ANAESTHESIA AND SEDATION

FOR

CARDIAC CATHETERISATION.

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"Catheterisation of the heart and angiocardiography with their attendant dangers are methods necessary in some but not every case and when they are applied they should be applied with discernment, humanity and respect for the patient." I. Mahaim (1949)

Extract from Presidential address to Swiss Society of Cardiology.

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I N T R O D U C T I O N.

INTRODUCTION

The great advances in surgical correction of congenital cardiac anomalies have necessitated ever increasing accuracy in the diagnosis of these conditions. To this end cardiac catheterisation is being widely practised. The validity of the measurements obtained during this procedure depend on the patient remaining throughout in a steady physiological state.

Initially Cournand (1945) claimed that, in adults, satisfactory results could be obtained without recourse to drugs but as the technique was extended to children it soon became obvious that some form of sedation was required to facilitate the procedure. (Baldwin et al. 1946.) As it was considered desirable that the child should remain asleep, basal narcosis by rectal injection was widely used. Thus an anaesthetist is now a member of most cardiac diagnostic teams.

The choice of anaesthetic technique is extremely difficult. A period of narcosis lasting for several hours must be induced in children, with a wide variety of congenital heart lesions, who would be regarded, under any circumstances, as poor anaesthetic risks. In addition the agents used must not interfere with the technical procedures involved, such as blood gas analysis.

Despite this, respiration and circulation should remain stable throughout the entire investigation and should approximate to the patient's resting state. Any departure from this "steady state" should be readily detectable, enabling appropriate action to be taken either by repeating the readings after the unsettled period or by making due allowance when the results are analysed.

By 1956 when this study was commenced, a number of techniques had been described in which sedation or anaesthesia was employed to provide suitable conditions for cardiac catheterisation. These were claimed by their exponents to provide the necessary "steady state", although in many cases it seemed highly unlikely that they would in fact do so.

The most notable feature of these reports was that no attempt was made to measure the condition of the circulation and respiration during the investigation to see whether a "steady state" was being obtained. Indeed although the expression "steady state" pervades the literature on cardiac catheterisation, there is little indication of exactly what it is intended to include; even less is there any suggestion of how this state can be measured. This seemed a highly unsatisfactory basis on which to work and this thesis describes an attempt to elucidate the problem involved.

In the first place the technique of cardiac

catheterisation and the information obtainable from the investigation are described. It is then possible to appreciate which physiological variables must be kept as constant as possible and thus to establish parameters for the "steady state".

Once the desirable "steady state" had been delineated it was possible to seek methods by which it could be measured easily, quickly and safely and these are evaluated.

An anaesthetic technique was then developed which on theoretical grounds should maintain stable conditions throughout the period necessary to complete the investigation.

A series of 63 patients were catheterised using the anaesthetic technique evolved and the nature of the conditions produced was measured. It was possible to estimate not only whether the physiological state was steady but what relationship it bore to the so called resting state.

A further series of 59 cases was examined using moderate to heavy doses of sedatives and a similar assessment of the conditions obtained was made.

Finally the results obtained in each series were compared to evaluate each method in the furtherance of cardiac catheterisation.

CHAPTER ONE

CARDIAC CATHETERISATION

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History.

Although cardiac catheterisation had been performed on animals in the middle of the nineteenth century, (Bernard 1855; Pavy 1857) the application of the technique to human beings dates from 1929. In this year Forssmann reported how, first with the help of a colleague and later unaided, he passed a catheter through an antecubital vein into his own right atrium (Forssmann 1929 a). He published with his report a radiograph showing the catheter in situ. Some weeks later (Forssmann 1929 b) he acknowledged a communication from Unger pointing out that venous catheterisation had been carried out in 1905 (Bleichroeder 1912) and he agreed with Unger's claim that from the length of catheter employed it was probable that it had entered Bleichroeder's heart in some of these experiments. Forssmann stated that this work had escaped his notice, and his own research had been performed independently. His bold experiments led to the award of a Nobel Prize in 1957.

In Prague, Klein (1930) applied the technique

to the determination of cardiac output by application of the Fick Principle and in the following year Forssmann (1931) attempted to obtain angiocardiograms using the new technique. Although he was successful in animals he failed to produce a good contrast film of his own heart. His pioneer work was however duly acknowledged when angiocardiography became successfully established (Robb and Steinberg, 1938; Castellanos et al., 1938). In their classic paper Cournand and Ranges (1941) reported their application of cardiac catheterisation to the determination of cardiac output and it is from this work that the acceptance and widespread use of the technique dates. Shortly afterwards, McMichael and Sharpey Schafer (1944) published their first results in this country.

The application of cardiac catheterisation to the diagnosis of congenital heart disease dates from the report by Brannon et al. (1945) of their findings in four cases of atrial septal defect. This was soon followed by a report of the findings in ventricular septal defects (Baldwin et al. 1946) and a footnote to this paper contains the first reference to the use of basal narcosis to facilitate the procedure. The first of Dexter's many contributions appeared in the same year (Dexter et al. 1946) and in the following year the first

British report was published (Howarth et al. 1947).

In Glasgow Royal Infirmary the first cardiac catheterisation was performed in 1946. This is believed to be the first time that the technique has been widely used by the Cardiology Department in the diagnosis of congenital cardiac defects.

Technique (As used in Cardiology Dept., Glasgow Royal Infirmary).

The patient, who has fasted for at least four hours, is brought to the X-ray room and placed comfortably on a soft foam mattress. A vein, preferably the left median cubital, is exposed under local anaesthesia and the largest possible Cournand catheter is introduced. Normally a catheter with a terminal opening is used but where angiocardiography is contemplated a catheter with lateral apertures is employed. Heparinised saline is allowed to drip slowly through the catheter which is manipulated under fluoroscopic control into the various chambers of the heart.

Intracardiac pressures are recorded by connecting the catheter through an electromanometer to an oscilloscope and suitable recorder. The oxygen content of serial blood samples removed in quick succession from adjacent sites with the heart is measured directly by means of a

cuvette oximeter. In addition samples are removed and placed under oil to enable further analysis to be carried out with a haemoreflector and by the Van Slyke method. (Van Slyke and Neill 1924).

Simultaneously with the withdrawal of blood from the middle of the right atrium (or pulmonary artery), a sample is taken from the femoral artery and the oxygen consumption is measured by means of a spirometer or Douglas bag. From this data cardiac output is estimated in accordance with the Fick Principle.

During the procedure the electrocardiogram is continuously monitored on an oscilloscope, standard limb leads being used. An ear oximeter enables the arterial oxygen saturation to be checked and serial pulse and systemic blood pressure readings are taken.

In addition in suitable cases dye has been injected through the catheter and by means of the oximeter connected to a suitable recorder dye dilution curves have been obtained. In other cases while the catheter is maintained at a preselected site in the heart, a radio opaque substance is injected through it and angiocardiograms obtained.

The procedure is carried out entirely in the X-Ray room and occupies about two hours.

Information obtained by Cardiac Catheterisation.

Cardiac catheterisation may provide both physiological and anatomical data although the latter may be more easily obtained by angiocardiography. The information may be grouped as follows:-

1) The catheter may be passed directly through a septal defect or into an aberrant vessel. Fluoroscopy in various positions will normally enable the path taken by the catheter to be accurately determined. By manipulating the catheter within any cardiac chamber it is often possible to estimate the size of that part of the heart (Crawford 1950).

2) Pressure recording. By serial recording of the pressures at adjacent sites within the heart and great vessels it is often possible to reveal gradients suggestive of valvular stenosis or intracardiac shunts. This is normally done by using the ordinary Cournand catheter connected through an electromanometer but Ellis (1950) and Wood (1954) claim greater accuracy by employing a manometric sound attached to the catheter

tip. Simultaneous readings from both sides of a valve may be obtained by using a double lumen catheter (Cournand et al., 1945).

3) Estimation of the oxygen content of blood samples taken in quick succession from adjacent sites in the heart and great vessels may reveal abnormal oxygenation suggestive of an arterio-venous shunt.

Within these three groups lies the basic information provided by cardiac catheterisation and it is with this that we are primarily concerned but some of the supplementary procedures also have a direct bearing on the anaesthetic technique.

4) Morrow et al. (1958) have suggested the administration of known concentrations of nitrous oxide to the patient, the subsequent estimation of nitrous oxide in the blood taken from adjacent sites within the heart and their comparison with arterial samples. This he claims is more accurate than comparison of oxygen contents.

5) The catheter may be used as an intracardiac electrode and electrograms typical of different sites within the heart may help to localise the catheter tip. (Emslie-Smith 1955).

6) Injection of dye through the catheter at various sites in the heart enables dye dilution curves to be recorded with a suitable photoelectric device such as an ear oximeter. The tracings to be expected in the various congenital lesions have been reported by Swan and Wood (1953). More recently radio-active elements have been similarly used.

7) Selective angiocardiography may be performed by injecting a suitable contrast medium through the catheter while the tip is maintained at a predetermined site and taking a series of films of the heart in rapid succession.

From the data obtained during cardiac catheterisation it is possible to estimate the cardiac output in accordance with the Fick principle. Formulae have been suggested by which the magnitude of intracardiac shunts may also be calculated (Bing et al. 1947; Cournand et al. 1949). In employing these however the potential sources of error (McMichael, 1949b; Rossier et al. 1949 and Barratt Boyes and Wood, 1957) must be borne in mind.

Indications for cardiac catheterisation.

Opinions on this subject vary considerably. Lewis (1948) recommends that all patients with suspected

anomalies should be catheterised whereas Taussig (1952) considers that the procedure should be reserved for a small minority. Dexter (1950) suggests that 65% of cases will require catheterisation but Rutledge (1949), Shapiro (1949) and Wittenborg and Neuhauser (1955) place the figure at 10-20%. Cournand (1951) and Adams et al. (1950) from wide experience advise that the procedure be restricted to those cases where the diagnosis is still in doubt after the less complicated methods have been applied.

Where, however, operative treatment of a congenital lesion is considered feasible a strong case for previous cardiac catheterisation can be made out. Patients with pulmonary stenosis may be found to have an associated septal defect which can be simultaneously repaired. Cosby et al. (1953) have shown that in patients with clinically diagnosed atrial septal defects, cardiac catheterisation may uncover a lesion of greater importance than the septal defect. Even in the apparently typical patent ductus arteriosus, cardiac catheterisation may reveal pulmonary hypertension such as would contra-indicate operation (Lancet 1956) or show the clinical diagnosis to have been completely wrong (Morgan & Burchell 1950).

In all cases valuable information may be gained as to the state of the pulmonary circulation (Marder et al. 1952, Warren 1953) which may sway the balance for or against operation. With these factors in mind it is the practice in Glasgow Royal Infirmary to submit to cardiac catheterisation all patients whose condition cannot be diagnosed accurately by other means. In addition virtually all cases submitted for surgery are investigated in this way.

Contra-indications to Cardiac Catheterisation.

These have been well expressed by the Committee on Cardiac Catheterisation and Angiocardiography of the American Heart Association. (Cournand et al. 1953). They are:-

1) Paroxysmal ventricular tachycardia
2) Recent myocardial infarction
3) Recent subacute bacterial endocarditis
4) Electrocardiographic manifestations of acute rheumatic fever.
5) Recent pulmonary embolism
6) A critically ill patient.

Less acceptable is their view that the procedure is contra-indicated in poorly co-operative or anxious patients. The view of Levine (1951) and

Bruce (1954) that small children should be excluded because they require a general anaesthetic has little to commend it. Many would agree with Harned (1955) that the procedure should not be carried out on patients suffering from Ebstein's disease.

In addition to observing these contra-indications most clinics postpone the investigation in the presence of any acute infection particularly when it affects the respiratory tract.

Complications of Cardiac Catheterisation.

Hebert et al. (1953) in a review of 973 cardiac catheterisations found complications in 30% of cases. Only 3% were however classified as serious. The complications fall into several groups:-

Arrythmias. These are so common that many workers consider them an almost inevitable accompaniment of the procedure. Patients with congenital cardiac lesions are twice as liable to develop significant arrythmias during cardiac catheterisation as patients with other lesions (Michel et al. 1950). Landtman (1950) in an extensive study found practically all known arrythmias occurring during cardiac catheterisation but was unable to associate any particular arrythmias with a specific congenital lesion. It has, however, been

shown that the type of arrythmia present may be related to the site of the catheter tip at the time of its occurrence (Fowler et al., 1951; Episcopu, 1952). Ventricular fibrillation is the most serious of the arrythmias and is one of the causes of death during cardiac catheterisation (Wood, 1950; Goldman et al., 1950). Recovery has been reported however following cardiac massage (Southworth et al., 1950; Schnabel, 1954) and indeed even without this treatment (Gordh et al., 1956).

Venospasm is an irritating complication which Wood (1950) considers is due to poor technique. It normally occurs only in the arm veins but spasm of a pulmonary vein has been reported (Jorgens et al., 1952).

Venous Thrombosis is also normally limited to the area of insertion of the catheter. Cournand et al. (1945) feel that this is also due to poor technique. Small emboli may arise from these localised thromboses and may cause pulmonary infarcts or even hemiplegia where there is an intracardiac shunt (McMichael 1949a). Of more serious import however are the vena caval thromboses reported by Johnson et al., (1947) and Peel et al. (1956). Both cases had a fatal outcome.

In animals damage to the heart wall occurs fairly frequently (Ellis et al., 1950; Banfield et al., 1950). Although this may be suspected on clinical

grounds in man (Biork and Krook, 1951), it has seldom been demonstrated at necropsy (Stern et al., 1952; Sancetta et al., 1953; Edwards et al., 1953; Goodwin, 1953; Escher et al., 1958).

Catheterisation of the coronary sinus may cause circulatory collapse (McMichael and Mounsey, 1951; Rivier et al., 1954) but it appears that this only occurs when the catheter passes deeply into the sinus and impacts in a coronary vein (Smith et al., 1951; Read et al., 1955).

Obstruction of a stenosed pulmonary valve by the catheter may cause circulatory collapse as a result of the acute anoxia which ensues. This has been reported where the pulmonary stenosis was an isolated lesion (Bing, 1952; Paul and Rudolph, 1958), where there was an associated septal defect (Cournand and Himmelstein, 1952) and in Fallot's Tetralogy (Mannheimer et al., 1951).

Pulmonary oedema due to over transfusion while the catheter is in the pulmonary artery has been reported by Skaggs and Chapman (1952) and Broustet et al. (1954). Pulmonary infarction may follow wedging of the catheter in a peripheral pulmonary artery (Houssay et al. 1952; Nightingale and Williams, 1955).

Pyrogenic reactions to catheterisation have disappeared with the introduction of thorough cleansing and autoclaving of the catheters. The cause was thought to be a heat labile plasma transfusion factor (Hernandez and Saslaw, 1953). Only once has infective endocarditis been reported after cardiac catheterisation (Winchell, 1953).

Rarely the catheter may loop in the heart or great vessels Johansson et al. (1954).

Conclusions.

It can be seen that the technique of cardiac catheterisation is complicated and the procedure may occupy several hours. In addition it is likely to be undertaken in patients with a varying degree of cardiac disability. Despite the many potential hazards the mortality rate from the procedure is only about 1 in 1,000 cases (Cournand et al., 1953) and the morbidity rate is also low. This is more than offset by the lives saved by avoiding surgery in unsuitable cases. (Ziegler, 1954; Kirklin and Ellis, 1956). The technique should therefore be a presursor of cardiac surgery.

Basically the technique provides information by enabling comparisons to be made of the oxygen contents and blood pressure levels at various sites within the heart and great vessels. If they are to be valid these

levels should be constant during the period of sampling and preferably during the entire investigation. Hence the need for a steady physiological state.

CHAPTER TWO

THE STEADY STATE

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The need to maintain the circulation and respiration in a steady state during the performance of cardiac catheterisation has been recognised since the clinical introduction of the procedure (Cournand and Ranges 1941). Many clinicians feel that the readings obtained should represent basal values for the patient. Thus what is required is the maintenance of the patient in a resting or basal state for a period of several hours.

Wide variations in circulatory dynamics during cardiac catheterisation are reflected in marked fluctuations in the oxygen content of the blood. This state of affairs can produce very misleading results and one unnecessary thoracotomy has been reported (Fowler et al., 1957). Circulatory instability is also of more immediate danger to life in children with congenital heart lesions (Rink et al., 1948; Potts, 1949; McQuiston, 1949; Keown et al., 1957).

The previous chapter has shown that the basic need is for constant levels of oxygen saturation throughout the period during which blood samples are being withdrawn and preferably throughout the entire investigation. In addition the pressures in both the systemic and

pulmonary circulations should show minimal fluctuations. In attempting to define the desirable steady state one must consider those factors known to cause fluctuations in these values.

Emotion.

Cournand and Ranges (1941) in their initial report of eight cardiac catheterisations in adult volunteers claim that the procedure is not accompanied by psychic disturbances. Later Cournand (1945) after performing 1,200 catheterisations reiterated that persistent anxiety in most subjects can be regarded as a negligible factor. In his later works, however, he recommends the use of Bromethol when carrying out the investigation in children (Cournand et al., 1949; Cournand, 1951).

Following the work of Stead et al. (1945), Altschule (1951) noted that emotion caused palpitations, marked fluctuations in cardiac rate, and a rise in cardiac output. Similar changes have been reported by Fishman et al. (1952).

The systemic circulation may show marked rises or falls in pressure due to anxiety (Wright, 1953) and it has recently been shown by Barratt Boyes and Wood (1958) that anxiety also raises the right ventricular pressure. This rise is normally reflected in a rise in

pulmonary artery pressure. Their work confirms the findings of Harris (1955) who noted considerable fluctuations in the pulmonary artery pressure of nervous or apprehensive patients. Both workers are at variance with Shephard (1954) who found that emotion had little effect on the pulmonary artery pressure.

Right atrial pressure is however little affected by emotion (Stead et al., 1945).

Oxygen consumption rises with anxiety, even where there are no outward signs of emotion (Ziegler and Levine, 1925).

Arterial oxygen saturation can be lowered by potentially stressful stimuli (Doust and Schneider, 1955). Extreme anxiety may in addition cause crying, struggling or irregular respiration and the deleterious effects of these are considered in separate sections.

Respiration.

During both phases of respiration impulses from the respiratory centre radiate to the vasomotor centre and minor changes in blood pressure result (Wright, 1953). The direct effects of the changes in intrathoracic pressure during respiration have been frequently reported (Hamilton et al., 1944; Bloomfield et al., 1946; Battro et al., 1949; Beard, 1950; Opdyke and Brecher, 1950; Borden et al., 1950 and Lee et al. 1954). Right heart pressures

in the auricle, ventricle and pulmonary artery rise during expiration and fall during inspiration. Forced expiration also produces a rise in systemic blood pressure. The variations in pressure increase markedly with increased depth of respiration and coughing and straining produce very wide fluctuations indeed. Barratt Boyes and Wood (1958) have shown that under these circumstances the right ventricular pressure varies less than the pulmonary artery and systemic pressures.

During deep breathing there is an unequal discharge from the ventricles, the right predominating during inspiration and left during expiration (Cournand, 1947). Dyspnoeic respiration in dogs with artificially produced atrial septal defects has been shown to cause bidirectional blood flow-being from right to left during inspiration and left to right during expiration. (Brecher and Opdyke, 1950).

Finally, it has been shown that hyperventilation in a person previously breathing quietly, will cause a rise in arterial oxygen saturation which does not regain its former level for five minutes (Van Lingen and Whidborne, 1952). Depression of respiration below the level required for adequate oxygenation of the blood is considered in relation to the various drugs which are normally responsible.

Straining and Struggling.

Straining and struggling during cardiac catheterisation cause marked fluctuations in intra cardiac pressures and blood oxygen values (Holling and Zak, 1950). This may alter the amount of blood flowing in a left to right shunt (Hickman, 1949) or even reverse the direction of the blood flow (Banghart and Lewis, 1948). In ill patients or those with large septal defects this may result in acute right heart failure (Beard and Goodwin, 1956).

The alteration in oxygen content of blood samples during struggling noted by Holling and Zak (1950) varied with the particular lesion present. An exceptionally sudden and severe fall is reported by Montgomery et al. (1948) in a child with Fallot's Tetralogy. Quite apart from the obvious danger to life which may be involved, such incidents if repeated may lead to the abandonment of the catheterisation (Zak, 1949).

Variation in Alveolar Oxygen and Carbon Dioxide Levels.

It has been shown that during normal quiet respiration a rise in oxygen percentage in the inspired air causes a rise in the arterial oxygen saturation (Preston and Ordway, 1948; Wood, 1949). Similarly a fall in the oxygen content of the inspiration air, causes a fall in arterial oxygen saturation. (Dripps

and Comroe, 1947).

The pulmonary artery pressure has been shown to be very sensitive to variations in the inspired oxygen percentage. Following the work of Von Euler and Liljestrand (1946) in cats, Motley et al. (1947); Doyle et al. (1951); Westcott et al. (1951) and Stroud and Rahn (1953) have shown that in man also the pulmonary artery pressure rises markedly as the inspired oxygen level falls. A fall in pulmonary artery pressure during inspiration of high percentage oxygen mixtures has been demonstrated by Dressler et al. (1952) and Barratt Boyes and Wood (1958).

Cardiac output, pulse rate and systemic blood pressure also vary inversely with the amount of oxygen in the inspired air. (Dripps and Comroe, 1947; Dressler et al., 1952; Barratt Boyes and Wood,1958).

It is not surprising therefore that in patients with congenital heart disease wide variations in oxygen saturation (Burchell, 1950) and reversal of shunts (Burchell et al. 1951, 1953; Downing, 1953 and Burchell, 1954) have been shown to follow the variation of oxygen content in inspired air.

Only a slight rise in pulmonary artery pressure follows severe carbon dioxide retention (Cournand, 1950) or the inhalation of 5% carbon dioxide (Shephard, 1954)

although the cardiac output and systemic pressure show a considerable rise in similar circumstances.

Sleep.

Important changes are associated with the onset of sleep. The heart rate and systemic blood pressure fall (Kleitman, 1929) as does the pulmonary artery pressure (Halmagyi et al., 1953). Robin et al. (1958) report a rise in alveolar carbon dioxide tension without any change in oxygen tension and a fall in oxygen consumption, carbon dioxide production, alveolar and total ventilation. Respiration is regular (Reed and Kleitman, 1926).

The changes occurring during sleep are considered more fully later in the appropriate section but it is important to note that in a patient who sleeps intermittently wide fluctuations may take place. In addition Steen (1954) has emphasised a point often overlooked. A child who falls asleep during the cardiac catheterisation may, by awakening in strange surroundings, struggle.

Drugs.

The effects of thiopentone sodium were studied by Barton et al. (1946). They found a transient drop in blood pressure and transient respiratory depression following intravenous injection of the drug. The arterial

oxygen saturation remained normal although the carbon dioxide tension was raised. The respiratory rate was normal but minute volume was decreased. Swerdlow (1957) found that this respiratory depression was greater and longer lasting with each subsequent injection of the drug. Similarly Bizard et al. (1952) reported greater depression of arterial oxygen saturation. Cardiac output, and blood pressure fell while the pulse rate rose in proportion to the depth of anaesthesia produced. (Etsten and Li, 1955).

The administration of pethidine has been shown to produce marked changes in the respiratory pattern in man but even when the minute volume was grossly decreased alveolar carbon dioxide levels remained normal (Elam and Brown, 1956; Tenney, 1956; Ausherman et al., 1956).

Morphine sulphate by injection causes a fall in right heart pressures lasting up to thirty minutes (Scebat and Lenegre, 1949).

Various combinations of opiate and belladonna derivatives used in premedication were shown by Shackman et al. (1951) to have an unpredictable effect on oxygen consumption. Similar results were produced by barbiturates (Fraser and Nordin, 1955). When the patients fell asleep, however, oxygen consumption invariably fell. This confirms the work of Quastel and Wheatley (1933) who found a fall in oxygen

consumption during anaesthesia.

Intermittent injection of drugs can therefore produce marked changes in respiration and in circulatory dynamics.

Miscellaneous.

Although Himmelstein and Cournand (1952) have pointed out that the state of oxygenation of the patient will depend on the oxygen consumption of the tissues, any increase in oxygen consumption is usually accompanied by hyperventilation. As this compensatory effect does not invariably occur it is desirable that the oxygen consumption should remain constant.

Finally Harned et al. (1952) noted a reduction in arterial oxygen saturation in the presence of cardiac arrythmias. We have found a reduction to accompany severe tachycardia even where the heart rate has remained regular.

These then are some of the factors which tend to upset the state of circulation and respiration and in their absence it is reasonable to postulate that the values for oxygen content and blood pressure within the heart and great vessels should remain constant or nearly so over a long period. We may therefore consider that for the purposes of cardiac catheterisation a steady state exists where over the period of

examination the following conditions are fulfilled:-

1) Anxiety is absent
2) Respiration is quiet and regular although of sufficient depth to allow normal gaseous exchange
3) The composition of inspired gases is constant
4) Oxygen consumption does not vary appreciably
5) Muscular movements do not take place
6) Changes in heart rate are minimal
7) Administration of drugs is avoided during the period of sampling and preferably during the whole examination
8) The patient remains asleep or awake during the investigation but does not lapse from one state to the other.

It is important at this stage to observe how many of the above criteria can be fulfilled by sedating the patient to the point of sleep. They are considered in order:-

1) During sleep anxiety is absent. Although circulatory changes have been noted where unpleasant dreams occur this is probably not relevant to the present problem.

2) Reed and Kleitman (1926) found that the most characteristic feature of sleep was the regularity of rate and depth of respiration. Robin et al. (1958) have shown that normal gaseous exchange is virtually unaltered.

3) The patient can breathe room air throughout.

4) Fraser and Nordin (1955) showed that in all patients sedated to sleep from which they were not rousable the oxygen consumption falls within normal limits and remains thus for some considerable time. An accurate estimation could therefore be obtained for any patient at any time. Robin et al. (1958) found that the alveolar carbon dioxide tension did not vary during sleep although a slight fluctuation occurred when repeated measurements were made with the patient awake.

5),6),7),8) Provided sleep is induced with adequate initial doses of narcotics these conditions should be fulfilled.

CHAPTER THREE.

PROVIDING THE STEADY STATE

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The choice of anaesthetic agents for any given patient is frequently a matter of compromise, the benefits of some agents being outweighed by undesirable side effects. In arriving at a decision as to what agents should be employed, the anaesthetist must consider the limitations imposed by the physical condition of the patient and by the requirements of the cardiac catheterisation. It is appropriate to consider these at this point.

The Patient.

Patients with congenital heart disease presented for cardiac catheterisation all suffer from some defect which impairs the efficiency of their circulation but their condition varies from those who are almost symptom free to some who are on the brink of cardiac failure. Inglis (1954) in his paper describes the patients as poor risk, undersized, cyanotic children subject to recurrent respiratory infections but while this description certainly fits some of the worst cases it is not by any means universally applicable. Brown (1945) points out that while the cyanotic cases are much more dramatic in appearance and in symptomatology,

it is often forgotten that they are in the minority.

Adams et al. (1954) using the Wetzel Grid found in 228 children with all types of congenital cardiac lesions that growth and development were essentially normal. Their opinion was that stunted growth followed repeated chest infections but was not an invariable sequel of anoxia. Chazan et al. (1951) found that the average intellect of these patients was about normal and where the intelligence quotient was low the children had frequently been deprived of schooling or had associated speech defects. Although they found many of the children emotionally well adapted to their illness they point out that parental anxiety may profoundly influence the child. Among cyanotic children Gibson (1952) found a large proportion who were irritable, fretful and unhappy.

