# THE PRODUCTION OF PERINATAL HEALTH IN SCOTLAND

by

John Franklin Forbes

A thesis submitted in fulfillment of the
requirements for the degree of
Doctor of Philosophy

Department of Social and Economic Research
Faculty of Social Sciences
University of Glasgow

January 1989



ProQuest LLC.
789 East Eisenhower Parkway
P.O. Box 1346
Ann Arbor, MI 48106 – 1346

CONTENTS

## ACKNOWLEDGEMENTS

My greatest debt is to my supervisor, Professor Laurie Hunter who provided insight, advice and encouragement throughout the course of my research. I would also like to thank the innumerable medical friends and colleagues who have improved my understanding of the health and health care of the newborn. My former colleagues in the Health Economics Research Unit at the University of Aberdeen and the Social Paediatric and Obstetric Research Unit at the University of Glasgow have also contributed in a variety of ways.

The econometrics in this thesis is a reflection of the ease in which estimation and testing of economic time series models can now be accomplished thanks to the path breaking work of Professor David Hendry at Nuffield College, Oxford. All of the results were generated using PC-GIVE, a suite of interactive microcomputer based programs kindly made available by Professor Hendry.

Most of the early work on this thesis began when I enjoyed the hospitality of two research institutes. Professor Victor Fuchs at the National Bureau of Economic Research (Stanford University) and Professor Leif Bakketieg and Dr. Karl-Erik Larssen at the Norwegian Institute of Hospital Research (University of Trondheim) gave me the opportunity to reflect on my Scottish topic from welcome vantage points.

Staff at the libraries of the Universities of Bristol, Glasgow and Edinburgh, the Royal College of Physicians in Glasgow, the Mitchell Library in Glasgow and the National Library in Edinburgh have also helped enormously throughout my research.

Finally, Christine Martyn not only prepared the bibliography but tolerated a project whose length greatly surpassed the normal period of gestation attained by the hundreds of thousands of Scottish newborns whose earliest experiences provided the subject matter for this thesis.

## SUMMARY

This thesis presents an economic perspective on the determinants of perinatal mortality in post-war Scotland. Unlike previous work which has used cross-sectional data, the focus is on developing and testing models which may provide a more informed explanation for the improvement in perinatal mortality over time.

The theory of investment in human capital and the application of this paradigm to the study of health and health related behaviour is surveyed as a way of developing a more precise taxonomy for identifying the principal sources of change in Scottish perinatal mortality. Empirical studies of health during the earliest stages of the life cycle which employ the human capital theoretical framework are reviewed.

Post-war trends in Scottish perinatal mortality are investigated using a time series of dis-aggregated perinatal mortality rates that have been constructed especially for use in the estimates of the health production functions presented in this thesis.

A model is also developed for describing the post-war pattern of public expenditure on perinatal hospital services in Scotland. A perinatal hospital service demand equation is specified in terms of the share of NHS hospital expenditure devoted to the perinatal hospital sector which is

related to real NHS hospital expenditure (income), relative hospital prices and policy targets and indicators of the need for perinatal services. Total public spending on hospital care appears to have had no consistent and clear impact on the perinatal hospital budget share. Relative prices or costs of perinatal care in relation to other forms of hospital care are important determinants of expenditure. A partial adjustment model suggested that virtually all the adjustment required to achieve desired perinatal budget shares occurs within five years. An error correction model also supported the hypothesis that disequilibirium between target and actual perinatal shares was an important determinant of the change in budget shares over time.

The health production function estimates quantify the relative impact of medical and nursing labour inputs, hospital capital inputs, hospital expenditure and population factors on perinatal mortality. A variety of functional forms are tested against each other using a general to specific approach to estimation and testing. Alternative ways of augmenting the production function by introducing empirical measures of technical change are investigated. Production functions are presented for a range of mortality rates in different age and birthweight groups.

The results confirmed that hospital medical and nursing inputs are important determinants of perinatal mortality across different birthweight strata. Increases in the num-

ber of obstetricians and nurses have a beneficial and similar impact on mortality in low birthweight infants. Nurses also appear to have a more important impact on mortality in the normal weight infant. Decreases in the bed stock are associated with improvements in perinatal mortality, particularly for low birthweight infants. In terms of cost per life year gained, investment in perinatal hospital services has generated health outcomes at a cost which is lower than many other health care programmes whose costs and effectiveness have been evaluated from an economic perspective.

Finally, the possible links between income, unemployment and infant health are investigated. The main findings failed to support the hypothesis that unemployment excerts an impact on perinatal and post-neonatal mortality, let alone an adverse impact. In addition, measures of economic instability (short and long-run changes in economic growth) seem to play little role in determining the chances of survival of new born children.

# CHAPTER 1 INTRODUCTION

The modern revival of economic research in the field of population which has occurred over the past 30 years represents a return to a traditional area of inquiry in political economy. The Malthusian theory of population was an integral though controversial part of classical economic theory which established a framework for the economic analysis of demographic phenomena. Paradoxically, however, considering the importance accorded by Marshall, Pareto and particularly Wicksell to population theory, economic analysis of population questions declined substantially and become increasingly preoccupied with a narrow range of problems for well over five decades following the ascendancy of neoclassical economics.

Although there were a few empirical studies on, for example, the influence of the trade cycle on marriage and fertility rates (Thomas, 1927), economists generally restricted their analysis to the economic consequences associated with demographic change. Analysis of the causes of population change was generally left to demographers, sociologists and statisticians; a distinction emphasised by Peacock (1954) in one of the first attempts to interpret Malthusian theory in terms of modern economic analysis. Although Peacock acknowledged that fertility decisions involved economic choices regarding the allocation of scarce resources and the oppor-

tunity costs of children, the primary interest of the economist was in the consequences of population change (Peacock, 1954).

This thesis presents an economic perspective on the determinants of perinatal mortality in post-war Scotland. The health and well being of infants has long provided a fertile area of inquiry for epidemiologists, demographers, sociologists and others interested in the public health of populations. There have been, however, relatively few attempts by economists to examine the determinants of perinatal health despite the proliferation of research in health economics and investment in human capital which has occurred over the past 25 years. Unlike previous work which has used cross-sectional data, the focus is on developing and testing models which may provide a more informed explanation for the improvement in perinatal mortality over time.

Whether conducted in an economic framework or not, studies of perinatal mortality traditionally distinguish possible sources of change in perinatal health outcomes. Broad distinctions are usually drawn between socio-economic, biological, demographic, environmental and health care determinants of mortality differences, at a point in time, or mortality changes over time. These distinctions reflect the longstanding concern, expressed most prominently by Thomas McKeown (1976) that the contribution of health care to improvements in mortality should be evaluated alongside

other concurrent changes occurring in the population itself, which may themselves be more important determinants of mortality change over time. McKeown's basic thesis that health is only marginally influenced by personal medical care has yet to be rigorously tested using data for infants, particularly during the post-war period (Reves, 1985).

This scepticism regarding the impact of innovations in health care has been shared by many observers of recent trends in perinatal care and perinatal mortality and those who are anxious to subject clinical practice in obstetrics and paediatrics to more rigorous clinical evaluation and assessment (Chalmers and Sinclair, 1985; Silverman, 1980). This concern is also a characteristic feature of epidemiological studies that attempt to assess the relative contribution of changes in inherent population risks and specific medical interventions to declining perinatal and neonatal mortality (Williams and Chen, 1982; Forbes, *et al*, 1982; David and Siegel, 1983).

These epidemiological studies can be contrasted with the economic approach to explaining population change that is surveyed in Chapter 2. Although the same set of variables is often examined by the economist and epidemiologist: the economic perspective imposes a framework for guiding the subsequent empirical analysis and interpretation of find-

ings. The distinctive feature of the economic approach is its emphasis on constrained choice which imposes a structure on subsequent analysis which is typically absent in empirical studies conducted within a different disciplinary framework. The theory of investment in human capital and the application of this paradigm to the study of health and health related behaviour is surveyed as a way of developing a more precise taxonomy for identifying the principal sources of change in Scottish perinatal mortality during the post-war period.

Although the Chicago/Columbia approach to household production has dominated empirical studies of infant health conducted by economists, this relatively small and slowly growing literature is dwarfed by decades of work by clinicians, epidemiologists, demographers, social scientists and others who have examined various aspects of infant health using different or less theoretical perspectives. Empirical studies of health during the earliest stages of the life cycle which employ the human capital theoretical framework are reviewed in Chapter 3. The primary focus is on studies which have either examined outcomes of pregnancy during the perinatal period (the immediate period surrounding birth) or investigated the determinants of health in infancy or between the period from birth to one year of age. Many of the methodological features of these studies are also common in investigations of the health of older children and the utilisation of paediatric care.

One of the limitations of many empirical studies of perinatal health is the reliance on relatively crude measures of mortality as output variables in production functions. Building on the work of Sir Dugald Baird in Aberdeen during the 1940's, numerous studies have described different aspects of perinatal mortality at different points of time in the Scottish population. Although some recent studies have examined trends in Scottish perinatal mortality during the 1970's (Forbes *et al*, 1982) and the impact of demographic and social factors on the risks of perinatal mortality over the period 1960-1982 (Forbes and Pickering, 1985) no overall review of Scottish perinatal mortality during the post-war period is available. Furthermore, no dis-aggregated series of perinatal mortality rates has been assembled for post-war Scotland which would allow an assessment of the differential impact of social and medical factors on the mortality experience of different population sub-groups.

Chapter 4 examines post-war trends in Scottish perinatal mortality and reports a time series of dis-aggregated perinatal mortality rates that have been constructed especially for use in the estimates of the health production functions presented in Chapters 6 and 7. Perinatal mortality rates are sub-divided according to specific cause of death. Neonatal mortality rates are dis-aggregated by age at death. Perinatal mortality rates are also calculated for

different birthweight groups. Using a variety of published and unpublished data a unique time series of birthweight specific perinatal mortality rates is assembled and used to examine how the relative risk of perinatal mortality has changed in different birthweight groups over time. Each specification of the dependent variable permits the estimation of a set of health production equations which may provide further insight into the determinants of different components of perinatal mortality.

Chapter 5 presents the results of estimating a demand equation for perinatal hospital services in Scotland. Using a version of the demand model introduced by Deaton and Muellbauer (1980) the perinatal hospital service demand equation is specified in terms of the share of NHS hospital expenditure devoted to the perinatal hospital sector which is related to real NHS hospital expenditure (income), relative hospital prices and policy targets and indicators of the need for perinatal services.

This specification attempts to explain the variance of the perinatal budget share rather than the level of expenditure on perinatal hospital services. The construction of a perinatal budget share requires data on perinatal hospital expenditure, total hospital expenditure and a relevant price deflator for NHS expenditure. The derivation and measurement of a unique expenditure series on perinatal hospital expenditure and NHS hospital expenditure in Scot-

land over the period 1951-1985 is described. Although the demand for perinatal hospital services is inevitably constrained by the overall level of hospital expenditure it seems unnecessarily restrictive to assume that demand depends only on income or total expenditure. The concept and measurement of relative prices in the hospital sector and their role in the demand equation is examined.

In addition to income and prices, the demand for hospital care may be influenced by evolving policy objectives and perceptions of the need for such services. Chapter 5 also outlines the Scottish evolution of hospital bed supply targets, expenditure norms and population need indicators for perinatal care and illustrates how they can be introduced into the demand equation.

A wide range of specifications have been used to directly estimate health production functions. One of the characteristic features of this literature is the virtual absence of any critical testing of alternative functional forms or models which could be used to summarise the relationship between inputs and health outputs. Burdened by the inertia of previous studies investigators have often simply assumed the existence of a particular function and an arbitrary set of regressors which is imposed on the data with little concern for the coherence and validity of their assumptions.

A second feature of the literature is its preoccupation with estimating static cross sectional production functions. Few studies have used time series data to examine the impact of changes over time in health care resources on mortality and other health outcomes. Despite the recognition of the impact of technical change and innovation on both the process and outcomes of health care this dynamic process is not usually captured in the traditional cross sectional approach. Consequently the investigation of the dynamics surrounding health production functions is in its infancy.

Both of these themes are addressed in the health production function estimates presented in Chapter 6 which attempts to quantify the relative impact of medical and nursing labour inputs, hospital capital inputs, hospital expenditure and population factors on perinatal mortality. A variety of functional forms are tested against each other using a general to specific approach to estimation and testing. Alternative ways of augmenting the production function by introducing empirical measures of technical change are investigated. Production functions are presented for a range of mortality rates in different age and birthweight groups. One additional feature of the health production analyses is the calculation of the cost per life year gained based on the regression results.

Over the past decade there has occurred a virtual explosion in studies examining the impact of economic factors on health. Although this topic had attracted the attention of researchers for well over a century the initial spark which re-kindled the most recent surge of interest was struck by the work of Harvey Brenner, who published a series of controversial time series studies in the late 1970's linking mortality with unemployment. Brenner's contribution was soon followed by growing concern over the implications of the economic recession of the early 1980's for health care programmes financed by government spending as well as the health consequencies (if any) arising from cuts in such programmes.

One major strand of this literature concerns the influence of fluctuations in economic activity on mortality. Two indices of economic activity which have received particular attention are income and unemployment. Chapter 7 examines the association between income, unemployment and perinatal and infant mortality in Scotland. Unlike previous studies, the relationship between unemployment and mortality is modelled using a general autoregressive distributed lag framework in conjunction with an unemployment series disaggregated by age and duration of unemployment.

# CHAPTER 2 ECONOMIC PERSPECTIVES ON PERINATAL HEALTH

## 2.1 Introduction

The health and well being of infants has long provided a fertile area of inquiry for epidemiologists, demographers, sociologists and others interested in the public health of populations. There have been, however, relatively few attempts by economists to examine the determinants of perinatal health despite the proliferation of research in health economics and investment in human capital which has occurred over the past 25 years. The modern revival of economic research in the field of population which has occurred over the past 30 years represents a return to a traditional area of inquiry in political economy. The Malthusian theory of population was an integral though controversial part of classical economic theory which established a framework for the economic analysis of demographic phenomena. Paradoxically, however, considering the importance accorded by Marshall, Pareto and particularly Wicksell to population theory, economic analysis of population questions declined substantially and become increasingly preoccupied with a narrow range of problems for well over five decades following the ascendancy of neoclassical economics.

Although there were a few empirical studies on, for example, the influence of the trade cycle on marriage and fertility rates (Thomas, 1927), economists generally restricted their analysis to the economic consequences associated with demographic change.[1] Analysis of the causes of population change was generally left to demographers, sociologists and statisticians; a distinction emphasised by Peacock (1954) in one of the first attempts to interpret Malthusian theory in terms of modern economic analysis. Although Peacock acknowledged that fertility decisions involved economic choices regarding the allocation of scarce resources and the opportunity costs of children, the primary interest of the economist was in the consequences of population change (Peacock, 1954).

---

1. The treatment of demographic phenomena as exogenous factors within the framework of orthodox economic analysis is well illustrated by the secular stagnation thesis advanced during the 1930s (Robbins, 1929; Keynes, 1937; Hansen, 1939) which examined the effects of stationary or declining population growth on investment, employment and economic growth. This pre-occupation with the effects of population changes can also be seen in the Neo-Malthusian concerns with the consequences of rapidly increasing population in developing economies (Coale and Hoover, 1958)

In this chapter an economic perspective on the determinants of health is presented to provide a framework for developing an economic analysis of the factors influencing perinatal mortality in Scotland. The theory of investment in human capital and Grossman's application of this paradigm to the study of health and health related behaviour is briefly outlined. General criticisms of Grossman's approach are reviewed and the extension of this model to perinatal health is discussed.

## 2.2 Sources of Change in Perinatal Mortality

Whether conducted in an economic framework or not, studies of perinatal mortality traditionally distinguish possible sources of change in perinatal health outcomes. Broad distinctions are usually drawn between socio-economic, biological, demographic, environmental and health care determinants of mortality differences, at a point in time, or mortality changes over time. These distinctions reflect the longstanding concern, expressed most prominently by McKeown (1976) that the contribution of health care to improvements in mortality should be evaluated alongside other concurrent changes occurring in the population itself, which may, in fact, be more important determinants of mortality change over time. McKeown's basic thesis that health is only

marginally influenced by personal medical care has yet to be rigorously tested using data for infants, particularly during the post-war period (Reves, 1985).

This scepticism regarding the impact of innovations in health care has been shared by many observers of recent trends in perinatal care and perinatal mortality and those who are anxious to subject clinical practice in obstetrics and paediatrics to more rigorous clinical evaluation and assessment (Chalmers and Sinclair, 1985; Silverman, 1980). This concern is also a characteristic feature of epidemiological studies that attempt to assess the relative contribution of changes in inherent population risks and specific medical interventions to declining perinatal and neonatal mortality (Williams and Chen, 1982; Forbes, *et al*, 1982; David and Siegel, 1983).

These epidemiological studies can be contrasted with the economic approach to explaining population change. Although the same set of variables is often examined by the economist and epidemiologist: the economic perspective imposes a framework for guiding the subsequent empirical analysis and interpretation of findings. The distinctive feature of the economic

approach is its emphasis on constrained choice. Fuchs (1974; 1983) adopting Lionel Robbins' definition of economics[2] summarises the distinctive assumptions of the economic perspective as

- resources are scarce relative to human wants
- resources have alternative uses
- individuals have diverse wants, not all of which can be satisfied

This emphasis on constrained choice imposes a structure on subsequent analysis which is typically absent in empirical studies conducted within a different disciplinary framework. The implications of adopting an economic perspective are illustrated by Schultz (1982) who suggested that sources of mortality change can be usefully classified into two main groups:

- those originating in the public health sector.

---

2. "Economics is a science which studies human behaviour as a relationship between [a given hierarchy] of ends and scarce means which have alternative uses" (Robbins, 1932).

■ those arising out of the market system

This broad classification can be extended and refined into a more precise taxonomy for identifying the principal sources of change in Scottish perinatal mortality during the post-war period. The vast array of variables confronting the researcher can be partitioned into those which are modelled (endogenous) and those which are not modelled (exogenous).[3] Health outcomes are naturally treated as modelled variables though not necessarily as the primary or ultimate object of choice. Other modelled variables include the consumption of goods and services; some of which may influence the production of health. The demand for goods/services and the production of health outcomes are constrained by prices and economic endowments[4] arising out of the

---

3. The definition of exogenous and endogenous will depend on the level of aggregation (individual versus community decisions).

4. Economic endowments are often measured in terms of current or lifetime (permanent) income or wealth. This conventional treatment has been challenged by Becker (1965, 1981) who argues that money income should not be regarded as "given" or exogenous since it is (partly) determined by individual choices regarding investment in human capital and the allocation of time between market and non-market activities.

market system. A further set of biological supply constraints or health endowments will also influence the production of health and the demand for health related goods and services. These will take the form of the genetic inhereitance passed on by parents to their children as well as other intrinsic biological factors which determine perinatal health.

This classification can be modified further by breaking factors down into those which are observed and unobserved by the researcher or investigator. For example, health endowments and other biological characteristics of parents and their children are typically not observed by the researcher. Individuals and populations themselves may not even be aware of certain elements of the vector of their own health endowments. This complicates the analysis of mortality determinants because the observed consumption of health related goods is likely to depend on the unobserved health endowments. Since the unobserved factors will be subsumed into the error term OLS estimates of the effects of health inputs on health outcomes will be biased and inconsistent. If health outcomes were independent of health endowments and/or the use of health inputs was independent of health endowments the bias would disappear. Since this scenario seems unlikely

compensating variations in health input use in response to corresponding variation in health endowments will obscure the effects of health inputs on health outcomes.

The bias will depend both on the influence which health endowments have on the use of health inputs and on the impact of health endowments on subsequent health outcomes. The direction of bias can be estimated using the following formula. Consider a simple misspecified regression of perinatal mortality rates (PNM) on expenditure on perinatal hospital care (E) given by

$$PNM = \beta_1 E + u \qquad (2.1)$$

Assuming the "true" equation also includes a measure of health endowments (H)

$$PNM = \beta_1 E + \beta_2 H + u \qquad (2.2)$$

The estimate of $\beta_1$ from equation 2.1 is given by

$$E(\beta) = \beta_1 + b_{21}\beta_2 \qquad (2.3)$$

where $b_{21}$ is the regression coefficient from a regression of health endowments on perinatal mortality and $\beta_2$ is the coefficient of the health endowments variable obtained from equation 2.2. The expected signs of the coefficients are $\beta_1 < 0$, $\beta_2 < 0$ and $b_{21} < 0$. As

$b_{21}\beta_2 > 0$ the direction of the bias will be positive. This positive bias will thus ensure that any beneficial impact of expenditure on perinatal mortality is underestimated. It is also possible for the bias to swamp the negative sign on $\beta_1$ thereby generating the counter-intuitive result that additional expenditure on perinatal care increases perinatal morbidity and mortality.[5]

## 2.3 Public Health Sector

From an economic perspective, the public health sector can influence the risk of perinatal mortality in two principal ways. First, some public health programmes may reduce the risk of exposure to disease for the entire community or population. This type of programme, exhibits the typical characteristics of a public good in so far as benefits are conferred on the

---

5. This is not to deny that some forms of care inadvertantly generate adverse health effects. One of the best known examples in the perinatal field is the unintended consequences of oxygen administration to premature infants in the early 1950's which influenced the risks of neonatal survival and the incidence of retrolental fibroplasia (Silverman, 1980). The same may be true of more recent perinatal interventions whose effectiveness is unknown..

community which are largely indivisible and non-excludable. Perhaps the best example of a public health activity which falls into this category is the quality control of water supplies. Other types of activities include effective immunisation strategies designed to control or eradicate infectious diseases and the control of radiation, toxins and other environmental hazards which can influence fetal and infant health. These public health programmes may also vary in terms of their degree of publicness and the extent to which they are independent of or exogenous to individual behaviour or decision making. At one extreme a policy may exhibit both the characteristics of a pure public good (radiation control) (Fuchs, 1980) and exert an independent influence on the risk of illness. Alternatively, the benefits of some programmes may be excludable and divisible amongst members of the community and/or may depend on decisions taken by individuals or households. For example, despite their potential for generating community wide benefits, immunisation campaigns rely on the participation of individuals if they are to succeed. Risks to the fetus and newborn, originating in the workplace, may require occupational health policies whose benefits may be confined to workers in particular industries or occupations.[6]

---

6. Occupational health policies designed to limit reproduction

Most activities in the public health sector, however, are of a different nature and fail to conform to the public good paradigm. Policies which control the content, organisation, supply and distribution of health services may influence infant health outcomes in two ways: either by subsidising the cost of services or by altering quantity constraints or ration levels facing individuals or groups. Schultz (1982) focuses exclusively on public health activities that subsidise the cost of health care. This orientation is less relevant in countries like the U.K. where health services are predominately financed, organised and provided using NHS-type or non-market institutions. While it is possible to regard direct public provision of health care as a form of non-market subsidy, it is important to recognise that the demand for and utilisation of health care by individuals (or their agents) is subject to quantity or rationing constraints imposed or conditioned by public policies.

---

hazards in the workplace also entail significant costs when employers adopt employment discrimination practices which threaten the jobs or earnings of fertile women. See Bayer (1982) for a discussion of the implications of fetal protection policies for the employment rights of women.

While exogenous to individuals, health care policies are endogenous when viewed from a community perspective. The nature and extent of cost subsidies or quantity constraints in health care reflect community preferences or, perhaps more realistically, the preferences of decision makers responsible for determining:

- overall levels of public expenditure, and

- the allocation of public expenditure to (and within) competing public programmes, of which health care is only one.

Public expenditure decisions on perinatal health services can be viewed as analogous to consumer choices in the household sector in so far as both the private and public sectors are assumed to behave as though they maximise some objective function subject to a budget constraint. The government's expressed demand for perinatal health services, derived from an underlying public sector demand for the commodity "perinatal health", is influenced by relative programme costs (prices), population indicators of need, the deviation of perinatal mortality from target levels and total public expenditure on health services. This approach also encorporates the Becker-Lancaster distinction between "goods" and "fundamental

commodities". Thus, the public sector commodity, perinatal health, is distinguished from a public sector good, the supply of perinatal health services.

This approach also recognises the influence which the household sector may have on the public health sector. The principal links are the quantity and quality of births, past (and expected) rates of perinatal mortality and other perinatal health outcomes such as low birthweight. The total number of births will influence the government's perception of the need for perinatal health services (demand side). Another indicator of need, entering in on the demand side, is the perceived quality of births as measured by, say, the maternal age, parity and social class distribution of births and other factors associated with differential risks of mortality. The distribution of risks inherent in the newborn population may also enter the supply (cost) side by increasing or decreasing the costs of achieving a target level of perinatal mortality. Thus, a change in the quality of births may shift both the demand and supply schedules for public health sector expenditure on perinatal care.

The notion of a desired or long run level of perinatal mortality can be introduced as one of the determinants of public expenditure on perinatal health care. Past and expected rates of perinatal mortality provide a

basis for comparing target and actual values of perinatal mortality. Stimulus for expenditure and programme innovation can be captured by the size and sign of the difference between some pre-determined target value and the actual rate of perinatal mortality. Both target and actual rates will change over time. Targets may relate to some hypothetical ideal rate[7] or to population rates observed in regions or countries[8] which have attained or maintained lower rates of fetal loss and mortality during early infancy. Mortality rates, dis-aggregated according to different causes or times of death, could also provoke different public health sector responses depending on the perceived deviation of cause specific mortality rates from target values based on the pattern of cause or age specific mortality in populations. One example of such targets is encapsulated in the notion of avoidable perinatal deaths popularised

---

7. For example, in the 1940s Baird (1947) suggested that the Scottish perinatal mortality rate could be reduced to 10 per 1000: a target that was not achieved until 1985.

8. Throughout the post-war period international comparisons of perinatal mortality have repeatedly demonstrated that mortality rates in the U.K. are higher and have fallen more slowly compared with the rates observed in Sweden and other industrialised countries.

by the House of Commons Social Services Committee Report on Perinatal and Neonatal Mortality published in 1980.[9] The report suggested that

"About one-third to one-half of the (perinatal) deaths are preventable, if modern knowledge and care were universally applied. This amounts to at least 3,000-5,000 avoidable baby deaths a year in England and Wales" (para 522.1-522.4)

Despite the reluctance of the Government to endorse what it regarded as an unrealistic speculation the Social Services Committee's most recent report on Perinatal, Neonatal and Infant Mortality issued in 1988 has re-stated the concept of avoidable deaths in terms of target rates of reduction in perinatal and infant mortality which it set out for the English Regional Health Authorities. The Committee also recommended that target rates should be reflected in the resource allocations for health authorities, involving if necessary, the earmarking of additional resources for maternal and neonatal by the Department of Health to enable health authorities to achieve their targets by 1993.

---

9. Second Report from the Social Services Committee, Perinatal and Neonatal Mortality, HC 663, 1979-80.

## 2.4 The Market System

### 2.4.1 Price Effects

Sources of mortality change originating in the market system can take a variety of forms. Patterns of consumption and the allocation of time between market and non-market activities are influenced by market prices. Relative prices influence adult consumption of tobacco, alcohol and other goods prior to or during the perinatal period which have a potentially adverse effect on health outcomes. Likewise, many public interventions in the perinatal health care market are directed towards subsidising or removing the prices which consumers face when utilising antenatal, intrapartum or post-partum care.

Another example of price effects is the impact of real female wage increases on life cycle decisions regarding female labour supply, marriage and fertility patterns. Building on the influential work of Leibenstein (1957), Becker (1960) and Mincer (1963) a micro-economic framework for analysing demographic phenomena has evolved which emphasises the role which socio-economic factors play in influencing the demand for children.[10]

---

10. See Blake (1968) for an early critical review of the economic

Within a time series context, real wage growth has emerged as a (partial) explanation for the post-war increase in labour force participation and corresponding changes in fertility.[11] Current fertility is (partly) determined by female wages as well as by lagged fertility, lagged female labour force participation and male wages (earnings). In fertility equations female wages are used as a proxy for the opportunity costs of children. The net effect of changes in female wages on fertility will depend on the usual substitution and income effects. As female wages and the opportunity cost of a woman's time increase the demand for leisure and children should decrease. This negative substitution effect will be accompanied by a positive income effect

---

analysis of fertility where the demand for children is treated as the demand for a consumer durable.

11. Following Mincer (1963) fertility and female labour supply are often treated as joint consumer-demand choices conditioned by prices (Conger and Campbell, 1978; Moffit, 1984; Sprague, 1988). This joint treatment of two closely inter-related variables overcomes many of the specification problems inherent in num erous labour supply studies which include fertility variables as regressors in labour supply equations and fertility studies which traditionally include participation rates in fertility equations

unless leisure and children are inferior goods. If, as most studies have found, substitution effects dominate an increase in female wages will be associated with a decline in fertility.

Standard labour supply equations employed in aggregate time series studies typically specify female labour supply as a function of female wages, past fertility, male wages (earnings) and male unemployment (O'Neil, 1981; Smith and Ward, 1985; Riboud, 1985).[12] The supply of female labour is assumed to be directly related to female wage increases. Increases in female labour force participation (or hours worked) may have an indirect impact on perinatal health outcomes by increasing the exposure of the fetus to health risks in the workplace. However, the linkage between adverse perinatal health outcomes and female employment is anything but transparent (Saurel-Cubizolles and Kaminski, 1986).

Throughout the corpus of work on the economic analysis of fertility emphasis is placed on explaining the numbers, timing or spacing of births. Whether any or all

---

12. A more complete specification would also treat female wage rates as endogenous using a wage-accumulation equation which relates the market wage to previous labour supply decisions, work experience and the stock of human capital (Mincer,1974).

of these quantitative indices of fertility are associated with perinatal outcomes depends on the strength of the relationship between the quantity and quality or characteristics of births which are known to influence perinatal health.

### 2.4.2 Income Effects

The impact of relative price changes on health related behaviour is also conditioned by increases/decreases in income or changes in the distribution of income over time. Changes in income may have a (dis) proportionate effect on the budget shares of total expenditure allocated to health related goods or services. Relative expenditure on health care, for example, may increase as a result of rising incomes. This may occur when individuals decide to devote a greater proportion of household income to health care or, in the case of more centralised decisions outwith the household, growth in real incomes spurs increased public expenditure on health care in accordance with rising community expectations regarding the level and distribution of public expenditure. International cross-sectional studies of the association between national income and health care expenditure have generally reported income elasticities greater than one (Kleiman, 1974; Newhouse, 1977; Leu, 1986. The classification of health care as a luxury good[13] based

on these estimated elasticities, however, should be tempered by the realisation that these aggregate level studies are bedeviled by a number of theoretical and empirical problems (Parkin, et al, 1987).[14]

Alternatively, higher incomes may engender life styles or patterns of consumption that have adverse consequences for health which may lead to increased expenditure on health care. While this positive and increasing relationship between income and expenditure on health care may hold in general, health care can not be classified as a luxury good over all income ranges, particularly in low income groups where the opposite

---

13. This refers to the technical definition of a luxury good based on the income elasticity of demand or the ratio of the marginal propensity to consume to the average propensity to consume and should not be confused with other more common defitions or notions of luxury, extravagance or indulgence.

14. The micro-foundations of these aggregate studies are usually suspect as is the consistency and reliability of data across countries with different systems of health care and national income accounting.

relationship may be true due to the deleterious effect of low income (poverty) on health and consequent need for and use of health services.

Changes in income (measured by male earnings) can also lead to changes in behaviour that may influence perinatal health outcomes. For decades, economists have argued in favour of a positive relationship between income and fertility.[15] Studies using post-war time series data generally support the hypothesis of a positive income effect (Butz and Ward, 1979; Ermish, 1983; Shields and Tray, 1986; Spraque, 1988). These results can be contrasted with other studies supporting the existence of a neagative relationship[16] as well as the long term decline in fertility which has occurred alongside economic growth. Following Becker (1960), economists have adopted a plausible, though problematic, explanation which relies on a trade-off between the quantity and quality of children. As the income effect on fertility is positive, assuming that children are not

---

15. See Thomas (1927) for an early study of aggregate economic activity and fertility supporting a positive relationship between fertility and income.

16. Negative income effects are usually found in cross-sectional and panel studies.

inferior goods, parents are assumed to react to increasing income by devoting more income to each child in an attempt to enhance child quality. Thus, an increase in income would lead to an increase in the demand for child quality at the expense of child quantity.[17]

As fertility patterns change so too may female labour supply which, in turn, may react to actual or expected unemployment of primary workers. For example, in addition to its adverse effect on household income, increases in male unemployment or decreases in male activity rates may influence female labour supply and fertility. Most studies of female labour supply find an inverse relationship between the labour force participation rate of married women and male unemployment. This "discouraged worker effect" is a consequence of the fact that the expected gains from

---

17. An auxiliary explanation forwarded by Becker for the inverse association between income and fertility hinged on the assumption that the effectiveness (or knowledge) of contraception increased with income. Hence, wealthier people were more able to control the "supply" of children. The inability of economists to concede that children are inferior goods has spawned a literature in its own right insisting on the "normality" of children.

female job search fall below the expected costs of obtaining a job. Increased competition and search duration by female re-entrants to the labour market may lead potential re-entrants to lower their expected future earnings and hence reduce their labour force participation rates with further adverse consequences for household income. Alternatively, in response to rising male unemployment rates female participation rates may increase as women attempt to maintain family income (added worker effect). The added worker and discouraged worker effects may occur both in households where males are currently unemployed and in households which may anticipate possible future unemployment of primary workers. The effect of male unemployment on birth outcomes is thus not confined to direct (family) income effects on patterns of household consumption but may operate indirectly via the impact on female labour supply and fertility.

## 2.5 "New" Home Economics

### 2.5.1 Investment in Perinatal Health Capital

The concept of human capital and the recognition that people may invest resources in themselves (or others) to enhance their education, skills, productivity, health or other characteristics has a long history in political economy. The modern theory of investment in human

capital, however, emerged with Schultz's (1960) work on education and economic growth and Becker's (1964) influential study of rates of return to education. These early studies, along with Becker's (1965) related work on the allocation of time and theory of household production and consumption, have stimulated a vast literature in labour economics, the economics of education and human resources and the economics of the household and family.[18]

In their early work, both Schultz and Becker acknowledged that investment in human capital could take the form of improvements in health which,in turn, generated a stream of consumption benefits (e.g., the direct utility arising from changes in health) and investment benefits (e.g., increases in productivity due to a healthier labour force). This conceptual separation was also apparent in empirical work, although virtually

---

18. However, despite encouraging and influencing analytical and empirical studies by economists on previously unexplored or "new" subjects, such as household formation, fertility and schooling, the theoretical propositions offered by the human capital approach have been largely ignored (and sometimes ridiculed) by disciplines such as demography whose traditional domain was invaded by the enthusiastic application of neoclassical economics to what was and is still regarded by some as beyond the outer limits of economic analysis.

all studies tended to focus on the impact of changes in health on the productive capacity of the economy. The evaluation of consumption benefits was relegated to a subordinate position reflecting a pre-occupation with the direct and more easily measured effects of ill-health on the economy or, as was characteristic of early cost-benefit studies of health programmes, the economic return arising from improvements in health.

### 2.5.2 Grossman's Model of the Demand for Health

Although elements of the human capital approach can be found in the econometric "health production" study of Austen *et al* (1969), the application of the human capital framework to health is primarily due to Grossman (1972) whose theoretical model of the demand for health has been used to examine a diverse range of health related behaviour. Grossman's main contribution was the development of a model, integrating the theory of household production with standard capital theory, which treats an individual's demand for medical care as a derived demand for a more fundamental commodity, namely health.[19] Individuals are assumed to inherit an initial stock of health which depreciates over time but may be

---

19. See Wagstaff (1986) for a non-technical introduction to Grossman's model.

augmented by investment in health. Such investment could occur in a variety of ways. Individuals may, for example, combine the use of medical services with inputs of their own time, modify their diet, engage in exercise or change other aspects of their behaviour regarded as either detrimental or beneficial to health. The stock of health at any point in time is also assumed to depend on exogenous environmental and biological factors, including initial genetic endowments. One of the most important environmental variables in Grossman's model is education which, following the household production paradigm, is hypothesised to influence the efficiency of productive activities in the household, one of which is investment in health.

Carefully distinguishing between the demand for health and the demand for medical care, Grossman utilised standard demand and capital investment theory to determine the optimal stock of health for individuals. Not surprisingly, given the assumptions of his model, the optimal stock is derived from setting the value of the marginal product of health capital equal to the user cost or supply price of health capital. The marginal benefits of gross investment in health capital must equal the marginal cost of such investment: a standard result in neoclasssical investment theory. The value of the marginal product of health capital will reflect both the investment (or production) aspects of improvements

in health and the consumption benefits of health represented by the direct impact of health on utility. This traditional dichotomy between the investment and consumption benefits of health is thus maintained by Grossman who concentrated on developing and testing an investment sub-model which ignored the consumption benefits arising from increases in the stock of health.

Having defined the optimal stock of health, Grossman investigated how the optimal stock should change over the life cycle in response to variations in depreciation rates, the influence of differences in age rates on the demand for health and medical care and the impact of education on both the production and demand for health. The principal predictions which emerge from his model are that:

- an increase in the rate of depreciation of health capital will raise the price of health capital and cause the demand for health to fall,

- an individual's demand for health and medical care should be positively associated with the wage rate,

- if education increases the efficiency of household production and gross investment in health, more educated individuals will demand a larger stock of health.

Various assumptions about the elasticity of the demand curve for health also enabled Grossman to make predictions about the impact of depreciation rates, wages and education on the demand for medical care.

### 2.5.3 Criticism of Grossman's Model

Grossman's model has dominated subsequent economics analyses of the demand for health and medical care and, to a much lesser extent, studies of the determinants of health. Despite its prominence there have been relatively few critical assessments of his theoretical model. Nor has there been a critical review of the empirical studies spawned by his model, in particular whether the testable predictions of this theory are confirmed or rejected by empirical evidence.

Most constructive criticism of his theoretical model takes the form of extensions to his original contribution. That is, the general theoretical framework is accepted but changed by relaxing one or more of the more unrealistic or unnecessary assumptions under-pinning his analysis. Most of the problems which have attracted attention, moreover, were initially acknowledged by Grossman himself. For example, despite Arrow's (1963) influential paper on the pervasive effects of uncertainty on the medical care market,

Grossman's model was formulated around the assumption that individuals possessed perfect information on the incidence of future illness and the marginal productivity of medical care, their own time and other market and non-market inputs on the production of health. This unrealistic assumption was later replaced by the more plausible assumption that individuals maximise expected utility.[20]

The reasonableness of Grossman's distinction and mutually exclusive interpretation of health benefits as either a pure investment of a pure consumption good has also been challenged. Usher (1975) questioned whether the motivation underlying an individual's demand for health and medical care is adequately captured by an investment sub-model which concentrates entirely on the monetary return (as measured by the wage rate) arising from marginal improvements in health. Muurinen's (1982)

---

20. Theoretical extensions of Grossman's model, encorporating the expected utility hypotheses, have been employed to analyse the joint demand for health insurance and preventive medicine (Nordquist and Wu, 1976; Phelps, 1978), the demand for prevention and the choice of occupations hazardous to health (Cropper, 1977) and the optimal consumption over the life cycle of goods (e.g. smoking, environmental pollutants) which may increase the probability of adverse health outcomes (Ippolito, 1981).

generalisation of Grossman's model also retains the consumption/investment distinction but treats each of these benefits as complements or joint products which are produced from increases in the stock of health.

Muurinen (1982) also examined the implications of relaxing two additional assumptions which Grossman acknowledged were limitations of his model. First, she allows for investment in health to occur without the use of medical care inputs. In Grossman's model, investment in health capital could only occur as a result of combining medical care with own time inputs. Although this is a common assumption in many production studies employing, for example a Cobb-Douglas production function where all inputs must be used in production, casual observations suggest that the non-use of medical care should not be interpreted as zero investment in health.

A second, more important, feature of her model is that she treats the health capital depreciation rate as dependent on a variety of decisions regarding health related behaviour (e.g. diet, smoking, exercise). This interpretation of use-related depreciation (again a concept borrowed from standard capital theory) enables her to abandon the household production paradigm which led Grossman to argue that education could only influence the production of health through its influence

on the technical efficiency of household production and consumption. Rather, she suggests that the role of education is more important in improving the choice of life style, etc., which in turn influences the rate at which the health capital stock depreciates and the extent of replacement investment which must occur to maintain the stock at a desired level. The concept of use-related depreciation also enables the analysis of additional factors, such as environmental variables, which at least at a conceptual level, can be treated as influencing rates of depreciation and thus raising the user cost of health capital, which could result in individuals demanding less health.

Grossman's hypothesis regarding the impact of education on health, based on no more than the assertion or belief that education enhances the efficiency of household production, has also attracted criticism which has led to alternative formulations or hypotheses which could account for the widely observed positive correlation between educational levels and health status. In addition to Muurinen's model which emphasises the 'allocative' as opposed to 'technical' effects of education, Fuchs (1982) explored the possible role played by differences in time preference as an explanation for the links between education and health. Fuchs argues that individual variation in time preference could explain both investment in education

and health. Individuals with low rates of time preference would tend to invest in more years of schooling and would also invest in health enhancing activities. Thus schooling would have no direct effect on health (or the efficiency of household production) since both schooling and health are jointly determined by rates of time preference. Fuchs also suggests that schooling could also affect rates of times preference, perhaps leading to situations where individuals with more education would be willing to engage in health investments with lower rates of return. Although Fuch's empirical study confirmed a correlation between schooling and time preferences and between time preference and health investment the true direction of causality was difficult to identify.

Working with the household production paradigm, Rosenzweig and Schultz (1981) also attempt to distinguish between the allocative and technical efficiency effects attributable to education in a study of the production of child health. Measuring child health in terms of birth outcomes (birthweight and birthweight standardised for gestational age) they found little empirical support for the hypothesis that mother's education augments child health, given her choice of health related behaviour and inputs. Rather, if education plays any role it seems to be that of improving the allocation of inputs.

Additional questions about Grossman's hypothesis that education improves the technical efficiency of investment in health concern the direction of causality between health and schooling. It could be that persons with better health endowments are more efficient in schooling activities. Poor health during childhood, for example, may limit later educational attainment. Other possible hypotheses linking education and health could build on the idea that common underlying variables could determine both education and health. Thus education may be associated with health simply because education is correlated with income, consumption patterns, life style, decision making ability, occupational health risks and other factors known to be associated with health.

A more fundamental problem with Grossman's model has risen as a by product of critiques levied at both the human capital approach (Blaug, 1976; 1980) and the household production model (Pollack and Wachter, 1975). The human capital research programme and, more specifically, the wide ranging contributions of Becker (1976, 1981) which provided the intellectual framework for Grossman's model of health, has been questioned by Ben-Porath (1982), Hannan (1982) and particularly Blaug (1976, 1980) who offers a methodological criticism of the human capital approach and models such as Grossman's

which represent one strand of the new home economics. Blaug's general criticisms about the intellectual imperialism of Becker, who seeks to extend traditional micro-economic theory into areas previously ignored or beyond the reach of economists yet fails to satisfy a number of methodological requirements which would enable the rigorous testing of his model's predictions and insights, can also be applied to Grossman's model and much of the empirical work based on it. Although not directed explicitly at Grossman, the inherent problems and limitations of the household production model discussed by Pollak and Wachter (1975) who argue that such models do not provide an appropriate nor logical framework for analysing activities involving joint production and Bockstael and McConnell (1983) who demonstrate that the household production function offers almost no testable hypotheses involving the production and consumption of 'basic' commodities (like health), raise serious doubts about the applicability of the household production framework in studies of health-related activities and decision-making. Further criticism of the household production approach has focused on the narrow neoclassical view of production which has been thrust upon the activities of households and families. By concentrating entirely on differences in prices and technologies as factors influencing the production of goods which families produce for their own consumption, Pollak (1985) argues that the household

production approach could be usefully supplemented and perhaps replaced by a transaction cost approach that recognised the importance of the internal organisation and structure of families and households.

Finally, the generality of Grossman's model, including its high degree of abstraction and its reliance on largely unobserved variables such as the demand for health, or the technology of household production, make it very difficult to assess whether any empirical evidence could be produced that would be inconsistent with his theory. The failure of the model to produce well specified empirical tests of its propositions has also led to the ad hoc inclusion or exclusion of variables, eccentric interpretations of results and to what Usher (1975) suggested was a body of empirical results which could probably stand regardless of the elaborate theoretical framework proposed by Grossman. If the basic insights offered by Grossman's model of the demand for health are no more illuminating (nor testable) than those generated by models of the demand for physical capital by firms where the desired level of capital is derived from equating the value of the marginal product of capital to the rental price of capital, it could be argued that although the theory may be applicable, its usefulness as way of furthering our understanding of the health related behaviour of individuals is questionable. Indeed, the technical

sophistication of the theoretical models proposed by Grossman and others do little more than provide a framework for clarifying thought and organising the structural relations which ideally should be estimated from available data.

### 2.5.4 Grossman's Model and Perinatal Health

Grossman's model, and subsequent theoretical extensions, have been largely formulated and tested using data on the demand for health and medical care by adults. The generality of the basic model, however, has led to several empirical studies in the field of child health which owe allegiance to his original contribution (Grossman and Jacobowitz, 1981; Corman and Grossman, 1985; Corman, Joyce and Grossman, 1987; Joyce 1987a; 1987b). No major changes or extensions to the model have emerged as result of applying the theory to the study of children's health. This probably reflects more the degree of abstraction which characterises the theoretical elaboration of his model rather than the evolution of the model in response to the particular characteristics of the demand for and production of children's health. Many of the empirical studies of child health, moreover, are more closely related to the economic models of fertility behaviour associated with either the Chicago/Columbia approach or that of Easterlin and his colleagues at the University of

Pennsylvania (Forbes, 1983). Considering the few testable predictions of Grossman's model it is important to consider the changes which the model must undergo if it is going to be applicable to the study of perinatal health.

First, the investment sub-model is not likely to be relevant in perinatal health studies. The marginal benefits of improvements in health are more sensibly regarded as consumption benefits enjoyed by the child's parents or caretakers. While it is true that investments in health during infancy and childhood may lead to financial or monetary returns later in life, this narrow measure of health benefits ignores what is arguably the more important direct impact on both the child's and the partners' utility derived from improvements in child health. This is not to deny that investment benefits, in the limited human capital sense, could occur but that, in relation to consumption benefits, they are likely to be less important for decisions regarding immediate investments in child health.[21]

---

21. One implication of this is that, given the absence of easily obtainable estimates of such consumption benefits, empirical studies will be constrained to use proxy measures of consumption benefits or forced to adopt the investment sub-model, which though of limited relevance, could be applied in much the same way as cost-benefit

A related feature of child health is that the principal decision-makers make decisions not about investment in their own health but but about the health of their children. When viewed from a family perspective the health of children can be regarded as public good whose benefits are conferred on both the immediate and (to a lesser degree) the extended family members. Encorporating this into models of health requires a framework for the interdependence of the utility functions of family members of auxiliary assumptions regarding the nature of the family's utility function or joint welfare function or the dominance of one family member's utility function which includes as arguments the levels of health and well-being enjoyed by other family members (Becker, 1981).

This latter approach, following the altruistic model of Becker (1981), tends to be employed in studies of child health. Inman (1976) for example, assumes that the decision maker for the family's demand for and production of child health is the mother who is concerned with both her and her family's direct consumption benefits associated with healthy children.

---

studies of child health programmes where the human capital approach to generate monetary values for health improvements is employed.

Other studies simply enter child health as arguments into their parent's utility functions and analyse the demand for child health as they would analyse the demand for a consumer durable which generates a stream of consumption benefits. The publicness of child health and its treatment in models of health related behaviour has yet to be fully examined. For example, the effect of differences in family composition and size on investment in child health could reflect the fact that single parent families will tend to invest in lower levels of child health since the benefits of such investment will extend to fewer individuals. Thus, while this lower level of investment is optimal for single parents, a similar level of investment for two parent families would be sub-optimal since the public nature of child health ensures that the sum of family benefits would be higher. If a higher level of child health were to be achieved in single-parent families the costs of investment in health capital faced by such families would have to subsidised.

## 2.6 Biological Supply Considerations

As it stands, the above taxonomy emphasises a demand side orientation to identifying possible sources of change in perinatal mortality and other reproductive outcomes. This approach, viewing child health as the consequence of household decisions constrained and

conditioned by prices and income, is consistent with the "Chicago-Columbian" economic model of fertility which treats birth outcomes as wholly demand determined. The usefulness of this demand side focus as a way of understanding fertility behaviour and outcomes, has been challenged by the emergence of competing, more general analytical approaches that recognise and explicitly encorporate the importance of exogenous supply side fertility determinants; many of which are biological in nature (Easterlin, Pollak and Wachter, 1980; Behrman and Wolfe, 1984).

Biological, as well as behavioural, factors are also important determinants of perinatal mortality. Just as household decisions are conditioned and constrained by prices and incomes, so too are household (community) allocative decisions and reproductive health outcomes conditioned and constrained by exogenous variation in biological supply factors. A variety of exogenous supply factors can be identified including women's health status (during and before pregnancy), previous reproductive outcomes and the genetic endowment inherited by the newborn.

Changes in the health and well being of the child bearing population over time may influence reproductive health outcomes. The health and physique of women has long been recognised as an important (pre-) determinant

of reproductive outcomes. Many perinatal health outcomes are associated with the health of women during their pregnancy. The relationship between the health of women and reproductive outcomes, however, is likely to be more complex and subtle. Pregnancy outcomes may be influenced by health both during and prior to pregnancy. Lags between past health and pregnancy outcomes may extend over a long time period and may even be linked over successive generations.[22]

Another biological constraint that may condition allocative behaviour and constrain the attainment of desired levels of perinatal health is a woman's past obstetric history. A variety of previous reproductive outcomes are closely linked to current reproductive outcomes. Longitudinal studies have demonstrated the likely repetition of low birthweight, pre-term delivery and intrauterine growth retardation in successive pregnancies in the same women. Since all of these factors influence the risks of perinatal mortality, a similar repetitive pattern has been established for perinatal deaths. The likelihood of maternal disease

---

22. Baird (1980), for example, suggests that the level of what he terms "reproductive efficiency" observed for a particular cohort of women may be a consequence of the level of health attained by that cohort during earlier stages of the life cycle, going as far back as infancy and even the period when the cohort was in utero.

occurring in pregnancy is also related to the occurrance of disease in preceding pregnancies. For example, the incidence pre-eclampsia (a toxaemia of late pregnancy) in second pregnancies is dependent on the pre-eclampsia state of the first pregnancy None of these studies, however, are able to distinguish whether the increased risk of adverse reproductive outcomes is related to the previous occurrance of these outcomes or is due to the predisposition of women to such outcomes. Nevertheless, irrespective of these competing explanations, the interdependence of pregnancy careers and outcomes represents an important, largely exogenous biological determinant of change in perinatal mortality.

A third biological supply factor that influences reproductive health outcomes is the genetic endowment of the fetus and newborn which may be inherited or de novo in origin. Recurrent fetal loss (spontaneous abortion), the incidence of certain congenital malformations (e.g., Downs Syndrome) and many other aspects of the health and development of the newborn are genetically determined. The genetic endowment of the fetus may act as a constraint on health during the perinatal period and also condition behaviour as genetic information (or the health outcome resulting from the genetic endowment) is detected during the pregnancy. For example, the pre-natal detection of chromosomal or neural tube defects is now commonly

followed by an induced abortion to prevent the birth of a child with a severe congenital malformation. This form of "supply-induced" demand may excert an influence on the observed perinatal mortality rate, depending on the population incidence of malformations, the associated risks of mortality in malformed fetuses and newborns and the behavioural reaction to newly acquired information during the pre-natal period.

Biological supply factors are not uniformly distributed in the population. The heterogenous and stochastic nature of exogenous supply factors may influence the demand for health and related allocative behaviour of decision-makers within and outwith the household. Women's past and present health status, obstetric history and the genetic endowment of the fetus and newborn are important supply-side signals which convey information that can lead to compensatory behaviour on the part of women and health care providers. Examples of such behaviour include the differential care and treatment offered to women with maternal disease, a history of perinatal loss or other characteristics who are classified as "high risk" cases and the impact of information on the health of the fetus acquired using electronic fetal monitoring on the management of labour and delievery. However,

whether compensatory behaviour or specific interventions conditioned by biological supply factors improve perinatal health is an empirical question.

## 2.7 Conclusions

This chapter surveyed the economic approach to the analysis of mortality determinants with particular reference to changes in perinatal survival in populations over time. An aggregate model which captures the important causes of the decline in perinatal mortality would distinguish the contribution of evolving population characteristics from that arising from changes in the nature of public health services provided for the care of the obstetric and newborn population. Ideally, interaction between the population and public health sectors would be allowed. The population or household sector relies on medical care inputs provided and rationed by the public health or government sector. The state influences income and other (e.g., information) constraints facing households. Unemployment, poor housing and other non-medical factors associated with adverse health outcomes may be consequences of government policies. Decisions taken by households will also play a role in influencing both the demand and supply of public health services. Fertility trends may shift the governments perceived need for maternity services. On the supply

side, the cost of attaining some target level of perinatal mortality may be influenced by changes in the level of risk inherent in the population.

The theoretical models based on the "new home economics" provide one description of individual behaviour. Blaug (1980) has described this approach, attempting to explain social phenomenon by tracing its roots back to individual behaviour, as that of "methodological individualism". This mode of economic analysis, beginning with the behaviour of individuals, has a long tradition in economics that can be traced back to Schumpeter. Throughout the human capital research programme, including that concerned with health, the emphasis is on testing the hypotheses of behavioural models constructed at the individual level. Consequently, little attention has been directed towards developing aggregate level models nor to investigating their possible micro-foundations. This is an important gap in the human capital literature since it is not readily apparent whether an individual level focus is the most appropriate or advantageous way of investigating collective behaviour or the ways in which households and governments may interact with each other in health, education and other public sectors.

Since no detailed specification of aggregate level models has yet emerged from the human capital research programme, previous studies conducted at the aggregate level use the human capital paradigm as the point of departure for empirical analyses in the spirit of previous work formulated at the micro-level. Thus aggregate studies of health, fertility behaviour and labour force participation tend to rely on the micro-level perspective to suggest explanatory variables that are subsequently included in empirical models.

The micro-based approach has undoubtedly improved the thinking (of economists at least) about health and population phenomena. However, it provides only limited information about the precise specification of aggregate models which allows for the dynamic, multi-variate nature of demographic change in populations. Perhaps inadvertantly because of an over-reliance on tenuous and generally unchallenged theoretical constructs the associated empirical analysis has tended to be overly restrictive. Models based on an edifice of untested restrictions can easily lead not only to less understanding but misunderstanding (Behrman and Wolfe, 1984). One thing though is certain. Despite their ambitions, economists like all other investigators from

other disciplines are unlikely to explain everything concerning the evolving pattern of health in newborn populations.

# CHAPTER 3 Empirical Studies of Perinatal Health

## 3.1 Introduction

Empirical studies of health during the earliest stages of the life cycle which employ the human capital theoretical framework are reviewed in this chapter. The primary focus is on studies which have either examined outcomes of pregnancy during the perinatal period (the immediate period surrounding birth) or investigated the determinants of health in infancy or between the period from birth to one year of age. Many of the features of these studies (i.e., methods, data limitations) are also common in investigations of the health of older children and the utilisation of paediatric care.

## 3.2 Theory and Empirical Models

Although the household production model has dominated emprical studies of infant health conducted by economists, this relatively small and slowly growing literature is dwarfed by decades of work by clinicians, epidemiologists, demographers,

social scientists and others who have examined various aspects of infant health using different or less theoretical perspectives.[1] However, despite their collective allegiance to the household production approach, most of the studies reviewed below closely resemble empirical studies which have traditionally employed little or no theoretical framework. This general feature of the literature echos the remarks of Usher (1975) who, commenting on an early study by Grossman, suggested that much if not all of the reported empirical results could stand independent of his elaborate theoretical model.

In principle, the theoretical models provide a context for structuring empirical studies and suggesting what variables could be important determinants of health. In practice, particularly in studies using aggregate data, they are rarely

---

1. Economists analyzing the determinants of health do not always use theoretical models to structure their empirical work. For example, see the series of studies of perinatal mortality conducted by Martin Feldstein: perhaps the most emminent economist ever to work in the field of health (Feldstein, 1965; Feldstein 1966; Feldstein and Butler, 1966).

used to define and estimate structural relationships between measurable variables as a way of establishing the causal influences on infant health or pregnancy outcomes. Furthermore, its use in formulating empirical models which convincingly test theoretical predictions and enable discrimination between competing theoretical models or alternative explanations for observed empirical regularities has been limited. This may reflect the embryonic development and consequent application of these models to infant health as well as the problems arising from the generality of the theoretical framework, a lack of competing theoretical models (economic or otherwise), the paucity of testable predictions concerning the health of infants and the formidable data requirements which must be satisfied to allow the estimation of structural relations between largely unobservable variables.

The lack of concordance between theoretical and empirical models of infant health is perhaps best illustrated by the fact that many studies fail both to distinguish between and explicitly model the simultaneous nature of the demand for infant health (or infant health care) and the production process which influences the supply curve of infant health. The distinctions between the demand

for health and the supply of health which is such a prominent feature of the theoretical framework is usually unclear and often ignored. For example, studies estimating the demand for child health inputs like medical care typically adopt a single equation approach to the estimation of demand functions which ignore the production of health. Likewise, studies which concentrate on estimating the technical relations between inputs and health outputs using the household production framework tend to ignore the simultaneous demand for health or incorporate information on prices and income into what Rosenzweig and Schultz (1983) refer to as "hybrid" health equations which are neither supply nor demand equations.

The general equation estimated using this "hybrid" approach takes the form of:

$$H = h(x,t,p,Y,u) \qquad (3.1)$$

where H is a measure of child health, x is a vector of market goods or services which may influence child health, t is parents' time input into the production of child health, p is a vector of exogenous prices, Y is a budget constraint and u represents family specific influences on child health, such as environmental or genetic factors,

which may be known to the family but are typically unobserved by researchers. Such equations have been employed to assess the effect of inputs such as medical care. Corresponding coefficients have been interpreted as though they represented marginal products derived from production functions. Rosenzweig and Schultz (1983) demonstrate that such hybrid equations provide biased estimates of the true technical relationship between an input like medical care and child health.[2] Since ordinary least squares estimates of equation (3.1) can be biased, two stage least squares (or other simultaneous equation methods) need to be employed in studies of infant health using the Grossman type distinction between the demand for and production of health. Increasingly, this two-stage approach is being adopted in empirical studies of infant health. The first application of this joint approach to estimation was that of Rosenzweig and Schultz (1983) who first estimate demand equations for health related inputs and then employ these

---

2. This bias essentially reflects the fact that the hybrid marginal products, estimated from an equation like (3.1), are influenced by both the technical properties of production and the families' preferences.

estimates to obtain second-stage estimates of birthweight and birthweight-for-gestational age production functions. This approach thus allows for the endogenous effect of pre-natal care, smoking and fertility on estimates of infant health production functions.[3]

An alternative approach, which recognises the interdependence of health production and input demand equations, was adopted by Inman (1976) in a study of ear, nose and throat (ENT) infections in children. Inman develops a model of family demand for preventive and curative health services in which health attribute production functions are integrated into specifications of the demand for health-related goods and services (including time inputs into the production of child health). Estimates of the average impact of curative doctor visits, preventive care and parents' time on ENT (as well as the variability or uncertainty of average effects) derived from estimating "hybrid" health production functions are used as

---

3. Studies which follow the lead of Rosenzweig and Schultz (1983) include Corman, Joyce and Grossman (1987) and Joyce (1987a, 1987b).

independent variables in the demand functions to test for the influence of differences in health care technology on the demand for health-related goods and services.

This two-part approach, however, beginning with production function estimates and then moving on to encorporating these estimates into demand functions simply introduces bias into the estimated demand functions arising from the hybrid nature of Inman's health production function which includes family income per person as inputs into the production of health. Thus, although Inman's intentions and explicit modelling of the interaction between the production of health and the demand for health are in the right direction, his model suffers from the bias demonstrated by Rosenzweig and Schultz (1983) in so far as both the average and variable effects of health care and parents' time on child health will be biased since they reflect both the technological properties of the health production process and the families' preference orderings.

### 3.3 Measuring Health Outcomes

Studies of the determinants of health in infancy have employed two different types of dependent variables as measures of perinatal and infant health. First, death rates have been used. This reflects a traditional approach to measuring health during the perinatal period and throughout the first year of life. Although death is a finite event whose measurement is less difficult, compared to other measures of illness and health, the interpretation of death rates, particularly during the perinatal period, can be problematic.[4]

---

4. Definitions of the perinatal period may differ, depending on the criteria used to distinguish late fetal deaths from spontaneous abortions and whether only first week deaths or all neonatal deaths during the first four weeks following birth are regarded as perinatal deaths. Definitions may also vary if the population at risk includes all births (irrespective of birthweight, gestation or other infant characteristics) or only those infants born, for instance, after a specific gestational age or weighing more than some arbitrary birthweight. The perinatal period in the U.S., for example, is the period from twenty weeks gestation up to seven days of life, following birth, whereas in the United Kingdom the lower age limit is 28 weeks gestation.

Various mortality rates have been employed as dependent variables in infant health production function studies.[5] Perinatal mortality rates for hospitals were investigated by Williams (1979) and the impact of prenatal care on the probability of survival during the perinatal period was examined by Harris (1982). Neonatal mortality has been frequently employed (Williams, 1975; Grossman and Jacobowitz, 1981; Goldman and Grossman, 1982; Hadley, 1982; Harris, 1982; Corman and Grossman, 1985; Corman, Joyce and Grossman 1987; Joyce, 1987a, 1987b). Post-neonatal mortality was analysed by Goldman and Grossman (1982) and Hadley (1982). Finally, infant mortality was investigated by Goldman and Grossman (1982), Hadley (1982) and Rosenzweig and Schultz (1983).

Although several of these studies examine sex and race specific mortality rates, no further adjustments to crude mortality rates tend to be made in these studies. The usefulness and interpretation of crude overall mortality rates in health production studies is questionable. First,

---

5. Definitions of perinatal, stillbirth, neonatal, post-neonatal and infant mortality rates are presented in a Glossary at the end of this Chapter.

unlike most age-specific mortality rates, perinatal, neonatal and post-neonatal mortality rates are defined over an age range in which the risk of death varies enormously. For example, pre-term infants born before 37 weeks gestation face a risk of mortality some 120-170 times greater than the risk faced by full term infants born at 38-42 weeks. Likewise, the risk of death during the first 24 hours following birth is much greater than at any time during the first year of life.

A more fundamental problem in using overall mortality rates in health production studies is the powerful impact of birthweight on the risk of death. Weight-specific mortality rates can vary 6-7 hundred fold depending on birthweight. Since the distribution of birthweight varies between different populations separated by space or time, the impact of birthweight distribution on overall mortality rates can be considerable.

Several basic approaches to adjusting for the influence of birthweight in studies of infant or perinatal mortality have been suggested. First, the effect of birthweight can be "controlled" by analysing mortality in different birthweight groups (Erkkola *et al*, 1982; Forbes *et al*, 1987).

Weight-specific mortality rates began to be reported in the 1940's when rates were compared for "premature" (birthweight below 2.5kg) and "mature" infants whose weight was greater than or equal to 2.5kg. Subsequent improvements in birthweight recording and statistics have enabled the reporting of weight-specific mortality rates in more refined weight categories conventionally divided into 500 or 250 gram intervals, or broader groups defined as low birthweight (less than 2.5kg) and very low birthweight (less than 1.5kg).

A second approach to the problem of adjusting for the effect of birthweight on mortality is standardisation (Chalmers *et al*, 1978; Kleinman, 1982). Both direct and indirect standardisation techniques and related procedures involving standardised mortality ratios have been employed. This popular technique, however, has been strongly criticised by Wilcox and Russell (1983)[6] who suggest that standardisation is more likely to confuse rather than illuminate differences in

---

6. Wilcox and Russell (1983) demonstrate that birthweight standardisation is inherently biased against populations with heavier birthweights (i.e., populations whose birthweights have a greater mean).

perinatal mortality. Nevertheless, other workers (Kleinman, 1984) have stressed the usefulness of standardised rates when comparing the mortality experience of different hospital or regional populations.

Another largely experimental approach to adjusting for birthweight effects involves the development of statistical models. Wilcox and Russell (1986) have proposed a statistical model of perinatal mortality which includes two components: the frequency distribution of birthweight and the curve of weight-specific mortality.[7]

Despite the importance of controlling for the effect of birthweight, only two health production studies have employed birthweight-specific or birthweight standardised mortality as dependent variables. Harris (1982) examined the impact of pre-natal care on neonatal mortality in two different weight groups (<2500g and >2500g). This approach involves dividing the total sample

---

7. Chapman and Fryer (1980) have also examined the utility of statistical models for examining the social and environmental influences on birthweight.

into two groups and regressing the weight-specific mortality rates on an identical set of independent variables. An alternative measure of perinatal health was employed by Williams (1979) who calculated birthweight standardised perinatal mortality ratios and then regressed this indicator on various hospital characteristics (e.g. size, teaching affiliation, urban location) thought to influence inter-hospital variations in perinatal mortality.

If mortality rates are employed in infant health production functions and adjustments for birthweight are made, weight-specific mortality rates are preferable to standardised rates. Compared to overall or total mortality rtes, weight-specific mortality offers a basis for estimating how the marginal impact of various medical care inputs may vary across different sub-groups of the newborn population. The activities associated with the care of a 1000g infant are vastly different from those routinely provided for the majority of newborns. Attempting to estimate the marginal product of an intensive neonatal care unit using a health production function that employs overall neonatal mortality as a dependent variable is likely to result in marginal products that are biased towards zero

since the impact of intensive care on the small risk of mortality in normal weight infants (>2.5kg), who account for 92-95 per cent of all births, will be negligible. Standardised mortality rates or ratios, such as that employed by Williams (1979) obscure these differences and suffer from the methodological weakness noted by Wilcox and Russell (1983).

Another aspect of using mortality rates as dependent variables in health production studies involves the disaggregation of mortality by gestation or cause of death within birthweight strata. Gestational age exerts an important independent influence on mortality which, particularly at very early stages of the life cycle (e.g. 32 weeks gestation) may be a more powerful determinant of survival than even birthweight.

The influence of various medical and non-medical inputs on the risk of mortality may also vary depending on cause of death. The value of analyses of perinatal and infant mortality without information on the causes of death will be limited as has been well recognised, at least in Scotland, for over 40 years due to the work of Baird and his colleagues in Aberdeen.

Causes of death can be classified using a number of schemes ranging from standard disease classifications[8] to various typologies with clinical or pathological orientations. Ideally, deaths should be distinguished to enable the evaluation of different aspects of medical and non-medical inputs on the risk of mortality. For example, a distinction should be drawn between deaths which could reflect varying degrees of effectiveness in antenatal screening programmes for congenital malformations, obstetric management and the delivery of intensive care to the high risk very low birthweight infant (Wigglesworth, 1980).[9]

---

8. For example, the World Health Organization's International Classification of Diseases, Injuries and Causes of Deaths (ICD) that is widely used throughout the world.

9. These classifications attempt to ascribe death to the initial factor that eventually led to the baby's death, not to the immediate cause of death.

### 3.4 Non-mortality health outcomes

Health production function studies that define health outcomes in terms of mortality use aggregate data for hospitals (Williams, 1979), counties (Grossman and Jocobowitz, 1981; Goldman and Grossman, 1982; Corman and Grossman, 1985; Corman, Joyce and Grossman, 1987; Joyce, 1987a, 1987b), county groups (Hadley, 1982) or states (Williams, 1975). Although two micro level studies (Rosenzweig and Schultz, 1983; Harris, 1982) have employed mortality rates, it is more common for studies based on pregnancy outcomes of individual women to focus on other indicators of health, principally birthweight and gestational age. Birthweight and gestational age can be regarded as intermediate outcomes of pregnancy which have an important impact on both the immediate risk of mortality and the subsequent health and development of the child.

Birthweight has been used as a health outcome measure in a variety of ways. Lewit (1983) and Rosenzweig and Schultz (1983), for example, estimate reduced form health production functions where the dependent variable is actual birthweight. This specification assumes that

birthweight is a linear indicator of infant health: a questionable assumption given the well established non-linear relationship between birthweight and mortality. Kehrer and Wolin (1979) adopt a different specification and measure birthweight as a qualitative dependent variable taking the values 1 or 0 depending on whether actual birthweight is below or above a threshold level defined as the lower limit of the optimal birthweight range (3001 grams or 6.6 pounds): optimal birthweight simply refers to the weight range (3001-4000 grams) where infants have the greatest expectation of survival.

A second birth outcome measure that has been employed in infant health production studies is gestational age or the duration of pregnancy. Although not starting from a theoretical model of health production, Harris (1982) develops a statistical model to test the hypothesis that prenatal care affects the length of gestation. Rosenzweig and Schultz (1982) estimate several health production functions for various birth characteristics, including gestational age.

Neither study provides details of the method used to calculate gestational age or the reliability and accuracy of estimated gestations. Gestation is

usually measured as the interval from the day of the last menstrual period to the day of delivery. However, since dates of the last menstrual period may be unknown or difficult to establish with certainty, the length of gestation will often be missing or measured with error. This problem of errors in variables, moreover, can not be completely overcome using other methods of estimating gestation based on serial ultrasound measures or clinical assessment of the newborn infant. Excluding cases where gestation is either unknown or uncertain, as was done by Harris (1982), will introduce bias into health production studies or epidemiological studies of the determinants of health outcomes.

Harris (1982), Lewit (1983) and Rosenzweig and Schultz (1982, 1983) examine birthweight conditional on gestational age. This approach attempts to develop a model of fetal growth as proxied by changes in the distribution of birthweight at different gestational ages. Rosenzweig and Schultz (1983) employ birthweight standardised for gestational age in their health production functions. Birthweight is standardised by calculating the ratio of actual to expected birthweight where expected birthweight is estimated from a polynomial fetal growth function

which relates birthweight to gestation. Harris (1982) develops a more sophisticated model of fetal growth, allowing for both the impact of gestation and several additional explanatory variables reflecting characteristics of women and their use of prenatal care on birthweight.

Both of these models, however, suffer from the difficulty of estimating true rates of fetal growth from a series of cross-sectional observations. The process of fetal growth is largely unobservable, despite the potential of using serial ultrasonic measures throughout pregnancy of the fetus in situ. The definition of small-for-gestational age infants is problematic since the true trajectories of normal (and abnormal) fetal growth may bear little resemblance to the actual distribution of birthweights observed for different gestational ages (Forbes and Smalls, 1983). The birthweight distribution of pre-term (less than 37 weeks gestation) infants is unlikely to be equivalent to the distribution of birthweight, at 36 weeks gestation, for fetuses who survive until term. This reflects the fetal selection process which occurs throughout pregnancy where the less healthy fetuses are progressively eliminated as gestation increases.[10]

## 3.5 Multiple outputs and joint products

Many health production studies analyse a single output, like neonatal mortality, in conjunction with a single production process (e.g. Grossman and Jacobowitz, 1981; Corman and Grossman, 1985; Lewit, 1983). By concentrating on a single output measure, other outputs are often ignored. A few studies extend this restrictive model to encompass a set of multiple outputs, such as birthweight and gestation, where each output is produced by a separate production process. Rosenzweig and Schultz (1982), for example, specify separate birth characteristics production functions for birthweight, gestation and birthweight normalised for gestation. Their production functions take the general form of,

$$H_1 = h_1 (X;u)$$
$$H_2 = h_2 (X;u) \qquad (3.2)$$

---

10. This may occur either naturally or as a result of selective medical interventions accelerating the delivery of fetuses with specific clinical indications (e.g. severe growth retardation) associated with higher risks of adverse health outcomes.

$$H_3 = h_3 (X;u)$$

where $H_1$, $H_2$ and $H_3$ represent birthweight, gestation and birthweight-for-gestation, X is a vector of inputs into the production process and u is a vector of genetic and environmental factors that influence production. This recognition of the multi-dimensional aspect of birth outcomes, however, fails to reflect the fact that multiple outputs may be both interdependent and simultaneously produced. Hence, a more appropriate specification of (3.2) could be written as,

$$\begin{aligned} H_1 &= h_1 (H_2, X, u) \\ H_2 &= h_2 (H_1, X, u) \\ H_3 &= h_3 (H_1, H_2, X, u) \end{aligned} \qquad (3.2a)$$

where birthweight ($H_1$) is determined partly by gestation ($H_2$), gestation is influenced by birthweight and birthweight normalised for gestation and ($H_3$) is determined by both birthweight and gestation.

Both single and multiple output approaches, however, are inappropriate if outputs are joint products, i.e., when two or more outputs are produced simultaneously from a single production process. Formally this can be expressed as,

$$f(H_1, H_2) = h(X,u) \qquad (3.3)$$

Whether at the level of individuals or populations, joint production is a pervasive feature of many, if not all, health production processes. The same is true for infant health production functions. Birthweight, gestation and many other attributes describing the health and development of newborn infants are jointly determined by what can best be interpreted as a single production process. Outputs such as birthweight and gestation are neither independent not produced in isolation from each other in separate production processes. Inputs into the production of one output (medical care, environmental factors, behavioural inputs such as smoking) can be shared or employed to produce several simultaneous outputs which collectively describe the health and development of the infant.

Although the implications of joint production for the household production approach have been addressed from a theoretical perspective, most of the discussion is focussed on the problems arising from the "confounding" of both technology and tastes within the shadow prices for fundamental commodities (such as health). Pollak and Wachter (1975), for example, demonstrate that if the household technology exhibits joint production, demand functions based on commodity prices will depend on the household's technology, the price of inputs into the household production function and the commodity bundle consumed. Since commodity prices will not be independent of household preferences (as reflected in differences in commodity bundles) they are not capable of playing the same role accorded to exogenous prices in traditional consumer theory.[11]

---

11. Pollak and Wachter's results were also extended by Bockstael and McConnell (1983) who showed that unique Marshallian demand curves cannot be derived for commodities produced in the household production frameowrk and, as a consequence, welfare measurement using commodity demand functions is not feasible.

Although Pollak and Wachter (1975) suggest that estimation of the household technology is possible, though more complicated, when joint production is present it does require an explicit approach using data on both inputs and commodity outputs. Indirect approaches applying dual methods to estimate production functions from cost functions are not applicable since the marginal cost of each commodity will depend on the composition of output or bundles of commodities chosen and produced by the household. Since the marginal costs, or shadow prices, of commodities cannot be regarded as independent of the vector of outputs, the dual relation between the cost function and the production function will not hold.

The conceptual complexity introduced by the presence of joint production is also accompanied by methodological problems arising from ignoring joint production completely or mis-specifying joint outputs as multiple (and independent) outputs produced by separate production processes. First, OLS techniques are generally appropriate if a single output process is analysed or when multiple outputs are independently and separately produced. If the production process is that given by (3.2a) where multiple products are produced

simultaneously, TSLS or some other simultaneous equation procedure is generally required, although OLS procedures will, under certain conditions, be applicable.

However, if joint production is a characteristic feature of the production process, neither OLS nor TSLS are strictly speaking appropriate for estimating equations like (3.3) since each procedure only allows for one dependent variable in each equation (Chizmar and Zak, 1983). In this case alternative, largely experimental, procedures have been suggested (Vinod, 1968, 1976) which specify a single production function encompassing the joint outputs. Vinod's (1976) joint production model, however, is not applicable to the case of infant health outcomes since it requires what he terms the "output transformation curve" to be declining; i.e., with resources fixed, more of one output must result in less of another. However, many birth outcomes violate this condition. The most obvious instance instance is the positive relationship between birthweight and gestational age.[12] Modelling of joint health outputs as

---

12. If OLS is used, interactions between the output variables are ignored and the estimated effects of various inputs on outputs will be biased if no account is taken of the

single or multiple outputs is thus likely to remain a characteristic feature of health production studies.

### 3.6 Inputs into production

The inputs into health production processes typically include an array of biological, social, environmental, behavioural and medical factors. It could be expected that Grossman's (or any other) model would provide a theoretical justification or some guidance regarding the selection of inputs. However, with the exception of education no particular arguments derived from the theoretical model are put forward to include or exclude specific inputs in infant health production studies. Variables are often introduced as inputs simply because, on the basis of clinical and/or epidemiological studies, they are already well established co-variates of infant health outcomes.

---

jointness of production. The direction and importance of such bias in estimated parameters, such as the marginal products and marginal rates of substitution of exogenous inputs, will depend on a variety of factors including the extent of jointness, the interaction and interdependence of different outputs and the estimation procedure employed.

The similarity of health production studies and empirical studies which are not dependent on (or even cognizant of) the "new home economics" emphasises the fact that, irrespective of the theoretical framework, a common set of inputs is used in empirical studies of infant health.

Another, perhaps more important, influence on the specification and measurement of inputs is data availability which inevitably limits the possible set of inputs included in empirical studies. No study estimating a health production function has been based on data collected specifically for the purpose of testing economic models of health production. Rather health production studies have been constrained to opportunistic use of existing registration, survey or administrative data, most of which was collected for different less ambitious purposes.[13]

---

13. To varying degrees, most studies are based on routinely collected registration data of births and infant deaths, ad hoc birth surveys (Rosenzweig and Schultz, 1982) or hospital activity data (Williams, 1979).

The range of inputs employed in infant health production studies can be catgorised into four main groups. First, all studies employ one or more measures of medical care expenditure, availability, quality or utilisation. In most cases, medical care inputs do not measure the use of specific services, but rather service availability. Generally, quantitative indicators of per-capita resource availability are employed. These may range from crude indices of per-capita distribution of doctors or hospital beds to more refined measures of specific services for the care of pregnant women and newborn children. Alongside these measures of availability several studies have also included expenditure on medical care which reflects both resource availability and the use of services.

A major weakness of health production studies which attempt to estimate the impact of medical services on infant health outcomes is that information in the use of specific services is frequently unavailable. Only four studies have included utilisation measures as co-variates of infant health. Of these, the two micro-level studies, using the individual as the unit of observation, have both analysed the effect of antenatal care on health outcomes (Harris, 1982;

Lewit, 1983). Furthermore, even when data on utilisation is available, information on the quality of services is not. No adjustments for qualitative differences in inputs into production can be made. This is an important omission and potential source of bias in health production studies.[14] Although several studies acknowledge that the quality as well as quantity of care could be an important determinant of infant health, explicit measure of quality have only been employed by Williams (1979) and Hadley (1982). Williams (1979) investigates whether several characteristics of hospitals (e.g. size, teaching affiliation) are related to perinatal mortality whereas Hadley (1982) analyses qualitative differences in labour inputs (physician age and certification status).

---

14. Feldstein's work on the components of cost escalation in the hospital sector emphasise the importance of qualitative changes in the context of health care (Feldstein, 1974). Goldman and Grossman (1978) employed a hedonic characteristic approach to measuring the qualitative differences in health care in a study of medical care utilisation by children.

A second set of inputs employed in infant health production studies are biological and demographic variables. These may relate to either the mother or newborn child. Characteristics of the mother include age, parity (birth order) race and prior obstetric history (previous fetal/infant loss). Several of these factors (maternal age and parity) can also be classified as behavioural outcomes which result from decisions taken by women regarding the numbers and timing of births. Maternal age, parity and previous fetal/infant loss are also interdependent.