Of the cyanotic group of abnormalities, Fallot's Tetralogy is the most common accounting for about 70% of cases (Shapiro, 1949; Brown, 1949). These patients are subject to syncopal attacks on the slightest exertion or emotional strain or even in the absence of any obvious precipitating factor (Parker, 1948; McQuiston, 1949; Olney, 1949; Brown, 1949). Such attacks which can have a fatal outcome may be brought about by infundibular

spasm increasing the already severe pulmonary stenosis (Brock, 1956; Wood, 1956).

Effort syncope is also a common manifestation of pulmonary hypertension (Dressler, 1952; Howarth and Lowe, 1953), and pulmonary hypertension has been found in conjunction with almost all of the acyanotic forms of congenital heart disease (Griswold et al. 1949; Adams, 1952 Wood, 1952; Shephard, 1954). Its importance is greater where the pulmonary artery pressure is more than twice normal (Selzer, 1954). Where there is a large left to right intracardiac shunt, blood accumulates in the lungs displacing air and the patients are subject to recurrent respiratory infections (Ordway, 1955; Edwards, 1954).

Cardiac arrythmias are present in some patients and they are particularly associated with atrial septal defects (Dry, 1948; Banghart and Lewis, 1948).

Blood oxygen saturation varies widely from case to case and even at different times in the same case. Burchell et al. (1950) found that where the patient was well nourished the oxygen saturation was over 70%. Gross and Jezer (1956) emphasized the importance of slow circulation through dilated peripheral veins as a cause of cyanosis and this type of cyanosis is mentioned by Campbell (1954) as a feature of

pulmonary stenosis. In some cases the cyanosis may be due to a thickening of the alveolar membrane with consequent slow diffusion through it (Ordway et al., 1950).

A low oxygen consumption is a feature of congenital heart disease (Bing et al., 1948) and Shephard (1955) has similarly found a low alveolar carbon dioxide level. He feels that this may be due to the resting hyperventilation found in these cases or to the youth of many of the patients.

The reaction of cyanotic patients to anaesthetic drugs varies considerably; thus Smith et al. (1958) give less "sedative mixture" to their cyanosed children but Lucas (1958) finds that they are resistant to anaesthesia and require larger than normal doses of any drugs used. My own experience in dealing with cyanotic children confirms the views held by Lucas.

It is clear therefore that while the condition of these patients will vary widely, it is reasonable to expect some or all of the following feature in the majority of cases.

1) Many of the patients will be young children.
2) They will frequently have been hospitalised once or more and will be accustomed to a great deal of attention from parents, relatives and

friends. This coupled with their natural anxiety in strange surroundings tends to make them emotionally unstable.

3) Many will be prone to syncopal attacks either spontaneously or on the least physical or emotional strain.

4) They will tolerate anoxia badly.

5) Some have rigidly fixed cardiac outputs, e.g. those with aortic or mitral stenosis or coarctation of the aorta and may develop severe hypotension with small doses of drugs.

6) A number will have pulmonary congestion and chronic respiratory disease.

7) Other congenital defects may be present.

Many of the cases are therefore poor risks for anaesthesia under any circumstances. Burchell et al. (1953) actually report three deaths in patients awaiting cardiac catheterisation.

The Procedure.

In an earlier chapter the technique of cardiac catheterisation as practised in the Royal Infirmary, Glasgow, is discussed and some of the limitations imposed by this procedure on the choice of anaesthetic or sedative agents have been considered. Although it

is now the practice to determine the blood oxygen values by drawing the blood direct from the catheter through the cuvette of a cuvette oximeter and to check the readings on samples in a Brinkman Haemoreflector, it has long been considered desirable to check these photoelectric readings against the standard gasometric analysis of Van Slyke and Neill (Burchell and Wood, 1950). Thus it is not possible to use anaesthetic gases or volatile liquids; even paraldehyde is excluded by Smith (1950) for the same reason. The effects of oxygen have already been considered and this gas is not given unless the patient's life is in danger.

Orcutt and Waters (1937) have described a method which will allow the Van Slyke analysis to be performed in the presence of nitrous oxide, cyclopropane or ethylene but this is time consuming especially where many samples have to be analysed. In any case inflammable agents are not considered to be safe when used in the X-ray department as even with the most modern apparatus there is the danger of a static spark (Simon, 1949). In addition as the examination is carried out in the darkened X-ray room it is desirable that apparatus should be kept to a minimum.

The early reports of the procedure emphasised its freedom from pain and discomfort (Cournand and

Ranges, 1941; Cournand, 1945), and Geigler et al. (1946) report that where students and doctors have been subjected to it they have reported no awareness of the catheter. This is however not always the case and pain albeit transitory has been reported when:-

1) There is venospasm (Sancetta, 1951)
2) The catheter tip is striking the endocardium (Whitelaw, 1948; Landtman, 1950).
3) The catheter is in the right ventricle (Holling and Zak, 1950).

This aspect of the procedure has been investigated as part of the present study and the results will be discussed in due course.

Finally the danger of exposure to radiation measured by Hills and Standford (1950) and Dornette and Ott (1958) make it desirable that the anaesthetist should not require to handle the patient unless with gloved hands during the screening period.

CONCLUSIONS.

It appears from consideration of the type of patient under investigation and the nature of the procedure to be performed that the ideal anaesthetic should:-

1) Allow easy induction even in nervous children.

2) Keep the patient asleep and quiet for 2-3 hours without undue depression of vital function.

3) Be non toxic, non explosive and neither gaseous nor volatile.

4) Not require complicated apparatus or complicated manoeuvres in the confined spaces available.

The anaesthetic methods which have in the past been employed together with their advantages and disadvantages must now be considered.

CHAPTER FOUR.

METHODS OF SEDATION AND NARCOSIS

EXPLANATORY NOTE.

The difference between sedation on one hand and basal narcosis or anaesthesia on the other is largely one of degree, different doses of the same drugs being capable of producing all three.

In this work however the terms are used to differentiate the two main methods used by the anaesthetist to facilitate cardiac catheterisation.

Where a narcotic or hypnotic drug is administered to allay anxiety without producing sleep the patient is described as having been examined under sedation only.

The terms basal narcosis and anaesthesia are used synonymously where a state of deep sleep (or light anaesthesia) is induced by rectal injection. Such a technique in anaesthetic practice was normally a preliminary to further anaesthesia but in the present study the Bromethol so administered has been the main - and in many cases the only true anaesthetic employed.

CHAPTER FOUR.

METHODS OF SEDATION AND NARCOSIS.

Baldwin et al. (1946) reporting the results of cardiac catheterisation in patients with ventricular septal defects, stated in a footnote that Cournand had reported examinations under Bromethol narcosis. This was the first reference to the use of Bromethol to facilitate the procedure but many more were to follow. While many of the earlier authors merely mentioned the use of Bromethol as an incidental part of their technique, and did not comment on its merits, Lehmann et al. (1951) considered it the drug of choice in children. Light et al. (1950) state that using it in a dose of 100 mgms/Kilo, they did not require to supplement it but Cournand et al. (1949); Cernuschi (1950); Sussmann et al. (1951) and Millar (1952) gave further doses of Bromethol during the catheterisation. This however, involves a rectal injection in the X-ray department, and thiopentone became popular as the supplementary agent as it can frequently be administered via the catheter (Holling and Zak, 1950; Adelman et al., 1952; Gibson, 1953 and Fieldman et al., 1955). Gibson in a footnote to his paper recommends alternatively the use of

small doses of pethidine intravenously and this has also found favour with Duncalf and Thompson (1956).

Rectal thiopentone supplemented by intravenous thiopentone although providing a shorter period of narcosis is preferred by some authors and its use is recommended by Green et al., 1950; Wood, 1950; Deuchar and Knebel, 1952; Carnegie, 1953; Inglis, 1954; Harned, 1955 and Fowler et al., 1957. Keown et al. (1957) in addition discuss the use of thioamyl and thiopentone administered intramuscularly, and intravenously. Smith (1950) uses pentobarbitone premedication followed by intravenous thiopentone while Lundy (1958) after rectal thiopentone administers a variety of sedative agents using their antidotes if required. Another barbiturate "Narcodorm" has been used intravenously by Boeson et al. (1956).

Oral barbiturates have been used to provide light sedation and the following have been reported as satisfactory:- Phenobarbitone (Bustamente et al., 1952); pentobarbitone (Brown et al., 1949); quinalbarbitone (Barratt Boyes and Wood, 1957) and amylobarbitone (Lucas and Short, 1952).

Morphine and scopolamine were used by Goldman et al. (1950) and Bing (1952) but Dickerson (1954) feels that opiates increase the incidence of venospasm

and he prefers pethidine. This drug is also the choice of Bruce (1954). More recently it has been combined with promethazine and chlorpromazine and given as a "lytic cocktail" by Smith et al. (1958) and Mitchell and Minor (1958).

Volatile agents have long been known to interfere with the performance of the Van Slyke analysis as have gases such as nitrous oxide and cyclopropane (Orcutt and Waters, 1937). Kepes et. al. (1955) however describe the use of nitrous oxide and oxygen in conjunction with photoelectric gas analysis. They consider this is safer than basal narcosis though they admit it is less accurate. Keats et al. (1958) recommend the use of Trichlorethylene in air as an anaesthetic and they suggest that this does not materially affect the Van Slyke analysis. Their figures do not however support their claim. When the results of the Van Slyke analysis with and without Trilene in the blood are compared, it is seen that there is a mean difference in oxygen values of one volume per cent, which is quite unacceptable. In addition, tachypnoea occurred in a high percentage of cases and respiratory rates of up to 84 are reported. Eggers et al. (1959) report the use of nitrous oxide, oxygen and Trilene as a light general anaesthetic but

again tachypnoea was a troublesome complication occurring in 36% of cases. Here respiratory rates over 100 are reported in 5 cases (out of 67). Trichlorethylene does not appear to offer much help to the problems of anaesthesia in this field. Halothane-Ether mixture has been used in a few cases by Adams and Parkhouse (1960) but appears to be of limited value.

Ziegler (1954), Kepes et al. (1955), Lima (1956) and Clark (1957) stress distraction by a kind and gentle nurse as the best method of dealing with a child undergoing cardiac catheterisation but on perusal of their papers it is obvious that this is intended mainly for older children and is combined with sedation.

Finally hypnosis has been recommended by Mason (1958) and the advantages of the method are enthusiastically described in some detail. However, Mason in a personal communication states that he has no experience in this field and cannot recall the source of his information.

From this survey it is evident that in children the majority of authors employ basal narcosis induced by rectal injection and where necessary supplement this by intravenous barbiturate or pethidine. The choice of agent for the initial injection varies from Bromethol

to thiopentone, the former being preferred where a longer period of narcosis is desired or where it is desired to minimise the use of supplementary anaesthesia. Inhalational anaesthesia is used only in the form of nitrous oxide and oxygen to facilitate the surgical "cut down" when it is impossible to perform a venipuncture and give thiopentone or where it is not reasonable to restrain the child while local anaesthesia is injected. The use of nitrous oxide or volatile agents is otherwise eschewed by all but the authors quoted and even they recognise the limitations thus imposed.

Where only sedation is aimed at, a wide variety of agents are used according to local preference. No specific claims are made for any particular drug. The use of a mixture of chlorpromazine, promethazine and pethidine by intramuscular injection appears to fall between the previous methods by providing deep sedation from which the patient may awake or be awakened. This so called "lytic cocktail" appears popular where an anaesthetist is not present at the time of catheterisation.

It seemed desirable to inquire what methods were used in other hospitals in this country and the following table shows the results (Information obtained over period 1957 - 59).

TABLE 1.

Name of Hospital	Main Agent(s)	Source of Information
Hospital for Sick Children, Great Ormond Street, London.	"Lytic Cocktail"	Dr. G. Graham
Westminister Hospital	Promethazine, Pethidine and Atropine + Nitrous Oxide	Dr. P. Cliffe.
Liverpool Hospitals	Thiopentone	Dr. G.J. Rees
Newcastle Hospitals	Thiopentone	Dr. J.Hutchison
Manchester Royal Infirmary	"Lytic Cocktail"	Dr. G. Howitt
Cardiff Royal Infirmary	Bromethol	Dr. S. Galloon
Belfast Hospitals	Rectal Thiopentone, Nitrous Oxide, Oxygen, Halothane	Dr. G.W. Black
GLASGOW		
Western Infirmary	Thiopentone	Dr. E. Ross
Victoria Infirmary	Thiopentone	Dr. J. Dickson
Royal Hospital for Sick Children	Phenobarbitone, Papaveretum and Scopolamine	Dr. G. France
Stobhill Hospital	Bromethol	Dr. D. Campbell
Southern General Hosp.	Thiopentone	Dr. D. Hart.

SELECTION OF ANAESTHETIC TECHNIQUE.

Having considered fully the theoretical requirements of any anaesthetic technique employed to facilitate cardiac catheterisation and having surveyed those which have been used, it was decided to try to evolve a method which would be applicable to all types of patients with congenital heart disease.

SEDATION.

The practice of merely sedating patients over the age of twelve so as to produce a drowsy state has normally been continued. The agents employed varied from barbiturates, when simple cardiac catheterisation was contemplated, to papaveretum and scopolamine when angiocardiography was to follow. An open mind was kept to the possibilities of using other sedative or tranquillising drugs if and when they became available.

BASAL NARCOSIS.

It is in the age groups below twelve that basal narcosis is primarily employed. Although it is possible to induce deep narcosis by giving repeated oral doses of barbiturate the results are notoriously unreliable. Methods involving injections are, in a varying proportion of children, associated with crying and struggling such as would be potentially dangerous

in many of the children likely to be encountered. Inhalational methods were not considered in view of the theoretical objections discussed. Rectal injection thus appeared to be the method of choice. It had been found to cause no upset in the vast majority of children and it enabled anaesthesia to be induced in the child's own bed by nurses whom he had known over a week or two. The fact that this method was used in thyrotoxicosis for many years before anti-thyroid drugs were available is a tribute to the ease with which anaesthesia can be induced in the most difficult patients. There are however the usual drawbacks associated with it, namely the uncertain rate of absorption of the drug and the difficulty of recovering the drug after it has been given. With careful limitation of dosage and slow administration of the enema however these have not been found to be troublesome.

The choice of agent lay between thiopentone, bromethol and paraldehyde. Experience with thiopentone and the reports quoted in the literature (pages 31 and 32) showed that this drug did not provide a long enough period of narcosis and supplementary intravenous thiopentone was required. Paraldehyde which had been extensively used in the Cardiology Department for cardiac catheterisation had also required supplementation

with thiopentone and it was considered to be theoretically undesirable in view of the possible interference with the gas analysis caused by its vapour in the blood and in the lungs through which it is partly excreted. Bromethol was therefore selected as the main agent in the narcotic technique.

Bromethol was first used in this country as a basal narcotic by Blomfield and Shipway (1929). It is marketed as a solution of tribromethol alcohol in amylene hydrate, one Gram being contained in one millilitre. This solution is diluted with sterile distilled water to a final concentration of 2½% and is administered rectally at body temperature, the injection occupying at least ten minutes. After the injection, the patient falls asleep and the transition is notably free from excitement (Hewer, 1953).

EFFECTS OF BROMETHOL.

With the onset of sleep the minute volume falls although the respiratory rate rises. The respiratory centre is depressed and its threshold to carbon dioxide is raised. The metabolic rate is lowered by about 15% (Adriani, 1954). Respiration is regular and continues thus during the period of narcosis.

The vasomotor centre is depressed and there is a fall in blood pressure, mainly systolic, of about 20%. The heart rate is raised. (Adriani, 1954).

Blomfield (1929) compares the drop in blood pressure to that encountered in normal sleep. Similar changes are reported by Goldschmidt and Hunt (1932) and Gorham (1933).

The effect of the drug on the heart itself is obviously of great importance and this has been studied extensively by Anschutz et al. (1930). As a result of their own work and an extensive review of studies in the German literature they conclude that Bromethol does not injure the healthy human heart. They also quote reports from colleagues who recommend it and have used it in cases with myocarditis and compensated cardiac lesions. They emphasise that it should not be used in cases of uncompensated heart disease or peripheral circulatory disease. Morton (1935) and Norris and Stevens (1936) have studied the electrocardiographic changes during Bromethol narcosis and found nothing beyond slight nyocardial depression. They share the views of Anschutz and his colleagues.

The effects of the drug on the liver were the subject of discussion for some years but most of the cases considered had been given other anaesthetics such as ether and had been subjected to surgical procedures which complicated the picture. Edwards (1954) considers that liver function is not impaired and McKim and Bourne (1933) reported no disturbance of liver function in a

patient to whom Bromethol was administered 23 times in less than ten weeks.

The dose of the drug to be given was a matter of some considerable thought and discussion. The normal range of dosage is given as 80-120 mgms./Kilo although doses up to 175 mgms./Kilo have been reported as being safely given to children in combination with morphine and scopolamine (Hewer, 1953). Tannenbaum (1934) found that the main difference with increasing dosage was an increase in the duration of sleep produced. However Fieldman et al. (1955) reported that using larger doses of Bromethol (125 mgms./Kilo) they required less supplementary anaesthesia and that the patients tolerated cardiac catheterisation better in addition to producing more reliable data. A standard dose of 120-125 mgms./Kilo has normally been used in the present series as being the most suitable.

It had been the custom prior to the beginning of this study to augment premedication for general surgical cases and for cardiac catheterisation with promethazine, given orally on the night before anaesthesia and on the morning of operation. This practice was based on the hope that the drug would minimise post-operative vomiting, reduce upper respiratory reflexes and augment the other sedative agents employed as claimed

by Beard (1954). The value of the drug as an adjuvant to pre-operative medication has recently been studied by Weiss and McGee (1956) and they consider that the sedative effect of pethidine is markedly enhanced. Light et al. (1957) also found that, although by itself it did not produce adequate sedation, it increased the sleepiness produced by other medicaments. Hopkin (1954) describes the effect as producing euphoria without hypnosis. Howarth and Owen (1954) studied the effects of intravenous injections of 25-50 mgms. of Promethazine during cardiac catheterisation. They found no significant change in systemic blood pressure and only small changes in pulmonary artery pressure and oxygen consumption. In all cases there was a rise in heart rate. The quinidine like action of promethazine in minimising arrythmias is commented on by Howarth and Owen and this effect is also noted by Hutcheon (1953) who reports that it is half as potent as quinidine in reducing the maximum rate of the driven auricles. This author reports that it has a slight drying effect on saliva and other secretions in animals and a marked antisialagogue effect has been demonstrated in man by Dobkin et al. (1958). Restlessness was noted by Eckenhoff et al. (1957) after the administration of

50 mgms. of promethazine to healthy volunteers. They felt sleepy but could not sleep and changed position continuously. This restlessness was accompanied by a rise in minute volume and respiratory rate and a fall in alveolar carbon dioxide. These changes persisted even after the addition of a barbiturate to the promethazine but disappeared when pethidine and promethazine were given together. The blood pressure was variable after promethazine but postural hypotension was marked. Tachycardia invariably occurred. It was decided to continue to give promethazine orally on the night prior to cardiac catheterisation and three hours before the catheterisation was due to commence. This in addition to the effects described produces mild sedation which allows the enema to be given with minimal upset. The dosage employed is 10-25 mgms., 10 mgms. being given to the smallest children up to 2 stones in weight, 20 mgms. up to 4 stones and 25 mgms. thereafter.

Pethidine was recommended as a supplement to the main basal narcosis by Gibson (1953) and Duncalf and Thompson (1956). They gave the drug intravenously during the procedure itself. The desirability of avoiding injections during the actual catheterisation

has been stressed and experience in the operating theatre has been shown that profound falls in blood pressure and bradypnoea may follow intravenous pethidine even in small doses. It was therefore decided to give pethidine intramuscularly 20 minutes before the catheterisation started and so to obtain the maximum analgesic effect at the time of the surgical "cut down". The effects of intramuscular injection of this as of any other drug are slower in onset and of longer duration than when the drug is given intravenously. This seems particularly advantageous in the present circumstances where sudden changes are undesirable. The duration of effect of intramuscular pethidine, two hours, (Lee, 1959) is almost ideal for the purpose.

In addition to the analgesic action for which pethidine is primarily used it has an atropine like action and thus dries secretions in mouth and bronchi (Adriani, 1954). It also has a quinidine like action which Mushin and Rendell Baker (1950) find of value in preventing cardiac arrythmias during thoracic surgery. Respiration is usually slower and deeper than normal and in common with Bromethol and promethazine, pethidine dilates the bronchi. In this present work it was decided to give pethidine in a dose of 1 mgm./lb. of body weight, the dose being rounded off to the nearest

10 mgms. below for ease of measurement and administration.

It is normal anaesthetic practice to administer atropine or scopolamine as part of routine premedication before a general anaesthetic is given. These diminish bronchial secretions, and minimise undesirable vagal reflexes such as cardiac slowing and bronchial constriction. In normal dose atropine causes a mild tachycardia - Lee (1959) claims a rise in pulse rate of 20-30 following the administration of gr. 1/60 th. While this is perhaps undesirable it seemed essential to include atropine in doses ranging from gr. 1/200 - gr. 1/100 to avoid the excessive mucus found by Heard (1933) when Bromethol was given without atropine and to protect the patient lest intravenous thiopentone was required during the course of the investigation.

SUPPLEMENTARY ANAESTHESIA.

Although it is undesirable on theoretical grounds to use supplementary anaesthesia - and where the initial dose of the basal narcotic has been carefully given it is seldom required. It was recognised that on occasion it might be desirable to use an intravenous agent to control restlessness. Since this need would only arise where the restlessness was causing serious concern it was considered that a rapidly acting agent

should be employed. Thus dilute thiopentone was selected. Where it is impossible to find a suitable vein it seemed reasonable to follow established practice and administer nitrous oxide and oxygen until the catheter was in situ. When this is done it is essential to allow ten minutes to elapse before drawing blood off for sampling. Nicloux (1908) found that ten minutes after discontinuing nitrous oxide administration the gas was undetectable by chemical means in the blood. This work has been amply confirmed by American workers (Adriani, 1958 personal communication).

SUMMARY OF PROPOSED TECHNIQUES.

(Timings are based on cardiac catheterisation starting 11 a.m.)

Under age 12.

Night before investigation	Promethazine 10-25 mgms. (orally)
Morning of investigation 8 a.m.	Promethazine 10-25 mgms. (orally)
10.20 a.m.	Bromethol 120-125 mgms./Kilo (rectally)
10.40 a.m.	Pethidine 1 mgm./lb. Atropine gr. 1/100 (intramuscularly)
During catheterisation	Thiopentone 2½% only if essential, Nitrous oxide if no vein available.

Over age 12.

For Cardiac Catheterisation only	Barbiturate orally.
If Angiocardigraphy in addition	Papaverteum/Scopolamine (subcutaneously)

In each case may reinforce with Promethazine.

PREVENTION OF ARRYTHMIAS DURING CARDIAC CATHETERISATION.

The almost invariable occurrence of cardiac arrythmias during the course of the investigation has been commented on at an earlier stage. Although these are rarely of serious import they may be troublesome or even dangerous. Accordingly it has, in the past, in many centres been the practice to administer drugs - usually quinidine sulphate or procaine amide - before or during the catheterisation to abolish or minimise the irregularities. Opinions on the worth or even the advisability of these measures vary widely. Carnegie (1951), Skaggs and Chapman (1952), Dickerson (1954) and Steen (1954) all give quinidine beforehand but do not comment on its merits. Similarly Pietrafesa (1954) gives procaine amide before catheterisation, Bustamente et al. (1952) give it during catheterisation.

Others again stress the value of the drugs and Heymans (1951), Hayward (1952), Goodwin (1952) and Scherf (1953) all found that one or other or both reduced the incidence of arrythmias.

The view that the drugs are of no value was expressed by Bing (1952) and this view was endorsed by the Committee on Cardiac Catheterisation and Angiocardiography of the American Heart Association of

which Bing was a member (Cournand et al. 1953). Even stronger are the views of Adelman et al. (1952) who acknowledge a personal communication from Lief that procaine amide is useless and they then state that they feel that quinidine being a cardiac depressant is positively dangerous. This statement is endorsed by Duncalf and Thompson (1956).

Lucas and Short (1952) report the results of a trial of procaine amide in the prevention of catheter induced arrythmias. They found that with doses up to 500 mgms. they produced a fall in systemic and pulmonary artery pressure but that the arrythmias were uninfluenced. However, Zinn et al. (1952) in a study of 113 cases concluded that where the stimulation from the catheter was mild or moderate the incidence of arrythmias was significantly reduced by either procaine amide or quinidine but that neither was of any value when the stimulation was severe. Lucas (1958) in a personal communication states that he has found that the use of quinidine will avoid a number of spontaneous arrythmias but if serious arrythmias arise they are made worse.

The effect of the oral administration of 800 mgms. of quinidine on normal persons was studied

by Ferrer et al. (1948). Cardiac catheterisation revealed no change in right ventricular pressure or cardiac output but a fall in systemic blood pressure due to vasodilatation. A group of cardiac patients similarly investigated showed also a fall in right heart pressure.

The wide diversity of views expressed is no doubt partly due to the wide variation in the occurrence of arrythmias from patient to patient, in the same patient at subsequent catheterisations and even during the performance of the procedure when, for example, exploration of the right ventricle may produce multiple extrasystoles on some occasions but not on others. Many feel that it is extremely difficult to evaluate the worth of the drugs in reducing arrythmias although there can be no doubt that neither drug prevents them. The practice of the cardiologists in Glasgow Royal Infirmary has been to give quinidine orally before catheterisation and this has been continued in the series of cases reported here. On the few occasions when supraventricular tachycardia has occurred, it has been treated by the intravenous administration of procaine amide.

The technique outlined for providing a steady state with basal narcosis has been developed therefore on a consideration of the requirements of the procedure,

the condition of the patients likely to be encountered and a knowledge of the pharmacology of the drugs which may be employed. It seemed possible, even probable, that this technique would provide a satisfactorily steady state - a claim made for many techniques - and to prove or disprove this an assessment of the condition of these patients has been made at intervals during the performance of the catheterisation.

CHAPTER FIVE.

ASSESSING THE STEADY STATE.

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ASSESSING THE STEADY STATE.

METHODS:

In discussing the "steady state" it was noted that conditions were aimed at under which the oxygen content and blood pressures at various sites within the heart and great vessels would remain constant over a considerable period of time. The factors likely to upset this stability were discussed.

It seemed essential to carry out some measurements to ascertain to what extent a steady state was being maintained and whether the factors likely to upset it were being suppressed. In addition by employing the same methods in the ward before cardiac catheterisation it was possible to obtain resting values for each patient and compare these with the conditions prevailing during catheterisation.

In deciding which measurements to make, many factors had to be taken into account. In the first place methods involving bulky apparatus and a great deal of time and assistance were impracticable. The checks could be made during the period when sampling of blood from the catheter and pressure recording were being carried out thus ensuring that the samples were indeed suitable for comparison. This, however, meant

that only a limited time was available in which to perform the selected tests. Again, if it was proposed to carry out these tests in the ward to obtain resting values they must be applicable to small children without upsetting them emotionally. They must of course be accurate, although as comparisons were being made, relative as opposed to absolute accuracy was the main object. To be of any value they had also to produce results rapidly so that in the event of an upset of the steady state new samples and readings could be obtained.

It was considered practicable to monitor before and repeatedly during the procedure:-

1) Arterial oxygen saturation
2) Systemic blood pressure
3) Heart rate.

and to note any respiratory variations, undesirable features, etc. as and when they occurred.