Several physiological characteristics of infants have also been included as inputs into the production of subsequent health outcomes. Infant sex, birthweight and gestation are amongst the most important determinants of survival during the perinatal period and throughout the first year of life. However, there are no firm rules as to whether these factors should be regarded as inputs into a production process, intermediate outputs or final outputs.

A third set of variables included in health production studies are socio-economic characteristics of individuals or populations. At an individual (or household) level it is difficult

to distinguish many socio-economic characteristics from behavioural factors. All of the socio-economic variables employed in infant health studies (education, income, wages, geographical area) summarise to some extent the outcomes of decisions regading investment in human capital. The theoretical justification for and interpretation of specific variables like education in health production functions can be ambiguous (see Section 2.4 in Chapter 2). For example, Rosenzweig and Schultz (1981) attempt to distinguish between the "efficiency" and "allocative" roles of education in production (Welch, 1970). That is, education can:

- enhance the efficiency of household production by augmenting the marginal product of one or more inputs in household production

and/or

- improve the allocation of resources within the household

The improved allocation of resources is assumed to be due to a decrease in the costs of information and changes in the perceptions of the technology

of health production arising from education which enable a more informed choice regarding health inputs.

Thus, education may have no impact on the efficiency of production but may influence health outcomes via the demand for goods (and activities) affecting health. Rosenzweig and Schultz's (1981) results support the hypothesis that education significantly affects health input choices but fail to support the efficiency role accorded to education, principally by Grossman, in health production studies. Thus, if education plays any role in infant health production it does not appear to be that of augmenting the marginal productivity of health inputs; a central tenet of the human capital theories of how education affects the efficiency of market and non-market activities.

Like the Rosenzweig and Schultz study of education and health outcomes, the analysis by Kehrer and Wolin (1979) of how income may affect the incidence of low birthweight provides or at least identifies a possible rationale for expecting socio-economic factors to have an impact on infant health outcomes. Kehrer and Wolin investigate whether the income transfers, provided as part of

the income maintenance experiment conducted in Gary, Indiana, affect the incidence of low birthweight among the poor. Three possible links between increased income and improvements in low birthweight were suggested. First, increased income could lead to more use of antenatal care, either by increasing ability to pay and/or subsidising true cost (loss of earnings) associated with antenatal care. This assumes that early and more frquent use of antenatal care could have a beneficial impact on birthweight.

A second mechanism linking income and birthweight is via the impact of income transfers on household diet and maternal malnutrition and weight gain during pregnancy. Finally, a third link involves the impact of work (paid employment) on birthweight. If employment during pregnancy influences reproductive outcomes like birthweight, the reduction in labour force activity permitted by the income maintenance may have a beneficial effect on birthweight. However, having specified these different channels, data limitations precluded any empirical tests which could be used to discriminate between and assess the relative inmportance of the likely mechanism linking income transfers and birthweight.

A final set of factors entered as inputs into infant health production processes are those measuring health related behaviour. Several of these have already been mentioned. Fertility decisions, choices regarding abortion and the use of antenatal care are all aspects of behaviour which may influence infant health outcomes. Cigarette consumption during pregnancy and women's preferences for infant feeding are two additional factors that have been entered into health production studies. Behavioural decisions taken by parents have been explicitly modelled most extensively by Rosenzweig and Schultz (1981, 1982, 1983). They estimated demand equations for several health related behavioural variables[15] and then used the fitted values of these variables in second stage estimates of infant health production functions.

---

15. Birth order, delay after conception in seeking medical care during pregnancy, mother's rate of smoking while pregnant, mother's age, duration of breast feeding and delay by the mother after the child's birth in returning to work.

## 3.7 Production sets and functional form

A variety of functional forms have been employed to characterise the relationship between inputs and health outputs. As in all production studies when the technology of production is not given or fixed by engineering considerations, the precise form of input/health output relations can not be unambiguously determined. All that can reasonably be accomplished is that restrictions on the production process can be identified and then tested. This aproach, however, is very much the exception, rather than the rule, in infant health production studies where functional forms are arbitrarily imposed on the data with little, if any, attention given to the corresponding restrictions and interpretation arising from different assumptions regarding the link between inputs and outputs.

Perhaps the most ambitious and potentially misleading endeavour in this area is the uncritical attempt to apply textbook concepts to an area like health. The concept of a production function, for example, as conventionally understood and applied may be an inappropriate way of characterising/modelling the determinants of health outcomes. Many of the criticisms levied at

the conventional economic theory of production when applied to the study of education effectiveness (Murnane and Nelson, 1984) also extend to the health sector.

For example, underlying the standard theory of production are several assumptions which may not be appropriate in studies of health "production", viz.,

- Inputs and techniques of production are rationally chosen from a set of well-defined and well-known possibilities so as to best achieve objectives. Organisations and firms (or households) thus operate at the frontier of their production sets or on their production functions,

- Observed inputs/output combinations are of situations when exploration of the technical efficiency of alternative techniques and input combinations is essentially complete. The process of production is essentially distinct from that of experimentation,

- The requisite inputs and techniques are generally available. All organisations face the same production function.

■ The creation of new techniques occurs through separate research and development activities. Innovation is thus due to research and development and there are no particular problems in transferring knowledge from the research and development setting to actual use.

Murnane and Nelson (1984) argue convincingly that the standard theory, embracing these four assumptions, is likely to describe the empirical reality of input-output relations in a field such as education or any activity or sector where techniques are idiosyncratic or poorly articulated.

Can the standard theory of production be applied, at least as a first approximation, to the production of infant health? While it is easy to identify health outcomes and examine the influence of various factors on these outcomes, it is erroneous to assume that household activities conform to a set of constructs that have been developed to analyse productive activities of firms (and industries) in the manufacturing, transportation and energy sectors. In particular, at the household level, it is difficult to see how the traditional theory is applicable to what is in reality a "lumpy" process involving very little if

any repetition. Many households may only engage once or twice in the process of "producing" a child, offering very little scope for investigating the impact of alternative combinations of inputs on outputs or potential for learning by doing. Many health inputs, particularly biological or genetic factors, are fixed whereas other resources may not be readily available. Education, information and other parental inputs may be rationed. The scope for innovation may be limited. Despite the proliferation of birth guides etc., techniques of production may not be well articulated, easily obtainable or transfer able from household to household. The production set will have a single point (or very small set of points) representing the input-output combinations which actually characterise the households activities and infant health outcomes - many of which may be unconnected.

If the familiar theory of cost and production is not applicable the measurement of efficiency, marginal products, elasticity of input substitution, etc., is problematic in the case of infant health. Several studies either implicitly, or explicitly proceed as if they were estimating health production functions. Rosenzweig and Schultz (1982, 1983) present a series of estimates

of "health technology" using linear, Cobb-Douglas and translog specifications of production derived from the generalised Leontief-Diewert (Diewert, 1971) production function. The flexible functional form provided by the generalised Leontief-Diewert enabled Rosenzweig and Schultz to test the restrictions imposed by different specifications of technology. Both the sign and magnitude of estimated marginal products are sensitive to the specification of technology within the household. The criterion for choosing one specification over another is not the reasonableness of the restrictions imposed on the input-output relations but rather statistical tests used to discriminate between competing models of production. This testing strategy, however, leads to the acceptance of Cobb-Douglas technology which seems implausible given the assumptions underlying this well known but restrictive functional form. Returns to scale may not be constant nor equal to one. Clearly, infant health can be produced without using all available inputs whereas the Cobb-Douglas specification dictates that all inputs must be employed otherwise output is zero.[16] The impact of

---

16. If one input level is zero, output will be zero by definition in the Cobb-Douglas case. Inputs such as cigarettes are clear examples where this condition is

an input like education or income may not be independent of the level of input utilisation.

### 3.8 Findings of previous studies

A common feature of infant health production studies is their poor explanatory power, particularly in cross-sectional investigations conducted at the individual level. With the exception of Lewit (1983), the series of individual level studies by Rosenzweig and Schultz (1981, 1982, 1983) fail to explain over 90% of the variation in birthweight, gestation or birthweight standardised for gestation. The aggregate level studies of Grossman and his co-workers manage to explain about 25 to 30% of the variation in white neonatal and infant mortality rates but can only account for 12-20% of the variation in neonatal and infant mortality rates in black infants. Hadley's (1982) analysis of variations in mortality at the county level achieved slightly greater explanatory power.

#### 3.8.1 Individual Based Studies

---

unlikely to be satisfied.

The results of individual based studies are generally consistent with the findings of clinical and epidemiological studies of the effects of biological and demographic characteristics of mothers (and infants) on infant health outcomes. Women at the extremes of the age and parity distribution tend to have the greatest risk of poor or adverse pregnancy outcomes. Black women and women who have a previous history of poor obstetric performance also face higher risks of low birthweight, pre-term delivery and perinatal and infant mortality. Many of these effects, however, are not significant, particularly when birthweight and, to a lesser degree, gestation are controlled for. Birthweight is by far the most important determinant of infant survival. The effects of factors such as maternal age and parity on perinatal mortality appear to act via shifts in the distribution of birthweight and gestation, which in turn, influence the probability of survival.[17] The results of studies which fail to

---

17. This finding is also consistent with recent epidemiological investigations of the utility of using birthweight-specific mortality rates as a way of standardising for the effects of demographic and socio-economic factors that have been traditionally associated with poor reproductive performance.

include birthweight as a regressor in an equation attempting to explain the variation in infant mortality (Rosenzweig and Schultz, 1983) are thus difficult to evaluate due to the bias arising from omitting birthweight.

The estimated effect of socio-economic factors on infant health outcomes generally fails to support the predictions of the Grossman model. Lewit (1983), for example, reports that more educated women have lighter babies; a finding which contradicts the expected impact of education on health. Rosenzweig and Schultz (1981) detect a slight effect of education on birthweight: a 10% increase in educational attainment is associated with a 0.77% increase in birthweight (25 grams). Given an average of 11 years of formal schooling, a woman who completed four years of post-secondary schooling could, according to these estimates, expect an increase of about 100 grams or 3% in the weight of her baby. Furthermore, no significant impact of education on birthweight standardised for gestation or on the marginal product of health inputs (efficiency hypothesis) was demonstrated in their study.

In contrast to education, the results of Kehrer and Wolin's (1979) study suggested that income maintenance programmes could have a beneficial impact on birthweight. Depending on a woman's age, pregnancy interval and smoking behaviour, income transfers resulted in an average weight gain of between 174 and 530 grams (5% to 15%). An important finding of their study is that the effect of a socio-economic variable like income is likely to be conditional on other household or individual characteristics. Their use of a Tobit model where the response of the dependent variable to a change or shift in an independent variable depends on the particular values taken by all other independent variables permits a more realistic, if more complex, assessment of the effects of specific factors.

Several studies examine the effect of antenatal care on health outcomes. In fact this is virtually the only health or medical care factor that is included in health production studies conducted at the individual level. Harris (1982) finds a weak positive, though statistically insignificant, effect of the timing of antenatal care on birthweight (60 to 100 grams). Contrary to expectations prevalent in the clinical literature, Harris demonstrated an inverse association between

the start of antenatal care and birthweight specific mortality: a result explained by the process of fetal selection which results in a smaller proportion of high risk fetuses in the mothers remaining pregnant into the third trimester. Alternately it could be that women who expect a poor outcome based, say, on their previous obstetric history, would attend earlier for antenatal care. After taking account of fetal selection, his results suggest that the duration of care has a small favourable effect, again not significant, on perinatal survival. The complete absence of care would result in a 20% proportional increase in perinatal mortality, conditional on birthweight and gestational age.

Rosenzweig and Schultz (1982, 1983) also examined the effect of antenatal care on various infant health outcomes. Delay in attendance for care was not significantly associated with birthweight or gestation. A six month delay in attendance appeared to only reduce birthweight by 45 grams and gestation by 1.6 weeks. Their results examining the effect of care on mortality are consistent with those reported by Harris (1982).

### 3.8.2 Aggregate Level Studies

Throughout the 1980's Grossman and his colleagues have published a series of related studies[18] on the determinants of black and white neonatal mortality rates in the U.S. All of these studies employ essentially the same cross-sectional regression model estimated using population based data. The level of aggregation is the "large" U.S. county: a geographical (and administrative) area with a population of at least 50,000.[19] The determinants examined include poverty, education (schooling), Medicaid, maternal and infant care projects, community health centres, federallly subsidized family planning services for low-income women, special supplemental Food Program for Women, Infants and Children, the legalization of abortion and the availability of physicians, obstetricians, gynecologists and neonatal intensive care. A characteristic feature of this

---

18. The complete series of studies published to date include Grossman and Jacobowitz (1981), Corman and Grossman (1985), Corman, Joyce and Grossman (1987), Joyce (1987a, 1987b).

19. The number of counties analysed ranges from 357 for neonatal mortality amongst black infants to 677 for neonatal mortality for white infants. These counties account for approximately 80 per cent of the U.S. population.

literature is the attempt to "explain" the recent improvement in U.S. neonatal mortality rates over time using the cross-sectional regression coefficients applied to national trends in this set of (partly) exogenous variables.

Several salient results emerge from these studies. The first is the sensitivity of findings to relatively small changes in model specification or variable measurement. This is perhaps best illustrated in the estimated impact of the legal abortion rate on the neonatal mortality rate. Grossman and Jacobowitz (1981) conclude that:

"the increase in the legal abortion rate is the single most important factor in reductions in both white and nonwhite neonatal mortality rates. Not only does the growth in abortion dominate the other public policies but it also dominates schooling and poverty"

Although not reported, the estimated elasticity of the neonatal mortality rate with respect to the legal abortion rate (per 1000 births) was -0.304 for whites and -0.365 for blacks. Using a different measure of abortion[20] Corman and Grossman

---

20. Abortion providers (public hospitals, private hospitals,

(1985) report results that suggest elasticities of -0.210 for whites and -0.625 for blacks. However, when abortion is treated as an endogenous factor and the percentage of low birthweight births is controlled for, the elasticity drops to -0.014 for whites and -0.024 for blacks (Corman, Joyce and Grossman, 1987). This dramatic change in the estimated impact of abortion on neonatal mortality was also confirmed by Joyce (1987a) who reported elasticities of -0.048 for whites and -0.068 for blacks.[21] A similar pattern of erratic estimates also emerges for the impact of family planning and neonatal intensive care.

---

non-hospital clinics and office based physicians per 1000 women aged 15-44)

21. The ambiguous effect of the abortion rate has also been confirmed by a recent report from the National Bureau of Economic Research on nutrition and infant health in Japan. Using cross-sectional data, Yamada, Yamada and Chaloupka (1987) found that the availability of abortion was positively (although insignificantly) related to infant mortality.

Second, female education is associated with lower neonatal mortality rates, particularly for whites. Corman and Grossman (1985) suggest that schooling affects mortality by "influencing the mix of inputs selected by families to produce healthy infants". This "allocative" role assigned to female education, however, is not explicitly tested. Schooling, in fact, is dropped from the models estimated by Corman, Joyce and Grossman (1987) and Joyce (1987a, 1987b) who focus instead on more direct inputs into the production of infant health such as smoking and prenatal care. Thus, even if there is a link between education and more informed choice or behavioural change, education has been largely superseded by more direct measures of inputs into infant health.

Third, several of these studies attempt to estimate the impact of different factors on the improvement in neonatal mortality over time. Essentially, the assumption of fixed coefficients is adopted: i.e., that the impact of factors such as neonatal intensive care, abortion and family planning on neonatal mortality is time invariant. This seems an unreasonably assumption, particularly in the case of neonatal intensive care. Over the last two decades care provided for sick newborns has changed dramatically culminating

in the highly technical, diagnostic and therapuetic techniques that characterise modern intensive care. As the mix of services change so too may the marginal product of newborn care.[22]

The same criticism can also be applied in the case of abortion. As the characteristics of women (and their aborted fetuses) change over time so too will the impact of abortion on neonatal mortality. Joyce (1987a), for example, suggests that abortion "lowers the rate of unfavourable birth outcomes". This assumes that allowing women to terminate an unwanted pregnancy reduces the distribution of high risk births. While this may be true for the 2.0 to 3.0 per cent of abortions conducted in Scotland due to risk of fetal abnormality, most of the increase in abortion has

---

22. Trends in weight-specific mortality are often cited as evidence of the changing effectiveness of neonatal care. However, the most convincing demonstration of how the mix of newborn care can influence outcomes is the economic evaluation of intensive care conducted by Boyle et al (1983) who showed that the introduction of a regional perinatal program in 1970 resulted in increased health outcomes for a 1973 to 1977 birth cohort compared to a 1964 to 1969 birth cohort.

occurred in women aged 16-24 who typically face below average risks of perinatal mortality. Ill timed pregnancies may not necessarily lead to intrinsically less healthy infants.

Another important cross-sectional analysis of infant mortality was reported by Hadley (1982). Using U.S. data for 1969-1973 aggregated into county groups alongwith a Cobb-Douglas specification, Hadley estimated a series of infant health production functions. Although not formally dependent on the "new home economics" approach the empirical analysis is virtually indistinquisable from the empirical models estimated by Grossman and his colleagues.

Infant mortality is broken down into its neonatal and post-neonatal components. Separate production functions are estimated for different infant sex and race groups. A host of medical care variables and social policy variables are introduced into the analysis as well as as measures of population risk which controll for variation in birthweight, age-sex-race differences in weight-specific mortality, mother's age and the percentage of births in hospitals. Following the lead of Grossman and Jacobowitz (1981) Hadley also

considers the impact of Medicaid coverage of unborn children and abortion law liberalization on infant mortality.

Not surprisingly, the most important factor explaining the variation in overall mortality amongst white infants is the variation in birthweight distribution. Medical care (Medicare expenditures per enrolles and the numbers of obstetricians and paediatricians per 1000 births) is negatively associated with mortality. An increase of 10 per cent in medical care spending per capita is associated with a 1.5 to 2.0 per cent reduction in mortality rates. A similar increase in the number of obstetricians and paediatricans per 1000 live births would lead to a 0.6 to 0.8 per cent decline in mortality.

Quality of care variables such as the age and qualifications of physicians had little impact on mortality. The percentage of hospitals with neonatal intensive care facilities was negatively but insignificantly associated with neonatal mortality. The results for black infants were generally similar. The coefficients in the black equations, however, were unstable when different specifications (i.e., different sets of variables)

were employed as regressors: a result attributed to the twin problems of multicollinearity and mis-specification.

The problems of multicollinearity and specification also constrained the robustness of Hadley's results regarding the impact of Medicaid coverage and abortion. After dropping expenditure on medical care from the equations, the restricted model suggests that Medicaid coverage and abortion law liberalisation improves infant health outcomes. The impact of abortion is small. The estimated elasticities (-0.04 for whites; -0.01 for blacks) correspond closely with those reported by Joyce (1987a).

Following the attempts by other workers to "explain" time series of mortality rates using cross-sectional results, Hadley considers the decline in infant mortality between 1969-1978 and concludes that the most important factor was the reduction in the incidence of low birthweight births, followed by the availability of obstetricians and paediatricians. Hadley does caution that the reliability of the predicted changes decreases as one moves further from the mean values of the variables used to generate the cross-sectional estimates.

## 3.9 Conclusions

This survey of empirical studies of perinatal and infant health conducted within an economic framework has highlighted a number of points. First, all of the studies are cross-sectional and are based on U.S. data. No time series or longitudinal study has been reported largely due to the fact that a consistent and reasonably long time series on infant health outcomes and inputs has not been assembled. Attempts to bridge this gap have tended to take the form of "explaining" the change in neonatal and infant mortality over time using cross-sectional regression coefficients in conjunction with national trends in the "exogenous" variables. The limitations of this approach highlight the need for a direct examination of time series data as a way of improving our understanding of the factors which have contributed to the improvement in health outcomes over time.

A common features of the aggregate level studies is the relatively limited set of mortality rates that have been used to measure infant health outcomes. Although mortality rates have been disaggregated by age, sex and race no study has analyzed how inputs may differentially influence the survival of infants in different birthweight groups or strata. Another obvious and long overdue disaggregation of mortality data is by cause of death. Although the results based on total or overall mortality rates provide an estimate of the average impact this may obscure a wide range of effects in different sub-groups of the population.

Relating inputs to outputs is a perennial problem in health production studies. Many characteristic features of input-output relations in infant health fail to conform to the standard models of production that have been routinely employed in previous health production studies. At the micro or individual level input substitution possibilities are limited, many inputs are fixed and there is often little scope for exercising choice over different input combinations. Inputs may have positive, negative or no impact on output. Many inputs have no observable effect on

output until a certain threshold level is passed. Furthermore, all inputs are not required for production.

The production process itself is not routine and largely unobservable during the production period. Long lags occur between the utilisation of inputs and eventual outputs. There is very little scope for innovation or learning by doing since the production process is unlikely to be replicated more than once for most individuals. The unobservable nature of production introduces an important element of qualitative uncertainty which is difficult for decision-makers to avoid.

At the aggregate level, there is greater scope for input substitution. Although the production process is more routine and standardised, there are possibilities for innovation and the development and adoption of new techniques over time. There is still a lag between the use of some inputs and eventual outputs. At the aggregate level, the marginal product of inputs also may be positive, negative or zero.

The choice of functional form for relating inputs to outputs should therefore not be based on convenience - a practice which imposes familiar

functional forms borrowed from textbook analyses of production functions - but rather according to careful empirical testing of different specifications which are flexible enough to accommodate realistic relationships between inputs and health outputs. This point can be expanded further into a more general criticism of the methods used in health production studies.

A characteristic features and inherent limitation of all of the empirical studies surveyed is their uncritical approach to model specification, validation and testing. This is particularly evident in the work of Grossman and his colleqgues who use econometrics to illustrate the theories of household production which they believe independently. This approach, recently characterised by Gilbert (1986), starts with the unproven assertion that the theory is correct, generates coefficient estimates for the variables of interest and then addresses the pathological features of the regression results (serial correlation, multicollinearity, etc) which presumably have led to the usual collection of disappointing/unexpected results (insignificant coefficients and "wrong" signs).[23] Thus poor

---

23. A graphic illustration of this approach is inherent in

results imply problems of consistency and efficient estimation of the "true" model. This approach, as Hendry (1980) suggested in his inaugural lecture at the London School of Economics too often constitutes a recipe for disaster due to:

"Simply writing down an "economic theory", manipulating it into a "condensed form" and "calibrating" the resulting parameters using a pseudo-sophisticated estimator based on poor data which the model does not adequately describe".

An alternative approach, derived in large part from the early work of Denis Sargan (1964) and most closely associated with the work of David Hendry, is to begin with a very general specification which is systematically reduced or simplified and then rigorously tested. This general to specific model simplication does not assume that one knows the "correct" or "true" model but rather that well conducted specification

---

the following quotation taken from the study by Grossman and Jacobowitz (1981): "Because the poverty rate has the "wrong" sign for blacks it is excluded in regressions A2, A4, B2 and B4".

searches and "destructive" model testing will increase the likelihood of discovering models that adequately characterise the available data. The hallmark of this approach involves the full disclosure of the path leading to a preferred model and the results generated by tests addressing the problem of "quality control" of the preferred model (McAleer et al 1985).

Perhaps one of the most succinct summaries of the potential benefits of Hendry's methodological approach is embodied in a question suggested by McAleer et al (1985) that should be posed in any empirical study, namely, "Is the Model a Lemon?". This question is never addressed, let alone answered, in the corpus of literature on the determinants of infant health. However, the empirical studies surveyed in this Chapter does leave one with a distinctive tart aftertaste.

## Glossary

**Stillbirth** - child born after 28 weeks gestation, not breathing nor showing any signs of life.

**Stillbirth rate** - stillbirths per 1000 total births (live births and stillbirths)

**Neonatal** - first 28 days of life.

**Neonatal mortality rate** - deaths at ages under 28 days per 1000 live births.

**Post-neonatal** - period from 28 days up to one year following live birth.

**Post-neonatal mortality rate** - deaths during first year of life (excluding neonatal deaths) per 1000 live births.

**Infant** - first year of life.

**Infant mortality rate** - deaths during first year of life per 1000 live births.

**Perinatal** - period from 28 weeks gestation to 7 or 28 days after birth.

**Perinatal mortality rate** - stillbirths and first week (or neontal) deaths per 1000 total births (live births and stillbirths).

# CHAPTER 4 POST-WAR TRENDS IN SCOTTISH PERINATAL MORTALITY

## 4.1 Introduction

Post-war research on perinatal mortality in Scotland has its roots in the work of Sir Dugald Baird in Aberdeen (Baird and Wyper, 1941; Baird, 1945; Baird, 1947; Baird, 1949) and in an important, though less prominent, study conducted in Edinburgh by Drillien (1947). These local investigations were complemented by a series of national reviews by Charlotte Douglas and Peter McKinlay throughout the 1940's and 1950's (Douglas, 1945; Douglas, 1954; Douglas and McKinlay 1959) which were subsequently updated in the 1960's by numerous studies.[1] In the late 1970's a series of annual national inquiries into perinatal deaths (McIlwaine et al, 1979) integrated Baird's obstetric classification of perinatal deaths and the population based approach epitomised by the early work of Douglas and McKinlay. These perinatal inquiries

---

1. This national perspective was followed by local studies which, for example, investigated the possible causes underlying the above average perinatal mortality traditionally recorded for residents of Glasgow (Richards et al, 1969; McIlwaine et al, 1974).

were superseded by similar national studies conducted on a routine basis as part of the information system of the Scottish National Health Service (McIlwaine et al 1985; Cole et al, 1986).[2]

These diverse reports describe different aspects of perinatal mortality at different points of time in the Scottish population. Although some recent studies have examined trends in Scottish perinatal mortality during the 1970's (Forbes *et al*, 1982) and the impact of demographic and social factors on the risks of perinatal mortality over the period 1960-1982 (Forbes and Pickering, 1985) no overall review of Scottish perinatal mortality during the post-war period is available. This chapter examines post-war trends in Scottish perinatal mortality and reports a time series of dis-aggregated perinatal mortality rates that have been constructed especially for use in the estimates of the health production functions estimated in Chapters 6 and 7. Perinatal mortality rates are sub-divided according to specific cause of death. Neonatal mortality rates

---

2. Other notable contributions include that of Sir Dugald Baird published towards the end of his long career where he restated his belief that perinatal mortality depends as much on antecedent social and economic factors as on effective perinatal care (Baird, 1980).

are dis-aggregated by age at death. Perinatal mortality rates are also calculated for different birthweight groups. Each specification of the dependent variable permits the estimation of a set of health production equations which may provide further insight into the determinants of different components of perinatal mortality.

Stillbirth and neonatal death rates can be specified in a variety of ways. The most general measure is stillbirths and neonatal deaths from all causes in all birthweight groups. Although these aggregate indices of the two main components of perinatal mortality are readily available from published statistics they easily obscure the changing pattern of perinatal mortality in post-war Scotland and provide a poor basis for assessing the influence which social and medical factors may have had on perinatal mortality in different population sub-groups.

The chapter is structured as follows. Section 4.2 describes data sources, variable definitions and summary measures used to examine trends in perinatal mortality over time. Section 4.3 presents a general review of long run trends in perinatal mortality. General trends in stillbirth rates and neonatal mortality rates are

distinguished. Additional insight into the changing pattern of neonatal deaths is provided by an analysis of age specific death rates during the neonatal period.

In Section 4.4 cause-specific perinatal mortality rates are reviewed in two ways. First, selected cause-specific still-birth and neonatal death rates are calculated over the period 1950-1985 using registered causes classified according to the Sixth to Ninth revisions of the International Statistical Classification of Diseases, Injuries and Causes of Death (World Health Organization, 1950; 1955; 1965; 1977). Second, the Aberdeen classification of perinatal deaths (Baird and Wyper, 1941; Baird, Walker and Thomson, 1954) is used as a comparative frame of reference for reviewing trends in specific causes of perinatal mortality. Reflecting an obstetricians view of perinatal events, the Aberdeen system classifies deaths according to their predisposing obstetric event. Since its introduction the Aberdeen classification has been employed in a variety of hospital and population based studies throughout the U.K. culminating in a series of national enquiries into perinatal death in Scotland.

Section 4.5 presents birthweight specific perinatal mortality rates. Using a variety of published and unpublished data a unique time series of birthweight specific perinatal mortality rates is constructed and

used to examine how the relative risk of perinatal mortality has changed in different birthweight groups over time. The chapter's conclusions are summarised in Section 4.6.

## 4.2 Data Sources and Methods

### The Perinatal Period

Since 1939, perinatal deaths in Scotland have been defined to include stillbirths or late fetal deaths, of at least 28 weeks gestation, and either (i) all babies dying within one week of birth or (ii) all neonatal deaths or babies dying within four weeks or one month of birth.[3] Baird and Wyper (1941) argued that since most neonatal deaths are related to pregnancy and childbirth they should be analysed alongwith stillbirths to gain an appreciation of the number of infants, who despite

---

3. In contrast to England and Wales, where data on perinatal deaths are available from 1927 onwards, the analysis of perinatal mortality is only feasible from 1939 for the Scottish population. Although the Registrar General (Scotland) began publishing data on first month deaths in 1910, first week deaths in 1922 and first day deaths in 1931, the registration of stillbirths was not initiated until 1939 following the Registration of Stillbirths (Scotland) Act of 1938.

reaching the stage of viability, failed to survive. Beginning in the 1950's, however, late neonatal deaths (between the ages of one week and one month) tended to be excluded from the calculation of perinatal mortality rates. This concentration on events during the immediate period surrounding birth can also be seen in the brief use of the term "obstetric death rate" (Duncan, Baird and Thomson 1952) to describe the number of stillbirths and deaths in the first week of life per 1000 total births (live and still). Although there were some notable exceptions such as the 1958 British Perinatal Mortality Survey (Butler and Bonham, 1963) which defined perinatal deaths as stillbirths and neonatal deaths, the narrow definition of perinatal mortality remained popular. Recognition that most neonatal and indeed many infant deaths have causes rooted in the perinatal period has coincided with a return to Baird and Wyper's original suggestion as can be seen in recent reports on perinatal death in Scotland which analyse stillbirths alongside both early and late neonatal deaths (Cole, 1988).

Unless otherwise stated throughout this chapter perinatal deaths include stillbirths and neonatal deaths. Stillbirths include all fetal deaths whose gestational age is estimated to be greater than 27 completed weeks. Neonatal death is defined to include all deaths occuring within four weeks of birth. Prior

to 1958 neonatal deaths include all deaths occurring within one month of birth. Neonatal deaths are sub-divided into three mutually exclusive age groups: less than 24 hours, 1-6 days and 7-27 days following birth. All data on stillbirths, neonatal deaths and live births was extracted from the annual reports of the Registrar General Scotland (various years).

Cause-specific Perinatal Mortality

The examination of national trends in cause-specific perinatal mortality requires a consistent classification over time of the certified causes of both stillbirths and neonatal deaths. Certified causes of death, however, are classified in Scotland according to conventions established by the International Statistical Classification of Diseases, Injuries and Causes of Deaths (ICD) which has been revised four times since 1948. Furthermore, although cause-specific stillbirth rates were reported in 1939 by the Registrar General, a perinatal classification was not introduced in Scotland until 1958 when an experimental scheme was adopted which classified neonatal deaths using the existing stillbirth list. This scheme continued until 1968 when the Eight Classification (ICD-8) introducted a "P" list of 100 causes of perinatal death for the common classification

of stillbirths and neonatal deaths. One consequence of ICD-9 was that the initial "P" list was superseded in 1979 by a revised version containing 62 causes.

These evolving classifications introduce a series of discontinuities which limit the number of cause-specific death rates that can be calculated on a consistent basis over the entire post-war period. Within the time domain of a specific ICD revision, however, consistent comparisions can be made. It is thus possible to examine cause-specific stillbirth and neonatal mortality rates over the periods 1958-1967, 1968-1978 and 1979-1985. By linking successive revisions, moreover, three important causes of neonatal mortality (congenital malformations, prematurity and asphyxia) can be monitored across as well as within these time periods. Data on stillbirth and neonatal deaths classified according to certified causes was extracted from the Annual Reports of the Registrar General Scotland (various years).

The Aberdeen clinical classification of perinatal mortality, introduced by Baird and Wyper (1941) and developed further by Baird, Walker and Thomson (1954), provides an alternative to a classification based on certified causes of death recorded by the Registrar General. This classification subdivides perinatal deaths according to clinically diagnosable

maternal/fetal conditions. Eight categories of perinatal death are distinguished The Aberdeen classification adopts an obstetric perspective on the causes of perinatal death in contrast to other classifications based on detailed pathological analysis (Bound, Butler and Spector, 1956) or simplified pathological sub-groups (Wigglesworth, 1980). Although the Aberdeen classification has been widely used it has not escaped criticism. One common complaint concerns the classification's lack of specificity since the commonest cause of perinatal death is invariably "birthweight < 2500g" which they suggest is as unsatisfactory as classifying most adult deaths to old age. The classification's maternal/obstetric focus also takes little account of the babies' individuality and the underlying clinico-pathological processes within the baby which may have culminated in perinatal death.

However, despite these difficulties, the Aberdeen classification represents the only alternative to registered causes of death when documenting trends in cause-specific post-war perinatal mortality in Scotland. Prior to 1977 data on (Aberdeen cause-specific perinatal mortality was assembled from a range of scattered studies reported in the literature. Later data was obtained from the published reports of the Scottish Perinatal Mortality Survey.

**Birthweight Specific Perinatal Mortality**

Prior to 1963 data on birthweight specific perinatal mortality in Scotland was confined to ad hoc local studies and one national survey conducted in 1954 (Douglas and McKinlay, 1955). Beginning in 1963 annual data on birthweight specific stillbirth and neonatal mortality rates can be obtained in disaggregated form from the Local Health Authorities Statistics (Form 15) completed for each county and burgh in Scotland. A detailed breakdown by 500 gram intervals is only available for births weighing less than 2501 grams. Weight specific mortality rates for all births weighing more than 2500 grams can be estimated by subtracting the sum of low birthweight births and perinatal deaths from the total births and perinatal deaths recorded by the Registrar General. The series was discontinued following the 1974 reorganisation of the NHS.

In 1969 birthweight began to be recorded for births occurring in maternity hospitals and wards which participated in a new hospital information scheme. The Scottish Morbidity Record - 2 (SMR2), a hospital record specially designed for maternity discharges, contained details of the other's health during pregnancy, a description of intrapartum events and birth outcomes. Population coverage gradually improved as more hospital

joined the SMR2 scheme. From 1976 onwards birthweight is recorded for more than 97 per cent of registered (total) births.[4] The series reported below thus represents a linkage of local surveys (1956-1962), the LHA Form 15 series (1963-1973) and the SMR-2 series (1974-1985).

## Perinatal Mortality: Rates and Indices of Change

A variety of mortality rates are presented in this chapter. Perinatal mortality rates are defined as the number of stillbirths and neonatal deaths per 1000 total births (live and still births). Stillbirth rates are defined as the number of stillbirths per 1000 total births. Age specific mortality rates throughout the neonatal period are defined as the number of deaths in a particular age group per 1000 live births. Cause-specific stillbirth, neonatal and perinatal mortality rates are defined as the number of stillbirths, neonatal deaths and perinatal deaths

---

4. Compared to the earlier LHA series, the birthweight intervals are shifted downwards by 1 gram (e.g. 1000-1499 versus 1001-1499). Due to rounding and digit preference when recording birthweight, this change in reported birthweight intervals introduces a negligible discontinuity into the data series.

attributed to a particular cause of fetal or neonatal death per 1000 total or, in the case of neonatal deaths, live births. Birthweight specific perinatal mortality rates are defined as the number of stillbirths and deaths of infants occurring within one week of birth in a specific birthweight interval per 1000 live and stillbirths whose birthweight falls within that interval.

Average annual percentage rates of change in mortality rates are calculated using geometric means. Mortality rates are also expressed using index numbers ($I_t$) where the rate in the base year (t) is set to 100. Denoting the base year by t and the terminal year of the series by t+n, the proportion of decline in mortality which occurred over the interval t to t+i is given by

$$[(I_t - I_{t+i})/(I_t - I_{t+n})]100$$

where $i < n$.

Proportional mortality rates, defined as the percentage of total deaths in a specific age, cause of death or birthweight group, provide another perspective on the changing composition of perinatal mortality over time.

All charts were produced using the Quattro spreadsheet on an IBM-AT compatible micro-computer.

## 4.3 Trends in Scottish Perinatal Mortality

Figure 4.1 presents annual stillbirth, neonatal and perinatal mortality rates from 1939 to 1985. In 1939 4.2 per cent of all births in Scotland were stillborn whereas 3.7 per cent of all live born babies failed to survive the first month of life. The Scottish perinatal mortality rate of 77.3 per thousand births was about 18% above that in England and Wales, primarily due to Scotland's higher neonatal mortality rate.

Scotland's neonatal mortality rate of 36.6 per thousand live births represented a static rate which had shown little improvement since 1911. During the interwar years neonatal mortality rates had ranged from a high of 39.9 (1921) to a low of 35.0 (1938). In contrast, the infant mortality rate declined from 112.5 in 1911 to 68.5 in 1939, reflecting a reduction in late infant deaths due to infections.

Over the period 1939 to 1985 the perinatal mortality rate declined by 86 per cent from 77.3 to 10.9 per thousand births. This represented an average rate of decline of -4.2 per cent per annum. By 1985 stillbirth and neonatal mortality rates were identical (5.5 per thousand), as a result of respective average annual rates of decline of -4.3 and -4.0 percent. These long

run average rates of change, however, were not uniform over this 46 year period (Table 4.1). During the war years (1939-45) both stillbirth and neonatal mortality rates declined by about 4 per cent per year. These rates of improvement were not sustained over the next two decades. Between 1955 and 1965, for example, the stillbirth rate declined by 3.1 per cent per annum whereas the neonatal mortality rate only declined by 2.1 per cent per annum. Rates of improvement began to accelerate during the late 1960's, first for stillbirths and then, particularly from 1975 onwards, for neonatal mortality rates. Over the period 1975 to 1985 neonatal mortality rates declined by 7.4 per cent per annum: a rate of decline more than double that of the previous decade (-3.0 per cent).

Another perspective on these differential rates of improvement is provided by Figure 4.2 where index numbers (1939 = 100) of perinatal mortality rates are presented alongside the proportion of overall change which occurred over time. Index numbers of stillbirth and neonatal death rates are contained in Figure 4.3 By 1945, 25 per cent of the overall decline in perinatal mortality recorded over the 1939-1985 period had already occurred. Another 25 per cent of the overall decline had occurred by 1953. Three quarters of the overall decline, however, was not registered for another 16 years in 1969 with the final quarter again taking 16

years. Thus, although 50 per cent of the overall decline in perinatal mortality had occurred in 14 years (1939-1953), the remaining 50 per cent required 32 years.

This general pattern of rapid decline in the 1940's and early 1950's followed by a relatively long period of less impressive falls in perinatal mortality during the mid-fifties and sixties which was supplanted by an accelerating rate of improvement beginning in the mid-seventies was true for both stillbirth and neonatal mortality rates (Figure 4.3). By 1957, 50 per cent of the overall declind in the stillbirth rate and 55 per cent of the overall decline in the neonatal mortality rate had occurred. By 1968, 75 per cent of the total improvement in both stillbirth and neonatal mortality rates had been achieved. Most of the remaining 25 per cent had occurred by 1979 in the case of stillbirths. Improvement in the neonatal mortality rate lagged behind that of stillbirths. However, over the period 1977-1985, an accelerating rate of decline in neonatal mortality accounted for a fifth of the overall improvement in neonatal mortality.

These rates of decline had relatively little impact on the distribution of perinatal deaths between stillbirths and neonatal deaths (Figure 4.4). Over the period 1939-1985 the proportion of perinatal deaths accounted

for by stillbirths ranged from 43.9 per cent in 1977 to 56.8 per cent in 1953. Until the early 1970's over half of the perinatal deaths in Scotland were due to stillbirths. The stillbirth proportion dropped below 50 per cent during the next decade due to the faster rate of improvement in the stillbirth rate compared to the neonatal mortality rate.[5] By 1985 perinatal deaths were equally distributed between stillbirths and neonatal deaths.

## Age Composition of Neonatal Mortality

The overall neonatal mortality rate obscures differential rates of improvement in component age-specific rates of mortality during the first four weeks after births. Age-specific morality rates are presented in Figure 4.5 for three mutually exclusive age groups (<1 day, 1-6 days and 7-27 days): corresponding annual rates of change are provided in Table 4.2. Index numbers (1939=100) of age specific neonatal mortality are presented in Figure 4.6 During the war years all

---

5. To an unknown extent this re-distribution of perinatal deaths may reflect the impact of technical change in perinatology over the last decade which encouraged earlier more active intervention amongst the babies who, instead of dying in utero, now risk dying soon after birth.

three age-specific mortality rates declined at a broadly similar rate of about 4 per cent per year. Over the next decade the <1 day rate dropped by only 1.4 per cent per year and the 1-6 day rate by just 2.6 per cent. The most dramatic change was the 10.1 per cent annual rate overall neonatal mortality over this period (1945-1955) was thus largely due to the fall in late neonatal deaths.

During the next ten years (1955-1965) annual rates of decline in mortality rates beyond the first 24 hours were about 2-3 times greater that those recorded for the first day. This differential pattern began to change over the period 1965-1975 when for the first time the drop in both < 1 day and 1-6 day mortality exceeded the fall in the late neonatal mortality rate. This new pattern continued throughout the late 1970's and early 1980's as a consequence of dramatic declines of around 8 per cent per year in both < 1 day and 1-6 day mortality rates: double the rate of change in late neonatal mortality.

These differences in growth rates had an important impact on proportional mortality rates during the neonatal period (Figure 4.7). Throughout the war years, deaths during the neonatal period were evenly distributed between the three age groups. Over the next decade, the proportion of deaths occurring after the

first week dropped from a third to less than a sixth of all neonatal deaths. The proportion of deaths between 1-6 days increased slightly from 35 per cent in 1945 to 39 per cent in 1955. In fact, throughout 1939-1985 period, deaths occurring between 1-6 days accounted for about a third of all neonatal deaths. First day deaths accounted for an increasing proportion of neonatal deaths, rising to 47 per cent in 1955. Between 1959 and 1973 just over 50 per cent of neonatal deaths occurred within the first 24 hours following birth. Beginning in 1974 this proportion of first day deaths was an increase in late neonatal deaths from 15 per cent in 1975 to 20 per cent in 1985. The proportion of deaths occurring between 1-6 days remained virtually constant over this period.

## 4.4 Perinatal Mortality: Causes of Death

### ICD-7 (1958-1967)

Figure 4.8 presents proportional mortality rates for nine broad causes of perinatal death for 1958 and 1967. These rates summarise the more detailed stillbirth list which was used to classify perinatal deaths prior to the introduction of the first "P" list in 1968. Over the period 1958-1967 there was very little change in proportional perinatal mortality rates. Placental and cord conditions, congenital malformations and maternal disease collectively accounted for two-thirds of

stillbirths. Malformations, prematurity and asphyxia accounted for 60 per cent of neonatal deaths. An increasing proportion of neonatal deaths was attributable to malformations and asphyxia over this period.

The proportion of perinatal deaths due to asphyxia nearly doubled from 7.4 per cent in 1958 to 13.6 per cent in 1967. Deaths due to atelectasis declined form 10.1 per cent to 4.9 per cent, mostly due to a reduction in this cause of neonatal mortality. Congenital malformations increased slightly from 20.7 per cent to 23.8 per cent. This was wholly the result of an increasing proportion of neonatal deaths due to malformations (18 per cent to 24 per cent) as there was a change in the proportion of stillbirths attributable to malformations.

**ICD-8 (1968-1978)**

Little change in proportional mortality rates occurred over the next decade. Figure 4.9 presents proportional perinatal mortality rates for 1968 and 1978 using a summarised version of the amalgamation of 'P' codes suggested by Edouard and Alberman (1980) in their analysis of perinatal mortality trends in England and Wales. Deaths due to difficult labour declined in importance from 4.2 per cent to 1.7 per cent of

perinatal deaths due to an improvement in stillbirth rates. There was no change in the proportion of perinatal deaths due to congenital malformations: this masked however a decline in proportional stillbirth rates and an increase in proportional neonatal mortality rates from this cause.

## ICD-9 (1979-1985)

The following six years again showed no dramatic change in the causes of perinatal death. Figure 4.10 presents cause-specific proportional perinatal mortality rates for 1979 and 1985 using a summarised version of the "P" codes introduced by ICD-9. There was a slight increase in the proportion of perinatal deaths due to placental and cord conditions, reflecting the increasing importance of this cause of stillbirths. By 1985 birth injury had virtually disappeared as a cause of perinatal death. Malformations while still an important cause of perinatal mortality accounting for about a fifth of all perinatal deaths decreased slightly due to a dramatic decline in stillbirths attributable to congenital malformations. No changes occurred in the proportion of perinatal deaths due to premature births and asphyxia.

## LINKING ICD 6-9 (1950-1985)

Using the Registrar General's bridging tabulations linking successive ICD revisions it is possible to construct a consistent time series of neonatal mortality rates for selected causes of death. Figure 4.11 presents cause-specific neonatal mortality rates for congenital malformations, prematurity and asphyxia over the period 1950-1985. These are the only important causes of neonatal mortality that can be feasibly documented using a linked series of data based on the 6th to 9th revisions of the ICD.

Neonatal mortality rates from congenital malformations remained virtually constant at around 3.5 per thousand until the mid 1970's when the rate began to fall, reaching a low of 1.8 per thousand in 1985. Mortality associated with prematurity declined form 6.2 per thousand in 1950 to 0.5 per thousand in 1985. Mortality rates from asphyxia showed little sign of improvement during the 1950.s and early 1960's, averaging about 5-6 per thousand. The mortality rate from asphyxia ranged form 2.8 to 3.7 during the 1970's and fell to around 2 per thousand by 1983-85.

Table 4.3 provides a breakdown of average annual rates of change in these cause-specific neonatal mortality rates. The greatest overall decline over this period occurred in the death rate due to prematurity (-6.9 per cent), particularly over the 1975-1985 decade when

average rates of decline accelerated to -11.5 per cent per year. The long run rate of decline in neonatal death rates due to congenital malformation was less than 2 per cent. Over the 1975-1985 period the congenital malformation death rate declined by just under 5 per cent per year. During this decade neonatal death rates due to asphyxia also decreased at a rate which exceeded its long run average decline.

These differential rates of decline in cause-specific neonatal mortality are compared in Figure 4.12 which provides index numbers (1950=100) of mortality rates over the period 1950-1985. The mortality rate from congenital malformations fluctuated around its 1950 level until 1978 when it finally dropped and stayed below its long run trend. Prematurity declined steadily as a cause of neonatal mortality in contrast to asphyxia which showed no significant improvement until the mid 1970's. The impact of these trends on proportional mortality rates is illustrated in Figure 4.13. The proportion of neonatal deaths due to malformations increased from 15 per cent in 1950 to 33 per cent in 1985. A similar pattern emerged in deaths due to asphyxia. Prematurity accounted for a declining proportion of neonatal deaths: 27 per cent in 1950 versus 9 per cent in 1985.

ABERDEEN CLASSIFICATION OF PERINATAL DEATH 1938-1976

Prior to 1977, the use of the Aberdeen classification in Scotland was confined to local or hospital based populations in Aberdeen, Dundee and Glasgow. Although the populations are not directly comparable the major changes over time were an increase in the proportion of deaths due to congenital malformations and a decrease in proportional mortality rates due to birth trauma and maternal disease. Unexplained low birthweight (< 2.5 kg) accounted for between one fifth and one quarter of all perinatal deaths in these populations. No changes occurred in the proportion of deaths due to antepartum haemorrhage.

## NATIONAL SURVEYS USING THE ABERDEEN CLASSIFICATION 1977-1985

Although the Aberdeen classification was used, alongwith a detailed pathological classification, in the 1958 British Perinatal Mortality Survey, it was not employed at the national level in Scotland until 1977 (McIlwaine et al, 1979). The Scottish perinatal mortality survey of 1977 was the first in a series of national inquiries into all perinatal deaths. Since 1979 successive inquiries have generated a short but consistent time series on the causes of perinatal death classified according to the Aberdeen set of clinical factors.[6]

Figure 4.14 presents mortality rates for eight clinical causes of perinatal death over the period 1977-1985. Perinatal death is defined as stillbirths and first week deaths. Proportional mortality rates are provided in Table 4.4. The principal changes were an increase in the proportion of deaths due to unknown causes in mature (i.e., birthweight > 2.5 kg) babies, a decrease in malformations and a slight increase in antepartum haemorrhage. No change occurred in the proportion of deaths attributable to unknown factors in low birthweight infants. One feature of the classification is that about 40 per cent of perinatal deaths have no readily identifiable cause. These changes in proportional mortality rates reflected differential rates of improvement in (Aberdeen) cause-specific perinatal mortality (Table 4.5). The greatest relative improvements occurred in deaths attributable to trauma, congenital malformation, maternal disease and toxaemia. Mortality rates within the unknown cause category (birthweight < 2.5 kg) declined in line with the overall

---

6. The Information and Statistics Division of the Common Services Agency of the Scottish National Health Service assumed responsibility in 1983 for conducting what evolved into a routine national survey of perinatal deaths in Scotland.

improvement in perinatal mortality from all causes whereas death rates from antepartum haemorrhage and unknown causes in mature babies lagged behind.

## 4.5 BIRTHWEIGHT SPECIFIC PERINATAL MORTALITY

Early data on the relationship between birthweight and perinatal mortality in Scotland tended to be hospital based and selectively presented. The distinction between booked and non-booked cases was obscure, cases were sometimes excluded below some arbitrary birthweight and data was often not available for stillbirths. Baird (1945) reported a neonatal survival rate of 0.743 in low birthweight infants booked for delivery and born in Aberdeen Maternity Hospital between 1938 and 1944. The average survival rate in babies who weighed more than 2.5 kg was 0.983. The heavier infants had a 32 per cent greater chance of survival. Expressed in terms of mortality rates, the unadjusted relative risk of neonatal death in low birthweight infants compared to normal weight infants was 15.2. Less than one third of babies weighing less than 1588 g (3.5 lb.) survived the neonatal period.

Two early studies reviewed the experience of total hospital populations where both booked and non-booked ("emergency") cases were investigated. Thomson (1952) reported perinatal mortality rates by birthweight groups

in an analysis of the influence of the varying incidence of singleton and twins, low birthweight and booked and non-booked cases on the perinatal mortality rate of the Simpson Memorial Maternity Pavillion in Edinburgh (SMMP) over the period 1939-40 to 1948-51. By 1948-51 the perinatal mortality rate for low birthweight births had fallen to 390.4 per thousand or 73 per cent of the 1939-40 rate of 533.3 per thousand. The perinatal mortality rate for normal weight births fell even more rapidly from 65.6 to 33.7 per thousand. As result the relative risk associated with low birthweight increased from 8.1 to 11.6 over this period.

Comparable neonatal mortality data based on total hospital cases is available for the Glasgow Royal Maternity Hospital beginning in the early 1950's (Glasgow Royal Maternity Hospital, various years). During 1951-52 the neonatal mortality rates within this hospital population were 7.8 per thousand normal weight live births and 210 per thousand low birthweight live births. The low birthweight survival rate of 79 per cent was similar to that recorded at the SMMP during 1942-45 (Drillien, 1947). The relative risk of neonatal mortality in low birthweight infants compared to normal weight infants was also very close in the two populations (27.0 versus 24.1).

## EARLY NATIONAL SURVEYS

National statistics on birthweight specific neonatal mortality in Scotland were first collected in 1951 as result of a special survey conducted under the auspices of the Chief Medical officer for Scotland (Douglas and McKinley, 1953). No detailed breakdown of birthweight was reported as infants were simply divided into premature and full-term groups. First month mortality rates (per 1000 live births) were 190.4 in the premature group, 13.5 in the full-term group and 22.3 for all babies. The ratio of first month mortality rates (premature:full-term) was 14.1 This ratio varies throughout the first month from 11.1 for deaths during the first 24 hours to 20.6 (days 1-6) and 10.2 (days 7-28).

A second national survey occurred in 1954 (Douglas and McKinley, 1955). No further breakdown of birthweight is available but unlike the 1951 survey information was collected on both still and live births which permitted the calculation of national birthweight specific perinatal mortality rates. The perinatal mortality rate amongst premature births was 428.1 per thousand or about 18 times the rate of 23.9 in full term births, the still birth rate was about 17 times greater for premature births (232 versus 13.7). The principal difference in the perinatal mortality rates was relative mortality during the first month following birth where

the premature births faced a risk of dying nearly 25 times that of the full-term babies (255.0 versus 10.4). The relative risk of mortality associated with prematurity increased from 14.6 during the first 24 hours to 57.8 over days 1-6 and dropped to 13.8 over the remainder of the neonatal period.

## BIRTHWEIGHT SPECIFIC PERINATAL MORTALITY 1963-1984

Figure 4.15 presents birthweight specific perinatal mortality rates for Scotland over the period 1956 to 1985. Prior to 1975 little improvement occurred in perinatal mortality rates recorded for infants weighing less than 1501 g. Most of the improvement in perinatal mortality was due to a uniform decline in mortality in the heavier infants. Perinatal mortality declined in all birthweight groups over the next decade, initially in the heavier birthweights and then with a lag in increasingly lighter infants.

Reviewing the 1956-85 period as a whole it can be seen that the greatest relative improvement in perinatal mortality occurred in the heavier infants weighing more than 2500 g (Figure 4.16). Relative improvement was directly related to birthweight; heavier infants recorded progressively greater survival during the perinatal period. However, over the past 5 years

chances of survival have increased most radically for the very low birthweight infant. In 1963 only 2 out of 10 infants weighing less than 1000 g could expect to survive; by 1985 survival in this weight category increased to 4 out of 10.

Although the very low birthweight infant has encountered decreasing absolute risk of mortality over the past few years, relative risks of mortality have increased over the past two decades (Figure 4.17). Using the infants weighing more than 2500 g as a reference group, the relative risk of perinatal mortality increased steadily for the very low birthweight infants weighing less than 1500 g, particularly for the extremely low birthweight baby (< 1000 g). This trend seems to be reversed since 1980. Very little change occurred in the relative risk of mortality in infants weighing between 1.5 and 2.5 kg.

## 4.6 Summary

Throughout the post-war period perinatal mortality has fallen in most, if not all, industrialised countries. Scotland is no exception. Over the period 1939-1985 Scotland sustained an annual decline in her perinatal mortality rate of just over 4 per cent per year: a rate of improvement twice as great as her post-war annual rate of economic growth (2.1 per cent).

Perinatal mortality has not declined at a constant rate over this period. Although impressive improvement occurred during the war years the 1950's and 1960's were characterised by sluggish rates of decline. The most rapid change in perinatal mortality rates occurred over the period 1975-1985 where the annual rate of change increased to 7 per cent. Although improvement in stillbirth rates tended to precede that of neonatal mortality rates, the general pattern of decline over time in perinatal mortality was true for both stillbirths and neonatal deaths.

For decades Scottish infants who fail to survive the neonatal period have contributed close to 50 per cent of perinatal deaths. The falling neonatal mortality rate reflects differential improvement in age-specific survival in early infancy. The time pattern of improvement was directly related to age at death. Improvement occurred first in the late neonatal mortality rate (7-27 days), followed by the 1-6 day rate and finally the < 1 day rate. By 1954 most of the decline (80 per cent) in late neonatal mortality had occurred reflecting rapid improvement during the 1940's and early 1950's. In contrast, it took another two decades until a similar proportion of the overall decline in the < 1 day and 1-6 day mortality rates had been accounted for. Throughout the late 1950's and early 1960's the < 1 day rate remained virtually static.

Although rates improved during the late 1960's, over 25 per cent of the overall decline in the < 1 day mortality rate occurred between 1976 and 1985. The improvement in the 1-6 day rate preceded that of the < 1 day rate, but lagged behind the late neonatal mortality rate.

Despite changes in successive classifications three major causes of perinatal mortality (congenital malformations, placental and cord conditions and problems of prematurity and respiration) can be reviewed over the period 1958-1985. Congenital malformations declined in importance as a cause of stillbirths. In 1958 just over one fifth of stillbirths were attributed to malformations; by 1985 this proportion had dropped to 10 per cent. The proportion of stillbirths associated with placental and cord conditions remained constant at around one third. Asphyxia accounted for an increasing proportion of stillbirths.

Congenital malformations and the problems associated with prematurity and respiratory function have accounted for between 63 to 74 per cent of neonatal deaths between 1958 and 1985. The major trends are the increasing importance of congenital malformations and the declining proportion of neonatal deaths due to prematurity and respiratory problems.

These trends in cause-specific stillbirth and neonatal mortality tended to exert a countervailing influence on each other. Thus, although the proportion of stillbirths due to malformations has declined this has been accompanied by an increase in the proportional neonatal mortality rate for malformations. As a result, the principal causes of perinatal death remained remarkably constant. For nearly three decades, congenital malformations have accounted for 20-24 per cent of perinatal deaths. Placental and cord conditions have been responsible for another 17-20 per cent of perinatal deaths. Finally, prematurity and respiratory problems have accounted for about a third of perinatal deaths.

In Scotland the only consistent time series of national data on the causes of perinatal mortality which provides an alternative to the ICD classification is the obstetric classification introduced by Baird and his co-workers in the 1950's. The Aberdeen classification has occasionally been cross-tabulated against other perinatal classification based on pathological findings. No study has compared the certified causes of perinatal death as recorded by the Registrar General using the ICD "P" codes against the Aberdeen classification. Although a complete cross-tabulation is outwith the scope of the

present study, it is possible to make comparisons across broad categories of perinatal death using the information presented in this chapter.

The general trend in the 1980's in the declining proportion of perinatal deaths due to congenital malformations as defined by the Aberdeen classification and ICD-9 are in close agreement. Similarly, the proportion of deaths due to premature birth and asphyxia coded using ICD-9 (32.8 per cent) is fairly close to the proportion of unknown cause (birthweight < 2.5 kg) deaths defined by the Aberdeen classification.

Birthweight is one of the most important factors influencing perinatal mortality. Although Scottish data on birthweight specific perinatal mortality is scarce in the 1940's and 1950's the 1954 survey data reported by Douglas and McKinlay (1955) can be linked with more recent information. The perinatal death rate (stillbirths and first week deaths) was 408.1 per thousand premature births compared to 22.1 per thousand premature births in 1954. The relative risk of perinatal mortality in the premature babies was 18.5. Ten years later this relative risk stood at 17.6 confirming a similar rate of improvement in perinatal mortality in premature and mature infants. Average

annual rates of decline were -4.9 per cent in premature and -4.4 per cent in mature infants over the period 1954-1963.

Over the next two decades, however, the average annual rate of decline dropped to -4.1 per cent in premature births but rose to -6.6 per cent in mature births. These divergent rates of improvement lay behind the increasing relative risk of perinatal mortality in the premature infant. Thus although the risk of perinatal mortality declined in all birthweight groups the most impressive gains occurred not in those infants facing the greatest risks of death but rather in the heavier infant who apparently benefited most from the concurrent changes in the social and medical determinants of perinatal mortality in Scotland.

Table 4.1 Average Annual Percentage Rates of Change in Scottish Perinatal Mortality Rates: 1939 - 1985

| Period | Stillbirth Rate | Neonatal Mortality Rate | Perinatal Mortality Rate |
|---|---|---|---|
| 1939-45 | -4.1 | -4.0 | -4.0 |
| 1945-55 | -2.9 | -3.6 | -3.2 |
| 1955-65 | -3.1 | -2.1 | -2.6 |
| 1965-75 | -4.6 | -3.0 | -3.8 |
| 1975-85 | -6.9 | -7.4 | -7.1 |
| 1939-85 | -4.3 | -4.1 | -4.2 |

Averages based on geometric means.

Table 4.2 Average Annual Percentage Rates of Change in Scottish Neonatal Mortality Rates: 1939 - 1985

| Period | < 1 day Rate | 1-6 Day Rate | 7-27 Day Rate | Neonatal 0-27 Day Rate |
|---|---|---|---|---|
| 1939-45 | -3.5 | -4.4 | -4.3 | -4.0 |
| 1945-55 | -1.4 | -2.6 | -10.1 | -3.6 |
| 1955-65 | -1.2 | -3.1 | -2.5 | -2.1 |
| 1965-75 | -3.5 | -2.7 | -2.0 | -3.0 |
| 1975-85 | -8.3 | -7.9 | -4.0 | -7.4 |
| 1939-85 | -3.6 | -4.2 | -4.7 | -4.1 |

Averages based on geometric means.

Table 4.3 Average Annual Percentage Rates of Change in Cause-specific Neonatal Mortality Rates: 1950 - 1985

| Period | Malformation Rate | Prematurity Rate | Asphyxia Rate | Other Causes Rate |
|---|---|---|---|---|
| 1950-65 | 0.2 | -4.9 | 4.0 | -3.3 |
| 1965-75 | -1.5 | -5.2 | -5.5 | -5.5 |
| 1975-85 | -5.0 | -11.5 | -8.0 | -7.9 |
| 1950-85 | -1.8 | -6.9 | -2.3 | -5.3 |

Averages based on geometric means.

Table 4.4 Cause-specific Perinatal Mortality
Aberdeen Classification (proportional mortality rates)

| Aberdeen Cause | Year 1977 | 1979 | 1981 | 1983 | 1985 |
|---|---|---|---|---|---|
| Unknown Birthweight < 2.5kg | 29.8 | 31.0 | 31.3 | 28.9 | 28.9 |
| Unknown Birthweight > 2.5kg | 10.5 | 13.1 | 11.8 | 15.5 | 14.4 |
| Malformation | 26.2 | 25.9 | 23.3 | 22.7 | 18.9 |
| Toxaemia | 8.8 | 7.7 | 8.6 | 5.2 | 7.8 |
| Antepartum Haemorrhage | 12.5 | 12.4 | 12.2 | 15.5 | 16.7 |
| Maternal Disease | 4.4 | 3.7 | 3.7 | 4.6 | 5.2 |
| Trauma | 4.9 | 3.2 | 3.2 | 3.5 | 2.1 |
| Other Causes | 2.9 | 2.3 | 2.3 | 3.1 | 5.6 |
| All Causes | 100 | 100 | 100 | 100 | 100 |

Table 4.5 Cause-specific Perinatal Mortality
Aberdeen Classification (Average Annual Rates of Change)

| Aberdeen Cause | 1977-85 |
|---|---|
| Unknown Birthweight < 2.5kg | -7.0 |
| Unknown Birthweight > 2.5kg | -3.3 |
| Malformation | -10.7 |
| Toxaemia | -8.3 |
| Antepartum Haemorrhage | -3.5 |
| Maternal Disease | -10.0 |
| Trauma | -11.5 |
| All Causes | -7.0 |

**Figure 4.1 Scottish Perinatal Mortality: 1939-1985**

**(rate per 1000 total births)**

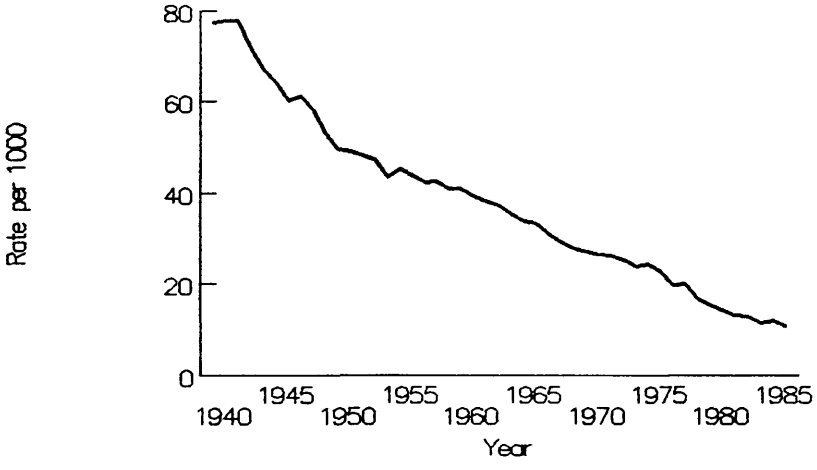



**Figure 4.2 Scottish Perinatal Mortality: 1939-1985**

**Index Numbers and Proportionate Decline**

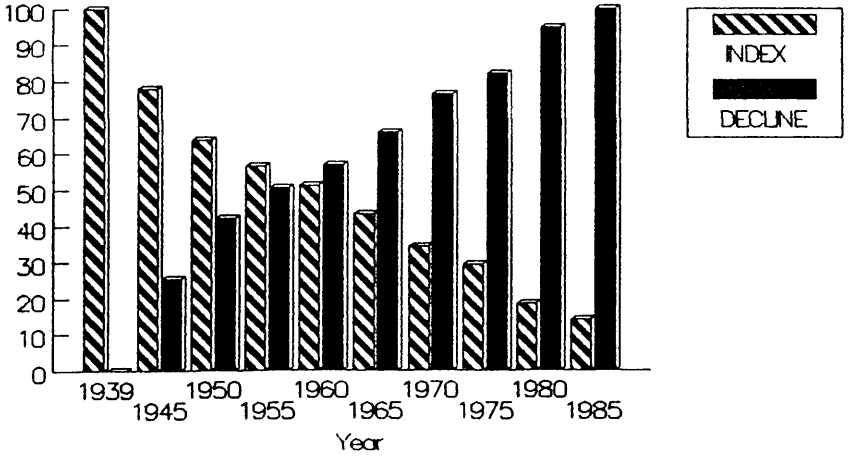


**Figure 4.3 Stillbirth and Neonatal Death Rates**

**(Index Numbers 1939=100)**

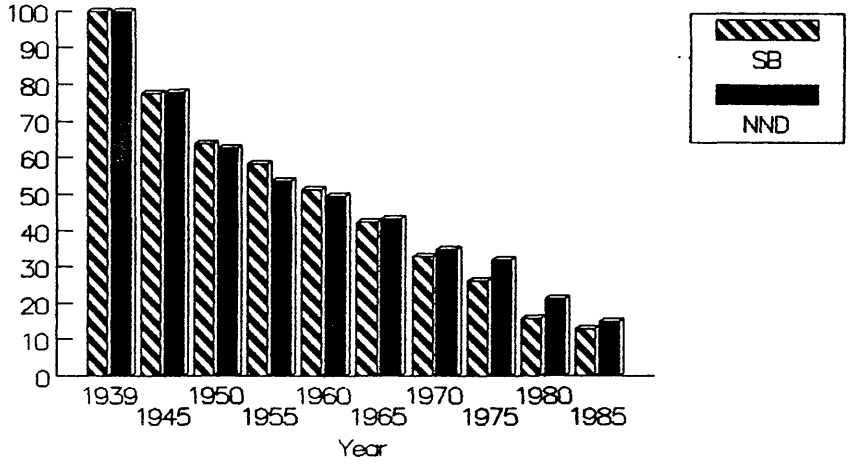
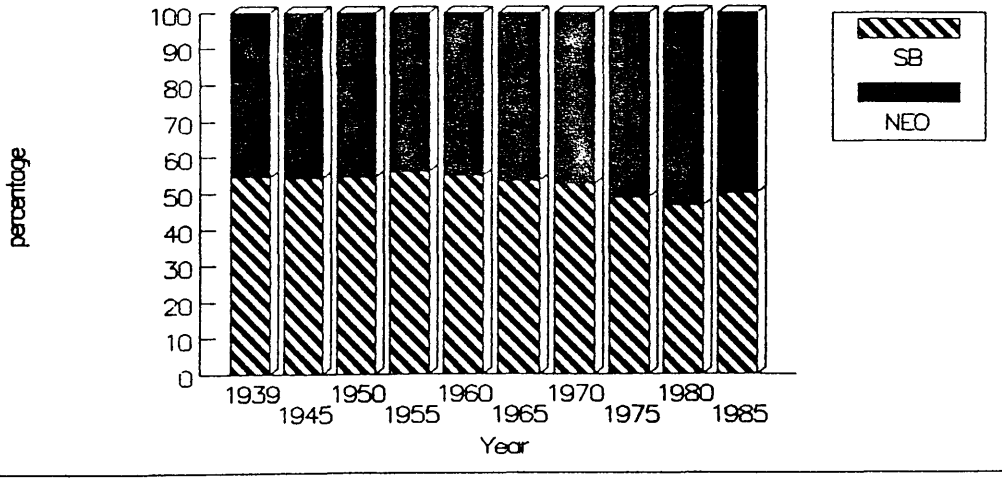


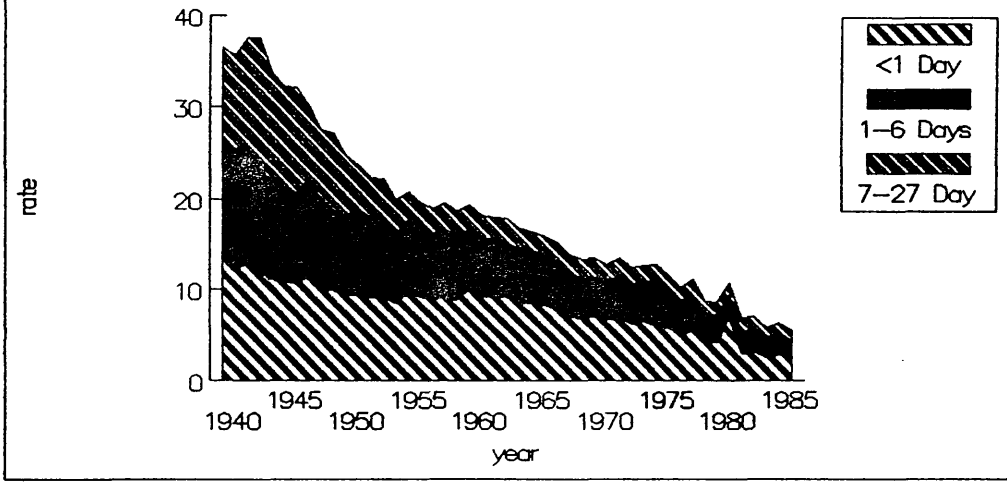

**Figure 4.4 Scottish Perinatal Deaths**

**Stillbirth and Neonatal Death Breakdown**



## Figure 4.5 Age-specific neonatal mortality

## (cumulative rate per 1000 live births)




## Figure 4.6 Neonatal Mortality Rates

## (Index Numbers 1939=100)

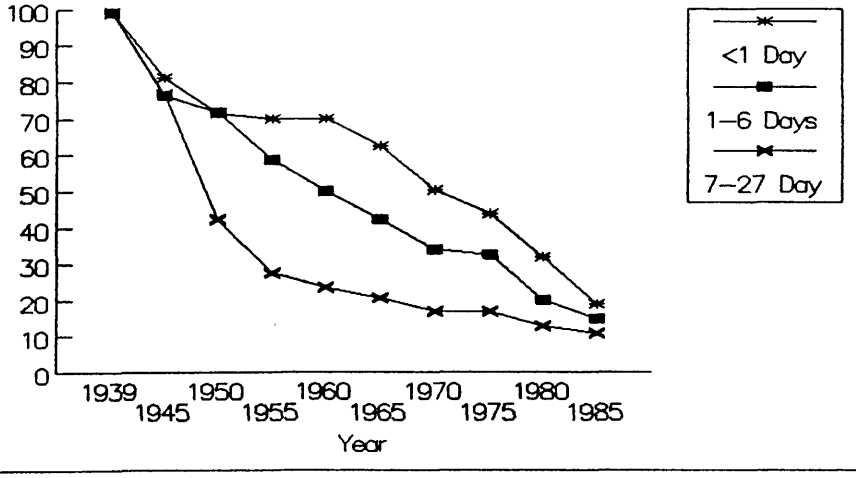


**Figure 4.7 Age Composition of Neonatal Mortality**

**(percentages)**

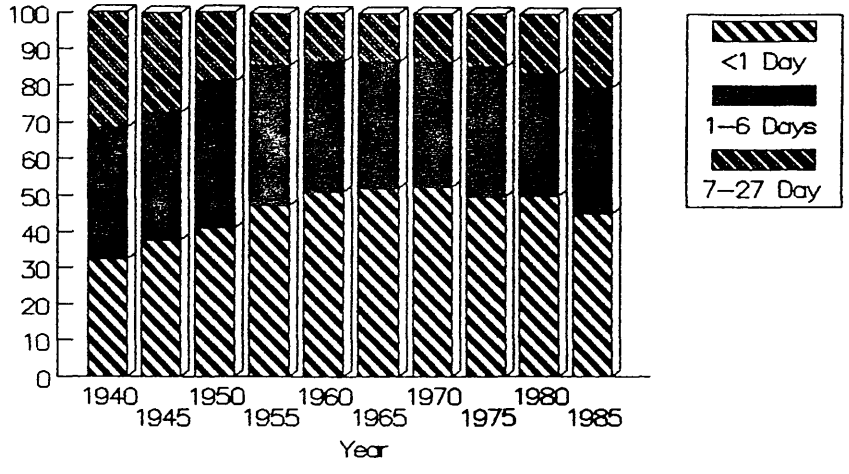
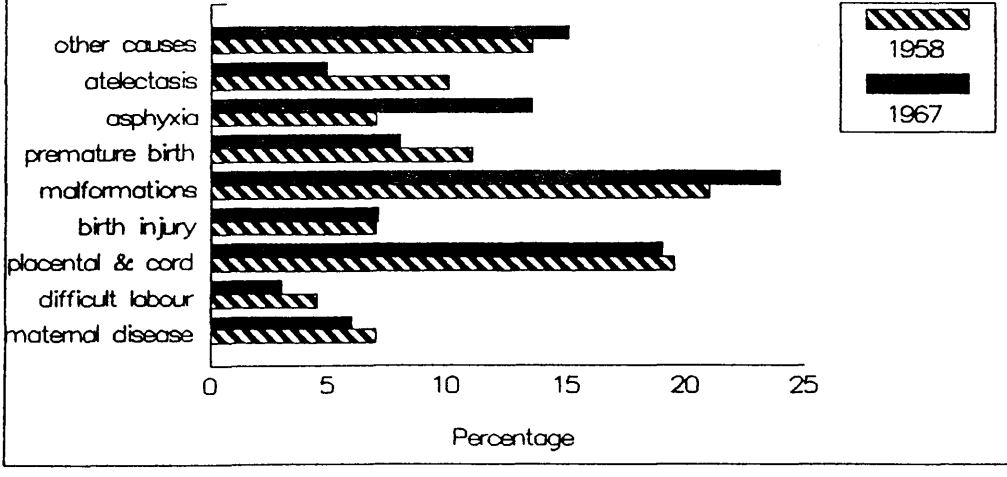


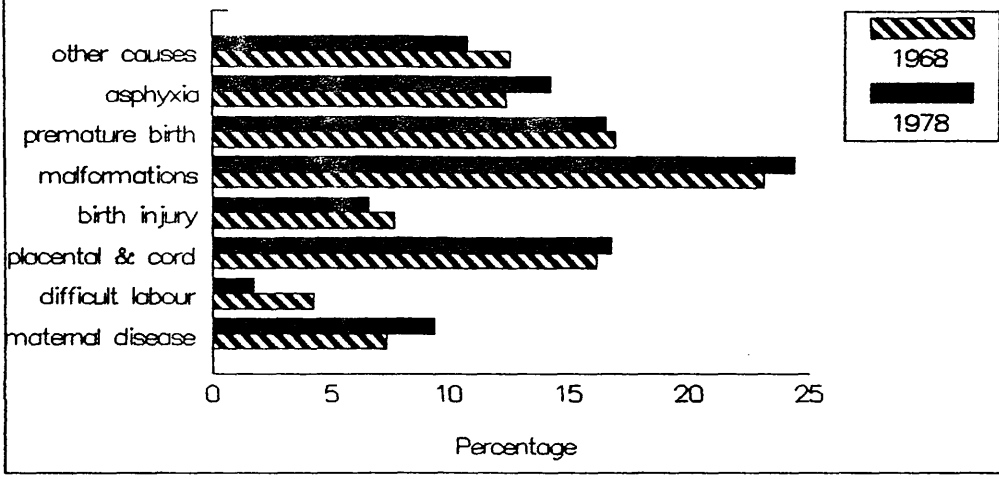

**Figure 4.8 Proportional Mortality Rates**

**(ICD 6 & 7: Perinatal Mortality)**



## Figure 4.9 Proportional Mortality Rates

## (ICD 8: Perinatal Mortality)




## Figure 4.10 Proportional Mortality Rate

## (ICD 9: Perinatal Mortality)

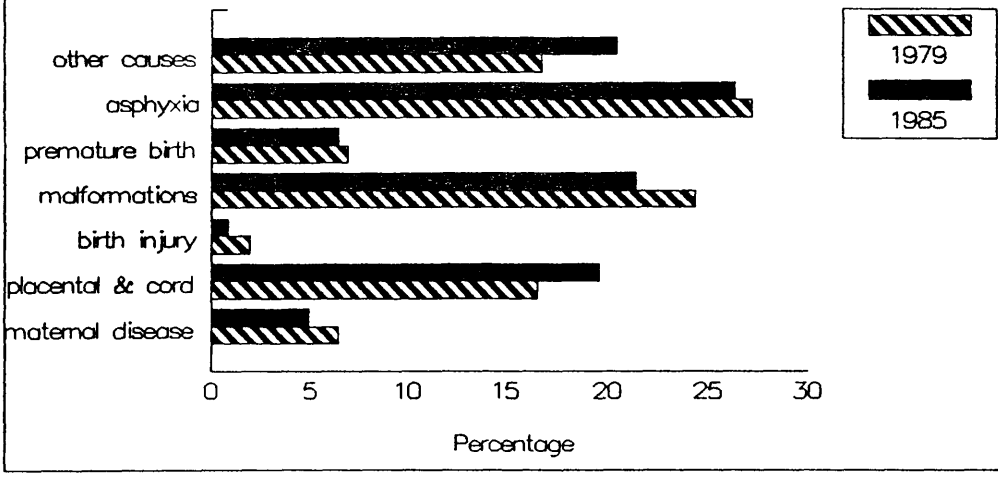


**Figure 4.11 Neonatal Mortality: 1950-85**

**Cumulative cause-specific rates**

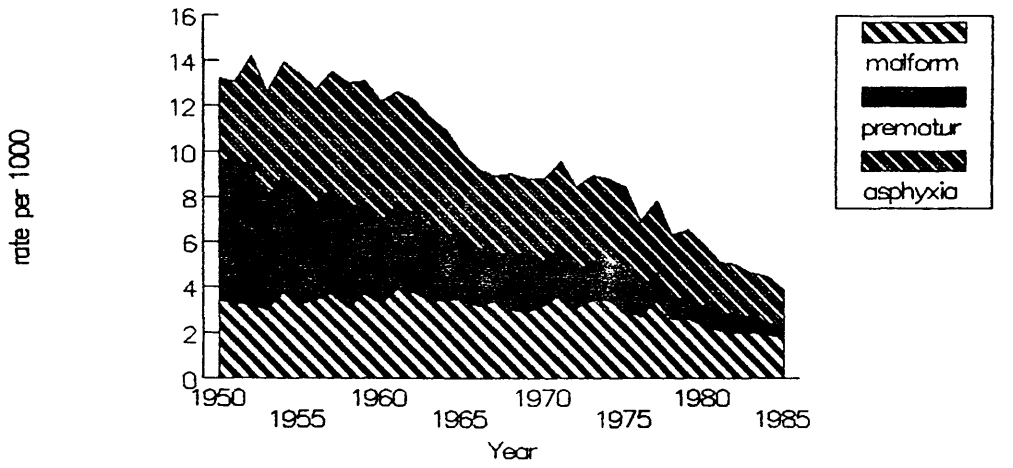
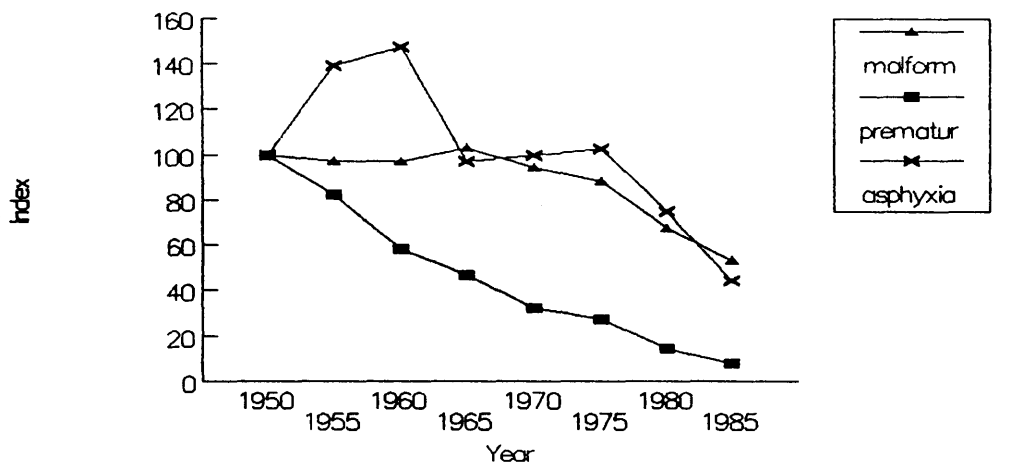


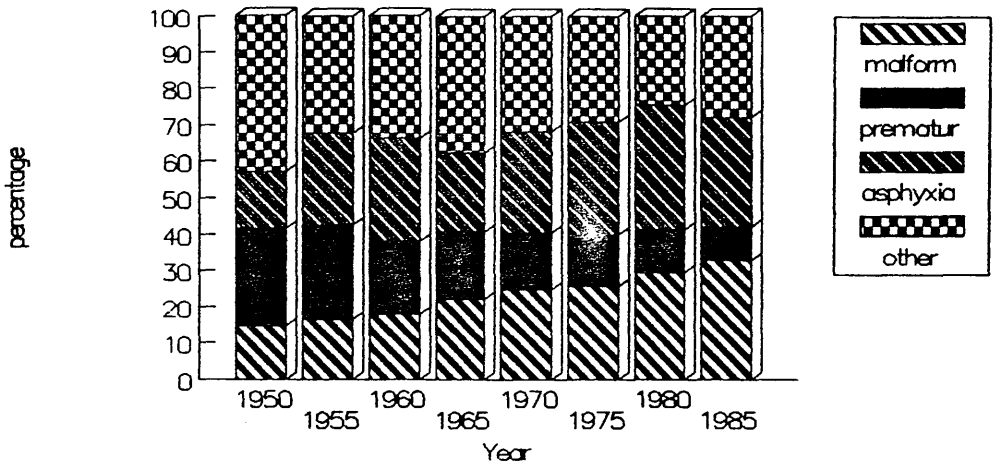

**Figure 4.12 Neonatal Mortality: 1950-85**

**Cause-specific rates (1950=100)**



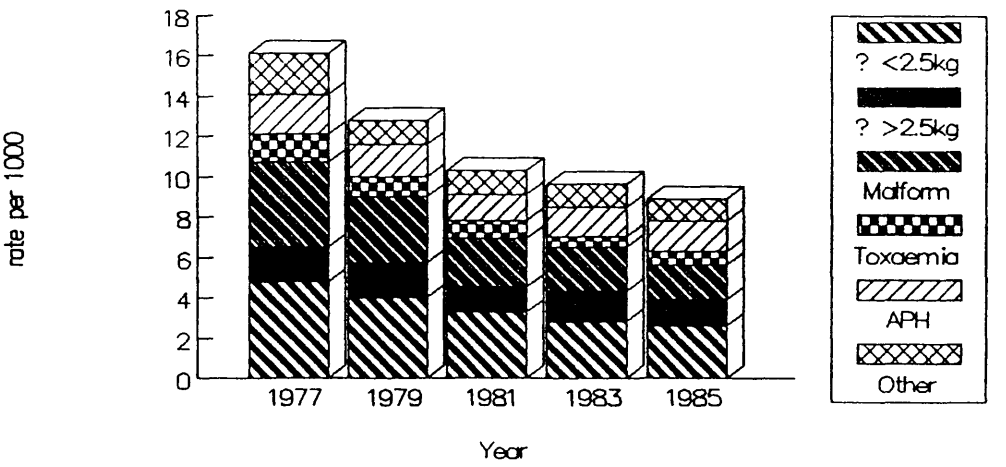
## Figure 4.13 Causes of Neonatal Death

### (cumulative proportional mortality)



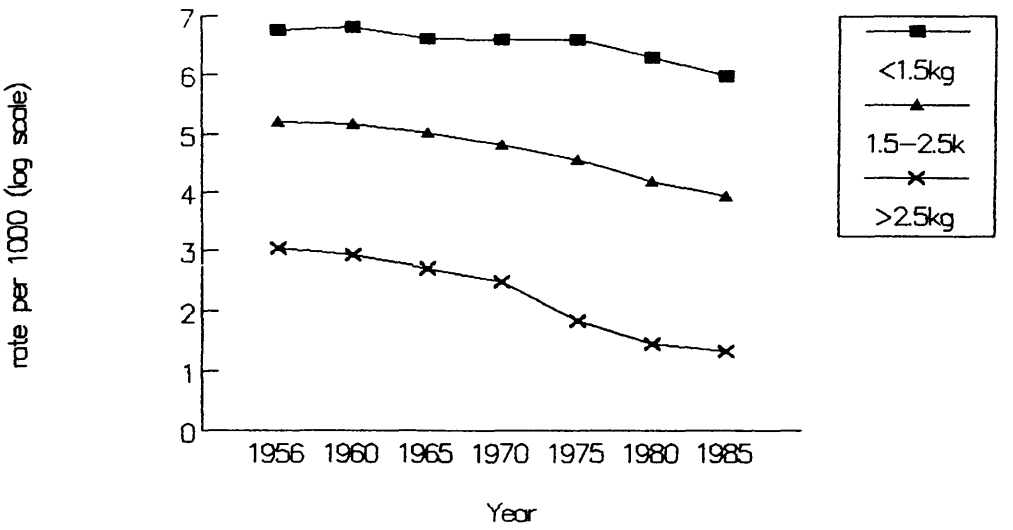

## Figure 4.14 Cause-specific Perinatal Mortality

### (Aberdeen Classification)



## Figure 4.15 Birthweight Specific Perinatal Mortality

## (Rate per 1000: Log Scale)




## Figure 4.16 Birthweight Specific Perinatal Mortality

## Rate per 1000 (Index 1956=100)

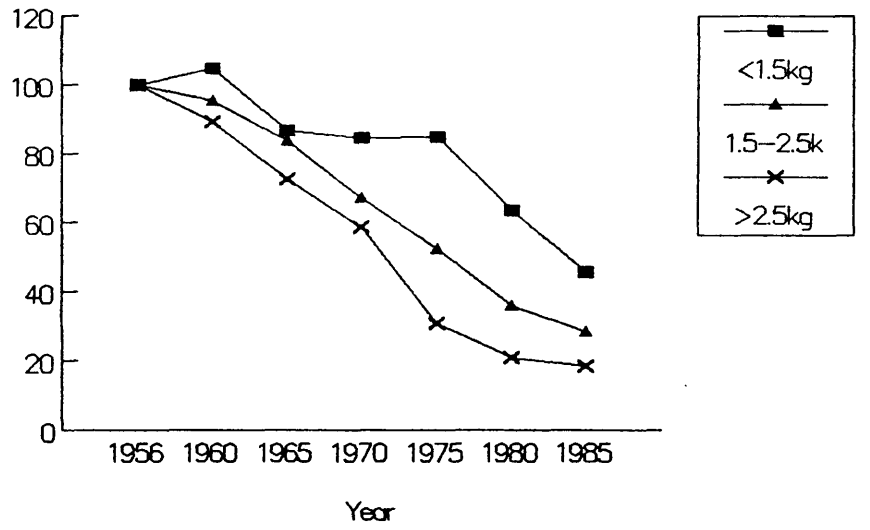


**Figure 4.17 Relative Risk of Perinatal Mortality**

**Reference Group = Birthweight > 2.5kg**

# CHAPTER 5 THE DEMAND FOR PERINATAL HOSPITAL CARE

## 5.1 INTRODUCTION

This chapter presents the results of estimating a demand equation for perinatal hospital services in Scotland. Using a version of the demand model introduced by Deaton and Muellbauer (1980) the perinatal hospital service demand equation is specified in terms of the share of NHS hospital expenditure devoted to the perinatal hospital sector which is linearly related to the logarithm of real (price deflated) NHS hospital expenditure, the logarithms of hospital prices and the logarithms of various policy targets and indicators of the need for perinatal services.