In addition it was possible to obtain a sample of arterial blood which provided information regarding the arterial pH and carbon dioxide tension, during the catheterisation. It was not considered practicable to do this repeatedly during the procedure or in the ward beforehand, for reasons discussed later.

Where the patient was sedated but remained awake a close watch was kept to note any sensations reported which might give a clue to the better management of basal narcosis in others.

Initially it was hoped to include further respiratory data such as respiratory rate and depth and oxygen consumption figures but for reasons discussed later this was abandoned.

The tests to be made and their application and limitations are now considered.

OXYGEN SATURATION.

Choice of Site for Measurement.

The suggestion of Johnson and his colleagues (1947) that the oxygen content at one site within the heart should be studied at intervals raises many problems and would involve the use of two catheters. In addition it would be difficult to know in which chamber the catheter tip should be placed. However, it seemed reasonable to monitor the arterial oxygen saturation, as much valuable information could be gained in this way. Thus, most respiratory disturbances, changes in the amount of right to left shunt or reversal of a left to right shunt would be reflected in changes in arterial oxygen saturation. Any change in the amount of blood contained in a left to right shunt is

not revealed but unless reversal takes place such a shunt would still be detected by catheterisation.

It will be seen that most of the changes likely to be produced by basal narcosis - or at least frequently attributed to it - such as respiratory depression or obstruction and severe blood pressure drops would cause a change in the arterial oxygen saturation.

Choice of Method of Measurement of Arterial Oxygen Saturation.

Visual estimation of cyanosis, it's presence, absence or degree, is virtually worthless as a means of measuring oxygen saturation, the classic paper of Comroe and Bothelo (1947) having demonstrated that even skilled observers showed considerable variation in the levels at which they detected cyanosis in different patients and even in the same patient at different times. Boere (1949) has shown that with a falling arterial saturation marked electrocardiographic changes may precede the onset of cyanosis.

Arterial puncture with subsequent estimation of the oxygen content has been rejected as impracticable for many reasons. Particularly in children, the emotional and respiratory upset which ensues makes a mockery of the estimation of oxygen content under

conditions of rest (Burchell, 1948, 1950; Hultgren and Hackett, 1950; Sekel, 1954) and Potts (1949) claims that false low readings are frequently obtained. Changes which take place rapidly could not be recorded because of the time needed to withdraw a sample of blood and the number of samples could be examined is limited by the blood loss involved (Livingston and Adams, 1952). The performance of the arterial puncture may be technically difficult especially in small children and it has caused circulatory collapse even when performed in stable co-operative adults under local anaesthesia (Rushmer, 1944).

In face of these difficulties it is usual to take advantage of the fact that the vasodilated ear contains blood which is equal in oxygen content to arterial blood and to use an ear oximeter to measure the oxygen saturation in the intact ear.

The theory practice, accuracy and limitations of ear oximeters and in particular the "Starco" oximeters are discussed in an appendix.

It has proved possible to use the oximeter even in small children without any upset whatever. Readings have been taken in the ward, at rest, and at frequent intervals from the time the patient arrives in

the X-ray room. In particular a reading has always been taken as blood is withdrawn from the catheter.

BLOOD PRESSURE.

Since the drugs used in the production of basal narcosis have been frequently accused of producing profound circulatory depression, it is essential to keep a careful record of the systemic blood pressure. Not only does this protect the child from danger but much valuable information can be obtained about the conditions under which the investigation is being carried out.

In discussing the steady state it was noted that changes in blood pressure are likely to interfere with the validity of the measurements obtained during cardiac catheterisation. They may alter the direction of flow of the shunt. In some cases the factors causing systemic changes alter the right heart pressures also - although to a lesser extent - and while this will lessen the effect of shunt flow it will give false pressure readings in the right heart and pulmonary vessels.

Factors causing Systemic Blood Pressure Change.

1) Anxiety is usually manifest by blood pressure fluctuations.
2) Muscular movement or restlessness may vary the pressure.
3) Disturbed respiration is similarly reflected.

4) Uncompensated change in either the cardiac output or peripheral resistance will by definition change the blood pressure.
5) Administration of depressant drugs may produce changes.
6) Alteration of the constitution of the respired gases is a further factor.
7) The pressure will fall during sleep.

It is obvious therefore that all the adverse factors mentioned in Chapter 2 will cause fluctuations in systemic blood pressure and it is possible to detect the influence of these factors - and their effects go far beyond the systemic blood pressure - by regularly measuring the brachial artery pressure.

Methods of Measuring Systemic Blood Pressure.

As with the measurement of arterial oxygen saturation the choice of method was governed by practical considerations. Thus while it is no doubt true that the most accurate method of measuring blood pressure is to cannulate a major vessel and thus connect it through an electromanometer to a recording device this would have added considerably to the technique, as it was normal practice merely to perform a single arterial puncture to obtain blood. The risk of arterial cannulation although perhaps not great was not considered justifiable unless

other methods were unsatisfactory. Furthermore resting values in the ward could obviously not be obtained by arterial cannulation and the method used should be the same throughout. Readings have therefore been taken in the ward and during the investigation by auscultation over the brachial artery in conjunction with a standard mercury column sphygmomanometer.

Accuracy of Method.

Compared with the direct measurement of blood pressure through a manometer the indirect method using a sphygmomanometer and stethoscope falls slightly short of ideal in providing absolute readings. In a series of thirty comparisons Hamilton et al. (1936) found the systolic pressure estimated by the indirect method to be 3-4 mms.Hg. too low while the diastolic pressure was recorded about 9 mm.Hg. too high. Bergen et al. (1954) found that the difference increased with higher pressures. Similar deviations were reported by Bordley et al. (1951).

In practice these deviations are probably not of great significance and it is tempting to suggest that the difference might be due to slight anxiety resulting from the arterial puncture when the effects of this procedure (Rushmer, 1944) are borne in mind. The present investigation is in any case concerned mainly

with changes in blood pressure rather than with absolute values and as with oximetry the accuracy of indirect measurements is greater when the readings are taken by the same observer in the same patient by the same method (Bergen et al., 1954). It is in these circumstances that the method has been used.

The recommendations of Bordley et al.,(1951) have been carried out in this work.

The Resting Blood Pressure.

Bordley et al. (1951) suggest that to measure the resting blood pressure the same conditions should be established as are used to measure the basal metabolic rate. Accepting this literally, in view of the work of Fraser and Nordin (1955), would involve sedating the patient to the point of sleep. It may well be therefore that we are only really measuring the "resting" blood pressure under basal narcosis. However in the present work a measure of the resting blood pressure as normally accepted has been sought to enable a comparison to be made with the values found under basal narcosis or sedation. The futility of relying on one estimation made under circumstances strange to the patient is only too well known. In this series therefore I have not attempted to take blood pressure readings on the pre-anaesthetic visit to the patient but have relied on the blood pressure chart where records have been taken at rest

in bed by the House Physician each morning over a week or so. The charts characteristically show a falling curve which levels off after 3-4 days and it is from this point that the resting level is taken. This obviously brings in question the error incurred by using different observers but the residents have had explained to them the recommendations discussed above and on frequent occasions I have checked their measurements during the investigation. The differences have almost invariably been less than 5 mm.Hg. and never more than 10 mm.Hg. This is much less than the error likely to be caused by some one relatively unknown to the patient taking the reading.

The indirect method of blood pressure recording described appeared to offer a satisfactory base line for the purposes required. The resting value was obtained as described and repeated readings were taken during the investigation particularly while sampling and pressure recording took place.

HEART RATE.

Most of the other factors which will upset the steady state are likely also to disturb the heart rate. A considerable fluctuation in heart rate is one of the sighs of anxiety which we must avoid if possible. In addition excessive stimulation of the heart by the catheter

is reflected by irregularities of pulse rate, rhythm or both, which are often accompanied by marked though temporary changes in haemodynamics.

Measuring the Heart Rate.

Pulse counters attached to a thumb or finger tip have been used in this and other fields of anaesthetic work but have not been uniformly reliable. A stethoscope strapped at the angle of Louis or the Telephonic Stethoscope (Inglis, 1954) may be employed. Perhaps the most obvious method of estimating heart rate, particularly if there is a pulse deficit, is by watching the oscilloscope on which the electrocardiogram is recorded. This or palpation of an artery have been the methods employed in this series.

Resting values were obtained as for blood pressure by studying the pulse chart kept during the week or so that the patient was in hospital before catheterisation. As before readings were taken repeatedly during the investigation while sampling was being carried out.

These measurements serve two purposes. Firstly, by comparing the resting values with the conditions prevailing during the catheterisation it is possible to delineate the state which is obtained by the sedation or basal narcosis. Secondly, by comparing the readings during the examination we can see to what extent this state

this state is steady.

OTHER TESTS.

Other information can be obtained to try to determine what conditions prevail during part at least of the cardiac catheterisation. In the latter part of the work reported, it was possible to estimate the carbon dioxide tension in the arterial blood by measuring the total carbon dioxide content and the pH and substituting in the Henderson - Hesselbach equation. Later the Astrup (1956) Method was used. This value has been measured only once during each catheterisation at the time that an arterial sample was being withdrawn to allow oxygen content to be measured prior to determining cardiac output. The reading was considered in relation to the normal range of values and no attempt was made to perform an arterial puncture in the ward to obtain so called resting values. Information regarding carbon dioxide tension and pH was considered desirable to demonstrate to what extent respiratory depression was taking place and whether carbon dioxide tensions would be reached such as would increase cardiac irritability.

In those cases examined under sedation but remaining awake careful note was taken of any complaint of pain or discomfort howevere slight or transient, as it was felt that some of these might explain the restlessness sometimes encountered during basal narcosis.

CHAPTER SIX.

MATERIAL

CHAPTER SIX.

MATERIAL.

The series here examined consists of 122 cardiac catheterisations carried out on 110 patients. They can be divided into two groups, namely those who were examined under basal narcosis "the anaesthetised group", and the remainder who were examined awake but under the effects of sedation "the sedated group". Details are shown in Table 2.

TABLE 2.

Number of Cardiac Catheterisations.

Under anaesthesia	63
Under sedation	59

Sex.

	Anaesthesia	Sedation.
Male	31	32
Female	32	27

Ages.

	Anaesthesia	Sedation.
Under 5	14	nil
5 - 10	38	2
11 - 15	8	13
16 - 20	2	21
21 - 25	1	9
26 - 30	nil	3
31 - 35	nil	5
36 - 40	nil	3
Over 40	nil	3

The two groups are comparable in numbers and sex distribution. The differences in ages are to be expected as the indications for anaesthesia are found in the younger patients while older patients are wherever possible examined under sedation. Two children aged ten were sedated but remained awake in the early part of the work as they were considered to be very placid children. Older patients were anaesthetised only when this was considered necessary - on account of extreme nervousness, mental deficiency or lack of co-operation or at the patients insistence. This was our normal practice and was not altered in any way for the purposes of the investigation.

DIAGNOSIS.

The nature of the lesions present in the patients examined can be seen from a study of Table 3. The series are comparable as regards the lesions present. The majority of cases in each group were acyanotic.

All cases prior to the examination were presumed to be cases of congenital heart disease. The patient with pulmonary fibrosis was considered to be a case of Eisenmenger's Syndrome while the patients with mitral stenosis were suspected of having in addition atrial septal defects.

TABLE 3.

DIAGNOSIS:	Anaesthesia	Sedation
Pulmonary Stenosis	9	14
Atrial Septal Defect	11	8
Ventricular Septal Defect	14	12
Pulmonary Hypertension	1	1
Coarctation of Aorta	1	4
Fallot's Tetralogy	7	5
Patent Ductus Arteriosus	5	3
Aortic Stenosis	3	3
Mitral Stenosis	2	5
Dextrocardia	0	1
Ebstein's Disease	0	2
Pulmonary Fibrosis	0	1
Not Established	8	0
Left Superior Vena Cava	2	0

Anaesthetic and Sedative Agents.

Anaesthetic Agents.

The agents used to produce basal narcosis and their dosage are listed in Table 4.

TABLE 4.

Basal Narcotic	Number of Cases	
	Supplemented (By Intravenous Thiopentone).	Unsupplemented
Paraldehyde 1 dr.(4 c.c.)/ stone	3	0
Bromethol 100 mgms/Kilo	7	5
Bromethol 110 mgms/Kilo	1	2
Bromethol 120-125 mgms/Kilo	5	40

All patients in this group had in addition to the basal narcotic

Promethazine 10-25 mgms. night and morning

Pethidine 1 mgm/lb of body weight

Atropine sulphate gr. 1/150 - 1/100 (0.4 - 0.6 mgms.)

Paraldehyde was used when Bromethol was temporarily unavailable. The smaller doses of Bromethol were employed in the early part of the series but were abandoned in favour of the larger ones when it became obvious that supplementation was frequently required. Supplements with the larger doses were needed only when part of the enema had been voided.

Sedative Agents.

These are shown in Table.5. The doses are included in the case sheets in an appendix.

TABLE 5.

Sedative Agents:	No. of Cases	
Pentobarbitone	9	
Pentobarbitone + Pethidine	1	
Pentobarbitone + Pethidine + Atropine	1	
Pentobarbitone + Pethidine + Promethazine	1	
Pentobarbitone + Promethazine	2	14
Amylobarbitone	7	
Amylobarbitone + Promethazine	13	
Amylobarbitone + Promethazine + Pethidine + Atropine	3	23
Promethazine + Pethidine + Atropine	1	1
Meprobamamte	2	
Meprobamate + Promethazine	2	4
Papaveretum + Scopolamine	10	
Papaveretum + Scopolamine + Promethazine	7	17

The group can be divided into four - those sedated mainly with pentobarbitone, amylobarbitone, papaveretum/scopolamine or meprobamate. In each case where there has appeared to be a need for greater sedation than that provided by the main agent, other agents have been added - normally pethidine or promethazine. Atropine has been given where it was feared that thiopentone might be required.

These groups, the anaesthetised and the sedated, are comparable in all respects other than age groups. They represent all the patients submitted to cardiac catheterisation under basal narcosis in Glasgow Royal Infirmary over a four year period and 59 of these examined under sedation. Those examined under sedation were not specially selected but were merely those examined at times at which I was able to attend the catheterisations. Many others have been examined under sedation at different times. This sedated group however included all those patients examined under sedation where it was intended to follow the catheterisation with angiocardiography.

The results of the tests described in the previous section when applied to these cases are now considered.

CHAPTER SEVEN.

RESULTS.

CHAPTER SEVEN.

RESULTS.

This chapter can be divided conveniently into three parts. In the first are presented the data showing the relationship of the state produced by anaesthesia or sedation to the resting state, measured as previously described; in the second the figures obtained by taking serial readings during cardiac catheterisations and thus giving a measure of the steadiness of the state obtained; in the third other relevant results.

PART ONE - Changes Produced by Anaesthesia or Sedation.

For comparison with the resting values, readings have been selected which were obtained:-

1) As the patient lay on the X-ray table while the preliminary procedures - attachment of electrocardiograph leads etc. - were carried out.
2) As the catheter entered the patient's heart.

In five patients of the anaesthetised group it was not possible to pass a catheter into the heart for technical reasons unrelated to the anaesthetic and thus readings as at (2) are not available. Where this is so the readings at (1) have been omitted from the comparisons

and analyses hereunder. These exclusions do not affect the results or the conclusions drawn from them.

A) OXYGEN SATURATION

The changes in the arterial oxygen saturation as measured by an ear oximeter are shown in Tables 6 and 7.

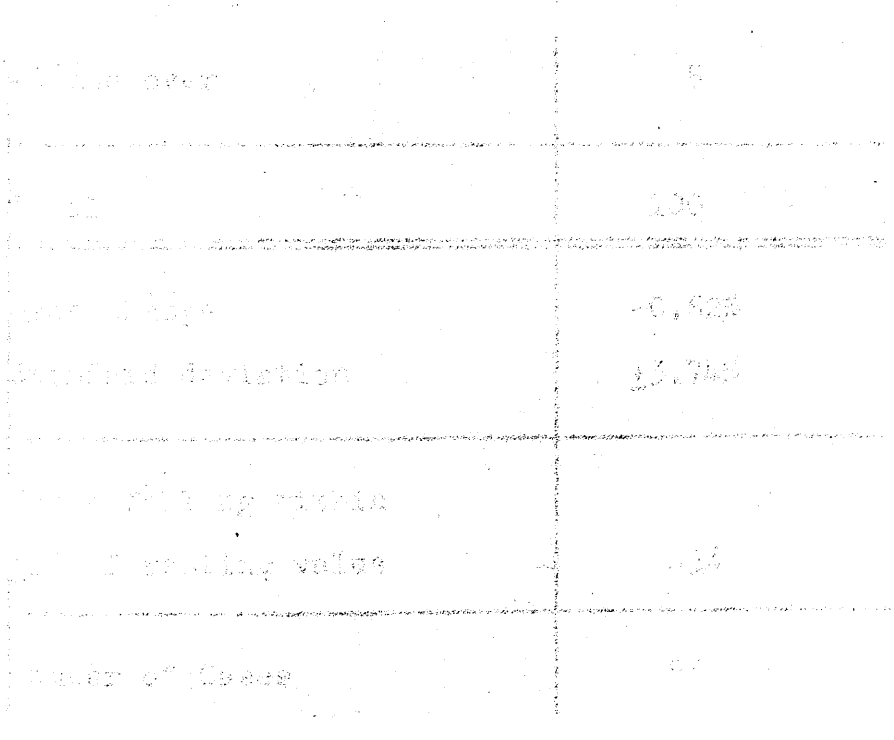

TABLE 6.

Changes in arterial oxygen saturation from resting state to beginning of cardiac catheterisation.

Change (Percentage Saturation)	Percentage of Cases Showing Change	
	Anaesthesia	Sedation
+3 and over	9 %	2 %
+2 and +1	17	11
Nil	38	49
-2 and -1	28	25
-3 and over	8	13
Total	100	100
Mean change	-0.62%	-0.59%
Standard deviation	±3.74%	±1.35%
Cases falling within ±2% of resting value	83%	85%
Number of Cases	53	55

At the beginning of catheterisation the only significant difference between the groups is in the greater variability of the results obtained under anaesthesia (F test $P < 0.05$).

These results give a measure of the safety of the methods employed as they reflect the maximum changes in oxygen saturation likely to be caused by depression of the patient by drugs or to stimulation by the surgical cut down to insert the catheter.

The conditions which prevail when the catheter enters the heart should be more favourable and it is these which may have a marked bearing on the interpretation of the values obtained by the physician during the investigation. They are presented in Table 7.

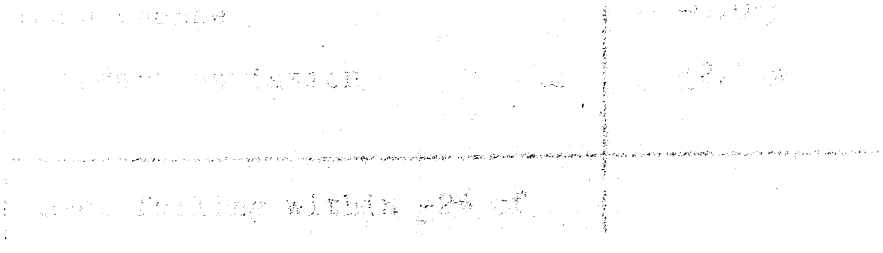

TABLE 7.

Changes in arterial oxygen saturation from resting values to time when cardiac catheter entered heart.

Change (Percentage Saturation)	Percentage of Cases Showing Change.	
	Anaesthesia	Sedation
+3% and over	10 %	2 %
+2% and +1%	12	11
Nil	40	36
-2% and -1%	34	38
-3% and over	4	13
Total	100	100
Mean change	-0.08%	-0.79%
Standard deviation	±2.24%	±1.8%
Cases falling within ±2% of resting values	86%	85%
Number of Cases	53	55

A χ^2 test reveals that there is no significant difference in the proportions of cases in the sub-divisions of the tables. A t test shows that there is no significant difference in the means and an F test shows that the variability of the methods is essentially the same.

Comparison with the conditions prevailing at the beginning of the procedure shows that in the anaesthetised group there is now less overall depression with less variation in the results, while the sedated cases show slightly greater scatter in the readings. Analyses reveal however that the differences between the results shown in Tables 6 and 7 are not significant.

B) CARBON DIOXIDE TENSION.

The samples removed in order to measure the oxygen content of the arterial blood were submitted to further analyses to determine their carbon dioxide tension and the results are presented in Table 8.

Using χ^2 a significantly larger proportion of cases (50% against 23%) have arterial carbon dioxide tensions below the normally accepted maximum of 40 mm.Hg. ($P < 0.01$). The variability of the results obtained is however greater with sedation and when the variances are compared by an F test this difference is found to be significant ($P < 0.05$). A t test shows that the means

are not however significantly different and are such as would be expected from the increase in arterial carbon dioxide tension (3.5 mm.) previously reported during sleep, by Robin et al. (1958).

TABLE 8.

Arterial carbon dioxide tension during cardiac catheterisation.

Tension (mm.Hg.)	Percentage of Cases	
	Anaesthesia	Sedation
30 - 39 mm.	23 %	50 %
40 - 49 mm.	59	28
50 mm. and over	18	22
Total	100	100
Mean tension	44.5 mm.	42.4 mm.
Standard deviation	±6.42 mm.	±10.2 mm.
Range	30-55 mm.	30-60 mm.
Total No. of cases	18	15

C) BLOOD PRESSURE.

Concurrently with the measurement of the arterial oxygen saturation with the oximeter, the systemic blood pressure was recorded as described previously. The changes from the resting levels to those prevailing at the beginning of catheterisation are set out in Table 9.

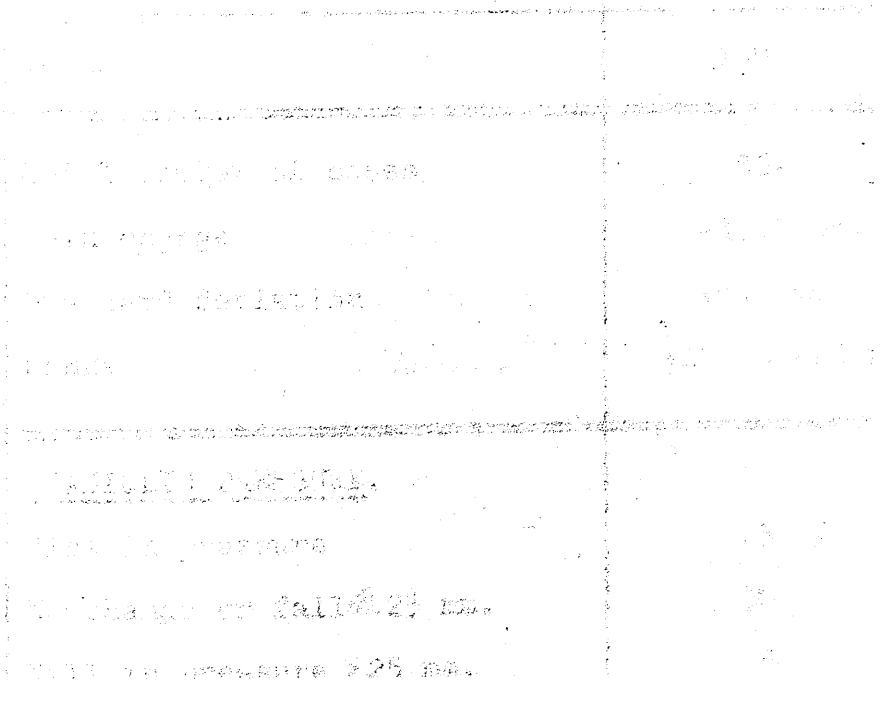

TABLE 9.

Blood pressure changes from resting levels to beginning of cardiac catheterisation.

SYSTOLIC PRESSURE.	Percentage of Cases Showing Change	
	Anaesthesia	Sedation
Rise in pressure	4%	42 %
No change or fall ≤ 25 mm.Hg.	87	51
Fall in pressure > 25 mm.Hg.	9	7
Total	100	100
Total number of cases	56	57
Mean change	-11.5 mm.	-1 mm.
Standard deviation	±9.3 mm.	±21.8 mm.
Range	+10 to -40 mm.	+40 to -105 mm.
DIASTOLIC PRESSURE.		
Rise in pressure	5 %	25 %
No change or fall ≤ 25 mm.	90	70
Fall in pressure > 25 mm.	5	5
Total	100	100
Total number of cases	55	57
Mean change	-8.5 mm.	-3 mm.
Standard deviation	±9.8 mm.	±13.2 mm.
Range	+20 to -30 mm.	+20 to -50mm.

Systolic Pressure.

The mean fall in pressure is greater after basal narcosis (11.5 mm.Hg.) than after sedation (1 mm.Hg.). A t test shows that this difference is highly significant ($P < 0.01$). The variability of the results obtained is much greater after sedation than after basal narcosis. An F test shows that this difference is again highly significant ($P < 0.01$).

A much larger proportion of patients show a rise in blood pressure after sedation (25 out of 57 = 42%) than after basal narcosis (2 out of 55 = 4%). A χ^2 test shows that the difference is highly significant ($P < 0.01$). This rise in pressure is probably a manifestation of uncontrolled anxiety.

Since it is the aim of the basal narcosis to obviate persistent anxiety by sedating the patient to the point of sleep one must expect the fall in blood pressure which accompanies sleep - up to 25 mm.Hg. Such a fall may be taken to show that the patient is relaxed and free from emotional stress. A study of the number of patients whose blood pressure falls but falls not more than 25 mm.Hg. shows that there are more of the anaesthetised patients than the sedated in this category (87% against 51%). A χ^2 test shows that this difference is highly significant ($P < 0.01$).

Severe falls in blood pressure seldom occur in the anaesthetised group, the maximum drop being 40 mm.Hg. in one patient. In the sedated group, falls greater than 25 mm.Hg. occurred in the same proportion (7% against 9%) but the maximum fall was greater at 115 mm.Hg.

Diastolic Pressure.

A t test shows that the difference in the means is still highly significant ($P < 0.01$) and an F test that the variability is significantly greater after sedation ($P < 0.05$). A χ^2 test shows that the proportion of patients showing a rise in pressure after sedation is significantly greater (25% against 5%) ($P < 0.05$) while the proportion showing a moderate fall in pressure is significantly greater after anaesthesia (90% against 70%) ($P < 0.05$).

As with the changes in oxygen saturation at the beginning of catheterisation, the blood pressure changes at this time are probably maximal due to the effects of the depressant drugs on one hand and anxiety on the other. However they indicate that neither method is likely to endanger life.

The conditions prevailing when the catheter enters the heart may be judged from the blood pressure changes summarised in Table 10.

TABLE 10.

Blood pressure changes from resting values to time of catheter entering heart.

SYSTOLIC PRESSURE.	Percentage of Cases showing Change	
	Anaesthesia	Sedation
Rise in pressure	2 %	37 %
No change to fall $\leq$ 25 mm.Hg.	89	54
Fall in pressure $>$ 25 mm.Hg.	9	9
Total	100	100
Number of cases	56	57
Mean change	-13.5 mm.	-4.5 mm.
Standard deviation	$\pm$ 9.9 mm.	$\pm$20.5 mm.
Range	+5 to -40 mm.	+25 to -115mm.
DIASTOLIC PRESSURE.		
Rise in pressure	7 %	23 %
No change or fall $\leq$ 25 mm.Hg.	88	75
Fall in pressure $>$ 25 mm.Hg.	5	2
Total	100	100
Number of cases	55	57
Mean change	-10 mm.Hg.	-3 mm.Hg.
Standard deviation	$\pm$10.6 mm.Hg.	$\pm$12.3 mm.Hg.
Range	+25 to -30 mm. Hg.	+20 to -50 mm. Hg.

Systolic Pressure.

The results at this stage show little change from those found at the beginning of the procedure. The differences in the means, variability and proportions of cases showing a rise in blood pressure and a moderate fall in pressure are all still highly significant ($P < 0.01$). There is in each series a slight fall in the mean blood pressure level as compared with that found at the beginning of catheterisation but the differences are not significant.

Diastolic Pressure.

The changes in the diastolic pressure follow the pattern set by the systolic but the differences between the series are not so great. A t test shows that the means are significantly different ($P < 0.02$) but the variability of the results with sedation although greater is now not significantly so. The proportion of cases showing a rise in pressure after sedation is still greater and the proportion of cases showing a moderate fall in pressure is greater after basal narcosis but these differences are not statistically significant. Again there is no significant change in the conditions from those prevailing at the beginning of the procedure.

D) HEART RATE.

As with the oxygen saturation and blood pressure the changes in heart rate are considered first as they show at the beginning of the procedure. These are summarised in Table 11.

TABLE 11.

Changes in heart rate from resting level to beginning of cardiac catheterisation.

Change	Percentage of Cases showing change.	
	Anaesthesia	Sedation
Fall in pulse rate	10 %	20 %
No change in rate or rise $\leq$ 20/min.	54	49
Rise in pulse rate $>$ 20/min.	36	31
Total	100	100
Mean rise in pulse rate/min.	15.5	15
Standard deviation	$\pm$14.3	$\pm$19.9
Range	-18 to +48	-10 to +82
Total number of cases	56	57

At this stage there is therefore little to choose between the methods as regards their effect on the pulse rate. The only significant difference is in the variability of the results obtained as measured by a comparison of the variances (F test). Here it is found that sedation produces more variable results ($P < 0.05$).

Finally one must consider to what extent these pulse changes have altered by the time that the catheter has reached the heart and sampling may be anticipated. The changes at this stage of the examination are summarised in Table 12.

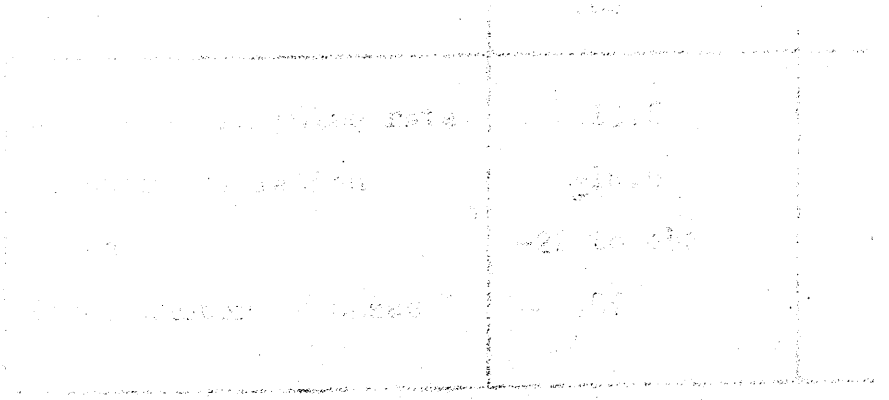

TABLE 12.

Changes in heart rate from resting level to catheter entering heart.

Change	Percentage of Cases showing change,	
	Anaesthesia	Sedation
Fall in pulse rate	10 %	21 %
No change or rise $\leq$ 20/minute	63	53
Rise in rate $>$ 20/min.	27	26
Total	100	100
Mean rise in pulse rate	13.6	12.6
Standard deviation	$\pm$14.6	$\pm$17.7
Range	-22 to +52	-16 to +62
Total number of cases	56	57

A comparison of the sedated and anaesthetised patients at this stage shows an almost similar picture. Again the only significant difference is in the variability of the results produced and again an F test shows that the variation within the sedated group is significantly greater than in the anaesthetised group ($P < 0.05$).

When the picture is compared with that existing at the beginning of the investigation it is seen that the sedated series is showing some improvement with a slightly smaller overall rise in pulse rate and a smaller variance. The anaesthetised patients are also slightly better, showing a slight fall in the mean rise in pulse rate. In neither case is the improvement of statistical significance.

PART TWO - Variation in The Resting State During Cardiac Catheterisation.

Having delineated the conditions of respiration and circulation which exist at the beginning of cardiac catheterisation and at the time when the catheter enters the patient's heart it is essential to determine what fluctuations take place during the entire investigation and in particular during the period when samples and readings are being taken or are liable to be taken. These are considered under the same headings as were the variations from the patients resting state.

ARTERIAL OXYGEN SATURATION.

The variations taking place are determined by measuring the difference between the highest and lowest readings on the oximeter.

Care has been taken to exclude from consideration any changes which are due to causes bearing no relation to the sedative or anaesthetic technique - such as obstruction of a stenosed pulmonary valve by the catheter. Such artefacts have occurred with the same frequency in each series and their exclusion does not affect the results. The changes recorded during the entire investigation are summarised in Table 13.

TABLE 13.

Variation in Arterial Oxygen Saturation During Cardiac Catheterisation (Entire Procedure).

Variation	Percentage of Cases showing Variation	
	Anaesthesia	Sedation
Nil, 1% and 2%	64 %	65 %
3%, 4% and 5%	24	27
6% and over	12	8
Total	100	100
Mean Variation	2.6%	1.4%
Standard deviation	± 3%	±2.0%
Range	0 - 15	0 - 10
Number of Cases	53	55

The fluctuation in arterial oxygen saturation during the entire procedure is similar in both series. An F test shows that the variability of the results under anaesthesia is however significantly greater ($P < 0.05$).

Many of the changes have been associated with the stimulation of the surgical cut down and one would anticipate better conditions when only the variations taking place during the period of sampling are considered. That this is so is shown in Table 14.

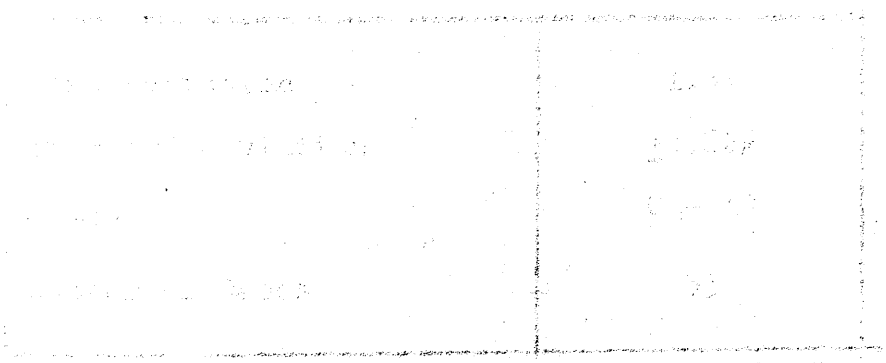

TABLE 14.

Variation in arterial oxygen saturation while catheter is in heart or great vessels.

Variation	Percentage of Cases showing Variations	
	Anaesthesia	Sedation
Nil, 1% and 2%	80 %	70 %
3%, 4% and 5%	16	22
6% and over	4	8
Total	100	100
Mean variation	1.5%	2.0%
Standard deviation	±1.82%	±2.2%
Range	0 - 10	0 - 10
Number of Cases	53	55

Better conditions are now obtained by the use of basal narcosis, but none of the differences are significant. Compared with the changes taking place during the entire period of the investigation an F test shows that there is a significant improvement in the variability of results in the anaesthetised group ($P < 0.05$).

BLOOD PRESSURE.

The variations in blood pressure during the entire period of cardiac catheterisation are shown in Table 15.

TABLE 15.

Blood pressure variations during cardiac catheterisation (entire procedure).

SYSTOLIC. Variation	Percentage of Cases showing Variation	
	Anaesthesia	Sedation
0 - 10 mm.Hg.	66 %	40 %
11 - 20 mm.Hg.	22	31
21 mm.Hg. and over	12	29
Total	100	100
Mean Variation	11 mm.Hg.	18.5 mm.Hg.
Standard Variation	± 7.94 mm.Hg.	± 13.9 mm.Hg.
Range	0 - 35 mm.	0 - 55 mm.
DIASTOLIC.		
0 - 10 mm.Hg.	80 %	65 %
11 - 20 mm.Hg.	11	23
21 mm.Hg. and over	9%	12
Total	100	100
Mean Variation	7 mm.Hg.	11.7 mm.Hg.
Standard Deviation	± 7.35 mm.	±9.38 mm.
Range	0 - 35 mm.	0 - 40 mm.
Number of Cases	56	57

Systolic Pressure.

It is clear that the conditions prevailing under anaesthesia are better than those obtained by the use of sedatives. A t test shows that the mean variation of pressure under anaesthesia is significantly less (11 mmHg. against 18.5 mmHg.) ($P < 0.01$). A χ^2 test reveals that the percentage of patients whose pressure varies 10 mmHg. or less under anaesthesia (66%) is significantly less than the percentage under sedation (40%) ($P < 0.05$). An F test shows that the variability of the results under sedation is also significantly greater than under anaesthesia (F test) ($P < 0.05$).

Diastolic Pressure.

The conditions prevailing are again better under anaesthesia. The t test again shows that the mean variation of pressure under sedation is significantly greater (11.7 against 7 mm.) ($P < 0.01$) but while under sedation, the variability of results remains greater and the proportion of patients whose pressure varies 10 mmHg. or less is less, these differences are not however statistically significant.

As with the arterial oxygen saturation, many of the wide fluctuations in blood pressure noted occurred

at the beginning of the procedure and were associated with the stimulation of the surgical cut down. To determine the effect that such upsets are likely to have on the interpretation of the catheter findings one must consider what fluctuations take place while the catheter is in the heart. These are summarised in Table 16.

TABLE 16.

Blood pressure variations while catheter is in heart or great vessels.

Variation	Percentage of Cases showing Variation.	
	Anaesthesia	Sedation
SYSTOLIC:		
0 - 10 mmHg.	86 %	75 %
11 - 20 mmHg.	12	12
21 mmHg. and over	2	13
Total	100	100
Mean Variation	6 mm.Hg.	8 mm.Hg.
Standard deviation	± 6.58 mm.	±10.8 mm.
Range	0 - 35 mm.	0 - 55 mm.
Number of Cases	56	57
DIASTOLIC:		
0 - 10 mmHg.	93 %	80 %
11 - 20 mmHg.	7	14
21 mmHg. and over	0	6
Total	100	100
Mean variation	3.2 mmHg.	7 mmHg.
Standard deviation	± 5.34 mm.	± 8.44 mm.
Range	0 - 20 mm.	0 - 40 mm.
Number of Cases	56	57

Systolic Pressure.

Although the results remain better under anaesthesia, the mean variation being less (6 against 8) and the proportion of cases showing a variation of 10 mmHg. or less being greater (86 against 75%), these differences are no longer statistically significant. An F test shows that the variability of the methods remains significantly greater under sedation however ($P < 0.05$).

Diastolic Pressure.

As with the systolic pressure the results are better under anaesthesia but the only significant difference is in the greater variability of the sedative method (F test) ($P < 0.05$).

When the results obtained during the period of sampling or potential sampling are compared with those obtained during the entire procedure, both series show a significant improvement in the standard error of the means in respect of systolic and diastolic variations (t test) ($P \leq 0.01$).

HEART RATE.

The variations in the heart rate during the entire procedure are summarised in Table 17.

TABLE 17.

Fluctuations in heart rate during cardiac catheterisation (entire procedure).

Fluctuation	Percentage of Cases showing Fluctuation.	
	Anaesthesia	Sedation
Nil to 10 per minute	36 %	32 %
11 to 20 per minute	43	44
21 to 30 per minute	12	17
31 and over	9	7
Total	100	100
Mean variation	17 per minute	15.5 per minute
Standard deviation	±11.18 per min.	± 10 per minute
Range	0 - 50	0 - 45
Number of Cases	56	57

It is obvious that there is no significant difference between the series so far as changes in cardiac rate are concerned. The means are almost identical (17 and 15.5), the variability of the procedures is very similar and the proportion of cases in each of the divisions of the table is again almost identical.

Even when the changes produced at the beginning of the procedure are excluded and only the variations taking place during the potential sampling period are considered the results are identical. These are summarised in Table 18.

TABLE 18.

Fluctuations in heart rate during period when caridac catheter is in heart.

Fluctuation	Percentage of Cases showing Fluctuation	
	Anaesthesia	Sedation
Nil to 10 per minute	64 %	65 %
11 to 20 per minute	24	25
21 to 30 per minute	5	8
31 and over	7	2
Total	100	100
Mean variation	10.5 per min.	9.5 per min.
Standard deviation	±10.7 per min.	±8.88 per min.
Number of cases	56	57

In both series the results are significantly better when only the sampling period is considered, the means being lower (t test) and the proportion of cases varying 10 beats per minute or less being greater (χ^2) ($P < 0.01$).

Results in Patients Catheterised Twice.

In twelve patients cardiac catheterisation has been performed twice for various reasons and it is interesting to compare the results obtained. Three patients were examined under sedation on each occasion. In one the sedation twice produced tranquility and the results were identical at each catheterisation. The other two patients show a similar pattern one to the other. Each had one catheterisation in which he experienced pain and discomfort associated with technical difficulties in inserting the catheter. When this occurred the blood pressure, pulse rate and arterial oxygen saturation were higher than on the occasions when the examination passed off smoothly and the patient slept. The difference in blood pressure was of the order 20 mm.Hg.

One patient was examined under sedation and a repeat catheterisation was performed under basal narcosis, at the patient's request. Under basal narcosis the blood pressure was 20 mmHg. lower, the pulse rate 20/minute

slower and the arterial oxygen saturation 3% higher.

Of the eight cases examined twice under basal narcosis most showed only minor differences when extraneous factors are excluded. Thus one child (a probable Fallot's tetralogy) who was a little restless on the first occasion was given fifty per cent more pethidine on the second occasion and although better sedated showed a fall in blood pressure of 20 mmHg. and a drop in arterial oxygen saturation of 3%. However a child with aortic stenosis who had suffered a fall in blood pressure of 40 mmHg. after a dose of bromethol of 125 mgm/kilo showed a similar fall when the dose was reduced to 100 mgms/kilo. One child with a Fallot's Tetralogy, well sedated, had an arterial oxygen saturation almost 14% higher when she was sound asleep than when she was examined awake. Two children developed a supra ventricular tachycardia which confused the results and three showed no differences when examined on a subsequent occasion. It is impossible to draw firm conclusions from a small number of repeat examinations but the results in the sedated cases support the view that mild depression follows adequate sedation no matter how produced. In the anaesthetised patients it is of interest to note that the child with aortic stenosis and a fixed cardiac output suffered the same fall in blood pressure even when the dose was reduced by 20%.

PART THREE - Other Results.

PAIN DURING CARDIAC CATHETERISATION.

Many indeed most children when examined under basal narcosis move as the local anaesthetic is injected into the skin prior to the surgical cut down. Quite a number appeared to move at intervals during the catheterisation and to find the cause of this, cases being examined, sedated but awake, were closely observed. As a result it was found that 28 out of 59 patients complained of pain at some time during the course of the investigation. The site and incidence of these pains was as follows:-

Chest	10 cases	
Abdomen	3 cases	
Arm	15 cases	In two cases pain was experienced at more than one site.
Shoulder	2 cases	

Chest pain was felt while the catheter was either in the heart or entering it. It was, on occasion, associated with cardiac arrythmias. Abdominal pain occurred when the catheter went into the inferior ven a cava or liver. Pain in the arm sometimes occurred as the catheter passed up the vein but was most frequently felt at the site of cutdown when the effects of the local anaesthetic were wearing off after about one hour. Normally

the pain was moderately severe but transitory. In seven cases however it was so severe and persistent that the patients wept openly for some time, a fact which could have prejudiced the whole investigation. A further cause of discomfort is anoxia following the obstruction by the catheter of a stenosed pulmonary valve. Such obstruction was noted in 10 cases, causing restlessness in two.

As stated most of these episodes of pain were transitory and passed off as the catheter was moved. The pain in the arm associated with the wearing off of the local anaesthetic responded of course to a further injection of local anaesthetic.

DIFFICULTIES AND COMPLICATIONS OF ANAESTHESIA.

The technique of basal narcosis described is an exacting one. If good results are to be obtained it must be followed carefully and meticulously. It has been noticeable that most of the minor troubles have arisen when cases were prepared in wards not accustomed to doing so. The total number of cases in the anaesthetised series was 63. The notes of complications arising refer to this series.

Voiding of Bromethol occurred in six cases. Usually this was due to failure to strap the buttocks adequately

after administering the enema. A little thiopentone given on arrival in the X-Ray department sufficed to rectify the trouble but in one case this was associated with some struggling and cyanosis. Three cases showed a poor response to their bromethol although there was no history of voiding the drug. It may be that they were resistant to bromethol - a fact noted by Lucas (1960) in cyanotic patients.

Respiratory Depression and Cyanosis occurred on three occasions following the administration of supplementary thiopentone and on three occasions when the patients were momentarily restless. This demonstrates the extreme sensitivity of the children to small doses of thiopentone when they are already heavily although inadequately sedated.

Respiratory obstruction has been found in four cases due to large tonsils, adenoids or nasal congestion. In these cases an artificial airway was inserted and the trouble rectified. In all other cases it has been found unnecessary and indeed undesirable to insert an airway as the patient usually resents it.

Coughing occurred albeit for a very short time on three occasions in patients with chronic chest infections but settled before any treatment could be instituted. One patient, on two occasions, had an excess of secretions in mouth and lungs despite apparently adequate atropinisation.

Poor Veins necessitated the administration of nitrous oxide and oxygen on three occasions (twice in one child) to facilitate the surgical cut down.

Venospasm has only proved troublesome on one occasion and it settled without treatment.

Vomiting at the end of the catheterisation occurred on two occasions - the same frequency with which it occurred in the sedated patients. On both occasions the patients were awake and no harm was done.

One patient who came near to disaster after the administration of his bromethol is considered in detail in view of the inferences drawn from the findings.

A boy aged 5, weighing 2 stones 5 lbs. was re-admitted for repeat cardiac catheterisation, the catheter having failed to pass up the inferior vena cava on a previous occasion. The anaesthetic had been uneventful on the previous occasion apart from a little trouble with airway obstruction due to his tongue falling back. He had been in addition given a double dose of pethidine but apart from sleeping rather longer than some other children, suffered no ill effects.

On the second occasion the same premedication was ordered (avoiding the extra pethidine) and as he was 4 lbs heavier on this occasion the dose in terms of body weight was relatively smaller. After he had been given

his enema he was left propped up on his pillow in error and he again developed a mild respiratory obstruction due to his tongue falling back. Before this was noted and corrected he had become extremely cyanosed and the insertion of an oral airway and administration of 100% oxygen did little to correct this. His arterial oxygen saturation (ear oximeter) was about 60% and his blood pressure had fallen to 80/55 mmHg. In addition his pulse rate had risen to 170. He was laid flat and given oxygen but in view of the dubious value of vasopressors (Wood, 1956) in an undiagnosed lesion no further active treatment was given at the time. Over the next few hours his arterial oxygen saturation rose to 85 and the tachycardia subsided to 150. The blood pressure rose to its resting level (110/75) and then settled at 90 mmHg. He responded by movement to mildly painful stimuli. By evening his blood pressure and arterial oxygen saturation were within normal limits but tachycardia persisted. During that night it became obvious that cerebral damage had occurred, the child being spastic, unconscious in-continent and having a high pitched cry. He recovered gradually however and apart from slight weakness in one leg which persisted for several weeks made a complete recovery.

This child was a poor risk at best. He was grossly underweight and for years had been subject to bouts

of extreme cyanosis during which he lost consciousness for up to 15 minutes at a time. He became cyanosed on the least exertion such as crying and the diagnosis suggested was either a pulmonary atresia or Fallot's Tetralogy. The full cause of the incident is not quite clear. There can be no doubt that two gross errors took place at the induction of basal narcosis, namely the fact that he was allowed to remain propped up after his enema and also that his respiratory obstruction passed unnoticed, even for a short period. The cyanosis was not however due to the respiratory obstruction alone as the airway was quickly cleared and oxygen under pressure administered without any appreciable improvement in colour or oximetry. Nor indeed was the hypotension extreme. In any event the blood pressure rose from 65/50 to 110/70 without any appreciable improvement in the child's oxygen saturation. It seems unlikely therefore that the cyanosis was due to reversal of the shunt following systemic hypotension but that the severe tachycardia impaired the action of a damaged heart and that the resultant picture was largely caused by this. Schnabel (1954) has reported a similar occurrence following the development of a ventricular tachycardia unassociated with anaesthesia. The effect of supraventricular tachycardia on the oxygen saturation and blood pressure of a patient already severely crippled can

can be seen in Case 51 while the effect on a patient with a corrected pulmonary stenosis is well illustrated in Case 55.

It seems unlikely that the child was unduly sensitive to any of the drugs as he had already had the same premedication without ill effect.

While it would appear that the main trouble in this case was the development of a severe tachycardia to which such patients are in any case prone one cannot exclude the anaesthetic as a possible cause of this and certainly the cerebral signs which developed may in part have been caused by the respiratory obstruction in a head up position which occurred at the induction. In retrospect more active treatment with procaine amide to reduce the pulse rate and dehydration therapy with or without hypothermia might have lessened the cerebral anoxia, but these measures are not without risk in a child in such a precarious state.

DIFFICULTIES AND COMPLICATIONS OF SEDATION.

Inadequate sedation was the most frequent complication. As stated more than half of the cases complained of pain or discomfort of mild degree. However seven patients whose ages ranged from 12 to 23 were so upset that they wept openly for varying periods. Such conditions are obviously not conducive to a satisfactory cardiac catheterisation.

Restlessness apart from the cases mentioned occurred in 4 cases all of whom had been given pentobarbitone. In two further cases the restlessness proceeded to severe excitement. These cases had also received pentobarbitone as premedication. Pentobarbitone had been the standard premedication for patients examined under sedation but early on in this study it was abandoned.

Nausea or Vomiting occurred in four cases. Three of these had been given promethazine as part of the premedication but were nauseated in spite of it.

DURATION OF CATHETERISATION

The duration of the cardiac catheterisation is a factor of importance in determining the choice of agents to sedate or anaesthetise a patient. Many factors influence the time taken and the investigations have taken from 45 minutes to 3 hours (Mean 70 minutes plus or minus 25 minutes). There was no difference between the anaesthetised and sedated cases in respect of time taken to complete the investigation and these results are calculated from the combined series.

TIME LAPSE BETWEEN SURGICAL CUT DOWN AND FIRST SAMPLE.

As it is sometimes desirable to give nitrous oxide to facilitate the surgical cut down this interval is of importance. Mean times of 25 minutes (Anaesthetised) and 27 minutes (Sedated) elapsed before the first sample of

blood was withdrawn and in only two (both sedated cases) was blood drawn within ten minutes. Nitrous oxide would be excreted safely therefore in all cases.

SUPPLEMENTARY ANAESTHESIA.

Supplementary anaesthesia in the form of dilute thiopentone has been required as follows:-

Basal Narcotic	Supplemented	Unsupplemented
Paraldehyde (4 ccs./stone)	3/3 (100%)	0/3 (0%)
Bromethol 100 mgms/kilo	8/12 (66%)	4/12 (34%)
120 mgms/kilo	8/45 (18%)	37/45 (82%)

The difference in the percentages requiring supplements after the smaller dose of bromethol is significatnly greater than after the larger dose had been given (Using χ^2 $P < 0.01$).

CHAPTER EIGHT

DISCUSSION

CHAPTER EIGHT.

DISCUSSION.

EFFECTS OF ANAESTHESIA ON "RESTING STATE".

(1) Conditions at the beginning of cardiac catheterisation.

Most of the cases examined in the present series arrived at the beginning of the cardiac catheterisation in a condition closely resembling their resting state in the ward. Thus 83% of the patients had an arterial oxygen saturation which was raised more than 2%, so that they were in fact better oxygenated than when at rest in bed and the remaining 8% (3 cases) were all briefly depressed by a dose of thiopentone given immediately before readings were taken. The mean arterial pCO_2 of 44.5 mmHg. is at the upper limit of normal. Thus although respiratory minute volume is diminished, the blood gases were still essentially normal. Circulatory changes were also minimal. The blood pressure fell less than 25 mmHg. in 87% of cases and only 4% showed a rise in pressure. In 9% of cases the fall in blood pressure was greater than 25 mmHg. but the maximum drop was 40 mmHg. When one considers that many of the cases had fixed cardiac outputs, such as in mitral or aortic stenosis, and that all were asleep, this must be considered satisfactory. A similar fall in pressure (up to 25 mmHg.) accompanies normal sleep (Kleitman 1929) and moderate to severe hypotension accompanies preoperative sedation in about

10% of surgical and gynaecological patients (Norris, 1960).

The heart rate at this stage showed a less satisfactory picture however as one third of the anaesthetised patients showed a rise in pulse rate of more than 30 per minute. Much of this must be attributed to the premedicant drugs.

2) Conditions as the cardiac catheter entered the patient's heart.

There was a slight though insignificant improvement compared with the conditions prevailing at the beginning of the procedure. The effects of the thiopentone, given to stop movement, had now worn off and so also had the adverse changes due to the preparation of the patient for the examination to come. Thus the mean fall in arterial oxygen saturation was slightly less, the mean fall in systemic blood pressure slightly greater and the rise in pulse rate slightly less. None of the improvements were statistically significant.

EFFECTS OF SEDATION ON "RESTING STATE".

(1) Conditions at the beginning of cardiac catheterisation.

The sedative methods used caused little respiratory upset as 85% of cases at this stage had arterial oxygen saturations within ±2% of resting

values. However, 13% had their arterial oxygen saturation depressed more than 2% for no obvious reason. The mean carbon dioxide tension of 42.4 mmHg. is within normal limits.

The circulatory changes were however extremely important. While 51% of the patients showed the mild hypotension which accompanies sleep, 42% showed a rise in systolic pressure which in 25% was accompanied by a rise in diastolic pressure. It would thus appear that sedation to a point short of sleep may be accompanied by at least one of the manifestations of persistent anxiety and thus fail in its purpose in over 40% of cases. A drop in pressure greater than 25 mmHg. was found in 7% of patients examined. This latter is in accord with the findings mentioned above in regard to surgical and gynaecological patients.

As with the anaesthetised patients one third of the sedated cases showed a rise in heart rate of more than 30 beats per minute.

Many of the changes found were no doubt due to the arrival of the patient in the strange surroundings of the X-Ray department and anxiety as the preliminary procedures were carried out but even as he became used to the surroundings and the initial stimulation of the cut down wore off there was little sign of improvement.

(2) Conditions as the cardiac catheter entered the patient's heart.

The respiratory changes were again minimal and were almost identical to those prevailing at the beginning of the catheterisation. Unfortunately the changes in blood pressure were also almost identical so that 37% of patients still had an elevated systolic blood pressure and 23% also had an elevated diastolic pressure. This suggests that even at the point where the catheter had entered the patient's heart and therefore at a time when sampling and blood pressure recording were imminent almost 40% of cases still showed signs of persistent anxiety. At the other end of the scale some 10% showed fairly severe falls in blood pressure, one patient having developed a fall in pressure of 115 mmHg.

These conditions do not augur well for a satisfactory cardiac catheterisation.

Comparison of conditions produced by Anaesthesia and Sedation.

At the beginning of the cardiac catheterisation and even as the catheter enters the heart, there is no significant difference in the respiratory depression produced by the two methods. The higher mean carbon dioxide tension in the arterial blood is no more than would be expected from the fact that the anaesthetised

patients are all asleep. Similarly the greater scatter of arterial oxygen saturation values is due to the marked rise in saturation in the cyanosed cases under anaesthesia and to the use of thiopentone. This latter could probably have been avoided.