The specification described in Section 5.2 attempts to explain the variance of the perinatal budget share rather than the level of expenditure on perinatal hospital services. The construction of a perinatal budget share requires data on perinatal hospital expenditure, total hospital expenditure and a relevant price deflator for NHS expenditure. Section 5.3 describes the derivation and measurement of an expenditure series on perinatal hospital expenditure and NHS hospital expenditure in Scotland over the

period 1951-1985. Section 5.4 outlines the price series that is employed to generate a series of real expenditure on hospital services in Scotland.

Although the demand for perinatal hospital services is inevitably constrained by the overall level of hospital expenditure it seems unnecessarily restrictive to assume that demand depends only on income or total expenditure. The concept and measurement of relative prices in the hospital sector and their role in the demand equation is examined in Section 5.5.

In addition to income and prices, the demand for perinatal hospital care may be influenced by evolving policy objectives and perceptions of the need for such services. Section 5.6 and the Appendix outlines the Scottish evolution of hospital bed supply and expenditure targets for perinatal care and illustrates how they can be introduced into the demand equation. Indicators of population need for perinatal hospital care are discussed in Section 5.7. Section 5.8 presents the results of estimating and testing different specifications of the demand model. Conclusions are presented in Section 5.9.

## 5.2 The AI Demand Model

Within the public health sector the principal endogenous variable is expenditure on perinatal hospital services. Following demand models developed for analyzing expenditure on public (government) programmes[1] expenditure on perinatal hospital services is viewed as conditional on relative programme prices, total expenditure (income), policy objectives and population need indicators. Expenditure on perinatal hospital services E, is given by a demand function taking the general form

$$E = f(P, X, N,) \qquad (5.1)$$

where P is a vector of health programme prices, X is total public expenditure on health services and N is a vector of policy objectives and population need indicators.

---

1. For examples of public expenditure demand studies see Deacon (1978); Smith (1980); Dunne and Smith (1983); Dunne, Pashardes and Smith (1984) and Hulten (1985).

A variety of functional forms could be used to specify equation 5.1. Using the Almost Ideal (AI) demand system introduced by Deaton and Muelbauer (1980), the demand function can be written in budget share form as

$$w_i = \theta_i + \Sigma_j \delta_{i\,j} \ln P_j + \beta_i \ln(X/P^*) \qquad (5.2)$$

where the *i*th budget share of total health care expenditure is given by

$$w_i = E/X \qquad (5.3)$$

$P_j$ is the price of service j, X is total health care expenditure and $P^*$ is a share weighted price index (Stone, 1953) defined as

$$\ln P^* = \Sigma_j \, w_j \ln P_j \qquad (5.4)$$

used to deflate total expenditure.

Policy targets and population need indicators $N_k$ can be introduced into the demand equation by expressing the intercept in 5.2 as

$$\theta_i = a_{0i} + \Sigma_k a_{ik} \ln N_k \qquad (5.5)$$

Substitution of 5.5 in 5.2 gives

$$w_i = a_{0i} + \Sigma_k a_{ik} \ln N_k + \Sigma_j \delta_{ij} \ln P_j + \beta_i \ln(X/P^*) \qquad (5.6)$$

which is the basic estimation equation for the demand function 5.1.

Interpretation of equation (5.6) is straightforward. Changes in population characteristics influence demand via the terms $a_{ik}$. Thus a change in fertility could influence the government's perceived need for perinatal hospital services which could be expressed in a shift of health sector resources in favour of perinatal services. The effect of changes in relative prices on demand is measured through the $\delta_{ij}$ terms where each $\delta_{ij}$ represents $10^2$ the effect on the $i$th budget share of a 1 percent change in the $j$th price with real expenditure X/P* held constant. The $\beta_i$ terms measure the impact of changes in real expenditure on demand. In the absence of changes in population characteristics, relative prices and real expenditure, budget shares are constant.

This specification allows for changes in budget shares, even when relative prices and real expenditure are constant, via the impact of changing population characteristics on government demand for health programmes and perinatal hospital services. A further point regarding the AI specification concerns its generality. Demand functions taking the form (5.6) are first-order approximations to any set of demand functions derived from utility-maximizing behaviour. However, as Deaton and Muelbauer (1980) stress, even if maximizing behaviour is not assumed but demands are simply regarded as continuous functions of prices and total expenditure the AI demand function still provides a first-order approximation. This last characteristic is particularly attractive since it enables the analysis of demand systems in the public sector where the assumption of maximizing behaviour may be suspect or more difficult to sustain.

## 5.3 PERINATAL BUDGET SHARE

### 5.3.1 Total Expenditure or Budget Shares?

Most studies of health care expenditure focus on total expenditure or some decomposition of expenditure into price and quantity components (Evans and Bauer, 1983).

In contrast, the dependent variable in the demand equation is the budget share of total NHS hospital spending which is devoted to perinatal care. The principal reason for this budget share specification is that it provides a sensible characterisation of the allocation process whereby total NHS expenditure is (re)distributed amongst competing programmes over time. Thus, in the absence of changes in total expenditure, relative prices, policy targets and need indicators the budget shares are constant. Changes in budget shares over time should reflect changes in any or all of these factors.

Two further econometric arguments can also be forwarded for this specification of the dependent variable in demand equations. First, heteroskedasticity is less likely to occur in equations which regress budget shares as opposed to quantities or expenditures. For example, if the dependent variable was total expenditure on perinatal hospital care the assumption of homoskedasticity is untenable since the rising trend in perinatal hospital expenditure is likely to be accompanied by a rising trend in the variance of the error terms. Second, the post-war increase in expenditure on hospital perinatal care ensures extremely high "explanatory

power" in poorly specified models which regress quantities or expenditures on aggregate variables which themselves have trended upwards over time.

Although the advent of programme budgeting in the NHS has resulted in the routine calculation of budget shares allocated to broad categories of expenditure such series only begin in the mid 1970s. Furthermore, no programme budget has ever been reported for the perinatal hospital sector in Scotland. The share of total NHS hospital expenditure allocated to the perinatal hospital sector was thus calculated as follows.

### 5.3.2 Expenditure on perinatal hospital care

Total expenditure on perinatal hospital services was defined to include both in-patient and out-patient care. In-patient expenditure on perinatal hospital services was computed by multiplying the average cost per in-patient discharge in maternity hospitals by the number of obstetric discharges in all NHS hospitals. Average cost per discharge was weighted to reflect the changing cost structure and mix of maternity hospitals over time. Obstetric in-patient discharges include both antenatal and postnatal discharges which typically exceeds the number of hospital births. Out-patient expenditure was computed

by multiplying the average cost per out-patient attendance in maternity hospitals by the estimated number of obstetric out-patient attendances in all NHS hospitals. Data on maternity hospital costs was obtained from the annual NHS (Scotland) accounts presented to Parliament and annual reports on Hospital Running Costs published by the Scottish Home and Health Department (SHHD). Numbers of obstetric in-patient discharges and out-patient attendances were extracted from Scottish Health Statistics.

Hospital cost data and patient activity data have traditionally been reported on a different yearly basis in Scotland. Given the six month difference in year ends (31 March for cost and expenditure data and 30 September for patient activity) and the need to ensure consistency with the data on perinatal mortality reviewed in Chapter 4 all data was rescaled to calender years.

Table 5.1 presents in-patient, out-patient and total hospital expenditure on perinatal care in Scotland over the period 1951 to 1985. All expenditure is reported in current prices with no adjustment for price changes over time. Over this period total expenditure on perinatal hospital care increased at an average rate of 11.3 per cent per year. Most of this

increase over time was not due to an increase in inpatient cases or outpatient activity but rather the increase in expenditure per case and per attendance. The number of inpatient cases actually reached their post-war peak in 1971.

### 5.3.3 NHS Hospital Expenditure

NHS hospital expenditure enters both sides of the demand equation: as the denominator in the budget share and as a constraint on the demand for perinatal hospital services. Data on NHS hospital expenditure was obtained from the annual NHS (Scotland) Accounts submitted to the House of Commons. As the expenditure on perinatal hospital services is derived from the estimated running costs of maternity hospitals, capital expenditure, which accounts for about 10 per cent of total hospital expenditure, is excluded from both the denominator of the budget share variable and the overall hospital budget constraint. Thus, the budget share expresses the proportion of current hospital expenditure (running costs) which is consumed by the perinatal sector. NHS expenditure was rescaled from financial years to calender years.

### 5.3.4 Perinatal Budget Shares

Figure 5.1 presents the share of total hospital expenditure and total NHS expenditure devoted to the perinatal hospital sector over the period 1951-1985. The share of hospital expenditure increased from 6.5 per cent in 1951 to a post-war peak of 8.9 per cent in 1966. Thereafter the share decreased steadily reaching a post-war low of 5.9 per cent in 1985. A similar pattern emerges when expenditure on perinatal hospital care is expressed as a share of total NHS expenditure in Scotland.

Although a change in budget shares reflects the allocation process, budget shares on their own provide little insight into the level of real expenditure on perinatal hospital services which in turn may influence perinatal mortality. A constant (or decreasing) share of an increasing NHS budget is commensurate with an increase in the actual level of perinatal inputs.[2]

## 5.4 PRICE SERIES

---

2. Using a simple accounting identity, expenditure on perinatal hospital services can be derived given the estimated budget share and real total expenditure.

The expenditure series in current prices were expressed in real prices using an implicit NHS deflator based on the NHS Pay and Prices Index rescaled to a 1980 base. Like the implicit GDP deflator at factor cost, the NHS deflator is an input price index based on expenditure data. Although over the period 1949-1985 the two price series follow a similar pattern the long run average annual rate of change in NHS pay and prices (8.1 per cent) was slightly greater than the general rate of inflation as measured by the GDP deflator (7.0 per cent). The difference in annual rates of change in these price series provides an indication of the relative price effect or the extent to which changes in the price of NHS inputs departed from the general rate of inflation.

The NHS deflator series was calculated from the constant and current series of UK NHS expenditure presented in Tables E6 and E7 of the Royal Commission on the National Health Service Report (Cmnd 7615). This series covering 1949-77 is based on health departments' statistics. A complete series covering the period 1949-85 was constructed by splicing the 1949-77 series to the series of implicit NHS deflators reported for 1974-85 in Table 2.3 of the Office of Health Economics Compendium of Health Statistics (1987 Edition).[3] Both series

were rescaled to a 1980 base. The implicit GDP deflator series (1980=100) was obtained from Economic Trends (1985 and 1986 Annual Supplements) published by the Central Statistical Office.

The contrast between hospital expenditure on perinatal care expressed in current and constant prices is depicted in Table 5.2. The long run average annual rate of increase in real expenditure is 2.6 per cent compared to 11.0 per cent when no adjustment is made for changes in the general level of NHS pay and prices. In real terms the level of hospital expenditure on perinatal care in 1985 was equivalent to that recorded in 1971. However, given the deflation of both hospital expenditure on perinatal care and total hospital expenditure the estimated budget shares remained the same irrespective of their calculation using expenditure series in current or constant prices.

## 5.5 RELATIVE PRICES

---

3. The original source for the 1974-85 series reported by the Office of Health Economics is the Department of Health and Social Security.

One simple and restrictive specification of the demand function is given by a model in which the budget share is expressed as a linear function of the logarithm of total real expenditure and the logarithms of policy objectives and population need indicators. In other words budget shares are independent of prices; a proposition which is testable by imposing and testing restrictions on the price parameters.

In addition to total hospital expenditure, two further explanatory variables in the demand equation are the logarithims of the price of perinatal hospital services and the price of all other NHS hospital services. The wage bill is the largest component of hospital and (and health service) expenditure. Labour costs account for roughly three quarters of hospital running costs in Scotland (Gray and McGuire, 1987). Given agreed national employment conditions and pay for NHS employees and the fact that hospitals and health boards cannot vary national agreements relative price differences will reflect varying levels and mixes of labour and capital which emerge over time.

However, in common with the accounting conventions adopted throughout the NHS, expenditure on different NHS programmes in Scotland has not been factored into price and quantity components. Consequently, price indexes which distinquish between the

perinatal hospital sector and other health service activities are not available over the time period of interest.

There is, however, a degree of conceptual ambiquity about the appropriate price index for health service activities. Price indexes rarely manage to overcome what can be called the comparability problem, namely that even if the quantity of items is held constant the quality and characteristics of both old and new commodities inevitably do change over time and, as such, prices may contain more than just information about changes in prices. As the inputs used to produce health care change so too should the price of the basket of health care inputs that in combination represent a day of hospital care.

Policy makers are perhaps tempted to rely on "psuedo" price series such as costs per day or per case (discharge) for information on the relative price of different types of health care. Although such indexes can be constructed throughout most of the post war period for selected groups of Scottish hospitals their utility as price indexes can be questioned. First, as the mix, quantity and quality of hospital inputs changes, costs per day will contain both price and quantity information (Bauer and Evans, 1983). This, however, may be precisely the type of

"price" information which informs the decision making process when policy makers consider the allocation of resources across different competing programmes. Second, even if prices, service volumne and treated patients are held constant, costs per day will vary with changes in the pattern of care as measured by lengths of hospital stay. Thus, when lengths of stay decrease, a psuedo price index such as cost per day will overstate price increases. Again, policy makers may find that, compared to other measures of movement in health care prices, this psuedo index is a more relevant measure of changes in the price of providing a day of patient care.[4]

---

4. The concept of price should relate to the price of producing health care which in turn is just one of many inputs into the production of health. Following Becker (1965) the decision maker could also be hypothesised to use inputs of "market goods" (nursing hours, medical hours, capital services) to produce "basic commodities" such as a hospital day or hospital case. Within this framework implicit commodity prices are defined as the marginal costs of producing commodities which will depend on both input prices and the technology adopted to produce output.

No series of marginal costs exist for the NHS hospital sector in Scotland. If the hospital sector was assumed to be cost minimizing marginal costs could be measured by average costs since they would in the long run be equivalent. This assumption has been embraced in a number of hospital cost studies, most recently in the work of McQuire and Gray (1987) who analyzed factor substitution in Scottish hospitals over the period 1951-1983.

Whether this assumption corresponds with reality is doubtful, however, given the findings reported by Feldstein (1967) and Barr (1974). Using data on 177 acute, non-teaching hospitals for the 1960/61 financial year, Feldstein (1967) demonstrated that the marginal cost of treating an additional case was substantially less than the average cost per case. Although obstetrics had one of the highest ratios of marginal to average costs (.45) in general the ratio of marginal to average costs was between .15 and .25 suggesting that hospitals could improve their efficiency by increasing the intensity of bed use. Similar results were reported by Barr (1974) in his study of all maternity hospitals in England and Wales using data for 1970/71. The ratio of marginal cost to average cost ranged from 0.147 in General Practitioner obstetric hospitals to 0.387 in Specialist obstetric hospitals.

Within the demand model for perinatal hospital care the focus, however, is not on changes in absolute prices but relative prices between different hospital or health service sectors and programmes. If the ratio of marginal to average costs remained the same over time within each sector then the ratio of marginal costs could be estimated by the ratio of average costs. That is, if the total cost elasticity with respect to output (the ratio of marginal to average cost) remained constant over time within each sector average costs which are readily available provide a proxy for relative marginal costs or prices between the perinatal and non-perinatal hospital sectors. Adopting this assumption, the ratio of average costs per case in the perinatal and non-perinatal hospital sectors are used as a proxy for relative prices. The time series of this measure of relative prices is presented in Figure 5.2. Over the period 1951-1985 the average cost per perinatal case was about 50 per cent of the average cost of a non-perinatal case. Over time the series is characterised by a slight upward trend which reflects the fact that the average cost of hospital care increased at a more rapid rate in the perinatal sector.

## 5.6 POLICY OBJECTIVES

### 5.6.1 Aiming at Moving Policy Targets[5]

Throughout the post-war period policy objectives have been variously stated in terms of bed targets, budget shares and rates of growth in expenditure. Introducing these myraid targets into the demand equation requires their translation using a common factor. Given the preoccupation with bed targets for about two-thirds of the sample period one candidate is the maternity bed gap defined as the difference between actual and target bed supply levels. Figure 5.3 illustrates the extent of the maternity bed gap using a splined set of targets proposed by the Orr Report for the period 1948-1959 and the Montgomery Report for the period 1960-1985.

As the Montgomery target was adopted as part of the 1962 Hospital Plan for Scotland it only served as an explicit target for the planning period up to 1975. Although the Montgomery target was superseded in the late 1970's by expenditure targets it does provide a useful measure of the relative over supply of

---

5. This section summarises the information contained in the Appendix which provides a comprehensive review of the evolution of policy targets for maternity services in Scotland.

maternity beds which is consistent with the government's declared intention to reduce expenditure on maternity services. It is also interesting to note that by 1980 parity had been achieved between the actual bed stock and the target bed supply based on the Montgomery recommendations. Simultaneously, the review of maternity resources presented in Scottish Health Authorities Priorities for the Eighties (Scottish Home and Health Department, 1980) argued that the existing bed stock was sufficient to meet all expected demand throughout the 1980's. Thus, an implicit bed target was endorsed which was consistent with the Montgomery target. The contemporary relevance of the Montgomery target is a reflection of two countervailing influences on expected bed needs: namely an increase from 70 to 100 per cent in the hospital confinement rate coupled with a decrease from 10 to 7 days in the average length of stay.

The bed gap can be expressed in terms of expenditure by multiplying the difference in target and actual beds by average hospital expenditure per obstetric bed. This expenditure gap can then be added to actual expenditure to give a target level of perinatal hospital care expenditure. The target level of expenditure when expressed as a share of total hospital expenditure represents a target budget share for perinatal hospital care. Figure 5.4 compares the

actual and target budget shares. Two distinct periods can be distinguished. First, between 1951 and 1971 the actual share of expenditure fell short of the target share. Second, beginning in 1972 actual shares were greater than or equal to target shares. The overshooting of targets in the mid 1970's perhaps reflects the inadvertent costs of adjusting to the relatively long period of inadequate supply which characterised the 1950's and 1960's.

## 5.7 POPULATION NEED INDICATORS

### 5.7.1 Introduction

Demographic variables have long been acknowledged as important determinants of demand. Changes in demographic characteristics have a direct effect on demand by shifting needs and inducing changes in patterns of consumption. Modelling demographic variables in demand equations can take a variety of forms. Using the AI specification demographic variables can be introduced into the perinatal hospital demand equation by letting the intercept or constant term depend on selected perinatal hospital need indicators.

A wide range of need indicators could be introduced into the demand equation. The limited number of observations in annual time series and consequent degrees of freedom dictated a parsimonious approach. Two indicators stand out which cogently summarise the expected case load generated by the fertile population and the level of perinatal risk in the newborn population. The first is the number of births per year. The second is the perinatal mortality rate.

### 5.7.2 Birth Events

One obvious candidate for a population need indicator is the number of births. This fertility measure summarises both the age distribution of the female population and profiles of fertility by age. In the post-war period the time series pattern of annual births in Scotland has essentially reflected changes in fertility profiles, not changes in the numbers and age composition of the female population. The choice of an event measure of fertility (total births) rather than fertility rates (per 1000 population at risk) assumes that both anticipated and actual expenditure on perinatal hospital care responds to birth events rather than shifts in fertility rates.

However, the total number of births already enters the demand equation via the policy objectives outlined in the previous section. Whether expressed in terms of target beds or target expenditure, the policy objectives are themselves a function of the total number of births. Thus, population need for maternity services, as measured by birth events, is subsumed in the target perinatal budget share.

Another population need indicator which was considered was expected birth numbers based on the official forecasts published by the Registrar General Scotland. Although post-war fertility in many countries has proven to be notoriously difficult to forecast with any degree of certainty. This largely reflects the limitations of forecasting methods that have traditionally been used to generate official fertility projections. Nevertheless, despite their unreliability, projected birth numbers have influenced plans and recommendations concerning the provision of post-war perinatal hospital services in Scotland and elsewhere throughout the U.K.[6]

---

6. Throughout the mid 1960's to the late 1970's the official five year forecasts consistently overestimated the actual Scottish birth numbers by 10,000 to nearly 30,000 births (11 per cent to 33 per cent).

Birth forecasts could be incorporated into the demand equation in place of actual births or indirectly using a variable such as the percentage deviation of actual birth numbers from expected birth numbers. The "birth gap" between actual and expected events should be positively related to perinatal budget shares. In periods where forecasts under estimate the actual number of births, expenditure on perinatal care should increase. Likewise, when forecasts persistently exceed birth numbers, expenditure on perinatal care should decrease as expenditure plans are accordingly revised.

However, a major problem with the use of forecast births is that Scottish estimates are only available from 1959; thus for five and ten year forecasts the sample period is severely restricted to 1964-1985 and 1969-1985 respectively. A further reason for not using birth forecasts in the demand equation derives from the fact that actual expenditure will reflect both expected and actual needs, since at the margin expenditure outturns will be influenced by the number of births, not by forecast births.

### 5.7.3 Does expenditure depend on mortality?

Although many studies test for a relationship between health care expenditure and mortality the direction of the relationship is usually assumed to be from expenditure to mortality. Including mortality as a need indicator permits the testing of a more general hypothesis in which expenditure itself is dependent on the recent mortality experience of the population (Hadley, 1982; Carr-Hill and Russell, 1987; Gravelle and Backhouse, 1987).

Mortality rates could be introduced into the demand equation in a variety of ways. Possibilities include perinatal mortality rates (perhaps disaggregated according to cause or birthweight group), the absolute or relative distance from a target mortality rate and the ratio of perinatal mortality to the mortality experience of different groups in the population.

Since only a limited number of variables could be included in the demand equation, the overall perinatal mortality rate was chosen as a summary measure. Both current and lagged perinatal mortality rates were used in estimation and testing.

## 5.8 SPECIFICATION, TESTING AND RESULTS

### 5.8.1 Introduction

Given the incremental nature of NHS planning, the fact that the NHS budgetary process sets levels of expenditure one year in advance of programme spending and policy decisions which establish future priorities for health care spending within the context of current and expected demographic, social and economic environments, it is clearly implausible to assume that allocation decisions wholly and instantaneously adjust to the current level of policy targets and need indicators. The demand equation is thus modelled to enable the test of different hypotheses about the dynamics of allocation decisions. The maintained assumption is that the observed share of hospital spending on perinatal care will reflect a mixture of realised plans, expectations, corrections of previous mistakes and perhaps, most importantly, the inertia of previous allocation decisions.

### 5.8.2 Modelling Approach

One of the characteristic features and difficulties of applied econometrics is that neither the precise set of variables to be included in an equation nor the appropriate dynamic specification is given a priori. These two issues (i) selecting the set of regressors and (ii) dynamic specification are also bound up with

a third, namely stochastic specification. Since all of these issues are germane in the demand function for perinatal hospital care they are addressed using the following modelling strategy which begins with a general model that is subsequently simplified. Alternative equation specifications are tested against a common nesting maintained hypothesis. This general to simple path follows the methodology for model specification and validation that can be traced to Denis Sargan's (1964) study of wages and prices in the United Kingdom. Due largely to the subsequent work of David Hendry (1980, 1983) this methodological orientation has had a growing influence on econometric time series modelling over the past decade. McAleer, Pagan and Volker (1985) summarise the main features of this approach as

- selection of a general model
- disclosure of how and why any general model was simplified to the preferred model
- quality control of the preferred model.

The emphasis is on the full presentation of the process which led to the selection of a preferred model and, perhaps more importantly, the rigorous testing and evaluation of the preferred model.[7]

**Time Series Properties and Cointegration**

Like many time series of aggregate economic data, the series used to estimate the perinatal hospital demand function can not be presumed to satisfy the assumption of stationarity. The mean and variance of several data series have not remained constant over time. This is particularly the case for real expenditure on hospital services, perinatal mortality and other variables with non-stationary means where the presence of a time trend is easily detected by visual inspection of the series. As perinatal mortality has changed over time it has exhibited the tendency to decrease in mean with a corresponding reduction in the dispersion around the declining mean.

The consequences of non-stationarity for the usual statistical properties of estimators and hypothesis tests are two-fold (Judge *et al*, 1985). Firstly when OLS is used to model non-stationary processes the estimates will be unbiased but will not in general be

---

7. See Gilbert (1986) for a concise survey of the essential features of this methodological approach which is contrasted against the traditional approach adopted in North America (as well as in Britain).

efficient. Thus, regression coefficients do not converge in probability as the sample size increases. A second major consequence is that the OLS variance estimator is biased, thereby invalidating the usual least squares test statistics.

Extending Granger and Newbold's (1974) results on the hazards of spurious regressions with time series data Phillips (1986) presented a formal analysis and asymptotic theory for regressions of non-stationary stochastic processes. Unlike the standard theory of regression for stationary processes, the regression coefficients do not converge to constants as the sample size increases. Furthermore the distributions of the test statistics $F$ and $t$ also diverge as the sample size increases. As a result there are no asympototically correct critical values for these conventional statistical tests. This implies that the use of conventional critical values leads to a rejection rate that increases with sample size. The rejection rate is also greater for the block $F$ test than for the individual $t$-tests. Another related result concerns the Durbin-Watson (DW) test of first order autocorrelation. Low values of the DW statistic are a well established feature of spurious regressions. Phillips (1986) demonstrates that under certain conditions the DW statistic in fact converges on zero.

One increasingly prominent method of addressing these problems in the analysis of non-stationary time series is through the use of "cointegrated" techniques (Granger, 1981). At an intuitive level, a vector of time series variables are said to be cointegrated if they move together. Certain variables should not drift apart from each other by too great an extent over time. Short term fluctuations are thus accompanied by a long run tendency back towards a close relationship. The rationale for such cointegrated processes could be a market mechanism based on equilibrium concepts relating supply and demand, goverment intervention or, more generally, error correction models which combine non-stationary and stationary elements to explain long-run equilibrium and short-run dynamics.[8]

Formally, a variable $z$ is said to be integrated of order $d$ [$z \approx I(d)$] if it has a stationary, invertible, non-deterministic autoregressive moving

---

8. An error correction model where some proportion of the disequilibrium from one period is corrected in the next period is developed below in Section 5.8.8.

average representation after differencing *d* times (Granger, 1986). Thus, an integrated series of order *d* is a time series that must be differenced *d* times before it becomes stationary. The simplest example of an I(0) series is white noise. An example of an I(1) series is given by a stationary AR(1) process where

$$x_t = \alpha x_{t-1} + \epsilon_t$$

and $|\alpha| < 1$ and $\epsilon_t$ is white noise with a zero mean.

The definition of cointegration proposed by Granger (1983) stated that *N* series in the vector $x_t$ are cointegrated or order $(d,b)$, ($x_t \approx CI(d,b)$) if all *N* series are integrated of order *d* (i.e., $x_{i\,t} \approx I(d)$) and there exists a linear combination of the *N* series $z_t = \alpha' x_t$ such that $z_t = \alpha' x_t \approx I(d-b)$ with $b > 0$. Within this modelling framework $z_t = \alpha' x_t$ is interpreted as a long-run equilibrium relation. Thus when two or more variables are cointegrated, longer-run responses can be aggregated out from their dynamic behaviour by a suitable linear combination of the original variables.

The importance of cointegration for econometric modelling is derived from a theorem originally stated and proved by Granger which shows that cointegrated series can always be represented by error correction

models. This result provides the foundation for a two-stage estimation procedure proposed by Engle and Granger (1987). The first stage involves explicit testing for cointegration. This is an important step which seeks to ensure that the right and left hand sides of the regression equation have compatible long-run properties; i.e. they must be integrated to the same order. A variety of integration tests have been proposed.[9] If the variables are indeed cointegrated, the second stage enters the residuals from the static (levels) regression used to test for cointegration into the error correction model in place of the levels terms.

The cointegration approach to the analysis of macroeconomic time series has grown in popularity over recent years, particularly in studies of the relationship between income and consumption (Engle and Granger, 1987; Drobny and Hall, 1987; Campbell, 1987; Osborn, *et al* 1988; MacDonald and Speight, 1989). Other work has concentrated on aggregate models of wage determination (Hall, 1986). Within the general

---

9. Two popular tests are the Dickey-Fuller (DF) and the Augmented Dickey-Fuller (ADF). These are both "t" tests and rely on rejecting the hypothesis that the series is a random walk in favour of stationarity (Maddala, 1988).

domain of population and health economics, however, the concept and application of cointegration remain novel. Ermisch (1988) develops a model of the economic determinants of British post-war birth rates using the Engle and Granger two-stage estimation procedure. His results confirmed that birth rates and virtually all of the economic "independent" variables were not stationary. Most of the time varying economic series were characterised by I(1) but other variables measuring relative cohort size and the proportion of a particular cohort at risk for a certain birth at a certain age (cohort heterogeneity) were I(2) variables. The evidence on whether the series were cointegrated was thus inconclusive. However, by selecting a subset of I(1) variables (either individually or in combination), Ermish was able to estimate a dynamic equation in first differences in which all the variables were stationary and cointegrated.[10]

---

10. Ermish also uses the dynamic model to assess the relative influence of various economic factors on the change in births over the period 1973-1985, estimate the impact of higher child benefit on fertility and develop birth forecasts for 1986-1990.

The time series properties of the variables used to estimate the perinatal hospital demand function were analysed using a variety of tests.[11] The battery of tests confirmed that the series are non-stationary. The sample autocorrelations of the data in levels and first differences were consistent with the hypothesis of a random walk or difference-stationary (DS) process. Sample autocorrelations of the residuals from a linear least squares regressions of the series on a time trend and a constant also supported the DS process against an alternative given by a trend-stationary (TS) process.[12] A more rigorous test using an Augmented Dickey-Fuller test provided additional evidence that the time series were all $I(1)$

---

11. Full test results are given in Appendix Tables A5.1 to A5.4. The data series are presented in Appendix Table A5.5.

12. See Nelson and Plosser (1982) for a discussion of the difference between difference-stationary and trend-stationary processes. Their empirical findings led them to conclude that the non-stationarity observed in many macroeconmic time series can be characterised by a random walk or difference-stationary process. For a rival view see Rappoport and Reichlin (1989) who argue in favour of an intermediate hypothesis, namely that many difference-stationary processes are in fact better described by a series of "segmented trends".

or $I(0)$ after differencing once. Thus all of the variables constitute a cointegrated vector which ensures that an error-correction model is a valid representation of the relationship between perinatal hospital shares and the set of explanatory variables used in the equations presented below (Section 5.8.8).

### 5.8.3 Selection of a General Model

The error correction model is only one of an infinite variety of models that could be used to estimate the demand equation. Following Hendry (1988) the AI demand equation is specified within a general Autoregressive-Distributed lag framework as

$$W_t = \beta_0 + \beta_1 W_{t-1} + \beta_2 x_t + \beta_3 x_{t-1} + \beta_4 rp_t + \beta_5 rp_{t-1}$$

$$+ \beta_6 tw_t + \beta_7 tw_{t-1} + \beta_7 pnm_t + \beta_8 pnm_{t-1} + e_t \quad (5.7)$$

where lower case letters denote logs of corresponding capitals and:

W = Share of Hospital Expenditure on Perinatal Care

X = Total Hospital Expenditure

RP = Relative price of perinatal and non-perinatal hospital care

TW = Target Perinatal Hospital Share

PNM = Perinatal Mortality Rate

e = error term.

This general specification actually encompasses several different types of model as further special cases which can be tested by imposing appropriate restrictions on the estimated parameters. Although it may be tempting to treat some models as uninteresting or eccentric and focus on the more familiar models (e.g., static regressions, distributed lags, first differences), this modelling framework imposes a discipline which encourages the assessment of competing specifications as a way of searching for an acceptable and robust, though not necessarily correct, model.

By including total hospital expenditure (income), relative prices, target perinatal shares and perinatal mortality as regressors (both current and lagged one period) the general model (equation 5.7) also lends itself to testing restrictions on subsets of the parameters thereby narrowing down the set of regressors prior to testing different dynamic specifications of a more parsimonious equation. This is a particularly attractive feature of the modelling approach given the relatively short annual time series and the large number of variables which potentially

could be included in the equation. It also avoids some of the pitfalls which can occur when an equation is sequentially revised and expanded by variable addition: a simple to general modelling approach which inadvertantly may exclude important variables, retain extraneous variables and thereby increase the likelihood of accepting (or at least failing to recognise) misspecified models.

### 5.8.4 Simplification of the General Model

The testing strategy proceeds in two steps. The first examines variable selection and considers a nested set of alternative specifications of Equation 5.7 listed in Table 5.3. The unrestricted version of Equation 5.7 is represented by Model 1. Setting ($\beta_i = 0$ for $i > 7$) gives Model 2, which excludes both current and lagged values of the perinatal mortality rate. Model 3 excludes perinatal mortality rates and the target perinatal hospital budget shares ($\beta_i = 0$ for $i > 5$). Model 4 extends the exclusion list to relative price variables ($\beta_i = 0$ for $i > 3$ ) leaving a model in which the share of hospital expenditure on perinatal hospital care is dependent on the lagged perinatal share and total hospital expenditure. Finally, Model 5, a univariate time series model, is obtained by setting $\beta_i = 0$ for $i > 1$.

Having selected the broad class of variables to be retained in the model the second step involves the imposition of further restrictions which are used to test different dynamic specifications of the simplified model.

The alternative models and their respective restrictions were tested using standard F-tests given by

$$F = [(\ddot{e}'\ddot{e} - \hat{e}'\hat{e})/(\hat{e}'\hat{e})]\,[(T - k)/r]$$

where $\ddot{e}'\ddot{e}$ is the sum of squared residuals from the restricted model,

$\hat{e}'\hat{e}$ is the sum of squared residuals from the unrestricted model,

$(T - k)$ is the degrees of freedom of the unrestricted model and

$r$ is the number of tested restrictions.

A 1% level of significance was chosen to reduce the chances of over-rejection of the competing specifications.

### 5.8.5 Tests of the Set of Regressors

Table 5.3 presents the F-statistics used to test the set of nested general models which sequentially restrict the set of regressors. The results suggest the rejection of all restrictions except those imposed

by Model 2: the equation which excludes the current and lagged values of the perinatal mortality rate. This supports the hypothesis that the share of expenditure on perinatal hospital care is independent of perinatal mortality.[13]

Reducing the set of regressors further is not justified given the value of the F-statistics associated with more restrictive sub-models. Thus, alongwith the lagged perinatal hospital share, current and lagged values of total NHS expenditure, relative prices and the target perinatal hospital share are included as regressors.

### 5.8.6 Tests of Dynamic Structure

Ordinary Least Squares (OLS) estimation of Model 2 over the period 1952 to 1985 (with 5 observations withheld for forecasts) yields

$$W_t = \underset{[2.211]}{0.075} + \underset{[3.858]}{0.690}W_{t-1} + \underset{[0.343]}{0.005}x_t - \underset{[-0.296]}{0.005}x_{t-1}$$

$$+ \underset{[6.840]}{0.048}rp_t - \underset{[-1.526]}{0.019}rp_{t-1} + \underset{[2.970]}{0.031}tw_t - \underset{[-0.976]}{0.016}tw_{t-1} \quad (5.8)$$

---

13. Similar results were obtained when dis-aggregated mortality rates were included as regressors in place of the overall perinatal mortality rate.

T = 29 R-squared = 0.9825 F(7,21) 168.90

s = 0.12% RSS = 0.000032

T = sample size

s = standard deviation of the residuals

RSS = residual sum of squares

[ ] = t-statistics calculated using heteroscedastic consistent standard errors (MacKinnon and White, 1985).

Figure 5.5 plots the actual and fitted values of $W_t$. The equation provides a good fit to the post-war series of perinatal hospital shares. The equation also performs well in the forecast period (1981 to 1985). The absolute value of the standardised forecast error (1-step forecast error divided by the standard error of the forecast) ranges from 0.248 to 1.157. A Chow test of parameter stability over the forecast period supported the null hypothesis of no structural change in the parameters (Chow statistic = 0.83, F[5,21] critical value = 4.04 (1%)).

Solving for the static long run parameters derived from the "equilibrium" solution of (5.8) yields

$$W = \underset{(2.870)}{0.241} + \underset{(0.217)}{0.003x} + \underset{(3.383)}{0.095rp} + \underset{(4.520)}{0.048tw} \qquad (5.9)$$

where t-statistics are given in parentheses.

The results suggest that the share of hospital expenditure devoted to perinatal care is positively related to total hospital expenditure, the relative price of hospital perinatal care and target perinatal shares. The null hypothesis that all long run coefficients are zero is easily rejected. A further test of the significance of individual variables was based on F-tests of the restriction setting the parameters of the coefficients of the current and lagged value of each variable to zero. These tests support the hypothesis that total hospital expenditure exerted no significant effect on the share of hospital expenditure devoted to perinatal care. In contrast, the relative price of perinatal and non-perinatal hospital care and the target perinatal share were both significant determinants of W .

The residuals also appear to be reasonably well behaved (Figure 5.6). A Wald Test rejected the hypothesis of common factors in the lag polynomials and first order autoregressive residuals.

This first order dynamic model is deliberately overparameterised. The results of testing dynamic structure are presented in Table 5.4. In general, the restrictions imposed by different dynamic

specifications are rejected by the F-statistics at the 1% level. Three popular specifications (static regression, distributed lag and leading indicator) are all decisively rejected. However, the partial adjustment model is not rejected by the data. It also seems reasonable to accept the restrictions imposed by the first difference model, considering the time series properties of the variables and the closeness of the F-statistic to the critical value and the possibility of over-rejection of the null hypothesis in small samples.

The estimated coefficients of the alternative models are presented for reference in Table 5.5. All models were estimated using annual observations for 1952 to 1985 with the last five observations used for forecasts. Detailed discussion of individual models will initially be confined to the partial adjustment and first difference models. The first difference model is also extended to include an error correction term.

### 5.8.6 Interpretation and Evaluation of the Partial Adjustment Model

The partial adjustment model provides one parsimonious version of the general autoregressive model. OLS estimates yield

$$W_t = \underset{[7.616]}{0.115} + \underset{[6.361]}{0.470W_{t-1}} + \underset{[0.805]}{0.003x_t} + \underset{[6.669]}{0.045rp_t} + \underset{[4.724]}{0.024tw_t} \qquad (5.10)$$

T = 29 R-squared = 0.9775 F(4,24) 260.46
s = 0.13% RSS = 0.000041

The share of hospital expenditure on perinatal hospital care is positively related to the lagged perinatal share and the logarithms of total hospital expenditure, relative prices and the target perinatal share. Total hospital expenditure has no significant impact on the perinatal budget share. The positive coefficient on the relative price variable suggests that as the price of perinatal care increases (relative to other hospital services) the share of expenditure on perinatal hospital care increases. One explanation for this counter-intuitive result is the relative inflexibilty of the quantity of existing hospital services in the short run, thus current relative price rises are transmitted directly into current expenditure shares. The current budget share is also directly related to the target perinatal share.

Within the present context, this model can be interpreted as one of habit persistence (or simple inertia) (Brown, 1952) where the perinatal budget share evolves over time as a function of previous budget shares and the current period values of total hospital expenditure, relative prices and target shares. Another possible interpretation is that, following Eisner and Strotz (1963), agents face adjustment costs when changing their plans from previous outcomes as well as to not achieving present targets. Both interpretations are inherent features of Nerlove's (1956) original formulation where the current values of the independent variable determines the desired value of the dependent variable but only a fraction of the desired adjustment is possible within the current period. Thus, decision makers respond to a changing perinatal share target or movement in relative prices, by sequentially adjusting the perinatal hospital share. A further important feature of the partial adjustment model is that past discrepancies between realised and target perinatal budget shares are transmitted into future behaviour.

One potentially problematic feature of this model is that the speed of adjustment may be very slow, particularly when the coefficient ($\pi$) on the lagged dependent variable is large. In fact, in the extreme case when $\pi = 1$, no adjustment occurs and the value

of the dependent variable never changes. At the other extreme when $\pi = 0$, adjustment is immediate. For intermediate values of $\pi$ the mean lag is given by $\pi/(1 - \pi)$. The mean lag, however, may disguise the distribution of the adjustment process over time. A more informative measure is the proportion of the desired adjustment which occurs after *n* time periods $(1 - \pi)^n$. The likely colinearity between the lagged dependent variable and one (or more) independent variables can also pose estimation problems. The existence of a stochastic regressor also invalidates the use of the Durban-Watson d test for first order serial correlation. Finally, with constantly growing (decreasing) targets decision makers consistently fall short of (or overshoot) their goals; a proposition which is perhaps not too alien in an organisation like the NHS.

The estimated parameter on the lagged perinatal budget share (0.470) gives a mean lag of 0.887 years. The lag distribution over time is presented in Figure 5.7. Just over half the total adjustment occurs within one year. After three years, 90 per cent of the total adjustment has occurred. Virtually all the adjustment required to achieve the desired perinatal budget share occurs within five years.

**Autocorrelation**

Figure 5.8 presents the actual and fitted values of W based on the partial adjustment model. The corresponding residuals scaled by the equation standard error are graphed in Figure 5.9. No dramatic visual evidence of autocorrelation is apparent. This observation is confirmed by a more strenuous examination using an appropriate test for general autoregressive errors when the regressors include a dependent variable. This Lagrange multiplier test, suggested by Godfrey (1978) and Breusch (1978), can be used to test for the existence of either autoregressive or moving average errors of any order. The error autocorrelation coefficients for lags 1 and 2 are 0.1821 and -0.3226, respectively. The value of the appropriate F-test is 1.48 (1% critical value F[2,22] = 5.72). Thus, the null hypothesis of uncorrelated errors is accepted.

Further investigation of the distribution of the residuals (scaled to a zero mean and unit variance) reveals a value of -0.291 for skewness and -0.628 for (excess) kurtosis. A test of the hypothesis of normality (i.e., zero skewness and kurtosis) was based on the statistic suggested by Jarque and Bera (1980). The value of the relevant chi-square statistic adjusted for degress of freedom is 0.734 (1% critical

value = 9.21), suggesting that the distribution of the residuals is similar to that of the normal distribution.

Parameter Stability

A test of parameter stability between the 1952-1980 (within sample) and 1981-1985 (post-sample) periods yielded a Chow test of 0.92 (1% critical value F[5,24]= 3.90) confirming the stability of estimated parameters. Further tests of parameter stability were generated by re-estimating (5.10) using recursive least squares. Following the estimation of the model using the first 16 observations (1952 to 1967) the coefficients and their standard errors (alongwith the residual sum of squares) are sequentially revised in light of extending the sample period by one year.

Figures 5.10 to 5.13 provide graphs of the recursively estimated coefficients and their standard errors over the period 1967 to 1985. Throughout the latter sample period, no significant change in the estimated parameters is revealed. Any changes seem to be confined to the period immediately following the initial estimation period. Although there is a slight upward drift in the coefficient on the lagged perinatal share from 1967 to 1969, the coefficient remained virtually constant after 1970. No

significant change occured in the coefficient on hospital expenditure which, in any case, hovers near zero. There is a slight downward drift in the relative price coefficient from 1967 to 1977. In the early 1970's the target perinatal share coefficient increased slightly from about 0.018 to 0.024. A further test of parameter constancy based on a sequence of Chow tests also confirmed the stability of the estimated parameters over time.

### 5.8.7 Differenced Data: An Alternative Specification

Although the partial adjustment model provides a reasonable description of the data and survives the tests of stochastic specification and parameter stability it faces (at least) one close contender, namely the differenced data model. Ordinary least squares estimates of the differenced data model (excluding a constant) yields

$$\Delta W_t = \underset{[1.094]}{.008}\,\Delta x_t + \underset{[7.137]}{.045}\,\Delta rp_t + \underset{[3.723]}{.034}\,\Delta tw_t \qquad (5.11)$$

T = 29 R-squared= 0.7829 F(3,26) = 31.26
s = 0.16% RSS= 0.000063

In this first difference form of the equation total hospital expenditure again exerts no significant impact on perinatal hospital budget shares. Relative prices and target shares are both significant determinants of the annual change in the perinatal

budget share. A decrease (increase) in the relative price of perinatal care and the target perinatal share results in a decrease (increase) in the perinatal budget share. These results are reminiscent of the partial adjustment model and the long run solution of the general model reported in (5.9).

Autocorrelation

Following the influential paper of Granger and Newbold (1974) the presence of first order autocorrelation in static regressions using time series data was often (mis)interpreted as a license to estimate first difference models as a way of identifing the true relationship between the variables under study. The first difference specification should not be seen as a "cure" for first order autocorrelation in the static regression model in this (or any other) study but rather as a legitimate specification based on a test of the restrictions imposed on the general autoregressive model by the first difference model.

The graph of fitted and actual values of $\Delta W_t$ alongwith the corresponding residuals of the first difference model are presented in Figures 5.14 and 5.15. No systematic pattern in the residuals is apparent from the graphical analysis. The null hypothesis of no autocorrelation is accepted using

Godfrey's LM test as the F-statistic is 0.70 (1% Critical Value F[2,24] = 5.61). The residuals were also normally distributed according to the analysis of the third and fourth moments of the distribution (skewness and kurtosis) of the scaled residuals using the Jarque and Bera (1980) test statistic.

Parameter Stability

No change in parameters was detected between the within sample and post-sample periods (Chow test = 0.97 1% critical value F[5,26] = 3.61). Recursive least squares estimates of equation 5.11 confirmed the stability of the estimated coefficients (Figures 5.16 to 5.18). Although the coefficients drift slightly between 1965 and 1970 no significant shifts in parameter values occur.

### 5.8.8 Error Correction Model

Another possible dynamic specification of the perinatal demand equation is an error correction model where perinatal budget shares are marginally adjusted over time to changes in total hospital expenditure, relative prices, target shares and a measure of

disequilibrium between actual and target shares. Ordinary least squares estimates of a simple error correction model yields

$$\Delta W_t = \underset{[0.079]}{-0.001}\,\Delta x_t + \underset{[5.191]}{0.040}\,\Delta rp_t + \underset{[2.748]}{0.031}\,\Delta tw_t + \underset{[0.948]}{0.005}\,\Delta pnd_t + \underset{[1.371]}{0.032}\,(TW - W)_{t-1} \qquad (5.12)$$

T = 29 R-squared = 0.8265 F(5,24) = 22.87
s = 0.15% RSS = 0.000051 DW = 1.780.

where $(TW - W)_{t-1}$ is a measure of the previous period disequilibirium between the target (TW) and actual (W) perinatal hospital budget shares. When target and actual shares are equal (or if the coefficient on the error correction variable is restricted to zero) the error correction model is equivalent to the differenced data model. The estimated coefficients on the relative price and target share variables are in fact virtually identical in the two specifications.

One interpretation of equation 5.12 that can be traced back to Phillips (1954, 1957) work on stabilization policy is that of distinctive types of feedback control. Using Phillips' terminology the parameters on relative prices and target perinatal shares represent "derivative" feedback control. Decision makers plan to allocate the same budget share

to perinatal care modified by the annual change in relative prices and target shares. Expenditure is also influenced by "proportional" feedback control arising from the difference between target and actual shares where in equilibrium the ratio of target and actual shares is one. Decision makers thus attempt to reach and maintain a constant ratio of one between target and actual shares; this desired proportionality in turn feedbacks to the year to year change in perinatal budget shares.

**Autocorrelation**

Figures 5.19 and 5.20 provide an illustration of the actual and fitted values of the annual change in perinatal budget shares alongwith the corresponding residuals. Visually, the residuals are characterised by an autoregressive process during the 1950's which is superseeded by a random pattern during the remaining sample period. The first, second and third order autocorrelation coefficients are 0.0147, -0.1774 and -0.3600, respectively. No statistical evidence of autocorrelation is apparent from Godfrey's test which gave an F-statistic of 1.22 (1% critical value F[3,21] = 4.87. The scaled residuals also were distributed in accordance with a normal variate (skewness = 0.738,

excess kurtosis = -0.3795). The Jargue and Bera (1980) test for normality was 0.166 (1% critical value, chi-square[2] = 9.21).

Parameter Stability

Like the partial adjustment and differenced data models, the parameters displayed no significant structural shifts over time. The within and post-sample parameters are stable (Chow-test = 1.05, 1% critical value F[5,24] = 3.90). Once again, the recursive least squares estimates confirmed the stability of estimated parameters over time.

Granger-Engle Two-Stage Estimates

The error correction model was re-estimated using the Granger-Engle two-stage approach outlined above in Section 5.8.2. Using the lagged residuals ($\hat{u}_{t-1}$) from a static regression of W on h, p, tw and pnd the error correction model over the period 1952-1985 with five observations held as forecasts is given by

$$\Delta W_t = \underset{[1.452]}{0.012\,\Delta x_t} + \underset{[6.721]}{0.048\,\Delta rp_t} + \underset{[2.917]}{0.030\,\Delta tw_t} +$$

$$\underset{[1.378]}{0.005\,\Delta pnd_t} - \underset{[1.910]}{0.353\hat{u}_{t-1}} \qquad (5.13)$$

T = 29 R-squared = 0.8517 F(5,24) 27.56

s = 0.13% RSS = 0.000043 DW = 1.611

An F-test failed to support the inclusion of a constant term in 5.13 [F(1,23) = 0.002]. Inspection of the residual correlogram alongwith the Godfrey and Jargue-Bera test statistics provided no evidence of autocorrelation.

A final point to recognise about the error correction model concerns the fact that the general equation given in 5.7 can be re-specified in an equivalent form using first differences and levels

$$\Delta W_t = \delta_0 + a_1 W_{t-1} + \delta_2 \Delta x_t + a_2 x_{t-1} + \delta_3 \Delta rp_t + a_3 rp_{t-1}$$

$$+ \delta_4 \Delta tw_t + a_4 tw_{t-1} + \delta_5 \Delta pnm_t + a_5 pnm_{t-1} + e_t \quad (5.7a)$$

By imposing appropriate restrictions on the parameters in 5.7a the ß's in 5.7 can be obtained (e.g., $\delta_2 = \beta_2$ and $\beta_3 = a_2 - \delta_2$). The typology of dynamic models presented in this chapter, including the error correction model, can also be derived by imposing restrictions on this general formulation. The error

correction model in turn provides a general specification that can be used to test rival models (Hendry, Pagan and Sargan, 1984; Hendry, 1988).[14]

## 5.9 Conclusions

This chapter developed a model for describing the post-war pattern of expenditure on perinatal hospital services in Scotland. The demand model encompasses the major factors that have influenced public expenditure on perinatal hospital care.

The general findings presented in this chapter can be summarised as follows. First, perinatal mortality does not appear to influence the proportion of total hospital expenditure devoted to perinatal care. Both

---

14. One suggested modelling approach within this general framework is to begin with the error correction model and then test the series of restrictions imposed by specific dynamic models. Hendry, Pagan and Sargan (1984) discuss how even if the data generation process was characterised by a rival model the error correction model would not be mis-specified but would facilitate the search for the special case(s) that provided a more parsimonous model.

current and lagged mortality rates were found to exert no impact on the perinatal hospital budget share. This could reflect the fact that due to a variety of institutional constraints and the sluggisness of the budgetary process, expenditure on hospital services simply does not react quickly enough to changes in population need as (crudely) measured by mortality rates. Another possible explanation is that decision makers use other targets or need indicators which dominate any short term fluctuations in mortality.

Second, income, measured by total NHS spending on hospital care, has no influence on the perinatal budget share. As total real expenditure on hospital services in Scotland has steadily increased over time, the budget share for perinatal hospital care has increased as well as decreased. Perinatal care can thus not be easily classified over the entire post-war period. Prior to the peak in the fertility rate in the 1960's perinatal care perhaps represented a necessity or even luxury good as expenditure struggled to keep up with the "baby boom" and growing demand for maternity services as norms changed for hospital delivery. The subsequent "baby bust" has ushered in an era where expenditure on perinatal care has increasingly been seen as an inferior good.

Relative prices or costs of perinatal care in relation to other forms of hospital care are important determinants of expenditure. As its price increases the share of hospital expenditure on perinatal care increases. This may reflect the inflexibility of the quantity of existing hospital services in the short run; thus current relative price changes are transmitted directly into current expenditure shares.

Several different specifications of the demand model provided a good fit for the data and survived tests of parameter stability and stochastic specification. The partial adjustment model suggested that virtually all the adjustment required to achieve desired perinatal budget shares occurs within five years: a plausible planning horizon for marginal shifts in the pattern of hospital expenditure.

Like many aggregate macroeconomic time series, the data used to estimate the perinatal hospital demand function is characterised by non-stationary. A battery of tests confirmed that the perinatal budget share and the vector of explanatory variables conformed to a difference stationary process; i.e., that after taking first differences the data were stationary. Using the insights generated by the technique of cointegration a model in first

differences was estimated and extended to include an error correction term. The error correction model supported the hypothesis that disequilibirium between target and actual perinatal shares was an important determinant of the change in budget shares over time. The cointegrated series also permitted the application of the Granger-Engle two stage estimation procedure which provides a firmer basis for inference and understanding of the process underlying the evolution of perinatal budget shares.

## APPENDIX 5.1 Policy Objectives for Maternity Care

### A.1 Pre-NHS

Policy objectives have their origin in the concern expressed at the turn of the century in the health and well being of mothers and young children. Beginning with the Notification of Births Act (1907) local authorities in Scotland were encouraged to introduce initiatives designed to reduce the high level of infant mortality and improve the general health of mothers and young children.[1] In the 1920's concern over the lack of maternity facilities was expressed by the Report on Maternal Morbidity and Mortality in Scotland, issued under the auspices of the Scientific Advisory Committee of

---

1. The powers of local authorities were widened by the Notification of Births (Extension) Act, 1915. Subject to the approval of the Local Government Board for Scotland local authorities could facilitate maternity hospital provision for abnormal conditions of pregnancy and parturition. This policy objective of establishing or subsidising beds for the care of women with extraordinary needs introduced the notion of maternal risk assessment which was to govern the allocation of maternity resources for decades.

the Department of Health for Scotland. Perhaps the most influential pre-war report on the health of women in pregnancy it stressed the need for more and better maternity hospital facilities, particularly for antenatal care. Soon after the publication of this report the Local Government (Scotland) Act, 1929 was passed which had an important impact on the inter-war growth of hospital maternity services and the stock of maternity resources inherited by the NHS in 1948.

The impact of this legislation on the stock of maternity beds can be seen in the three-fold increase in local authority maternity beds from 191 in 1930 to 673 in 1935 (Department of Health for Scotland, 1936a). Confinements increased from 3281 to 9365 over this period.[2] Despite the increase in beds, the need for more and better maternity services was a theme reinforced by the Cathcart Report on Scottish

---

2. The increased number of maternity beds in local authority hospitals was mainly due to additional beds having been made available for this purpose in their general hospitals. There were 633 maternity beds in voluntary institutions and 92 beds private maternity homes giving a total of 1398 compared to between 250 and 300 beds for maternity care in 1926.

Health Services (Department of Health for Scotland, 1936b). The Committee's recommendations regarding maternity care largely corroborated those contained in the Report on Maternal Morbidity and Mortality (Department of Health for Scotland, 1935). Anticipating the eventual vogue for large maternity units which decades later was endorsed by a succession of special committees and reports, the Cathcart Report emphasised the importance of ensuring that maternity units were large enough to deal with all types of cases and to justify the employment of a resident medical officer. Although the committee stated that hospital facilities were in seriously short supply, no explicit indication of the target bed level is given. Implicitly, however, a target bed level of around 1100 beds is inherent in proposals for financing a comprehensive maternity service based on the assumption of 24000 institutional births per year.[3]

---

3. Even after excluding the private maternity homes, the 1935 bed stock exceeded this target figure by 200 beds suggesting a decrease in supply which curiously offers little support for the committee's assertion about the shortfall in maternity beds.

## A.2 Bed Supply Targets

The first explicit bed supply target for maternity hospital services appeared in the Orr Report on Infant Mortality in Scotland (Department of Health for Scotland, 1943). Alongside pleas to increase the number of beds in maternity hospitals and nurseries for premature infants, the committee presented wide ranging recommendations to the Secretary of State for Scotland directed towards improving housing conditions, the diet of mothers and infants, child welfare services, antenatal care (via the introduction of clinics staffed by obstetricians) and medical and general education.

The committee argued that all first births, births to mothers with bad obstetrical histories and births to women from "unsuitable" or poor houses should occur in hospital. Although precise estimates of the number of births falling into these categories were not presented the committee recommended a tripling of the number of beds from their 1937 level of 15 per 1000 births to 43 per 1000 births. To underline their recommendation, the committee suggested that the 1937 bed allocation was not even sufficient to accomodate all first births which accounted for 35 per cent of all births. They also

added that this target supply level represented nothing more than a lower estimate of the number of required beds.

## A.3 Post-war targets

The immediate post-war years were characterised by an increasing concern with the level of maternity hospital resources. The post-war increase in fertility, housing difficulties which limited the scope of domiciliary confinements and the screening function of antenatal care which identified an increasing number of cases for hospital delievery were cited by the Department of Health for Scotland as factors which increased the demand for maternity beds.[4]

---

4. This concern was hightened following the publication of the first comprehensive survey of hospital bed resources in Scotland conducted in 1946 which revealed a short-fall in maternity beds, particularly in the Western Region. This finding was not surprising considering that the Survey endorsed the maternity bed targets recommended by the Orr Report.

With the inception of the National Health Service, virtually all the hospitals in Scotland passed into State ownership on the 5th July, 1948. The Scottish NHS inherited 63880 hospital beds of which 2698 were categorised as maternity beds (4.2 per cent). Although the stock of maternity beds had increased by 26 per cent between 1943 and 1948,additional maternity bed provision was regarded as a priority for the new service. As a result investment in new or converted beds for maternity care was a prominent feature of the hospital building programme in the early years of the NHS.

The degree of priority accorded to maternity beds however gradually eroded in the early 1950s as other programmes and client groups[5] began to compete for available resources. Another factor which may have influenced decision making was the report of the Royal Commission on Population published in 1950 which forecast a gradual and steady decline in fertility: a prediction which, ironically for the commission and those who planned services based on

---

5. Such programmes included the special campaign against respiratory tuberculosis, treatment of the aged and chronic sick and the institutional care of the mentally disabled).

their forecast, heralded the start of one of the most dramatic recorded increases in fertility experienced by the Scottish population.

In May 1953 the Minister of Health and Secretary of State for Scotland appointed a committee to examine the present and projected cost of the National Health Service with a view to making recommendations that would improve the efficiency of the service, reduce the financial burden and maintain an adequate service. The eventual report submitted by the Committee of Enquiry into the Cost of the National Health Service (Chairman, C. W. Guillebaud) was presented to Parliament in January 1956 (Ministry of Health, 1956). Although the committee did not offer any policy recommendations regarding the provision of maternity care, their report concluded that the maternity services were in "a state of some confusion, which must impair their usefullness" and recommended that the organisation of maternity services under the National Health Service be reviewed at an early date.

One immediate consequence of the Guillebaud Report was the appointment in 1956 of two committees with similar terms of reference which respectively included a review the maternity services in Scotland (Montgomery Committee) and in England and Wales

(Cranbrook Committee). Three years later both committees recommended similar maternity bed supply targets which, in Scotland, superseded and revised downwards by 14 per cent the previous target of the Orr Committee.

The bed target of 37 maternity beds per 1000 total births set out in the Montgomery Report (Department of Health for Scotland, 1959) was based on several assumptions. The first concerned the proportion of hospital deliveries (70-80 percent of all births). This proportion did not emerge from a detailed assessment of the advantages and disadvantages of hospital confinement but rather reflected a concensus view of the medical opinion expressed before the committee regarding the need for hospital confinement for (a) women with medical or obstetrical conditions requiring it; (b) primigravidae; (c) fourth and subsequent pregnancies; and (d) women requiring admission on social grounds. The second assumption was an average length of stay in hospital of 10 days which simply reflected the wide spread preference of the professionals involved in providing maternity care. The third was an antenatal bed complement of 8 beds per 1000 total births which the committee itself acknowledged as nothing more than a rough estimate of the need for antenatal hospitalisation.

Essentially the Montgomery Committee acknowledged the regional imbalances in the maternity services inhereited by the NHS and set out a strategy for reducing the degree of inequality in service access and presumably the outcomes of maternity care. By 1957 70 per cent of Scottish women delivered their babies in institutions. The chances of an institutional confinement, however, varied dramatically throughout the country. Between local health authorities hospital confinement rates per 1000 births ranged from 964 to 504 with the lowest rates recorded in the West of Scotland. Consequently the greatest gap between the supply of maternity beds and the estimated demand (given the above assumptions) was in the Western Regional Hospital Board where demand exceeded supply by 546 beds or about one third of the 1957 bed complement of 1598 beds.

**A.4 1962 Hospital Plan for Scotland**

In 1962 the Government set out an investment strategy for hospital building in England and Wales (Cmnd. 1604) and in Scotland (Cmnd. 1602) over the period up to 1970. One of the main objectives of the Scottish hospital plan was the provision of maternity hospital beds in line with the

recommendations of the Montgomery Committee. The maternity bed target was somewhat curiously restated as 0.69 maternity beds per 1000 of population so as to permit the extrapolation of bed requirements given population projections up to 1975.[6]

The Hospital Plan assumed that by 1970 the maternity bed stock would reach 3400, an increase of 15 per cent over the 1960-1970 period. In order to bring supply in line with expected demand, investment was concentrated in the Western Regional Hospital Board. Although the largest absolute bed deficit fell within the boundary of the Western RHB (484 beds or 29 per cent of the 1961 maternity bed complement), relative shortfalls in supply, however, were actually larger in the South-Eastern RHB (-32.2 per cent) and particularly, the Eastern RHB (-54.6 per cent). In contrast, the level of maternity

---

6. Regional maternity bed requirements were based on the assumption that changes in the size of the fertile population, fertility rates, trends in service utilisation (hospital confinement, length of stay) and other factors influencing the need for maternity hospitals were directly proportional to regional population size. No further allowance was made for differences in the size or composition of the obstetric population.

bed provision in 1961 in the Northern RHB was only 7 per cent below the 1975 target whereas the population of the North-Eastern RHB enjoyed access to a level of provision in 1961 which was 7 per cent above the target.

The 1962 Hospital Building programme was revised in 1964 and reviewed in 1966 (Cmnd. 2877). The 1966 review essentially supported the investment priorities outlined in the 1962 programme. Maternity services were still planned in accordance with the Montgomery Committee recommendations with the following qualification. Responding to studies which suggested that, at least in Glasgow, the need for hospital confinement exceeded the confinement ratio established by the Montgomery Committee, the review emphasised the importance of concentrating further maternity hospital development in areas of greatest need as (inversely) measured by the proportion of hospital confinements.

Throughout the 1960's the maternity bed:population ratios recommended by the Cranbrook and Montgomery Reports and the 1962 Hospital Plans were questioned. Newell (1964) presented an elaborate statistical model of the demand for maternity beds and demonstrated the sensitivity of bed ratios to differing fertility rates and length of stay in

hospital. Paige (1962) argued that a 60 per cent increase in the stock of maternity beds was a more appropriate target than the increase of 32 per cent outlined in the 1962 Hospital Plan for England and Wales. Her argument revolved around a higher proportion of hospital confinement (80-85 per cent) and birth forecasts by the National Institute of Economic and Social Research which exceeded official birth projections by 12 per cent. Feldstein (1965) demonstrated that the selection criterion for hospital confinement was inefficient and could be improved by a multivariate approach to assessing the risk of perinatal mortality. McEwan (1967) presented revised bed ratios of between 35-48 maternity beds per 100,000 population compared to the 58 ratio suggested by Cranbrook. The planning of maternity provision on the basis of uniform bed:population ratios was criticised by Marshall (1967) who proposed alternative criterion based on a more sophisticated appraisal of the number of births and the proportion of hospital deliveries. Golding (1967) suggested that because birth rates were notoriously difficult to predict maternity units should be multi-purpose so that beds could be used for other specialties.

### A.5 Tennent Committee

The death knell of the home confinement and the predominance of maternity care provided in specialist obstetric units was heralded by the Tennent Report on the Integration of Maternity Work in Scotland (Scottish Home and Health Department, 1973). Published soon after the Peel Report on Domiciliary Midwifery and Maternity Bed Needs in England and Wales, the Tennent Committee recommended the establishment of maternity service districts, co-terminous with the districts introduced by Health Boards in accordance with the 1974 administrative reorganisation of the health and local authorities in Scotland. The report discusses the function and administration of the matenity service and the respective roles and authority of the hospital medical staff, general practitioners, midwives and other health professionals involved in maternity care. Although the level of provision is not made explicit, all pregnant women were to have access to the "complete range of maternity services" based on specialist obstetric units with at least 80 beds. The report endorsed the rapidly approaching 100 per cent institutional confinement rate and went so far as to recommend that the resources of health education be mobilised to reduce the proportion of home confinements (4 per cent in 1971). Unlike the Montgomery Report and the 1962 Hospital Plan no maternity bed ratios are presented and no indication

is given as to whether the current stock of maternity resources was sufficient to satisfy existing and expected needs. Furthermore, in contrast to the detailed calculations presented in the Peel Report, policy recommendations were not based on statistical studies of maternity bed needs conducted (or commissioned) by the Tennent Committee. Acknowledging that Scotland had nearly achieved one longstanding maternity target (universal hospital confinement) the committee set out policy objectives which concentrated on changing the mix of maternity services in favour of more specialised care.

### A.6 Expenditure Targets

By the mid 1970s NHS spending was subject to public expenditure constraints which coincided with the slow down in economic growth. At the same time explicit bed targets were largely superseded by expenditure targets. This reflected an emerging trend in favour of policy objectives stated in the form of expenditure shares. These novel expenditure targets typically took the form of conventional programme budgets for specific client groups or global budgets for total NHS expenditure derived from the resource allocation formulae adopted by the Health Departments throughout the U.K. for distributing resources amongst health authorities.

The introduction of expenditure targets heralded a new era characterised, at least as far as the Government was concerned, by an oversupply of maternity beds. For example, the DHSS consultative document, Priorities for Health and Social Services in England (Department of Health and Social Security, 1976), recommended a cut in expenditure on maternity services as the birth rate was falling and the service was overprovided. Similar sentiment was expressed by "The Way Ahead" a planning document issued by the Scottish Home and Health Department (1976) which outlined a shift of resources away from the acute and maternity hospital sectors. Noting the dramatic fall in Scottish births from 104,000 in 1964 to around 70,000 in the mid 1970's the report observed that 99 per cent of all births took place in hospital. Despite the declining birth rate, expenditure on obstetrics and related services had grown over the past decade. This apparent excess supply in maternity resources was not wholly due to falling birth numbers. Alongside a decline in average length of stay, the supply of beds increased between 1962 and 1975 in line with the strategy proposed by the 1962 Hospital Plan. The report also suggested that shorter lengths of stay in hospital would also enable expenditure to be reduced

without compromising maternal and infant health. However, no quantitative estimate of the likely reduction in expenditure is provided.

In 1980 Scottish NHS expenditure targets were restated in terms of rates of growth in expenditure on specific health services set out in Scottish Health Authorities Priorities for the Eighties (SHAPE) (Scottish Home and Health Department, 1980). Rehabilitating the notion of a target bed supply figure, this report argued that the existing maternity bed stock was sufficient to cater for the forecast peak in births which according to the Registrar General's 1977 projection was due to occur in 1988/89 (88,300 births). By reducing the average length of stay from 7 to 6 days and increasing the occupancy ratio from 64 to 80 per cent this remarkable projected increase (42 per cent) in births from their 1977 level could be accommodated. Consequently, no increase in maternity beds was anticipated and maternity services were categorised as "B" programmes (i.e., revenue expenditure would increase, but at a lower rate than the top priority programmes such as services for the elderly, the mentally ill and the handicaped).

Finally, maternity services continued their migration down the league table of priorities in 1988 when SHARPEN (Scottish Health Authorities Review of Priorities for the Eighties and Nineties) appeared (Scottish Home and Health Department, 1988). SHARPEN concluded that absolute savings could be made by a more effective and efficient allocation of available resources, rather than by an increase in the quantity of resources devoted to perinatal care.