Changes in heart rate are again almost identical although the tachycardia in the anaesthetised cases is almost certainly due to the effects of the premedicant drugs while in the sedated cases it is again probably a manifestation of anxiety.

The changes in blood pressure produced by the methods are however very different. The mean fall in blood pressure after anaesthesia is significantly greater than after sedation. This is however largely due to the fact that a significantly higher proportion of the sedated cases have persistently raised blood pressures after sedation while a significantly higher proportion of the anaesthetised cases fall into the group showing the mild hypotension which normally accompanies sleep. In each case there is a small group (about 10%) who show a more severe fall in blood pressure. The results after sedation are very widely scattered about the mean and the variability of the method is significantly greater than after anaesthesia.

It is clear therefore that the use of basal narcosis will produce minimal depression of respiration and circulation in over 80% of cases and that these results are reproducible. Where however the patient is sedated to a point short of sleep the results will be almost unpredictable and in half the cases examined persistent anxiety will be found. Thus, while it has been the practice in some centres to condemn the use of basal narcosis to facilitate cardiac catheterisation because severe circulatory and respiratory depression are produced, the results of the present study do not substantiate this charge. As has been stated, no reports have been published giving measurements in support of the condemnation of basal narcosis and it appears that the rather unsavoury reputation which it has acquired may be based on false premises.

It cannot be disputed that the respiratory minute volume is decreased after the administration of Bromethol or other basal narcotic. Such a change however accompanies normal sleep without apparent ill effect. What is often forgotten is that in addition to this depression of minute volume, the patient's oxygen consumption and carbon dioxide production are depressed and the lack of change in the blood gases suggests that

these two mechanisms run closely parallel paths. The reports of cyanosis which are to be found in the early publications on basal narcosis may well have been due to respiratory obstruction caused by the tongue falling back. This should not be a problem when an anaesthetist is in attendance.

As regards depression of systemic blood pressure it has been shown that this seldom exceeds the hypotension which accompanies normal sleep and is presumably the price which must be paid for having the patient asleep. Occasional severe hypotension may occur but no more often than after routine sedation.

These changes appear less formidable in any event when they are compared with the changes which are found after the administration of doses of sedative drugs given merely to allay anxiety without producing sleep. Frequent monitoring of blood pressure in anaesthetic practice has shown how often unsuspected hypotension may occur in these circumstances.

Basal narcosis has in particular been condemned because of its potential dangers in patients with right to left shunts where it is feared that by causing systemic hypotension on a marked increase in the shunt would take place. Kepes et al. (1955) indeed abandoned the method for this reason. However these patients do not invariably react in this way and

many such cases in the anaesthetised group showed a rise in arterial oxygen saturation despite a fall in blood pressure. This may be due to the decreased oxygen consumption associated with their sleeping rate or it may be that the fall in systemic blood pressure is due to a fall in cardiac output caused by myocardial depression and affecting the right heart output at least to the same extent.

This improvement in the oxygen saturation of cyanosed children after heavy premedication is a well recognised clinical entity and has been commented on by Harmel and Lamont (1946), Smith (1950), Adelman et al. (1952) and Fieldman et al. (1955). Thus they, Rink et al. (1948), Potts (1949) and Smith (1952) specifically recommend the use of heavy premedication in the preparation of such cases.

Although severe drops in blood pressure can cause a precipitous fall in saturation, raising the systemic blood pressure with vasoconstrictors does not affect the oxygen saturation of patients with Fallot's Tetralogy (Burchell and Wood, 1949; Wood, 1956 and 1958). This is not perhaps surprising when one considers that vasoconstrictors have been shown to raise the pressure on both sides of the heart. (Cournand, 1947 and Besterman, 1951). It would seem tempting to suggest that many of

the factors causing minor falls in systemic pressure will also cause mild falls in right heart pressure.

It appears that so far as changes from the resting state are concerned, the patients examined under basal narcosis form the more satisfactory group, showing minimal depression which is reproducible with fair accuracy. The extent to which the steady state varies during the examination must now be considered.

FLUCTUATIONS IN THE "STEADY STATE" UNDER BASAL NARCOSIS.

(1) Fluctuations occurring during the entire procedure.

A high proportion of the cases (64%) showed a variation of only 2% in their arterial oxygen saturation during the examination. Only 12% of cases varied more than 4% and half of these longer fluctuations followed the administration of thiopentone at the time of the surgical cut down. Similarly 66% of the cases showed a variation of 10 mmHg. or less in their systolic blood pressure, only 12% varying 20 mmHg. or more. These larger variations were once more associated with the administration of thiopentone. The diastolic variations were smaller 80% varying 10 mmHg. or less. The fluctuations in heart rate were similar, 80% of all patients showing a variation of 20 beats or less per minute.

Thus despite the stimulation of the surgical cut down and the measures taken to facilitate it, most patients showed remarkably little upset during the cardiac catheterisation. No fluctuations likely to endanger life were found. To determine whether the fluctuations were likely to effect the results obtained by the physician, the changes which took place while the catheter was in the heart were studied.

(2) Fluctuations occurring while the catheter is in the patient's heart.

As one would expect, when the results obtained during the cut down were excluded, conditions were now more favourable. Only 3 out of 53 patients showed variations in arterial oxygen saturation of more than 4%. Of these, one had a marked right to left shunt and two followed the administration of thiopentone.

Blood pressure fluctuations were 10 mmHg. or less in 86% of cases and only one child, who woke up, had a swing in pressure greater than 20 mmHg. Changes in heart rate improved significantly but they were still not ideal, although 88% of cases showed variations of less than 20 beats per minute.

FLUCTUATIONS IN THE STEADY STATE DURING SEDATION.

(1) Fluctuations taking place during the entire procedure.

As in the anaesthetised cases two thirds of the patients have a variation in their arterial oxygen

saturation of less than 2%, while 12% showed a variation of more than 4%. The heart rate again shows almost identical fluctuations varying not more than 20 beats per minute in over 80% of cases.

The fluctuation in the systolic blood pressure is however of considerable significance. Thus only 40% of the sedated cases showed a variation of less than 10 mmHg., 31% showed a variation of 10-20 mmHg. and 29% fluctuated more than 25 mmHg. The maximum variation was 55 mmHg. None of the patients appeared to suffer any ill effects from this marked change in blood pressure but in an ill child it is obviously potentially dangerous. Almost all of these changes were associated with the cut down or the perception of pain during the early stages of the procedure. The likely effect of the variations on the results obtained from the catheterisation can be judged by considering only the variations which occur during the period that the catheter is in the patients heart.

(2) Fluctuations occurring while the catheter is in the patient's heart.

There are surprisingly few differences in the variations in arterial oxygen saturation compared with the variations during the entire procedure. Thus only 5% more patients are in the group varying 2% or less and the patients varying more than 4% are as before.

The fluctuations in heart rate as with the anaesthetised series have diminished considerably. This suggests that the variations are due to anxiety rather than to any stimulation from the procedure itself.

The blood pressure changes which were so marked during the entire procedure have diminished significantly and during the sampling period 75% of cases show a variation of 10 mmHg. or less. It appears that the variations are seldom likely to be so severe as to interfere with the results of the catheterisation.

COMPARISON OF THE FLUCTUATIONS FOUND UNDER ANAESTHESIA AND SEDATION.

As regards oxygen saturation variations, the patients fared better under basal narcosis during the later stages of the procedure, that is during the sampling period. The differences however were not statistically significant. When the entire procedure is considered there was no difference between the methods.

So far as the fluctuations in heart rate are concerned there is no difference between the methods either during the sampling period or indeed the entire procedure.

The fluctuations in blood pressure are however very different in each series. When the changes

taking place during the entire procedure are considered, significantly fewer patients varied 10 mm.Hg. or less under sedation while significantly more varied 25 mm.Hg. or more. The variability of the results was also significantly greater. During the sampling period the results remained better under basal narcosis but the differences were statistically significant, only in regard to the greater number of sedated patients who varied more than 20 mm.Hg. These results suggest that in the absence of contra-indications more reliable conditions and therefore more reliable data from the cardiac catheterisation are to be expected in patients examined under basal narcosis. During the preliminary part of the procedure as the patient is being prepared and the cut down is taking place the patient is under less strain and exposed to less risk than when he is only sedated to a point short of sleep.

CHANGES TAKING PLACE IN CYANOSED PATIENTS.

The patients in each series who come for examination with cyanotic heart disease present perhaps the most valuable information in this study. They are subjects who will reflect by a change in arterial oxygen saturation any adverse effects of th eir sedation or anaesthesia. Thus respiratory depression, circulatory depression or instability and excitement will all be detectable.

In the anaesthetised series results are available for eight such cases while there were six comparable cases among those sedated.

After the administration of Bromethol five out of the eight cases showed a rise in arterial oxygen saturation ranging from 1% to 8%, one showed no change, and two had a fall in saturation of 2% and 3% respectively. The mean change in the series was a rise of 1.9%.

After sedation two out of the six cases showed a rise in arterial oxygen saturation (3% and 2%) two showed no change and two showed falls (1% and 5%). The mean change was a fall in saturation of 0.33%. This appears to be a pointer to the changes produced by each of the two methods and would appear to refute the charge that dangerous, circulatory and respiratory depression follow the use of basal narcosis (Kepes et al. 1955).

Further confirmation of the value of basal narcosis, particularly in this type of patient, is found when the fluctuations taking place during the examination are studied. Under basal narcosis only two out of the eight patients showed a fluctuation in arterial oxygen saturation greater than 5% during the entire procedure

and when the sampling period alone is considered only one remained in this category. Under sedation however four out of six cases varied more than 5% during the entire procedure and remained in this category during the sampling period. The difference in these proportions is significant ($P < 0.01$).

These findings are of great importance as they indicate that changes in intra cardiac shunts are probably taking place in a high proportion of the cases examined under sedation. While they are probably not great enough to mask the diagnosis except in patients with pressures almost equal on both sides of the heart, they may make a mockery of attempts to measure shunt flow. This is of importance as some authorities place great stress on this value when assessing the need for operative closure of septal defects.

THE USE AND ABUSE OF SUPPLEMENTARY INJECTIONS OF THIOPENTONE.

It was considered that supplementary injections of thiopentone during cardiac catheterisation were undesirable on theoretical grounds, as periodic respiratory and circulatory depression might be produced. So it has proved in practice. Much of the depression found at the beginning of cardiac catheterisation

performed under basal narcosis early on in this work was due to the exhibition of thiopentone in an attempt to control the movement associated with injection of local analgesic prior to the surgical cut down. In the same way thiopentone caused variations in the steady state when given to combat restlessness during the procedure.

On the basis of the results found in this work it appears that much of the thiopentone given during cardiac catheterisation is in fact unnecessary. The use of the full doses of bromethol, recommended in the technique described earlier, have been shown to do away with the need for supplements of barbiturate and almost all the thiopentone given in this series of cases was used when, for one reason or other, a reduced dose of bromethol was given initially. There is seldom any need to cut down the dosage in this way as it does not appear to reduce the hypotension or respiratory depression which may result. Indeed by producing a restless patient it may cause much more harm than it avoids.

The two main indications in the literature for supplementary thiopentone are movement and restlessness. It fs for this reason that the sedated cases were asked to report any pain noted during the catheterisation as such pain in a lightly anaesthetised patient would cause movement and restlessness. The results show that pain

albeit transitory is not uncommon during the course of a cardiac catheterisation but on examination of the of the causes few of these would appear to be indications for supplementary thiopentone. On the contrary restlessness following obstruction of a stenosed pulmonary valve by the catheter or the discomfort associated with arrythmias or tachycardia following stimulation of the ventricular septum must be considered contr-indications. Any of the other pains assoociated with the passage of the catheter into or around the heart are so transitory that they will have passed off long before an injection could become effective.

It is of particular interest to note that many of the sedated patients complained of pain in the arm at the site of the surgical cut down after about one hour of the investigation had passed. This is almost certainly a cause of restlessness at this time and in the past thiopentone has been given to combat it. It appears probable that at this stage it is the lignocaine and not the bromethol whose effect is wearing off. This source of trouble has been eliminated by routinely reinfiltrating the area of the cut down after one hour.

Probably there are only two main indications for the administration of intravenous thiopentone. One is the appearance of restlessness at the beginning of

of catheterisation unassociated with any stimulation but often associated with a history that part of the enema has been voided. The slow administration of dilute thiopentone can very often produce tranquillity for the remainder of the procedure. Any depression which may follow will have passed off long before sampling takes place. The other indication is the occurrence of deepening cyanosis and restlessness during the procedure where the cause appears to be infundibular spasm, as described by Wood (1956). He found that this relaxed on deepening the level of anaesthesia with cyclopropane (the anaesthetic in use at the time) but on two occasions I have found that the patient improved quite dramatically after the exhibition of a small dose of thiopentone.

If one accepts the submissions regarding the indications for thiopentone, there remains the problem of dealing with movement of the patient as the local analgesic is injected prior to insertion of the cardiac catheter.

There are three ways of dealing with this situation. Of these the simplest is merely to restrain the patient's arm gently as the first wheal is raised and then to inject the remainder of the local analgesic through this. Unless in very cyanosed patients this method is both safe and effective. Where it is

considered that the amount of struggling which may ensue is dangerous the most effective method of facilitating the injection is to administer nitrous oxide and oxygen until the local analgesic is safely deposited. After adequate infiltration there is no further need for nitrous oxide and as has been shown, the ten minutes required for excretion of the nitrous oxide is usually long exceeded before sampling takes place. This technique has only occasionally been used where it was felt to be indicated but it is now routine practice at the Westminister Hospital. A third possible course in older children is to infiltrate the arm while the patient is still awake. This is very often tolerated by a child who could not face up to the full investigation in the conscious state.

If it appears necessary at any time during the catheterisation to administer thiopentone, the most suitable route must be chosen. Most authorities recommend that the obvious one - the catheter-be employed but some feel that it is better avoided. This is in part due to fear of contamination but also, in part, due to the dangers of administering drugs through the catheter when the tip is beyond the venae cavae. The latter fear is probably amply justified and on the only occasion on which I have in fact given thiopentone under such

circumstances (into a main pulmonary artery due to a misunderstanding of the position of the catheter tip) a bronchospasm ensued. There is however very little occasion to give thiopentone when the catheter tip is beyond the atrium as it is probable that any restlessness present is not due to inadequate depth of anaesthesia but to one of the other causes mentioned earlier. In any event it is better to withdraw the tip of the catheter or inject into a peripheral vein.

ANAESTHETIC AND SEDATIVE AGENTS.

Much of the evidence obtained from the two series suggests that choice of agents used to produce sedation or anaesthesia does not have a great bearing on the results obtained. What matters rather is the extent to which they supress anxiety. Bromethol was used therefore merely because, in adequate doses, it acted for a long enough time to allow completion of the examination without further supplements. In addition the rectal route of administration has in practice been found to be very satisfactory and is only rarely disturbing to the patient. Thus a child can be put to sleep in his own familiar surroundings by a nurse he knows well. (The anaesthetist should of course prepare the drugs and be immediately at hand.)

The drugs employed for sedation have included barbiturates, opiates and the so called "tranquillisers". The use of these drugs for cardiac catheterisation was paralleled by their use for routine premedication of other, mainly gynaecological, patients. The tranquillisers although in theory a promising group of drugs, have proved rather disappointing. Meprobamate, which was the drug principally used, was not found to be satisfactory unless given repeatedly in large doses - the method used in psychiatric practice. When it was thus used the tachycardia and hypotension were indistinguishable from that found with other agents. Two patients, both gynaecological, developed a severe skin rash after one single dose and were kept in hospital for a week longer than their original condition required. At this time Raymond et al. (1957) published a report showing that amylobarbitone was superior to a group of tranquillisers which were in turn indistinguishable from a placebo. I thus used amylobarbitone thereafter. There is of course a wide variation in the individual response to the drugs. This will almost certainly continue until a more satisfactory standard is found on which to calculate dosage. It is usual in adults

to give a fixed dose of a sedative to a wide range of patients, while in children or with more potent drugs the dose is related to body weight. This method has been shown by Anderson (1952) and others to be faulty as the nervous temperament of the patient is of great importance in this connection. There is however no sure way of measuring nervous temperament and short of sedating the patient to the point of sleep variable results with present methods appear inevitable. It may be, as is currently suggested, that the dose of a drug should be related to body surface area but this method has not been explored in the present work.

The only methods which have been described since this present study was commenced have not appeared to offer any advantage over the methods which were practised. Thus the administration of a mixture of chlorpromazine, and pethazine and pethidine as recommended by Smith et al. (1958) and Mitchell and Minor (1958) appeared to have no great benefit. The authors describe the patients' condition as "satisfactory in most cases" during catheterisation and state that many slept intermittently during the course of the procedure. Such intermittent sleep has been shown to be undesirable. Well over one third of their patients were restless during the cut down and many required intravenous supplements of

the drug mixture. While it is stated that no respiratory depression took place and that there was an inconstant drop in blood pressure, no figures or measurements are given in support of these claims. It is however well recognised that the intravenous administration of chlorpormazine and pethidine in anaesthetic or psychiatric practice is frequently associated with marked tachycardia and hypotension and Beard and Goodwin (1956) specifically condemn the use of chlorpromazine by any route in cardiac cases for this reason. Intramuscular injections of this mixture of drugs are usually painful but one report states that the whimpering of the child usually subsided within five minutes! Mitchell and Minor in their paper state that the recovery period extends over 10-12 hours, a period probably slightly longer than that found after bromethol. This is probably no disadvantage in either case and indeed Fieldman et al. (1955) consider that the prolonged sleep is beneficial.

As this 'lytic cocktail' had been found in general anaesthetic work to produce marked circulatory changes and was in any case painful to administer, uncertain in its action and had an equally long recovery period it seemed inferior to the bromethol technique. This view was also expressed by Adams and Parkhouse (1960)

who after personal experience with the phenothiazine mixture were unable to repeat the results claimed by Smith et al. and found the complications which one would anticipate - restlessness, hypotension and tachycardia.

Other reports deal with the use of volatile anaesthetic agents administered with air or nitrous oxide in oxygen. Keats et al. (1958) recommend the use of trichlorethylene in air following heavy sedation and provide figures allegedly supporting their claim to a steady state. They and Eggers et al. (1959), however, who give the trichlorethylene with nitrous oxide and oxygen both found marked tachypnoea with the technique and this and the high incidence of cardiac arrythmias caused Fleming (1955, 1957) and Duncalf and Thompson (1956) to abandon trichlorethylene for cardiac catheterisation.

The use of volatile agents necessitates endotracheal intubation if dead space is to be minimised and this in turn may cause coughing during the catheterisation. This difficulty is overcome in a compromise by Adams and Parkhouse (1960)

who, in a small number of cases, employed controlled respiration with endotracheal intubation, using the azeotropic mixture of halothane and ether. They claim that the method does not affect the intra-thoracic pressures and thus the flow of blood in the heart and great vessels. This latter seems unlikely in the extreme and indeed they quote a personal communication from Hodgson warning them of the dangers of controlled respiration in poor risk children with finely balanced shunts. Much more evidence will be needed to substantiate their rather remarkable claims and in the meantime spontaneous respiration on air seems preferable.

The objections to volatile agents on the grounds of their interference with Van Slyke analyses become less as more oxygen saturation readings are obtained with an oximeter or haemoreflector but it is still desirable to be able to check these instruments against gas analysis which remain the standard by which such instruments are judged.

There seems therefore to be no technique free from some disadvantages and basal narcosis with Bromethol is at least as satisfactory as most and better by far than many.

CARDIAC OUTPUT.

No attempt has been made to compare the cardiac output during basal narcosis or sedation with that found in the resting state. There are many reasons for this omission. Most of the methods used to study cardiac output are not suitable for application to young children with any expectation of obtaining basal readings. Indeed McMichael (1949) and Cosby et al. (1952) emphasise the difficulty of ever obtaining true "basal" flows. All that can be obtained is a reading at a given time and referable to the circumstances obtaining at that time. After heavy sedation or anaesthesia Winchell et al. (1951) and Quilligan et al. (1957) report a fall in cardiac output of 20-30%. Brotmacher and Fleming (1958) however did not find any material difference between the cardiac output of normal children without anaesthesia and a case examined under anaesthesia. This is in accord with the findings of Patrick and Faulconer (1952).

While Winchell et al. (1951) found considerable variation in the cardiac output during four hours of amylobarbitone narcosis, Etsten and Li (1955) found it to be fairly steady during thiopentone anaesthesia. The latter authors suggest that the fall in cardiac output is probably explained by the fall in

oxygen consumption. This would confirm the view of Graham (1958) that the fall in cardiac output after a period of thiopentone narcosis is a fall to and not of the basal value, a similar statement having been made regarding oxygen consumption during sleep by Benedict (1915).

It would seem probable that the readings obtained under basal narcosis will probably fall within normal limits for each patient while those under sedation will like all the other measurements show wide variation, depending on the effectiveness of the sedation produced.

MORTALITY AND MORBIDITY.

It is difficult if not impossible in many cases to distinguish the complications due to the anaesthetic or sedative method from those due to the investigation and indeed from those liable to develop spontaneously in the type of patient encountered in this type of work.

There have been no deaths in the present series either under anaesthesia or sedation although many of the patients examined were very poor risks. One, examined under sedation, and found to have a severe pulmonary fibrosis died a few days after the investigation.

Although these patients have all escaped serious harm I have heard through colleagues of deaths occurring under a variety of techniques all apparently correctly used. I know also of at least one fatality during a catheterisation performed under local anaesthesia with minimal sedation.

When one considers that these patients are liable to premature and sudden death and that the procedure by its very nature must always be liable to produce sudden and unexpected circulatory changes occasional deaths appear almost inevitable during the investigation no matter what sedative or anaesthetic is employed or however carefully it is administered.

Morbidity has also been slight in each series. The most serious complication has been the occurrence of supraventricular tachycardia which has necessitated the cancellation of the catheterisation on each occasion. This trouble has arisen with almost equal frequency in each series and is once more a complication which has been found in more than one of the patients at times other than the period of investigation. It is not therefore considered to be related to one or other technique.

These remarks apply only to the anaesthetic technique described when carried out under expert

supervision by all concerned and it must be obvious that any error in technique will be fraught with disaster. With this proviso it has not been shown that the techniques differ in mortality or morbidity.

THE FUTURE.

No attempt is made to suggest that the methods of sedation or anaesthesia employed are ideal and improvements could be made in both techniques.

In the basal narcosis the most obvious improvement would be to reduce the tachycardia which often accompanies the administration. It may be, as has been suggested by a colleague, that reduction of the dose of atropine would help in this way but so far I have been unable to prove or disprove this suggestion. A further possibility is that in place of pethidine a mixture of pethidine and levallorphan might be used to reduce the respiratory depression. So far experience with such mixtures suggests that not only the respiratory depression but also the analgesic effects of the pethidine are reduced. In view of the slight alteration in blood gas values from normal this line has not been pursued. It would be an advantage also to have a drug which produced adequately deep sleep for the same length of time as Bromethol but

which allowed for more rapid recovery after for example three hours. This would reduce the nursing care required in the ward. The alternative use of thiopentone has not proved satisfactory for two reasons. The first is that the period of sleep, deep enough to facilitate the catheterisation, is not sufficiently long and when supplementary intravenous thiopentone has been used the recovery times are virtually indistinguishable from those following Bromethol.

As regards sedation, the search continues for a drug which will produce relief from anxiety in most cases without the accompanying depression of respiration and circulation. As the changes produced are almost all found accompanying mental relaxation, it is felt that this may be a fruitless search.

It would also be of great value to know how much drug would produce the same degree of sedation in different patients. The only recognised method of doing this involves intravenous injection of the drug until speech slurring ensues or certain electroencephalographic changes appear. This method is not practical at present and in any case the effects of intravenous sedatives are usually fairly transitory and the need for furthersupplements arises. This has already been shown to be undesirable.

Monitoring of physiological variables could be improved and continuous recording of those variables measured would be an advantage. With the development of better apparatus both may be possible.

CHAPTER NINE.

CONCLUSIONS

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CONCLUSIONS.

After full consideration of the technical problems involved in cardiac catheterisation, it appears that the conditions most likely to yield reliable data to the physician are those found during normal sleep. During sleep basal values are obtained for many physiological variables and these values remain constant for a considerable period.

It should therefore be the aim of the anaesthetist to induce sleep with minimal disturbance to the patient, his circulation and respiration. This sleep should last throughout the period of the examination without the need for further recourse to drugs. It should be sufficiently deep to inhibit spontaneous movement but not of sufficient depth to mask the effects of undesirable stimuli such as the anoxia resulting from pulmonary valve obstruction. During the investigation the systemic blood pressure, arterial oxygen saturation, heart rate and electrocardiogram should be monitored, either continuously or at frequent intervals. This is particularly important while blood pressure and oxygen content readings are being obtained from the various sites within the heart.

The method of anaesthesia employed in the present study appears to meet most of the requirements set out above. After administration of the basal narcotic the patient was in a condition closely resembling deep sleep, apart from the increase in heart rate. The changes from the resting state were accurately predictable and could be reproduced in different patients and in the same patient on different occasions. This allows for greater accuracy in comparing the results one with another.

During the examination and in particular during the period when sampling was likely to take place, minimal variations occurred in the patient's resting state and such changes as did occur were unlikely to interfere with the interpretation of the data obtained during the examination. At no time did the patients' lives appear to be endangered by the technique per se.

Where the patient was merely given sedative drugs to allay his anxiety but not to produce sleep, the results were much less satisfactory. At the beginning of the procedure and even when sampling was due to commence, manifestations of anxiety were to be found in 40% of the cases examined. After

administration of the same dose of a drug to different patients and even to the same patient on subsequent occasions there were wide variations in the results produced, and the results were almost entirely unpredictable.

During the early stages of the examination the fluctuations in the patients' blood pressure and arterial oxygen saturation were so wide that in some life might have been endangered, although during the sampling period the fluctuations in the steady state were less marked and normally not sufficient to interfere with interpretation of the data obtained.

All the results obtained suggest that the conditions existing under basal narcosis are more stable and can be more accurately predicted beforehand than when the patients are sedated to a point short of sleep. This is in accord with the conclusions drawn earlier on in the study when the theoretical background to the evaluation of a suitable anaesthetic technique was discussed.

While many of the differences between the series, although favouring anaesthesia, are not statistically significant it is important to remember that the circumstances of the investigation were weighted in favour of sedation and against anaesthesia. Thus the anaesthetised patients were all in the younger age group and would have been unmanageable under light sedation while all the sedated

cases could easily - perhaps more easily - have been examined under anaesthesia. Many cases in the older age group who appeared apprehensive were examined under anaesthesia, some at their own request.

The benefits of having the patient asleep during the catheterisation are considerable. As has been shown the results obtained by the physician are likely to be more reliable and the patient will be in a true resting state. In addition and of great importance particularly in young children is the fact that the patients are spared what must be frightening experience to most of them. This investigation has shown that it is disturbing not only to the very young.

Providing a sleeping patient for cardiac catheterisation is a task which should never be undertaken lightly and calls for the greatest care both from the anaesthetist and from the nursing staff. The patient must be watched as carefully as if he was having a major operation and the price of respiratory obstruction or other complications must be fully appreciated. Where such skilled anaesthetic and nursing cover is not available the method is probably better avoided.

In prescribing the basal narcotic it is well to use adequate doses of the drugs initially as this significantly diminishes the need for intravenous supplements.

There must be a point where further increase in dosage will cause dangerous depression but this point does not appear to have been reached with the present technique. There is evidence, however, that it is being closely approached.