Table 5.1 Expenditure on Perinatal Hospital Services in Scotland

| Year | Inpatient Cases | Average Cost Per Case (£) | Total Inpatient Cost (£m) | Outpatient Attendances | Average Cost per Attendance (£) | Total Outpatient Cost (£m) | Total Hospital Cost (£m) |
|---|---|---|---|---|---|---|---|
| 1951 | 69893 | 22.75 | 1.590 | 279570 | 0.29 | 0.081 | 1.671 |
| 1952 | 72810 | 24.06 | 1.752 | 291240 | 0.26 | 0.076 | 1.828 |
| 1953 | 73873 | 24.39 | 1.802 | 295490 | 0.29 | 0.086 | 1.887 |
| 1954 | 75343 | 25.33 | 1.908 | 301370 | 0.30 | 0.090 | 1.999 |
| 1955 | 76460 | 26.92 | 2.058 | 305840 | 0.34 | 0.104 | 2.162 |
| 1956 | 81653 | 30.17 | 2.463 | 326610 | 0.39 | 0.127 | 2.591 |
| 1957 | 85750 | 33.65 | 2.885 | 343001 | 0.46 | 0.158 | 3.043 |
| 1958 | 90308 | 35.51 | 3.207 | 361232 | 0.47 | 0.170 | 3.377 |
| 1959 | 94023 | 36.91 | 3.470 | 379026 | 0.54 | 0.205 | 3.675 |
| 1960 | 97501 | 38.76 | 3.779 | 398550 | 0.58 | 0.231 | 4.010 |
| 1961 | 101992 | 41.26 | 4.208 | 396614 | 0.51 | 0.202 | 4.410 |
| 1962 | 108020 | 41.92 | 4.528 | 400392 | 0.52 | 0.208 | 4.736 |
| 1963 | 104796 | 45.68 | 4.787 | 419951 | 0.58 | 0.244 | 5.031 |
| 1964 | 107668 | 48.35 | 5.206 | 432799 | 0.60 | 0.260 | 5.465 |
| 1965 | 109110 | 52.74 | 5.754 | 439689 | 0.72 | 0.317 | 6.071 |
| 1966 | 112747 | 57.37 | 6.468 | 444955 | 0.77 | 0.343 | 6.811 |
| 1967 | 114392 | 56.66 | 6.481 | 427569 | 0.79 | 0.338 | 6.819 |
| 1968 | 113166 | 59.53 | 6.737 | 410751 | 0.84 | 0.345 | 7.082 |
| 1969 | 111178 | 65.8 | 7.316 | 407614 | 0.89 | 0.363 | 7.678 |
| 1970 | 112767 | 75.93 | 8.562 | 425442 | 1.10 | 0.468 | 9.030 |
| 1971 | 117358 | 86.98 | 10.208 | 414008 | 1.26 | 0.522 | 10.729 |
| 1972 | 113302 | 104.94 | 11.890 | 384409 | 1.43 | 0.550 | 12.440 |
| 1973 | 101685 | 124.04 | 12.613 | 385244 | 1.71 | 0.659 | 13.272 |
| 1974 | 98462 | 168.24 | 16.565 | 418885 | 2.15 | 0.901 | 17.466 |
| 1975 | 97949 | 224.45 | 21.985 | 410360 | 2.82 | 1.157 | 23.142 |
| 1976 | 97133 | 269.44 | 26.172 | 408695 | 3.46 | 1.414 | 27.586 |
| 1977 | 97419 | 294.6 | 28.700 | 401448 | 3.77 | 1.513 | 30.213 |
| 1978 | 101883 | 317.71 | 32.369 | 411889 | 4.00 | 1.648 | 34.017 |
| 1979 | 104452 | 348.66 | 36.418 | 431773 | 4.57 | 1.973 | 38.391 |
| 1980 | 106276 | 429.78 | 45.675 | 436922 | 5.62 | 2.456 | 48.131 |
| 1981 | 107196 | 500.73 | 53.676 | 430275 | 6.40 | 2.754 | 56.430 |
| 1982 | 105330 | 552.85 | 58.232 | 407139 | 6.81 | 2.773 | 61.004 |
| 1983 | 104873 | 563.62 | 59.109 | 391594 | 11.85 | 4.640 | 63.749 |
| 1984 | 103823 | 560.64 | 58.207 | 374030 | 11.93 | 4.462 | 62.670 |
| 1985 | 105553 | 569.85 | 60.149 | 284006 | 11.79 | 3.348 | 63.498 |

Note: All expenditure data in current prices

Table 5.2 Expenditure on Perinatal Hospital Services in Scotland
(Current and Real [1980] Prices)

| Year | Current Prices £m | 1980 Prices £m | NHS Implicit Price Deflator |
|---|---|---|---|
| 1951 | 1.671 | 18.990 | 0.088 |
| 1952 | 1.828 | 20.306 | 0.090 |
| 1953 | 1.887 | 20.516 | 0.092 |
| 1954 | 1.999 | 21.264 | 0.094 |
| 1955 | 2.162 | 22.064 | 0.098 |
| 1956 | 2.591 | 24.675 | 0.105 |
| 1957 | 3.043 | 27.417 | 0.111 |
| 1958 | 3.377 | 29.362 | 0.115 |
| 1959 | 3.675 | 30.626 | 0.120 |
| 1960 | 4.010 | 31.828 | 0.126 |
| 1961 | 4.410 | 32.914 | 0.134 |
| 1962 | 4.736 | 34.572 | 0.137 |
| 1963 | 5.031 | 35.427 | 0.142 |
| 1964 | 5.465 | 36.929 | 0.148 |
| 1965 | 6.071 | 37.944 | 0.160 |
| 1966 | 6.811 | 40.541 | 0.168 |
| 1967 | 6.819 | 38.096 | 0.179 |
| 1968 | 7.082 | 38.074 | 0.186 |
| 1969 | 7.678 | 38.779 | 0.198 |
| 1970 | 9.030 | 42.002 | 0.215 |
| 1971 | 10.729 | 45.082 | 0.238 |
| 1972 | 12.440 | 46.942 | 0.265 |
| 1973 | 13.272 | 46.083 | 0.288 |
| 1974 | 17.466 | 47.078 | 0.371 |
| 1975 | 23.142 | 49.767 | 0.465 |
| 1976 | 27.586 | 51.179 | 0.539 |
| 1977 | 30.213 | 51.122 | 0.591 |
| 1978 | 34.017 | 51.076 | 0.666 |
| 1979 | 38.391 | 48.844 | 0.786 |
| 1980 | 48.131 | 48.131 | 1.000 |
| 1981 | 56.430 | 51.160 | 1.103 |
| 1982 | 61.004 | 50.879 | 1.199 |
| 1983 | 63.749 | 50.474 | 1.263 |
| 1984 | 62.670 | 46.979 | 1.334 |
| 1985 | 63.498 | 45.130 | 1.407 |

Table 5.3 Tests of the Set of Regressors

| | Regressors | F-stat | CV* | DF** | Restrictions |
|---|---|---|---|---|---|
| MODEL 1 | W,x,rp,tw,pnm | - | - | - | 0 |
| MODEL 2 | W,x,rp,tw | 1.01 | 5.93 | (2,19) | 2 |
| MODEL 3 | W,x,rp | 7.92 | 4.50 | (4,19) | 4 |
| MODEL 4 | W,x | 17.16 | 3.94 | (6,19) | 6 |
| MODEL 5 | W | 20.46 | 3.63 | (8,19) | 8 |

Notes: Lagged and current values of regressors are included in each model.
All models also include an intercept.
* Critical Value (1 per cent)
** Degrees of Freedom

Table 5.4 Tests of Dynamic Structure

| MODEL | F-stat | CV* | DF** | Restrictions |
|---|---|---|---|---|
| GENERAL | - | - | - | 0 |
| STATIC REGRESSION | 21.09 | 4.50 | (4,19) | 4 |
| UNIVARIATE | 24.40 | 3.94 | (6,19) | 6 |
| PARTIAL ADJUSTMENT | 1.83 | 5.01 | (3,19) | 3 |
| DISTRIBUTED LAG | 12.66 | 8.19 | (1,19) | 1 |
| LEADING INDICATOR | 29.52 | 4.50 | (4,19) | 4 |
| DIFFERENCED DATA | 4.62 | 4.50 | (4,19) | 4 |

Notes: * Critical Value (1 per cent)
** Degrees of Freedom

Table 5.5 Alternative Models of the Demand for Perinatal Hospital Care

| MODEL | GENERAL MODEL | STATIC REGRESSION | UNI-VARIATE | PARTIAL ADJUST | DISTR LAG | LEADING INDICAT | 1ST DIFFER |
|---|---|---|---|---|---|---|---|
| REGRESSOR | | | | | | | |
| $W_{t-1}$ | 0.690 | | 0.967 | 0.470 | | | |
| | [6.468] | | [15.434] | [8.775] | | | |
| $x_t$ | 0.005 | 0.013 | | 0.003 | 0.018 | | |
| | [0.373] | [1.883] | | [0.668] | [0.723] | | |
| $x_{t-1}$ | -0.005 | | | | -0.005 | 0.016 | |
| | [-0.347] | | | | [-0.226] | [1.916] | |
| $rp_t$ | 0.048 | 0.067 | | 0.045 | 0.055 | | |
| | [6.447] | [5.312] | | [6.663] | [4.374] | | |
| $rp_{t-1}$ | -0.019 | | | | 0.012 | 0.047 | |
| | [-1.884] | | | | [0.781] | [2.947] | |
| $tw_t$ | 0.031 | 0.046 | | 0.024 | 0.020 | | |
| | [4.890] | [7.201] | | [5.901] | [1.990] | | |
| $tw_{t-1}$ | -0.159 | | | | 0.026 | 0.048 | |
| | [-1.869] | | | | [2.797] | [6.961] | |
| $\Delta x_i$ | | | | | | | 0.009 |
| | | | | | | | [0.590] |
| $\Delta rp$ | | | | | | | 0.046 |
| | | | | | | | [5.088] |
| $\Delta tw$ | | | | | | | 0.035 |
| | | | | | | | [5.246] |
| CONSTANT | 0.075 | 0.152 | 0.002 | 0.115 | 0.158 | 0.128 | -0.000 |
| | [2.869] | [3.962] | [0.495] | [5.861] | [4.134] | [2.715] | [-0.122] |
| R-SQ | 0.983 | 0.905 | 0.882 | 0.977 | 0.948 | 0.874 | 0.783 |
| SER*100 | 0.124 | 0.264 | 0.310 | 0.131 | 0.209 | 0.304 | 0.159 |
| F | 168.900 | 79.590 | 238.210 | 260.460 | 66.550 | 57.940 | 30.080 |
| DW | 2.040 | 0.550 | 1.190 | 1.718 | 1.029 | 1.611 | 1.487 |
| RSS | 0.000032 | 0.000175 | 0.000308 | 0.000041 | 0.000096 | 0.000232 | 0.000063 |

Notes: t-statistics in brackets
W = share of hospital expenditure on perinatal care
x = log total hospital expenditure
rp = log relative price of perinatal and non-perinatal hospital care
tw = log target perinatal hospital share

**Figure 5.1 Perinatal Hospital Budget Shares**

**Scotland: 1951-85**

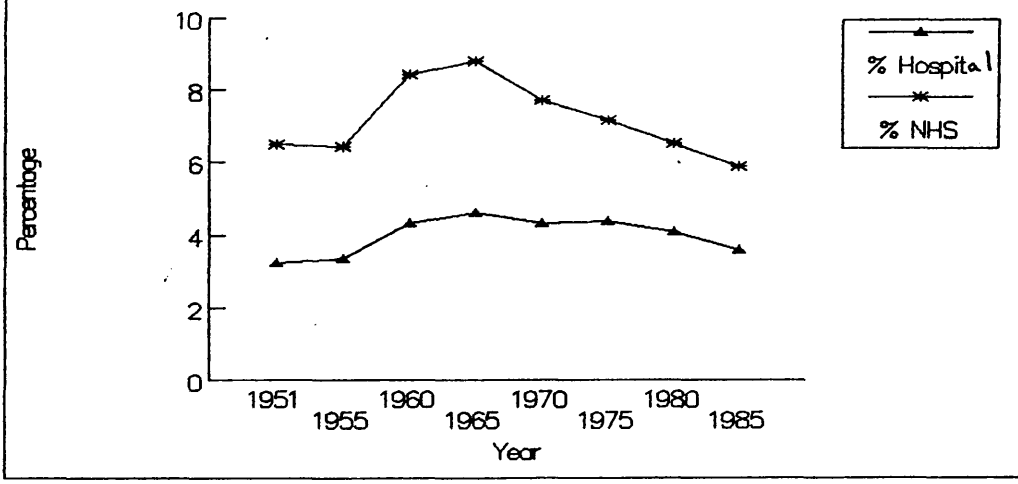
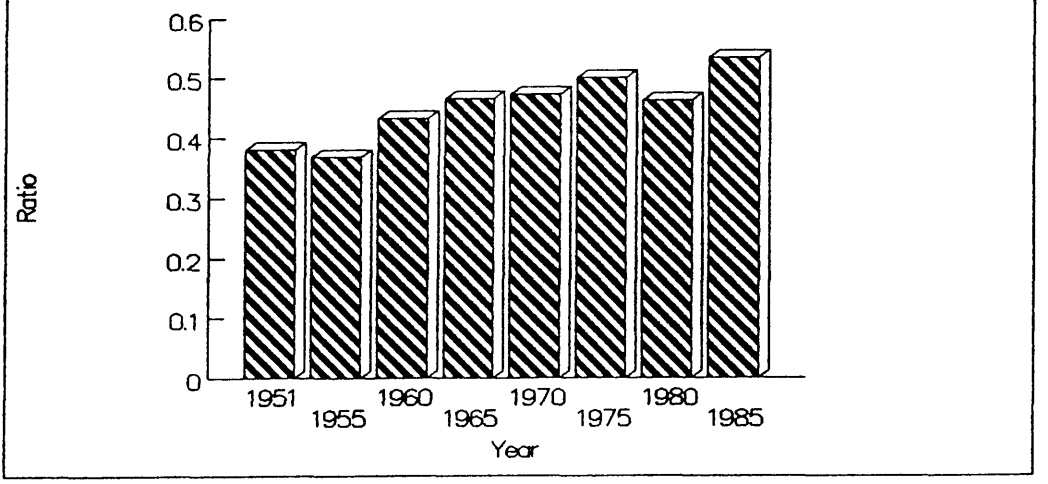


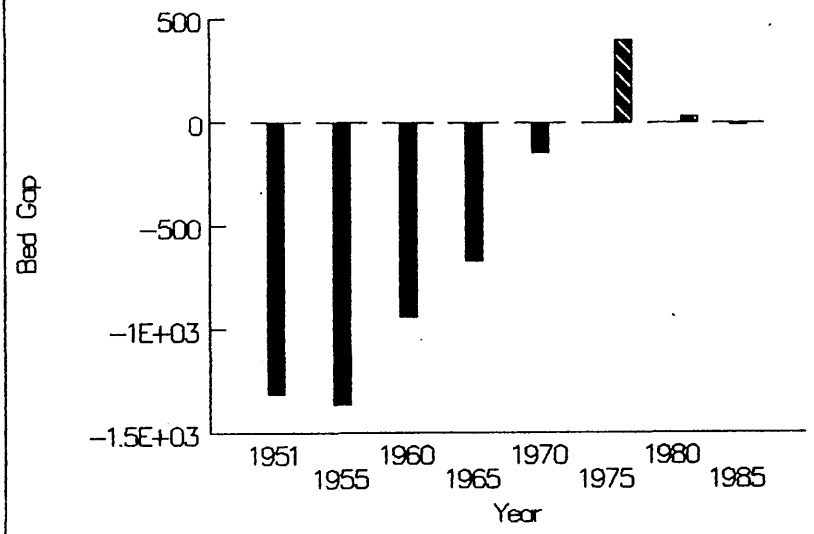

**Figure 5.2 Ratio of Average Cost per Case**

**Perinatal/Non-Perinatal Hospital Cases**



## Figure 5.3 Obstetric Bed Gap

### (Splined Orr and Montgomery Bed Targets)




## Figure 5.4 Perinatal Budget Shares

### Actual and Target Shares

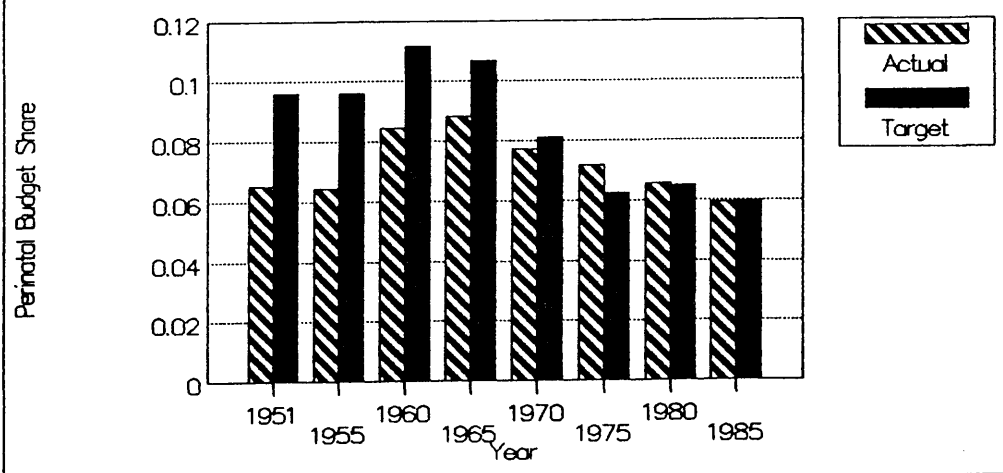


Figure 5.5 Actual and Fitted Values (General Model)

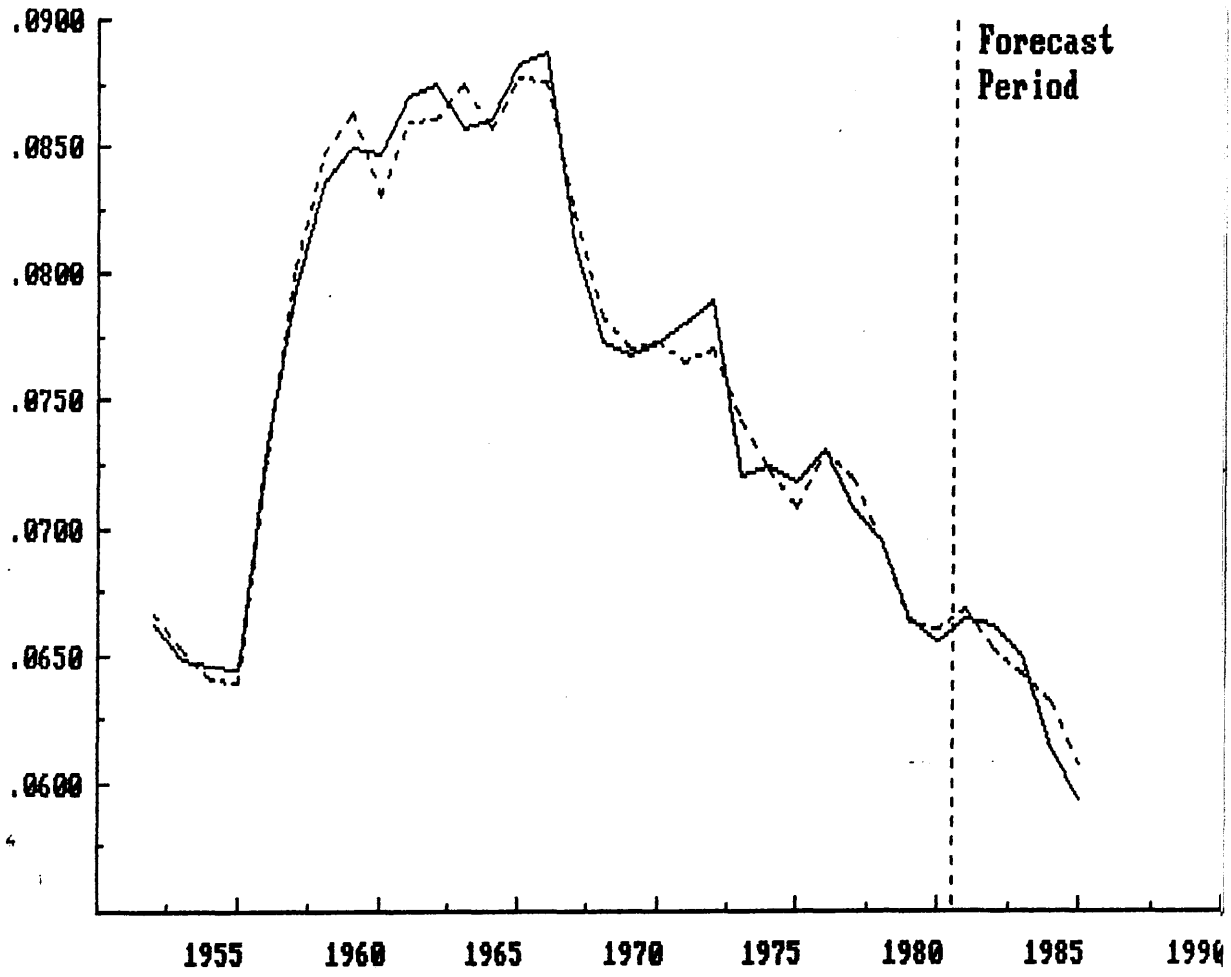
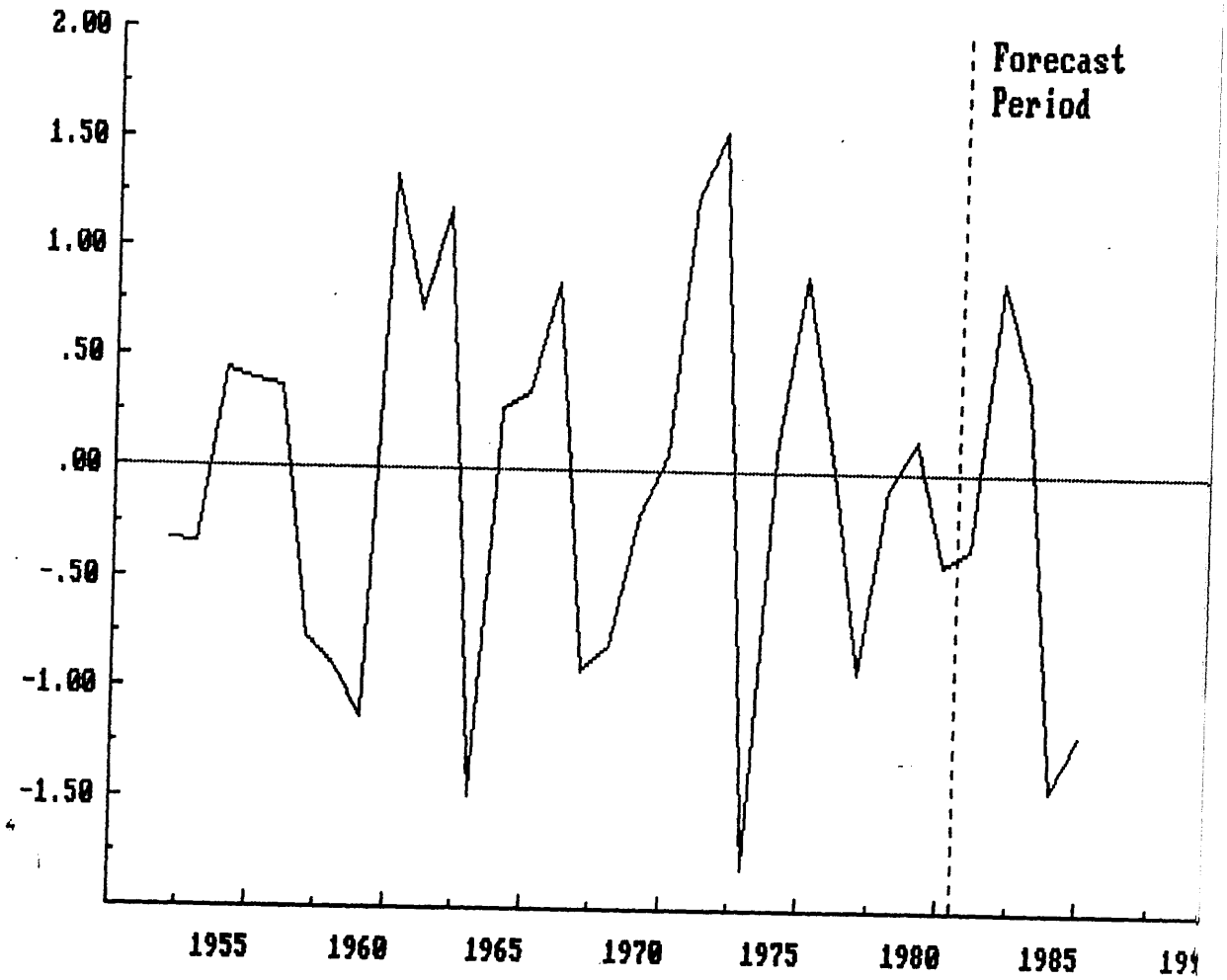


Figure 5.6 Scaled Residuals (General Model)



## Figure 5.7 Partial Adjustment Model

## Lag Distribution (Mean Lag = 0.887)

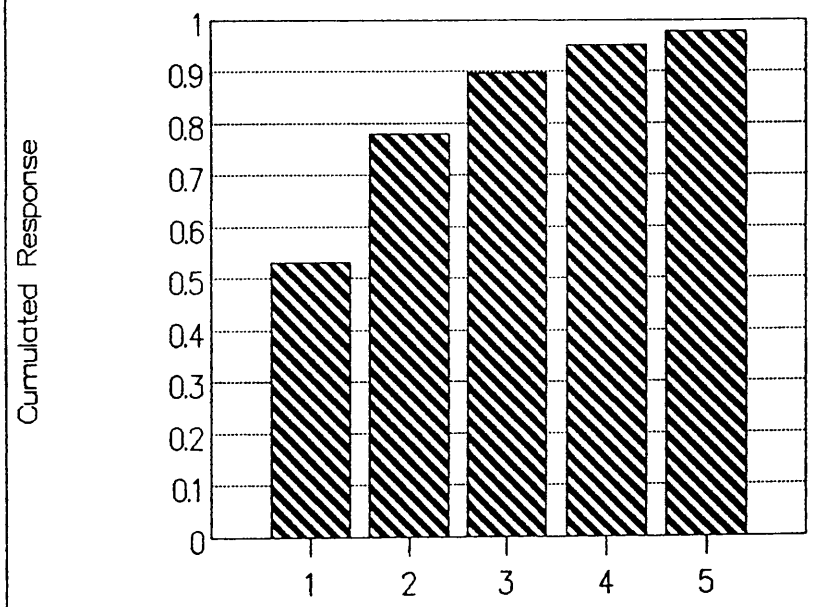


Figure 5.8 Actual and Fitted Values (Partial Adjustment Model)
W =____ FITTED =-- --

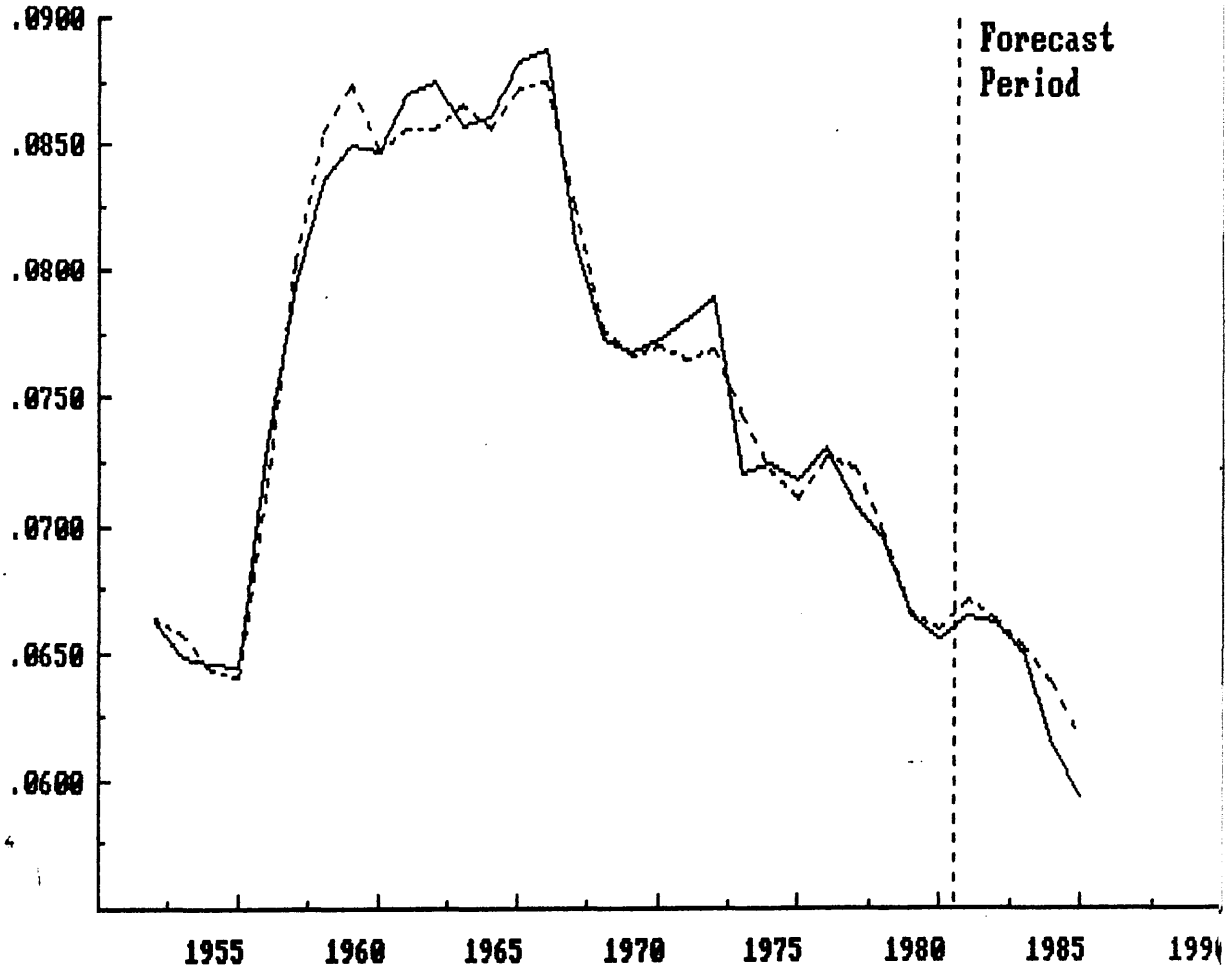


Figure 5.9 Scaled Residuals (Partial Adjustment Model)

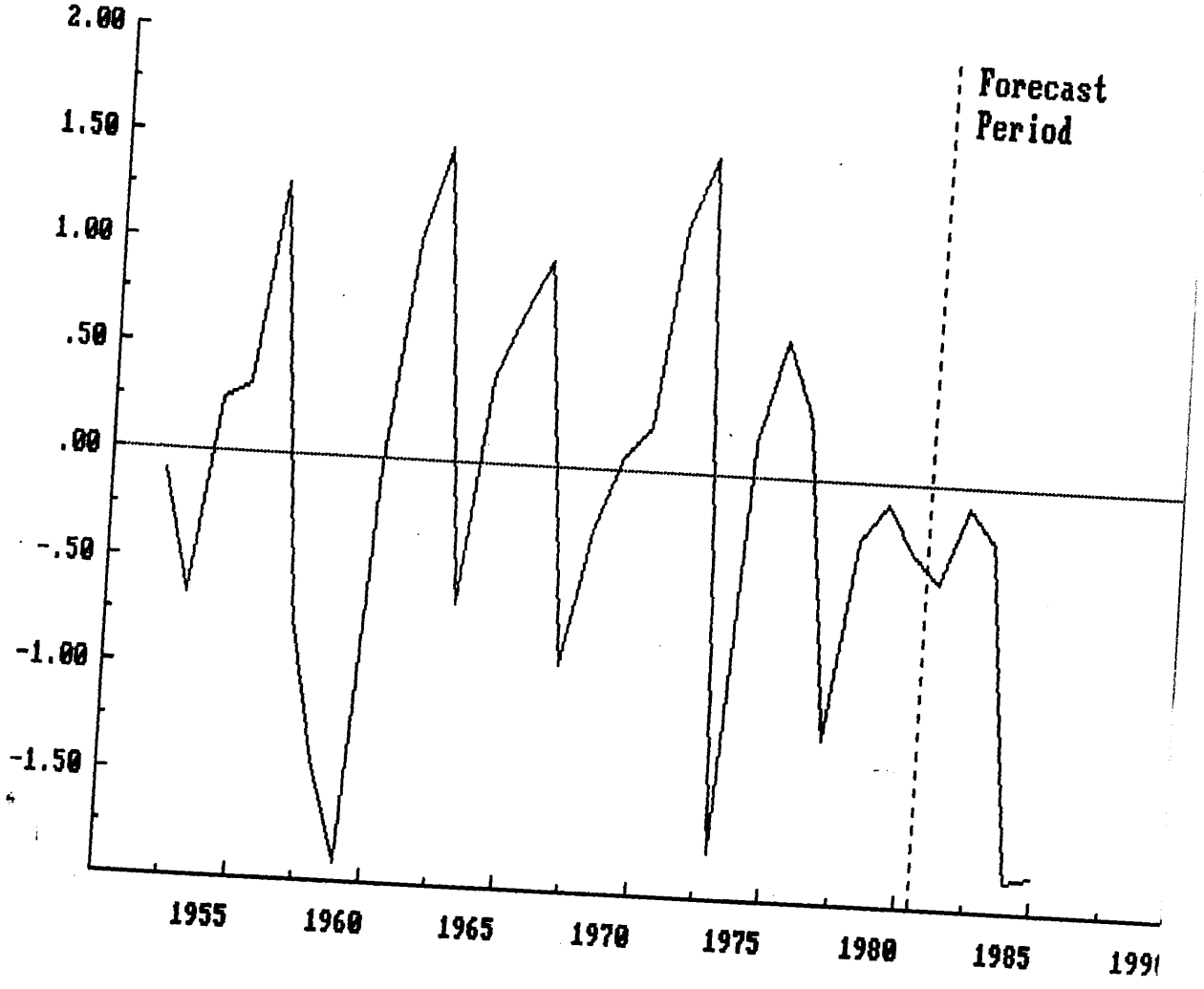


Figure 5.10 Parameter Stability over Time (Partial Adj Model)
W =____ ± 2*S.E.=-- --

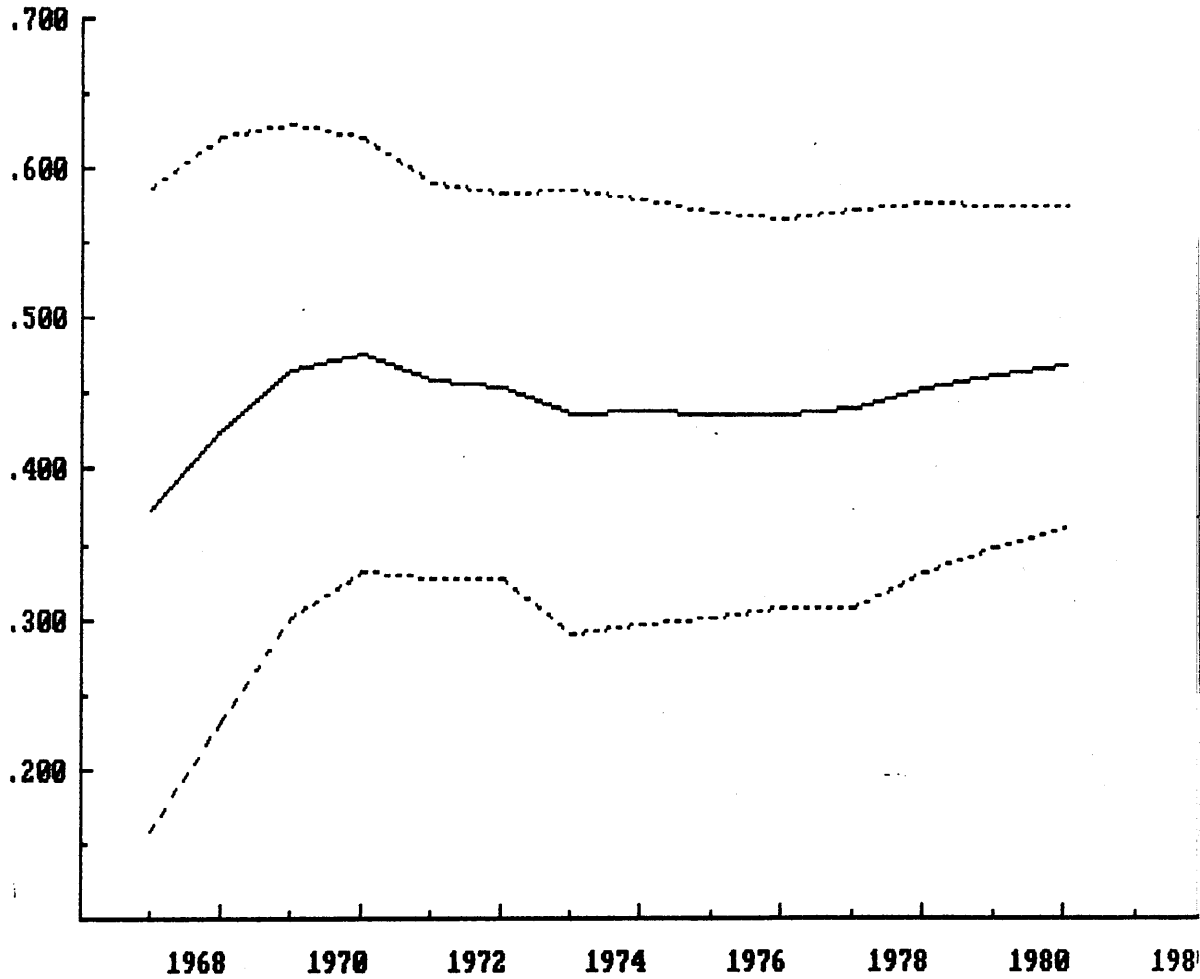


Figure 5.11 Parameter Stability Over Time (Partial Adj Model)

h =____ ± 2*S.E.=— —

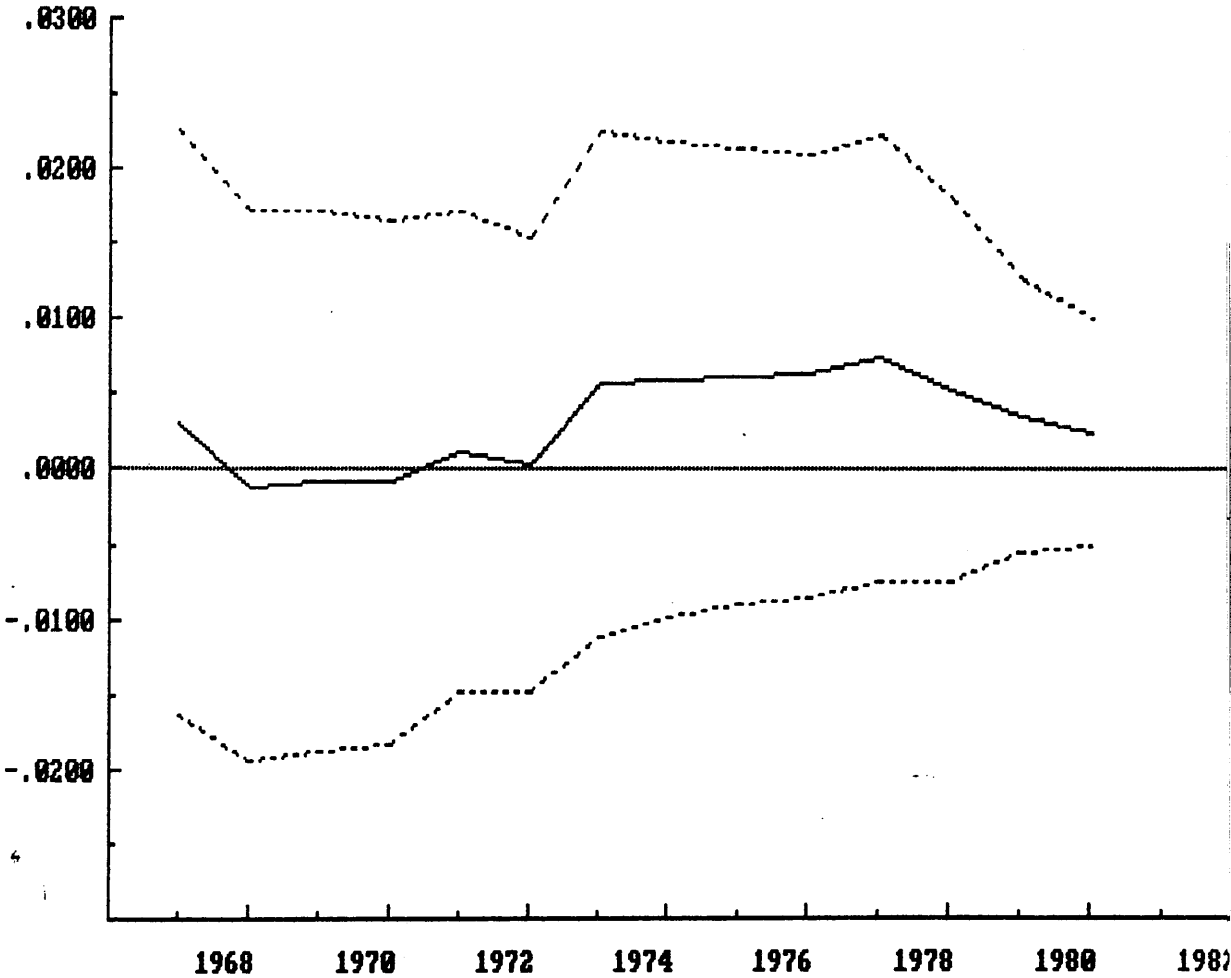


Figure 5.12 Parameter Stability Over Time (Partial Adj Model)
rp =_____ ± 2*S.E.=— —

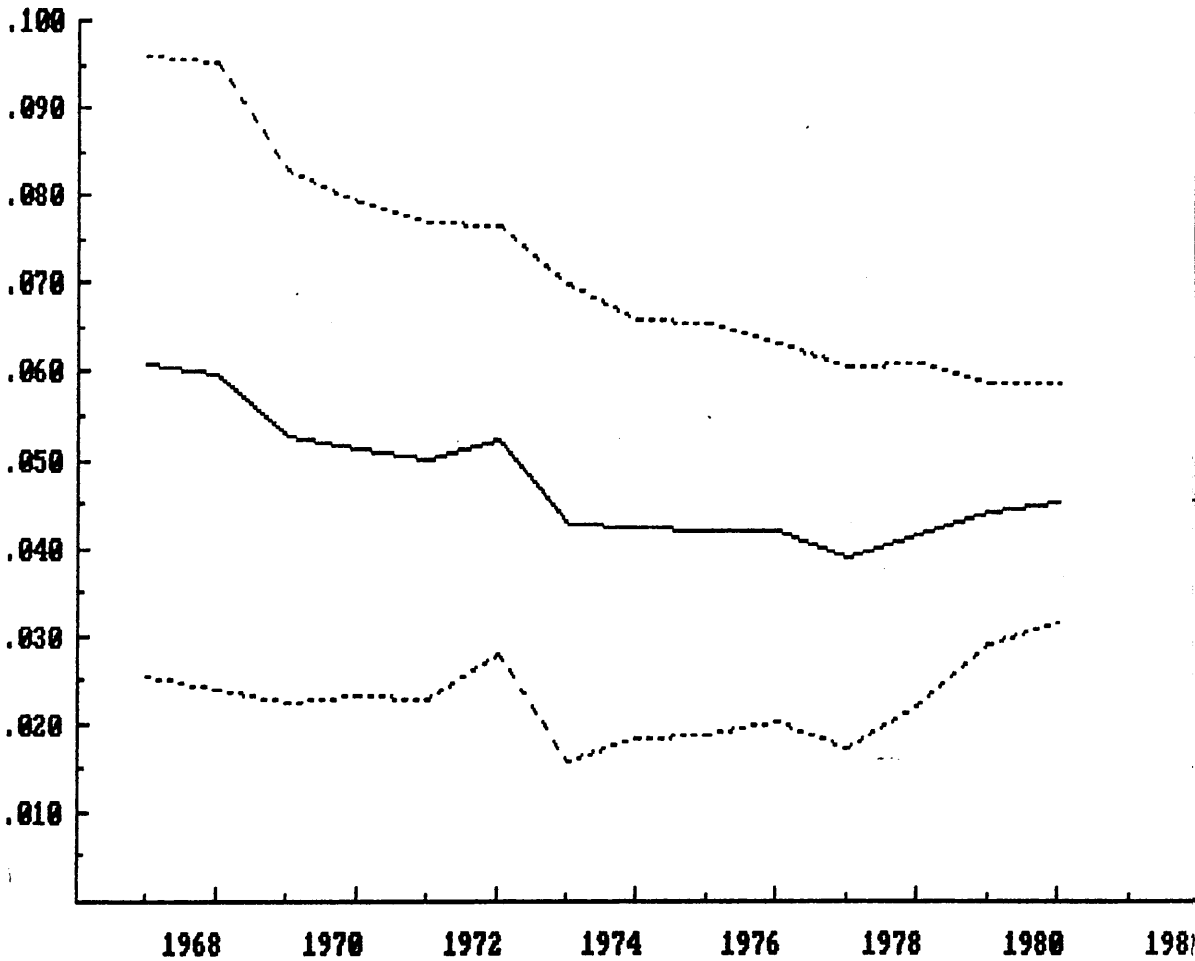


Figure 5.13 Parameter Stability Over Time (Partial Adj Model)

tw =____ ± 2*S.E.=— —

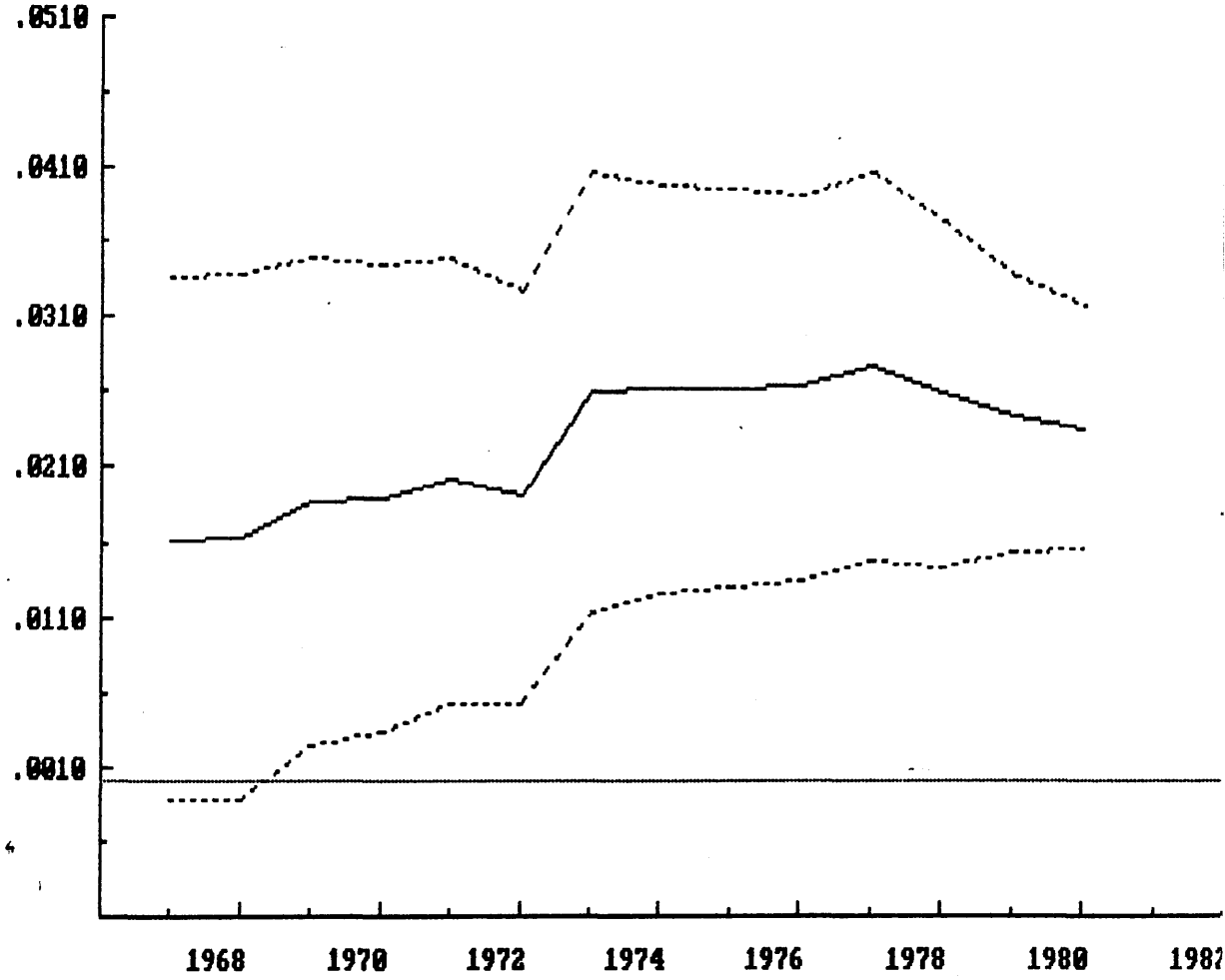


Figure 5.14 Actual and Fitted Values (Differenced Data Model)

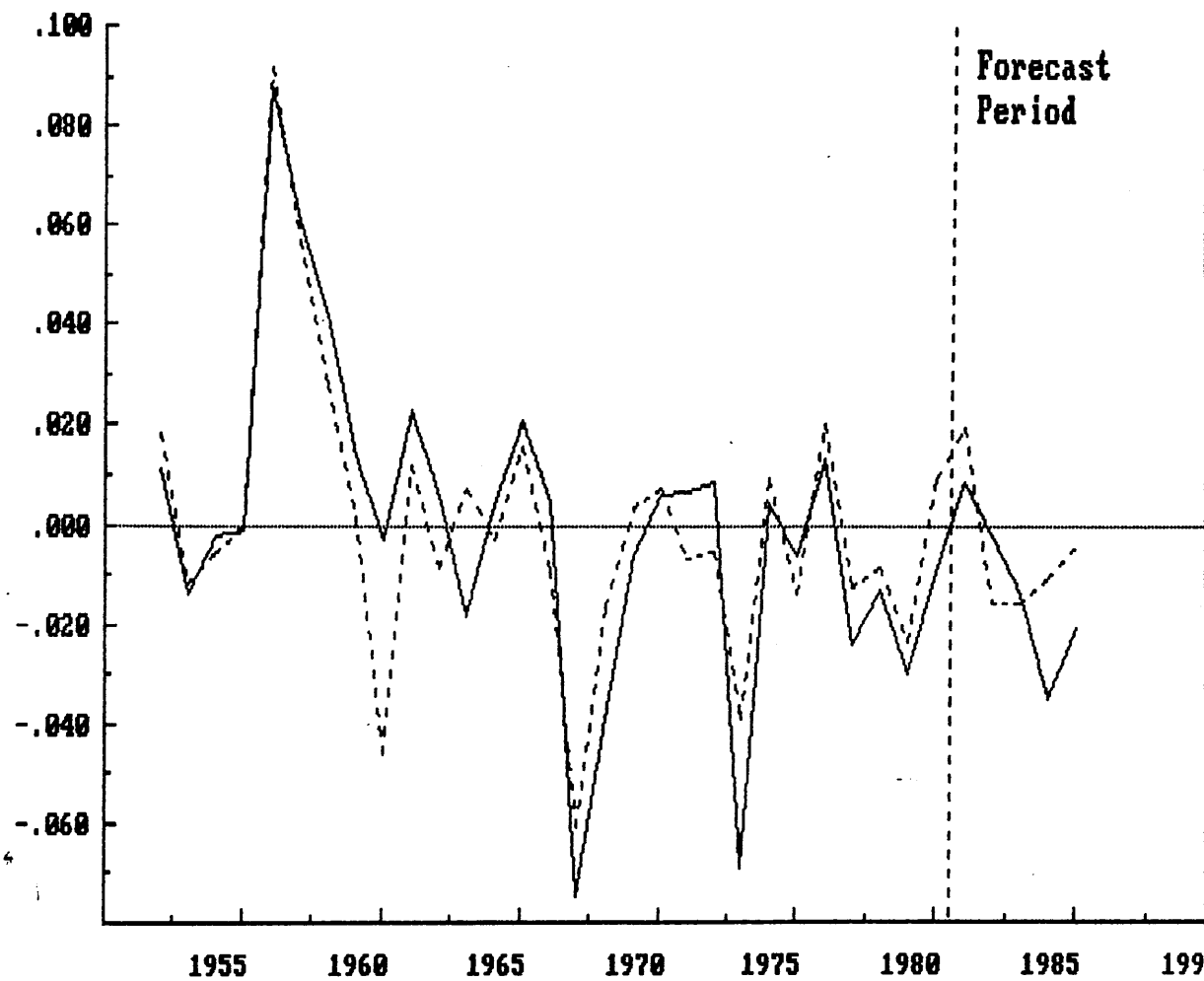


Figure 5.15 Scaled Residuals (Differenced Data Model)

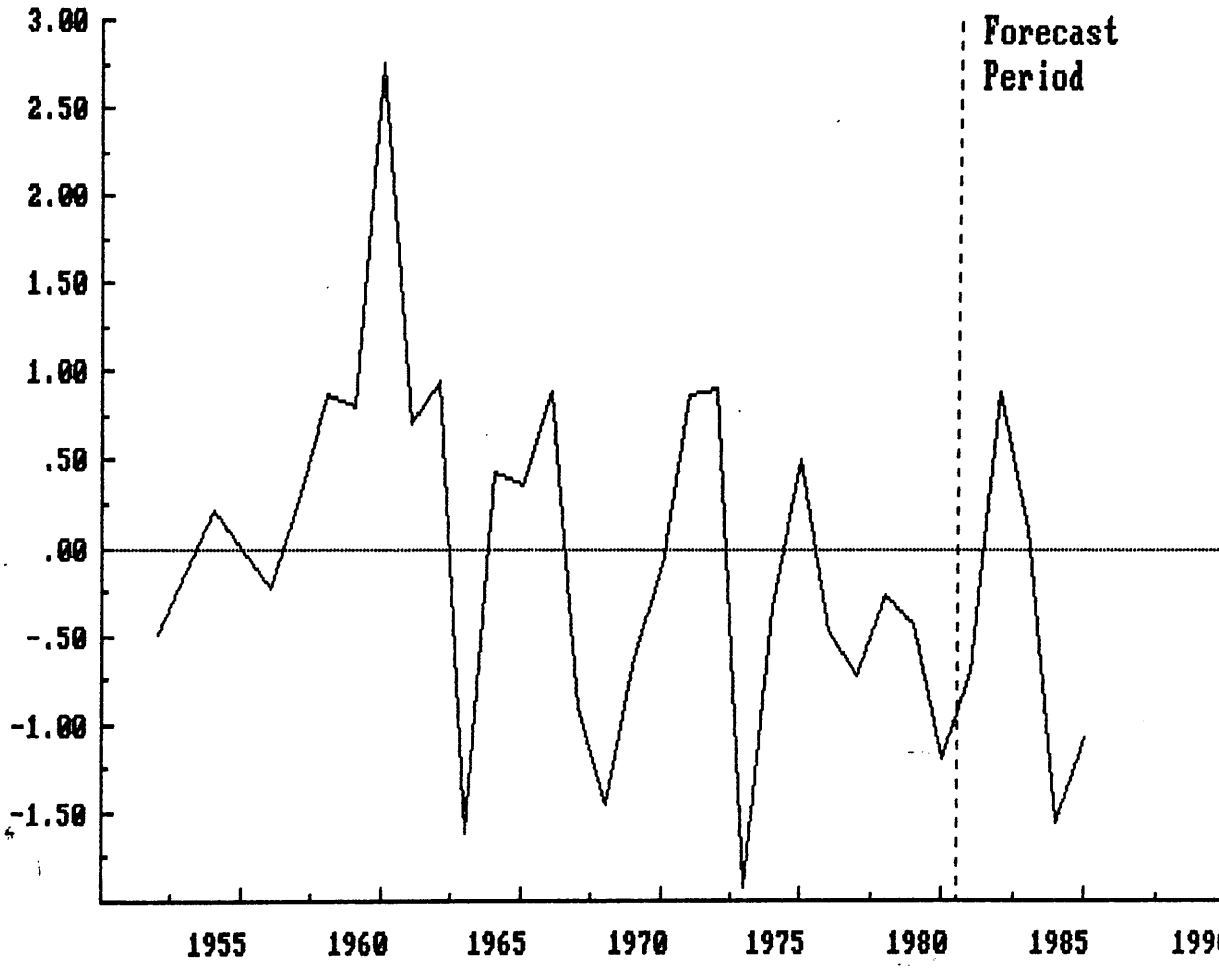


Figure 5.16 Parameter Stability Over Time (Differenced Data Model)

$\Delta x$ =_____ ± 2*S.E.=— —

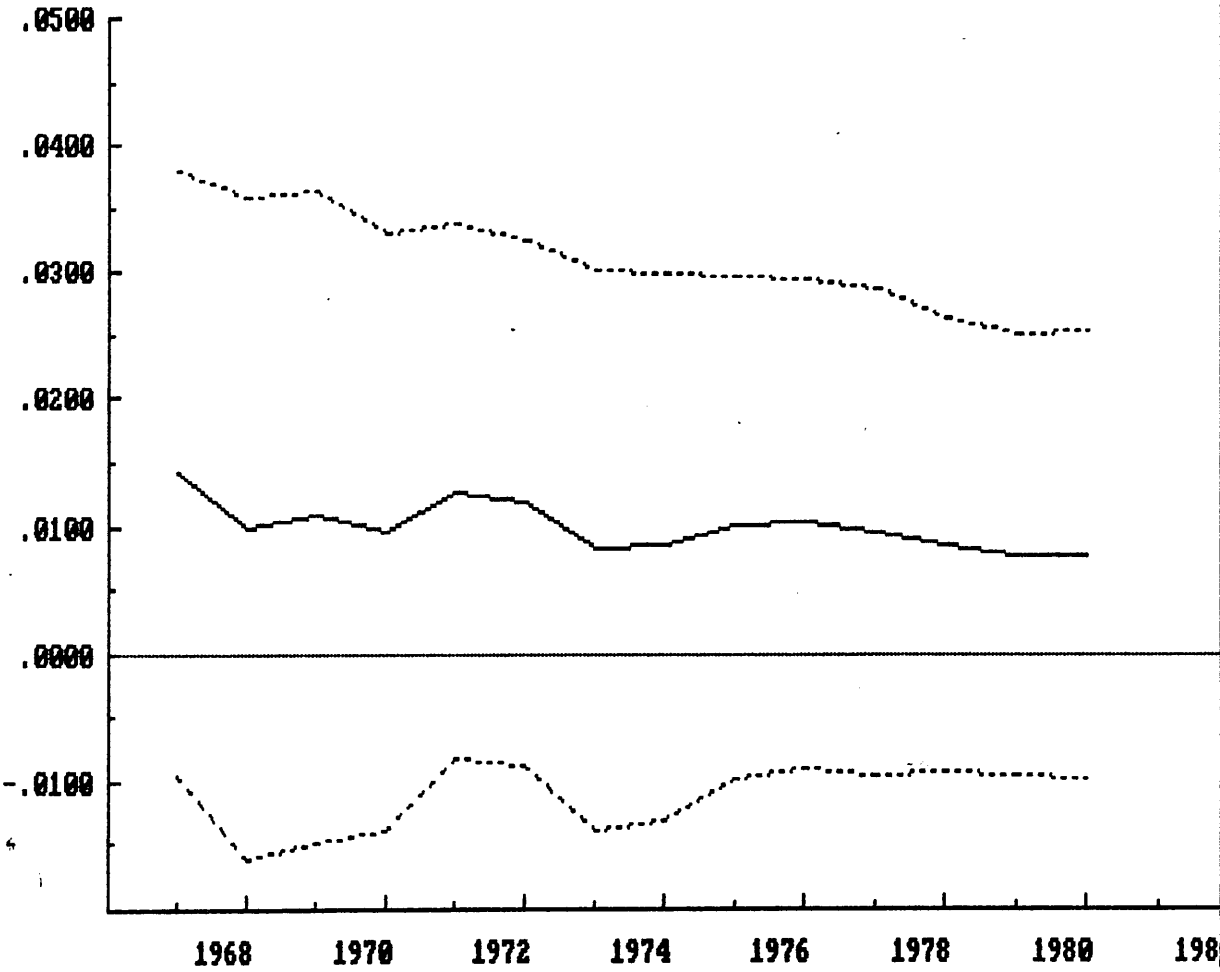


Figure 5.17 Parameter Stability Over Time (Differenced Data Model

Δ rp =_____ ± 2*S.E.=-- --

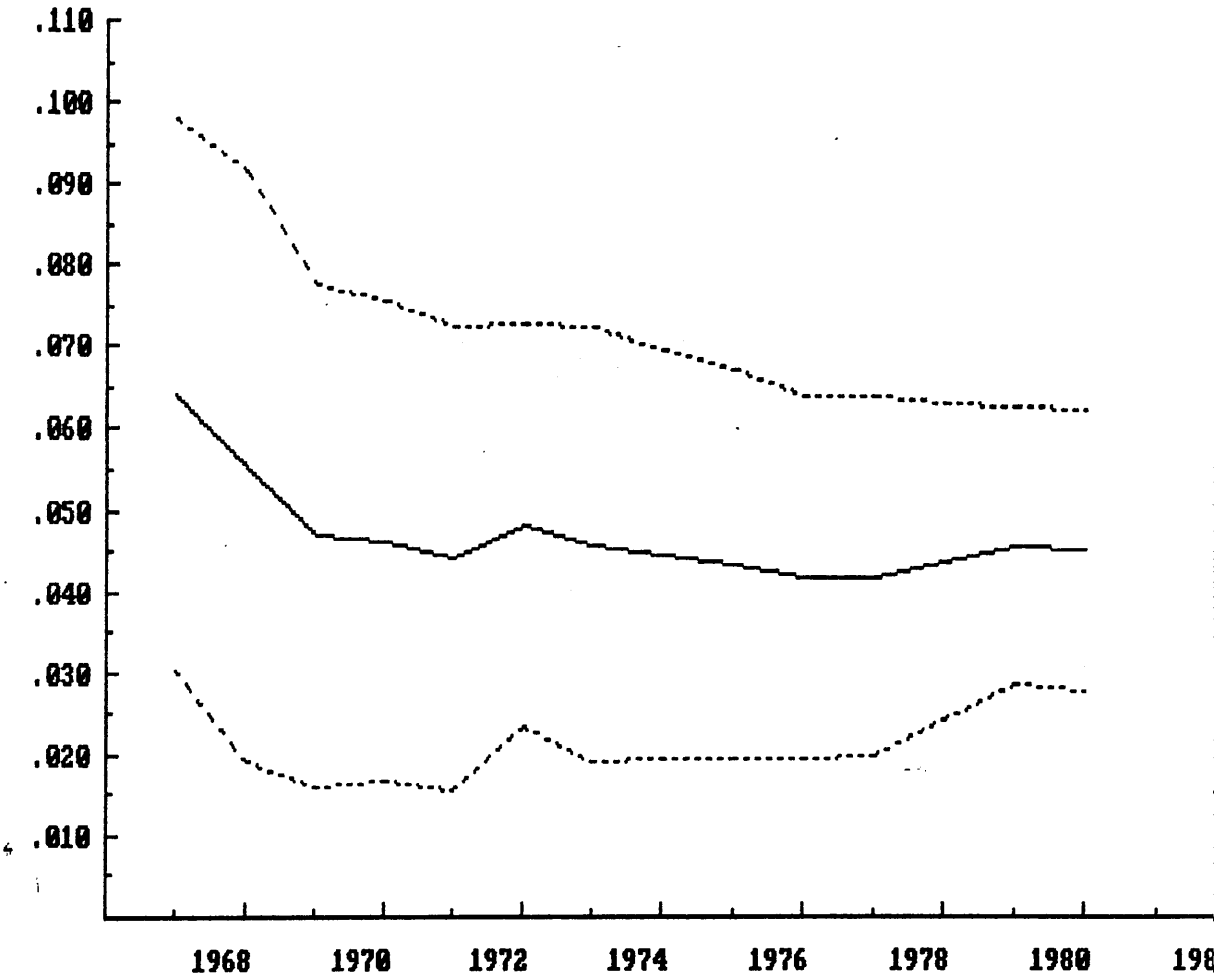


Figure 5.18 Parameter Stability Over Time (Differenced Data Model)

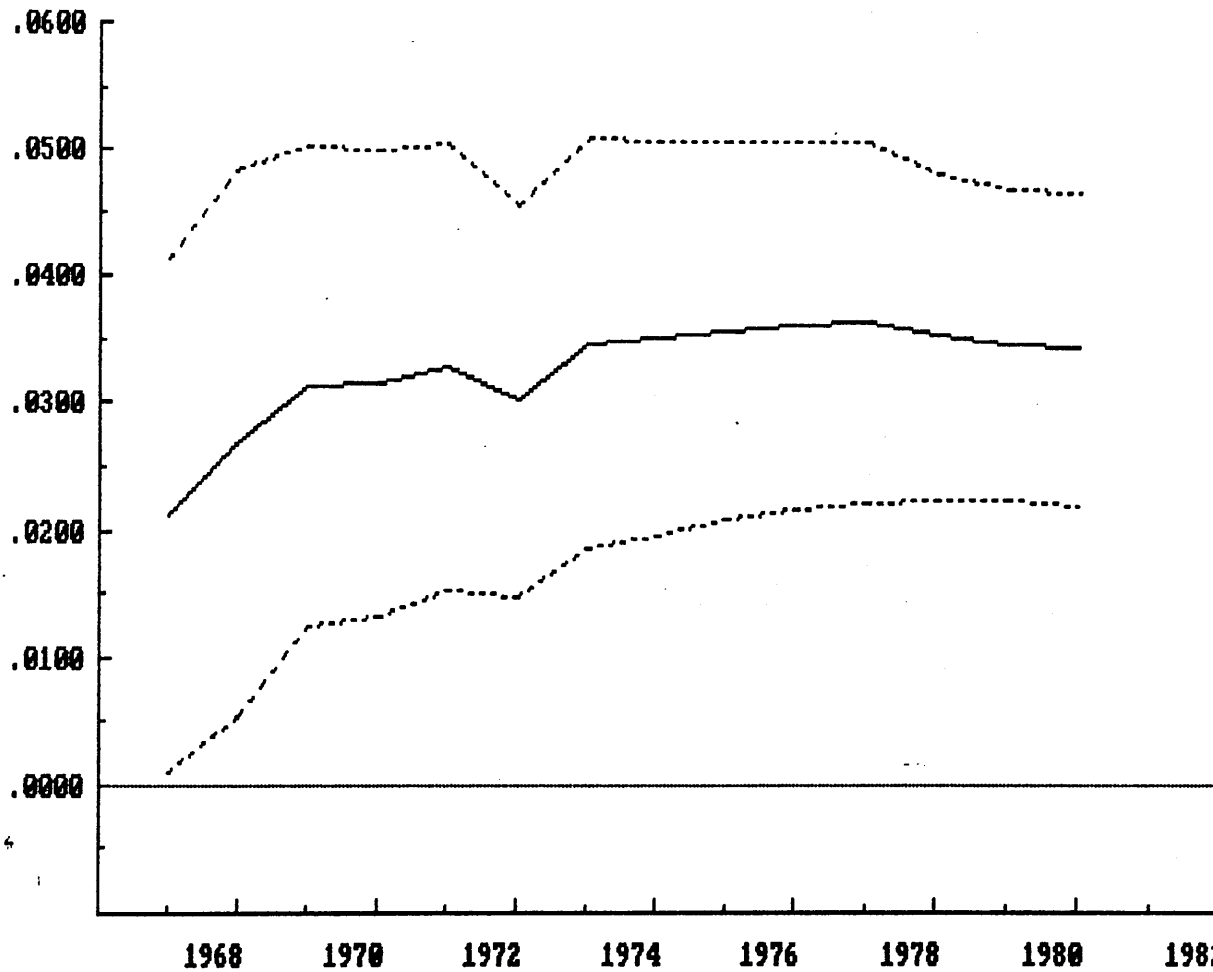


Figure 5.19 Actual and Fitted Values (Equation 5.12)

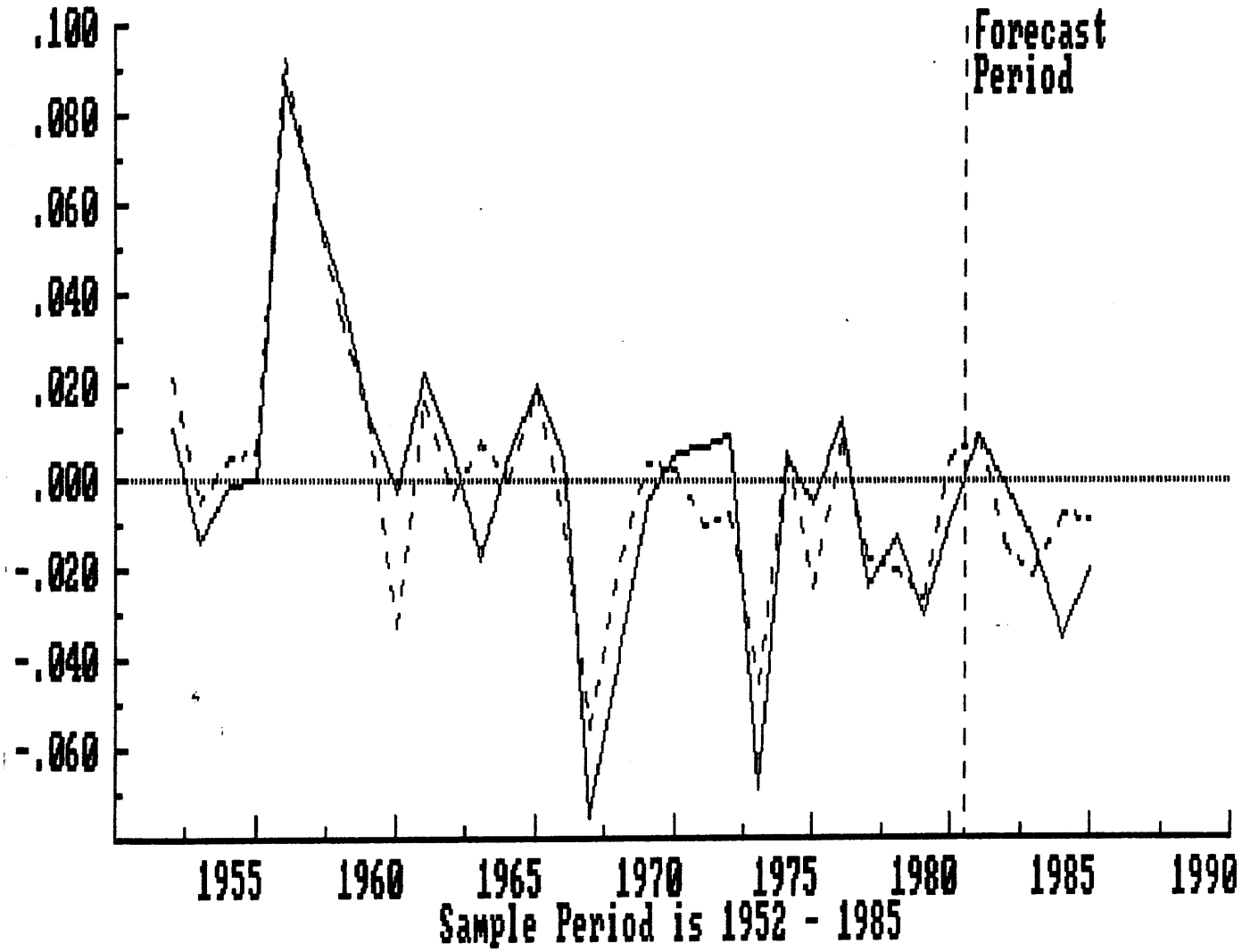


Figure 5.20 Scaled Residuals (Equation 5.12)

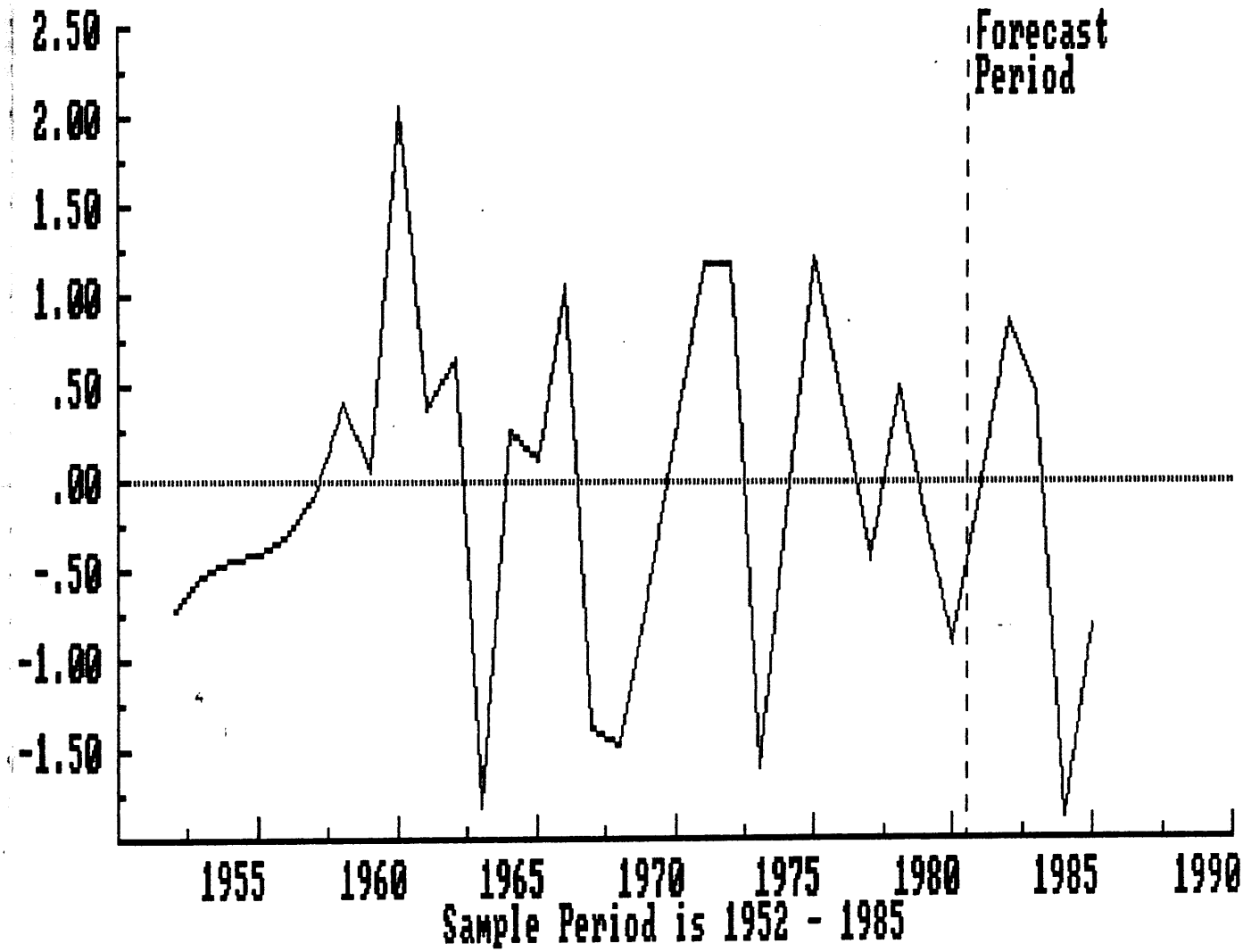


Table A5.1 Sample Autocorrelations of the Demand Function Variables

| Variable | Sample Autocorrelations (lag) | | | | |
|---|---|---|---|---|---|
| | 1 | 2 | 3 | 4 | 5 |
| W | 0.9469 | 0.8707 | 0.8209 | 0.8023 | 0.7598 |
| x | 0.9968 | 0.9933 | 0.9873 | 0.9800 | 0.9703 |
| rp | 0.7691 | 0.5160 | 0.3683 | 0.3367 | 0.2145 |
| tw | 0.9850 | 0.9620 | 0.9426 | 0.9292 | 0.8986 |
| pnm | 0.9942 | 0.9922 | 0.9874 | 0.9784 | 0.9748 |
| random walk | 0.9500 | 0.9000 | 0.8500 | 0.8100 | 0.7600 |

Notes: Sample autocorrelations from a random walk obtained from Nelson and Plosser (1982).

W = share of hospital expenditure on perinatal care
x = log total hospital expenditure
rp = log relative price of perinatal and non-perinatal care
tw = log target perinatal hospital share
pnm = log perinatal mortality rate

Table A5.2 Sample Autocorrelations of the Demand Function Variables First Differences

| Variable | Sample Autocorrelations (lag) | | | | |
|---|---|---|---|---|---|
| | 1 | 2 | 3 | 4 | 5 |
| $\Delta$ W | 0.3015 | -0.0664 | -0.1870 | 0.2252 | 0.2054 |
| $\Delta$ x | 0.0618 | 0.4644 | 0.1697 | 0.3178 | 0.1118 |
| $\Delta$ rp | 0.0716 | -0.1726 | -0.2675 | 0.1474 | -0.0847 |
| $\Delta$ tw | 0.3123 | -0.0616 | -0.1358 | 0.1934 | -0.0035 |
| $\Delta$ pnm | -0.2802 | 0.3455 | 0.2755 | -0.2093 | 0.2054 |
| random walk | 0.2500 | 0.0000 | 0.0000 | 0.0000 | 0.0000 |

Notes: Sample autocorrelations from a random walk obtained from Nelson and Plosser (1982).

W = share of hospital expenditure on perinatal care
x = log total hospital expenditure
rp = log relative price of perinatal and non-perinatal care
tw = log target perinatal hospital share
pnm = log perinatal mortality rate

Table A5.3 Sample Autocorrelations of the Deviations from a Time Trend

| Variable | Sample Autocorrelations (lag) | | | | |
|---|---|---|---|---|---|
| | 1 | 2 | 3 | 4 | 5 |
| W | 0.9300 | 0.7955 | 0.6349 | 0.4714 | 0.2508 |
| x | 0.8843 | 0.7535 | 0.5527 | 0.3336 | 0.1291 |
| rp | 0.8149 | 0.5445 | 0.3002 | 0.1424 | -0.0620 |
| tw | 0.9094 | 0.7418 | 0.5313 | 0.3070 | 0.0609 |
| pnm | 0.8875 | 0.8344 | 0.6708 | 0.4398 | 0.2146 |
| Detrended random walk | 0.8500 | 0.7100 | 0.5800 | 0.4700 | 0.3600 |

Notes: Sample autocorrelations from a detrended random walk obtained from Nelson and Plosser (1982).

W = share of hospital expenditure on perinatal care
x = log total hospital expenditure
rp = log relative price of perinatal and non-perinatal care
tw = log target perinatal hospital share
pnm = log perinatal mortality rate

Table A5.4 Tests for Autoregressive Unit Roots (Augmented Dickey-Fuller Test)

| | Dependent Variable | | | | |
|---|---|---|---|---|---|
| Regressor | w | x | rp | tw | pnm |
| constant | 0.0111 | 0.0328 | -0.1682 | -0.3029 | 0.2974 |
| | [2.164] | [0.058] | [-1.749] | [-2.237] | [0.876] |
| time trend | -0.0001 | -0.0006 | 0.0007 | -0.0043 | -0.0054 |
| | [-2.334] | [-0.180] | [0.768] | [-2.275] | [-1.534] |
| lagged dependent variable | 0.8913 | 1.0015 | 0.7789 | 0.8413 | 0.9286 |
| | [14.787] | [9.653] | [6.968] | [12.090] | [11.788] |
| first difference lagged dependent variable | 0.2874 | -0.0046 | 0.2737 | 0.3727 | -0.3896 |
| | [1.6843] | [-0.022] | [1.495] | [2.312] | [-2.271] |
| R-squared | 0.9157 | 0.9945 | 0.8304 | 0.9707 | 0.9918 |
| SER | 0.0027 | 0.0248 | 0.0321 | 0.048 | 0.0427 |
| Durbin-Watson | 1.94 | 1.95 | 1.92 | 1.99 | 1.87 |
| First order autocorrelation of residuals | -0.181 | 0.081 | -0.076 | -0.205 | 0.1012 |

Notes: The hypothesis of interest is whether the coefficient on the lagged dependent variable is significantly different from one. The values of this coefficient ranges from 0.7789 to 1.0015 and are consistent with the value of 0.90 that would be realised with a random walk process.