The methods of sedation employed have not been entirely satisfactory. It is true that the changes produced have not apparently interfered with the interpretation of the results obtained but many of the changes in blood pressure and arterial oxygen saturation were potentially dangerous and a method which allows almost half the patients to arrive at the point where pressure recording and sampling may take place with apparently uncontrolled anxiety, obviously leaves much to be desired.

This appears almost inevitable until a more satisfactory basis can be found on which to calculate the correct doses of the drugs to be given. At present it is clear that a given dose of sedative administered to apparently similar individuals may produce adequate sedation, little sedation at all or marked sedation. When adequate sedation is produced it is likely to be accompanied by all the changes found after basal narcosis.

These criticisms apply to all the drugs tested and there is no reason to expect any improvement

with other drugs. The fault lies in our inability to calculate accurately beforehand the dose of the drug which will provide adequate sedation unless we sedate the patient to the point of sleep.

It may well be that better methods of anaesthesia and sedation will be found to facilitate cardiac catheterisation in the future. It may also prove possible to measure other physiological parameters to obtain a broader view of the changes produced by these methods and of the fluctuations which take place during cardiac catheterisation.

At present however the method of basal narcosis employed seems to offer as much if not more than the available alternatives and the investigations performed have been all that it was possible to make without interfering with the investigation or neglecting the prime duty of the anaesthetist - to care for his unconscious patient.

It appears from this investigation that where adequate staff and facilities are available there is no need to withhold basal narcosis from patients undergoing cardiac catheterisation and, by making more accurate diagnosis possible, the extra labour involved may well save time, labour and indeed life at a later date.

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REFERENCES.

BIBLIOGRAPHY:

Adams, F.H. (1952) Pulmonary Hypertension in Children due to Congenital Heart Disease: J. Pediat. 40:42.

Adams, F.H. LaBree, J.W. Stauffer, H.M. (1950) Right heart catheterisation in acyanotic congenital heart disease: J-Lancet 70:159.

Adams, F.H. Lund, G.W. Disenhouse, R.B. (1954) Observations on physique and growth of children with congenital heart disease: J. Pediat. 44:674.

Adams, A.K. and Parkhouse, J. (1960) Anaesthesia for cardiac catheterisation in children: Brit. J. Anaesthesia 22:69.

Adelman, M.H. Rosenthal, A.I. Jacobson, E. Millis, A. (1952) Anaesthetic management in cardiac catheterisation: N.Y. St. J. Med. 52:1866.

Adriani, J. (1954) Pharmacology of Anaesthetic Drugs, Thomas, Springfield, Illinois.

Adriani, J. (1958) Personal Communication.

Altschule, M.D. (1951) Emotion and the circulation: Circulation 3:444.

Anschutz, W. Spechtk, K. Tiemann, F. (1930) Die Avertin narkose in der Chirurgie. Ergeb. chir. 23:406.

Astrup, P. (1956) A simple electrometric technique for the determination of carbon dioxide tension in blood and plasma total content of carbon dioxide in plasma and bicarbonate content in separated plasma at a fixed carbon dioxide content: J. clin. Lab. Invest. 8:33.

Ausherman, H.M. Nowill, W.K. and Stephen, C.R. (1956) Controlled analgesia with continuous drip meperidine J.A.M.A. 160:178.

Baldwin, E de F. Moore, L.V. Noble, R.B. (1946) Demonstration of ventricular septal defect by means of right heart catheterisation: Amer. Heart J. 32:152.

Banfield, W.G. Hackel, D.B. Goodale, W.T. (1950) Cardiac lesions following venous catheterisation: J. Lab. Clin. Med. 35:287.

Banghart, A.W. Lewis, J.A. (1948). Cardiac catheterisation in interauricular septal defects: Canad. Med. Ass. J. 58:605.

Barratt Boyes, B.G. and Wood, E.H. (1957). The oxygen saturation of blood in the venae cavae, right heart chambers and pulmonary vessels of healthy subjects: J. Lab. Clin. Med. 50:93.

Barratt Boyes, B.G. Wood, E.H. (1958). Cardiac output and related measurements in healthy subjects: J. Lab. Clin. Med. 1:72.

Barton, G.D. Wicks, W.R. Livingstone, H.M. (1946) Effects of pentothal alone or with oxygen or nitrous oxide and oxygen on arterial blood gases: Anaesthesiology 7:505.

Bates, D.V. (1957) Personal Communication.

Battro, A. Bidoggia, H. Pietrafesa, E.R. Labourt F.E. (1949). Intra cardiac blood pressure in human subjects and its relation to respiratory phases: Amer. Heart J. 37:11.

Beard, A.J.W. (1950) Cardiac Catheterisation: Anaesthesia 5:136.

Beard, A.J.W. (1954) Antihistamines in Anaesthesia: Proc. Roy. Soc. Med. 47:407.

Beard A.J.W. Goodwin, J.F. (1956) The assessment of the cardiac patient for anaesthesia: Brit. J. Anaesth. 28:557.

Benedict, F.G. (1915) J. biol. Chem. 20:288. The factors affecting metabolic rate.

Van Bergen, F.H. Weatherhead, S. Treloar, A.E. Dobkin, A.B. Buckley, J.J. (1954) Comparison of indirect and direct methods of measuring arterial blood pressure: Circulation 10:481.

Bernard, C. (1855) Lecons de Physiologie Experimentale Appliquee a la Medecine: Paris, Bailliere.

Bing, R.J. (1952) Catheterisation of heart: Advanc. Intern. Med. 5:59.

Bing, R.J. Vandam, L.D. Gray, F.D. (1947). Physiological studies in congenital heart disease: Bull Johns Hopk. Hosp. 80:107.

Bing, R.J. Vandam, L.D. Handelsman, J.C. Campbell, J.A. Spencer, R. Griswold, H.E. (1948). Physiological studies in congenital heart disease: Bull Johns Hopk. Hosp. 83:439.

Biork, G. Krook, H. (1951) Acta. cardiol. (Brux.) 6:101 Myocardiol injury at cardiac catheterisation.

Bizard, G. Vanlerenberghe, J. Robelet, A. (1952) Effects of pentothal on respiration: C.R. Soc. Biol. (Paris) 146:1121.

Bleichroeder, F. (1912) Intra arterial therapy: Berlin klin Wschr 49:1503.

Blomfield, J. (1929) Discussion on Avertin anaesthesia: Proc. roy. Soc. Med. 23:1-10.

Blomfield, J. and Shipway, F. (1929) Use of Avertin for anaesthesia: Lancet 516 - 546.

Blomfield, R.A. Lawson, H.D. Cournand, A. Breed, E.S. Richards, D.W. (1946) Recording of right heart pressures in normal and abnormal subjects: J. clin. Invest. 25:639.

Boere, L.A. (1949) Observation and interpretation of the percentage oxygen saturation during anaesthesia: Arch. Chir. neerl. 1:192.

Boeson, I. Lind, J. Merrild Hansen, B. Rosendal, T. Storm, O. Wegelius, C. (1956) The diagnosis of congenital heart disease in infants by catheterisation and selective angiocardiography: Brit. Heart J. 18:355.

Borden, C.W. Wilson, R.H. Ebert, R.V. Wells, H.S. (1950) Pulmonary hypertension in chronic emphysema: Amer. J. Med. 8:701.

Bordley, J. Connor, C.A.R. Hamilton, W.F. Kerr, W.J. Wiggers, C.J. (1951) Recommendations for the determination of human blood pressure recording by sphygmomanometer: Circulation 4:503.

Brannon, E.S. Weens, H.S. Warren, J.V. (1945) Atrial septal defect: Study of haemodynamics by technique of right heart catheterisation: Amer. J.med.Sci. 210:480.

Brecher, G.A. Opdyke, D.F. (1950) Amer. J. Physiol. 162:507. Effects of normal and abnormal respiration on the haemodynamics of experimental I.A.S.D.

Brock, R.C. (1956) Pulmonary Circulation and Respiratory Function: University of St. Andrews. 10.

Brotmacher, L. Fleming, P. (1958) Cardiac output and vascular pressures in 10 normal children and adolescents: Guys Hosp. Rep. 106:268.

Broustet, P. Castaing, R. Bricaud, H. Martin, C. Feillard, H. Lacour, R. (1954) Cardiac Catheterisation: J. Med Bordeaux 131:6.

Brown, B.C. England, N.J. Hauch, P.P. Lewis, J.A. (1949) Study of congenital heart disease by cardiac catheterisation: Canad. med. Ass. J. 60:50.

Brown, J.W. (1945) Diagnosis of congenital heart disease: Postgrad. med. J. 21:267.

Bruce, R.A. (1954) Cardiac dynamics and diagnostic role of cardiac catheterisation: North W. med. (Seattle) 53:577.

Burchell, H.B. (1948) Application of oximetry and cardiac catheterisation to diagnosis of congenital heart disease: J. Iowa St. med. Soc. 8:364.

Burchell, H.B. (1950) Introduction to clinical applications of oximetry: Proc. Mayo Clin. 25:377.

Burchell, H.B. (1954) Cardiac catheterisation in diagnosis of congenital heart disease: Minn. Med. 37:;20.

Burchell, H.B. Helmholtz, H.F. Wood, E.H. (1953) Overall experiences with cardiac catheterisation: Proc. Mayo Clin. 28:50.

Burchell, H.B. Swan, H.J. Wood, E.H. (1953) Demonstration of differential effects on pulmonary and systemic arterial pressure by variation in oxygen control of inspired air: Circulation 8:681.

Burchell, H.B. Taylor, B.E. Knutson, J.R.B. Wood, E.H. (1950) Circulatory adjustments to hypoxoemia in cyanotic congenital heart disease: Circulation 1:404.

Burchell, H.B. Wood, E.H. (1949) Reproducibility of values for oxygen saturation of arterial blood in patients with congenital cardiac disease: J. appl. Physiol. 1:560.

Burchell, H.B. Wood, E.H. (1950) Technique and applications of cardiac catheterisation: Proc. Mayo Clin. 25:41.

Burchell, H.B. Wood, E.H. (1951) Congenital heart disease: Fed. Proc. 10:21.

Bustamente, R.A. Perez-Stable, E. Barrera, F. Casas, R. Beltran, O.S. Boudet, L. Edelstein, J. (1952) Cardiac catheterisation: Rev. cubana Cardiol. 13:1.

Campbell, M. (1954) Simple pulmonary stenosis: Pulmonary valvulor stenosis with a closed ventriculor septum: Brit. Heart J. 16:273.

Carnegie, D.M. (1951) Angiocardiography in congenital heart disease: Brit. med. J. 1:1230.

Carnegie, D.M. (1953) Anaesthesia for cardiac catheterisation: Proc. roy. Soc. Med. 46:423.

Castellanos, A. Pereiras, R. Garcia, A. (1938) Angiocardiography in the infant: Presse Med. 46:1474.

Cernuschi, G. (1950) Diagnosis of congenital heart disease: Clin. Pediat. 32:273.

Chazan, M. Harris, T. O'Neill, D. Campbell, M. (1951) Intellectual and emotional development of children with congenital heart disease: Guy's Hosp. Rep. 100:331.

Clark, H.V. (1957) Music, Mobiles, and Cardiac Catheterisation: Amer. J. Nurs. 57:1026.

Comroe, J.H. Bothelo, S. (1947) Unreliability of cyanosis in recognition of arterial anoxaemia: Amer. J. med. Sci. 214:1.

Cosby, R.S. Griffith, G.C. Levinson, D.C. Oblath, R.W. Zinn, W.J. Dimitroff, S.P. Herman, L.M. Reynolds, T.B. (1952) Physiology of the lesser circulation: Med. Clin. N. Amer. 36:1035.

Cosby R.S. Griffith, G.C. Zinn, W.J. Levinson, D.C. Dimitroff, S.P. Oblath, R.W. Jacobson, G. (1953) Cardiac catheterisation in I.A.S.D. Amer. J. Med. 14:4.

Cournand, A. (1945) Measurement of cardiac output in man using right heart catheterisation: Fed. Proc. 4:207.

Cournand, A. (1947) Recent observations on dynamics of pulmonary circulation: Bull. N.Y. Acad. Med. 23:27.

Cournand, A. (1950) Some aspects of pulmonary circulation: Circulation. 2:641.

Cournand, A. (1951) Newer methods of diagnosis in congenital heart disease: Bull. N.Y. Acad. Med. 27:277.

Cournand, A. Baldwin, J.S. Himmelstein, A. (1949) Cardiac Catheterisation in congenital heart disease, New York, Commonwealth Fund.

Cournand, A. Bing, R.J. Dexter, L. Dotter, C. Katz, L.N. Warren, J.V. Wood, E. (1953) Report of Committee on Cardiac Catheterisation and Angiocardiography of American Heart Association: Circulation 7:769.

Cournand, A. Bloomfield, R.A. Lauson, H.D. (1945) Double lumer catheter for intravenous and intracardiac blood sampling and pressure recording: Proc. Soc. exp. Biol. (N.Y.) 60:73.

Cournand, A. and Ranges, H.A. (1941) Catheterisation of the right auricle in man: Proc. Soc. exp. Biol. N.Y. 46:462.

Cournand, A. Riley, R.L. Breed, E.S. Baldwin, E. de F. Richards, D.W. (1945) Measurement of cardiac output in man using right heart catheterisation: J. clin. Invest. 24:106.

Crawford, J.H. (1950) Recent advances in diagnosis and treatment of congenital heart disease: Brooklyn Hosp. J. 8:14.

Deuchar, D.C. Knebel, R. (1952) Pulmonary and systemic circulation in congenital heart disease: Brit. Heart J. 14:225.

Dexter, L. (1950) Cardiac catheterisation in diagnosis of congenital heart disease: Bull. N.Y. Acad. Med. 26:93.

Dexter, L. Burwell, C.S. Haynes, F.W. Seibel, R.E. (1946) Venous catheterisation for diagnosis of congenital heart disease: Bull. New Engl. Med. Cent. 8:113.

Dickerson, R.B. (1954) Performance of angiocardiography and cardiac catheterisation as a combined procedure: Amer. Heart J. 47:252

Dobkin, A.B. Wyant, G.M. Aasheim, G.M. (1958) Antisiologogue drugs in man: Anaesthesia 13:63.

Dornette, W.H.L. Ott Theodore (1958) Radiation safety for the anaesthesiologist: Curr. Res. Anesth. 37:167.

Doust, J.W.L. Schneider, R.A. (1955) Studies on physiology of awareness: Canad. J. Psychol. 9:67.

Dow, J.W. Levine, H.D. Elkin, M. Haynes, F.W. Hellem, H.K. Whittenberger, J.W. Ferris, B.G. Goodale, W.T. Harvey, W.P. Eppinger, E.C. Dexter, L. (1950) Studies of congenital heart disease: Circulation 1:267.

Downing, D.F. (1953) Diagnosis in congenital heart disease: Dis. Chest 24 157.

Dressler, W. (1952) Effort syncope manifesting pulmonary hypertension: Amer. J. med. Sci. 223:131.

Doyle, J.T. Wilson, J.S. Warren, J.V. (1951) Pulmonary vascular response to hypoxia: Circulation 5:263.

Dressler, S.H. Slonim, N.B. Balchum, O.J. Bronfin, G.J. Ravin, A. (1952) Effect of breathing ozygen on pulmonary artery pressure: J. clin. Invest. 31:807.

Dripps, R.D. Comroe, J.H. (1947) Effects of inhalation of high and low oxygen concentrations on oximeter readings in normal man. Amer. J. Physiol. 149:277.

Dry, T.J. (1948) Atrial septal defects: Med. Clin. N. America 32:895.

Duncalf, D. and Thompson, P.W. (1956) Anaesthesia for Cardiovascular and Neurosurgical Radiological Investigations. Brit. J. Anaesth. 28:450.

Eckenhoff, J.E. Helrich, M. Rolph, W.D. (1957) The effects of promethazine upon respiration and circulation in man.

Edwards, J.E. (1954) Functional pathology of congenital heart disease: Pediat. Clin. N. Amer. 1:13.

Edwards, J.E. Helmholz, H.F. Dushane, J.W. Burchell, H.B. (1953) Pathologic study of hearts previously catheterised: Proc. Mayo Clin. 28:113.

Eggers, G.W.N. Stoeckle, H.G.E. Allen, C.R. (1959) General anaesthesia for cardiac catheterisation: Anesthesiology 20:817.

Elam, J.O. and Brown, E.S. (1956) / CO_2 homeostasis during anaesthesia: Anesthesiology 17:116.

Elam, J.O. Neville, J.F. Sleator, W. Elam, W.N. (1949) Sources of error in oximetry: Ann. Sur. 130:755.

Ellis, E.J. Essex, H.E. Edwards, J.E. (1950) Proc. Mayo Clin. 25:73. Lesions of heart in dogs following cardiac catheterisation.

Ellis, E.J. Gauer, O. Wood, E.H. (1950) Application of manometric sound for recording intracardiac pressures: Proc. Mayo Clin. 25:49.

Emslie-Smith, D. (1955) The intracardiac electrogram as an aid to cardiac catheterisation: Brit. Heart J. 17:219.

Episcopo, U. (1952) Arrythmias during cardiac catheterisation: Acta cardiol. (Brux.) 7:595.

Escher, D.J.W. Shapiro, J.H. Rubinstein, B.M. Hurwitt, E.S. Schwartz, S.P. (1958) Perforation of the heart during cardiac catheterisation and selective angiocardiography: Circulation 18:418.

Etsten, B. and Li, T.H. (1955) Haemodynamic changes during thiopental anaesthesia in humans: J. clin. Invest. 34:500.

Von Euler, U.S. Liljestrand, G. (1946) Observations on pulmonary artery pressure in the cat: Acta physiol. Scand. 12:301.

Ferer, M.I. Harvey, R.M. Werko, L. Dreesdale, D.T. Cournand, A. Richards, D.W. (1948) Some effects of quinidine sulphate on the heart and circulation: Amer. Heart J. 36:816.

Fieldman, E.J. Lundy, J.S. DuShane, J.W. Wood, E.H. (1955) Anaesthesia for children undergoing diagnostic cardiac catheterisation: Anesthesiology 16:868.

Fishman, A.P. McClement, J. Himmelstein, A. Cournand, A. (1952) J. clin. Invest. 31:770. Effects of acute anoxia on respiration and circulation.

Fleming, H.A. (1955) Correspondence: Lancet, 1:617.

Fleming, H.A. (1957) Cardiac arrest during trichlorethylene anaesthesia: Brit. Med. J. 1:1062.

Forssman, W. (1931) Ueber Kontrastdarstellung der Hohler des Lebeden Rechten Hertzen und der Lungenschlagaden: Munsch. med. Wschr. 78:489.

Forssman, W. (1929) (a) The sounding of the right heart: Klin. Wschr. 8:2085.

(b) Correspondence: Klin. Wschr. 8:2287.

Fowler, N.O. Mannix, E.P. Noble, W. (1957) Difficulties in interpretation of right heart catheterisation data: Amer. Heart J. 53:343.

Fowler, N.O. Westcott, R.N. Scott, R.C. (1951) Disturbances in cardiac mechanisms complicating cardiac catheterisation: Amer. Heart J. 42:652.

Fraser, R. Nordin, B.E.C. (1955) Lancet 1:532. Basal metabolic rate during sleep.

Geiger, A.J. Anderson, H.C. Winkler, A.W. Kaplan, H.S. (1946) Cardio-vascular catheterisation as an aid in diagnosis of abnormal cardio-vascular communications: Conn. med. J. 10:895.

Gibson, J.B. (1953) Anaesthesia for cardiac catheterisation: Anaesthesia 4:269.

Gibson, S. (1952) Diagnosis of congenital heart disease: Med. Clin. N. Amer. 36:37.

Gilmore, H.R. Hamilton, M. Kopelman, H. Sommer, L.S. (1954) The ear oximeter: its use clinically and in the determination of cardiac output.

Godfrey, L. Pond, H.S. Wood, F.C. (1948) Millikan oximeter in recognition and treatment of anoxaemia in man: Amer. J. med. Sci. 216:605.

Goldman, I.R. Blount, S.G. Friedlich, A.L. Bing, R.J. (1950) Electrocardiographic observations during cardiac catheterisation: Bull Johns Hopk. Hosp. 86:141.

Goldschmidt, E.F. Hunt, A.M. (1932) Amer. J. Surg. 15:1. Tribromethanol - Amylene Hydrate - report of 314 basal anaesthetics administered to 225 patients.

Goodwin, J.F. (1953) Fatality following cardiac catheterisation injury: Brit. Heart J. 15:330.

Goodwin, J.F. (1952) The practical value of cardiac catheterisation: Practitioner 169:40.

Gordh, T. Linderholm, H. Strom, G. (1956) A transient attack of ventricular fibrillation during right heart catheterisation: Anesthesiology 17:358.

Gorham, A.P. (1933) Variations in blood pressure and pulse rate during basal anaesthesia: Brit. J. Anesth. 11:12.

Graham, G.R. (1958) Paper read to Association of Anaesthetists Meeting at Southport.

Green, D.M. Johnson, A.D. Bridges, W.C. Lehmann,J.H. Michel, J. Gray, F. Field, E. (1950) Use of cardiac catheterisation in medical practice: North W. Med. (Seattle) 49:27.

Griswold, H.E. Bing, R.J. Handelsmann, J.C. Campbell, J.A. Lebrun, E. (1949) Physiological studies in congenital heart disease: Bull Johns Hopk. Hosp. 84:76.

Gross, H. Jezer, A. (1956) Treatment of Heart Disease Saunders London and Philadelphia.

Halmagyi, D. Felkai, B. Ivanyi, J. Zsoter, T. Tanyi, M. Szucs, Zs. (1953) The role of the nervous system in the maintenance of pulmonary arterial hypertension in heart failure: Brit. Heart J. 15:15.

Hamilton, W.F. Woodbury, R.A. Harper, H.T. (1936) Physiologic relationships between intra thoracic, intra-spirol and arterial pressures:J. Amer. med. Ass. 107:853.

Hamilton, W.F. Woodbury, R.A. Harper, H.T. (1944) Arterial cerebro spinal and venous pressures in man during cough and strain: Amer. J. physiol. 141:43.

Harmel, M.H. Lamont, A. (1946) Anaesthesia in surgical treatment of congenital pulmonary stenosis: Anesthesiology 7:477.

Harned, H.S. (1955) Cardiac catheterisation in diagnosis: Conn. State med. J. 19:745.

Harned, H.S. Lurie, P.R. Crothers, C.H. Wittemore, R. (1952) Use of whole blood oximeter during cardiac catheterisation: J. Lab. clin. Med. 40:445.

Harris, P. (1955) Patent ductus arteriosus with pulmonary hypertension: Brit. Heart J. 17:85.

Hayward, G.W. (1952) Anaesthesia and the cardiac patient: Anaesthesia 7:67.

Hayward, G.W. (1952) Clinical applications of cardiac catheterisation: Bull Ayer clin. Lab. 20:131.

Heard, K.M. (1933) Clinical experiences with Avertin anaesthesia: Canad. med. Ass. J. 28:151.

Hebert, J. Scebat, L. Lenegre, J. (1953) Incidents and accidents during right heart catheterisation: Arch. Mal. Coeur 46:325.

Hemingway, A. Taylor, C.B. (1944) Laboratory tests of oximeter with automatic compensation for vasomotor changes: J. Lab. clin. Med. 29:987.

Hernandez, F.A. Saslaw, M.S. (1953) Pyrogenic reaction during cardiac catheterisation: Amer. J. med. Sci. 225:626.

Hewer, C.L. (1953) Recent Advances in Anaesthesia, 7th Edition, Churchill, London.

Heymans, G. van den Heuvel (1951) Procaine amide and cardiac arrythmias during heart catheterisation: Acta cardiol. (Brux.) 6:53.

Hickam, J.B. (1949) Atrial septal defects: Amer. Heart J. 38:801.

Hills, T.H. Stanford, R.W. (1950) Problem of excessive radiation during routine investigations of heart: Brit. Heart J. 12:45.

Himmelstein, A. Cournand, A. (1952) Cardiac catheterisation - an evaluation: Amer. J. Med. 12:349.

Holling, H.E. & Zak, G.A. (1950) Cardiac catheterisation in diagnosis of congenital heart disease: Brit. Heart J. 12:153.

Hopkin, D.A.B. (1954) Action of chlorpromazine and promethazine: Brit. Med. J. 1:1036.

Houssay, H.E.J. Haynes, F.W. Dexter, L. (1952) Pulmonary infarction from cardiac catheterisation: Proc. Soc. exper. Biol. (N.Y.) 79:444.

Howarth, S. Lowe, J.B. (1953) The mechanism of effort syncope in primary pulmonary hypertension and cyanotic congenital heart disease: Brit. Heart J. 15:47.

Howarth, S. McMichael, J. Sharpey Schafer, E.P. (1947) Cardiac catheterisation in cases of A.S.D. primary pulmonary hypertension, Fallot's Tetralogy, pulmonary stenosis: Brit. Heart J. 9:292.

Howarth, S. and Owen, S.G. (1954) Actions of promethazine on heart and systemic pressure: Brit. Med. J. 2:1266.

Hultgren, H.N. Hackett, A.J. (1950) Determination of oxygen content of capillary blood in congenital heart disease: Pediatrics 6:93.

Hutcheon, D.E. (1953) Comparison of actions of phenothiazine drugs: J. Pharmacol. exp. Ther. 108:340.

Inglis, J.M. (1954) Anaesthesia for cardiac catheterisation in children: Anaesthesia 9:25.

Johansson, L. Malmstrong, G. Uggla, L.G. (1954) Intracardiac knotting of catheter in heart catheterisation: J. Thorac. Surg. 27:605.

Johnson, A.L. Wollin, D.G. Ross, J.B. (1947) Heart catheterisation in investigation of congenital heart disease: Canad. med. Ass. J. 56:249.

Jorgens, J. LaBree, J. Adams, F. Rigler, L.G. (1952) Radiological aspects of heart catheterisation: Amer. J. Boentgenol. 68: 610.

Keats, A.S. Telford, J. Kurosu, Y. Latson, J.R. (1958) Providing a steady state for cardiac catheterisation under anaesthesia. J. Amer. med. Ass. 166:215.

Keown, K.K. Fisher, S.M. Downing, D.F. Hitchcock, P. (1957) Anaesthesia for cardiac catheterisation in infants and children: Anesthesiology 18:270.

Kepes, E.R. Livingstone, M. Escher, D.J.W. (1955) Management of children during cardiac catheterisation with inhalational anaesthesia: Curr. Res. Anesth. 54:5:299.

Kirklin, J.W. Ellis, F.H. (1956) Surgical implications of left heart catheterisation: Proc. Mayo Clinic 31:144.

Kleitman, N. (1929) Sleep, Physiol. Rev. 9:624.

Klein, O. (1930) Measurement of circulatory minute volume by Fick Principle (using cardiac catheterisation: Munsch. med. Wschr. 31:1311.

Lancet (1956) Annotation 2:1252.

Landtman, B. (1950) Mechanically induced disturbances in heart action: Acta Paediat. (Basel) 39:1.

Lee, J.A. (1959) Synopsis of Anaesthesia, Wright, Bristol.

Lee, G. de J. Matthews, M.B. Sharpey Shafter E.P. (1954) Effect of Valsalva manoeuvre on systemic and pulmonary arterial pressure in man: Brit. Heart J. 16:311.

Lehmann, J.H. Johnson, A.D. Bridges, W.C. Michel, J. Freed, D.M. (1951) Cardiac catheterisation in diagnosis of congenital heart disease: North W. med. 50:170.

Levine, E.B. (1951) Congenital heart disease in children: Ann west Med. Surg. 5:1027.