TABLE A5.5 VARIABLES USED IN PERINATAL HOSPITAL DEMAND MODEL

| | VARIABLE | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|
| YEAR | W | X | x | RP | rp | TW | tw | PNM | pnm |
| 1951 | 0.065 | 291.736 | 5.676 | 0.425 | -0.856 | 0.0961 | -2.342 | 48.3 | 3.877 |
| 1952 | 0.066 | 307.058 | 5.727 | 0.428 | -0.848 | 0.0991 | -2.312 | 47.3 | 3.857 |
| 1953 | 0.065 | 316.196 | 5.756 | 0.421 | -0.866 | 0.0975 | -2.328 | 43.6 | 3.775 |
| 1954 | 0.065 | 329.455 | 5.797 | 0.412 | -0.886 | 0.0976 | -2.327 | 45.5 | 3.818 |
| 1955 | 0.064 | 342.467 | 5.836 | 0.414 | -0.881 | 0.0959 | -2.344 | 43.8 | 3.780 |
| 1956 | 0.073 | 336.783 | 5.819 | 0.452 | -0.793 | 0.1119 | -2.190 | 42.5 | 3.750 |
| 1957 | 0.079 | 345.464 | 5.845 | 0.480 | -0.735 | 0.1215 | -2.108 | 42.8 | 3.757 |
| 1958 | 0.084 | 351.226 | 5.861 | 0.492 | -0.710 | 0.1271 | -2.063 | 41.1 | 3.716 |
| 1959 | 0.085 | 360.338 | 5.887 | 0.491 | -0.712 | 0.1272 | -2.062 | 41.1 | 3.716 |
| 1960 | 0.085 | 375.778 | 5.929 | 0.486 | -0.721 | 0.1116 | -2.193 | 39.6 | 3.679 |
| 1961 | 0.087 | 378.621 | 5.937 | 0.495 | -0.703 | 0.1125 | -2.185 | 38.3 | 3.645 |
| 1962 | 0.088 | 394.650 | 5.978 | 0.483 | -0.728 | 0.1124 | -2.186 | 37.5 | 3.624 |
| 1963 | 0.086 | 413.148 | 6.024 | 0.496 | -0.701 | 0.1096 | -2.211 | 35.6 | 3.572 |
| 1964 | 0.086 | 428.253 | 6.060 | 0.501 | -0.692 | 0.1065 | -2.240 | 34.0 | 3.526 |
| 1965 | 0.088 | 429.423 | 6.062 | 0.517 | -0.659 | 0.1067 | -2.238 | 33.5 | 3.512 |
| 1966 | 0.089 | 456.714 | 6.124 | 0.517 | -0.660 | 0.1026 | -2.277 | 31.2 | 3.440 |
| 1967 | 0.081 | 468.669 | 6.150 | 0.482 | -0.729 | 0.0935 | -2.370 | 29.4 | 3.381 |
| 1968 | 0.077 | 491.917 | 6.198 | 0.484 | -0.725 | 0.0877 | -2.434 | 27.9 | 3.329 |
| 1969 | 0.077 | 505.096 | 6.225 | 0.505 | -0.683 | 0.0834 | -2.484 | 27.3 | 3.307 |
| 1970 | 0.077 | 542.709 | 6.297 | 0.519 | -0.656 | 0.0810 | -2.513 | 26.5 | 3.277 |
| 1971 | 0.078 | 577.157 | 6.358 | 0.512 | -0.669 | 0.0797 | -2.529 | 26.4 | 3.273 |
| 1972 | 0.079 | 593.928 | 6.387 | 0.533 | -0.629 | 0.0740 | -2.604 | 25.5 | 3.239 |
| 1973 | 0.072 | 638.966 | 6.460 | 0.529 | -0.636 | 0.0656 | -2.724 | 24.1 | 3.182 |
| 1974 | 0.072 | 649.780 | 6.477 | 0.548 | -0.602 | 0.0642 | -2.746 | 24.6 | 3.203 |
| 1975 | 0.072 | 692.623 | 6.540 | 0.537 | -0.622 | 0.0624 | -2.774 | 22.8 | 3.127 |
| 1976 | 0.073 | 698.991 | 6.550 | 0.563 | -0.575 | 0.0621 | -2.779 | 19.8 | 2.986 |
| 1977 | 0.071 | 722.128 | 6.582 | 0.560 | -0.579 | 0.0597 | -2.818 | 20.0 | 2.996 |
| 1978 | 0.069 | 735.208 | 6.600 | 0.531 | -0.633 | 0.0623 | -2.776 | 16.8 | 2.821 |
| 1979 | 0.067 | 734.719 | 6.599 | 0.494 | -0.706 | 0.0641 | -2.747 | 15.5 | 2.741 |
| 1980 | 0.066 | 734.188 | 6.599 | 0.500 | -0.694 | 0.0649 | -2.735 | 14.4 | 2.667 |
| 1981 | 0.067 | 769.437 | 6.646 | 0.509 | -0.676 | 0.0664 | -2.712 | 13.1 | 2.573 |
| 1982 | 0.066 | 767.998 | 6.644 | 0.508 | -0.677 | 0.0635 | -2.757 | 12.9 | 2.557 |
| 1983 | 0.065 | 777.497 | 6.656 | 0.509 | -0.676 | 0.0604 | -2.807 | 11.6 | 2.451 |
| 1984 | 0.061 | 765.162 | 6.640 | 0.508 | -0.678 | 0.0590 | -2.830 | 12.1 | 2.493 |
| 1985 | 0.059 | 761.295 | 6.635 | 0.500 | -0.693 | 0.0595 | -2.822 | 10.9 | 2.389 |

NOTES: W = Share of hospital expenditure on perinatal care
X = Total hospital expenditure
RP = Relative price of perinatal and non-perinatal hospital care
TW = Target perinatal hospital share
PNM = Perinatal mortality rate

Full derivation of variables is given in text.
Lower case letters denote logs of corresponding capitals.

# CHAPTER 6 THE PRODUCTION OF PERINATAL HEALTH

## 6.1 INTRODUCTION

As the survey in Chapter 3 illustrated, a wide range of specifications have been used to directly estimate health production functions. One of the characteristic features of this literature is the virtual absence of any critical testing of alternative functional forms or models which could be used to summarise the relationship between inputs and health outputs. Burdened by the inertia of previous studies investigators have often simply assumed the existence of a particular function and an arbitrary set of regressors which is imposed on the data with little concern for the coherence and validity of their assumptions.

A second feature of the literature is its preoccupation with estimating static cross sectional production functions. Few studies have used time series data to examine the impact of changes over time in health care resources on mortality and other health outcomes. Despite the recognition of the impact of technical change and innovation on both the process and outcomes of health care this dynamic process is not usually captured in the traditional cross sectional approach. Consequently the investigation of the dynamics surrounding health production functions is in its infancy.

Both of these themes are addressed in the following health production function estimates. A variety of functional forms are tested against each other using the general to specific approach to estimation and testing consistent with that employed in Chapter 5. Section 6.2 discusses a menu of possible choices for functional forms. Alternative ways of augmenting the production function by introducing empirical measures of technical change are investigated in Section 6.3. The measurement and definition of output variables is presented in Section 6.4. Inputs into the production of perinatal health are discussed in Section 6.5. Results of testing and estimating a linear production function for perinatal mortality are presented in Sections 6.6 to 6.10. Section 6.11 estimates the cost per life year gained based on the regression results. Production function estimates for fetal and neonatal mortality are contained in the next two sections. Conclusions are presented in the final section.

## 6.2 FUNCTIONAL FORM

### 6.2.1 Linear Production Function

The simpliest and most popular form is the linear or ordinary n factor Leontief production function given by

$$Y = \Sigma_{i=1} a_i x_i \qquad (6.1)$$

This functional form imposes fixed factor proportions on the production process which assumes away the possibility of immediate input substitution. Output can be changed by varying the level of inputs but not the mix of inputs. Constant returns to scale are also implied. Doubling all inputs should lead to a doubling of output.

Marginal products may be positive, zero, or negative. Whether coefficients are fixed over time is an empirical issue that can be tested.

Although the fixed coefficient model does not possess the elegance and generality of more complicated functional forms it does have a number of desirable features when applied to health production functions. Perhaps one of the most attractive features of the linear production function is that it does not restrict the marginal products to be non-negative. This allows for the possibility that additional medical care may not have the expected positive impact on health outcomes. The marginal product of medical care on health may be zero or even negative. Several studies suggest that the marginal product of medical care on health is indeed close to zero. The history of perinatal care in the post-war period is also littered with dubious innovations of unproven effectiveness. The

notion of negative marginal products is often alleged in the perinatal field but rarely demonstrated with any degree of confidence.

One potentially less attractive feature of the linear production function is the impossibility of factor substitution. At the micro level the assumption of fixed factor proportions may be a legitimate characterisation of health care technology. The scope for factor substitution at the plant (hospital or ward) level may be severely limited by past decisions and the exercise of professional control over the process of providing health care. At the macro level, however, the possibility of input substitution may be a reality since over time it may be possible to re-allocate production amongst different units. For example, the staffing levels and bed/staff ratios of maternity hospitals and wards may vary over time. As small hospitals are gradually replaced by larger centralised units, this change in the mix of institutions will be accompanied by aggregate changes in factor proportions. This process of re-distributing births to larger more specialised maternity units has occurred in Scotland over the past two decades. The distribution of births and total production amongst different types of maternity hospital and wards thus provides scope for substitution at the aggregate level.

### 6.2.2 Generalised Leontief-Diewert Model

The simple model given by (6.1) can be extended using the generalised Leontief function introduced by Diewert (1971). This function takes the form

$$y = \Sigma_{i=1}\Sigma_{j=1} a_{ij} x_i^{\frac{1}{2}} x_j^{\frac{1}{2}} \qquad (6.2)$$

This general specification includes as a special case, the linear (fixed coefficient) model ($a_{ij} = 0$ for $i = j$). F-ratio tests are used to assess the validity of the restrictions imposed by the different specifications.

In Equation 6.2 the marginal product of an input is a function of the other inputs. Interaction between biological or population inputs (e.g. birthweight) and health care inputs is captured allowing for the possibility of differential impact of perinatal care as population determinants of perinatal mortality vary over time. Likewise, the marginal impact of changing population risks on perinatal mortality will depend on the availablity of perinatal health care inputs.

The elegant generality of the Leontief-Diewert function should be balanced against the econometric problems that can arise in practice. With even a small number of inputs, the number of estimated parameters grows rapidly.

The Leontief-Diewert's appetite for parameters is thus difficult to satisfy with the relatively short (and highly collinear) aggregate data series that most investigators use in time series studies of production functions. Although the Leontief-Diewert can be easily specified it is perhaps a bit naive to expect a sensible and robust estimate of such a complicated function when there are a relatively large number of inputs.

### 6.2.3 Translog Model

Another functional form recently used by Rosenzweig and Schultz (1982) to estimate infant health production functions is the Transcendental Logarithmic Function (Translog). Also known as the log-quadratic function (Sargan, 1971) the general form of this function is given by

$$y = \tfrac{1}{2}\Sigma_{i=1}\Sigma_{j=1}\beta_{ij}\, x_i\, x_j + \Sigma_{i=1}\beta_i\, x_i \qquad (6.3)$$

where all variables are expressed in logarithms.

The translog represents a second order approximation to any production function. The interaction and higher order terms allow for a general specification in which the elasticity of scale changes with factor proportions and with

the level of production. When the restriction ($\beta_{i,j} = 0$ for all i,j) is imposed, (6.3) collapses to the familiar Cobb-Douglas specification.

Like the Leontief-Diewert and most generalised production functions, the translog suffers from the disadvantage of a large number of parameters. Estimates of economic parameters such as marginal products and elasticities of substitution will depend upon functions of these parameters. The corresponding asymptotic standard errors for these economic parameters may not conform to a Gaussian distribution except in large samples (Sargan, 1971).

## 6.3 TECHNICAL CHANGE

### 6.3.1 Techological Change and Innovation in the Health Sector

Perinatal medicine has been characterised by a variety of process-innovations which have dramatically changed the medical management of pregnancy and childbirth during the post war period. However, as in other medical specialities, the effects of new techniques, procedures, or simply re-organisation of existing resources, on health and other outcomes are often unclear. Despite this uncertainty, rapid adoption and extensive diffusion of new

techniques has led to the present situation where novel untested innovations quickly become standard features of perinatal health services.

In contrast to the standard economic definition of a process-innovation; viz, "any adopted improvement in technique which reduces average costs per unit of output" (Blaug, 1963), the effect of many "new" techniques in perinatal care on either costs or output is usually unknown. Innovations may have positive (and negative) effects on both costs and output. The current controversy surrounding many perinatal innovations concerns precisely this point. Innovations may have little or no positive effect on output yet may actually increase, rather than decrease, costs. Indeed a substantial proportion of the increase in hospital costs and health service expenditure has been attributed to the introduction and widespread adoption of new medical techniques and procedures. Even when an innovation can be shown to lead to an increase in output, say measured in terms of improved survival, such an innovation may nevertheless increase average costs per unit of output. A good example of such an innovation in the perinatal field is the development and introduction of neonatal intensive care which is associated with an improvement in the survival of very low birthweight infants and an increase in the cost per unit of output, expressed in terms of cost per quality adjusted life year gained.

From the perspective of identifying sources of change in perinatal health Blaug's definition of process innovation should be restated as any change in technique which increases the effectiveness of medical interventions or programmes of care, where effectiveness is defined in terms of impact on mortality and other health outcomes. New techniques, whether innovative or not may (i) induce factor substitution resulting in changing capital-labour ratios depending on whether the innovation is labour or capital saving, (ii) raise (or lower) the marginal productivity of different factors of production, or, (iii) make some factors of production obsolete (e.g. the declining role of midwives may reflect an increasing preoccupation with techological obstetrics).

Technical change in perinatal care has undeniably transformed the care of women and their babies. What is important to recognise is that innovations in perinatal care can fail to conform to standard textbook definitions or economic interpretations. Although technical change and increases in knowledge may be responsible for some of the improvement in perinatal mortality, the individual and collective contribution of specific interventions remains largely unknown. Ironically, some innovations, despite their widespread diffusion throughout the perinatal care sector, may have simultaneously reduced output and increased costs.

### 6.3.2 Perinatal Care: The case for neutral technical progress

Like many types of health care, the process of perinatal care has changed dramatically over the past three decades. Innovations have been introduced, often without a clear understanding as to how they will influence, let alone improve, the productivity of labour and capital inputs employed in the perinatal sector.

Outside of the health arena, virtually all production functions estimated using time series data make some concession to measuring the impact of technical change on the production process. A familiar assumption in this literature is that technical change is autonomous, neutral and growing at a constant rate (Nadiri, 1970). If this assumption was adopted in the perinatal sector, technical change could, in principle, be easily measured by incorporating a time trend in any of the specifications of the production function outlined above.

This approach to accounting for technical change is indeed beguiling but is it appropriate for the perinatal sector where some degree of technical progress may be endogenous, non-neutral and growing at different rates? Innovations may develop both within and outwith the perinatal sector. Not all inputs may share in the fruits of technical

change. Some inputs, in fact, may be rendered obsolete as new techniques are introduced. Certain periods of time may be marked by rates of technical change which are greater (or less) than the long-run average rate.

The simple time trend may be a proxy for a more subtle process of change and innovation. From an econometric point of view the time trend approach can present problems when inputs are themselves increasing over time. This makes it difficult to distinguish the influence of changing technology from the impact due to differences in the scale of production.

### 6.3.3 Learning by doing?

One alternative to the time trend approach was suggested by Arrow (1962). Extending earlier studies which examined "learning curves" or "progress ratios" in production processes, Arrow formalised the notion of learning by doing whereby technical change is related to the sum of past activities or experience.

The learning by doing hypothesis can be presented as a relationship between input productivity and cumulated output (or investment). This formulation can be described as learning by doing or producing more. Applying this version of hypothesis to the perinatal sector requires a measure of cumulated experience. One proxy for doing and

learning more over time in the perinatal field is the lagged perinatal mortality rate. The key assumption is that the stock of learning or technological knowledge is proportional to immediate past output. Improvements in perinatal survival represent the accumulation and application of knowledge. No increase in the stock of knowledge is assumed to occur in periods characterised by a static perinatal mortality rate. Corresponding lagged dependent variables assume the role of technological proxies in the production functions for stillbirth rates and neonatal mortality rates.

The production function with a lagged dependent variable resembles a partial adjustment model. The perinatal mortality rate evolves over time as a function of previous mortality rates and the current period values of inputs used in the production process. When the coefficient on the lagged mortality rate is one, no technical change is assumed to occur and the perinatal mortality rate never changes. At the other extreme when the coefficient is zero, any change in perinatal mortality due to changes in inputs occurs immediately. The impact of technical change on perinatal mortality over time is given by the mean lag or, alternatively, the proportion of change in the dependent variable arising from technical change which occurs after n time periods.

Arrow's hypothesis has been interpreted as learning not by doing more but by doing it longer (Fellner, 1969). As in the simple time trend model, productivity is dependent on time alone. This specification assumes that experience is acquired with the passage of time, irrespective of the change in perinatal survival. Epochs in which no improvement in perinatal survival occurred would be accorded the same weight as those in which survival had increased significantly.

## 6.4 MEASUREMENT AND DEFINITION OF OUTPUT

### 6.4.1 Perinatal Mortality Rates

A host of possible candidates for output measures in perinatal mortality production functions were surveyed in Chapter 4. Several different specifications of mortality rates are used to estimate the production functions. The first is the perinatal mortality rate from all causes in all birthweight groups defined as the number of stillbirths and neonatal deaths per 1000 total (live and still) births.

Perinatal hospital services may also influence birthweight specific perinatal mortality in different ways over time. The introduction and deployment in the 1970's of intensive

care for the most vulnerable newborns is just one example where the impact of health care expenditure on perinatal survival may vary in different birthweight groups.

This hypothesis is tested by estimating the production functions using a time series of birthweight specific mortality rates in Scotland covering the period 1956-1985. Although weight-specific perinatal mortality can be disaggregated in 500g groups for shorter time series, the analysis is confined to mortality rates in three mutually exclusive weight categories: very low birthweight (<1.5 kg) intermediate low birthweight (1.5-2.5kg) and "normal" birthweight (>2.5 kg). The weight-specific perinatal mortality rates include all fetal deaths (of 28 or more weeks of gestation) and infant deaths occuring during the first week following birth. The derivation of the series is described in more detail in Chapter 4.

### 6.4.2 Years of Potential Life Gained

Improvements in perinatal health and mortality will extend well beyond the perinatal period. The concept of years of potential life gained (YPLG) provides a way of measuring these longer run changes in health attributable to a reduction in perinatal mortality. The product of the differenced annual series of perinatal mortality rates (per

1000 total births) and the total number of births gives an estimate of the incremental lives gained as a result of changes in the risk of perinatal mortality:

$$LG = PMR * [TB *.001] \qquad (6.4)$$

where LG is incremental lives gained, PMR is the annual change in perinatal mortality and TB is the number of total births in year t.

Since the lives gained will depend on the size of the newborn population LG can be re-stated per 1000 births to adjust for population size. However, since LG /[TB *.001] is equivalent to the differenced perinatal mortality rates, a production function estimated using differenced data will allow a direct estimate of the lives gained due to investment in the perinatal hospital sector. Alternatively the lives gained (or lost) can be easily derived from the estimated elasticity of perinatal mortality with respect to a selected perinatal hospital input. Given lives gained the YPLG due to improvements in perinatal mortality can be estimated using estimates of life expectancy at age 0.

### 6.4.3 Fetal Mortality Rate

Although the perinatal mortality rate is the primary output measure the production functions are also estimated using the fetal mortality rate as the dependent variable. The standard British definition of fetal mortality is employed, viz, the number of fetal deaths (of 28 or more weeks of gestation) per 1000 total births.

### 6.4.4 Neonatal Mortality Rate

The neonatal mortality rate is also used to complement the fetal mortality regressions. To capture any differential effects over the neonatal period the overall neonatal mortality rate is also dis-aggregated into three age-specific rates (< 1 day, 1-7 days and 7-27 days).

The impact of investment in perinatal hospital services on neonatal mortality may vary across different categories of infants defined according to their inherent risk of mortality. To some extent this is captured by the age-specific production function estimates since the distribution of mortality risks and the characteristics of the neonatal population varies throughout the neonatal period. One way of addressing this issue is to estimate production functions using cause-specific neonatal mortality rates based on the international classification of registered causes of death presented in Chapter 4. Separate produc-

tion functions are presented for neonatal mortality due to (i) congenital malformations (ii) prematurity (iii) asphyxia and (iv) those causes not listed in (i) to (iii).

## 6.5 Inputs

Conventionally, in direct estimates of production functions, inputs are specified in physical terms or in expenditure (cost) terms defined as the product of input prices and input quantities. Both approaches are used in the estimation of perinatal mortality production functions.

### 6.5.1 Population Inputs

The attributes of the newborn population constitute an important set of inputs into the production process. Newborn population size can be introduced into the production function using a separate variable or interactively with the other inputs. In the equations reported below, all hospital input quantity variables are expressed per 1000 total births. Hospital expenditure is expressed per birth.

The quality of the newborn population is summarised using the incidence of low birthweight (LBW) per 1000 total births. Changes in living standards, nutrition, smoking, fertility and other household characterstics are encapsulated in this variable. In the birthweight specific

production functions the birthweight distribution is further broken down into very low birthweight (VLBW), intermediate low birthweight (ILBW) and normal birthweight (NBW).

### 6.5.2 Hospital Input Quantities

Expressing hospital inputs in terms of quantity measures allows a dis-aggregate approach which permits investigation of how different classes of inputs may influence perinatal mortality. Depending on the precise formulation of the production function it also enables the inputs to interact with each other in the process of producing perinatal health.

#### Labour Inputs

Ideally, a precise measure of the flow of services from all labour inputs used in the process of providing perinatal care would be included in the production function. In most production function studies the flow of labour services is measured by man hours which in turn are often assumed to be directly proportional to the stock of labour. This approach was adopted for the following classes of labour.

The first labour variable is the number of nursing staff devoted to maternity care. Nursing accounts for the majority of expenditure on perinatal hospital services.

Data on numbers of whole (full) time equivalent maternity nursing staff were extracted from Scottish Health Statistics and unpublished tables supplied by the Information Services Division of the Scottish National Health Service. Whole time equivalent figures, however, overstate the flow of nursing services since the numbers of hours supplied per week per nurse have steadily declined during the post-war period. For example, standard weekly hours of nursing staff have declined from 48 in 1950 to 37.5 in 1985. Total full time equivalent numbers were adjusted to reflect changes in standard weekly hours and holiday entitlements using the series on nursing hours presented in Smail and Gray (1982).

The second labour group is the stock of obstetricians and gynecologists measured by the number of whole time equivalent medical staff in this specialty per 1000 births. The flow of labour services provided by this labour group is assumed to be proportional to the stock of labour. No consistent time series data exists on standard hours supplied by NHS hospital medical staff, let alone a break-down by specialty. However, sporadic surveys of the workload of hospital doctors suggest that average hours supplied has not varied substantially over time in the NHS.

Perinatal care, however, is not the sole province of the obstetrician. Paediatrics has long constituted an important class of labour inputs into the production of neona-

tal health. One candidate for measuring this category of medical manpower is the number of whole time equivalent doctors in the specialty of medical paediatrics. However, unlike obstetricians the flow of paediatric labour services to perinatal care can not be proxied by the stock of pediatricians. The flow of services from paediatricians is distributed over a client group that ranges in age from the neonatal period to late adolescence. No data is available which permits a partitioning of the medical paediatric stock by hours supplied to different age groups.

In common with other countries in the early 1970s Scotland witnessed the introduction and deployment of intensive neonatal care. Although there are now several full time medical posts in neonatology in Scotland the whole time equivalent complement of staff in this paediatric subspecialty would require information on the hours supplied by paediatricians who are not fully engaged in neonataology.

An alternative measure of the specialised paediatric resources devoted to neonatal care is given by a dummy variable (INTENSE) which corresponds to the epoch characterised by the availability of neonatal intensive care (1975-85).

**Capital Input**

Capital constitutes another input into the production of perinatal health. Ideally the production function requires a measure of the flow of capital services. Conventionally, in the health sector, the flow of capital services can only be indirectly measured by proxies such as capital expenditure. An alternative measure is the net accumulated stock of capital expenditure as embodied in the bed stock or complement. Available staffed beds provides a more accurate measure of capital inputs since it reflects the maximum stock or bed capacity that can be utilised in the production process.

The flow of capital services from the bed stock, however, depends on their utilisation. For example, a fixed stock of beds could be associated with an increase in service flow if more cases were treated per bed. A further adjustment for the utilisation of available bed capacity is required. Changes in the intensity of bed use can be measured by the case-flow rate (case/bed ratio). The case-flow rate (also known as the throughput or turnover rate) is defined as the number of hospital births per staffed bed per year. An index of relative case-flow rates is used to correct the bed stock series.

**Input Proportions**

The increasing labour intensity of perinatal hospital production can be measured by the change in input proportions (Figure 6.1 - 6.3). Over the period 1956-85 the mean nurse/bed ratio was 1.00. The ratio of nurses to beds increased from a low of 0.72 in 1956 to a high of 1.25 in 1981 and then decreased to around 1.10 in the mid-1980s. The overall increase in the nurse/bed ratio between 1956 to 1985 was 53 per cent. The ratio of nurses to beds did not increase at a uniform rate over time. Following a rapid increase in the late 1950s the ratio remained static around the long run mean level of one nurse per bed between 1965 to 1975.

The mean obstetrician/bed ratio was 0.08 (range 0.05 to 0.13). This average masks a steady increase which saw the number of obstetricians per bed increase by 160 per cent over three decades. The impact of these differential rates of change in labour/bed ratios can be seen in the declining nurse/obstetrician ratio. From a high of 16.5 nurses per obstetrician in the late 1950's the nurse/obstetrician ratio gradually declined to its present level of 8.7 in 1985. The principal change in input proportions is one of increasing labour intensity with a change in the mix of labour towards obstetricians and away from nurses.

### 6.5.3 Aggregate Expenditure on Perinatal Hospital Care

An alternative specification of perinatal inputs is also considered. Following Hadley (1982) the production functions for perinatal mortality are also specified using expenditure data. Total real expenditure on perinatal hospital services was calculated over the period 1956-1985 using the data sources and methodology outlined in Chapter 5. Total expenditure can be expressed as a ratio using a variety of denominators. Three different expenditure measures are considered. The first is defined as real expenditure on perinatal hospital services per birth. The second measure adjusts for the proportion of births occuring in hospital and is defined as real expenditure per hospital birth. A third option is expenditure per staffed perinatal hospital bed.

The expenditure per bed series was rejected since it fails to make any allowance for changes in the size of the obstetric population. This measure of expenditure is also directly influenced by the supply of beds and their utilisation. The per-capita series gradually drift closer together as domicillary deliveries become less prominent and eventually converge in the early 1970's. Given their high correlation the two series are virtually interchangable as per-capita measures of varying levels of expenditure on perinatal hospital inputs. To ensure consistency

with the measurement of the output (dependent) variables the series expressed per total birth was the preferred measure.

A list of variable names, definitions, means and standard deviations for all output (mortality) variables is presented in Table 6.1. Corresponding information for the input variables is contained in Table 6.2. The data series are contained in Appendix Tables A6.1 and A6.2.

## 6.6 Linear Production Functions

### 6.6.1 Perinatal Mortality

The first stage in the testing process focussed on the set of input variables to be included in the linear production function. The most general model included two labour variables (OBS and NUR), one capital variable (BED), a measure of the availability of neonatal intensive care (INTENSE) and one population variable (LBW). With the exception of the dummy variable (INTENSE), all variables are expressed per 1000 total births. The competing model excluded the set of health service inputs. The relevant F-test of 1.607 (5 per cent critical value = 2.80) suggested that the joint influence of the health service variables on perinatal mortality from all causes was negligible.

OLS estimates of the simple linear version of the perinatal mortality production function over the period 1956 - 1985 are given by

$$PMR = \underset{[4.898]}{6.2504} - \underset{[-27.976]}{1.0625TREND} + \underset{[3.488]}{0.2370LBW} \qquad (6.5)$$

R-squared = 0.9940 SER = 0.8339 F(2,27) = 2222.76
DW = 1.657 RSS = 18.7777

SER = standard deviation of the residuals
RSS = residual sum of squares
DW = Durbin-Watson statistic
[ ] = t-statistics calculated using heteroscedastic consistent standard errors (MacKinnon and White, 1985).

The signs on the coefficients are in accordance with expectations. The important influence of the simple proxy for technical change is illustrated by the negative time trend. The incidence of low birthweight births is positively related to the perinatal mortality rate. A 10 per cent increase in the incidence of low birth weight births is associated with a 6.3 per cent increase in the perinatal mortality rate. The elasticity of perinatal mortality with respect to the time trend is -0.6185. At the sample mean (which coincides with 1972) the annual rate of change in perinatal mortality attributable to a unit change in the time variable alone was -4.0 per cent.

The linear production function was also re-estimated using a lagged dependent variable in place of the time trend. F-tests again supported the exclusion of the health service inputs. OLS estimates give

$$PMR = \underset{[1.152]}{-6.8932} + \underset{[23.565]}{0.9549PNM} + \underset{[1.013]}{0.0992LBW} \qquad (6.6)$$

The estimated elasticity of perinatal mortality with respect to the incidence of low birthweight is 0.265. The average annual change in perinatal mortality attributed to technical change as proxied by the lagged dependent variable is -4.5 per cent.

### 6.6.2 Birthweight Specific Perinatal Mortality

Separate regressions were estimated for perinatal mortality rates in different birthweight groups. In the birthweight specific equations which included a time trend F-tests generally supported the specification which included the four health service variables (Table 6.3). OLS estimates of the general models for three different weight groups are presented in Table 6.4. The signs of the OBS and NUR variables are generally consistent with the hypothesis that these labour inputs exert a beneficial impact on perinatal mortality. The dummy variable for the availability of neonatal intensive care is also negatively related to weight specific perinatal mortality.

Decreases in the bed stock, however, are associated with improvements in perinatal mortality, particularly for low birthweight births. At first sight this may appear counter-intuitive but there are a number of possible ex-

planations for this result. Over time the mix of obstetric beds has shifted from small maternity institutions and wards towards larger and newer specialist units. If there is a positive relationship between the vintage of the capital stock and the marginal product of the capital stock then a decline in the stock would be associated with an improvement in perinatal mortality. An increase in the (average) scale of maternity units accompanied by a reduction in the total capital stock could also lead to an improvement in perinatal outcomes in the infants who face the greatest risks of mortality.

Shifts in the birthweight distribution have no significant impact on birthweight specific perinatal mortality rates. The sign on the respective birthweight variables is negative in all equations suggesting that an increase in the incidence of very low birthweight births is associated with a decrease in the perinatal mortality rate amongst very low birthweight births. This association between the incidence of birthweight and weight-specific mortality has been observed in other populations. For example, multiple births have lower birthweights and lower weight-specific mortality. American studies of race-specific mortality rates reveal that blacks have lower weight specific mortality but a higher incidence of low birthweight infants.

When the equations are re-estimated with a lagged dependent variable in the place of the linear time trend the joint influence of the health service variables emerges in a consistent fashion. Although the dummy variable for intensive neonatal care is insignificant the stock of obstetricians and nurses is negatively and significantly associated with mortality in births weighing less than 2.5 kg. Obstetricians are also negatively associated with perinatal mortality in births weighing over 2.5 kg. Nurses do not appear to be a significant determinant of perinatal mortality amongst normal weight births. Once again the incidence of specific birthweight categories has no significant impact on weight specific mortality rates. The bed stock is positively and significantly related to the mortality rates in the two weight groups below 2.5 kg. This is further support for the hypothesis that more recent bed vintages represent more effective capital inputs into the production of perinatal health in low birthweight infants.

### 6.6.3 A Dynamic Autoregressive Specification

The inclusion of the lagged dependent variable as a measure of technical change introduces a dynamic element into the perinatal mortality equations. This equation can be extended to include current and lagged inputs in a general autoregressive-distributed lag formulation of the linear production function. The results of estimating

this general formulation and solving for the long-run coefficients are given in Table 6.5. The Wald test for each equation easily rejects the null hypothesis that all long run coefficients are zero.

Table 6.6 presents the elasticities calculated using the long run coefficients. The impact of obstetricians on perinatal mortality is similar across birthweight groups. A ten per cent increase in obstetricians is associated with a seven per cent decrease in the perinatal mortality rate in very low birthweight births. The elasticity of perinatal mortality with respect to obstetricians increases slightly with increasing birthweight. For births weighing more than 1.5kg the long run elasticity is -0.95. A similar pattern of elasticities emerged for nurses in the birthweight groups < 2.5 kg. The elasticity for nurses is about 50 per cent greater, compared to that for obstetricians, in the normal weight group (> 2.5 kg). The elasticities for beds suggest that a ten per cent reduction in the stock of beds is associated with a 12 to 13 per cent reduction in weight-specific perinatal mortality rates.

As was the case in Chapter 5, the host of variables used to estimate the production function are not stationary. As such the caveats concerning the validity of statistical tests and inference in general are applicable, particularly in specifications involving non-stationary vari-

ables measured in levels. By including the current and lagged variables, and the lagged dependent variable, in the general specification a dynamic element is introduced into the production function which addresses this problem. Rival restricted models of this general specification could be sequentially tested as a way of exploring different dynamic specifications possibly involving lagged inputs and the first differences of input levels.

However, at an intuitive level, a production function dynamically linking inputs and perinatal mortality can be challenged. This is particularly true for specifications involving lagged inputs which are unlikely to play an important role in influencing current levels of mortality. The production process is more immediate: current output depends on current inputs not on past levels of inputs. If the learning by doing hypothesis underpinning technical change is accepted, current mortality may depend on past mortality. Thus, the choice of rival models can be narrowed down to a static regression versus the learning by doing (partial adjustment) model. Although more complex dynamic models could be specified the focus throughout this chapter is on less ambitous and arguably more realistic specifications of the production process. This trade-off between correct dynamic specification, the time series properties of the data and sensible specifications of production is explicitly acknowledged.

## 6.7 Generalised Leontief-Diewert Production Functions

The linear production model can be extended to the general case of the Leontief-Diewert by including an additional set of input interaction terms. F-tests used to assess the restrictions imposed by excluding these terms from the production function are reported in Table 6.7. The restrictions inherent in the linear production model are acceptable. One important consideration here is that the estimated Leontief-Diewert equations display the classic symptoms of multicollinearity making it difficult to determine the effect of inputs on perinatal mortality with any acceptable degree of precision. Due to the lack of sufficient sample information and large sampling variances for estimators of the unknown parameters inputs may be inappropriately dropped from the equation even if they have an effect on output.

A number of tests have been suggested for diagnosing multicollinearity. One of the more obvious symptoms is a high $R^2$ in conjunction with low partial correlations between the explantory variables and the dependent variable. In particular if the partial correlations fall when additional variables are added to the equation multicollinearity is indeed suspect.

Table 6.8 reports the partial correlation coefficients and R-squared for the Leontief and Leontief-Diewert specifications of the weight-specific production functions. As the number of input terms increases many of the partial correlations fall thus suggesting that multicollinearity does present a problem in the Leontief-Diewert specification. Given these test results and the problem of multicollinearity the generalised linear specification was rejected in favour of the more restrictive Leontief production function.

## 6.8 Testing linear versus log-linear production functions

### 6.8.1 Choosing Competing Specifications

The generalised Leontief-Diewert production function can be specified in log-linear form as

$$\log y = a_0 + \Sigma_{i=1}\Sigma_{j=1}\beta_{ij}\log((x_i + x_j)/2)) \qquad (6.13)$$

When $[\beta_{ij}]$ is a diagonal matrix, 6.13 reduces to the familiar Cobb-Douglas production function. Although the log-linear specification was used by Hadley (1982) in his cross-sectional study of mortality rates no attempt was made to test this specification against the linear production function. On logical grounds the inherent assumptions of the Cobb-Douglas specification are suspect. Smooth continuous substitution between obstetricians, nurses and

beds is one such assumption. Restricting the elasticity of substution between pairs of inputs to -1 by definition is another log-linear assumption that can be challenged in the case of perinatal hospital care.

Although these a priori arguments are convincing reasons not to adopt the log-linear model, it is possible to discriminate between the linear and log-linear specifications using statistical criteria. However, testing the alternative specifications requires more than simply comparing the R-squares from the linear and log-linear models since the variance of the log transformed dependent variable will be less than that of the dependent variable in the linear model. A variety of tests have been proposed to inform the choice between these rival models (Andrews, 1971; Godfrey and Wickens, 1981; Bera and McAleer, 1983; Davidson and Mackinnon, 1985).

### 6.8.2 The Pe Test

Given this menu of tests, the perinatal mortality production function models were investigated using the extended P (Pe)test suggested by Mackinnon, White and Davidson (1983) which was chosen on the grounds of simplicity and computational ease. The Pe test consists of two steps. The first involves estimating both specifications,

$$\log PNM = \beta_0 + \Sigma_{i=1} \beta_i \log x_i \qquad (6.14)$$

$$PNM = \delta_0 + \Sigma_{i=1} \delta_i x_i \qquad (6.15)$$

retaining the fitted values of each equation (logPNM and PNM) and calculating the transformed fitted values exp(logPNM) and logPNM. The second step estimates the artificial regressions

$$logPNM = \beta_0 + \Sigma_{i=1} \beta_i \log x_i + \theta_0 [PNM - \exp(logPNM)] \qquad (6.16)$$

$$PNM = \delta_0 + \Sigma_{i=1} \delta_i \log x_i + \theta_1 [logPNM - logPNM] \qquad (6.17)$$

followed by a test of $\theta_0 = 0$ and $\theta_1 = 0$ using t-tests. If the hypothesis that $\theta_0 = 0$ is accepted then the log-linear model is selected. If the hypothesis that $\theta_1 = 0$ is accepted than the linear model is opted for. If both hypotheses are accepted or rejected no clear cut choice can be made on statistical grounds. In the case where both hypotheses are rejected the inadequacy of the linear and log-linear models provides a strong incentive to investigate alternative functional forms as well as additional tests for omitted variables or other possible sources of model misspecification.

The results of the Pe test for birthweight specific perinatal mortality production functions are presented in Table 6.9. The linear specification is accepted for the

VLBW and ILBW regressions. For NBW regressions the linear specification was rejected. The rejection of the linear specification, however, should not be interpreted as evidence "in favour" of the log-linear specification (Davidson and Mackinnon, 1985). It merely means that the linear specification is unlikely to be correct not that the log-linear model is correct!

## 6.9 Expenditure on perinatal hospital care

An alternative specification of the perinatal mortality production function was investigated using expenditure on perinatal hospital care as a summary measure of hospital inputs. The impact of expenditure on perinatal hospital care on perinatal mortality (PNM) was first estimated using the Leontief-Diewert model. The F-tests rejected the Leontief-Diewert model as well as the inclusion of the expenditure variable in favour of the simple linear model given by

$$\underset{}{PMR} = \underset{[4.898]}{26.2505} - \underset{[27.976]}{1.0625TREND} + \underset{[3.488]}{0.2370LBW} \qquad (6.18)$$

R-squared = 0.9940 SER = 0.8339 F(2,27) = 222.76

DW = 1.657 RSS = 18.7777.

This simple equation which is identical to equation 6.5 reported above, did not provide a satisfactory summary of the influence of expenditure on perinatal mortality when separate regressions were estimated for weight-specific mortality. Table 6.10 presents the F-tests used to discriminate between different specifications of the weight-specific equations. Corresponding OLS estimates are presented in Table 6.11. The distribution of birthweight has no significant effect on weight-specific mortality rates. Once again the most important variables are the proxies for technical change as measured by the time trend or the lagged dependent variable. The dummy variable for intensive care enters most equations with a negative sign but is only significant in the case of births weighing between 1.5 and 2.5kg.

The negative marginal product for expenditure per birth in the intermediate low birthweight group and normal birthweight group is consistent with the hypothesis that increases in expenditure are associated with improvements in perinatal mortality (Table 6.12). This association is not, however, confirmed for births weighing less than 1.5kg. The estimated marginal product derived from the simple Leontief equations for normal weight births are very similar to those based on the Leontief-Diewert equations which allow for input interaction. The elasticity of perinatal mortality with respect to expenditure per birth is greatest for normal weight births. A ten per cent in-

crease in expenditure per birth is associated with a 4 to 5 per cent decrease in the perinatal mortality rate in births weighing more than 2.5kg. A similar increase in expenditure has a negligible impact on mortality in lower weight births (1.5 - 2.5kg).

## 6.10 Parameter Stability and Forecasting Performance

The traditional assumption of fixed coefficients in the linear regression model can be challenged within the context of perinatal mortality production functions. The impact of hospital inputs and expenditure on perinatal mortality may vary over time. After some point improvements in perinatal mortality may usher in a period of declining marginal products. Varying coefficients over time could provide support for the impact of technical change on the marginal productivity of inputs which is not fully captured by the time trend or lagged dependent variable. Coefficient "regimes" may also emerge when the production function is estimated using different sample periods, particularly if the estimated equation is unstable.

The stability of the weight-specific production functions was examined by re-estimated the equations using recursive least squares (RLS) over the period 1956-1985, retaining 5 observations for forecasts. The RLS estimates are first presented for the Leontief-Diewert model with birthweight, hospital expenditure, a birthweight-expenditure interac-

tion term, a constant term and a time trend included as regressors. This specification was chosen in preference to using input quantities as regressors given the relatively small number of observations used to estimate the intial coefficient estimates using the RLS method. The dummy variable for intensive neonatal care was also dropped from all equations to ensure a non-singular design matrix of input variables since the RLS coefficients are initialised using the observations for 1956-1971 a period where INTENSE was by definition always equal to zero. Dropping the intensive care variable from the production functions does, however, introduce the possibility of omitted variable bias in the RLS estimates.

**Time trend equations**

Table 6.13 presents the RLS estimates over the period 1956 - 1980. The estimated coefficients and their 95 per cent confidence intervals for the VLBW perinatal mortality production function are graphed for 1970 to 1980 in Figures 6.4 to 6.6. These graphs alongwith the Chow statistic (Figure 6.7) demonstrate that the coefficients are stable and increasingly different from zero over time. For the ILBW production function the coefficients begin to drift towards zero particularly after 1975 (Figures 6.8 to 6.10). Although the coefficients still differ significantly from zero a significant shift in the coefficients occurs around 1977 (Figure 6.11). The RLS es-

timates for mortality in normal weight births support the hypothesis of statistically stable coefficients, although the sign and significance of the coefficients is far from convincing (Figures 6.12 to 6.15).

The forecasting performance of the time trend equations is less than satisfactory. Over the forecast period of 1981 to 1985 the gap between the observed and forecast perinatal mortality rates widens for all birthweight groups (Figures 6.16 to 6.18). The best forecasting performance is recorded for the very low birthweight equation where the average absolute difference between forecasts and observed mortality rates is 36 deaths per 1000 live births or less than 10 per cent of the actual perinatal mortality rate in this weight group. The deterioration in forecasts is greatest for the normal weight births. The forecasts consistently overestimate the actual fall in perinatal mortality by about 50 per cent.

Learning by Doing Equations

One possible reason for the poor forecasting performance of the above equations is the prominence of the linear time trend. As can be seen by the graphs of actual and fitted mortality rates (Figures 6.22 to 6.24) the forecast rates march steadily downwards alongwith the passage of time. This optimistic expectation, inherent in using

the time trend as a proxy for technical change, obviously was not realized throughout the first half of the 1980's in all birthweight groups.

Re-estimating the equations using the lagged dependent variable in place of the time trend improves the forecasting performance. The RLS estimates of the Leontief partial adjustment equation are given in Table 6.14. The Leontief-Diewert specification was also estimated and found to give virtually identical forecasts to the simple Leontief model with the lagged mortality rate included as a regressor. Graphs of forecast and actual mortality rates are presented in Figures 6.19 to 6.21. The mean absolute forecasting error is 10.6 per cent for perinatal mortality in VLBW births compared to 10 per cent in the time trend equation (Table 6.15). In the heavier weight groups the partial adjustment equation with the lagged dependent variable is superior. The mean absolute forecasting error is about 50 per cent less in the ILBW equation. The forecasting performance in the normal weight group is nearly six times better in the partial adjustment equation compared to that recorded for the time trend equation. The improvment in forecasting performance can also be seen in the graphs of actual and fitted values (Figures 6.25 to 6.27). Similar results were obtained when the equation was re-estimated using a general autoregressive distributed lag model.

The stablity of the estimated parameters of the partial adjustment equation was examined both visually and statistically using the Chow test. No structural shifts occurred in the estimated coefficients. The parameters also remained constant over the forecast period.

## 6.11 Cost per Life Year Gained

The RLS estimates of the partial adjustment production function suggest that the elasticity of perinatal mortality with respect to expenditure per birth ranges from -0.443 in VLBW births to -1.005 in NBW births. Applying these elasticities to the mean number of births and the distribution of birthweight calculated over the sample period 1957 - 1985 suggests that a ten per cent increase in expenditure per birth would result in 31 fewer VLBW perinatal deaths, 43 fewer ILBW perinatal deaths and 95 fewer perinatal deaths in normal weight births. A 10 per cent increase in expenditure per birth would mean an increase of just over £4.169m in 1980 prices (£5.866m in 1985 prices).

Table 6.16 presents the incremental cost per year of potential life gained in the three birthweight groups. Assuming that the extra expenditure is spent exclusively on births in the specific weight category and a life expectancy of 70 years the incremental cost per (undiscounted) life year gained ranges from £1897 in the VLBW group to

£686 in the NBW category. The incremental cost per discounted life year gained in VLBW births is £6866 and £13291 for discount rates of 0.05 and 0.10, respectively. Similar figures for ILBW births are £4979 and £9639. The cost per discounted life year gained in normal weight births is about one third below that for very low birthweight births. The corresponding figures for 1985 prices are also given in Table 6.16.

These figures should be qualified by two points. First, no adjustment is made for any differences which may exist in the quality of life years gained by the different weight cohorts. Although opinion remains divided many investigators have argued that enthusiasm for the increased survival of the very low birthweight infant should be tempered by the array of long term health problems which many of these survivors encounter. Compared to heavier babies, the very low birthweight baby faces a greater likelihood of developmental handicap which, according to the preferences of parents, diminish the quality of life (Boyle et al, 1983). Adjusting life years gained for their expected quality will tend to widen the differences in incremental cost per life year gained or quality adjusted life year gained. Although the incremental cost per unadjusted life year gained will be very similar to the quality adjusted figure in the normal weight infants, such an assumption is questionable in the lower weight

groups. The unadjusted differences reported above will thus tend to under estimate the true difference in cost per quality adjusted life year gained.

A second point to consider is the single equation assumption that the increase in perinatal hospital expenditure is restricted to specific birthweight groups. An increase in expenditure per birth (and any beneficial health consequences) may be distributed across all births. Thus summing across the birthweight specific equations, 169 fewer perinatal deaths are associated with one 10 per cent increase in expenditure. The overall figure of £24612 per life gained or £352 per undiscounted life year gained is very much lower compared to the weight-specific estimates when expenditure does not spillover birthweight groups. The incremental cost per discounted life year gained ranges from £1272 (5 per cent) to £2464 (10 per cent).

## 6.12 Fetal Mortality

The perinatal mortality rate was disaggregated into its two primary components: fetal mortality and neonatal mortality. Linear fetal mortality production functions were estimated using the same general to specific testing procedure. When the time trend was included in the equation, F-tests supported the inclusion of the health service variables. However, when the time trend was replaced by

the lagged fetal mortality rate, the health service inputs had no apparent significant impact on the fetal mortality rate. The respective equations are given by

$$\begin{array}{lll} FMR = 12.4876 - 0.5731TREND + 0.1656LBW + \\ \quad\quad [4.339] \quad [10.251] \quad\quad [4.714] \\ \\ \quad\quad 0.4651OBS - 0.1214NUR \\ \quad\quad [0.791] \quad\quad [2.728] \end{array} \qquad (6.25)$$

R-squared = 0.9969 SER = 0.3743 F = 1536.01 DW = 1.835

$$\begin{array}{lll} FMR = -6.3789 + 0.9156FMR + 0.0980LBW \\ \quad\quad [2.428] \quad [30.561] \quad\quad [2.291] \end{array} \qquad (6.26)$$

R-squared = 0.9944 SER = 0.4728 F= 2399.98 DW = 2.679

The estimated elasticity of fetal mortality with respect to the incidence of low birthweight births is 0.850 and 0.504 depending on the respective LBW coefficients in equation 6.25 and 6.26. A ten per cent increase in the number of nurses per 1000 births is associated with a 3 per cent decrease in the fetal mortality rate. Obstetricians have very little impact on the fetal mortality rate (elasticity = 0.096). The stock of beds exerts a positive, though insignificant, influence on fetal mortality: a ten per cent reduction in the stock of beds is associated with a one per cent improvement in fetal mortality.

## 6.13 Neonatal Mortality

### 6.13.1 All Causes

Unlike the fetal mortality production function, F-tests supported the inclusion of the health service inputs in both specifications of the linear neonatal mortality production function. OLS estimates of the equation controlling for technical change using a time trend are given by

$$\begin{aligned} NMR = {} & 15.6720 - 0.4538TREND + 0.0329LBW - 0.4030OBS - \\ & [2.680] \quad [4.529] \qquad [0.453] \qquad [0.364] \\ & 0.0134NUR + 0.1118BED - 0.6289INTENSE \qquad (6.27) \\ & [0.137] \qquad [1.051] \qquad [1.120] \end{aligned}$$

R-squared = 0.9850 SER = 0.59251 F = 266.67 DW = 2.067

Although the health service inputs are jointly significant their individual effects are not very impressive. The signs on OBS, NUR and INTENSE are in accordance with the hypothesis that these factors are negatively related to the neonatal mortality rate. The positive BED coefficient again suggests that a decrease in the bed stock is associated with an improvement in neonatal mortality. A further equation was estimated replacing the time trend with the neonatal mortality rate lagged one period. OLS estimates over the period 1957 to 1985 are given by

$$\begin{aligned} NMR = {} & 6.7660 + 0.5285NMR + 0.0082LBW - 1.9317OBS - \\ & [1.255] \quad [3.016] \qquad [0.098] \qquad [1.990] \\ & 0.1237NUR + 0.2475BED - 0.3327INTENSE \qquad (6.28) \\ & [1.562] \qquad [2.254] \qquad [0.491] \end{aligned}$$

R-squared = 0.9818 SER = 0.6723 F = 206.28 DW = 2.880

Once again the signs on the labour variables are both negative, suggesting that an increase in both obstetricians and nurses per 1000 births is associated with a reduction in the neonatal mortality rate. The negative coefficient on the dummy variable for intensive neonatal care is not significantly different from zero. The elasticities for OBS, NUR and BED are -0.432, -0.339 and 0.669 respectively. One implication of these results is that, from the perspective of reducing the neonatal mortality rate, a ten per cent reduction in the bed stock would be as effective as a ten per cent increase in both the number of obstetricians and nurses. Both policies would result in an decrease in the bed/labour ratio and increase labour intensity in the perinatal hospital sector.

### 6.13.2 Neonatal Mortality: Age specific

The production functions were also re-estimated for deaths in three different stages of the neonatal period: <24 hours, 1-6 days and 7-27 days. For deaths within the first 24 hours and between 7 - 27 days following birth, F-tests supported a simple model including only a time trend, LBW and a constant (Table 6.17). When the equation was re-estimated for deaths between 1 and 6 days following birth the health service variables could not be excluded on the basis of the F-test (F = 6.457, CV = 2.80).

The results of re-specifying the age specific equations using the dependent variable lagged one year as a measure of technical change in place of the simple linear time trend are also presented in Table 6.17. OBS and NUR enter all age-specific equations with a negative sign. Corresponding elasticities are given in Table 6.18. The elasticity of neonatal mortality with respect to nursing inputs increases steadily throughout the neonatal weeks, reaching a peak during the late neonatal period. A comparison of the elasticities for the labour variables and the bed variable suggests that a ten per cent in labour intensity, achieved by either increasing labour inputs holding the bed stock constant or decreasing the bed stock holding labour inputs constant, would improve age-specific neonatal mortality rates.

### 6.13.3 Neonatal Mortality: Cause specific

The precise impact of nursing and medical resources per bed, however, may be obscured due to the aggregate measure of output: neonatal deaths from all causes. The results of testing and re-estimating the equations for different causes are given in Tables 6.19 and 6.20. Three specific causes of neonatal mortality were examined. Equation 6.35 reports the OLS estimates of the neonatal mortality production function for deaths due to congenital malforma-

tions. Although several of their individual coefficients are not significantly different from zero, the health service variables as a set could not be excluded from the equation. The most important determinant is the availability of intensive neonatal care. The marginal product of intensive care is about 5 fewer deaths per 10,000 live births. When the equation is respecified to include a lagged dependent variable (Equation 6.39) the intensive care variable remains significant. The number of obstetricians per 1000 births also emerges as a significant influence on neonatal mortality from congenital malformations. The incidence of low birthweight has no significant impact on this cause of neonatal death.

The results for equations where the dependent variable was neonatal mortality due to prematurity (Equation 6.36 and 6.40) or asphyxia (Equation 6.37 and 6.41) confirmed the acceptance of a simplified model excluding the health service variables. The incidence of birthweight is a significant factor influencing deaths due to prematurity or asphyxia. A prominent characteristic of these equations is the significant coefficients on the time trend and lagged dependent variable proxies for technical change which explain the vast majority of the variation over time in neonatal mortality from these specific causes.

A fourth category of cause-specific neonatal mortality, namely all other causes, was also investigated (Equations 6.38 and 6.42). The general model including the health service variables was accepted using the F-test criteria. The signs on the labour variables are negative as is that on the INTENSE dummy. BEDS enter the equations with a significant positive coefficient. The incidence of low birthweight is not significantly related to this residual and heterogenous collection of neonatal deaths not classified as due to malformations, prematurity or asphyxia.

The elasticities of cause-specific mortality with respect to the labour and capital input variables are presented in Table 6.21 for neonatal deaths due to malformations or other causes. The elasticities calculated for the malformation mortality rate are sensitive to the specification of the proxy for technical change. The positive elasticity calculated for the nursing variable is not significantly different from zero. The estimated elasticities in the case of deaths due to other causes are well defined and invariant to the equation specification. The elasticities with respect to obstetricians and nurses are virtually identical: suggesting that a ten per cent increase in these inputs per 1000 births results in a 7 to 8 per cent decrease in mortality. A similar reduction in the bed stock would lead to a 13 per cent improvement in mortality from this residual group of causes.

## 6.14 Conclusions

This chapter presented the first set of perinatal mortality production functions estimated using time series data. A wide range of different functional forms and specifications were carefully tested and evaluated. In general the restrictions imposed by a simple linear Leontief production function were found to be acceptable compared to more complicated and flexible functional forms. The widely used Cobb-Douglas specification was found to be suspect both on *a priori* and empirical grounds.

The results confirmed that hospital medical and nursing inputs are important determinants of perinatal mortality across different birthweight strata. The elasticity of perinatal mortality with respect to the number of obstetricians ranges from -0.820 in very low birthweight infants to -0.950 in infants of normal birthweight. Nurses have a similar impact on mortality in low birthweight infants but appear to have a more important impact on mortality in the normal weight infant (elasticity = -1.499). Decreases in the bed stock are associated with improvements in perinatal mortality, particularly for low birthweight infants. This supports the hypothesis that the shift over time in the mix of obstetric beds away from small maternity units and wards towards larger and more specialised units is associated with an improvement in mortality. Thus, an increase in the

(average) scale of maternity units accompanied by a reduction in the total maternity capital stock appears to have led to an improvement in the health outcomes of those infants who faced the greatest risks of mortality.

The estimates also suggest that the elasticity of perinatal mortality with respect to expenditure per birth ranges from -0.443 in very low birthweight infants to -1.005 in normal weight babies. Applying these elasticities to the mean numbers of births and the distribution of birthweight suggests that the incremental cost per year of potential life gained ranges from just under £7000 pounds in the very low birthweight infant to around £2300 in the normal birthweight infant (1985 prices and 5 per cent discount rate). In terms of cost per life year gained, investment in perinatal hospital services has generated health outcomes at a cost which is lower than many other health care programmes whose costs and effectiveness have been evaluated from an economic perspective (Torrance, 1986).

Table 6.1 OUTPUT VARIABLE NAMES, DEFINITIONS, MEANS AND STANDARD DEVIATIONS

| VARIABLE | DEFINITION | MEAN* | STANDARD DEVIATION* |
|---|---|---|---|
| PMR | Fetal deaths and neonatal deaths per 1000 total births | 26.63 | 10.36 |
| PMR(vlbw) | Fetal deaths and first week deaths weighing less than 1.5kg per 1000 total births weighing less than 1.5kg | 712.98 | 152.3 |
| PMR(ilbw) | Fetal deaths and first week deaths weighing between 1.5 - 2.5kg per 1000 total births weighing between 1.5 - 2.5kg | 122.71 | 46.82 |
| PMR(nbw) | Fetal deaths and first week deaths weighing more than 2.5 kg per 1000 total births weighing more than 2.5kg | 11.14 | 5.87 |
| FMR | Fetal deaths per 1000 total births | 13.83 | 6.1 |
| NMR | Neonatal deaths between 0 - 27 days per 1000 live births | 13.02 | 4.43 |
| VENMR | Neonatal deaths within 24 hours of birth per 1000 live births | 6.46 | 2.34 |
| ENMR | Neonatal deaths between 1 and 6 days following birth per 1000 live births | 4.66 | 1.62 |
| LNMR | Neonatal deaths between 7 and 27 days per 1000 live births | 1.91 | 0.52 |
| MAL | Neonatal deaths attributed to congenital malformations per 1000 live births | 3.03 | 0.61 |
| PREM | Neonatal deaths attributed to prematurity per 1000 live births | 2.21 | 1.28 |
| ASPH | Neonatal deaths attributed to asphyxia per 1000 live births | 3.67 | 1.14 |
| OTHER | Neonatal deaths not attributed to congenital malformations, prematurity or asphyxia per 1000 live births | 4.09 | 1.63 |

*Calculated over the period 1956-1985

TABLE 6.2 INPUT VARIABLE NAMES, DEFINITIONS, MEANS AND STANDARD DEVIATIONS

| VARIABLE | DEFINITION | MEAN* | STANDARD DEVIATION* |
|---|---|---|---|
| LBW | Total births weighing less than 2.5kg per 1000 total births | 71.07 | 4.67 |
| VLBW | Total births weighing less than 1.5kg per 1000 total births | 11.09 | 1.57 |
| ILBW | Total births weighing between 1.5 - 2.5 kg per 1000 total births | 59.98 | 3.54 |
| NBW | Total births weighing more than 2.5kg per 1000 total births | 928.93 | 4.67 |
| OBS | WTE obstetricians and gynecologists per 1000 total births | 2.91 | 1.12 |
| NUR | WTE maternity nurses (hour adjusted) per 1000 total births | 35.73 | 9.58 |
| BED | Available staffed maternity beds (case flow adjusted) per 1000 total births | 35.18 | 7.05 |
| INTENSE | Dummy variable = 0 (1956 - 1974) 1 (1975 - 1985) | 0.37 | 0.49 |
| EXPEND | Real expenditure on perinatal hospital care per total birth | 532.03 | 194.11 |

*Calculated over the period 1956-1985

TABLE 6.3 LEONTIEF PRODUCTION FUNCTION
F-TESTS FOR INCLUSION OF HEALTH SERVICE INPUTS

| MODEL SPECIFICATION | F 5% CV | BIRTHWEIGHT GROUP < 1.5KG | 1.5 - 2.5 KG | > 2.5KG |
|---|---|---|---|---|
| TIME * TREND | 2.80 | 3.042 | 3.609 | 8.096 |
| LAGGED** DEPENDENT VARIABLE | 2.82 | 2.789 | 10.303 | 1.667 |

NOTES: * GENERAL MODEL = CONSTANT, TREND, BW, OBS, NUR, BED, INTENSE
RESTRICTED MODEL = CONSTANT, TREND, BW

** GENERAL MODEL = CONSTANT, LAGGED MORTALITY RATE, BW, OBS, NUR, BED, INTENSE
RESTRICTED MODEL = CONSTANT, LAGGED MORTALITY RATE, BW

In these regressions the birthweight variables (BW) correspond with the the weight-specific mortality rate.

TABLE 6.4 BIRTHWEIGHT SPECIFIC PRODUCTION FUNCTIONS (Leontief Model)

| | (TIME TREND EQUATIONS) | | | ("LEARNING BY DOING" EQUATIONS) | | |
|---|---|---|---|---|---|---|
| EQUATION | 6.7 | 6.8 | 6.9 | 6.10 | 6.11 | 6.12 |
| DEPENDENT VARIABLE | PNM < 1.5 KG | PNM 1.5-2.5 KG | PNM > 2.5 KG | PNM < 1.5 KG | PNM 1.5-2.5 KG | PNM > 2.5 KG |
| REGRESSOR | | | | | | |
| BW* | -21.1263 [1.330] | -0.6464 [0.474] | -0.0687 [0.630] | -20.5560 [1.133] | -0.5536 [0.381] | -0.0394 [0.327] |
| OBS | -10.9480 [0.155] | -22.1940 [1.601] | 0.7119 [0.675] | -95.5030 [2.077] | -33.7710 [2.948] | -1.6540 [1.433] |
| NUR | 1.0055 [0.157] | -1.999 [1.680] | -0.1831 [1.706] | -8.29 [1.540] | -2.846 [2.289] | -0.1349 [0.951] |
| BED | 4.924 [0.879] | 3.196 [3.099] | -0.019 [0.183] | 14.1909 [2.475] | 4.4582 [3.817] | 0.1146 [0.844] |
| INTENSE | -58.9206 [1.567] | -9.3123 [1.210] | -1.044 [1.517] | -25.237 [0.619] | -4.859 [0.508] | 0.0112 [0.011] |
| TREND | -18.8299 [2.552] | -2.1303 [2.154] | -0.4731 [4.575] | - | - | - |
| LAGGED DEPENDENT VARIABLE | - | - | - | 0.4173 [1.650] | 0.1595 [0.804] | 0.5396 [1.690] |
| CONSTANT | 1083.475 [4.317] | 221.5203 [2.455] | 87.811 [0.876] | 722.875 [1.655] | 180.936 [2.091] | 47.009 [0.412] |
| R-squared | 0.9473 | 0.9776 | 0.9884 | 0.945 | 0.9763 | 0.9822 |
| SER | 39.236 | 7.8732 | 0.7105 | 40.2 | 8.0057 | 0.8545 |
| F | 69 | 167.06 | 325.57 | 63.05 | 151.34 | 202.04 |
| DW | 1.983 | 1.875 | 2.038 | 2.814 | 2.3 | 2.973 |
| RSS | 35406.82 | 1425.711 | 11.6108 | 35552.9 | 1410.01 | 16.0627 |

* Birthweight variable is consistent with weight category of dependent variable.

TABLE 6.5 LONG RUN ESTIMATES (AUTOREGRESSIVE DISTRIBUTED LAG MODEL)
BIRTHWEIGHT SPECIFIC PERINATAL MORTALITY PRODUCTION FUNCTIONS

| | BIRTHWEIGHT GROUP | | |
|---|---|---|---|
| VARIABLE | < 1.5 KG | 1.5-2.5 KG | > 2.5 KG |
| BW* | -59.473 | 0.538 | -0.011 |
| | (3.688) | (0.449) | (0.072) |
| OBS | -196.000 | -36.555 | -3.466 |
| | (4.344) | (3.220) | (2.504) |
| NUR | -13.838 | -2.682 | -0.447 |
| | (2.180) | (1.632) | (1.929) |
| BED | 24.052 | 4.420 | 0.385 |
| | (7.958) | (4.000) | (2.577) |
| INTENSE | -27.721 | -10.069 | 0.345 |
| | (0.673) | (1.064) | (0.275) |
| CONSTANT | 1555.000 | 140.000 | 32.849 |
| | (6.063) | (1.790) | (0.233) |
| WALD TEST STATISTIC | 5349.825 | 4158.718 | 1083.008 |

* Birthweight variable is consistent with weight category of dependent variable.

TABLE 6.6 LONG RUN ELASTICITIES DERIVED FROM AUTOREGRESSIVE DISTRIBUTED LAG MODEL BIRTHWEIGHT SPECIFIC PERINATAL MORTALITY PRODUCTION FUNCTIONS

| | BIRTHWEIGHT GROUP | | |
|---|---|---|---|
| INPUT | < 1.5 KG | 1.5-2.5 KG | > 2.5 KG |
| OBS | -0.820 | -0.898 | -0.950 |
| NUR | -0.708 | -0.806 | -1.499 |
| BED | 1.202 | 1.297 | 1.261 |

ALL ELASTICITIES EVALUATED AT SAMPLE MEANS CALCULATED OVER THE PERIOD 1957 TO 1985

TABLE 6.7 LEONTIEF AND LEONTIEF-DIEWERT PRODUCTION FUNCTIONS
F-TESTS FOR TESTING RESTRICTIONS OF LEONTIEF MODEL (NO INPUT INTERACTION)

| MODEL SPECIFICATION | F 5% CV | BIRTHWEIGHT GROUP < 1.5KG | 1.5 - 2.5 KG | > 2.5KG |
|---|---|---|---|---|
| TIME TREND | 2.74 | 1.750 | 0.827 | 0.345 |
| LAGGED DEPENDENT VARIABLE | 2.79 | 0.914 | 0.440 | 0.409 |

NOTE: GENERAL MODEL = LEONTIEF-DIEWERT
RESTRICTED MODEL = LEONTIEF

TABLE 6.8 MULTICOLLINEARITY AND THE LEONTIEF-DIEWERT PRODUCTION FUNCTION
BIRTHWEIGHT SPECIFIC PERINATAL MORTALITY

| | BIRTHWEIGHT GROUP | | | | | |
|---|---|---|---|---|---|---|
| | < 1.5 kg | | 1.5-2.5 kg | | > 2.5 kg | |
| VARIABLE | LEONTIEF | LEONTIEF DIEWERT | LEONTIEF | LEONTIEF DIEWERT | LEONTIEF | LEONTIEF DIEWERT |
| BW* | 0.1306 | 0.4540 | 0.0284 | 0.0252 | 0.0458 | 0.0037 |
| OBS | 0.0013 | 0.0124 | 0.1157 | 0.0013 | 0.0165 | 0.0031 |
| NUR | 0.0015 | 0.0000 | 0.1651 | 0.0301 | 0.1502 | 0.0014 |
| BED | 0.2820 | 0.0021 | 0.2433 | 0.0284 | 0.0013 | 0.0574 |
| √(BW*OBS) | - | 0.0830 | - | 0.0257 | - | 0.0109 |
| √(BW*NUR) | - | 0.0566 | - | 0.0391 | - | 0.0598 |
| √(BW*BED) | - | 0.0002 | - | 0.0311 | - | 0.0392 |
| √(BW*NUR) | - | 0.0010 | - | 0.0244 | - | 0.0032 |
| √(BW*BED) | - | 0.0009 | - | 0.0349 | - | 0.0003 |
| √(BW*BED) | - | 0.0031 | - | 0.0037 | - | 0.0295 |
| INTENSE | 0.1394 | 0.0017 | 0.9010 | 0.0235 | 0.1331 | 0.0699 |
| TREND | 0.2753 | 0.6326 | 0.1081 | 0.2399 | 0.4349 | 0.3181 |
| CONSTANT | 0.5990 | 0.7324 | 0.4525 | 0.4707 | 0.0845 | 0.0250 |
| $R^2$ | 0.9474 | 0.9799 | 0.9776 | 0.9841 | 0.9884 | 0.9898 |

NOTES: * BW variables are consistent with weight-specific mortality rates
Numbers in table cells are partial correlation coefficients

TABLE 6.9 PE TESTS OF LINEAR VERSUS LOG-LINEAR MODEL

| | BIRTHWEIGHT GROUP | | |
|---|---|---|---|
| HYPOTHESES | < 1.5 KG | 1.5-2.5 KG | > 2.5 KG |
| LINEAR | 0.0016 | 0.0104 | 0.0556 |
| $\theta_1 = 0$ | [1.040] | [2.021] | [1.240] |
| LOG-LINEAR | 753.8870 | -11.9473 | 4.1340 |
| $\theta_0 = 0$ | [0.797] | [0.197] | [2.265] |

NOTE: t-statistics in brackets

TABLE 6.10 F TESTS OF RESTRICTIONS: LEONTIEF AND LEONTIEF-DIEWERT MODELS
(BIRTHWEIGHT SPECIFIC PERINATAL MORTALITY)

| | BIRTHWEIGHT GROUP | | | | | |
|---|---|---|---|---|---|---|
| | < 1.5KG | | 1.5-2.5KG | | > 2.5KG | |
| F-TEST FOR ADDING | TIME* TREND | LDV** | TIME* TREND | LDV** | TIME* TREND | LDV** |
| EXPEND & INTENSE | 5.095 | 1.140 | 3.359 | 2.808 | 16.133 | 3.926 |
| ∮(EXPEND*BW) | 16.526 | 0.144 | 0.936 | 3.675 | 6.020 | 7.027 |

NOTES: * RESTRICTED MODEL INCLUDES BW, CONSTANT AND TIME TREND
** RESTRICTED MODEL INCLUDES BW, CONSTANT AND LAGGED DEPENDENT VARIABLE

TABLE 6.11 BIRTHWEIGHT SPECIFIC PRODUCTION FUNCTIONS (Leontief-Diewert Model)

| | (TIME TREND EQUATIONS) | | | ("LEARNING BY DOING" EQUATIONS) | | |
|---|---|---|---|---|---|---|
| EQUATION | 6.19 | 6.20 | 6.21 | 6.22 | 6.23 | 6.24 |
| DEPENDENT VARIABLE | PNM < 1.5 KG | PNM 1.5-2.5 KG | PNM > 2.5 KG | PNM < 1.5 KG | PNM 1.5-2.5 KG | PNM > 2.5 KG |
| REGRESSOR | | | | | | |
| BW* | -124.432 [4.132] | -0.4739 [0.364] | -0.0156 [0.280] | 6.1958 [0.937] | 3.7407 [1.539] | 0.0627 [0.770] |
| EXPENDITURE | -1.6365 [2.855] | - | 0.0463 [2.017] | - | 0.4790 [1.746] | 0.0685 [1.786] |
| (BW*EXPEND) | 30.3255 [3.856] | - | -0.0873 [2.661] | - | -2.9305 [1.880] | -0.1194 [1.992] |
| INTENSE | 32.39 [0.842] | - | -2.004 [1.457] | - | -32.7309 [2.121] | -2.0659 [1.018] |
| TREND | -33.5911 [11.439] | -5.3691 [10.385] | -0.274 [4.731] | - | - | - |
| LAGGED DEPENDENT VARIABLE | - | - | - | 0.9767 [13.500] | 0.6213 [2.909] | 0.4608 [1.972] |
| CONSTANT | 1213.82 [7.203] | 234.3482 [2.732] | 66.3003 [1.264] | -69.9301 [0.965] | 89.3153 [1.123] | -5.6554 [0.072] |
| R-squared | 0.9661 | 0.9635 | 0.9902 | 0.9172 | 0.9525 | 0.9866 |
| SER | 30.8069 | 9.2707 | 0.6377 | 45.3956 | 11.0937 | 0.7248 |
| F | 136.96 | 356.26 | 485.97 | 143.96 | 92.27 | 338.51 |
| DW | 2.02 | 1.691 | 1.982 | 2.467 | 2.744 | 2.835 |
| RSS | 22777.58 | 2320.56 | 9.758 | 53579.73 | 2830.63 | 12.0815 |

* Birthweight variable is consistent with weight category

TABLE 6.12 MARGINAL PRODUCTS (mp) AND ELASTICITIES (e) OF PERINATAL MORTALITY WITH RESPECT TO PERINATAL HOSPITAL EXPENDITURE PER BIRTH

| | | MODEL SPECIFICATION | | | |
|---|---|---|---|---|---|
| | | TIME TREND | | LEARNING BY DOING | |
| WEIGHT GROUP | | LEONTIEF | LEONTIEF-DIEWERT | LEONTIEF | LEONTIEF-DIEWERT |
| < 1.5KG | mp | 0.372 | 0.553 | - | - |
| | e | 0.278 | 0.413 | | |
| 1.5-2.5KG | mp | - | - | -0.039 | -0.008 |
| | e | | | -0.175 | -0.035 |
| > 2.5KG | mp | -0.010 | -0.011 | -0.008 | -0.010 |
| | e | -0.497 | -0.546 | -0.396 | -0.486 |

TABLE 6.13 RECURSIVE LEAST SQUARES (Leontief-Diewert Model)

(TIME TREND EQUATIONS)

| EQUATION | 6.19A | 6.20A | 6.21A |
|---|---|---|---|
| DEPENDENT VARIABLE | PNM < 1.5 KG | PNM 1.5-2.5 KG | PNM > 2.5 KG |
| REGRESSOR | | | |
| BW* | -122.898 [4.853] | -5.298 [2.057] | 0.0168 [0.015] |
| EXPENDITURE | -1.4040 [3.507] | -0.426 [2.718] | -0.0265 [1.060] |
| (BW*EXPEND) | 28.5487 [4.199] | 3.2012 [2.648] | 0.0351 [0.771] |
| TREND | -35.7341 [7.447] | -8.763 [4.026] | -0.684 [3.042] |
| CONSTANT | 1245.383 [8.048] | 239.398 [2.709] | -4.5297 [0.038] |
| R-squared | 0.9298 | 0.9706 | 0.9880 |
| SER | 31.1462 | 7.3158 | 0.6473 |
| F | 66.1900 | 165.3400 | 409.9500 |
| DW | 2.0480 | 2.0570 | 2.3310 |
| RSS | 19401.6780 | 1070.4300 | 8.3793 |

* Birthweight variable is consistent with weight category

TABLE 6.14 RECURSIVE LEAST SQUARES (Leontief Model)

(LEARNING BY DOING EQUATIONS)

| EQUATION | 6.22A | 6.23A | 6.24A |
|---|---|---|---|
| DEPENDENT VARIABLE | PNM < 1.5 KG | PNM 1.5-2.5 KG | PNM > 2.5 KG |
| REGRESSOR | | | |
| BW* | -1.6166 | -0.0717 | -0.0669 |
| | [0.123] | [1.670] | [0.577] |
| EXPEND | -0.1526 | -0.0717 | -0.0092 |
| | [1.198] | [1.670] | [2.049] |
| LAGGED DEPENDENT VARIABLE | 0.7728 | 0.5831 | 0.6246 |
| | [5.150] | [1.636] | [2.978] |
| CONSTANT | 256.7991 | 22.476 | 70.883 |
| | [0.918] | [0.281] | [0.639] |
| R-squared | 0.8461 | 0.9160 | 0.9758 |
| SER | 45.0981 | 11.9508 | 0.8692 |
| F | 36.6500 | 72.7400 | 268.9100 |
| DW | 2.0790 | 2.7840 | 2.7800 |
| RSS | 40676.7900 | 2856.4400 | 15.1100 |

* Birthweight variable is consistent with weight category

TABLE 6.15 RELATIVE FORECASTING PERFORMANCE
(MEAN ABSOLUTE FORECASTING ERROR)

| MODEL SPECIFICATION | BIRTHWEIGHT GROUP | | |
|---|---|---|---|
| | < 1.5 KG % | 1.5-2.5 KG % | > 2.5 KG % |
| TIME TREND | 10.00 | 39.20 | 58.60 |
| LEARNING BY DOING | 10.60 | 21.00 | 9.50 |

TABLE 6.16 INCREMENTAL COST (£) PER YEAR OF POTENTIAL LIFE GAINED

| DISCOUNT RATE (%) | BIRTHWEIGHT GROUP | | |
|---|---|---|---|
| | < 1.5 KG | 1.5-2.5 KG | > 2.5 KG |
| 0.0 | 1897<br>(2669) | 1376<br>(1925) | 626<br>(881) |
| 5.0 | 6866<br>(9660) | 4979<br>(6966) | 2267<br>(3190) |
| 7.5 | 10021<br>(14100) | 7267<br>(10167) | 3309<br>(4655) |
| 10.0 | 13291<br>(18701) | 9639<br>(13485) | 4389<br>(6175) |

NOTE: COSTS IN 1980 (1985) PRICES

TABLE 6.17 AGE SPECIFIC NEONATAL PRODUCTION FUNCTIONS (Leontief Model)

| | (TIME TREND EQUATIONS) | | | ("LEARNING BY DOING" EQUATIONS) | | |
|---|---|---|---|---|---|---|
| EQUATION | 6.29 | 6.30 | 6.31 | 6.32 | 6.33 | 6.34 |
| DEPENDENT VARIABLE | VENMR < 1 DAY | ENMR 1 - 6 DAYS | LNMR 7 - 27 DAYS | VENMR < 1 DAY | ENMR 1 - 6 DAYS | LNMR 7 - 27 DAYS |
| REGRESSOR | | | | | | |
| LBW | -0.0135 [1.330] | 0.0368 [1.606] | -0.0016 [0.113] | 0.0104 [0.214] | 0.0145 [0.568] | -0.0094 [0.455] |
| OBS | - | 0.2371 [0.614] | - | -1.1564 [1.730] | -0.5429 [1.299] | -0.4203 [3.012] |
| NUR | - | -0.0261 [0.829] | - | -0.0409 [0.627] | -0.0445 [1.473] | -0.0414 [1.794] |
| BED | - | 0.0447 [1.319] | - | 0.1159 [1.668] | 0.0863 [2.745] | 0.059 [3.164] |
| INTENSE | - | -0.0969 [0.324] | - | -0.2083 [0.473] | -0.1963 [0.641] | -0.1119 [0.671] |
| TREND | -0.2661 [15.221] | -0.188 [5.011] | -0.0574 [8.339] | - | - | - |
| LAGGED DEPENDENT VARIABLE | - | - | - | 0.5107 [2.646] | 0.5698 [3.915] | 0.1196 [0.710] |
| CONSTANT | 11.5409 [4.598] | 3.6627 [1.806] | 2.9082 [2.655] | 3.1359 [0.926] | 1.071 [0.559] | 2.927 [1.844] |
| R-squared | 0.9522 | 0.9866 | 0.906 | 0.9672 | 0.9808 | 0.8978 |
| SER | 0.5313 | 0.21089 | 0.16643 | 0.4771 | 0.2522 | 0.1881 |
| F | 268.92 | 282.38 | 130.16 | 112.91 | 196.22 | 33.67 |
| DW | 0.985 | 2.493 | 2.188 | 2.844 | 2.382 | 2.093 |
| RSS | 7.62202 | 1.0229 | 0.74791 | 5.2357 | 1.4632 | 0.8134 |

TABLE 6.18 ELASTICITIES OF NEONATAL MORTALITY WITH RESPECT TO OBSTETRIC, NURSING AND BED INPUTS

| INPUT | MODEL SPECIFICATION | MORTALITY RATE VENMR | ENMR | LNMR |
|---|---|---|---|---|
| OBS | TIME TREND | - | 0.148 | - |
| | LBD | -0.521 | -0.339 | -0.642 |
| NUR | TIME TREND | - | -0.020 | - |
| | LBD | -0.226 | -0.342 | -0.776 |
| BED | TIME TREND | - | 0.338 | - |
| | LBD | 0.631 | 0.652 | 1.089 |

TABLE 6.19 CAUSE SPECIFIC NEONATAL MORTALITY PRODUCTION FUNCTIONS

(TIME TREND EQUATIONS)

| EQUATION | 6.35 | 6.36 | 6.37 | 6.38 |
|---|---|---|---|---|
| DEPENDENT VARIABLE | MAL | PREM | ASPH | OTHER |
| REGRESSOR | | | | |
| LBW | -0.0085<br>[0.224] | 0.0365<br>[1.427] | 0.0609<br>[1.900] | -0.0551<br>[1.251] |
| OBS | -0.1998<br>[0.370] | - | - | -1.0510<br>[1.496] |
| NUR | 0.0336<br>[0.841] | - | - | -0.0839<br>[1.668] |
| BED | 0.0259<br>[0.476] | - | - | 0.1486<br>[3.535] |
| INTENSE | -0.5332<br>[2.030] | - | - | -0.3507<br>[1.494] |
| TREND | -0.0594<br>[1.514] | -0.1258<br>[10.019] | -0.0932<br>[4.882] | -0.0603<br>[1.161] |
| LAGGED DEPENDENT VARIABLE | - | - | - | - |
| CONSTANT | 3.2195<br>[1.111] | 1.5716<br>[0.786] | 0.7905<br>[0.310] | 9.8995<br>[2.836] |
| R-squared | 0.8374 | 0.9686 | 0.8942 | 0.9535 |
| SER | 0.27659 | 0.23463 | 0.38371 | 0.39361 |
| F | 19.74 | 416.75 | 114.05 | 78.57 |
| DW | 2.232 | 1.674 | 0.732 | 1.472 |
| RSS | 1.7596 | 1.4865 | 3.97533 | 3.56345 |

TABLE 6.20 CAUSE SPECIFIC NEONATAL MORTALITY PRODUCTION FUNCTIONS

("LEARNING BY DOING" EQUATIONS)

| EQUATION | 6.39 | 6.40 | 6.41 | 6.42 |
|---|---|---|---|---|
| DEPENDENT VARIABLE | MAL | PREM | ASPH | OTHER |
| REGRESSOR | | | | |
| LBW | -0.0030<br>[0.076] | 0.0188<br>[0.607] | 0.0377<br>[1.544] | -0.0450<br>[1.140] |
| OBS | -0.6622<br>[1.744] | - | - | -1.1629<br>[3.327] |
| NUR | 0.0122<br>[0.283] | - | - | -0.0886<br>[1.806] |
| BED | 0.0649<br>[1.404] | - | - | 0.1527<br>[3.778] |
| INTENSE | -0.4395<br>[1.782] | - | - | -0.2176<br>[0.880] |
| TREND | - | - | - | - |
| LAGGED DEPENDENT VARIABLE | 0.1005<br>[0.491] | 0.8742<br>[8.704] | 0.8602<br>[7.318] | 0.2623<br>[1.762] |
| CONSTANT | 2.3002<br>[0.747] | -1.192<br>[0.598] | -2.2595<br>[1.673] | 7.4351<br>[2.526] |
| R-squared | 0.8267 | 0.9563 | 0.9282 | 0.9573 |
| SER | 0.2855 | 0.2768 | 0.3161 | 0.377 |
| F | 18.29 | 295.61 | 174.47 | 86.01 |
| DW | 2.537 | 2.801 | 2.067 | 2.000 |
| RSS | 1.8753 | 2.06903 | 2.6974 | 3.2681 |

TABLE 6.21 ELASTICITIES OF CAUSE-SPECIFIC NEONATAL MORTALITY WITH RESPECT TO OBSTETRIC, NURSING AND BED INPUTS

| INPUT | MODEL SPECIFICATION | MORTALITY RATE MAL | PREM | ASPH | OTHER |
|---|---|---|---|---|---|
| OBS | TIME TREND | -0.192 | - | - | -0.748 |
| | L8D | -0.636 | - | - | -0.781 |
| NUR | TIME TREND | 0.397 | - | - | -0.732 |
| | L8D | 0.144 | - | - | -0.773 |
| BED | TIME TREND | 0.301 | - | - | 1.277 |
| | L8D | 0.753 | - | - | 1.311 |

Figure 6.1 Maternity Nurse/Bed Ratio

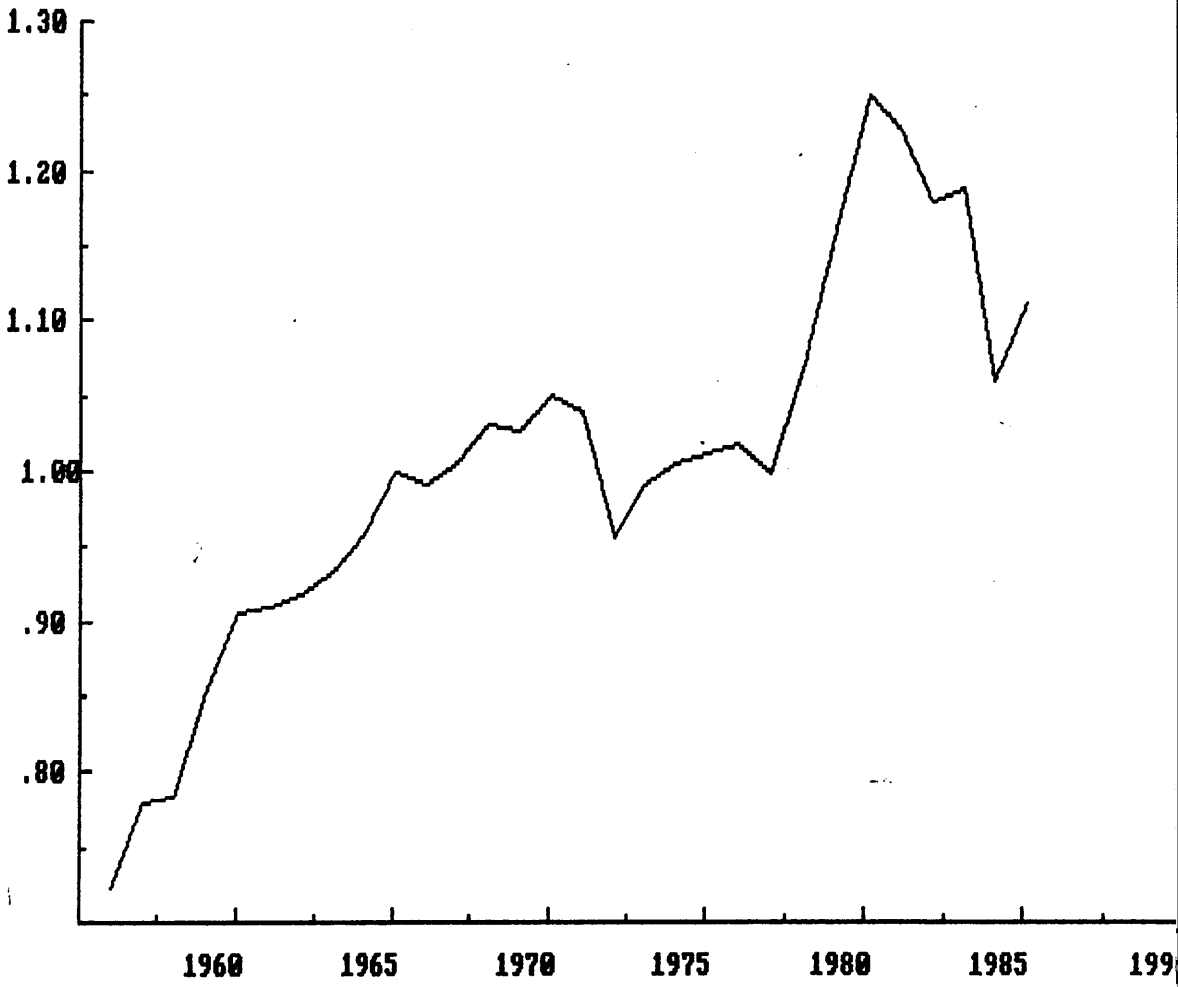


**Figure 6.2 Obstetrician/Bed Ratio**

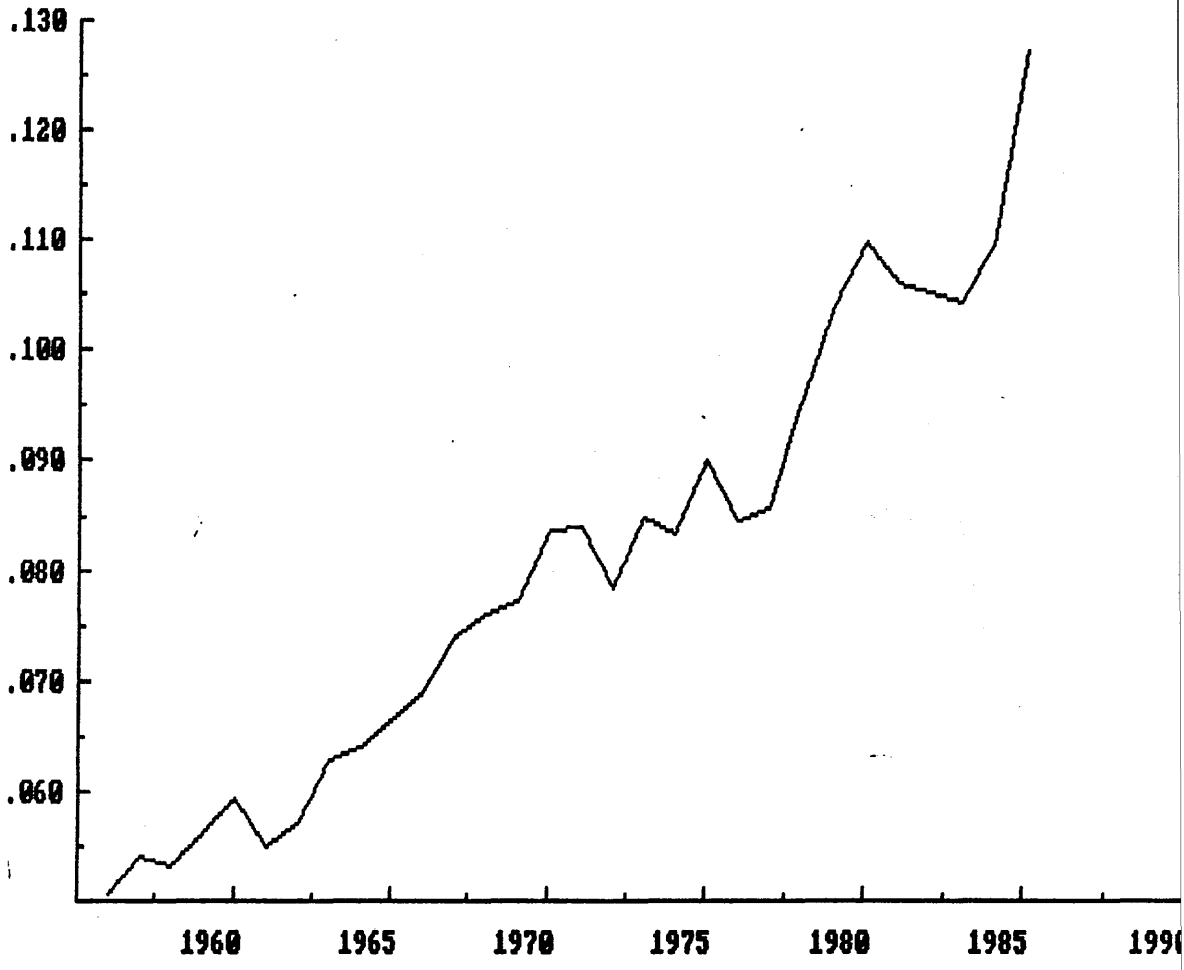


Figure 6.3 Maternity Nurse/Obstetrician Ratio

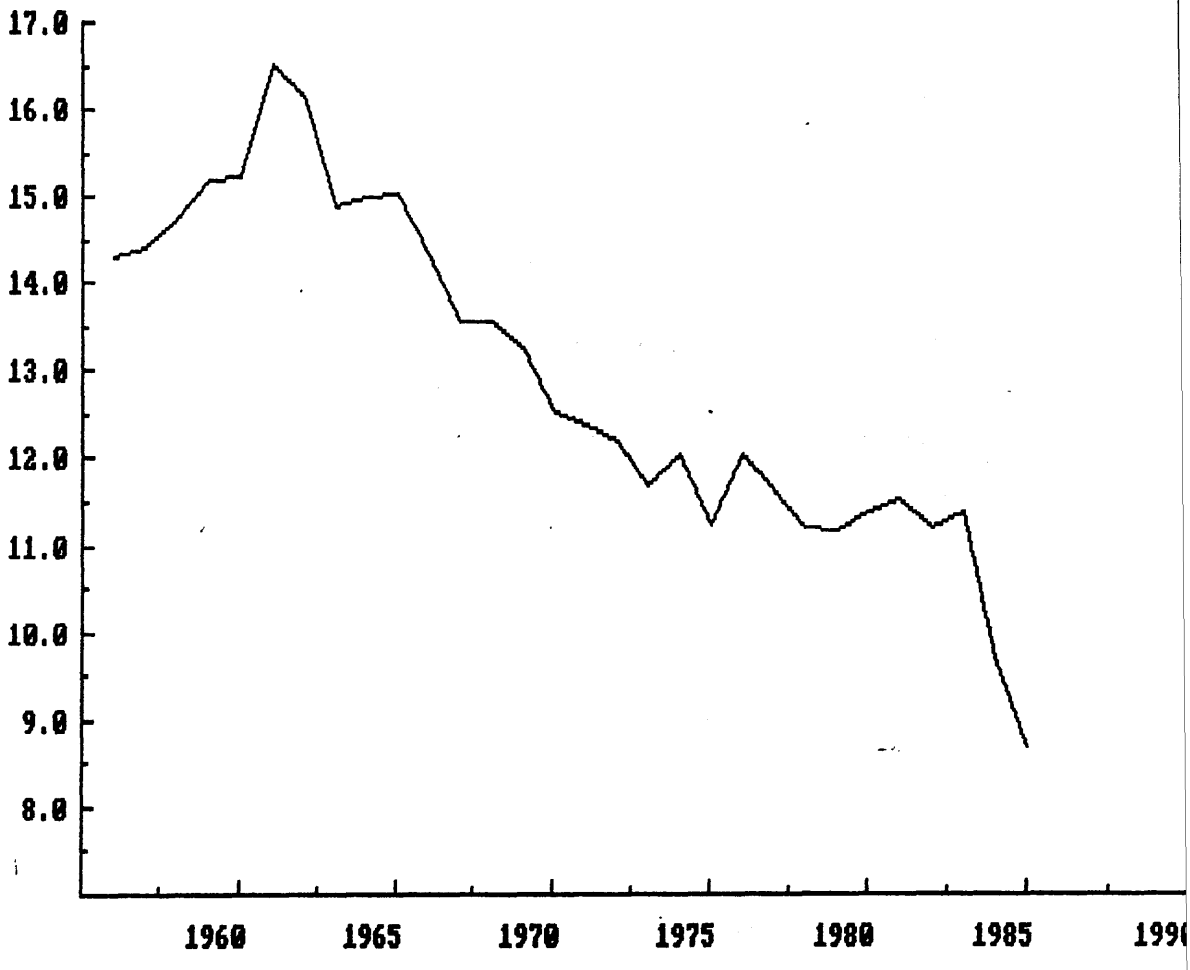


Figure 6.4 Parameter Stability Over Time [Equation 6.19a)

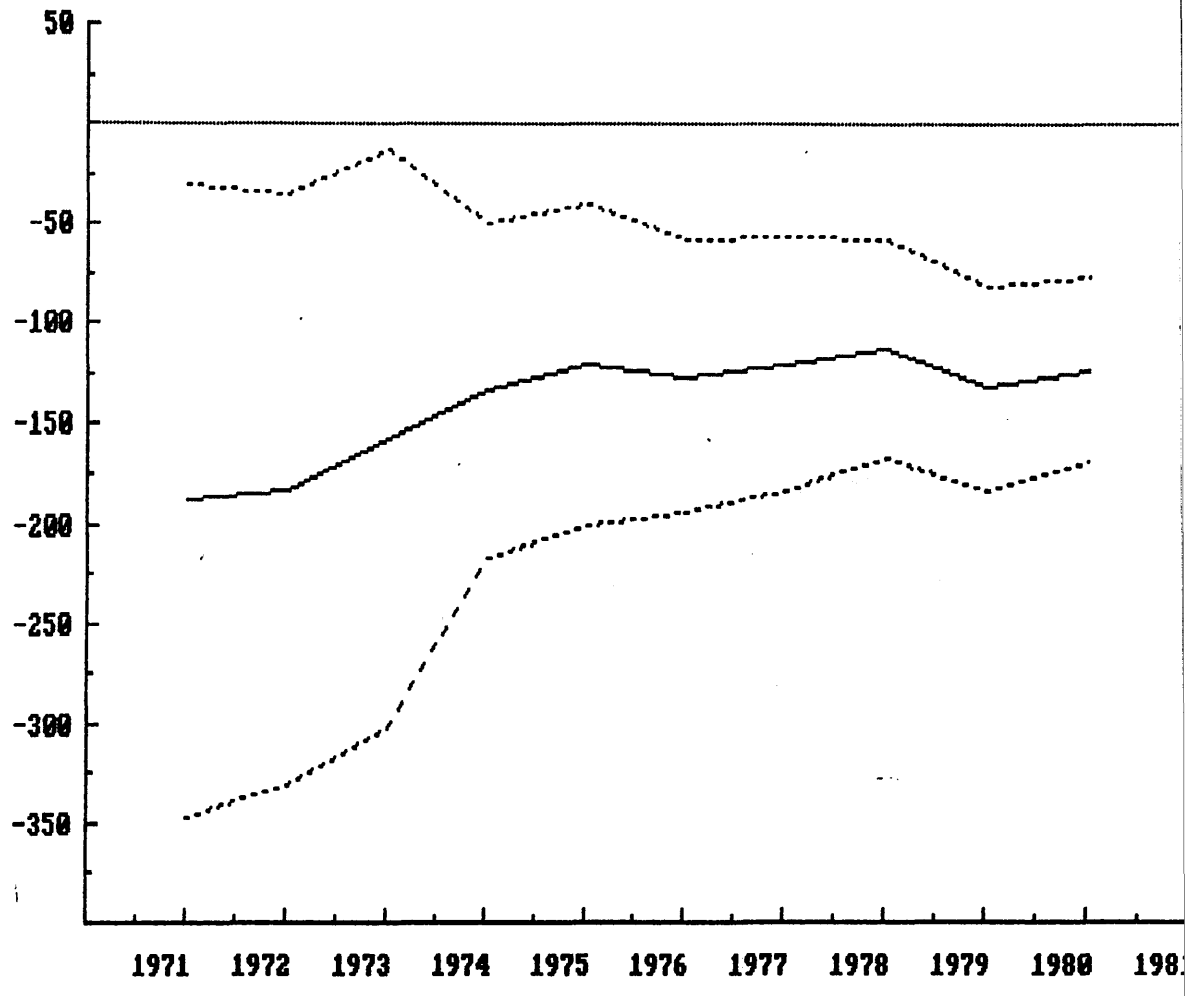


Figure 6.5 Parameter Stability Over Time [Equation 6.19a]
EXPEND =____ ± 2*S.E.=-- --

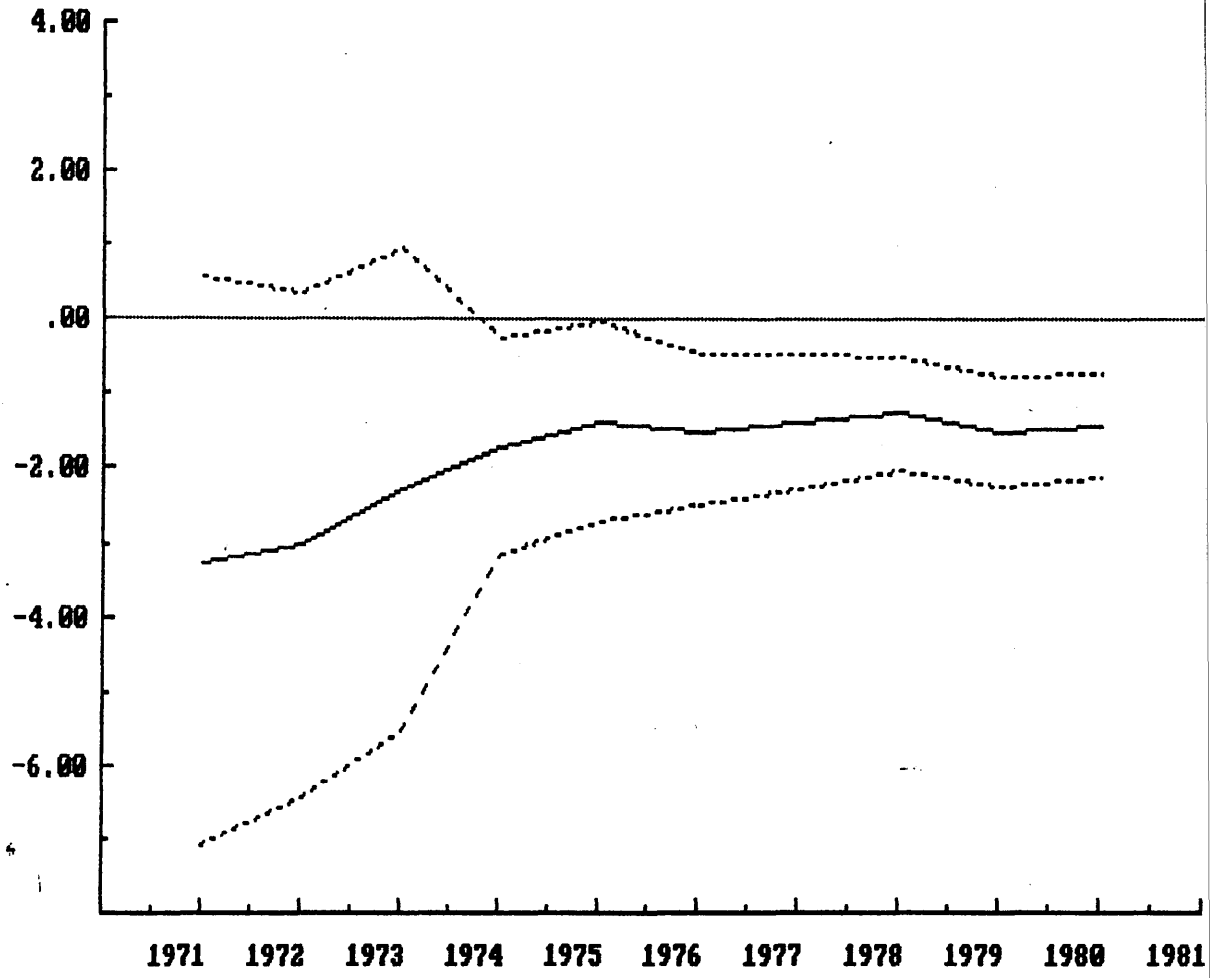


Figure 6.6 Parameter Stability Over Time [Equation 6.19a]
J(VLBW*EXPEND)=____ ± 2*S.E.=-- --

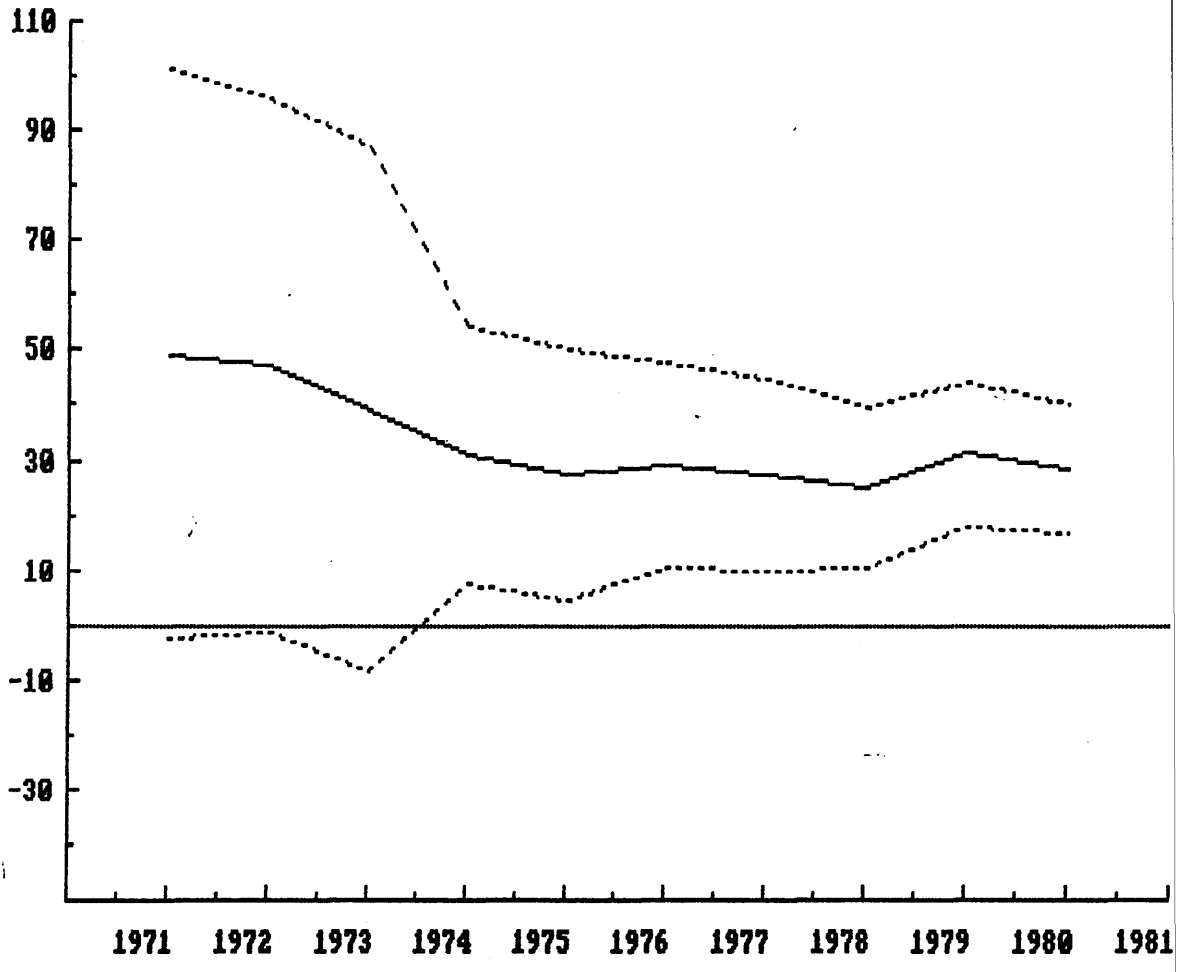


Figure 6.7 Chow Statistics [Equation 6.19a]

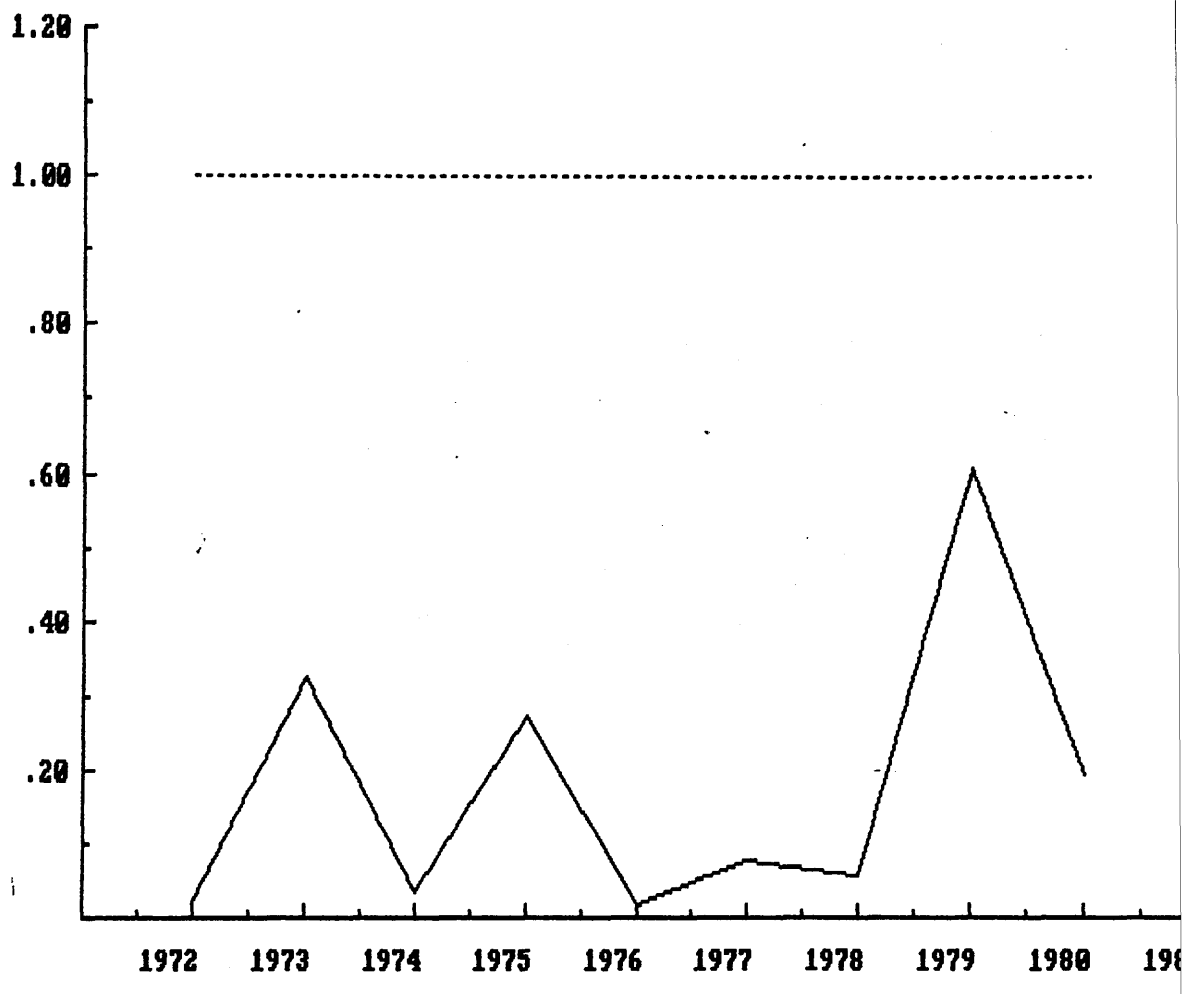


Figure 6.8 Parameter Stability Over Time [Equation 6.28a]
ILBW =____ ± 2*S.E.=— —

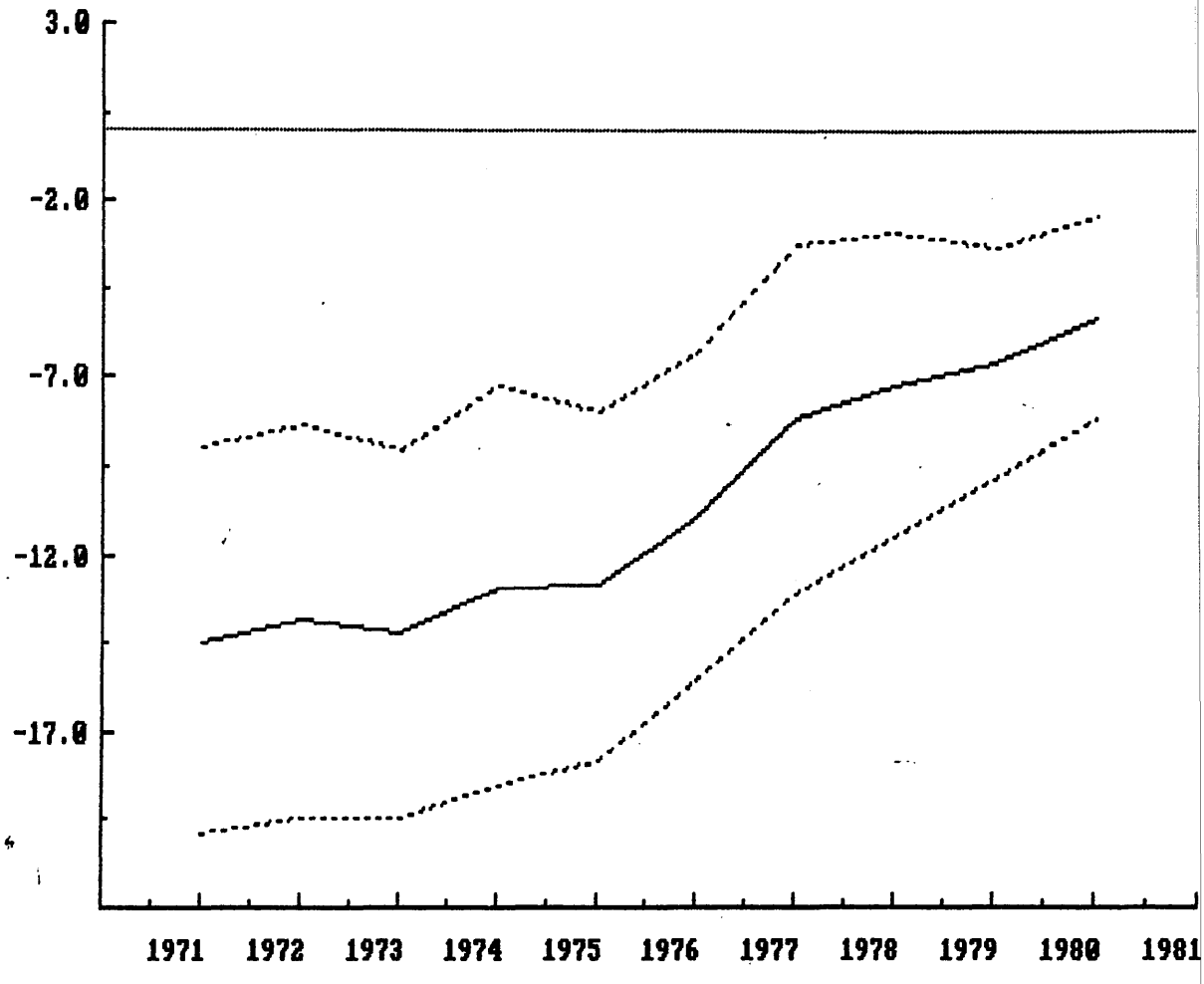


Figure 6.9 Parameter Stability Over Time [Equation 28a]

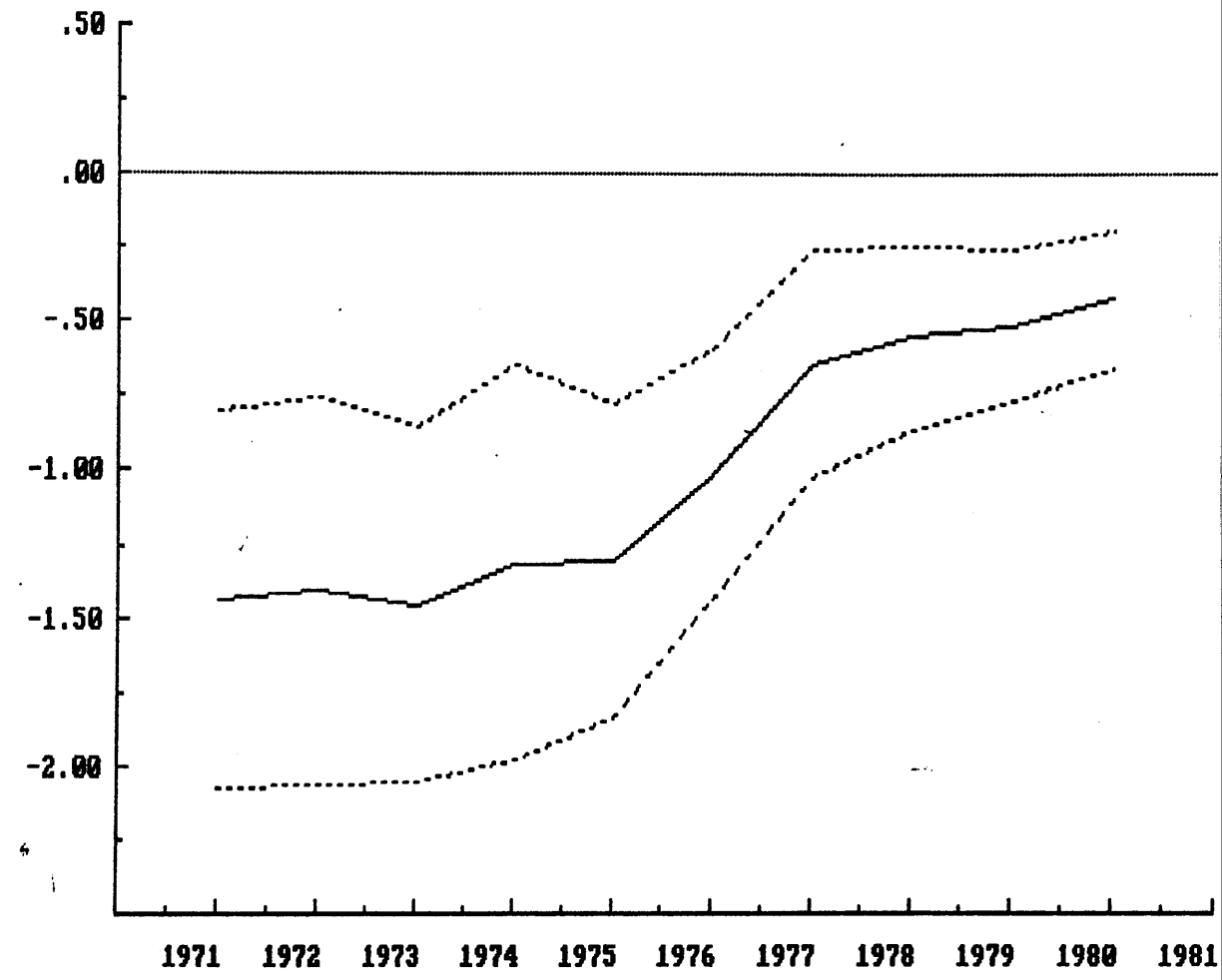


Figure 6.10 Parameter Stability Over Time [Equation 6.20a]
√(ILBW*EXPEND)____ ± 2*S.E.=-- --

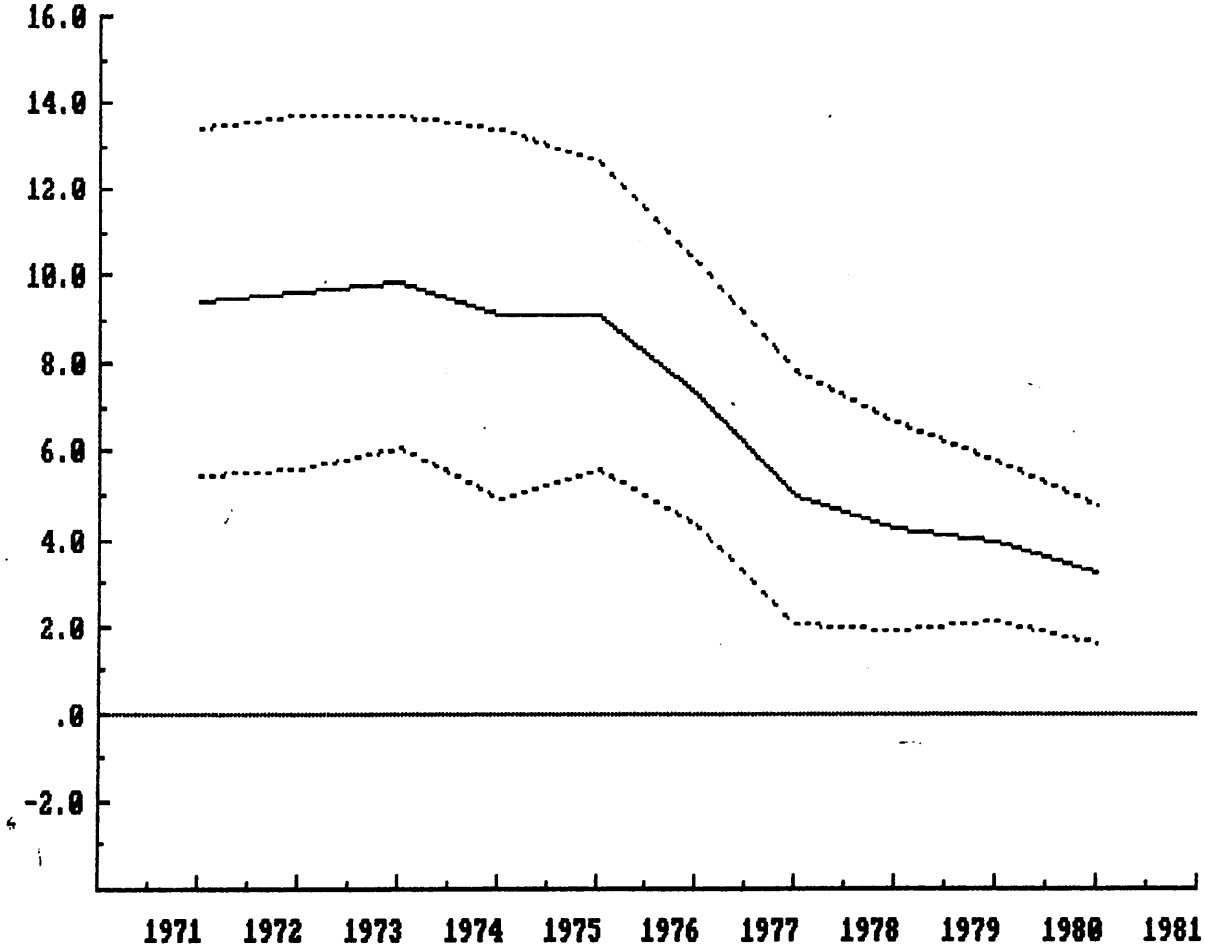


Figure 6.11 Chow Statistics [Equation 6.20a]

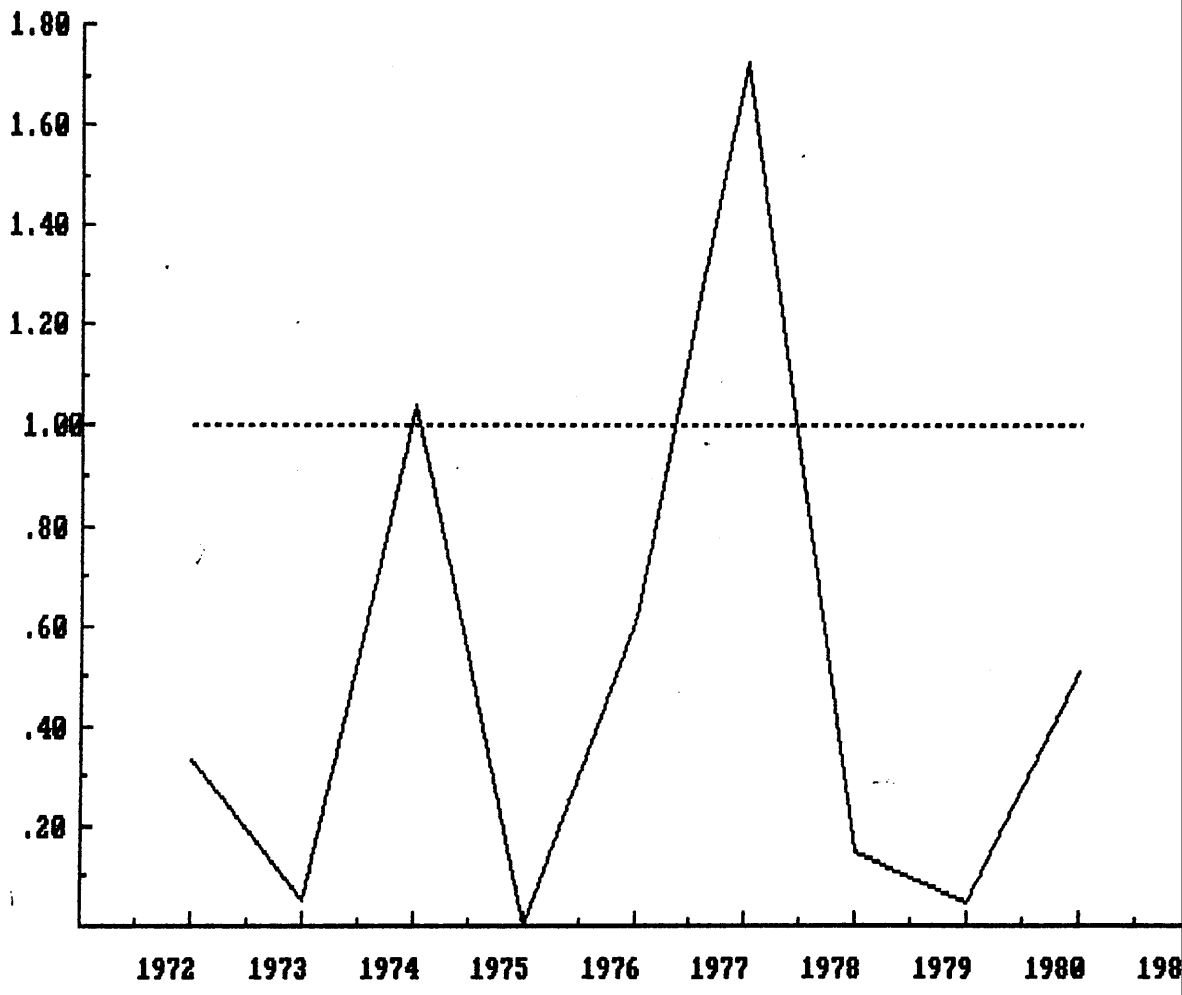


Figure 6.12 Parameter Stability Over Time [Equation 6.21a]

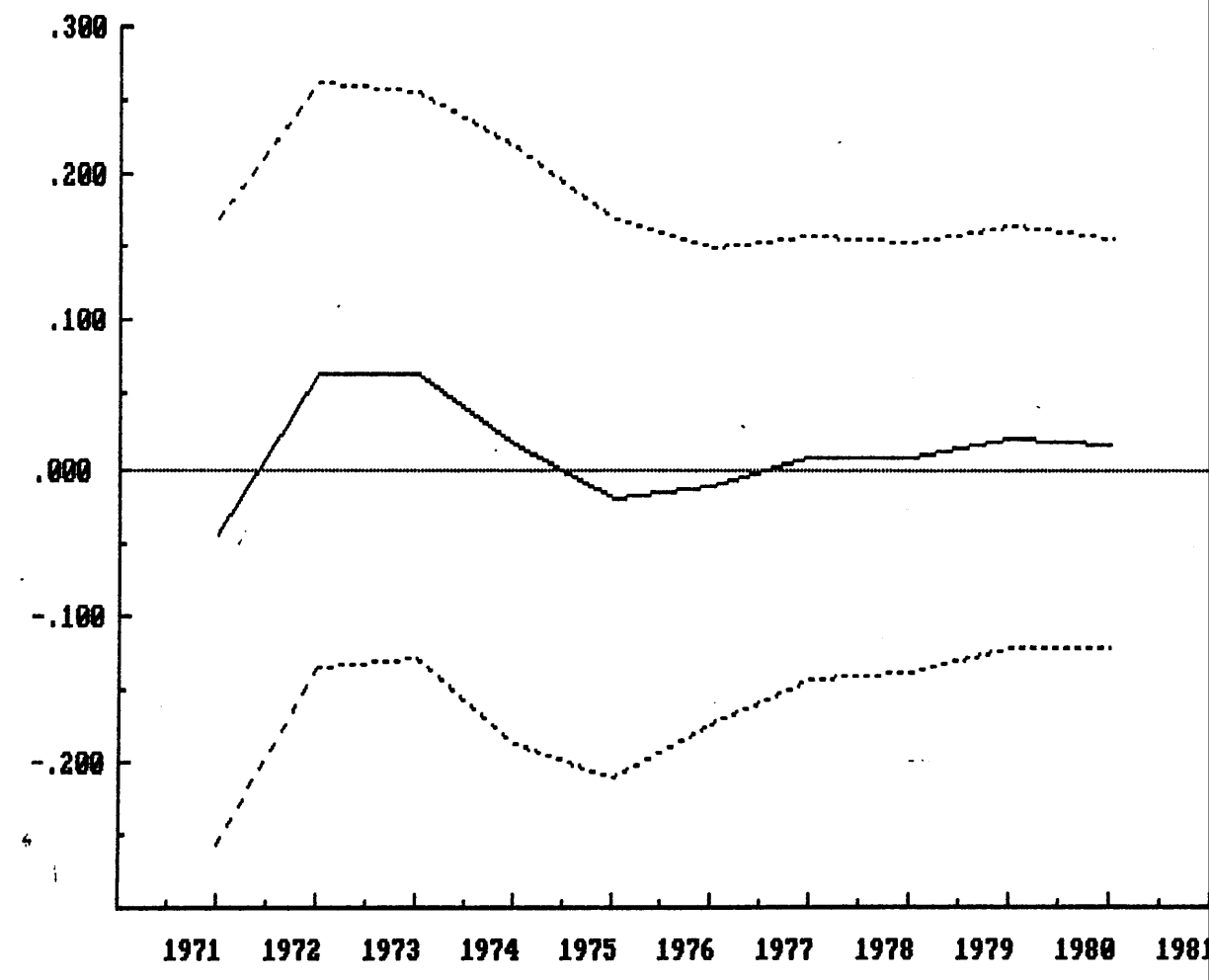


Figure 6.13 Parameter Stability Over Time [Equation 6.21a]

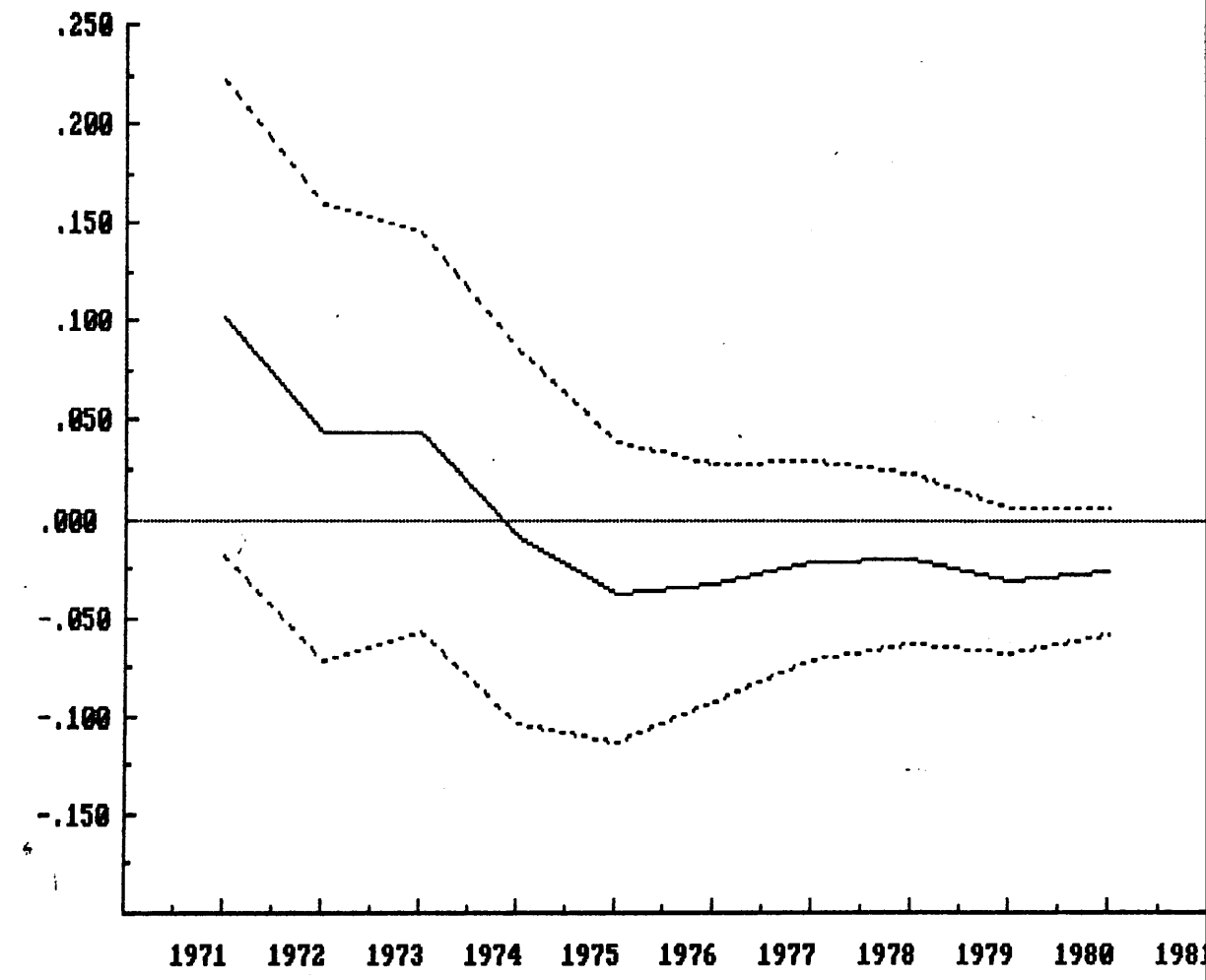


Figure 6.14 Parameter Stability Over Time [Equation 6.21a]

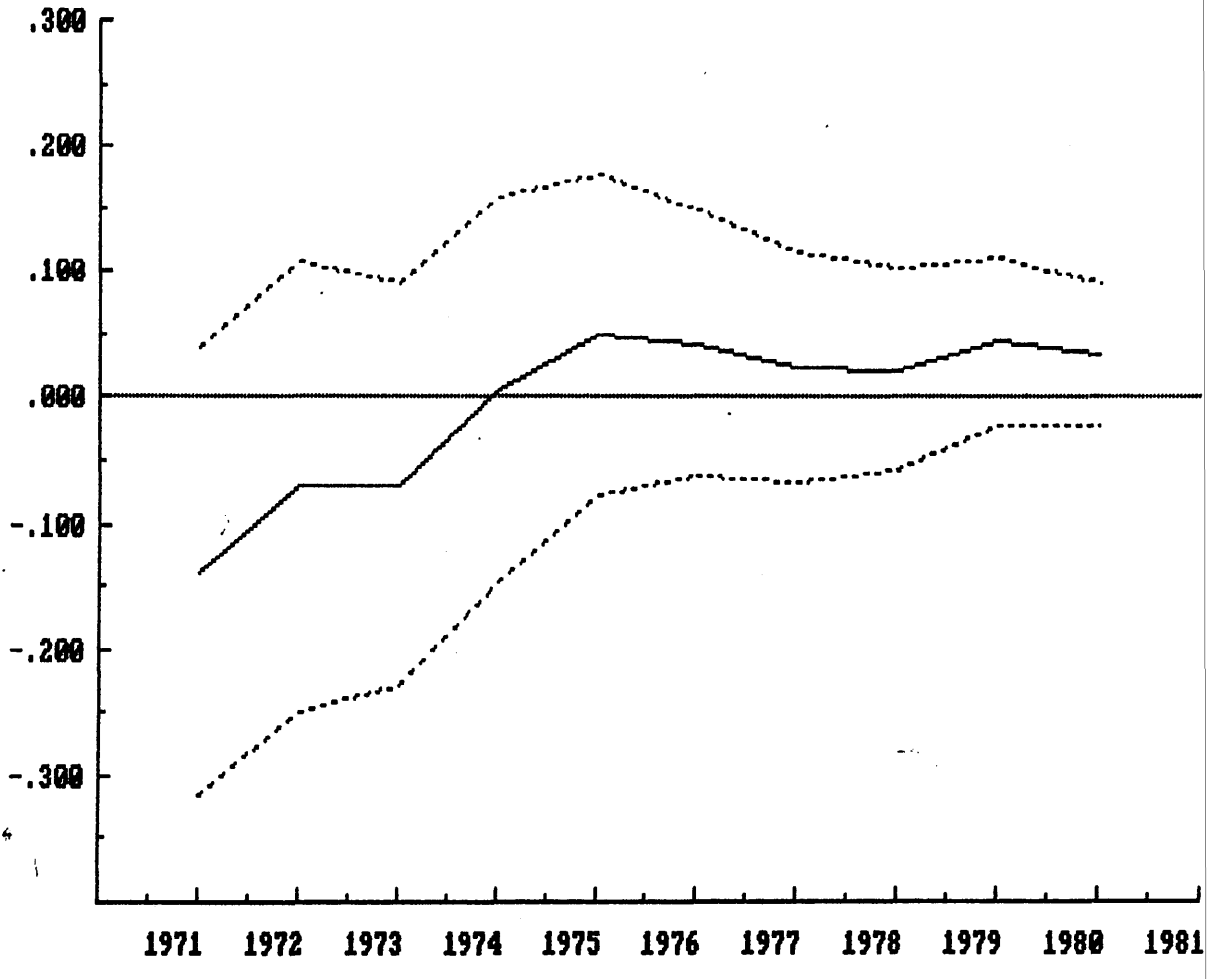


Figure 6.15 Chow Statistics [Equation 6.21a]*

1† CHOWs=____ 5.000%=-- --

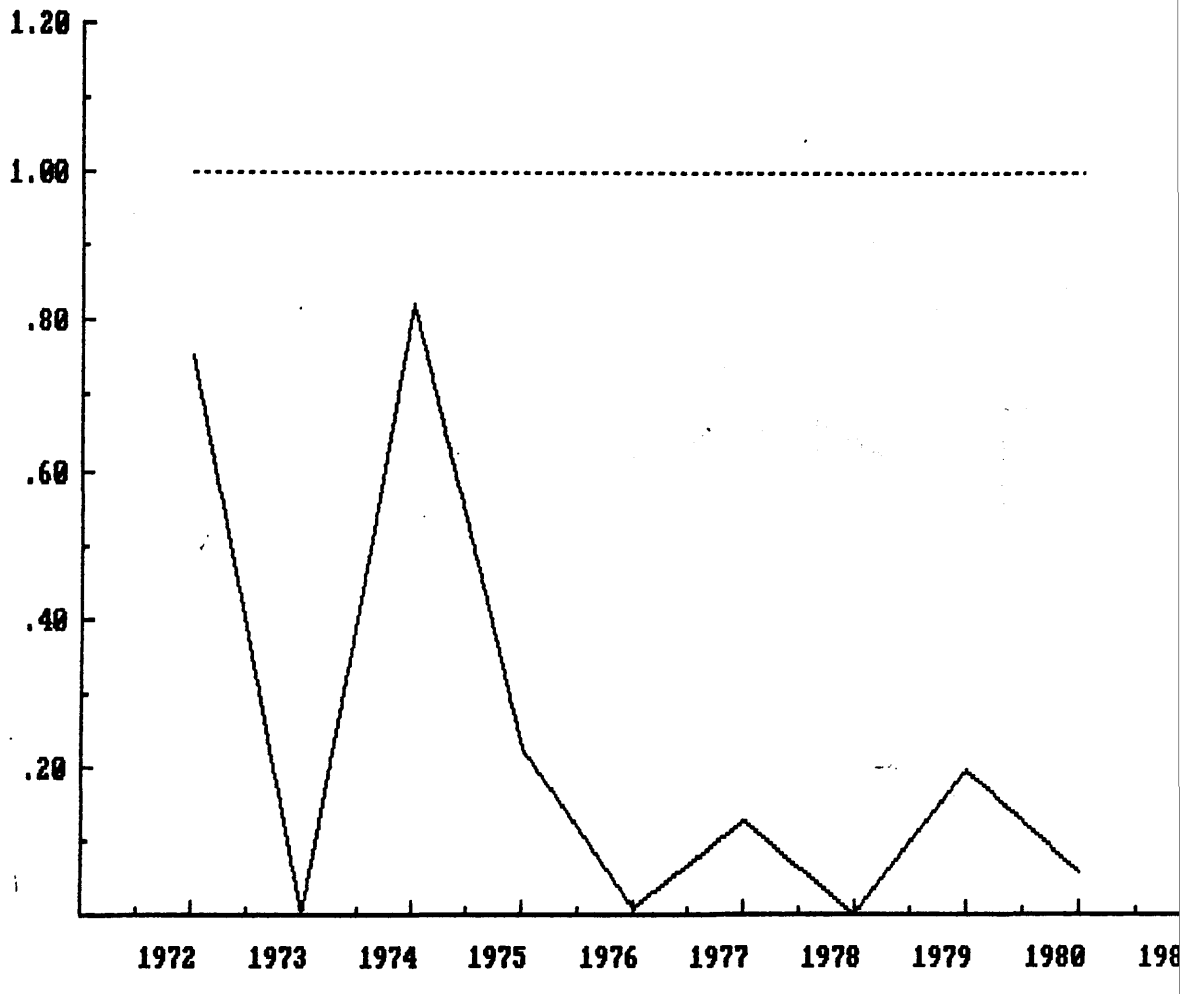


Figure 6.16 Forecast Performance [Equation 6.19a]

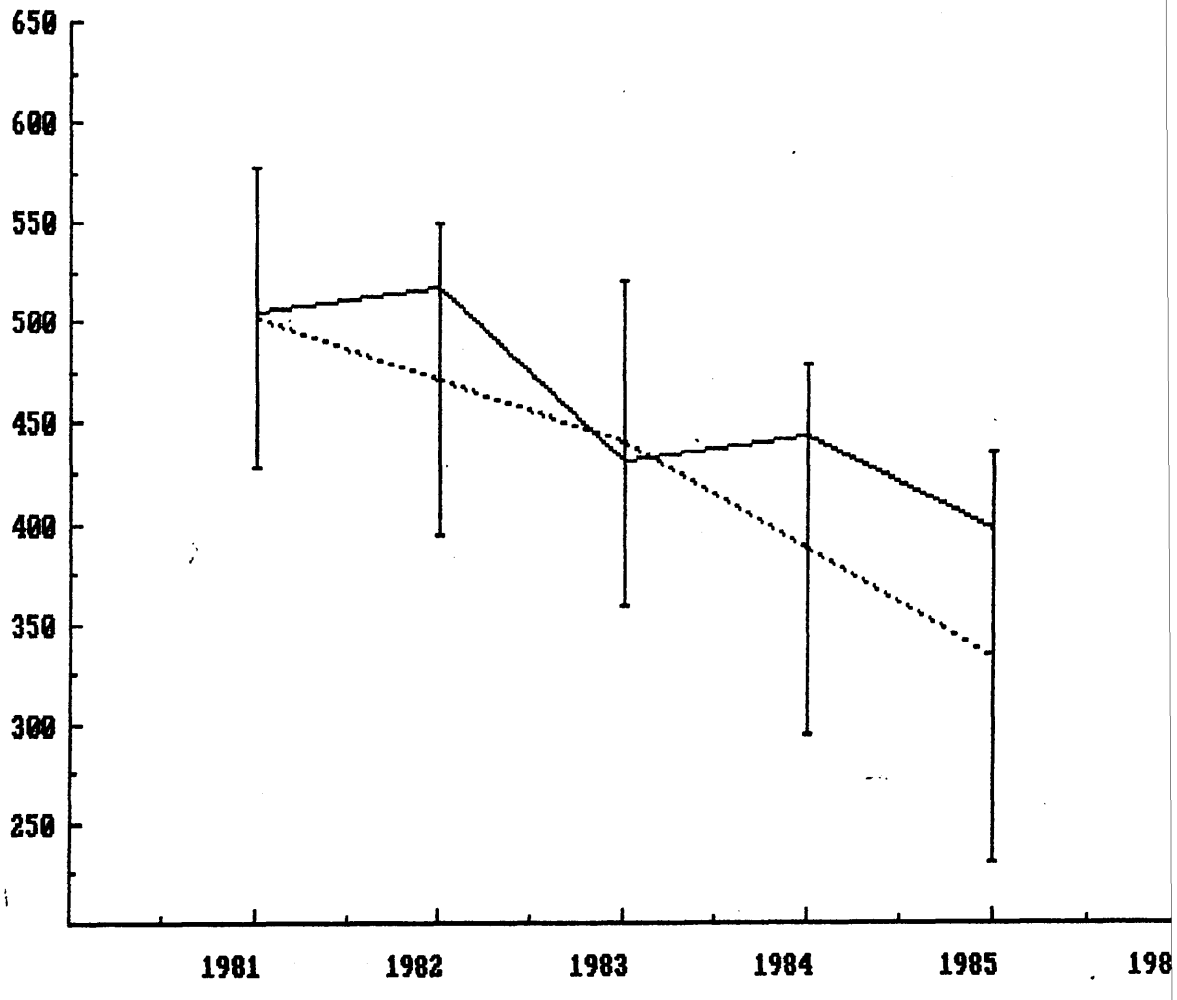


Figure 6.17 Forecast Performance [Equation 6.20a]
PMR(ilbw) =____ FORECAST=— —

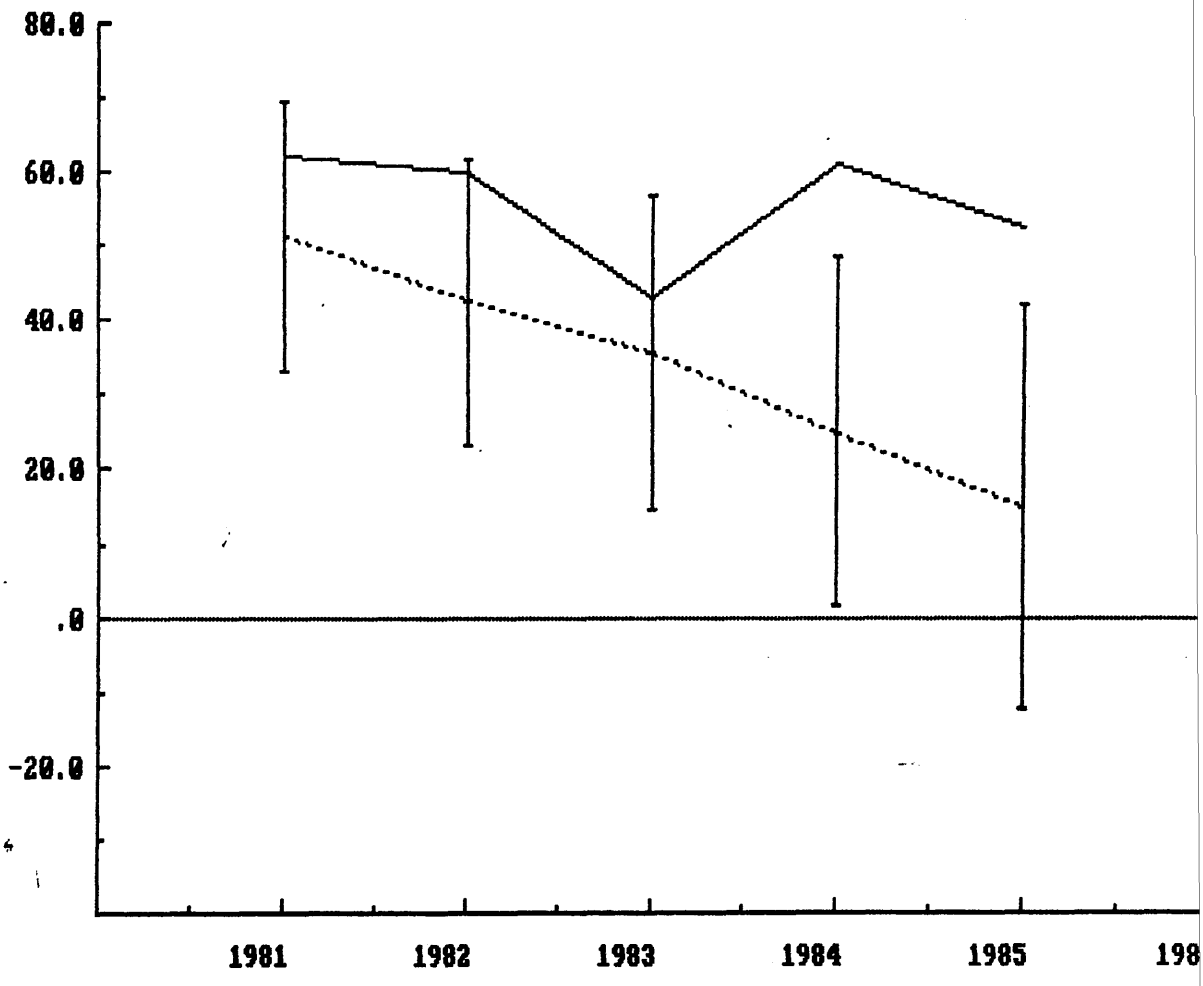


Figure 6.18 Forecast Performance [Equation 6.21a]

PNM(nbw)=____ FORECAST=— —

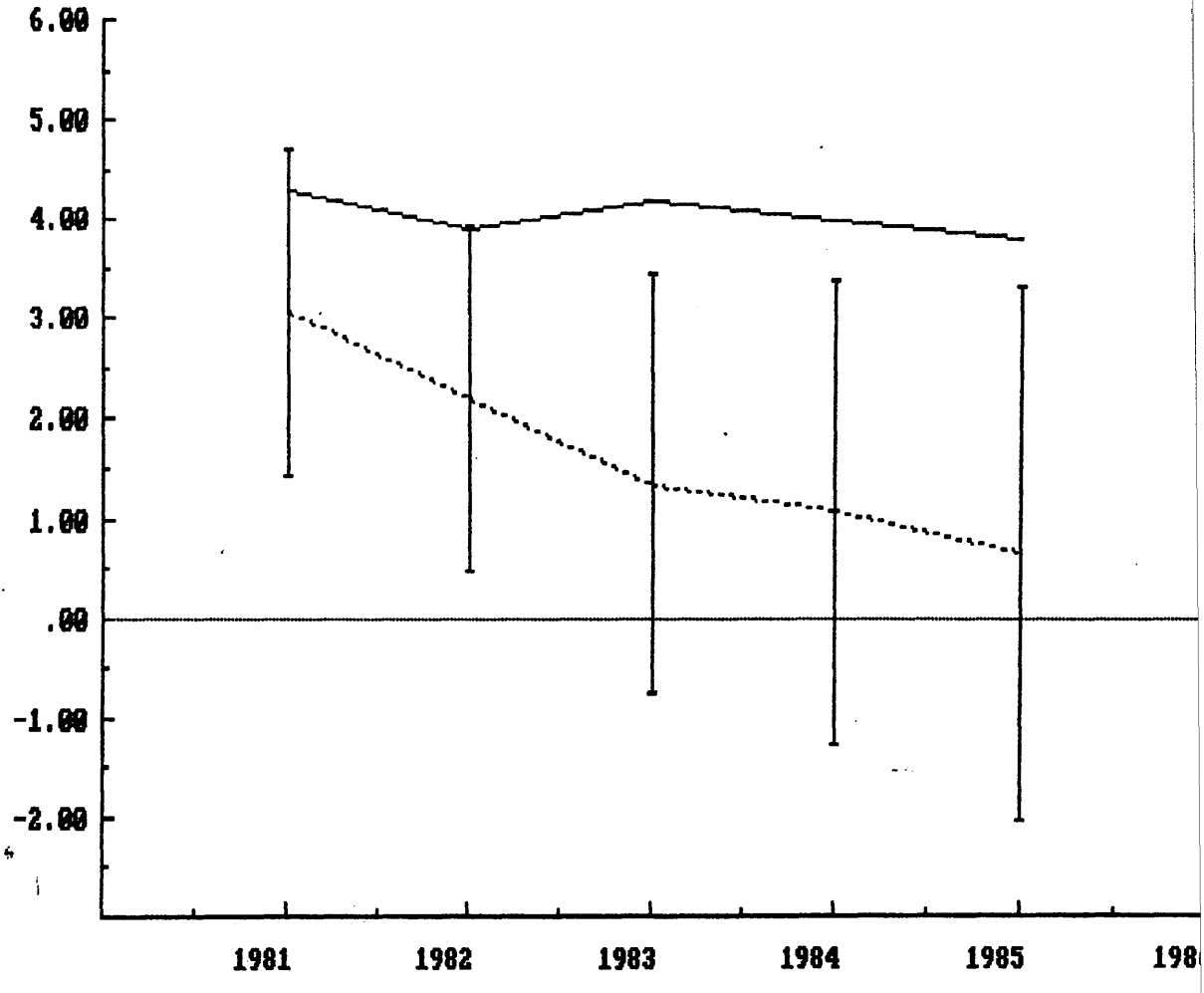


Figure 6.19 Forecast Performance [Equation 6.22a]
PNM(vlbw)=____ FORECAST=-- —

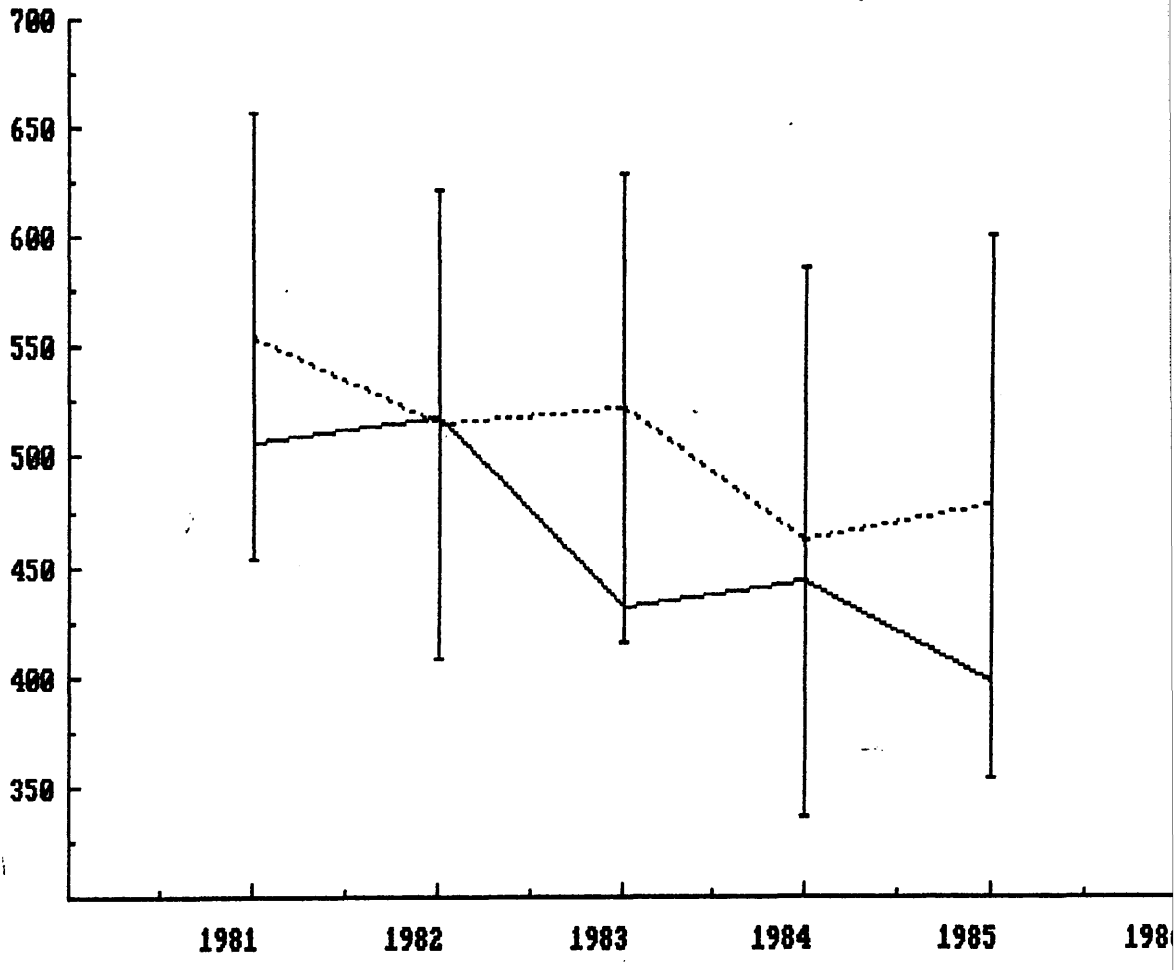


Figure 6.20 Forecast Performance [Equation 6.23a]
PNM(ilbw)=____ FORECAST=— —

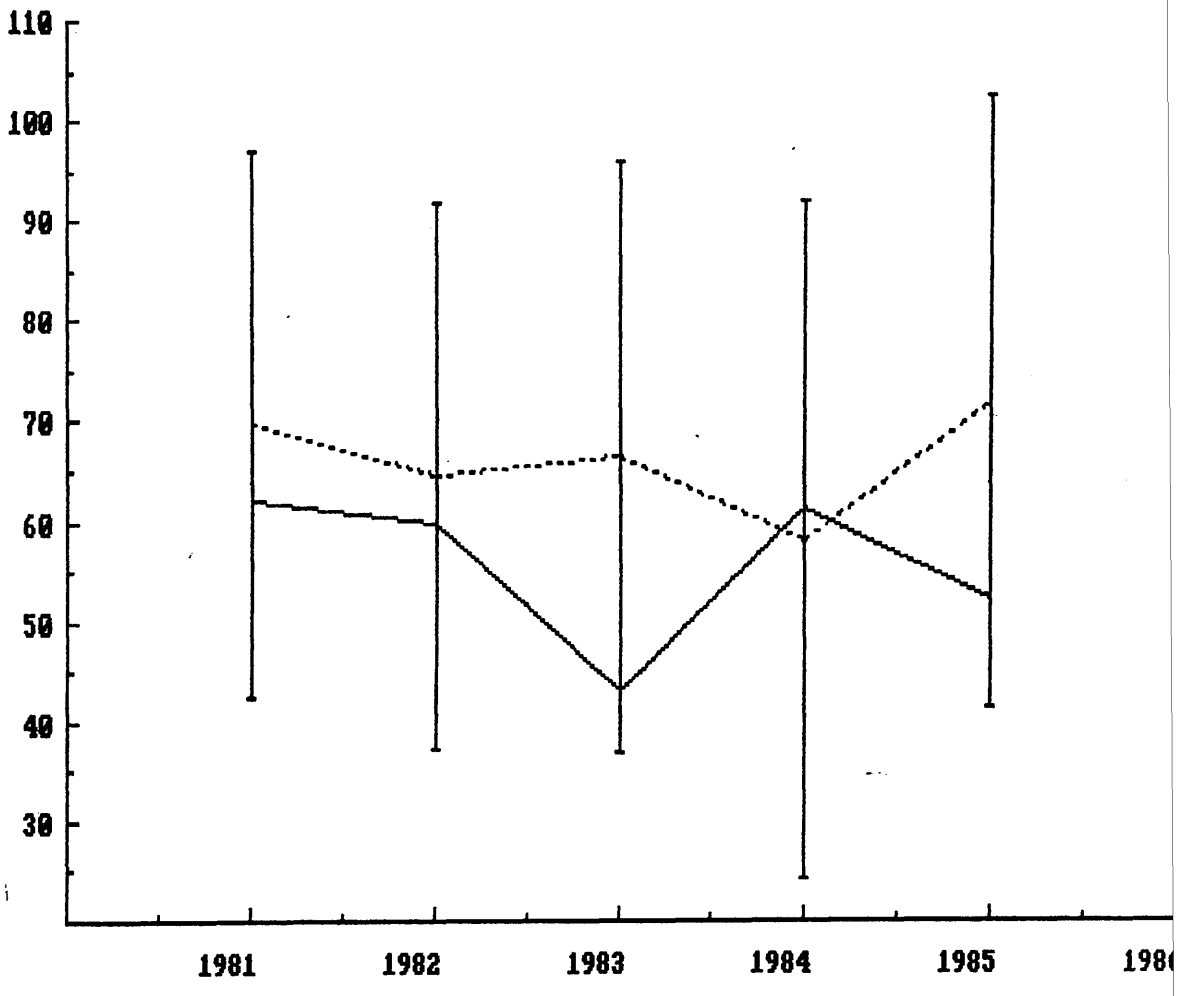


Figure 6.21 Forecast Performance [Equation 6.24a]
PMR(nbw)=____ FORECAST=— —

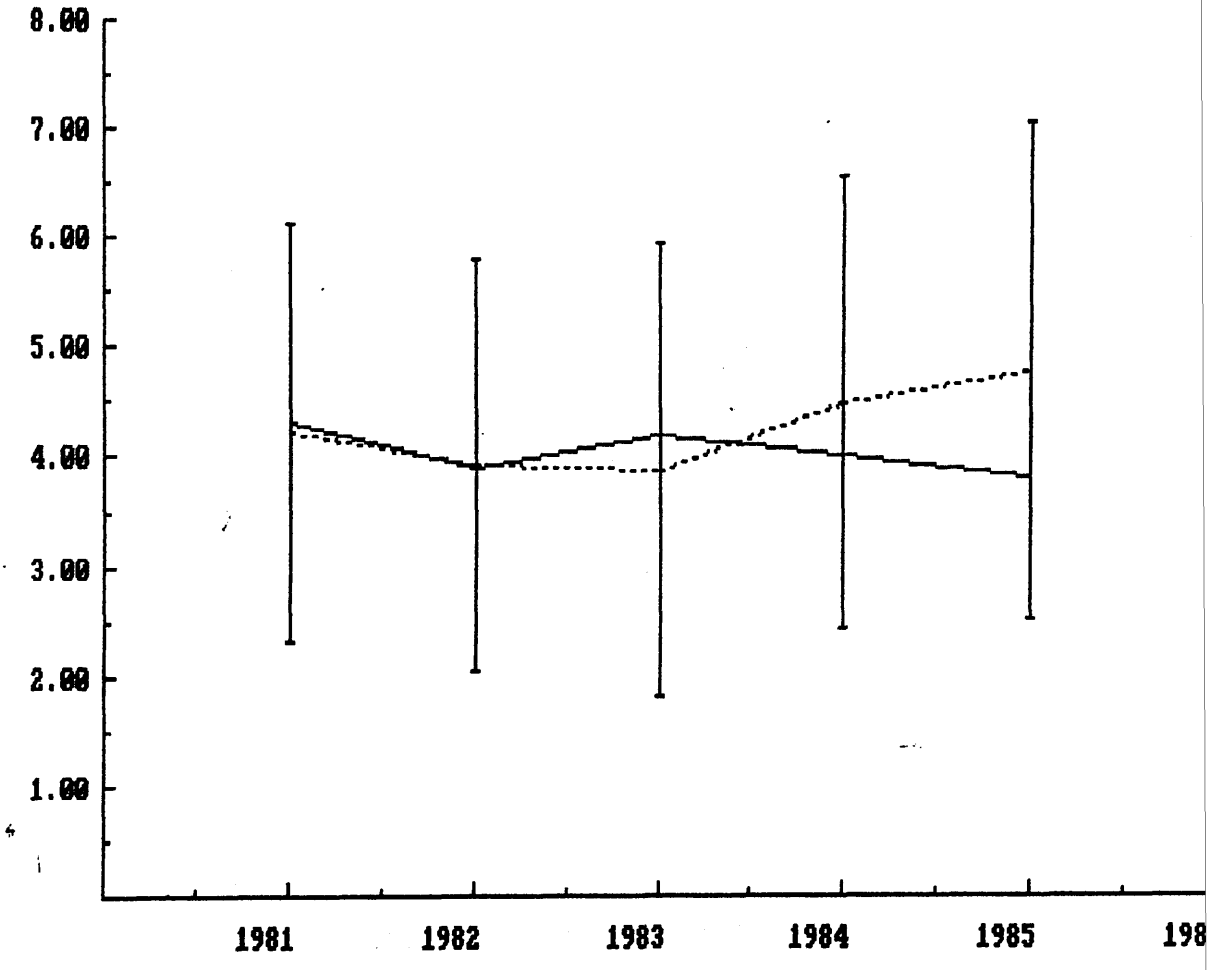


Figure 6.22 Actual and Fitted Values [Equation 6.19a]
PNM(vlbw) =_____ FITTED =— —