Lewis, J.A. (1948) Cardiac catheterisation: Treat. Serv. Bull 3:35.

Light, G.A. Livingstone, J.M. Adams, W.E. (1950) Anaesthesia for operations on heart and great vessels: A.M.A. Arch. Surg. 60:42.

Light, G.A. Morch, E.T. Engel, R. Cunningham, J. (1957) Promethazine hydrochloride as an adjunct to anaesthesia: J. Amer. med Ass. 164:1648.

Lima, A.B. (1956) Cardiac catheterisation in congenital heart disease: Diseases of Chest 29:62.

Lindgren, I. (1948) Continuous measurement of arterial oxygen saturation in man: Cardiologia (Basel) 13:226.

Lingen, B. Van Whidborne, J. (1952) Oximetry in congenital heart disease: Circulation 6:740.

Livingstone, H.M. Adams, W.E. (1952) Oximetry during anaesthesia in patients with limited pulmonary reserve: Curr. Res. Anesth. 31:229.

Lucas, B.G.B. (1958) Personal communication.

Lucas, B.G.B. (1958) Anoxic states and their management: Butterworth, London. Modern Trends in Anaesthesia: p. 290-291.

Lucas, B.G.B. Short, D.S. (1952) Procaine amide in control of cardiac arrythmias. Brit. Heart J. 14:470

Lundy, J.S. (1958) Amnesia-Analgesia for Management of children too young to co-operate. J. Amer. med. Ass. 166:453.

McKim, L.H. Bourne, W. (1933) Use of Avertin in multiple dressings - effects on liver: Canad. med. Ass.J. 28:149.

McMichael, J. (1949) a. Catheterisation of the heart in congenital heart disease: Cardiologia 15:134.

McMichael, J. (1949) b. Observations on right ventricular pressure: Cardiologia (Basel) 15:182.

McMichael, J. Mouhsey, J.P.D. (1951) A complication following coronary sinus and cardiac vein catheterisation in man: Brit. Heart J. 13:397.

McMichael, J. Sharpey Schafer, E.P. (1944) Cardiac output in many by direct Fick method: Brit. Heart J. 6:33.

McQuiston, W.O. (1949) Anaesthetic problems in cardiac surgery in children: Anesthesiology 10:590.

Mannheimer, E. Landtman, B. Melin, K.A. (1951) Complications of cardiac catheterisation: Cardiologia (Basel) 19:337.

Marder, S.N. Seaman, W.B. Wilson, H.M. (1952) Pulmonary circulation in diagnosis of congenital heart disease: J. Thor. Surg. 25:305.

Michel, J. Johnson, A.D. Bridges, W.C. Lehamnn, J.H. Gray, F. Field, L. Grenan, D.M. (1950) Arrythmias during intracardiac catheterisation: Circulation 2:240.

Millar, E. Joan (1952) Present position of anaesthesia for thoracic surgery: Proc. roy. Soc. Med. 45:52.

Millikan, G.A. (1942) Rev. Sci. Instrum. 13:434. Oximeter: instrument for measuring continuously oxygen saturation of arterial blood in man.

Mitchell, F.N. Minor, G.R. (1958) An analgesic mixture for minor surgical procedures in young children. J. Pediat. 52:16.

Montgomery, G.E. Wood, E.H. Burchell, H.B. Dry, T.J. Parker, R.L. Helmholz, H.F. (1948) Continuous measurements of arterial oxygen saturations at rest and during exercise in congenital heart disease: Amer. Heart J. 36:668.

Morgan, E.H. and Burchell, H.B. (1950) Ventricular septal defect simulating patent ductus arteriosus.

Morrow, A.G. Sanders, R.J. Braunwald, E. (1958) The Nitrous Oxide Test. Circulation 17:284.

Morton, W.R.M. (1935) Effects of tribromethanol on electrical charges in the human heart.

Motley, H.L. Cournand, A. Werko, L. Himmelstein, A. Dresdale, D. (1947) Influence of short periods of acute anoxia on pulmonary artery pressures in man.

Mushin, W.W. and Rendell Baker, L. (1950) Intravenous pethidine in anaesthesia for thoracic operations: Brit. J. Anaesth. 22:235.

Nicloux, M. (1908) Les Anestesique Generaux: O.Doin, Paris.

Nightingale, J.A. Williams, B.L. (1955) Pulmonary artery thrombosis following cardiac catheterisation: Brit. Heart J. 17:113.

Norris, L.D. Stevens, A.H. (1936) Tribromethonal in combination with other general anaesthetics including some electrocardiographic studies: Curr. Res. Anesth. 15:268.

Norris, W. (1960) Unpublished observations.

Nunn, J.F. (1958) Paper delivered to Association of Anaesthetists Meeting, Southport.

Olney, M.B. (1949) Diagnosis and surgical treatment of tetralogies: Calif. Med. 70:453.

Opdyke, D.F. Brecher, G.A. (1950) Effect of normal and abnormal intrathoracic pressures on effective right and left heart pressures: Amer. J. Physiol. 160:556.

Orcutt, F.S. Waters, R.M. (1937) Method for determination of cyclopropane, ethylene and nitrous oxide in blood with Van Slyke - Neill manometer apparatus. J. biol. Chem. 117:509.

Ordway, N.K. (1955) J. med. Ass. Ga 44:60.
Diagnosis and management of congenital heart disease.

Ordway, N.K. Bagnetto, R.L. Hyman, A.L. Levy, L. (1950) Recent developments in dagnosis of congenital heart disease: New Orleans med. Surg. J. 103:1.

Parker, R.L. (1948) Pulmonary stenosis: tetralogy of Fallot: Med. Clin. N. Amer. 32:855.

Patrick, R.T. Faulconer, A. (1952) Respiratory studies during anaesthesia: Anesthesiology 13:252.

Paul, M.H. Rudolph, A.M. (1958) Pulmonary valve obstruction during cardiac catheterisation: Circulation 18:53.

Pavy, F.W. (1857) An experimental enquiry into the alleged sugar forming function of the liver: Proc. roy. Soc. B. 9:300.

Peel, A.A.F. Seple, T. Kelly, J.C. Blum, K. (1956) Anomolous venous drainage with death from cardiac catheterisation: Scot. Med. J. 1:83.

Pietrafesa, E. (1954) Cardiac catheterisation: technique and elements of judgement: Dia Med. 26:337.

Potts, W.J. (1949) Diagnosis and surgical treatment of congenital pulmonary stenosis: J. Iowa St. med. Soc. 39:187.

Preston, S.N. Ordway, N.K. (1948) Observations of arterial oxygen content in children during inhalation of air and 100 per cent oxygen: Amer. J. Physiol. 152:696.

Quastel, J.H. Wheatley, A.H.M. (1933) Narcosis and oxidations of brain: Proc. roy. Soc. B. 112:60.

Quilligan, E.J. Hendricks, C.H. Hingson, R. (1957) Cardiac output: The acute effects of various anaesthetic agents and techniques as measured by the pulse pressure method: Res. Anesth. 36:33.

Raymond, M.J. Lucas, C.J. Beesley, M.L. O'Connell, B.A. Roberts, J.A.F. (1957) A trial of five tranquillising drugs in psychoneuroses. Brit. med. J. 2:63.

Reed, C.I. Kleitman, N. (1926) Studies on physiology of sleep: effect of sleep on respiration: Amer. J. Physiol. 75:600.

Read, J.L. Bond, E.G. Porter, R.R. (1955) Hazard of unrecognised catheterisation of the coronary sinus: A.M.A. Arch. intern. Med. 96:176.

Rink, E.H. Helliwell, P.J. Hutton, A.M. (1948) Anaesthesia for operation relief of congenital pulmonary stenosis: Guy's Hosp. Rep. 97:48.

Rivier J-L Desbaillets, P. Baudraz, B. West, R. (1954) The dangers of catheterising the coronary sinus: Cardiologia (Basel) 24:101.

Robb, G.P. Steinberg, I. (1938) A practical method of angiocardiography: J. clin. Invest. 17:507.

Robin, E.D. Whaley, R.D. Crump, C.H. Travis, D.M. (1958) Alveolar gas tensions, pulmonary ventilation and blood pH during physiologic sleep in normal subjects: J. clin. Invest. 37:981.

Rossier, P.H. Maier, C. Volkmann, M. (1949) Critical study of the measurement of cardiac output and intracardiac shunts by the Fick method: Cardiologia (Basel) 15:147.

Rushmer, R.F. (1944) Circulatory collapse following mechanical stimulation of arteries: Amer. J. Physiol. 141:722.

Rutledge, D.I. (1949) Approach to diagnosis of common types of congenital heart disease: J. Amer. med. Ass. 141:1290.

Sancetta, S.M. (1951) Local procaine block to overcome venospasm during cardiac catheterisation: J. Lab. clin. Med. 38:638.

Sancetta, S.M. Hackel, D.B. Lynn, R.B. (1953) Sub endocardial trouma produced by right sided catheterisation of the heart in man: Amer. Heart J. 45:491.

Scebat, L. and Lenegre, J. (1949) Action of morphine on intracardiac pressures: Arch. Mal Coeur 42:1154.

Scherf, D. (1953) Treatment of cardiac arrythmias: Circulation 8:756.

Schnabel, T.G. (1954) Cardiac catheterisation in heart disease: Med. Clin. N. Amer. 38:1617.

Sekel, J.P. (1954) Further studies on oximetry: Amer. Heart J. 48:746.

Selzer, A. (1954) Pulmonary hypertension and its relation to congenital heart disease: Dis. Chest 25:253.

Shackman, R. Graber, G.I. Redwood, C. (1951) Oxygen consumption and anaesthesia: Clin. Sci. 10:219.

Shapiro, M.J. (1949) Diagnosis of congenital heart disease by ordinary methods: Radiology 53:469.

Shephard, R.J. (1954) a. Pulmonary arterial pressure in acyanotic congenital heart disease: Brit. Heart J. 16:361.

Shephard, R.J. (1954) b. The effect of carbon dioxide on the pulmonary circulation in congenital heart disease: Brit. Heart J. 16:451.

Shephard, R.J. (1955) Brit. Heart J. 17:153.
The resting hyperventilation of congenital heart disease:

Simon, G. (1949) Radiology in cardiac disease: Postgrad. med. J. 25:543.

Skaggs, R.H. and Chapman, D.W. (1952) Technique of cardiac catheterisation: Texas med. J. 48:685.

Smith, C. Rowe, R.D. Vlad, P. (1958) Sedation of children for cardiac catheterisation with an ataractic mixture: Can. Anesth. Soc. J. 5:35.

Smith, J.A. (1950) Anaesthesia for cardiac catheterisation in children: Brit. Med. J. 1:705.

Smith, R.M. (1952) Circulatory factors affecting anaesthesia in surgery for congenital heart disease: Anesthesiology 13:38.

Smith, W.W. Albert, R.E. Radar, B. (1951) Myocardial damage during cardiac catheterisation: Amer. Heart J. 42:661.

Southworth, J.L. McKusick, V.A. Peirce, E.C. Rawson, F.L. (1950) Ventricular fibrillation precipitated by cardiac catheterisation: J. Amer. med. Ass. 143:717.

Stead, E.A. Warren, J.V. Merril, A.J. Brannon, E.S. (1945) Cardiac output in male subjects as measured by cardiac catheterisation: J. clin. Invest. 24:326.

Steen, R.E. (1954) Team diagnosis of congenital heart disease: Irish J. med. Sci. Jan. 1954 p. 1.

Stern, T.N. Tacket, H.S. Zachary, E.G. (1952) Penetration into pericardial cavity during cardiac catheterisation: Amer. Heart J. 44:448.

Stroud, R.C. Rahn, H. (1953) Effect of O_2 and CO_2 tensions on resistance of pulmonary blood vessels in man: Amer. J. Physiol. 172:211.

Sussman, M.L. Gordon, A.J. Brahms, S.A. Schwartz, B.M. Grishman, A. Steinberg, M.F. King, F.H. (1951) Technique of cardiac catheterisation: J. Mt. Sinai Hosp. 17:272.

Swan, H.J.C. and Wood, E.H. (1953) Localisation of cardiac defects by dye dilution curves: Proc. Mayo Clin. 28:95.

Swerdlow, M. (1957) Anaesthesia 12:42. Respiratory effects of anaesthetics.

Tannenbaum, L. (1934) Avertin Anaesthesia - resume of 150 cases: Curr. Res. Anaesth. 13:15.

Tausig, H.B. (1947) Congenital Malformations of the heart, New York, Commonwealth Fund.

Tausig, H.B. (1952) Diagnosis and management of common malformations of the heart: Circulation 6:930.

Tenney, S.M. (1956) Anesthesiology, 17:82.

Van Slyke, D.D. and Neill, J.M. (1924) Determination of gases in blood and other solutions by vacuum extraction and manometric measurement: J. Biol. Chem. 61:523.

Warren, J.V. (1953) N. C. med. J. 14:574.

Weiss, W.A. McGee, J.P. (1956) Promethazine, an adjunct to preoperative medication: Ann. Surg. 144:861.

Westcott, R.N. Fowler, N.O. Scott, R.C. Hauen Stein, J.D. McGuire, J. (1951) Anoxia and human pulmonary artery resistance: J. clin. Invest. 30:957.

Whitelaw, D.M. (1948) Discussion of cardiac catheterisation in diagnosis of congenital heart disease: Bull Vancouver med. Assoc. 24:312.

Winchell, P. (1953) Infectious endocarditis as a result of cardiac catheterisation: New Eng. J. Med. 248:245.

Wittenborg, M.H. Neuhauser, E.B.D. (1955) Diagnostic roentgenology in congenital heart disease: Circulation 11:462.

Wood, E.H. (1949) J. appl. Physiol. 1:567.
Normal oxygen saturation of arterial blood during inhalation of air and oxygen:

Wood, E.H. (1950) Single scale absolute reading ear oximeter: Proc. Mayo Clin. 25:385.

Wood, E.H. Geraci, J.E. (1949) Photoelectric determination of arterial oxygen saturation in man: J. Lab. clin. Med. 34:387.

Wood, E.H. Leusen, I.R. Warner, H.P. Wright, J.L. (1954) Measurement of pressures in man by cardiac catheters: Circulation Res. 2:294.

Wood, P. (1950) Congenital heart disease: Brit. Med. J. 2:639.

Wood, P. (1952) Pulmonary hypertension: Brit. med. Bull 8:348

Wood, P. (1956) Pulmonary Circulation and Respiratory Function, University of St. Andrews 10.

Wood, P. (1957) Personal communication.

Wood, P. (1958) Symposium on Congenital Heart Disease: Brit. Heart J. 20.

Wright, S. (1953) Applied Physiology, 9th Edition: Oxford Medical Press, London.

Zak, G.A. (1949) Appendix on cardiac catheterisation: Brit. Heart J. 11 198.

Ziegler, L.H. Levine, B.S. (1925) Influence of emotional reactions on basal metabolism: Amer. J. med. Sci. 169:68.

Ziegler, R.F. (1954) Clinical cardiac catheterisation in infants and children: Paediat. Clin. N. Amer. 1:93.

Zinn, W.J. Cosby, R.S. Levinson, D.C. Miller, H. Dimitroff, S.P. Dramer, F.B. Griffith, G.C. (1952) Use of oral quinidine and procaine amide as premedicants for cardiac catheterisation: Amer. Heart J. 43:451.

ANAESTHESIA AND SEDATION

FOR

CARDIAC CATHETERISATION.

WALTER NORRIS

M.B., Ch.B., F.F.A.R.C.S.

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APPENDIX 'A'.

EAR OXIMETER.

APPENDIX 'A'.

THE EAR OXIMETER:

Theoretical Background.

The first practical ear oximeter was described by Millikan in 1942. In his article he outlines the theory on which the instrument is based.

The oxygen saturation of the blood is the ratio between the concentration of oxygaemoglobin and total haemoglobin. It has been shown that this can be accurately estimated by measurement of the light intensities transmitted by the ear at two selected wavelengths. At one of these in the infra red region of the spectrum the absorption coefficient for oxyhaemoglobin and reduced haemoglobin are the same and thus a measure of the total haemoglobin is obtained. At the other in the red region there is a maximum difference between the absorption coefficients and a measure of oxyhaemoglobin is obtained. The percentage desaturation is a linear function of the ratio of the logarithms of these intensities.

The ear oximeter consists essentially therefore of a light source, photoelectric cells with filters and an amplifier with one or more indicating devices. The original oximeter gave only relative readings and it was set to 97-100% while the subject breathed pure oxygen or standardised against a single blood sample analysed by the Van Slyke method. In 1949 however Wood and Geraci

described what is claimed to be the first oximeter giving absolute as opposed to comparative readings and it is this type of oximeter which has been developed subsequently in many centres the world over.

Accuracy of Oximeters.

It is essential to understand the possible degree of accuracy and limitations of use of oximeters. In attempting to obtain absolute values of arterial oxygen saturation under ideal circumstances blood gas analysis remains the yard stick by which all other methods are judged. Millikan (1942) claimed that his instrument was accurate to 5% at the top of the scale but only to 8% at the lower end. Lindgren (1948) claims that the single scale model is accurate to 1.7% - 2.3% in the higher ranges falling off to 9% in the lower ranges. Many other reports also emphasise what is now a recognised characteristic of oximeters, namely good correlation with gas analysis at high levels with diminishing accuracy as the saturation falls. Hemingway and Taylor (1944) in defence of oximeters point out, however, that technical difficulties with gas analysis may lead to a 2% error and, in addition, the time lag in taking off a blood sample makes it very difficult to compare simultaneous samples.

It is however not with the absolute accuracy of the oximeter that we are primarily concerned but with its accuracy in showing changes in oxygen saturation in any individual. On this point all authors, including those quoted, above areagreed that the relative accuracy is much greater than the absolute. This is understandable when it is remembered that the calculated values are linear and that any initial inaccuracy is reflected equally throughout subsequent readings. Used in this way Barratt Boyes and Wood (1957) claim accuracy to 1% of the correct value.

Sources of Error in Oximetry.

The accuracy of the oximeter as of any scientific instrument depends on it being carefully and correctly used. In particular certain obvious sources of error must be avoided. In order of occurrence during the technique they are as follows:-

1) Failure to allow the amplifier to become thermally stable. This process takes about ten minutes and if the oximeter is used within ten minutes of being switched on false results may be obtained.

2) Vasoconstriction. This is perhaps the greatest difficulty encountered in the use

of an oximeter. As stated the ear in a state of vasodilation contains blood skin to arterial blood in oxygen content. It should therefore be obvious that vasodilation must be maintained before worthwhile results can be expected. In particular when using the Mark 1 model, where the cells were balanced to read 97% through the bloodless ear, it is natural that in the presence of severe vasoconstriction the reading would be high no matter what the oxygen saturation might be. Failure to appreciate this fact led many people to condemn the instrument in the early days. Cardiac catheterisation does not however normally produce the vasomotor changes seen for example during anaesthesia and surgery and many of the patients under sedation or basal narcosis are in a state of vasodilation when they arrive for examination.

Active steps to promote and maintain vasodilation are however taken and they include the application to the ear of a histamine cream or xylol and switching on the earpiece

light for some minutes before taking a reading. These steps are normally adequate to ensure that vasodilation will be attained and maintained (Millikan, 1942; Elam et al. 1949; Gilmore et al. 1954).

3) Earpiece cleanliness. Any dirt or clouding of the earpiece will prevent light from passing. The commonest source of this is the cream used to promote vasodilation.

4) Poor positioning of the earpiece so that the cells are not covered with tissue will give false readings. So will the inclusion of fibrous or cartilaginous tissue between the light and the cells.

5) Extraneous light must be kept from striking the cells. This is normally done by placing the light on the inside of the ear lobe. During catheterisation there is little difficulty as the fluoroscopic screen acts as a shield.

6) Movement of the earpiece will alter the amount of tissue between light and cells resulting in inaccurate readings. This difficulty can be obviated by standardising the instrument before taking each reading. The process takes about thirty seconds and adds greatly to the value of the results.

THE "STANCO" OXIMETER.

The instruments used in the present investigation have been Stanco oximeters which were available in the Royal Infirmary in 1955. The makers claim that their oximeter is accurate to ±2% of absolute values, with greater accuracy for relative readings. Before embarking on this project I contacted at the suggestion of the makers - Stanley Cox of London - two prominent physicians who had extensive clinical experience of this type of oximeter. Dr. Paul Wood, President of the Institute of Cardiology, stated that provided the cells of the oximeter matched it would be suitable for the purpose intended and Dr. D.V. Bates, then of St. Bartholomew's Hospital, opined that if it was frequently checked it should be accurate. The instrument in question was replaced as soon as the cells failed to balance and it and its successor have been frequently checked.

Two types of oximeter have been used. Initially the direct reading model was used. Here the two photoelectric cells were matched and balanced to read 97% through the bloodless ear. As time passed the quality of selenium available fell, so that it was difficult to provide matching cells. A later model was therefore introduced in which the cells provide

independent readings which are then interpolated via a nomogram. This Mark 2 model was used immediately the original cells showed evidence of deterioration. Approximately half of the series to be described has been examined with each instrument.

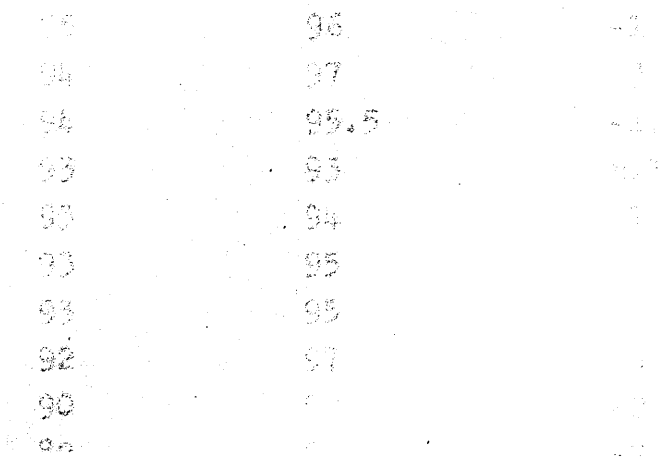
Sensitivity and Accuracy.

The sensitivity of the oximeter can be easily checked. If the earpiece is attached and the oximeter set up as described the percentage saturation reading can be quickly ascertained. If then the subject hyperventilates or breathes 100% oxygen for a few breaths the saturation will normally rise about 2%. If he holds his breath for over 40 seconds the saturation can be dropped to around 85%, and it will rapidly return to normal after a few breaths of air. This test I have carried out on myself repeatedly and have demonstrated it successfully to the most sceptical colleagues. In addition when nitrous oxide and oxygen have been administered to a patient the needle rapidly rises or falls in response to an alteration in the oxygen percentage administered.

Although the absolute accuracy of readings was of secondary importance in the investigation herein

described it was considered important to know how accurate the oximeter with regard to absolute readings so that one could perhaps take the readings in place of a direct blood analysis where this was not possible or desirable. It would also establish confidence in the use of the oximeter in other cases where it was considered to be valuable.

It will be seen that indeed the oximeter does give fairly accurate absolute values and it would certainly seem that the Stanco oximeter used, is as accurate as "the occasional use of the Van Slyke apparatus" - the claim made for their oximeters by Godfrey et al. (1948) and Gilmore et al. (1954).



Comparison of arterial oxygen saturation values obtained by using Stanco Oximeter against (1)Van Slyke (2) Haemoreflector

Mark 1 Oximeter			Mark 2 Oximeter		
Oximeter	Van Slyke	Deviation	Oximeter	Van Slyke	Deviation
96 %	96 %	-0 %	97 %	96 %	+1 %
94	95	-1	95	97	-2
94	91	+3	95	92.5	+2.5
93	89	+4	93	90.5	+2.5
92	90	+2	93	93	nil
88	86	+2	92	90.5	+1.5
81	76	+5	92	90	+2

Mark 2 Oximeter		
Oximeter	Haemoreflector	Deviation
96 %	96 %	nil %
95	96	-1
94	97	-3
94	95.5	-1.5
93	93	nil
93	94	-1
93	95	-2
93	95	-2
92	97	-5
90	92	-2
82	75	+7

APPENDIX 'B'.

CASE REPORT.

This case report is included as an example of what is considered to be faulty reasoning. The authors consider that their patient died from circulatory depression following administration of heavy barbiturate sedation. The figures which they quote indicate that minimal depression occurred during the catheterisation itself and it seems extremely unlikely that depression at the time of death would be as great as that immediately following the original administration of drugs.

It would appear probable that this patient's death was associated with the restlessness reported and probably was unrelated to the method of sedation employed.

APPENDIX 'B'

CASE REPORT (KEPES ET AL. 1955).

"A 9 year old white female was admitted for cardiac catheterisation.

History - The patient a premature baby weighing 3½ lbs. at birth, was kept in an incubator until she weighed 6 lbs. At 14 months of age she developed broncho-pneumonia and was hospitalised for the following 4 years. At 5 years of age she was sent home with no restriction in her activity. Occasionally she would develop cyanosis of lips and fingers when exerting herself. Otherwise, she was well.

Physical Examination - Significant findings were confined to the heart. Blood pressure was 105/60 mm.Hg. The point of maximum impulse was in the 5th intercostal space 2 cm. to the left of the midclavicular line. There was a harsh, loud, diastolic murmur heard best at the 2nd intercostal space to the left of the sternum, accompanied by a very harsh thrill. The murmur was transmitted to the mid-back region where it was poorly heard. There was no cyanosis or clubbing of fingers or toes.

Course - On June 19, 1952, the patient was given sedation consisting of oral seconal 100 mg. (gr. 1½), atropine .00027 gms. (gr. 1/250) intramuscularly. The anaesthetic consisted of sodium pentothal 20 mg./lb. of body weight

administered rectally in a 5 per cent solution using a total of 1 gm. of the drug.

Twice during the catheterisation the patient became cyanotic. Considerable EKG variability also was noted during the procedure. The pulse rate during the four hours of anaesthesia was around 100 beats per minute, and the blood pressure 100/60 mm.Hg. The pulmonary artery pressure and the right ventricular pressure were high, 95-105/62-75 and 80-95/8-10 mm.Hg. The diagnosis during the catheterisation was a probable Eisenmenger's syndrome. The left pulmonary artery was reached but no shunt, indicating a patient ductus was found at the time.

Following the catheterisation, the patient was seen several times in the recovery room. She had not fully reacted from the anaesthesia, but was moving about and reacted to the airway. About three and a half hours after the catheterisation, the child developed cyanosis and tachycardia. The administration of 100 per cent oxygen did not improve the colour. An endotracheal tube was inserted and respiration was assisted with 100 percent oxygen. Despite this, the cyanosis deepened and the heart rapidly slowed and stopped.

Autopsy showed an interventricular septal defect with dextraposition of aorta, dilation and hypertrophy of cardiac ventricles, dilation of pulmonary artery, patent ductus arteriosus, and pulmonary arteriosclerosis.

In this non-cyanotic patient with high pressures in the right side of the heart a reversal of her usual left to right shunt must have occurred through the interventricular septal defect and/or the patent ductus arteriosus when the pressure dropped in the peripheral circulation. This pressure drop can be explained by the large amount of barbiturates used. As a result of this reversal of flow most of the blood shunted to the left, without ever circulating through the lungs for oxygenation. Obviously, methods of resuscitation directed toward oxygenation alone were bound to be futile."

APPENDIX 'C'.

EXPANDED TABLES OF RESULTS.

EXPANDED TABLES OF RESULTS.

From these expanded tables of results, as from the chapter in the main body of the thesis, changes from the resting state and fluctuations during the procedure due to extraneous causes have been excluded. Such extraneous causes have been the development of supraventricular tachycardia and obstruction of a stenosed pulmonary valve by the catheter.