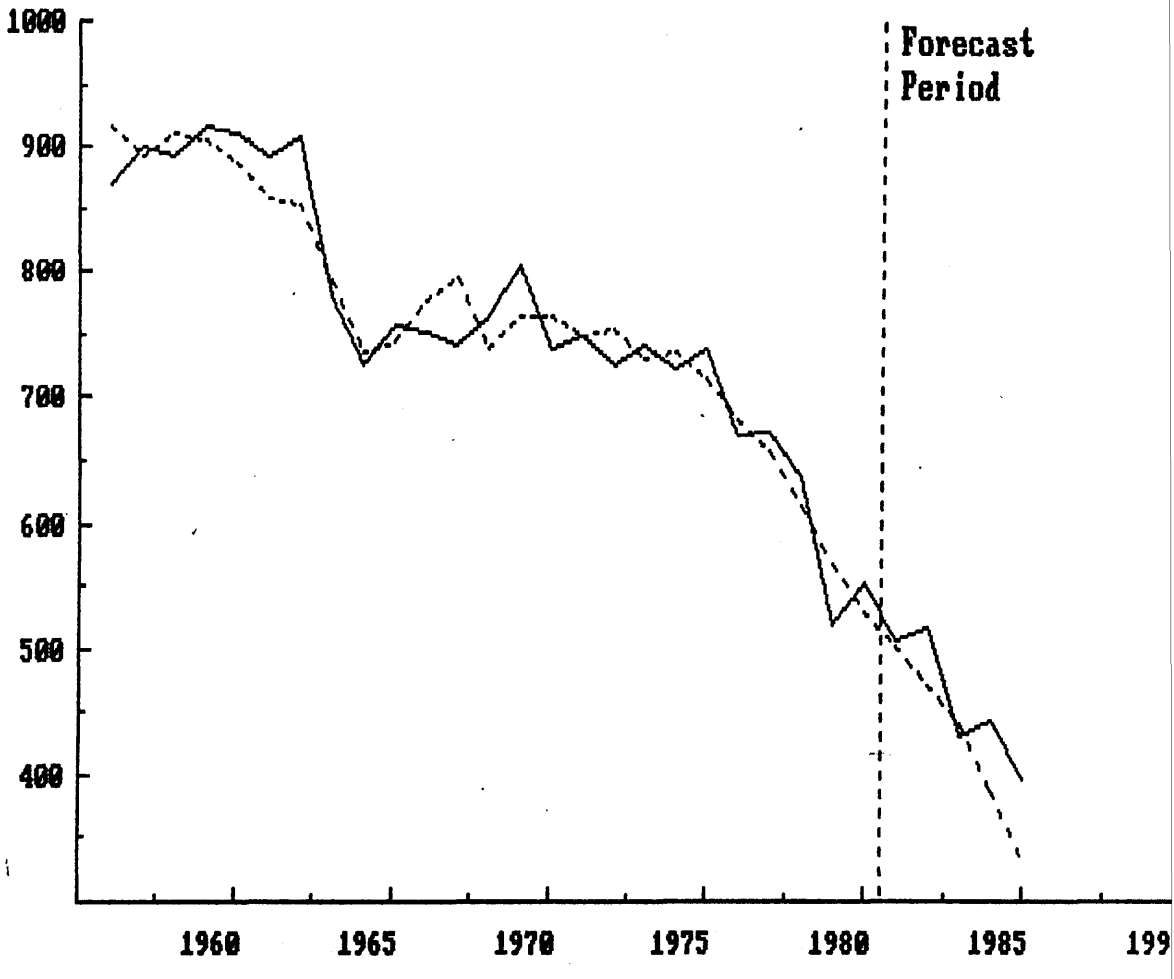


Figure 6.23 Actual and Fitted Values [Equation 6.20a]
PMR(ilbw) =_____ FITTED =-- --

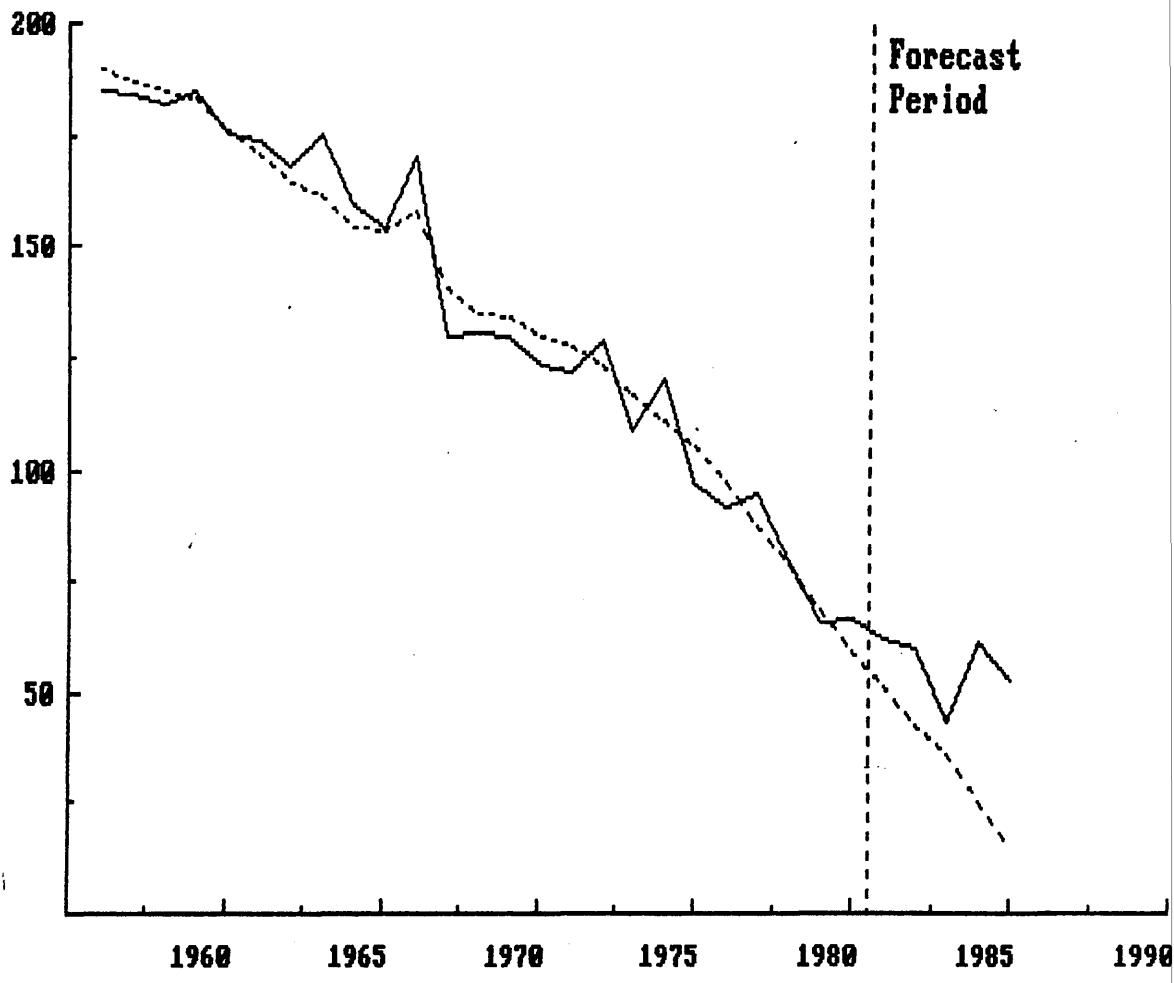


Figure 6.24 Actual and Fitted Values [Equation 6.21a]

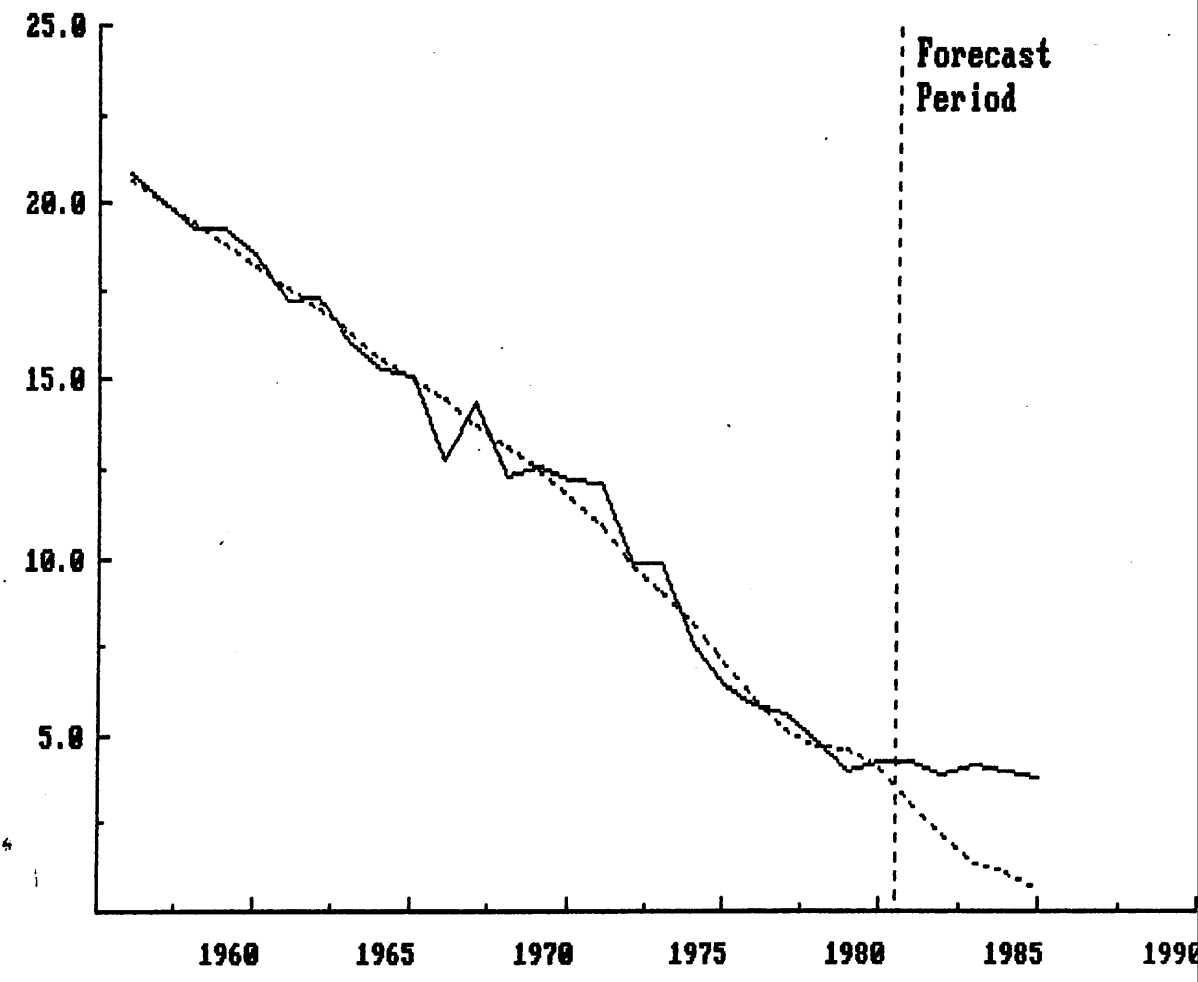


Figure 6.25 Actual and Fitted Values [Equation 6.22a]

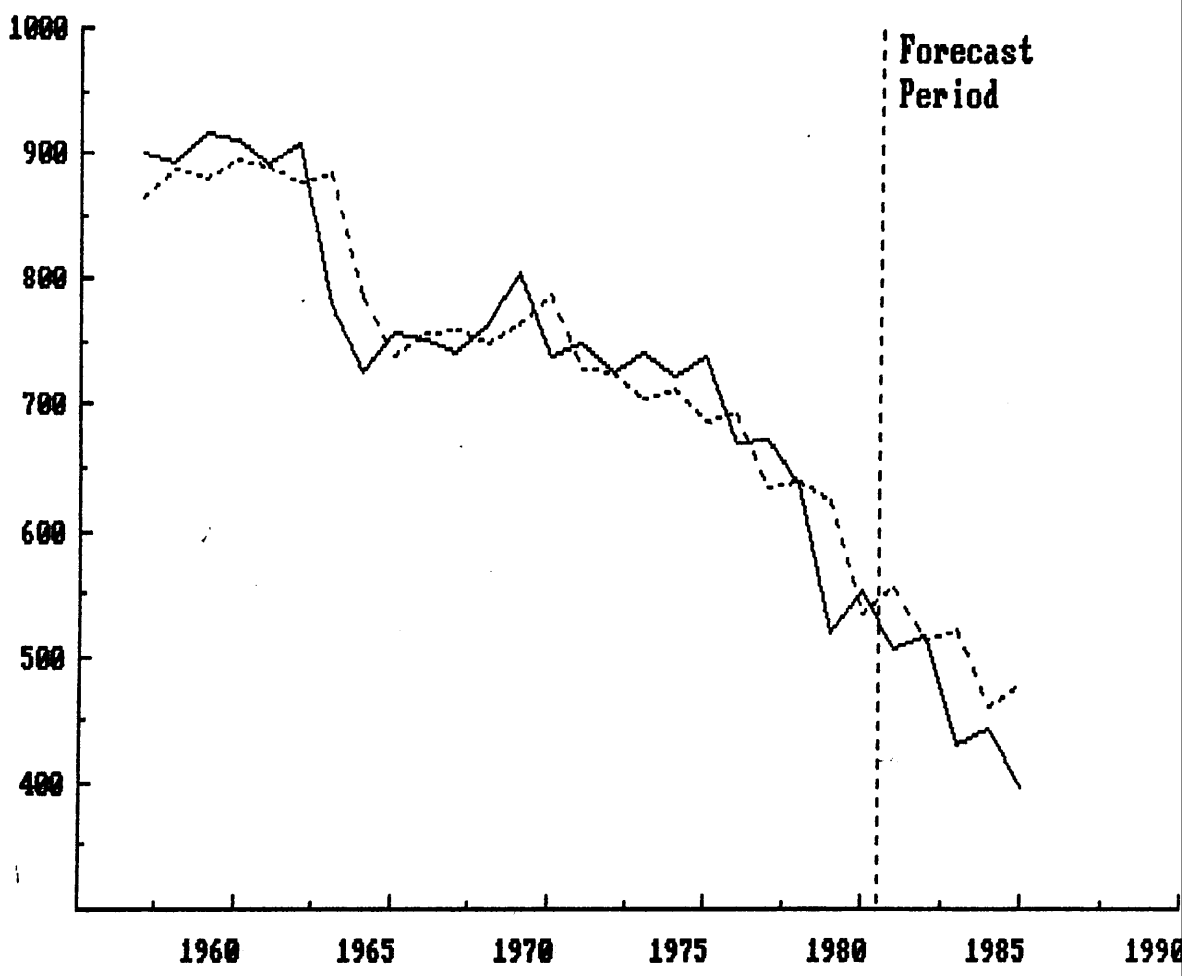


Figure 6.26 Actual and Fitted Values [Equation 6.23a]

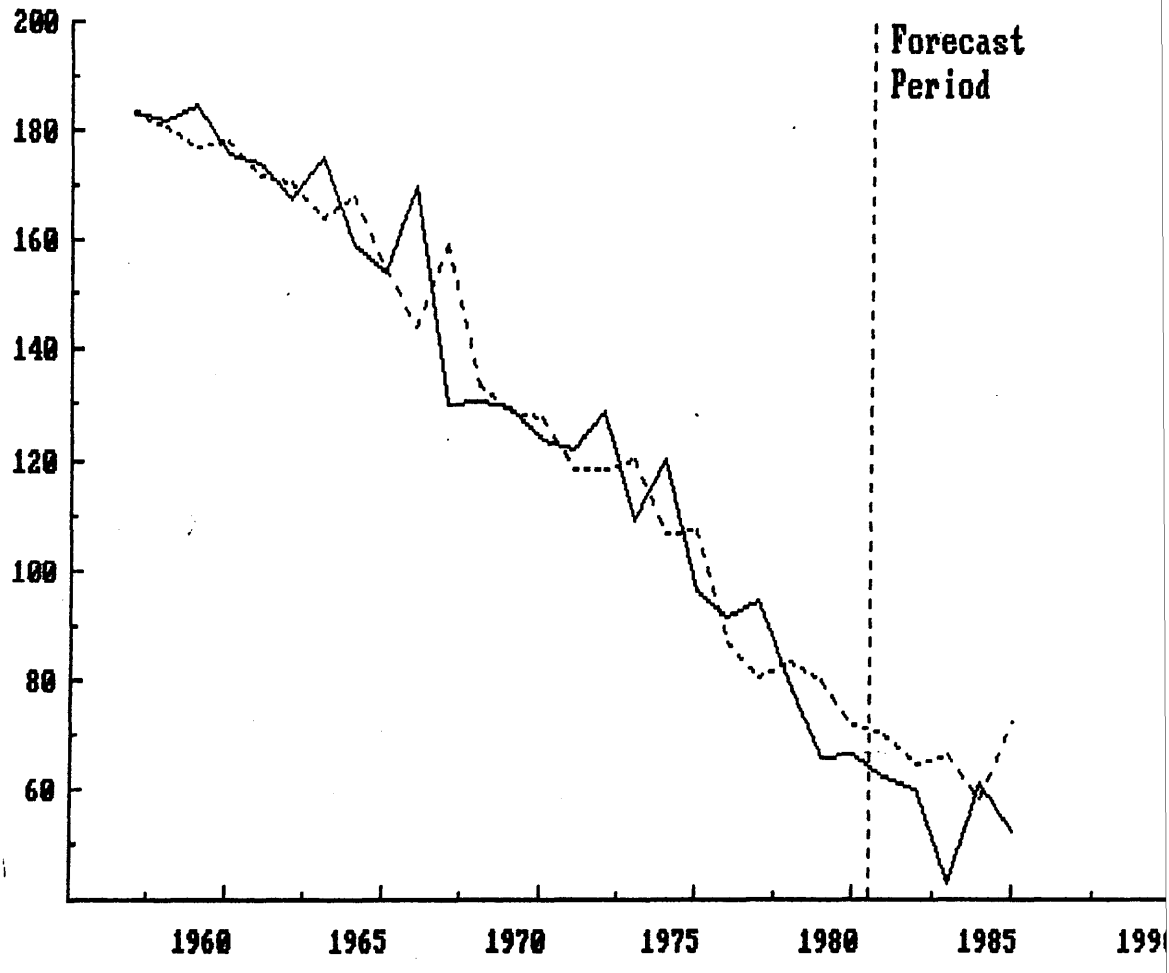


Figure 6.27 Actual and Fitted Values [Equation 6.24a]

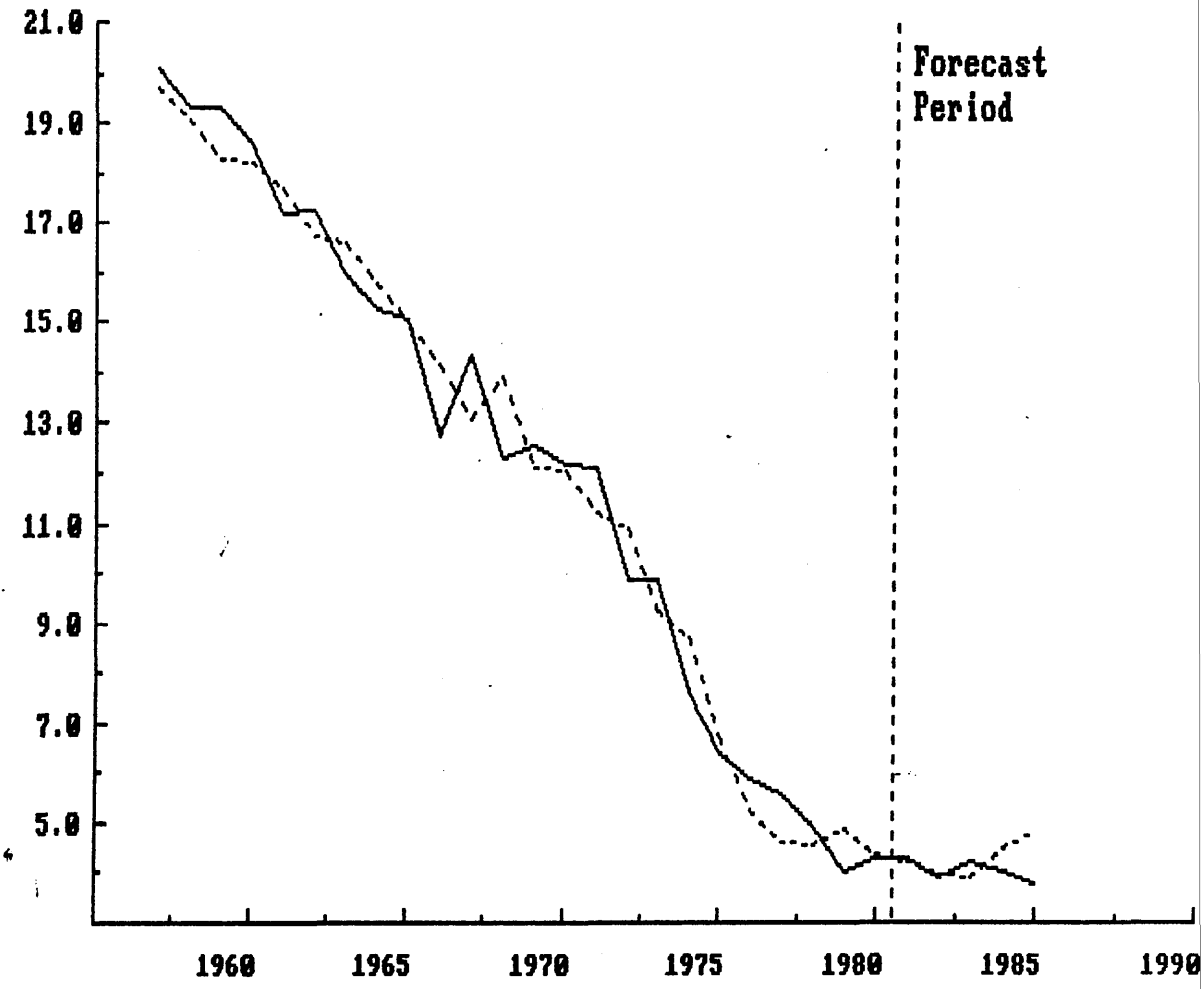


TABLE A6.1 OUTPUT VARIABLES USED IN PRODUCTION FUNCTION ESTIMATES

| | VARIABLE | | | | |
|---|---|---|---|---|---|
| YEAR | PMR | PMR(vlbw) | PMR(ilbw) | PMR(nbw) | FMR |
| 1956 | 42.5 | 870.8 | 184.5 | 20.8 | 23.9 |
| 1957 | 42.8 | 899.7 | 183.8 | 20.1 | 23.7 |
| 1958 | 41.1 | 893.1 | 181.6 | 19.3 | 22.8 |
| 1959 | 41.1 | 915.3 | 184.8 | 19.3 | 22.2 |
| 1960 | 39.6 | 911.2 | 175.8 | 18.6 | 21.7 |
| 1961 | 38.3 | 893.5 | 174.3 | 17.2 | 20.8 |
| 1962 | 37.5 | 908.6 | 168.0 | 17.3 | 19.9 |
| 1963 | 35.6 | 781.1 | 175.5 | 16.0 | 19.1 |
| 1964 | 34.0 | 726.9 | 159.4 | 15.3 | 17.9 |
| 1965 | 33.5 | 756.1 | 154.5 | 15.1 | 17.9 |
| 1966 | 31.2 | 752.9 | 170.3 | 12.8 | 16.2 |
| 1967 | 29.4 | 742.6 | 130.3 | 14.4 | 15.8 |
| 1968 | 27.9 | 765.9 | 130.9 | 12.3 | 14.8 |
| 1969 | 27.3 | 804.8 | 129.7 | 12.6 | 14.0 |
| 1970 | 26.5 | 738.8 | 123.7 | 12.2 | 13.9 |
| 1971 | 26.4 | 749.0 | 121.8 | 12.1 | 13.1 |
| 1972 | 25.5 | 726.0 | 129.2 | 9.9 | 13.2 |
| 1973 | 24.1 | 743.2 | 109.3 | 9.9 | 11.6 |
| 1974 | 24.6 | 724.0 | 120.5 | 7.6 | 12.0 |
| 1975 | 22.8 | 740.0 | 96.5 | 6.4 | 11.1 |
| 1976 | 19.8 | 670.0 | 91.6 | 5.9 | 9.6 |
| 1977 | 20.0 | 672.2 | 94.6 | 5.6 | 8.8 |
| 1978 | 16.8 | 636.1 | 79.2 | 4.9 | 8.1 |
| 1979 | 15.5 | 518.9 | 65.7 | 4.0 | 6.9 |
| 1980 | 14.4 | 552.0 | 66.6 | 4.3 | 6.7 |
| 1981 | 13.1 | 505.9 | 62.2 | 4.3 | 6.3 |
| 1982 | 12.9 | 518.3 | 59.9 | 3.9 | 5.8 |
| 1983 | 11.6 | 431.2 | 43.1 | 4.2 | 5.8 |
| 1984 | 12.1 | 443.7 | 61.4 | 4.0 | 5.8 |
| 1985 | 10.9 | 397.7 | 52.6 | 3.8 | 5.5 |

NOTES: Variables are defined in Table 6.1 and in text.

TABLE A6.1 (continued) OUTPUT VARIABLES USED IN PRODUCTION FUNCTION ESTIMATES

| | VARIABLE | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| YEAR | NMR | VENMR | ENMR | LNMR | MAL | PREM | ASPH | OTHER |
| 1956 | 19.1 | 8.9 | 7.4 | 2.8 | 3.4 | 4.3 | 5.0 | 6.4 |
| 1957 | 19.6 | 9.3 | 7.4 | 2.9 | 3.7 | 4.6 | 5.2 | 6.1 |
| 1958 | 18.7 | 8.9 | 7.3 | 2.5 | 3.1 | 4.3 | 5.6 | 5.8 |
| 1959 | 19.4 | 10.0 | 6.6 | 2.8 | 3.7 | 4.2 | 5.6 | 5.8 |
| 1960 | 18.2 | 9.3 | 6.5 | 2.4 | 3.3 | 3.6 | 5.2 | 6.1 |
| 1961 | 18.0 | 9.2 | 6.4 | 2.4 | 3.9 | 3.7 | 5.3 | 5.0 |
| 1962 | 17.9 | 9.3 | 5.9 | 2.7 | 3.8 | 3.5 | 5.0 | 5.7 |
| 1963 | 16.9 | 8.6 | 6.0 | 2.3 | 3.4 | 3.3 | 4.8 | 5.4 |
| 1964 | 16.4 | 8.6 | 5.9 | 1.9 | 3.4 | 2.9 | 4.6 | 5.5 |
| 1965 | 15.9 | 8.3 | 5.5 | 2.1 | 3.5 | 2.9 | 3.5 | 6.0 |
| 1966 | 15.2 | 7.9 | 5.4 | 1.9 | 3.2 | 2.6 | 3.4 | 6.0 |
| 1967 | 13.9 | 6.9 | 5.0 | 2.0 | 3.4 | 2.1 | 3.4 | 4.9 |
| 1968 | 13.4 | 6.8 | 4.5 | 2.1 | 3.0 | 2.5 | 3.5 | 4.3 |
| 1969 | 13.5 | 6.9 | 4.5 | 2.1 | 2.9 | 2.6 | 3.3 | 4.7 |
| 1970 | 12.8 | 6.7 | 4.4 | 1.7 | 3.2 | 2.0 | 3.6 | 4.1 |
| 1971 | 13.5 | 6.8 | 4.7 | 2.0 | 3.7 | 2.1 | 3.7 | 4.0 |
| 1972 | 12.5 | 6.3 | 4.4 | 1.8 | 3.1 | 1.6 | 3.7 | 4.0 |
| 1973 | 12.7 | 6.5 | 4.5 | 1.7 | 3.4 | 1.8 | 3.7 | 3.8 |
| 1974 | 12.8 | 6.0 | 4.9 | 1.9 | 3.4 | 1.8 | 3.6 | 4.0 |
| 1975 | 11.7 | 5.8 | 4.2 | 1.7 | 3.0 | 1.7 | 3.7 | 3.4 |
| 1976 | 10.3 | 5.0 | 3.8 | 1.5 | 2.7 | 1.2 | 3.0 | 3.3 |
| 1977 | 11.2 | 5.6 | 3.9 | 1.7 | 3.4 | 1.2 | 3.2 | 3.5 |
| 1978 | 8.8 | 4.1 | 3.3 | 1.4 | 2.6 | 0.9 | 2.8 | 2.4 |
| 1979 | 8.7 | 4.2 | 3.1 | 1.4 | 2.6 | 0.9 | 3.0 | 2.2 |
| 1980 | 10.8 | 6.9 | 2.6 | 1.3 | 2.3 | 0.9 | 2.7 | 1.9 |
| 1981 | 6.9 | 3.0 | 2.4 | 1.5 | 2.1 | 0.7 | 2.3 | 1.8 |
| 1982 | 7.1 | 3.1 | 2.6 | 1.4 | 2.0 | 0.8 | 2.2 | 2.1 |
| 1983 | 5.9 | 2.6 | 2.3 | 1.0 | 2.0 | 0.7 | 1.9 | 1.2 |
| 1984 | 6.4 | 2.8 | 2.4 | 1.2 | 1.9 | 0.5 | 2.1 | 1.9 |
| 1985 | 5.5 | 2.5 | 1.9 | 1.1 | 1.8 | 0.5 | 1.6 | 1.5 |

NOTES: Variables are defined in Table 6.1 and in text.

TABLE A6.2 INPUT VARIABLES USED IN PRODUCTION FUNCTION ESTIMATES

| | VARIABLE | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| YEAR | LBW | VLBW | ILBW | NBW | OBS | NUR | BED | EXPEND |
| 1956 | 78.3 | 12.8 | 65.5 | 921.7 | 1.5 | 21.3 | 29.5 | 259.0 |
| 1957 | 79.8 | 13.2 | 66.6 | 920.2 | 1.6 | 22.6 | 29.1 | 279.9 |
| 1958 | 78.5 | 12.7 | 65.8 | 921.5 | 1.5 | 22.8 | 29.1 | 295.2 |
| 1959 | 76.8 | 12.5 | 64.3 | 923.2 | 1.6 | 24.9 | 29.3 | 308.7 |
| 1960 | 76.4 | 12.3 | 64.1 | 923.6 | 1.6 | 24.8 | 27.4 | 314.2 |
| 1961 | 76.5 | 12.5 | 64.0 | 923.5 | 1.5 | 25.5 | 28.0 | 325.4 |
| 1962 | 75.7 | 12.0 | 63.7 | 924.3 | 1.6 | 25.7 | 27.9 | 331.4 |
| 1963 | 75.1 | 12.9 | 62.2 | 924.9 | 1.7 | 25.5 | 27.3 | 345.1 |
| 1964 | 76.4 | 13.6 | 62.8 | 923.6 | 1.8 | 27.2 | 28.3 | 353.9 |
| 1965 | 74.0 | 13.3 | 60.7 | 926.0 | 1.9 | 28.5 | 28.5 | 376.8 |
| 1966 | 69.2 | 12.9 | 56.3 | 930.8 | 2.1 | 29.9 | 30.1 | 420.1 |
| 1967 | 71.0 | 10.9 | 60.1 | 929.0 | 2.2 | 29.8 | 29.6 | 396.0 |
| 1968 | 70.1 | 11.6 | 58.5 | 929.9 | 2.3 | 30.8 | 29.9 | 401.6 |
| 1969 | 65.9 | 10.6 | 55.3 | 934.1 | 2.5 | 32.7 | 31.8 | 429.4 |
| 1970 | 69.4 | 10.8 | 58.6 | 930.6 | 2.8 | 35.4 | 33.6 | 480.8 |
| 1971 | 66.7 | 11.1 | 55.6 | 933.3 | 2.9 | 36.3 | 34.9 | 522.3 |
| 1972 | 73.2 | 11.3 | 61.9 | 926.8 | 3.2 | 39.2 | 41.0 | 597.5 |
| 1973 | 71.9 | 11.3 | 60.6 | 928.1 | 3.7 | 42.7 | 43.1 | 619.5 |
| 1974 | 71.2 | 8.6 | 62.6 | 928.8 | 3.8 | 45.5 | 45.3 | 671.7 |
| 1975 | 70.6 | 9.8 | 60.8 | 929.4 | 4.2 | 47.6 | 47.0 | 732.5 |
| 1976 | 67.8 | 8.8 | 59.0 | 932.2 | 4.2 | 50.1 | 49.2 | 788.6 |
| 1977 | 66.7 | 9.4 | 57.3 | 933.3 | 4.3 | 49.7 | 49.7 | 820.0 |
| 1978 | 65.6 | 8.8 | 56.8 | 934.4 | 4.2 | 47.0 | 44.0 | 794.4 |
| 1979 | 65.9 | 9.0 | 56.9 | 934.1 | 3.9 | 44.1 | 37.9 | 714.4 |
| 1980 | 64.7 | 9.3 | 55.4 | 935.3 | 4.0 | 45.1 | 36.0 | 698.6 |
| 1981 | 65.3 | 9.3 | 56.0 | 934.7 | 3.8 | 43.5 | 35.4 | 740.9 |
| 1982 | 64.9 | 9.3 | 55.6 | 935.1 | 4.0 | 45.4 | 38.4 | 768.6 |
| 1983 | 68.8 | 10.0 | 58.8 | 931.2 | 4.3 | 48.6 | 40.8 | 775.6 |
| 1984 | 67.8 | 11.0 | 56.8 | 932.2 | 4.2 | 40.6 | 38.2 | 721.6 |
| 1985 | 67.9 | 11.1 | 56.8 | 932.1 | 4.5 | 39.0 | 35.0 | 676.8 |

NOTES: Variables are defined in Table 6.2 and in text.

# CHAPTER 7 ECONOMIC INSTABILITY, UNEMPLOYMENT AND INFANT HEALTH

## 7.1 Introduction

Over the past decade there has occurred a virtual explosion in studies examining the impact of economic factors on health. Although this topic had attracted the attention of researchers for well over a century the initial spark which re-kindled the most recent surge of interest was struck by the work of Harvey Brenner (1973, 1979) who published a series of controversial time series studies in the 1970's linking mortality with unemployment. Brenner's contribution was soon followed by growing concern over the implications of the economic recession of the early 1980's for health care programmes financed by government spending as well as the health consequences (if any) arising from cuts in such programmes.

One major strand of this literature concerns the influence of fluctuations in economic activity on mortality. Two indices of economic activity which have received particular attention are income and unemployment. Most of the British literature has examined the influence of one or both of these economic factors on overall age-adjusted mortality rates from all causes in all age groups or in adults (Brenner, 1979;

Gravelle et al, 1981; McAvinchey, 1983; Forbes and McGregor, 1984). Forbes and McGregor (1987) have also considered the impact of unemployment and income on selected cause-specific mortality rates, again in the adult population.

Although there is a long tradition of examining the effect of socio-economic factors on perinatal and infant mortality, most of the studies are cross-sectional and thus do not consider socio-economic effects over time. The British literature, again mostly cross-sectional has shown less willingness to examine how income and unemployment may influence mortality compared with its enthusiasm for demonstrating associations between social class (based on occupational status) and mortality. The predominant concern in most of these studies is the description of, rather than explanation for, social class differences in mortality.

Several important exceptions to these cross-sectional descriptive studies deserve mention. Winter's (1983) investigation of unemployment and infant mortality in Britain between 1921 and 1950 found no evidence to suggest that fluctuations in infant mortality were related to unemployment. Brenner (1979), however, using time series data for England and Wales over the period 1936-1976 claimed that unemployment did influence in-

fant mortality confirming his earlier findings (Brenner, 1973) for the United States. Brenner's findings regarding the influence of economic change on infant mortality were also replicated for the Federal Republic of Germany (John, 1983).

This chapter examines the association between income, unemployment and perinatal and infant mortality in Scotland. Unlike previous studies, the relationship between unemployment and mortality is modelled using a general autoregressive distributed lag framework in conjunction with an unemployment series disaggregated by age and duration of unemployment. The impact of tobacco and alcohol consumption on health outcomes is also considered. Sections 7.2 and 7.3 describe the income variables and the tobacco and alcohol data series. The unemployment variables are discussed in Section 7.4. Results and conclusions are presented in the next two sections.

## 7.2 Measuring Income or Consumption?

### 7.2.1 Gross Domestic Product

A variety of different measures of income have been used in health production functions. The expected impact of income on infant health, however, is far from transparent and depends on the rationale for including income in the production function. Rising income may generate (or at least be a reasonable proxy for) improvements in environmental conditions that are conducive to both maternal and infant health. On the other hand, health may have a negative income elasticity. Changes in income may be associated with behavioural change which is detrimental to health. Positive income elasticities for tobacco (Godfrey and Maynard, 1988), alcohol (Selvanathan, 1988) and other inputs which may compromise health provide some support for this hypothesis. The deleterious effect of income on health has also been stressed by Eyer (1977) who argues that economic prosperity itself can be a threat to health. Catalano and Serxner (1987) pursue this hypothesis further in a time series study which demonstrated that the rate of disabling work accidents among manufacturing employees increases with the size of the manufacturing labour force.

Many studies following the model specification associated with the work of Brenner breakdown the overall business cycle (as measured by a per-capita income series) into different components. The most popular components expoused by Brenner are

- a long run exponential income trend
- deviation in actual from trend income
- the annual change in income.

The trend component is included to measure the beneficial impact of long term economic growth (Brenner, 1987). The second two components attempt to measure "economic instability" which is believed to have an adverse effect on health.

The effects of income on mortality are modelled within this framework using three measures of real per capita Scottish GDP which allow comparison with earlier studies and enable a test of the Brenner and Eyer conjectures concerning the possible competing influences of economic growth on perinatal and infant mortality. The measurement of these variables is also consistent with the criticism of Brenner's specification voiced by Gravelle et al (1981) and Wagstaff (1985).

The first income variable is the trend level of real per capita GDP for the Scottish economy (GDPTREND). Unlike Brenner's (1981, 1987) and McAvinchey's (1983) studies of Scottish mortality and economic factors where they both rely on estimates of Scottish net in-

come based on UK personal income, an annual time series of Scottish GDP is used. The series in current prices is converted into 1980 prices using a GDP deflator. Despite the enthusiastic use of exponential income trends in previous studies, a simple linear trend was found to be a better summary of the long run trend level of real GDP in Scotland. This essentially treats the time series of real GDP as a series of cyclical fluctuations around a deterministic trend. Alternativly the trend level of real GDP could be modelled as a stochastic trend which would be appropriate if real GDP was difference stationary or, in the terminology of the cointegrated approach, integrated of order 1 [ $I(1)$]. Whether or not real GDP is best characterised as a trend stationary or difference stationary process is an open question. Rappoport and Reichlin (1989), for example, challenge the long-standing belief that real GNP is best characterised as a difference stationary process and argue that an approach based on segmented trends offers promise. Their results provide support for the use of a deterministic trend in the present study.

The second variable captures short run changes in economic growth measured by the absolute value of the annual percentage change in per capita GDP ($\Delta$GDP/GDP). This definition of short run change in economic activity emphasises the point that it is not the direc-

tion of change but change itself which influences health outcomes. As such the impact of a rapid increase in per-capita income is assumed to be equivalent to a similar proportional decrease in income. Percentage changes are used to allow for the growth in the level of GDP over time. Within this specification of Brenner's model mortality should be positively related to this variable.

The third variable is defined as the percentage deviation in the actual and trend level of per-capita Scottish GDP (GDPDEV). This variable is introduced into the equation to capture medium term departures from the long run trend in GDP. The expected sign of this variable on perinatal and infant motality is

- positive if, as Eyer (1977) suggests, mortality rises with prosperity and falls during periods when incomes are below their long run trend level

or

- negative if economic prosperity is accompanied by, for example, improvments in living standards which have positive health consequences.

The linear trend in real GDP is given by the fitted values derived from the equation

$$GDP = \underset{[57.15]}{1830.85} + \underset{[22.06]}{59.41}TREND \qquad (7.1)$$

R-squared = 0.9425 SER = 131.363 F = 459.66 DW = 0.340.

Figure 7.1 presents the actual and fitted values from this regression estimated over the period 1956 to 1985. Four distinct sub-periods can be distinguished (Figure 7.2). Above trend levels of GDP were sustained during the 1970's and between 1956-58. The 1960's and 1980's share the honor of below trend level GDP. Throughout the 1960's actual GDP was around 2.5 per cent below its long run trend level. During the early 1980's , which ushered in Scotland's biggest post-war economic recession, the percentage deviation below trend GDP increased dramatically to -5 per cent in 1981.

The annual short run change in Scottish real GDP is illustrated in Figure 7.3. The absolute value of the percentage change in GDP averaged 2.6 per cent throughout 1956-1985. This period can be divided into two phases, the first half corresponding to a period of fairly steady economic growth during the 1950's and 1960's. Widening fluctuations in economic growth (both positive and negative) have characterised the 1970's and the 1980's.

### 7.2.2 Income as a Composite Input?

Despite its popularity, this specification attributes two different interpretations to income that arguably are erroneous in a production function. If income is regarded as a composite inpute or proxy representing the bundle of goods which comprise a "standard of living" the budget shares and relative prices (e.g., for housing, food, etc) have to remain constant for this variable to make any sense in a technical relationship between inputs and health outputs.

Evidence from surveys of household and family expenditure in Scotland over time does not support this argument. Table 7.1 describes the composition of Scottish household/family expenditure over the period 1953-54 and 1985-85. Expressed in terms of budget shares the major changes in consumption reflect a shift away from food, tobacco and clothing/footwear towards housing, transport, alcohol and consumer durables. This change in budget shares is not surprising considering the results of aggregate studies of consumer demand in post-war Britain. Deaton and Muellbauer (1980) using annual British data from 1954 to 1974 estimated that a proportional increase in prices and expenditure decreased expenditure on food and on clothing and increased expenditure on housing, transportation and communication. Similar changes in the pattern of

family expenditure are reported by Muellbauer and Pashardes (1988) using a longer data series (1954-1980).

So much for budget shares. The composite good argument assumes that increases in income results in a proportional increase in the quantity of commodities (presumably adjusted for quality). A further assumption is that the marginal product of different commodities on health remains constant. A shift in budget shares may obscure shifts in the mix of commodities and services consumed by households. This quantity mix, moreover, may respond in different ways to long and short run changes in income. To test this composite good hypothesis the minimum data set would be an income series and a series of household consumption quantities appropriately adjusted for quality. Given the absence of such a quantity series inferences can only be made from the data on budget shares which suggests that the conditions for treating income as a proxy for a health input composite good are unlikely to be satisfied.

### 7.2.3 An Alternative Interpretation of Income Effects

A rival interpretation excludes income as an argument in the production function. Given that household consumption patterns are conditioned by household income

an alternative approach would be to first model the demand for health related commodities and then include the commodities themselves, rather than income, in the production function. This second approach is preferable on econometric grounds as it lessens the impact of the simultaneous equation bias introduced when both income and specific household commodities are included as inputs in equations which are neither production nor demand functions.

This still leaves the question of what commodities to include as arguments in the production functions. The list of candidates is endless. However, no theory, economic or otherweise, provides unambiguous rules for excluding or including particular variables. The choice of variables thus has to rely on other criteria, namely commonsense.

### 7.3 Tobacco and Alcohol Consumption

In the mortality production functions estimated below two health related commodities are included as inputs. The justification for these inputs is based partly on their known (or suspected cross sectional impact on infant health and partly on their long standing importance within the context of public health policy. The first input is the consumption of tobacco. Maternal smoking during pregnancy has been associated with a

wide range of adverse perinatal and infant health outcomes. Abel's (1980) survey of the available evidence demonstrated that smoking increased the likelihood of:

- various pregnancy complications
- fetal distress
- spontaneous abortion
- low birthweight
- malformations
- perinatal and neonatal mortality

The most likely mechanism linking these adverse outcomes and maternal smoking is via (i) direct exposure of the fetus to the constituent ingredients in cigarette smoke and (ii) physiological changes in the mother during pregnancy. The major intervening variable in this equation is fetal hypoxia which arises as a result of the increased levels of carboxyhemoglobin which reduce the oxygen carrying capacity of blood.

No annual time series exists for cigarette consumption amongst pregnant women in Scotland. An approximation can be derived using an annual series on age specific per-capita cigarette consumption in British women aged 16-34 (Wald et al, 1988). A weighted average of estimated cigarette consumption is derived by combining the age-specific British consumption data with the age

distribution of the Scottish obstetric population. The age distribution of the Scottish obstetric population was obtained from the Registrar General Scotland. This weighted average of overall consumption is also adjusted upward in line with the higher consumption of cigarettes in the Scottish female population. A further control can also be made for changes over time in the carbon-monoxide yield of cigarettes.

The second input is alcohol consumption. Compared to maternal smoking the evidence linking consumption with perinatal and infant health outcomes is less conclusive. Although excessive alcohol consumption during pregnancy has achieved the notority of a syndrome (Fetal Alcohol Syndrome) characterised by the increased likelihood of facial anomalies, organ disorders and growth retardation in babies born to alcoholics, moderate consumption of alcohol poses less of a threat to the fetus. Abel's (1982) trawl through the literature found no evidence of a significant relationship between alcohol consumption and perinatal mortality. However, maternal alcohol consumption is associated with fetal hypoxia and growth retardation, though the magnitude of effect is less than that observed for maternal smoking.

Data on UK per capita consumption of beer, wine and spirts are taken from Selvanathan (1988). Over the period 1955 to 1985 the average annual growth in per capita consumption was 1.0, 5.3 and 3.6 per cent for beer, wine and spirits, respectively. In terms of consumer expenditure budget shares wine has gained at the expense of beer whereas the spirit share has remained fairly constant over this period. Using standard conversion ratios for the alcohol content of different beverages the quantity series are re-expressed in terms of a single series on per capita alcohol consumption.

As Chapter 3 noted a common criticism of econometric studies of mortality rates is that the hybrid nature of the estimated equations is often ignored. For example, in a recent study of economic instability, unemployment and mortality in Scotland, Brenner (1987) regresses mortality rates on per capita income, unemployment rates and several indicators of "behavioural risk" including the per-capita consumption of cigarettes and alcohol (alongwith January temperature!). By including both income and the "behavioural risk" variables in the same equation this specification inadvertently embraces simultaneous equation bias.

One obvious way around this problem is to model the demand for the health related commodities using a simultaneous equation approach in which demand for "behavioural risks" is conditional upon prices and income and perinatal mortality is conditional upon health sector inputs and behavioural inputs such as smoking and alcohol consumption. If these behavioural inputs can be sensibly modelled then they may have some role to play in the health production function.

Standard demand equations for cigarette and alcohol consumption typically regress per-capita consumption of these commodities on their own prices and real disposable income per capita. In the case of cigarettes, investigators have also included a variety of additional variables designed to capture the effects of advertising and anti-smoking publicity on consumption.

Although many studies express a (untested) preference for log-linear specifications, Leu (1984) justified the use of a linear functional form for the demand equation on the basis of a likelihood ratio test of these rival specifications. A linear specification is adopted for the cigarette and alcohol demand equations given by

$$C_t = \pi_0 + \pi_1 PC_t + \pi_1 PDI_t + e_t \qquad (7.2)$$

$$A_t = \beta_0 + \beta_1 PA_t + \beta_1 PDI_t + e_t \qquad (7.3)$$

where $C_t$ = annual per-capita consumption of ciagarettes by Scottish women aged 16-34, $A_t$ = annual per capita alcohol consumption; $PC_t$ = real price of cigarettes; $PA_t$ = real price of alcohol and $PDI_t$ = real per capita Scottish personal disposable income. The real price variables are calculated by respectively dividing the tobacco and alcohol price indices by the all items index of retail prices (Department of Employment, 1987). Real per-capita personal disposable income for Scotland was estimated by applying the UK ratio of real personal disposal income and real GDP to the Scottish series of real GDP.

Figures 7.4 to 7.8 present the series for consumption, prices and personal disposable income over the period 1956 to 1985. Annual cigarette consumption amongst Scottish women aged 16-34 increased steadily during the 1960's and early 1970's eventually peaking at just over 4100 cigarettes per year (79 per week). Since 1975 consumption has declined by 3.6 per cent per year. The real price of cigarettes fluctuated within a narrow range in the early 1960's and then fell dramatically between 1965 and 1973. The 1980's have been characterised by a sharp increase in the real price of cigarettes. Although the real price of alcohol follows a roughly similar path to that of

tobacco, per-capita alcohol consumption has not declined over the past decade but has increased in line with the growth in personal disposable incomes.

A more precise summary of the effect of prices and income on the consumption of cigarettes and alcohol is given by the OLS estimates of equations 7.2 and 7.3

$$C_t = \underset{[2.328]}{3848.103} - \underset{[-2.010]}{24.2675PC_t} + \underset{[3.553]}{1.0220PDI_t} \quad (7.2a)$$

R-squared = 0.5335 SER = 520.2516 F= 15.44 DW = 0.36

$$A_t = \underset{[1.251]}{1.003} - \underset{[-2.141]}{0.0120PA_t} + \underset{[28.917]}{0.0035PDI_t} \quad (7.3a)$$

R-squared = 0.9880 SER = 0.1619 F= 1109.61 DW = 1.560

Evaluated at the sample means over the 1956-1985 period the price elasticity for cigarettes is -0.939. This is about twice as large as that reported in most of the early studies based primarily on data for the 1950's and 1960's. However, it is well within the range reported by Godfrey and Maynard (1988) using more recent data (-0.4 to -1.5). The income elasticity for cigarettes (0.633) is also consistent with the results of previous studies.

The price elasticity for alcohol is -0.226. The corresponding income elasticity is 1.059. Despite using a much simplier linear specification these results are

similar to those reported for the UK by Selvanathan (1988) who estimated the demand for alcoholic beverages using the Rotterdam demand system.

Although it was tempting to stop at this point and accept these results without further testing, a closer examination of these demand equations generated the following findings. First, the demand equation for cigarettes is very unstable. When the estimation period is truncated from 1985 to 1980 the estimated coefficients change sign. One by product of this instability is a ludicrous set of forecasts over the period 1981 to 1985 (Figure 7.11). As the forecast rates of cigarette consumption march steadily upward, actual consumption begins to fall. When the equation is re-estimated using recursive least squares over the period 1956 to 1985 the coefficient on the price of cigarettes follows a rather disconcerting pattern (Figure 7.12) which not surprisingly is graphically emphasised by the Chow statistics (Figure 7.13) pointing towards a major shift in parameters towards the end of the 1970's.

The demand equation for alcohol was better behaved in terms of stability and forecasting performance. However, this crude measure of consumption is several stages removed from a more refined index of alcohol consumption amongst the fertile population. Although

crude per-capita consumption of alcohol has risen over time whether this is also true for women and particularly women during pregnancy is an open question. Thus, despite the expectation that these behavioural inputs would reduce some of the bias inherent in relatively simple specifications of perinatal health production functions, problems of both measurement and modelling stand in the way of making much progress in this area.

### 7.4 Unemployment

With two exceptions (Forbes and McGregor, 1984; 1987) studies of the impact of unemployment on mortality use aggregate unemployment rates with no adjustment for changes in the age and duration composition of unemployment which occur over time. The consequences of ignoring changes in the age composition of the unemployed in studies of the effect of unemployment in adults are obvious and need not be repeated here (Forbes, 1981). Simply stated if unemployment has a direct impact on infant health, increases in unemployment amongst younger workers are likely to be more important than a bulge in unemployment amongst 55 to 60 year olds who have entered a stage of their life cycle where child raising is a feature of their distant past.

If the incidence of unemployment was independent of age, increases in unemployment amongst the actively "fertile" population would be reflected in aggregate unemployment rates. The available evidence on unemployment rates in Britain over the period 1976 to 1985 reveals a three fold increase in male unemployment rates. Although unemployment has increased in all age groups the greatest increases have occurred amongst those aged 55-59 where unemployment has gone up by nearly four-fold (Layard, 1986). This disproportionate increase in unemployment amongst older workers effectively undermines the use of aggregate unemployment rates in the analysis of perinatal and infant mortality.

A similar consideration applies with respect to the importance of controlling for different durations of unemployment. The recent rise in unemployment is partly explained by an increased flow into unemployment. A more significant factor, however, is the decline in outflow rates among all durations but particularly in the long term unemployed. The dynamics of the sharp rise in aggregate unemployment in Great Britain between 1980 and 1983 reflect a rapid increase in long term unemployment from 1981 to 1983. The fraction of unemployment attributable to spells of greater than 9 months has risen from 0.50 in 1978 to 0.87 in 1985 (Haskel and Jackman, 1987). The average

uncompleted duration of current spells has increased from just under nine months in 1975-79 to nearly 16 months in 1984 (Layard and Nickell, 1986). The explanation for the increase in long term unemployment is thus a high inflow rate accompanied by a much lower exit probability.

A similar picture emerges for Scottish male unemployment. Figure 7.9 presents the numbers of unemployed males aged 20-54 expressed as a percentage of the male population in this age group. These population based unemployment rates will understate the conventional rates defined using the labour force as a denominator. The labour force participation rates amongst men in this age range are nevertheless high. The main feature is the general upward trend in unemployment culminating in a rapid increase beginning in 1980 particularly in long term unemployment (defined as a continuous spell of 26 weeks or more). The differential rates of increase in short and long term unemployment in this age group are graphically illustrated by the ratio of long to short duration unemployment rates (Figure 7.10). For nearly three decades beginning in 1948 this ratio remained fairly static, hovering around 0.60. In the late 1970's it departed from this steady historical pattern reaching a post-war peak of 1.64 in 1983-85.

In the equations presented below these two measures of male unemployment in Scottish men aged 20-54 are used to allow for the possible impact of different durations of unemployment on perinatal and infant mortality. Short term is defined as a period of continuous unemployment of up to 26 weeks. Long term refers to a period of continuous unemployment of more than 26 weeks. The numbers of unemployed men in these age and duration categories reported at June from 1950 to 1961 and July from 1962 to 1985 are used in these calculations. The age and duration data was obtained from the Ministry of Labour Gazette and the Scottish Abstract of statistics (various years). Because of changes in the categories used to cross-classify age and duration in the published unemployment statistics no further consistent disaggregation of age and duration is possible for Scotland over the period 1950-1985. The population figures used in the denominators are based on the Registrar General's annual estimates of the age and sex distribution of the home population for Scotland.

Several points should be noted about these unemployment series. First, they measure the incidence of unemployment in the population and are not synonymous with conventional age-specific unemployment rates. The choice of population-based denominators was due to the absence of annual time series data on the age dis-

tribution of the labour force. Age-specific unemployment rates using the labour force as a denominator are only available in Britain since 1975 (Garside, 1980). Age-specific activity rates for males are high in the age group 20-54 and have shown only small declines during the post-war period (Thomson and Hunter, 1978).

The series are also confined to males. No consistent series of British or Scottish female unemployment over time is available. Because of the changes which have occurred in women's entitlement to unemployment benefit the number of women registered for work does not provide an accurate measure of trends in female job seeking nor the number of unemployed women. This male bias is a common feature of most aggregate time series analysis of unemployment in Britain (Layard and Nickell, 1986).

A third point concerns the changes since 1979 in the official British definition and measurement of unemployment. Most of the 29 changes which have occurred do not affect this age group. However, the change in October 1982 from a registered count of the unemployed (those registering at employment exchanges) to a claimant count of those people claiming benefits or National Insurance credits has introduced a discontinuity into the unemployment series. This change in the official definition of unemployment had the effect

of reducing aggregate male unemployment in Scotland from 232000 to 214000, a reduction of 18000 or 7.8 per cent. Nearly 90 per cent of this apparent decline in unemployment, however, was confined to men at the extremes of the labour force age distribution (i.e., those aged less than 19 or more than 59). Thus, compared to school leavers and those aged 60+ where changes have occurred in benefit entitlements, this change in the official definition of unemployment has had little impact on the recorded level of unemployment amongst men aged 20-54. Finally, it also be noted that the results presented below are based on an estimation period ending in 1980. Any errors in the unemployment series induced by definitional changes will influence the forecasting performance of the model over the years 1983-1985 and corresponding tests of parameter constancy.

Table 7.2 presents variable names, definitions, means and standard deviations for the income, unemployment, cigarette, alcohol and price variables. Appendix Table A7.1 contains the data series. Information for the mortality, birthweight and health care variables used in the equations is contained in Tables 6.1 and 6.2 and the corresponding data appendix in Chapter 6.

## 7.5 Results

### 7.5.1 General and Restricted Specifications of the Brenner Model

The first stage involved estimated a general specification of the Brenner model and then testing the restrictions imposed by a set of nested sub-models. The models, defined in Table 7.3, range from the general (Model 1) to the restrictive (Model 4) which excludes all income and unemployment variables. A lagged dependent variable is included in all specifications.

**Birthweight specific perinatal mortality**

The results of testing different specifications using birthweight specific perinatal mortality rates as the dependent variable are presented in Table 7.4. The F-tests support the acceptance of the restrictions imposed by Model 2 which excludes the current and lagged

unemployment rates. This restriction is valid irrespective of whether short-term or long-term unemployment rates are introduced into the model. Model 3, which excludes unemployment rates and the two measures of economic change (GDPDEV and $\Delta$ GDP/GDP), is also accepted for all birthweight groups. The most restrictive model, excluding all of the "economic" variables routinely included in models based on Brenner's specification (Model 4), is accepted for birthweights less than 1.5kg. Model 4 is, however, rejected for birthweights greater than 1.5kg.

OLS estimates over the period 1957-1980 of the most restrictive models consistent with the data are presented in Table 7.5. Birthweight distribution is not a significant factor influencing birthweight specific mortality. Expenditure on perinatal care has a negative impact on mortality in the VLBW (<1.5kg) and NBW (>2.5kg) equations. The most important factor influencing mortality in births weighing more than 1.5kg is the long run trend level of GDP. In births weighing more than 2.5kg the estimated elasticity of weight specific mortality with respect to trend GDP is -1.979, suggesting that a 2.5 per cent increase in income is associated with a 4.9 per cent reduction in weight specific mortality in this group of normal weight babies. The corresponding elasticity for perinatal hospital expenditure is -0.253.

These differences in estimated elasticities, however, should not encourage the blind acceptance of the notion that, compared to expenditure on perinatal hospital services, economic growth per se is a more powerful policy instrument for improving perinatal mortality. Consider the magnitude of expenditure associated with the following two options. The first is 2.5 per cent increase in real per-capita Scottish GDP. Evaluated at the sample mean this would represent £65.82 per person or around £337m for the Scottish population. The second option would be 10 per cent increase in perinatal hospital expenditure per birth. Again, evaluated at the sample mean, this would among to an increase of 50.07 per birth or £3.75m for the Scottish obstetric population. Although the benefits (as well as the costs) associated with an increase in real per-capita GDP would extend beyond the perinatal health sector it would be difficult to justify option 1 as an efficient policy directed towards reducing perinatal mortality considering the more direct option of investment in the perinatal hospital sector.

The OLS estimates for the general model are reported for comparison in Table 7.6. The coefficients on the unemployment variable are generally insignificant. No consistent patten emerges in the signs of the unemployment variables: a positive coefficient is just

as likely as a negative coefficient. Although the sign on the Δ GDP/GDP variable is consistent with the hypothesis that short run economic change poses a risk to perinatal survival, the coefficients are not significantly diferent from zero. The GDPDEV coefficients are also consistent with Brenner's conjectures regarding the impact of deviations from long run economic growth. However, most of the coefficients are again insignificant.

**Fetal, Neonatal and Post-neonatal Mortality**

The F-tests for adding the unemployment and income terms in equations for fetal, neonatal and post-neonatal mortality rates generally confirmed the results for birthweight specific mortality (Table 7.7). The restrictions imposed by excluding the unemployment variables were accepted for fetal mortality and mortality throughout the first year of life. The income change variables again could be excluded, on the basis of the F-tests, from the most parsimonous specification which usually supported including the GDP trend. F-tests failed, however, to justify the inclusion of the GDP trend in the post-neonatal mortality equation. This somewhat surprising result contradicts on the long-standing beliefs in the epidemiology of infant health that economic factors

are more important determinants of mortality in child in the post-neonatal as opposed to the perinatal period. One possible explanation for this finding is that, despite the steady growth in real per-capita GDP, post-neonatal mortality rates in Scotland have stayed fairly static over the past two decades.

Cause-specific neonatal mortality

Table 7.8 presents the results of testing the set of restrictive models against the general model for different causes of neonatal death. On statistical criteria no support could be found for including the unemployment or income variables in these cause-specific equations.

### 7.5.2 Smoking and Alcohol Consumption

Despite the reservations outlined above, a series of tests were conducted to see whether the data supported the inclusion of smoking and alcohol variables in a simple model relating birthweight specific and cause-specific mortality rates to expenditure on perinatal hospital services, lagged mortality and current and lagged values of per-capita cigarette consumption and

per-capita alcohol consumption. The results confirmed a general lack of enthusiasm for embracing these behavioural inputs in the production function estimates. The F-tests supported the exclusion of these inputs from all equations with the single exception of the equation for neonatal deaths due to prematurity (in the latter case the sign on the cigarette variable was negative or positive depending on whether current and lagged values of cigarette consumption were included in the equation). The insignificant and indeterminant impact of these factors on mortality was also confirmed by TSLS estimates.

## 7.6 Conclusions

This chapter investigated the possible links between income, unemployment and infant health. The main findings failed to support the hypothesis that unemployment excerts an impact on perinatal and post-neonatal mortality, let alone an adverse impact. These results, based on the analysis of time series data, cast serious doubt on the robustness of the intuitively appealing notion that health outcomes in infancy are intimately linked with the economic health of a population as measured by the level of unemployment. In addition, measures of economic instability (short and long-run changes in economic growth) seem to play little role in determining the chances of sur-

vival of new born children. These results for mortality at the start of life confirm the general findings reported for the impact of unemployment on adult mortality by workers who have used a critical approach to model specification and testing (Gravelle et al, 1981; Forbes and McGregor, 1984).

Table 7.1 Composition of Household/Family Expenditure in Scotland

| CATEGORY | YEAR 1953-54 | 1964-66 | 1974-75 | 1984-85 | 1984-85 (1953-54=100) |
|---|---|---|---|---|---|
| HOUSING | 0.0714 | 0.0845 | 0.1106 | 0.1190 | 167 |
| FUEL, LIGHT & POWER | 0.0539 | 0.0682 | 0.0576 | 0.0645 | 120 |
| FOOD | 0.3484 | 0.2924 | 0.2583 | 0.2127 | 61 |
| ALCOHOL | 0.0302 | 0.0432 | 0.0582 | 0.0556 | 184 |
| TOBACCO | 0.0803 | 0.0715 | 0.0453 | 0.0378 | 47 |
| CLOTHING & FOOTWEAR | 0.1247 | 0.1047 | 0.0960 | 0.0815 | 65 |
| CONSUMER DURABLES | 0.0543 | 0.0696 | 0.0750 | 0.0788 | 145 |
| OTHER GOODS | 0.0645 | 0.1075 | 0.0697 | 0.0744 | 115 |
| TRANSPORT | 0.0743 | 0.1075 | 0.1189 | 0.1400 | 188 |
| SERVICES | 0.0917 | 0.0960 | 0.1051 | 0.1170 | 128 |
| MISCELLANEOUS | 0.0063 | 0.0043 | 0.0051 | 0.0043 | 68 |
| TOTAL | 1.0000 | 1.0000 | 1.0000 | 1.0000 | |
| EXPENDITURE/WEEK (current £) | 11.43 | 20.83 | 49.47 | 152.47 | |

TABLE 7.2 VARIABLE NAMES, DEFINITIONS, MEANS AND STANDARD DEVIATIONS

| VARIABLE | DEFINITION | MEAN* | STANDARD DEVIATION* |
|---|---|---|---|
| GDPTREND | Linear trend of per-capita Scottish GDP (1980) prices | 2751.67 | 522.99 |
| GDPDEV | Percentage deviation in actual and trend GDP | -0.03 | 4.34 |
| Δ GDP/GDP | Annual percentage change in GDP | 2.65 | 1.76 |
| PDI | Per-capita Scottish personal disposable income (1980) prices | 1822.63 | 386.35 |
| UNEMsd | Population incidence of short-duration (< 26 weeks) unemployment in male population aged 20-54 | 2.97 | 1.29 |
| UNEMld | Population incidence of short-duration (> 26 weeks) unemployment in male population aged 20-54 | 2.60 | 2.36 |
| C | Annual per-capita cigarette consumption (Scottish women aged 16-34) | 2944.33 | 734.94 |
| PC | Real price of cigarettes (1980=100) per 1000 live births | 114.00 | 10.03 |
| A | Annual per-capita alcohol consumption (litres per year) | 5.97 | 1.43 |
| PA | Real price of alcohol (1980=100) per 1000 live births | 112.67 | 8.20 |

*Calculated over the period 1956-1985

Table 7.3 General and Restricted Specifications of Brenner's Model

| | MODEL 1 (general) | MODEL 2 | MODEL 3 | MODEL 4 (restrictive) |
|---|---|---|---|---|
| | CONSTANT | CONSTANT | CONSTANT | CONSTANT |
| | LDV | LDV | LDV | LDV |
| | BW | BW | BW | BW |
| REGRESSORS | EXPEND | EXPEND | EXPEND | EXPEND |
| | GDPTREND | GDPTREND | GDPTREND | |
| | GDPDEV | GDPDEV | | |
| | Δ GDP/GDP | Δ GDP/GDP | | |
| | UNEM | | | |
| | UNEM | | | |

NOTES: LDV = lagged dependent variable
UNEM is replaced with UNEMsd and UNEMld to test for the differential impact of short and long durations

Table 7.4 F-tests of model restrictions
Birthweight specific perinatal mortality

| | | < 1.5kg | | 1.5-2.5KG | | > 2.5kg | |
|---|---|---|---|---|---|---|---|
| MODEL | Unem Measure | Short Duration | Long Duration | Short Duration | Long Duration | Short Duration | Long Duration |
| 2 versus 1<br>F(2,15)<br>CV=3.68 | | 0.01 | 1.98 | 1.55 | 1.96 | 3.01 | 0.27 |
| 3 versus 1<br>F(4,15)<br>CV=3.06 | | 0.54 | 1.67 | 1.46 | 1.70 | 2.30 | 0.73 |
| 4 versus 1<br>F(5,15)<br>CV=2.90 | | 1.50 | 2.68 | 4.10 | 4.43 | 5.14 | 3.02 |

Table 7.5 Restricted Regression Results
Birthweight specific perinatal mortality

| | Birthweight Group | | |
|---|---|---|---|
| REGRESSOR | < 1.5kg | 1.5-2.5KG | > 2.5kg |
| BW | -1.6166 | -0.8848 | 0.0230 |
| | [-0.125] | [-0.827] | [0.229] |
| EXPEND | -0.1526 | 0.0357 | -0.0062 |
| | [-1.152] | [0.860] | [-2.065] |
| LAGGED DEPENDENT VARIABLE | 0.7728 | 0.1209 | 0.0608 |
| | [5.156] | [0.555] | [0.294] |
| GDPTREND | - | -0.0991 | -0.0092 |
| | - | [-3.658] | [-3.599] |
| CONSTANT | 256.7990 | 413.6349 | 17.3740 |
| | [1.016] | [3.546] | [0.245] |
| R-SQUARED | 0.8461 | 0.9507 | 0.9856 |
| SER | 45.0981 | 9.3919 | 0.6877 |
| F | 36.6500 | 91.6800 | 325.4600 |
| DW | 2.0800 | 2.0500 | 2.1700 |
| Residual Autocorrelation Coefficients | | | |
| LAG 1 | -0.0672 | -0.0690 | -0.0765 |
| LAG 2 | -0.1182 | 0.2269 | 0.0284 |
| LAG 3 | -0.0962 | 0.1061 | -0.0403 |
| LAG 4 | -0.1728 | -0.2942 | -0.2263 |

Table 7.6 Unrestricted Regression Results
Birthweight specific perinatal mortality

| REGRESSOR | Unem Measure | < 1.5kg Short Duration | < 1.5kg Long Duration | 1.5-2.5KG Short Duration | 1.5-2.5KG Long Duration | > 2.5kg Short Duration | > 2.5kg Long Duration |
|---|---|---|---|---|---|---|---|
| BW | | -23.4010 | -25.0400 | -0.7066 | -0.5068 | 0.0231 | 0.5720 |
| | | [-1.378] | [-1.889] | [-0.664] | [-0.466] | [0.300] | [0.617] |
| EXPEND | | 0.2285 | 0.6077 | 0.1744 | 0.1914 | -0.0119 | -0.0051 |
| | | [0.601] | [1.842] | [2.085] | [2.491] | [-2.042] | [-0.830] |
| LAGGED DEPENDENT VARIABLE | | 0.3011 | 0.2551 | 0.0966 | 0.0050 | -0.1066 | 0.0643 |
| | | [1.72] | [1.218] | [0.438] | [0.022] | [-0.523] | [0.295] |
| GDPTREND | | -0.3338 | -0.3393 | -0.1315 | -0.1415 | -0.0113 | -0.0102 |
| | | [-2.113] | [-2.929] | [-4.071] | [-4.191] | [-3.910] | [-3.005] |
| GDPDEV | | -2.4094 | -8.0665 | -2.7702 | -2.8977 | 0.0629 | -0.0144 |
| | | [-0.374] | [-1.519] | [-2.094] | [-2.362] | [0.700] | [-0.143] |
| GDP/GDP | | 7.9828 | 10.8610 | 0.4186 | 0.3906 | 0.3051 | 0.1269 |
| | | [1.230] | [2.077] | [0.315] | [0.331] | [0.384] | [1.329] |
| UNEM | | 2.6226 | -42.3040 | -8.0925 | -12.1365 | 0.6066 | 0.3479 |
| | | [0.112] | [-1.677] | [-1.678] | [-1.953] | [1.861] | [0.725] |
| UNEM | | 0.9822 | 7.9167 | 3.1103 | 5.7272 | 0.3948 | -0.1866 |
| | | [0.044] | [0.329] | [0.758] | [1.008] | [1.277] | [-0.396] |
| CONSTANT | | 1523.8400 | 1624.7500 | 436.6000 | 453.1250 | 25.0130 | -13.0200 |
| | | [2.456] | [3.366] | [3.589] | [3.695] | [0.353] | [-0.153] |
| R-SQUARED | | 0.8974 | 0.9187 | 0.9645 | 0.9661 | 0.9910 | 0.9879 |
| SER | | 45.5268 | 37.8483 | 8.9669 | 8.7697 | 0.6092 | 0.7084 |
| F | | 16.3900 | 21.1900 | 51.0200 | 53.4300 | 208.5300 | 153.6800 |
| DW | | 1.9100 | 2.1700 | 1.6400 | 2.0200 | 2.3600 | 2.3700 |
| Residual Autocorrelation Coefficients | | | | | | | |
| LAG 1 | | 0.0596 | -0.1570 | 0.2118 | 0.0301 | -0.2269 | -0.1616 |
| LAG 2 | | -0.2085 | -0.1560 | 0.2138 | 0.0561 | -0.1404 | 0.1731 |
| LAG 3 | | 0.1726 | 0.0406 | 0.2433 | 0.1272 | 0.0323 | 0.0989 |
| LAG 4 | | 0.0610 | -0.0167 | -0.5683 | -0.3611 | 0.1398 | -0.0842 |

Table 7.7 F-tests of model restrictions
Fetal, Neonatal and Post-Neonatal Mortality

| | | Fetal | | Neonatal | | Post-Neonatal | |
|---|---|---|---|---|---|---|---|
| MODEL | Unem Measure | Short Duration | Long Duration | Short Duration | Long Duration | Short Duration | Long Duration |
| 2 versus 1<br>F(2,16)<br>CV=3.63 | | 1.21 | 1.53 | 0.25 | 2.49 | 0.16 | 0.84 |
| 3 versus 1<br>F(4,16)<br>CV=3.01 | | 1.63 | 1.83 | 0.44 | 1.65 | 0.26 | 0.61 |
| 4 versus 1<br>F(5,16)<br>CV=2.85 | | 6.92 | 7.27 | 2.33 | 3.83 | 0.67 | 0.99 |

Table 7.8 F-tests of model restrictions
Cause-specific Neonatal Mortality

| | | Malformations | | Prematurity | | Asphyxia | |
|---|---|---|---|---|---|---|---|
| MODEL | Unem Measure | Short Duration | Long Duration | Short Duration | Long Duration | Short Duration | Long Duration |
| 2 versus 1<br>F(2,16)<br>CV=3.63 | | 0.13 | 0.88 | 0.69 | 0.21 | 0.58 | 0.40 |
| 3 versus 1<br>F(4,16)<br>CV=3.01 | | 0.24 | 0.62 | 0.37 | 0.13 | 1.11 | 1.00 |
| 4 versus 1<br>F(5,16)<br>CV=2.85 | | 0.27 | 0.59 | 1.93 | 1.64 | 1.18 | 1.08 |

Figure 7.1 GDPTREND ___ and GDP — — in Scotland

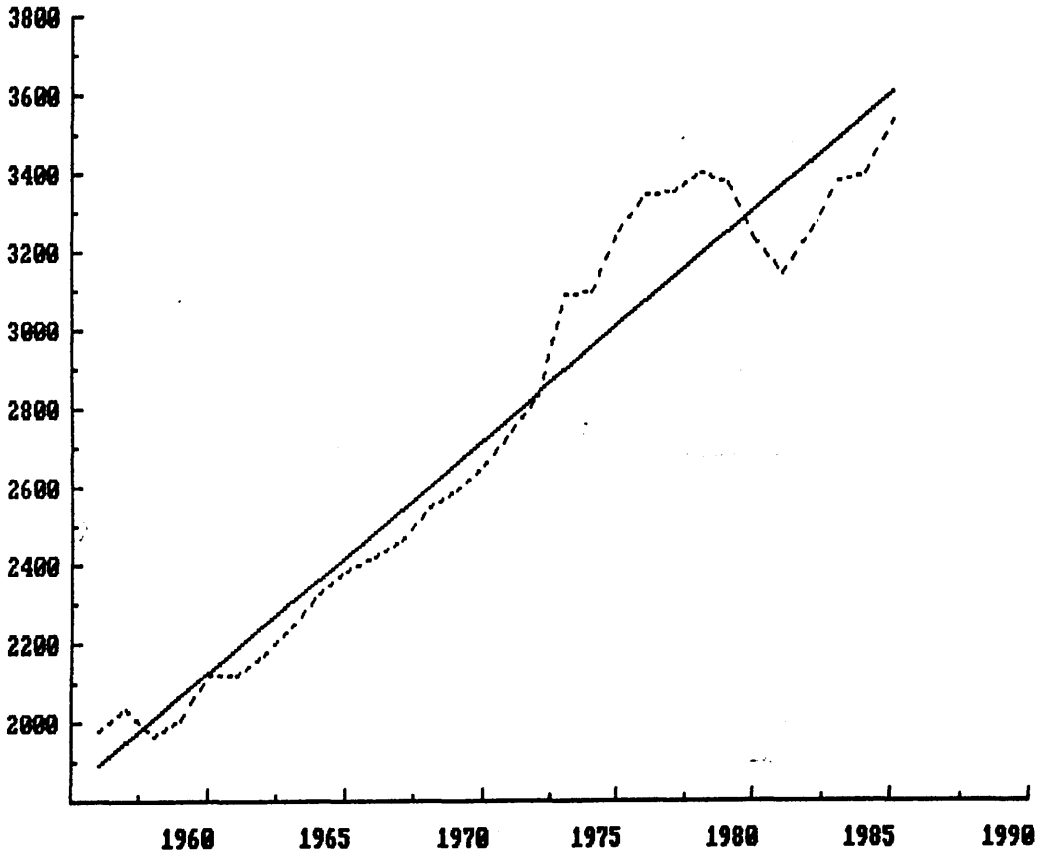


Figure 7.2 GDPDEV = _____ (Percentage Deviation from Trend)

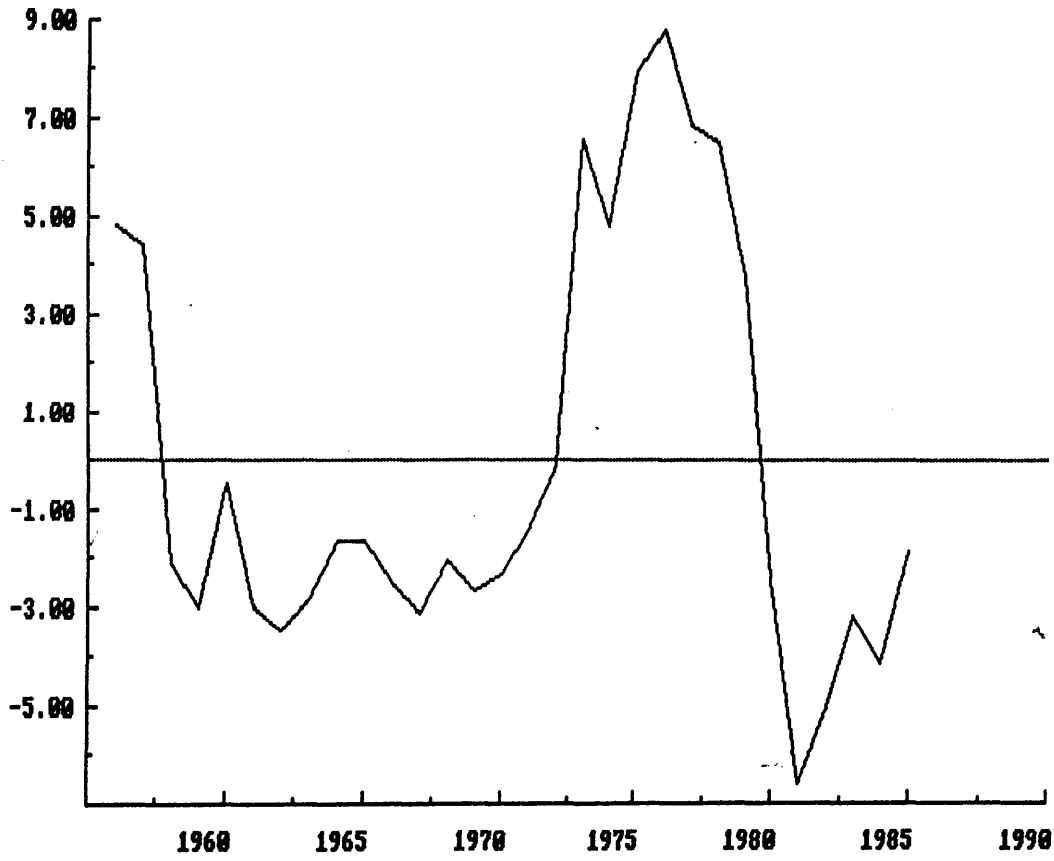


Figure 7.3 Δ GDP/GDP = _____ (Absolute value of annual percentage change in GDP)

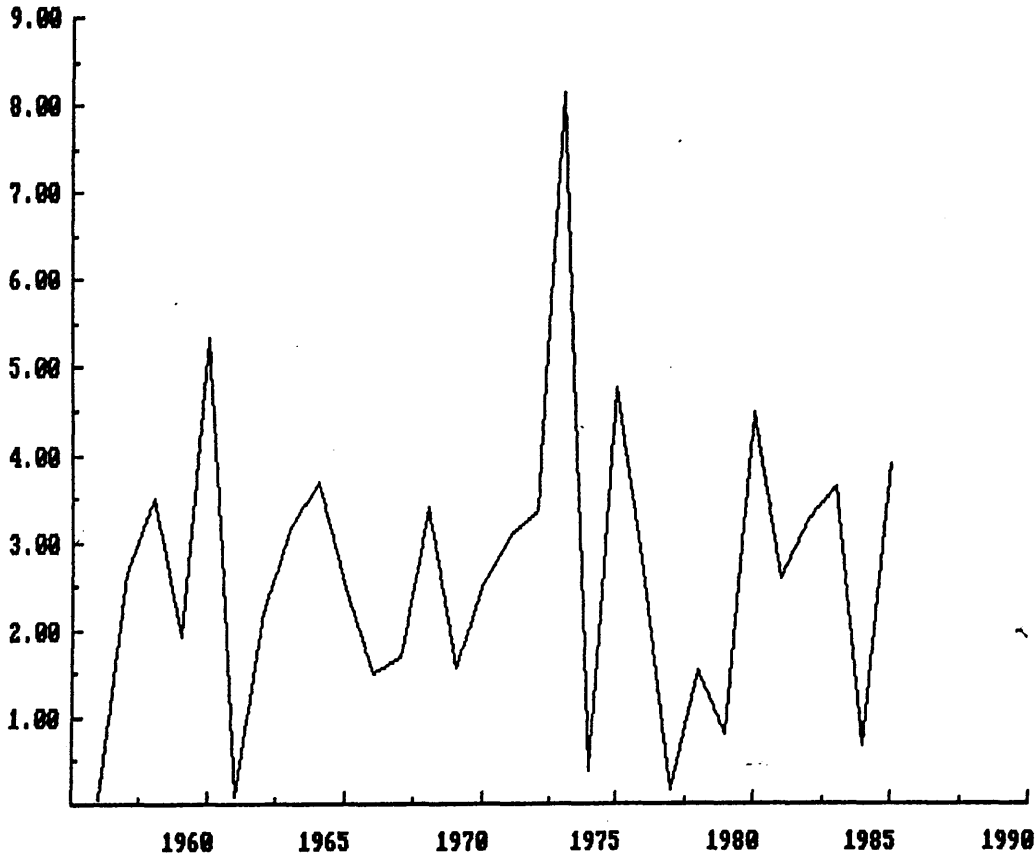


Figure 7.4 Annual Per-capita Cigarette Consumption
(Scottish Women Aged 16-34)

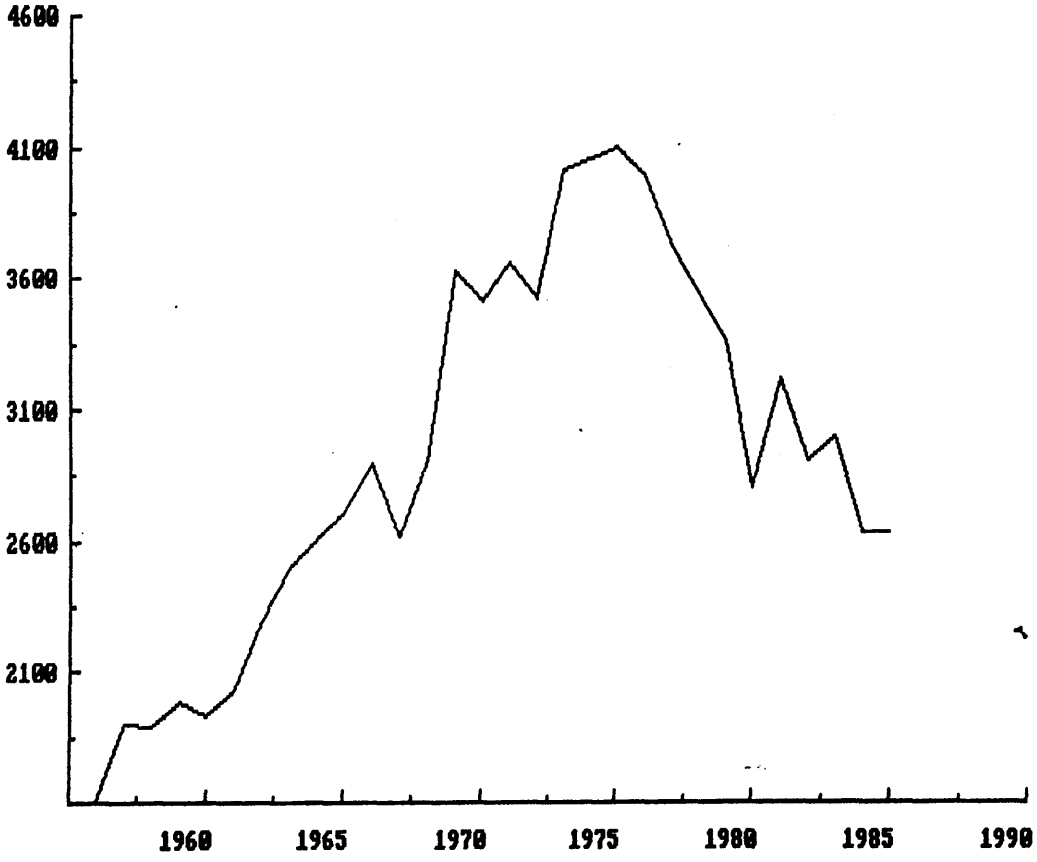


Figure 7.5 Real Price of Cigarettes (1980=100)

Figure 7.6 Per-capita Alcohol Consumption (U.K)
(litres/year)

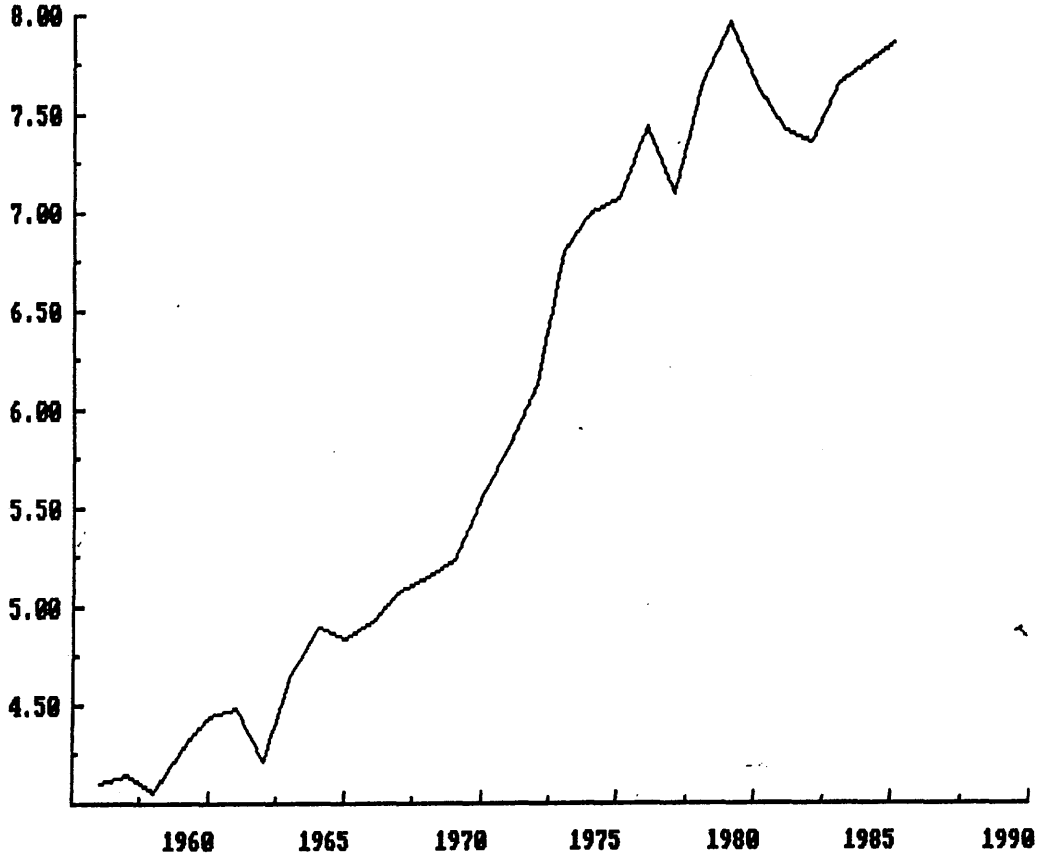


Figure 7.7 Real Price of Alcohol (1980=100)

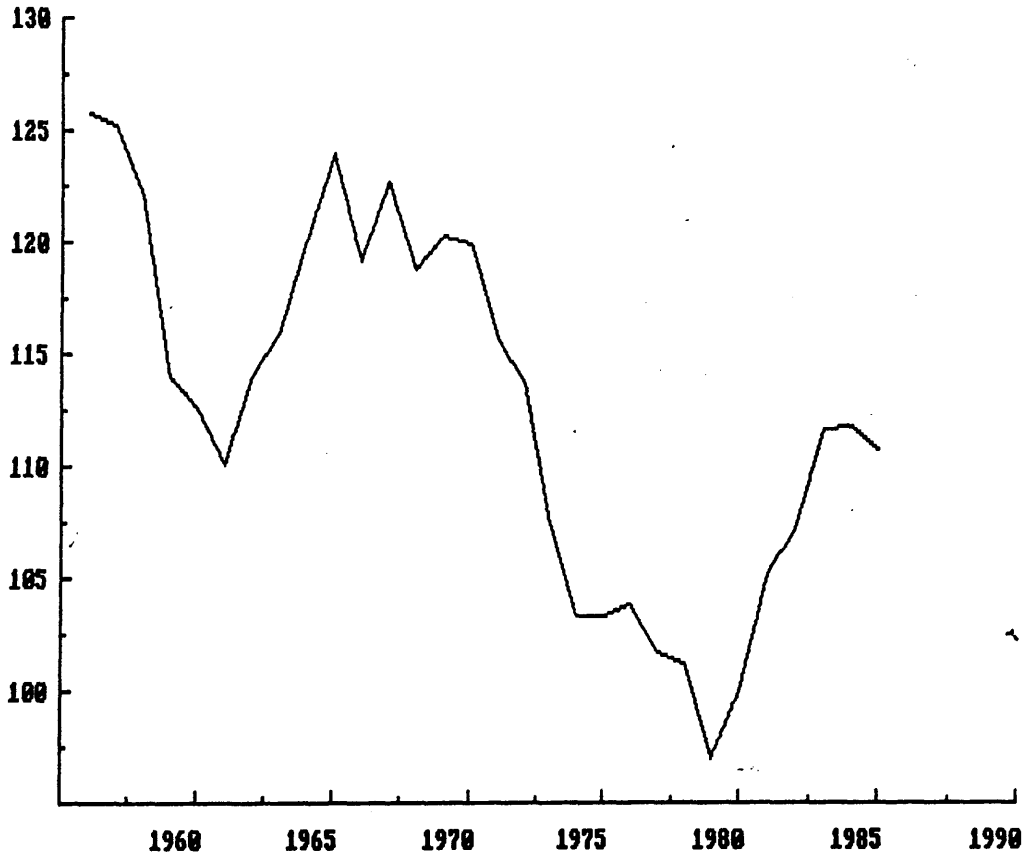


Figure 7.8 Scottish Personal Disposable Income (per-capita)
(1988 prices)

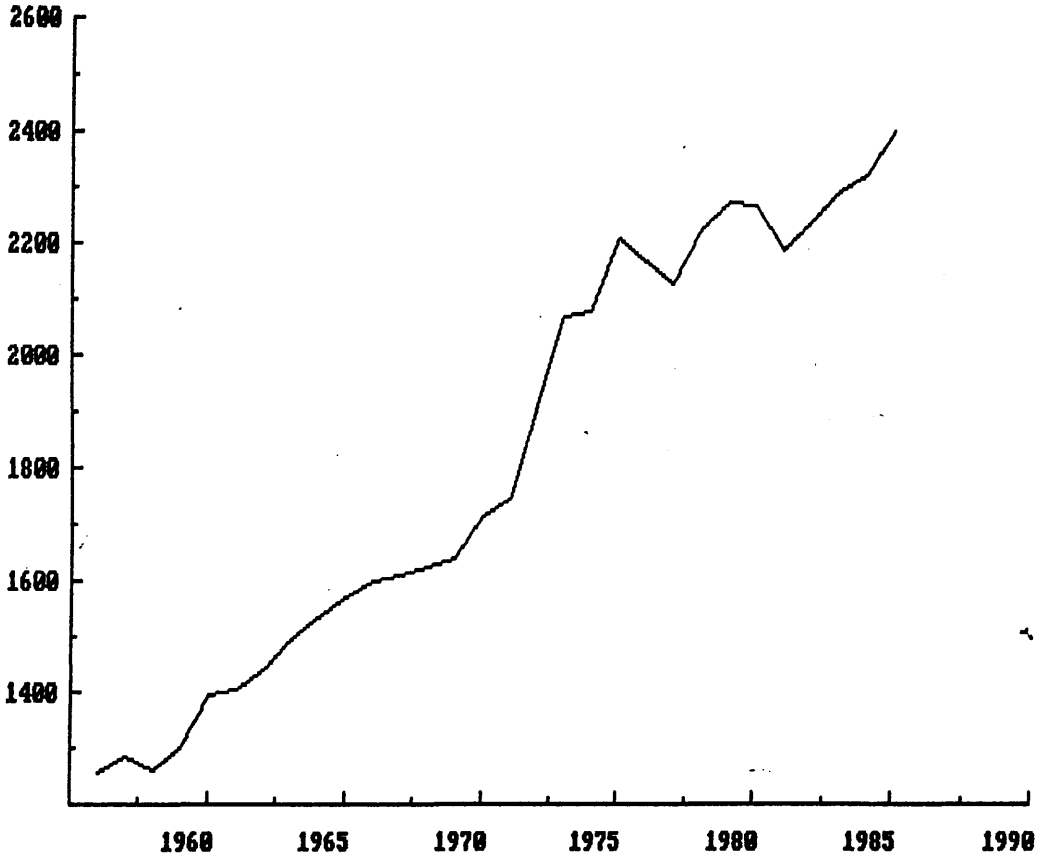


Figure 7.9 Male Unemployment in Scotland

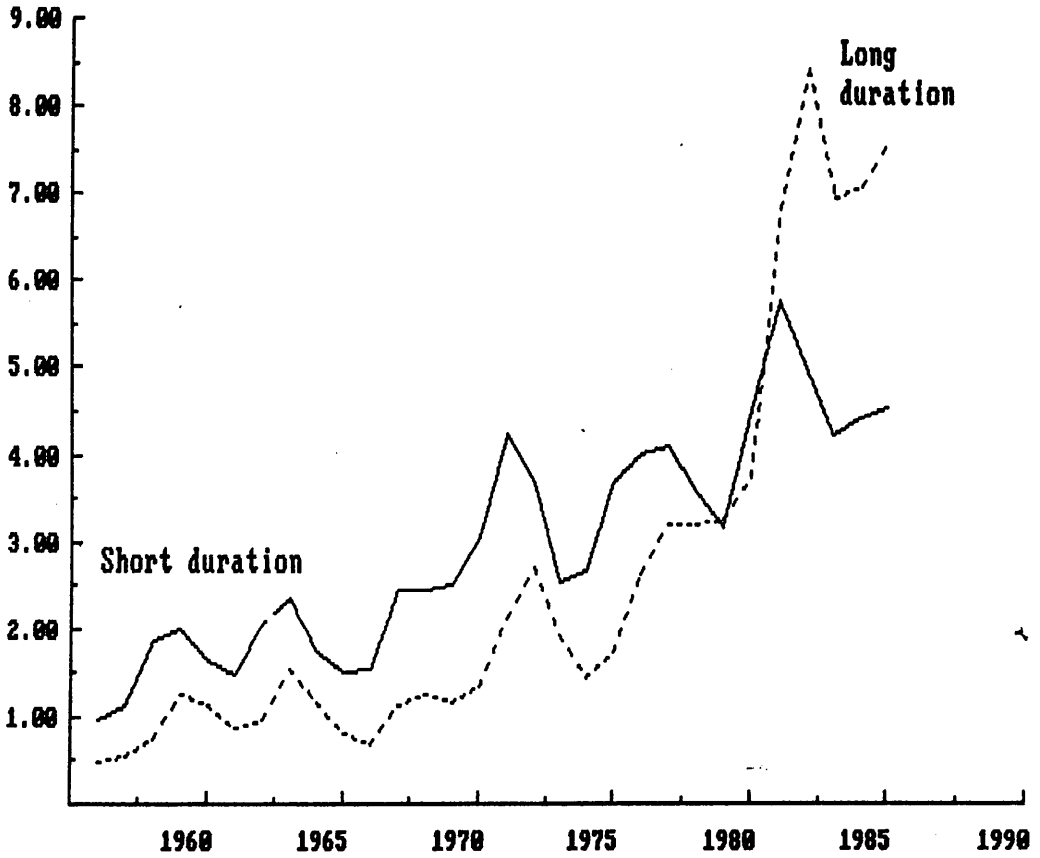


Figure 7.10 Ratio of Long to Short Duration Unemployment Rates

**Figure 7.11 Actual and Fitted Values (Equation 2)**
C =____ FITTED =-- --

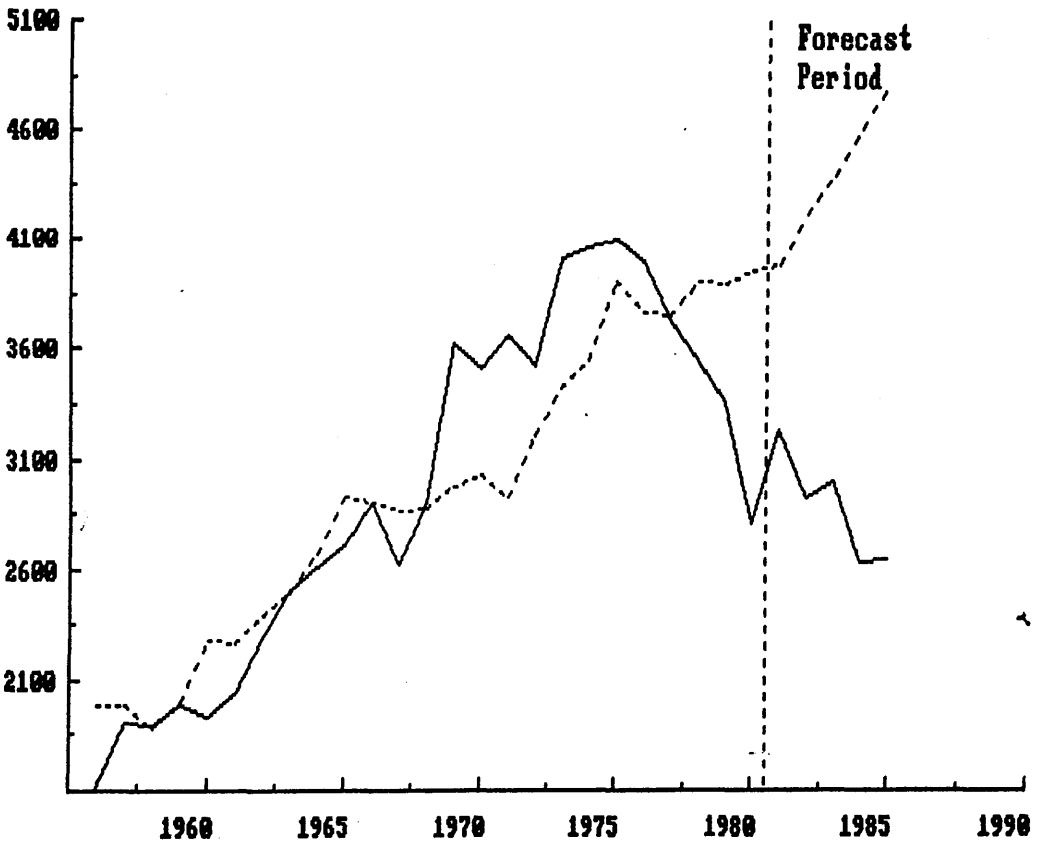


Figure 7.12 Parameter Stability Over Time (Equation 2a)
PC =____ ± 2*S.E.=— —

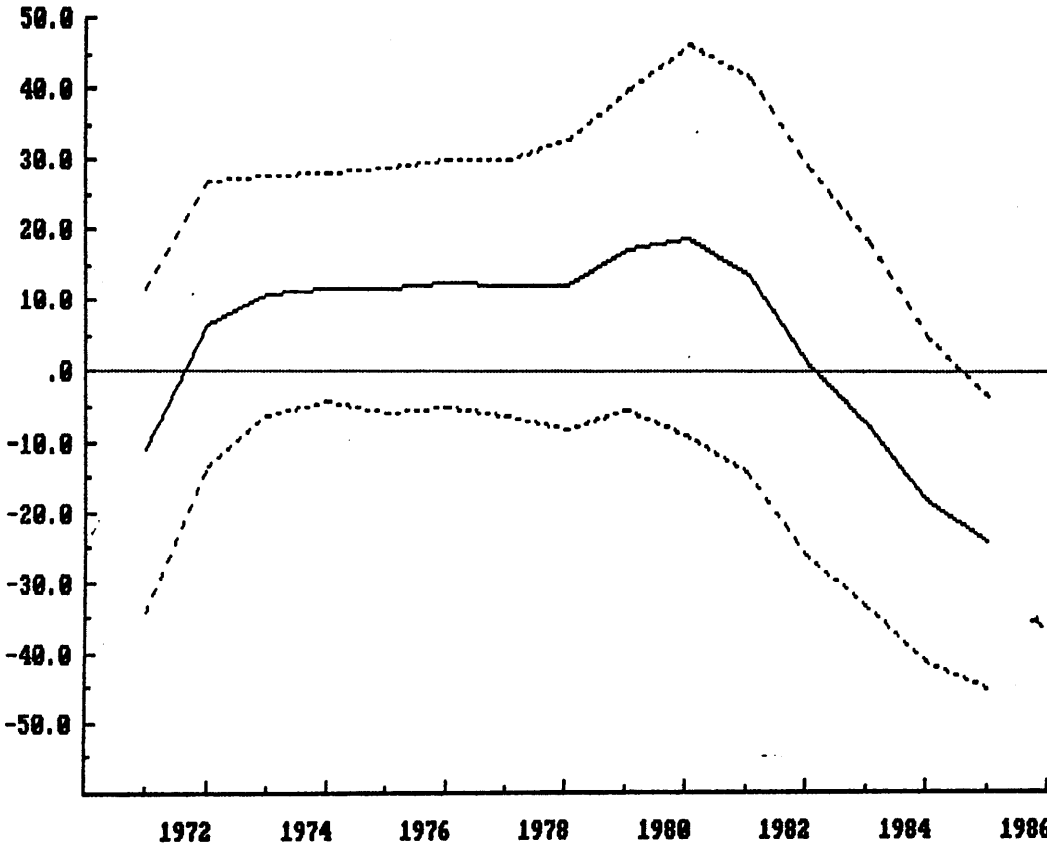


Figure 7.13 Chow Test of Parameter Stability (Equation 2a)

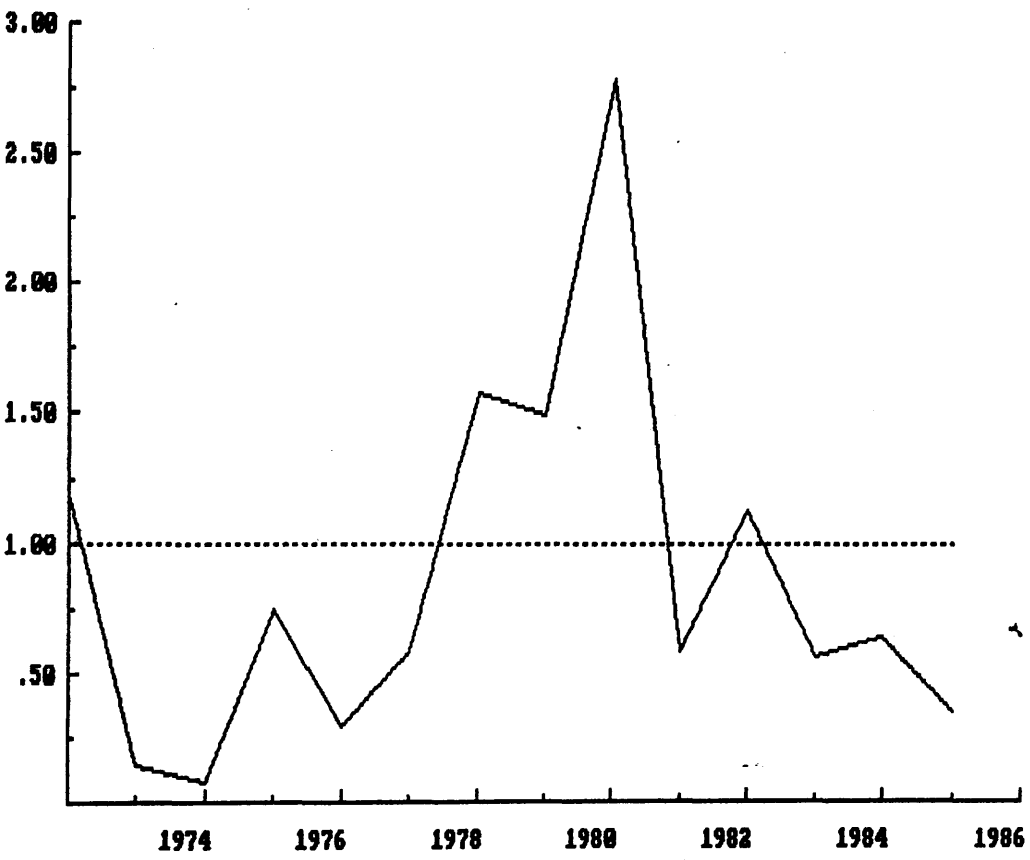


TABLE A7.1 INCOME, UNEMPLOYMENT AND BEHAVIOURAL VARIABLES

| | VARIABLE | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| YEAR | GDPTREND | GDPDEV | ΔGDP/GDP | PDI | UNEMsd | UNEMld | C | PC | A | PA |
| 1956 | 1890 | 4.86 | 0.06 | 1257 | 0.96 | 0.48 | 1610 | 119.00 | 4.10 | 125.80 |
| 1957 | 1950 | 4.44 | 2.66 | 1287 | 1.11 | 0.55 | 1902 | 115.30 | 4.14 | 125.20 |
| 1958 | 2009 | -2.08 | 3.51 | 1261 | 1.87 | 0.74 | 1893 | 113.20 | 4.05 | 122.10 |
| 1959 | 2068 | -3.02 | 1.93 | 1301 | 1.98 | 1.25 | 1983 | 114.20 | 4.27 | 114.00 |
| 1960 | 2128 | -0.41 | 5.34 | 1393 | 1.65 | 1.14 | 1930 | 118.10 | 4.45 | 112.60 |
| 1961 | 2187 | -3.02 | 0.10 | 1406 | 1.47 | 0.86 | 2030 | 115.60 | 4.49 | 110.10 |
| 1962 | 2247 | -3.44 | 2.22 | 1440 | 2.07 | 0.97 | 2295 | 118.30 | 4.22 | 114.00 |
| 1963 | 2306 | -2.84 | 3.18 | 1494 | 2.35 | 1.54 | 2502 | 117.20 | 4.66 | 116.00 |
| 1964 | 2366 | -1.65 | 3.69 | 1536 | 1.73 | 1.16 | 2603 | 121.50 | 4.90 | 120.10 |
| 1965 | 2425 | -1.64 | 2.46 | 1570 | 1.50 | 0.79 | 2710 | 130.50 | 4.84 | 124.00 |
| 1966 | 2484 | -2.51 | 1.52 | 1599 | 1.53 | 0.66 | 2898 | 125.60 | 4.93 | 119.30 |
| 1967 | 2544 | -3.13 | 1.71 | 1610 | 2.44 | 1.11 | 2623 | 122.70 | 5.09 | 122.70 |
| 1968 | 2603 | -2.02 | 3.40 | 1623 | 2.45 | 1.25 | 2904 | 121.80 | 5.16 | 118.90 |
| 1969 | 2663 | -2.68 | 1.56 | 1642 | 2.52 | 1.17 | 3633 | 124.90 | 5.24 | 120.40 |
| 1970 | 2722 | -2.34 | 2.53 | 1716 | 3.05 | 1.34 | 3522 | 118.10 | 5.57 | 120.00 |
| 1971 | 2781 | -1.38 | 3.08 | 1744 | 4.25 | 2.12 | 3663 | 109.30 | 5.83 | 115.80 |
| 1972 | 2841 | -0.07 | 3.38 | 1917 | 3.71 | 2.71 | 3533 | 103.00 | 6.14 | 113.70 |
| 1973 | 2900 | 6.59 | 8.17 | 2068 | 2.54 | 1.93 | 4017 | 96.00 | 6.82 | 107.50 |
| 1974 | 2960 | 4.85 | 0.38 | 2080 | 2.68 | 1.45 | 4068 | 101.00 | 7.01 | 103.40 |
| 1975 | 3019 | 7.96 | 4.79 | 2209 | 3.69 | 1.72 | 4105 | 104.30 | 7.08 | 103.40 |
| 1976 | 3078 | 8.82 | 2.70 | 2172 | 4.02 | 2.62 | 4000 | 101.40 | 7.46 | 103.90 |
| 1977 | 3138 | 6.91 | 0.15 | 2128 | 4.12 | 3.20 | 3735 | 106.30 | 7.12 | 101.80 |
| 1978 | 3197 | 6.56 | 1.53 | 2226 | 3.60 | 3.23 | 3553 | 102.60 | 7.68 | 101.30 |
| 1979 | 3257 | 3.80 | 0.79 | 2277 | 3.19 | 3.27 | 3363 | 95.30 | 7.98 | 97.00 |
| 1980 | 3316 | -2.45 | 4.50 | 2269 | 4.54 | 3.75 | 2815 | 100.00 | 7.66 | 100.00 |
| 1981 | 3375 | -6.60 | 2.61 | 2192 | 5.78 | 6.86 | 3232 | 110.50 | 7.45 | 105.20 |
| 1982 | 3435 | -5.09 | 3.29 | 2239 | 5.03 | 8.44 | 2923 | 117.20 | 7.39 | 107.30 |
| 1983 | 3494 | -3.17 | 3.65 | 2295 | 4.23 | 6.96 | 3007 | 119.70 | 7.69 | 111.70 |
| 1984 | 3554 | -4.15 | 0.67 | 2324 | 4.45 | 7.09 | 2634 | 128.20 | 7.78 | 111.90 |
| 1985 | 3613 | -1.87 | 3.93 | 2404 | 4.57 | 7.64 | 2644 | 129.20 | 7.89 | 110.90 |

NOTES: Variables are defined in Table 7.2 and in text.

## CHAPTER 8 SUMMARY

**"The death rates of young children are, in my opinion, among the most important studies in sanitary science...their tender young lives, as compared with the more hardened and acclimatised lives of the adult population, furnish a very sensitive test of sanitary circumstances..."**

**(Simon, 1858)**

The health of the newborn has long been and will undoubtedly continue to be an important topic. Beginning with early studies examining or simply speculating on the causes of infant and perinatal deaths, an increasing array of biological demographic, socio-economic, behavioural, environmental and health service factors have been associated with infant health outcomes. However, the distinctions between such factors, the extent to which they interact with and influence each other and their precise impact on the risk of mortality is often unclear.

To a large extent this can be traced to the descriptive, non-experimental methods of analysis traditionally employed in studies of perinatal mortality. Numerous studies, for instance, have demonstrated that the association between specific factors and reproductive outcomes depends on a variety of methodological considerations: for example, whether cross-sectional or longitudinal methods of analysis are employed or whether the direct and interactive effects of other variables are accounted for. A further explanation is the variety of perspectives adopted by investigators from different disciplines. For example, even when addressing

similar topics, clinicians, epidemiologists and social scientists tend to pose different research questions, use different conceptual and methodological approaches and draw different interpretations and conclusions from their research findings.

This thesis presented a new perspective on the determinants of perinatal health in post-war Scotland. Unlike virtually all previous work which has examined cross-sectional data this study developed and tested a variety of time series models which collectively provide a more informed explanation for the improvement in perinatal mortality over time.

Chapter 2 surveyed the economic approach to the analysis of mortality determinants. Unlike econometric studies where the selection and relationship between variables can, to some extent, be guided by economic theory, models of health production are not easily derived from the economic theory typically used to analyze the behaviour and production activities of firms. Uncritical acceptance of the assumptions and theoretical framework of traditional economic models can easily increase the likelihood of ignoring important determinants of health or imposing implausible relationships on observed associations between inputs and health outcomes.

The theoretical model based on the "new home economics" provides one description of individual behaviour. Blaug (1980) has described this approach, attempting to explain

social phenomenon by tracing its roots back to individual behaviour, as that of "methodological individualism". Throughout the human capital research programme, including that concerned with health, the emphasis is on testing the hypotheses of behavioural models constructed at the individual level. Consequently, little attention has been directed towards developing aggregate level models nor to investigating their possible micro-foundations. This remains an important gap in the human capital literature since it is not readily apparent whether an individual level focus is the most appropriate or advantageous way of investigating population or group phenomenon.

Since no detailed specification of aggregate level models has yet emerged from the human capital research programme, previous studies conducted at the aggregate level use the human capital paradigm as the point of departure for empirical analyses in the spirit of previous work formulated at the micro-level. However, it provides only limited information about the precise specification of aggregate models which allows for the dynamic, multi-variate nature of demographic change in populations. Perhaps inadvertantly because of an over-reliance on tenuous and generally unchallenged theoretical constructs the associated empirical analysis has tended to be overly restrictive.

Despite their collective allegiance to the household production approach, most of the studies reviewed in Chapter 3 closely resemble empirical studies which have traditionally employed little or no theoretical framework. In principle, the theoretical models provide a context for structuring empirical studies and suggesting what variables could be important determinants of health. In practice, particularly in studies using aggregate data, they are rarely used to define and estimate structural relationships between measurable variables as a way of establishing the causal influences on infant health or pregnancy outcomes. Furthermore, its use in formulating empirical models which convincingly test theoretical predictions and enable discrimination between competing theoretical models or alternative explanations for observed empirical regularities has been limited. This may reflect the embryonic development and consequent application of these models to infant health as well as the problems arising from the generality of the theoretical framework, a lack of competing theoretical models (economic or otherwise), the paucity of testable predictions concerning the health of infants and the formidable data requirements which must be satisfied to allow the estimation of structural relations between largely unobservable variables.

This survey of empirical studies of perinatal and infant health conducted within an economic framework highlighted a number of points. First, no time series or longitudinal

study has been reported largely due to the fact that a consistent and reasonably long time series on infant health outcomes and inputs has not been assembled. Attempts to bridge this gap have tended to take the form of "explaining" the change in neonatal and infant mortality over time using cross-sectional regression coefficients in conjunction with national trends in the "exogenous" variables. The limitations of this approach highlight the need for a direct examination of time series data as a way of improving our understanding of the factors which have contributed to the improvement in health outcomes over time.

A common feature of the aggregate level studies is the relatively limited set of mortality rates that have been used to measure infant health outcomes. Although mortality rates have been disaggregated by age, sex and race no study has analyzed how inputs may differentially influence the survival of infants in different birthweight groups or strata. Another obvious and long overdue disaggregation of mortality data is by cause of death. Although the results based on total or overall mortality rates provide an estimate of the average impact this may obscure a wide range of effects in different sub-groups of the population.

Chapter 4 generated a time series of dis-aggregated perinatal mortality rates which overcomes some of these limitations. Throughout the post-war period perinatal mortality has fallen in most, if not all, industrialised countries.

Scotland is no exception. Over the post-war period Scotland sustained an annual decline in her perinatal mortality rate of just over 4 per cent per year: a rate of improvement twice as great as her post-war annual rate of economic growth (2.1 per cent).

Perinatal mortality, however, has not declined at a constant rate. Although impressive improvement occurred during the war years the 1950's and 1960's were characterised by sluggish rates of decline. The most rapid change in perinatal mortality rates occurred over the period 1975-1985. Improvement occurred first in the late neonatal mortality rate (7-27 days), followed by the 1-6 day rate and finally the < 1 day rate. Throughout the late 1950's and early 1960's the < 1 day rate remained virtually static. Although rates improved during the late 1960's, over 25 per cent of the overall decline in the < 1 day mortality rate occurred between 1976 and 1985.

Trends in cause-specific stillbirth and neonatal mortality have tended to exert a countervailing influence on each other. Thus, although the proportion of stillbirths due to malformations has declined this has been accompanied by an increase in the proportional neonatal mortality rate for malformations. As a result, the principal causes of perinatal death remained remarkably constant. For nearly three decades, congenital malformations have accounted for 20-24 per cent of perinatal deaths. Placental and cord con-

ditions have been responsible for another 17-20 per cent of perinatal deaths. Finally, prematurity and respiratory problems have accounted for about a third of perinatal deaths.

Birthweight is one of the most important factors influencing perinatal mortality. Although mortality has declined in all birthweight strata over time, the post-war period has been characterised by divergent rates of improvement which have increased the relative risk of perinatal mortality in the premature infant. The most impressive gains occurred not in those infants facing the greatest risks of death but rather in the heavier infant who apparently benefited most from the concurrent changes in the social and medical determinants of perinatal mortality in Scotland.

These trends in perinatal mortality, however, do not appear to have influenced the proportion of total hospital expenditure devoted to perinatal care in Scotland. This result emerged from the analysis presented in Chapter 5 which developed a model for describing the post-war pattern of public expenditure on perinatal hospital services in Scotland. Total public spending on hospital care in Scotland appears to have had no consistent and clear impact on the perinatal hospital budget share. Prior to the peak in the fertility rate in the 1960's perinatal care perhaps represented a necessity or even luxury good as expenditure struggled to keep up with the "baby boom" and growing demand

for maternity services as norms changed for hospital delivery. The subsequent "baby bust" has ushered in an era where expenditure on perinatal care has increasingly been seen as an inferior good.

Relative prices or costs of perinatal care in relation to other forms of hospital care are important determinants of expenditure. As its price increases the share of hospital expenditure on perinatal care increases. This may reflect the inflexibility of the quantity of existing hospital services in the short run; thus current relative price changes are transmitted directly into current expenditure shares.

Several different specifications of the demand model provided a good fit for the data and survived tests of parameter stability and stochastic specification. The partial adjustment model suggested that virtually all the adjustment required to achieve desired perinatal budget shares occurs within five years: a plausible planning horizon for marginal shifts in the pattern of hospital expenditure. An error correction model also supported the hypothesis that disequilibirium between target and actual perinatal shares was an important determinant of the change in budget shares over time.

**"Until the laws of thermodynamics are repealed, I shall continue to relate outputs to inputs, i.e., to believe in production functions"**

**(Samuelson, 1966)**

Relating inputs to outputs is a perennial problem in health production studies. Many characteristic features of input-output relations in infant health fail to conform to the standard models of production that have been routinely employed in previous health production studies. The choice of functional form for relating inputs to outputs should therefore not be based on convenience - a practice which imposes familiar functional forms borrowed from textbook analyses of production functions - but rather according to careful empirical testing of different specifications which are flexible enough to accommodate realistic relationships between inputs and health outputs.

A characteristic features and inherent limitation of all of the empirical studies surveyed is their uncritical approach to model specification, validation and testing. An alternative approach, derived in large part from the early work of Denis Sargan (1964) and most closely associated with the work of David Hendry, is to begin with a very general specification which is systematically reduced or simplified and then rigorously tested. This general to specific model simplication does not assume that one knows the "correct" or "true" model but rather that well conducted specification

searches and "destructive" model testing will increase the likelihood of discovering models that adequately characterise the available data. The hallmark of this approach involves the full disclosure of the path leading to a preferred model and the results generated by tests addressing the problem of "quality control" of the preferred model

Adopting this methodological approach Chapter 6 presented a number of perinatal mortality production functions estimated using time series data. A wide range of different functional forms and specifications were carefully tested and evaluated. In general the restrictions imposed by a simple linear Leontief production function were found to be acceptable compared to more complicated and flexible functional forms. The widely used Cobb-Douglas specification was found to be suspect both on *a priori* and empirical grounds.

The results confirmed that hospital medical and nursing inputs are important determinants of perinatal mortality across different birthweight strata. Increases in the number of obstetricians and nurses have a beneficial and similar impact on mortality in low birthweight infants. Nurses also appear to have a more important impact on mortality in the normal weight infant. Decreases in the bed stock are associated with improvements in perinatal mortality, particularly for low birthweight infants. This supports the hypothesis that the shift over time in the mix of

obstetric beds away from small maternity units and wards towards larger and more specialised units is associated with an improvement in mortality. Thus, an increase in the (average) scale of maternity units accompanied by a reduction in the total maternity capital stock appears to have led to an improvement in the health outcomes of those infants who faced the greatest risks of mortality.

The estimates also suggest that the elasticity of perinatal mortality with respect to expenditure per birth ranges from -0.443 in very low birthweight infants to -1.005 in normal weight babies. Applying these elasticities to the mean numbers of births and the distribution of birthweight suggests that the incremental cost per year of potential life gained ranges from just under £7000 pounds in the very low birthweight infant to around £2300 in the normal birthweight infant (1985 prices and 5 per cent discount rate). In terms of cost per life year gained, investment in perinatal hospital services has generated health outcomes at a cost which is lower than many other health care programmes whose costs and effectiveness have been evaluated from an economic perspective (Torrance, 1986).

But what of changes outwith the health sector? Chapter 7 investigated the possible links between income, unemployment and infant health. The main findings failed to support the hypothesis that unemployment excerts an impact on perinatal and post-neonatal mortality, let alone an adverse impact.

These results cast serious doubt on the robustness of the intuitively appealing notion that health outcomes in infancy are intimately linked with the economic health of a population as measured by the level of unemployment. In addition, measures of economic instability (short and long-run changes in economic growth) seem to play little role in determining the chances of survival of new born children. These results for mortality at the start of life confirm the general findings reported for the impact of unemployment on adult mortality by workers who have used a critical approach to model specification and testing (Gravelle et al, 1981; Forbes and McGregor, 1984).

This thesis has ranged across a number of topics. Despite the array of methodological problems, I do believe that the empirical analysis has made a small contribution to improving our understanding of the impact of perinatal health services on health outcomes. Obviously, the analysis could be improved. The crude measures of medical care inputs need to be replaced by more refined indices of the resources going into the production process. Health indicators which, unlike mortality rates, can apply to more than 1 per cent of the perinatal population need to be developed and routinely used in the evaluation of perinatal health services. The relationship between inputs and outputs, moreover, should be carefully investigated and the traditional modes of analysis

which perhaps were sufficient to analyse production in manufacturing and industrial sectors of the economy should be critically reviewed and modified as required.

Like many time series of aggregate economic data, the series used in this study did not satisfy the assumption of stationarity. The consequences of non-stationarity for the usual statistical properties of estimators and hypothesis tests are well known. One increasingly prominent method of addressing these problems in the analysis of non-stationary time series is through the use of "cointegrated" techniques (Granger, 1981). The rationale for such cointegrated processes could be a market mechanism based on equilibrium concepts relating supply and demand, goverment intervention or, more generally, error correction models which combine non-stationary and stationary elements to explain long-run equilibrium and short-run dynamics. The cointegration approach to the analysis of macroeconomic time series has grown in popularity over recent years. One promising area of further research in the population and health field is likely to be the application of such models within a time series modelling framework.

Finally, perhaps the greatest barrier to progress in this area is that imposed by economists on themselves. More work by economists should be directed towards, and ideally should proceed in collaboration with, non-economists who are approaching the same problems from a different perspective.

Economists do have a contribution to make in this field. Their contribution may be enhanced if they take the time to reflect on what they can learn from their clinical and epidemiological colleagues who I believe can benefit from the insights offered by the economic perspective.

BIBLIOGRAPHY

Abel EL. (1980) Smoking during pregnancy: a review of effects on growth and development of offspring. Human Biology; 52: 593-625.

Abel EL. (1982) Consumption of alcohol during pregnancy: a review of effects on growth and development of offspring. Human Biology; 54: 421-453.

Andrews DF. (1971) A note on the selection of data transformations. Biometrica; 58: 249-254.

Arrow KJ. (1962) The economic implications of learning by doing. Review of Economic Studies; 29: 153-173.

Arrow KJ. (1963) Uncertainty and the welfare economics of medical care. American Economic Review; 53: 941-773.

Austen R, Leveson I, Savachek D. (1969) The production of health: an exploratory study. Journal of Human Resources; 4: 411-436.

Baird D, Wyper JFB. (1941) High stillbirth and neonatal mortality. Lancet; ii: 657-659.

Baird D. (1945) The influence of social and economic factors on stillbirths and neonatal deaths. Journal of Obstetrics and Gynaecology of the British Empire; 52: 219-234.

Baird D. (1947) Social class and foetal mortality. Lancet; ii: 531-534.

Baird D. (1949) Social factors in obstetrics. Lancet; i: 1079-1083.

Baird D, Walker J, Thomson AM. (1954) The causes and prevention of stillbirths and first week deaths. Journal of Obstetrics and Gynaecology of the British Empire; 61: 433-448.

Baird D. (1980) Environment and reproduction. British Journal of Obstetrics and Gynaecology; 87: 1057-1067.

Barr A. (1974) Maternity Hospital Costs. (Occasional paper No 1) Oxford: Oxford Regional Health Authority.

Bayer R. (1982) Reproduction hazards in the workplace: bearing the burden of fetal risks. Millbank Memorial Fund Quarterly. 60: 633-656.

Becker GS. (1960) An economic analysis of fertility in Demographic and Economic Change in Development Countries. NBER special conference series No. 11 Princeton: Princeton University Press.

Becker GS. (1964) Human Capital. New York: Columbia University Press for the National Bureau of Economic Research.

Becker GS. (1965) A theory of the allocation of time. Economic Journal; 75: 497-517.

Becker GS. (1976) The Economic Approach to Human Behaviour. Chicago: University of Chicago Press.

Becker GS. (1981) A Treatise on the Family. Cambridge Massachusetts. Harvard University Press.

Behrman JR, Wolfe BC. (1984) A more general approach to fertility determination in a developing country: the importance of biological supply considerations, endogeneous tastes and unperceived jointness. Economica; 51: 319-339.

Ben-Porath Y. (1982) Economics and the family - match or mismatch? A review of Becker's *A Treatise on the Family.* Journal of Economic Literature; 20: 52-64.

Bera AK, McAleer M. (1983) Further results on testing linear and log-linear regression models. Paper presented at the SSRC Econometric Group Conference on model specification and testing.

Blake J. (1968) Are babies consumer durables? A critique of the economic theory of reproduction motivation. Population Studies; 22: 5-25.

Blaug M. (1963) A survey of the theory of process innovation. Economica; 30: 13-32.

Blaug M. (1976) Human capital theory: A slightly jaundiced survey. Journal of Economic Literature; 14: 827-855.

Blaug M. (1980) The Methodology of Economics or How Economists Explain. Cambridge: Cambridge University Press.

Bockstael NE, McConnell KE. (1983) Welfare measurement in the household production framework. American Economic Review; 73: 806-814.

Bound JP, Butler NR, Spector NG. (1956) Classification and causes of perinatal mortality. British Medical Journal; 2: 1191-1196 and 1260-1265.

Boyle MH. et al (1983) Economic evaluation of neonatal intensive care of very low birthweight infants. New England Journal of Medicine; 308: 1330-1337.

Brenner MH. (1973) Fetal, infant and maternal mortality during periods of economic instability. International Journal of Health Services; 3 145-159.

Brenner MH. (1979) Mortality and the national economy: A review and the experience of England and Wales, 1936-76. Lancet; 2: 568-573.

Brenner MH. (1987) Economic instability, unemployment rates, behavioral risks and mortality rates in Scotland, 1952-1983. International Journal of Health Services; 17: 475-487.

Breusch TS. (1978) Testing for autocorrelation in dynamic linear models. Australian Economic Papers; 17: 334-355.

Brown TM. (1952) Habit persistence and lags in consumer behavior. Econometrica; 20: 355-371.

Butler NR, Bonham DG. (1963) Perinatal Mortality. Edinburgh: Churchill Livingston.

Butz WP, Ward MP. (1979) The emergence of countercyclical U.S. fertility. American Economic Review; 69: 318-328.

Campbell JY. (1987) Does saving anticipate declining labor income? An alternative test of the permanent income hypothesis. Econometrica; 55: 1249-1274.

Carr-Hill RA, Hardman GF, Russell IT. (1987) Variations in avoidable mortality and variations in health care resources. Lancet; i: 789-791

Catalano R, Serxner S (1987) Time series designs of potential interest to epidemiologists. American Journal of Epidermiology; 126: 724-731.

Chalmers I. et al (1978) Adjusted perinatal mortality rates in administrative areas of England and Wales. Health Trends; 10: 24-29.

Chalmers I, Sinclair JC. (1985) Promoting perinatal health: is it time for a change in emphasis in research? Early Human Development; 10: 171-191.

Chapman PF, Fryer JG. (1980) Birthweight and the linear statistical model. Bulletin in Applied Statistics; 7: 267-306.

Chizman JF, Zak TA. (1983) Modelling multiple outputs in educational production functions. American Economic Review; 73: 18-22.

Coale AJ, Hoover EIM. (1958) Population Growth and Economic Development in Low Income Countries: A Case study of India's Prospects. Princeton: Princeton University Press.

Cole SK. et al (1986) Classifying perinatal death: an obstetric approach. British Journal of Obstetrics and Gynaecology; 93: 1204-1212.

Cole SK. (1988) Scottish Stillbirth and Neonatal Death Report 1987. Edinburgh: Information and Statistics Division, Common Services Agency for the Scottish Health Service.

Conger AJ, Campbell JM. (1978) Simultaneity in the birth rate equation: The effects of education, labour force participation, income and health. Econometrica; 46: 631-642.

Corman H, Grossman M. (1985) Determinants of neonatal mortality rates in the U.S.: A reduced form model. Journal of Health Economics; 4: 1985.

Corman M, Joyce TJ, Grossman M. (1987) Birth outcome production function in the United States. Journal of Human Resources; 22: 339-360.

Cropper ML. (1977) Health, investment in health and occupational choice. Journal of Political Economy; 85: 1273-1294.

David RJ, Siegel E. (1983) Decrease in neonatal mortality, 1968-1977: Better babies or better care? Paediatric Research; 14:487.

Davidson R, Mackinnon JG. (1985) Testing linear and log-linear regressions against Box-Cox alternatives. Canadian Journal of Economics; 18: 499-517.

Deacon RT. (1978) A demand model for the local public sector. Review of Economics and Statistics; 60: 184-192.

Deaton A, Muellbauer J. (1980) An almost ideal demand system. American Economic Review; 70: 312-326.

Diewert WE. (1971) An application of the Shephard duality theorem: A generalised Leontief production function. Journal of Political Economy; 79: 481-507.

Douglas CA. (1945) Maternal and child health under war conditions. Transactions of the Royal Sanitary Association of Scotland.

Douglas CA, McKinlay PL. (1953) The incidence of prematurity in Scotland and its importance in early infant mortality. Health Bulletin; 11: 4-6.

Douglas CA. (1954) Trends in the risks of childbearing and in the mortalities of infants living the last 30 years. British Journal of Obstetrics and Gynaecology; 61: 216-223.

Douglas Ca, McKinlay PL. (1955) A further note on prematurity in Scotland. Health bulletin; 12: 22-24.

Douglas CA, McKinlay PL. (1959) Maternal and infant mortality in Scotland. Health Bulletin; 17: 17-44.

Drillien CM. (1947) Studies in prematurity, stillbirth and neonatal death. Journal of Obstetrics aand Gynaecology of the British Empire; 54: 300-323 and 443-468.

Drobny AD, Hall SG (1987) An investigation of the long-run properties of aggregate non-durable consumers' expenditure in the UK. National Institute Discussion Papers Number 128. London: National Institute of Economic and Social Research.

Duncan E, Baird D, Thomson A. (1952) The causes and prevention of stillbirths and first week deaths. Part I. The evidence of vital statistics. Journal of Obstetrics and Gynaeocology of the British Empire; 59: 183-196.

Dunne JP, Smith RP. (1983) The allocative efficiency of government expenditure. European Econommic Review; 20: 381-394.

Dunne JP, Pashardes P, Smith RP (1984) Needs, costs and bureaucracy: the allocation of public consumption in the UK. Economic Journal; 94: 1-15.

Easterlin R, Pollak RA, Wachter MC. (1980) Towards a more general economic model of fertility determination: endogenous preferences and natural fertility in Easterlin R (ed) Population and Economic Change in Less Developed Countries. Chicago: University of Chicago Press.

Edouard L, Alberman E. (1980) National trends in the certified causes of perinatal mortality, 1968-1978. British Journal of Obstetrics and Gynaecology; 87: 833-838.

Eisner R, Strotz RH. (1963) Determinants of business investment in Commision of Money and Credit: Impacts of Monetary Policy; Englewood Cliffs, N.J.: Prentice-Hall.

Engle RF, Granger CWJ (1987) Cointegration and error-correction: representation, estimation, and testing. Econometrica; 55: 251-276.

Erkkola R. et al (1982) Monitoring perinatal mortality by birthweight specific mortality rates. International Journal of Gynaecology and Obstetrics; 20: 231-235.

Ermisch J. (1983) The Political Economy of Demographic Change. London: Heinemann.

Ermish J. (1988) Economic influences on birth rates. National Institute Economic Review; November 1988: 71-81.

Evans RG, Bauer ML. (1983) Prices, proxies and productivity: an historical analysis of hospital and medical care in Canada in Diewert WE, Montmarquette (eds) Price Level Measurement; Ottowa: Minister of Supply and Services Canada.

Eyer J. (1977) Prosperity as a cause of death. International Journal of Health Services; 7: 125-150.

Feldstein MS. (1965) Improving the use of hospital maternity beds. Operational Research Quarterly; 16: 65-76.

Feldstein MS. (1965) A method of evaluating perinatal mortality risk. British Journal of Preventive and Social Medicine; 19: 135-139.

Feldstein MS. (1966) A binary variable multiple regression method of analysing factors affecting perinatal mortality and other outcomes of pregnancy. Journal of the Royal Statistical Society (Section A); 129:61-73

Feldstein MS, Butler NR. (1966) Analysis of factors affecting perinatal mortality: a multivariate statistical approach. British Journal of Preventive and Social Medicine; 19: 128-134.

Feldstein MS. (1967) Economic Analysis for Health Service Efficiency; Econometric Studies of the British National Health Service. Amsterdam : North-Holland.

Feldstein MS. (1974) Econometric studies of health economics in Intrilligator MD, Kendrick DA.(eds) Frontiers of Quantitative Economics (vol II); Amsterdam: North Holland.

Fellner W. (1969) Specific examples of learning by doing. Journal of Economic Theory; 1: 119-140.

Forbes JF. (1981) Unemployment and health. Lancet; ii: 875.

Forbes JF. et al (1982) Perinatal mortality in Scotland: 1970-9. Journal of Epidemiology and Community Health; 36: 282-288

Forbes JF. (1983) Population Economics: A survey of recent research. Scottish Journal of Political Economy; 30: 88-96.

Forbes JF, Smalls MR. (1983) A comparative analysis of birthweight for gestational age standards. British Journal of Obstetrics and Gynaecology; 99:297-303.

Forbes JF, McGregor A (1984) Unemployment and mortality in post-war Scotland. Journal of Health Economics; 3: 239-257.

Forbes JF, Pickering RM. (1985) Influence of maternal age, parity and social class on perinatal mortality in Scotland: 1960-83. Journal of Biosocial Science; 17:339-349.

Forbes JF, McGregor A. (1987) Male unemployment and cause-specific mortality in post-war Scotland. International Journal of Health Services; 17: 233-240.

Forbes JF, Lassen KE, Bakketeig LS. (1987) Access to intensive neonatal care and neonatal survival in low birthweight infants; A population study in Norway. Paediatric and Perinatal Epidemiology; 1: 33-42.

Fuchs VR. (1974) Who Shall Live? Health, Economics and Social Choice. New York: Basic Books.

Fuchs VR. (1981) Low-level radiation and infant mortality. Health Physics; 40: 847-854.

Fuchs VR. (1982) Time preference and health: an exploratory study in Fuchs VR. (ed) Economics Aspects of Health; Chicago: University of Chicago Press for the NBER.

Fuchs VR. (1983) How We Live: An Economic Perspective on Americans from Birth to Death. Cambridge, Massachusetts: Harvard University Press.

Garside W. (1980) The Measurement of Unemployment. Oxford: Basil Blackwell.

Gilbert CL. (1986) Professor Hendry's econometric methodology. Oxford Bulletin of Economics and Statistics; 48: 283-307.

Godfrey LG. (1978) Testing against autoregressive and moving average error models when the regressors include lagged dependent variables. Econometrica; 46: 1293-1301.

Godfrey LG, Wickens MR. (1981) Testing linear and log-linear regressions for functional form. Review of Economic Studies; 48: 487-496.

Godfrey C, Maynard A. (1988) Economic aspects of tobacco use and taxation policy. British Medical Journal; ii: 339-343.

Goldman F, Grossman M. (1978) The demand for paediatric care: an hedonic approach. Journal of Political Economy; 86: 259-280.

Goldman F, Grossman M. (1982) The Impact of Public Health Policy: The Case of Community Health Centers. National Bureau Economic Research, Working Paper Series No 1020.

Granger CWJ, Newbold P. (1974) Spurious regressions in econometrics. Journal of Econometrics; 2: 11-120.

Granger CWJ (1981) Some properties of time series data and their use in econometric model specification. Journal of Econometrics; 121-130.

Granger CWJ (1983) Co-integrated variables and error-correcting models. Mimeo, UCSD.

Granger CWJ (1986) Developments in the study of cointegrated economic variables. Oxford Bulletin of Economics and Statistics; 48: 213-227.

Gravelle HSE, Hutchinson G, Stern J. (1981) Mortality and unemployment: a critique of Brenner's time-series analysis. Lancet; ii: 675-679.

Gravelle HSE, Backhouse ME. (1987) International cross-section analysis of the determination of mortality. Social Sciences and Medicine; 25: 427-441.

Gray A, McGuire A. (1987) Factor Trends in the NHS Hospital Sector. Paper presented at the Health Economists' Study Group; University of Warwick, January 1987.

Grossman M. (1972) The Demand for Health: A Theoretical and Empirical Investigation. New York: National Bureau of Economic Research.

Grossman M, Jacobowitz S. (1981) Variations in infant mortality rates among countries of the United States: the roles of public policies and programs. Demography; 18: 695-713.

Hadley J. (1982) More Medical Care, Better Health? An Economic Analysis of Mortality Rates. Washington: The Urban Institute.

Hall SG (1986) An application of the Granger and Engle two-step estimation procedure to United Kingdom aggregate wage data. Oxford Bulletin of Economics and Statistics; 48: 229-239.

Hannan MT. (1982) Families, markets and social structures: an essay on Becker's *A Treatise on the Family*. Journal of Economic Literature; 20: 65-72.

Hansen AH. (1939) Economic progress and declining population growth. American Economic Review; 29: 1-15.

Harris JE. (1982) Prenatal medical care and infant mortality in Fuchs VR. (ed) Economic Aspects of Health. Chicago: University of Chicago Press.

Haskel J, Jackman R. (1987) Long-term Unemployment and Special Employment Measures in Britain. Centre for Labour Economics: London School of Economics (Discussion Paper No. 297).

Hendry DF. (1980) Econometrics - Alchemy or science? Economica; 47: 387-406.

Hendry DF. (1983) The econometric analysis of economic time series. International Statistical Revies; 51: 111-163.

Hendry DF, Pagan AR, Sargan JD (1984) Dynamic Specification in Handbook of Econometrics, Volumne II (eds, Griliches Z, Intriligator MD) Amsterdam: North-Holland.

Hendry DF. (1988) PC-GIVE: An Interactive Econometric Modelling System. Oxford: Institute of Economics and Statistics and Nuffield College.

Hutten CR. (1985) Productivity change in state and local governments. Review of Economics and Statistics; 67: 256-266.

Inman RP. (1976) The family provision of children's health: an economic analysis in Rosett R. (ed) The Role of Health Insurance in the Health Services Sector. New York: Neale Watson Academic Publications.

Ippolito PM. (1981) Information and the life cycle consumption of hazardous goods. Economic Inquiry; 19: 529-558.

Jaque CM, Bera AK (1980) Efficient tests for normality, homoscedasticity and serial independence of regression residuals. Economics Letters; 6: 255-259.

John J. (1983) Health effects of unemployment: approaches and results of empirical research in the Federal Republic of Germany in Influence of Economic Instability in Health; John J, Schavefed D, Zollner H. (eds) Berlin: Springen-Verlag.

Joyce T. (1987a) The impact of induced abortion on black and white birth outcomes in the United States. Demography; 24: 229-244.

Joyce T. (1987b) The demand for health inputs and their impact on the black neonatal mortality rate in the U.S. Social Science and Medicine; 24: 911-918.

Judge GG, Griffiths WE, Carter-Hill R, Lutkepohl H, Lee TC (1985) The Theory and Practice of Econometrics New York: John Wiley and Sons.

Kehrer BH, Wolin CM. (1979) Impact of income maintenance on low birthweight: evidence from the Gary experiment. Journal of Human Resources; 14: 434-462.

Keynes JM. (1937) Some economic consequences of a declining population. Eugenics Review; 29: 13-18.

Kleiman E. (1974) The determinants of national outlay on health in Perlman M(ed) The Economics of Health and Medical care. London: Macmillan.

Kleinman JC. (1982) Indirect standardisation of neonatal mortality for birthweight. International Journal of Epidemiology; 11: 146-154.

Kleinman JC. (1984) Re: "Perinatal mortality: standardizing for birthweight is biased". American Journal of Epidemiology; 120: 798-799.

Layard PRG. (1986) How to Beat Unemployment. Oxford: Oxford University Press.

Layard PRG. and Nickell SJ. (1986) Unemployment in Britain. Economica,(Special Issue); 53: 5121-5169.

Leibenstein K. (1957) Economic Backwardness and Economic Growth. New York: Wiley.

Leu RE. (1984) Anti-smoking publicity taxation and the demand for cigarettes. Journal of Health Economics; 3: 101-116.

Leu RE. (1986) The public-private mix and international health care costs in Culyer AJ, Jonsson B. (eds) Public and Private Health Services Complementarities and Conflicts. Oxford: Basil Blackwell.

Lewit E. (1983) The demand for prenatal care and the production of healthy infants in Sirageldin I. (ed) Research in Human Capital and Development: JAI Press Inc.

McAleer M. et al (1985) What will take the con out of econometrics? American Economic Review; 75: 293-307.

McAvinchey ID. (1983) Unemployment and mortality: some aspects of the Scottish Case, 1950-1978. Scottish Journal of Political Economy; 31: 1-27.

MacDonald R, Speight AEH (1989) Consumption, saving and rational expectations: some further evidence. Economic Journal; 99: 83-91.

McIlwaine GM. et al (1974) A study of perinatal deaths in Glasgow. Health Bulletin; 33:1-4.

McIlwaine GM et al (1979) The Scottish perinatal mortality survey. British Medical Journal; ii: 1103-1106.

McIlwaine GM et al (1985) A routine system for monitoring perinatal deaths in Scotland. British Journal of Obstetrics and Gynaecology; 92: 9-13.

McKeown T. (1976) The Role of Medicine: Dream, Mirage or Nemesis? London: Nuffield Provincial Hospital Trust.

MacKinnon JG, White H, Davidson R. (1983) Tests for model specification in the presence of alternative hypotheses. Journal of Econometrics; 21: 53-70.

Maddala GW (1988) Introduction to Econometrics. New York: Macmillan.

Mincer J. (1963) Market prices,opportunity costs and income effects in Christ C. et al (ed), Measurement in Economics: Studies in Mathematical Economics in Memory of Yehuda Grunfeld; Stanford, California: Stanford University Press.

Mincer J. (1974) Schooling, Experience and Earnings. New York: National Bureau of Economic Research.

Moffit R. (1984) Profiles of fertility, labour supply and wages of married women: a complete life-cycle model. Review of Economics Studies; 51: 263-278.

Muellbauer J, Pashardes P. (1988) Tests of Dynamic Specification and Homogeneity in a Demand System. (Working paper No 88/6) London: Institute for Fiscal Studies.

Murnane RJ, Nelson RR. (1984) Production and innovation when techniques are tacit. Journal of Economic Behaviour and Organization; 5: 353-373.

Muurinen JM. (1982) Demand for health: a generalised Grossman model. Journal of Health Economics; 1: 5-28.

Nadiri MI. (1970) Some approaches to the theory and measurement of total factor productivity: A survey. Journal of Economic Literature; 8: 1137-1177.

Nelson CR, Plosser C (1982) Trends and random walks in macroeconomic time series. Journal of Monetary Economics; 10: 139-162.

Nerlove M. (1956) Estimates of the elasticities of supply of selected agricultural commodities. Journal of Farm Economics; 38: 496-509.

Newell DJ. (1964) Statistical aspects of the demand for maternity beds. Journal of the Royal Statistical society; 127: 1-41.

Newhouse J. (1977) Medical care expenditures: a cross-national survey. Journal of Human Resources; 12: 115-125.

Nordquist G, Wu SY. (1976 The joint demand for health insurance and preventive medicine in Rossett RN. (ed) The Role of Health Insurance in the Health Services Sector; New York: National Bureau of Economic Research.

Office of Health Economics (1987) Compendium of Health Statistics. London: Office of Health Economics.

O'Neil J. (1981) A time-series analysis of women's labour force participation. American Economic Review; 71: 76-80.

Osborn DR, Chui APLm Smith JP, Birchenhall CR (1988) Seasonality and the order of integration for consumption. Oxford Bulletin of Economics and Statistics; 50: 361-377.

Paige DC. (1962) Births and maternity beds in England and Wales, 1970. National Institute Economic Review; 22: 22-37.

Parkin D, McGuire A, Yule B. (1987) International comparisons of expenditure on health care and its relationship to national income: a critique and some new evidence. Health Economics Research Unit, University of Aberdeen.

Peacock AT. (1954) Theory of population and modern economic analysis II. Population Studies; 7: 227-234.

Phelps CE. (1978) Illness prevention and medical insurance. Journal of Human Resources; 13: 183-209.

Phillips AW. (1954) Stabilisation policy in a closed economy. Economic Journal; 64: 290-323.

Phillips AW. (1957) Stabilisation policy and the time form of lagged responses. Economic Journal; 67: 265-277.

Phillips PCB (1986) Understanding spurious regressions in econometrics. Journal of Econometrics; 33: 311-340.

Pollak RA, Wachter ML. (1978) The relevance of the household production function and its implications for the allocation of time. Journal of Political Economy; 85: 255-277.

Pollak RA. (1985) A transaction cost approach to families and households. Journal of Economic Literature; 23: 581-608.

Rappoport P, Reichlin L (1989) Segmented trends and non-stationary time series. Economic Journal (supplement); 99: 168-177.

Reves R.(1985) Declining fertility in England and Wales as a major cause of the twentieth century decline in mortality. American Journal of Epidermiology; 122: 112-126.

Riboud M. (1985) An analysis of women's labour force participation in France: Cross-section estimates and time series evidence. Journal of Labour Economics; 3: 5177-5200.

Richards ID. et al (1969) Perinatal mortality in Glasgow. Health Bulletin; 27:1-9.

Robbins L. (1929) Notes on some probable consequences of the advent of a stationary population in Great Britain. Economica; 9: 71-82.

Robbins L. (1932) An Essay on the Nature and Significance of Economic Science. London: Macmillan.

Rosenzweig MR, Schultz TP.(1981) Education and the household production of infant health in Proceedings of the American Statistical Association, Social Statistical Section. Washington: American Statistical Association.

Rosenzweig MR, Schultz TP. (1982) The behaviour of mothers as inputs to child health: the determinants of birthweight, gestation and rate of fetal growth in Fuchs VR. (ed) Economic Aspects of Health; Chicago: University of Chicago Press.

Rosenzweig MR, Schultz TP. (1983) Estimating a household production function: heterogeneity, the demand for health inputs, and their effects on birthweight. Journal of Political Economy; 91: 723-746.

Samuelson P. (1966) Rejoinder: agreements, disagreements, doubts and the case of induced, Harrod-Neutral technical change. Review of Economics and Statistics; 48: 444-448.

Sargan JD. (1964) Wages and prices in the United Kingdom: a study in econometric methodology in Econometric Analysis for National Economic Planning. Hart PE, Mills G, Whitaker JK. (eds); London: Butterworths.

Sargan JD. (1971) Production functions in Layard PRG. et al (eds) Qualified Manpower and Economic Performance. London: Heinemann.

Savrel-Cubizolles M, Kaminski M (1986) Work in pregnancy: its evolving relationship with perinatal outcome (A Review). Social Science and Medicine; 22: 431-442.

Schultz TW. (1960) The formation of human capital by education. Journal of Political Economy; 68: 571-583.

Schultz TP. (1982) Interpretation of relations among mortality, economics of the household and the health environment in Proceedings of the Meetings on Socioeconomic Determinants and Consequences of Mortality. New York and Geneva: United Nations and World Health Organization.

Selvanathan EA. (1988) Alcohol consumption in the UK, 1955-85: a system analysis. Applied Economics; 20: 1071-1086.

Shields MP, Tray RL. (1986) Four themes in fertility research. Southern Economic Journal; 53: 210-216.

Silverman WA. (1980) Retrolental Fibroplasia: A Modern Parable. New York: Grune and Stratton.

Smail R, Gray A. (1982) Nurses' pay in the NHS; Nursing Paper No. 3. Health Economics Research Unit, University of Aberdeen.

Smith RP. (1980) The demand for military expenditure. Economic Journal; 90: 811-820.

Smith JP, Ward MP. (1985) Time-series growth in the female labour force. Journal of Labour Economics; 3: 559-590.

Sprague A. (1988) Post-war fertility and female labour force participation rates. Economic Journal; 98: 682-700.

Stone JRN. (1953) The measurement of Consumer's Expenditure and Behaviour in the United Kingdom, 1920-1938, (vol 1). Cambridge: Cambridge University Press.

Thomas D. (1927) Social Aspects of the Business Cycle. New York: Knopf.

Thomson A, Hunter L. (1978) Great Britain in Dunlop JT, Galenson W. (eds) Labour in the Twentieth Century. New York: Academic Press.

Thomson J. (1952) The incidence of foetal and infant mortality in 13085 births in a maternity hospital. Journal of Obstetrics and Gynaecology of the British Empire; 59: 483-490.

Torrance GW. (1986) Measurement of health state utilities for economic appraisal. Journal of Health Economics; 5: 1-30.

Usher D. (1975) Comment on: The correlation between health and schooling in Terleckyj NE. (ed) Household Production and Consumption; New York: Columbia University Press for the National Bureau of Economic Research.

Vinod H. (1968) Econometrics of joint production. Econometrica; 36: 322-336.

Vinod H. (1976) Canonical ridge and econometrics of joint production. Journal of Econometrics; 4: 147-166.

Wagstaff A. (1985) Time series analysis of the relationship between unemployment and mortality. Social Science and Medicine; 21: 985-996.

Wagstaff A. (1986) The demand for health: a simplified Grossman Model. Bulletin of Economic Research; 38: 93-95.

Wald N et al (eds) (1988) UK Smoking Statistics. Oxford: Oxford University Press.

Welch F. (1970) Education in Production. Journal of Political Economy; 78: 35-59.

Wigglesworth JG. (1980) Monitoring perinatal mortality: a pathophysiological approach. Lancet: ii: 684-686.

Wilcox AJ, Russell IT. (1983) Perinatal mortality: standardising for birthweight is biased. American Journal of Epidemiology; 11: 146-154.

Wilcox AJ, Russell IT. (1986) Birth weight and perinatal mortality: toward a new method of analysis. International Journal of Epidemiology; 15.

Williams RL. (1975) Exploring a health care paradox. Policy Sciences; 6: 91-101.

Williams RL. (1979) Measuring the effectiveness of perinatal medical care. Medical Care; 17: 95-110.

Williams RL, and Chen PM. (1982) Identifying the sources of the recent decline in perinatal mortality rates in California. New England Journal of Medicine; 306: 207-214.

Winter JM. (1983) Unemployment, nutrition and infant mortality in Britain: 1920-1950, in John J, Schavefel D, Zollner H. (eds) Influence of Economic Instability in Health. Berlin: Springer- Verlag.

Yamada T, Yamada T, Chaloupka F. (1987) Nutrition and Infant Health in Japan Working Paper No. 244; New York: National Bureau of Economic Research.

Goverment Documents

Central Statistical Office (various years ) Economic Trends. London: HMSO.

Department of Employment (1987) Retail Prices Indices: 1914-1986. London: HMSO

Department of Health for Scotland (1935) Report on Maternal Mortality and Morbidity in Scotland. Edinburgh: HMSO.

Department of Health for Scotland (1936a). Eighth Annual Report of the Department of Health for Scotland. Edinburgh: HMSO.

Department of Health for Scotland (1936b) Committee on Scottish Health Services Report (Cathcart Report). Edinburgh: HMSO.

Department of Health for Scotland (1943) Infant Mortality in Scotland (Orr Report). Edinburgh: HMSO.

Department of Health for Scotland (Montgomery Committee) (1959). Maternity Services in Scotland. Edinburgh: HMSO.

Department of Health for Scotland (1962) Hospital Plan For Scotland (cmnd. 1602). Edinburgh: HMSO.

Department of Health and Social Security (1976) Priorities for Health and Social Services in England. London: HMSO.

Guillebaud Committee (1956) Report of the Committee of Enquiry into the Cost of the National Health Service. London: HMSO.

Ministry of Health (Cranbrook Committee) (1959) Report of the Maternity Services Committee. London: HMSO.

Ministry of Health (1962) A Hospital Plan for England and Wales (cmnd. 1604). London: HMSO.

Ministry of Health (Peel Committee) (1970) Domiciliary Midwifery and Maternity Bed Needs. London: HMSO.

Registrar General Scotland (1939-1985) Annual Reports of the Registrar General for Scotland. Edinburgh: HMSO.

Royal Commission on the National Health Service (1979). Report. London: HMSO.

Scottish Home and Health Department (1973) Maternity Services: Integration of Maternity Work. Edinburgh: HMSO.

Scottish Home and Health Department (1976) Health Services in Scotland: The Way Ahead. Edinburgh: HMSO.

Scottish Home and Health Department (1980) Scottish Health Authorities Priorities for the Eighties. Edinburgh: HMSO.

Scottish Home and Health Department (1988) Scottish Health Authorities Priorities for the Eighties and Nineties . Edinburgh: HMSO.

Simon J. (1858) General Board of Health Papers relating to the Sanitary State of England. London: HMSO.

World Health Organisation (1950) International Classification of Diseases (6th revision). Geneva: World Health Organisation.

World Health Organisation (1955) International Classification of Diseases (7th revision). Geneva: World Health Organisation.

World Health Organisation (1965) International Classification of Diseases (8th revision). Geneva: World Health Organisation.

World Health Organisation (1977) International Classification of Diseases (9th revision). Geneva: World Health Organisation.