Under the heading of "no reading" I have included in column 1 those cases where the catheter has failed to enter the heart and therefore no reading is available at this time.

The changes following the extraneous causes mentioned in the first paragraph and the readings excluded in the second paragraph are of course available in the individual case records.

Changes in arterial oxygen saturation from resting level to the beginning of catheterisation.

Change	Number of Cases	
	Anaesthesia	Sedation
≥ +6 %	0	0
+5	0	0
+4	4	0
+3	1	1
+2	3	2
+1	6	4
0	20	27
-1	10	8
-2	5	6
-3	1	6
-4	0	0
-5	0	1
≤ -6	3	0
No reading	10	4
Total	63	59

Changes in arterial oxygen saturation from resting level to catheter entering heart.

Change	Number of Cases	
	Anaesthesia	Sedation
≥ +6 %	2	0
+5	0	0
+4	2	1
+3	1	0
+2	3	5
+1	3	1
0	21	20
-1	11	14
-2	8	7
-3	0	4
-4	1	0
-5	0	2
≤ -6	1	1
No reading	10	4
Total	63	59

Blood pressure changes from resting level to beginning of catheterisation.

Systolic Pressure.

Change	Number of Cases	
	Anaesthesia	Sedation
≥ +30 mm Hg.	0	3
+25	0	2
+20	0	2
+15	0	7
+10	1	8
+5	1	2
No change	9	12
-5	7	5
-10	17	5
-15	9	3
-20	5	2
-25	2	2
-30	2	0
-35	2	1
≤ -40	1	3
No reading	7	2
Total	63	59

Blood Pressure changes from resting level to beginning of catheterisation.

Diastolic Pressure.

Change	Number of Cases	
	Anaesthesia	Sedation
+30 mm. Hg	0	0
+25	0	0
+20	1	5
+15	0	0
+10	1	5
+5	1	4
No change	13	18
-5	9	7
-10	15	10
-15	5	2
-20	5	2
-25	2	1
-30	3	1
-35	0	1
-40	0	1
No reading	8	2
Total	63	59

Blood pressure changes from resting level to catheter entering heart.

Systolic Pressure.

Change	Number of Cases	
	Anaesthesia	Sedation
≥ +30 m.m.Hg.	0	0
+25	0	2
+20	0	1
+15	0	4
+10	0	8
+5	1	6
No change	8	8
-5	4	7
-10	13	7
-15	16	6
-20	4	2
-25	5	1
-30	3	1
-35	0	3
≤ -40	2	1
No reading	7	2
Total	63	59

Blood pressure changes from resting level to catheter entering heart.

Diastolic Pressure.

Change	Number of Cases	
	Anaesthesia	Sedation
≥ +30 m.m. Hg.	0	0
+25	1	0
+20	0	5
+15	0	0
+10	3	3
+5	0	5
No change	10	19
-5	8	5
-10	16	9
-15	5	6
-20	7	3
-25	2	1
-30	3	0
-35	0	0
≤ -40	0	1
No reading	8	2
Total	63	59

Change in heart rate from resting level to beginning of catheterisation.

Change	Number of Cases	
	Anaesthesia	Sedation
≥ -14 beats/min.	5	0
-13 to -10	0	1
-9 to -6	0	4
-5 to -1	1	6
No change	1	9
+1 to +4	9	5
+5 to +8	2	3
+9 to +12	7	4
+13 to +16	5	3
+17 to +20	6	4
+21 to + 24	4	6
+25 to +28	6	1
+29 to +32	6	2
+33 to +36	2	1
+37 to +40	0	1
≥ +42	2	7
No reading	7	2
Total	63	59

Changes in heart rate from resting level to catheter entering heart.

Change	Number of Cases	
	Anaesthesia	Sedation
≤ -14 beats/min.	2	3
-13 to -10	1	2
-9 to -6	0	3
-5 to -1	3	4
No change	4	4
+1 to +4	9	8
+5 to +8	4	3
+9 to +12	6	6
+13 to +16	3	5
+17 to +20	9	4
+21 to +24	4	4
+25 to +28	4	1
+29 to +32	3	4
+33 to +36	2	0
+37 to +40	0	3
≥ +42	2	3
No reading	7	2
Total	63	59

Fluctuation in arterial oxygen saturation during entire procedure.

Fluctuation	Number of Cases	
	Anaesthesia	Sedation
Nil	10	7
1 %	15	13
2	9	16
3	7	8
4	6	4
5	0	3
⩾ 6	6	4
No reading	10	4
Total	63	59

Fluctuation in arterial oxygen saturation while catheter is in patient's heart.

Fluctuation	Number of Cases	
	Anaesthesia	Sedation
Nil	18	13
1 %	18	16
2	6	9
3	5	7
4	3	4
5	1	2
≥ 6	2	4
No reading	10	4
Total	63	59

Fluctuations in blood pressure during entire procedure.

Systolic Pressure.

Fluctuation	Number of Cases	
	Anaesthesia	Sedation
Nil	9	6
5 mm.Hg.	16	6
10	12	11
15	6	10
20	6	8
25	4	5
≥ 30	3	12
No reading	7	2
Total	63	59

Fluctuations in blood pressure during entire procedure.

Diastolic Pressure.

Fluctuation	Number of Cases	
	Anaesthesia	Sedation
Nil	24	9
5 mm.Hg.	10	11
10	11	17
15	3	5
20	3	8
25	3	3
≥ 30	2	4
No reading	7	2
Total	63	59

Fluctuations in blood pressure while catheter is in heart.

Systolic Pressure.

Fluctuation	Number of Cases	
	Anaesthesia	Sedation
Nil	19	17
5 mm.Hg.	20	18
10	9	8
15	6	4
20	1	2
25	0	3
⩾ 30	1	4
No reading	7	2
Total	63	59

Fluctuations in blood pressure while catheter is in heart.

Diastolic Pressure.

Fluctuation	Number of Cases	
	Anaesthesia	Sedation
Nil	36	24
5 mm.Hg.	9	11
10	7	11
15	2	4
20	2	4
25	0	2
30	0	1
No reading	7	2
Total	63	59

Fluctuations in heart rate during entire procedure.

Fluctuation	Number of Cases	
	Anaesthesia	Sedation
Nil	4	3
1 - 4 beats/min.	6	7
5 - 8	7	8
9 - 12	9	10
13 - 16	12	8
17 - 20	6	7
21 - 24	3	5
25 - 28	2	4
29 - 32	3	3
$\geq$ 33	4	2
No reading	7	2
Total	63	59

Fluctuations in heart rate while catheter is in heart.

Fluctuation	Number of Cases	
	Anaesthesia	Sedation
Nil	9	10
1 - 4 beats/m.n.	16	13
5 - 8	9	14
9 - 12	8	8
13 - 16	3	3
17 - 20	4	3
21 - 24	0	3
25 - 28	2	2
29 - 32	2	0
$\geqslant$ 33	3	1
No reading	7	2
Total	63	59

APPENDIX 'D'

CASE RECORDS

CASE RECORDS.

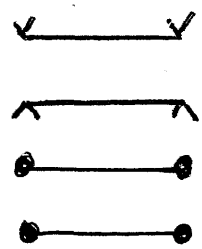
Symbols.

Systolic blood pressure (mm.Hg.)

Diastolic blood pressure (mm.Hg.)

Arterial oxygen saturation (%)

Heart rate (beats/minute)



Abbreviations.

P.S.	Pulmonary stenosis
I.A.S.D.	Atrial Septal Defect
I.V.S.D.	Ventricular Septal Defect
P.H.	Pulmonary hypertension
C. of A.	Coarctation of Aorta
F.T.	Fallot's Tetralogy
P.D.A.	Patent Ductus Arteriosus
A.S.	Aortic Stenosis
M.S.	Mitral Stenosis
L.S.V.C.	Left Superior Vena Cava
C.D.	Cut down being performed
C.E.H.	Catheter entering heart.
D.N.E.	Diagnosis not established.

The resting values obtained in the ward are marked on the line immediately to the left of the main chart.

Supplementary injections of thiopentone have been indicated by red arrows at the appropriate point on the top of the chart, the dose in mgms. being given after the letter 'T'.

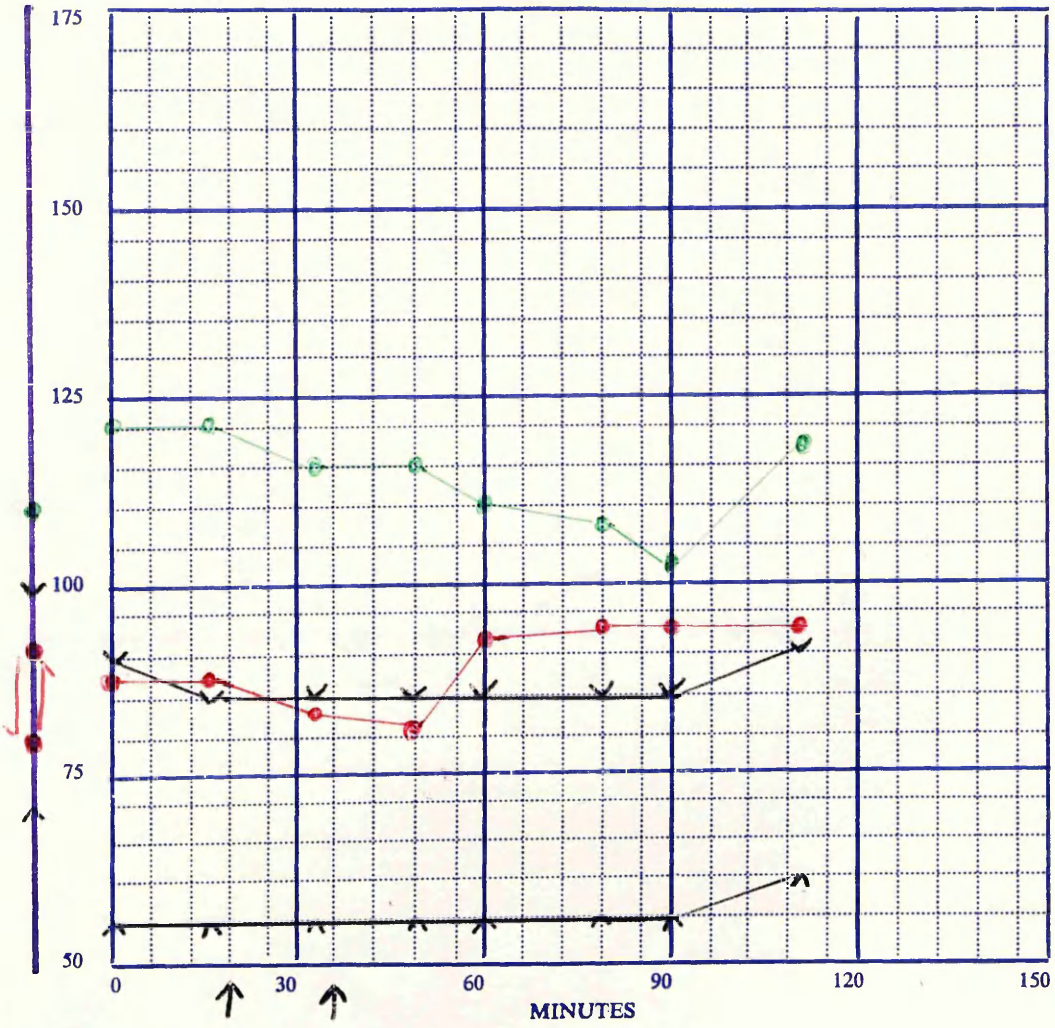
Where any dramatic changes have taken place, a red arrow on the lower part of the chart is used to indicate the position of the catheter in the heart at that time.

CASE RECORD No.....1.....

Name W.D. Age 2½ Diagnosis F.T.

Weight 14.2 Kilos. Haemoglobin 120%

Premedication Promethazine 10 mgms.
Bromethol 1.5 c.c.
Pethidine 30 mgms.
Atropine 0.6 mgms.



C.D. C.E.H.

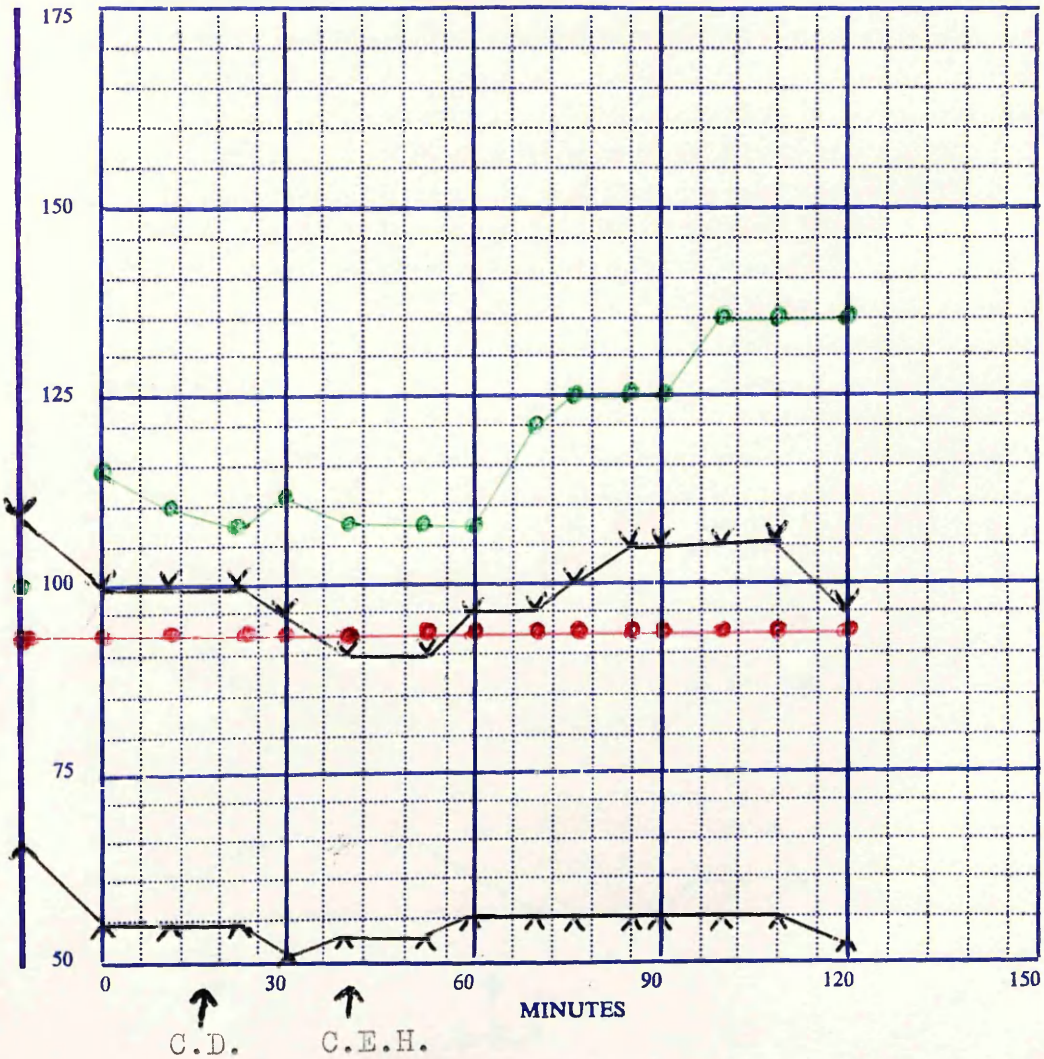
NOTES The fluctuation in oxygen saturation noted at the pre-operative examination was caused by the child crying.

CASE RECORD No. 2.

Name S.R. Age $2\frac{1}{2}$ Diagnosis P.D.A.

Weight 13 Kilos. Haemoglobin 100%

Premedication Promethazine 10 mgms.
Bromethol 1.6 c.c.
Pethidine 25 mgms.
Atropine 0.6 mgms.



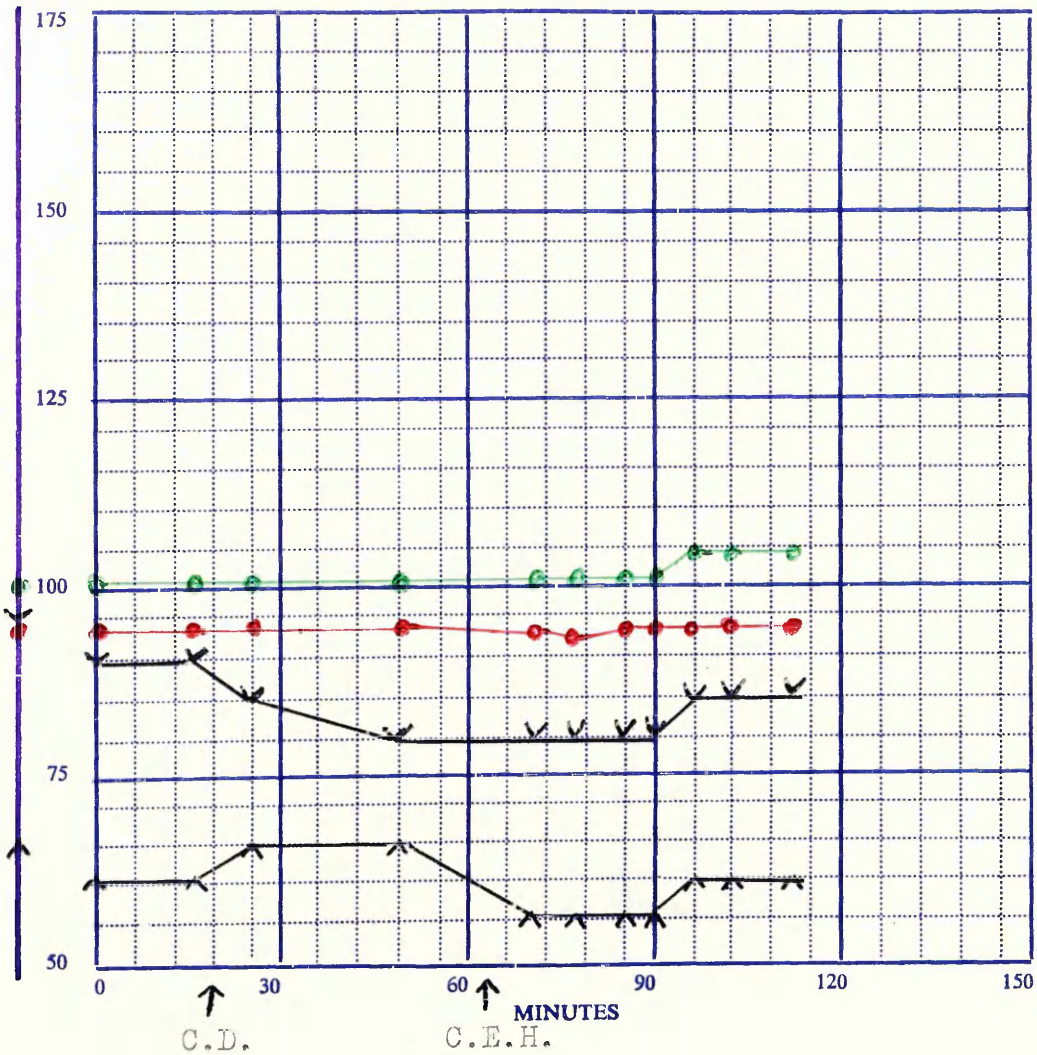
NOTES Arterial analysis pCO_2 40 mm.Hg.

CASE RECORD No. 3.

Name M. McS. Age 2½ Diagnosis I.A.S.D.

Weight 12 Kilos. Haemoglobin 98%

Premedication Promethazine 25 mgms.
Bromethol 1.5 c.c.
Pethidine 20 mgms. (Intravenously)



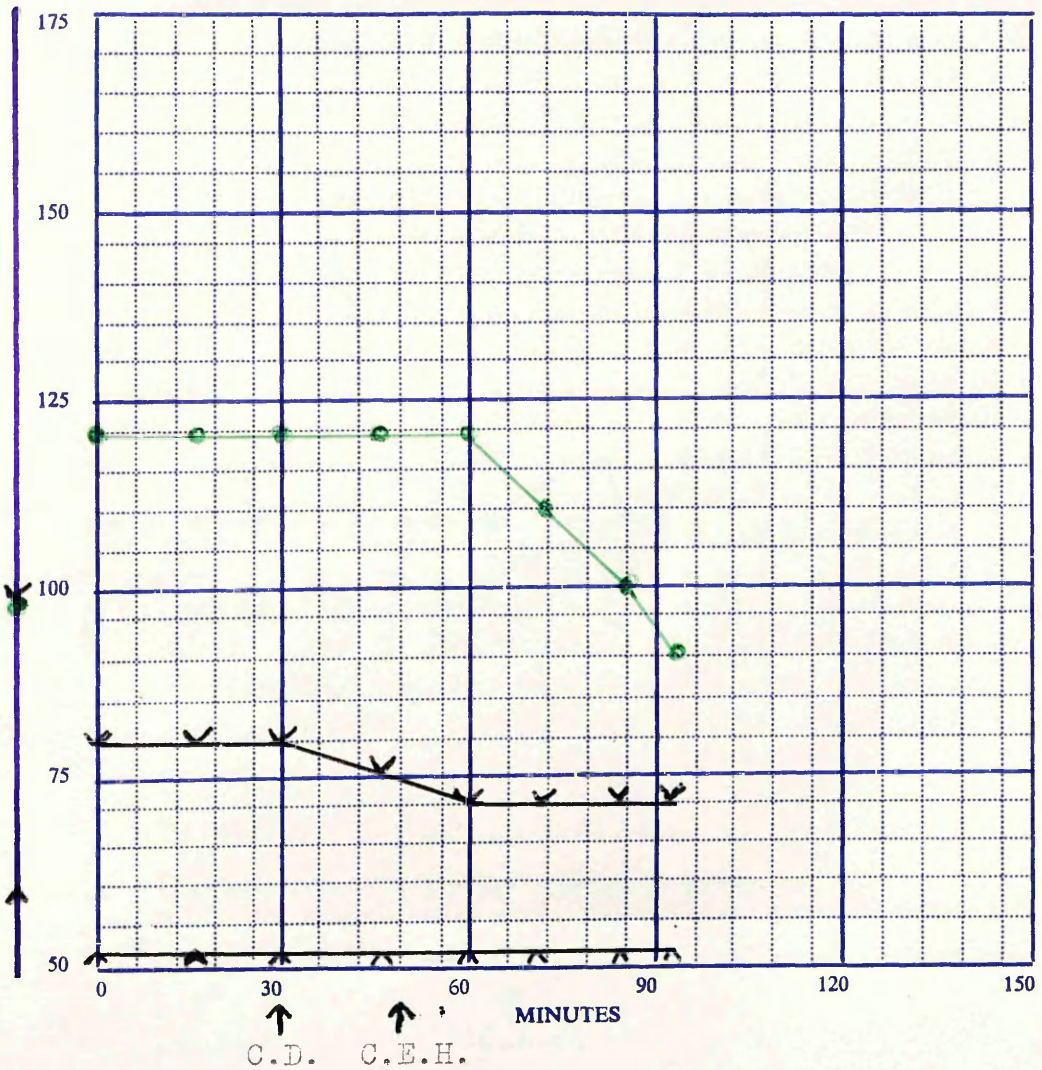
NOTES Arterial analysis - oxygen saturation 94%
(oximeter reading 93%).
pCO_2 39 mmHg.

CASE RECORD No.....4.....

Name G.D. Age 3 Diagnosis I.V.S.D.

Weight 14 Kilos. Haemoglobin 92%

Premedication Promethazine 20 mgms.
Bromethol 1.5 c.c.
Pethidine 30 mgms.
Atropine 0.6 mgms.



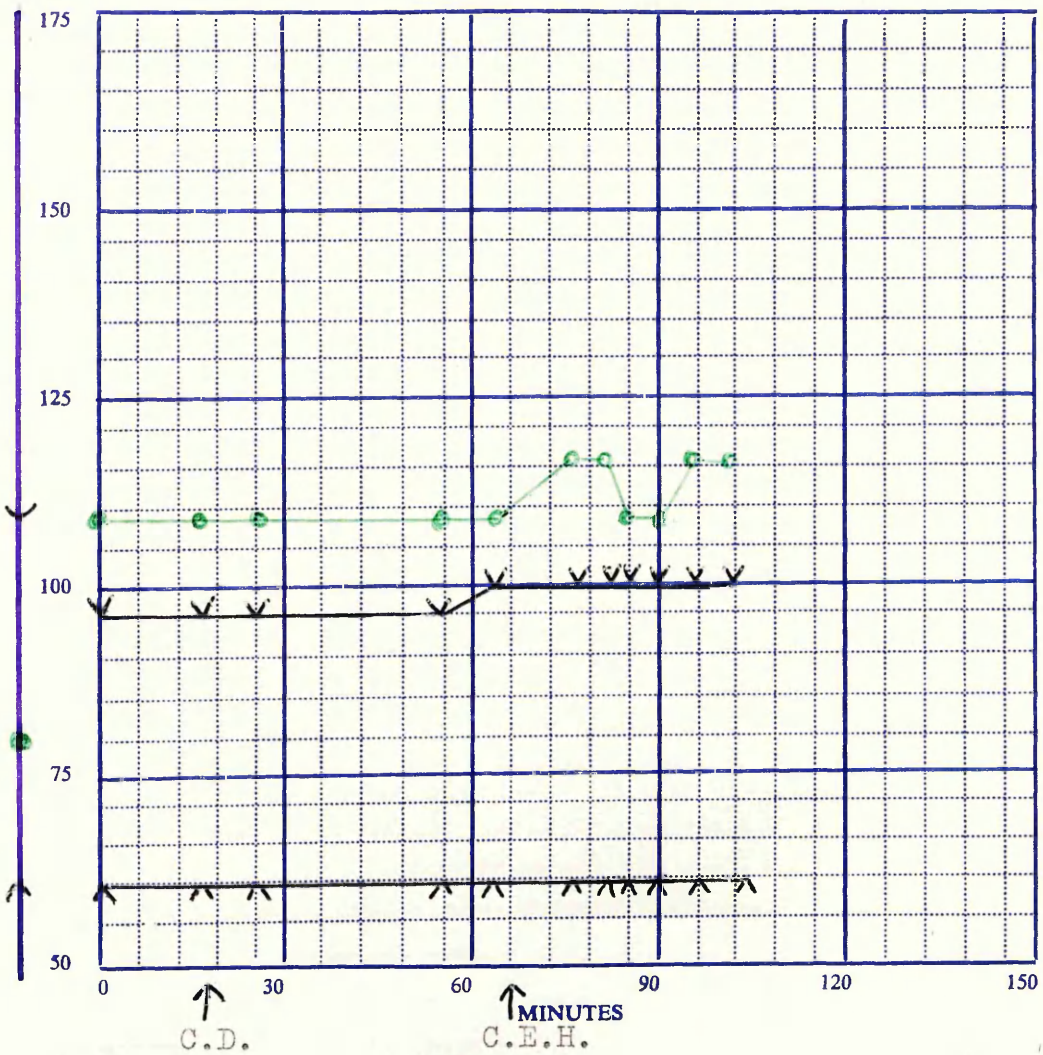
NOTES The ear piece of the oximeter was under repair at this time and readings were not possible.

CASE RECORD No. 5.

Name G.R. Age 3 Diagnosis D.N.E.

Weight 15 Kilos. Haemoglobin 85%

Premedication Promethazine 10 mgms.
Bromethol 1.8 c.c.
Pethidine 25 mgms.
Atropine 0.6 mgms.



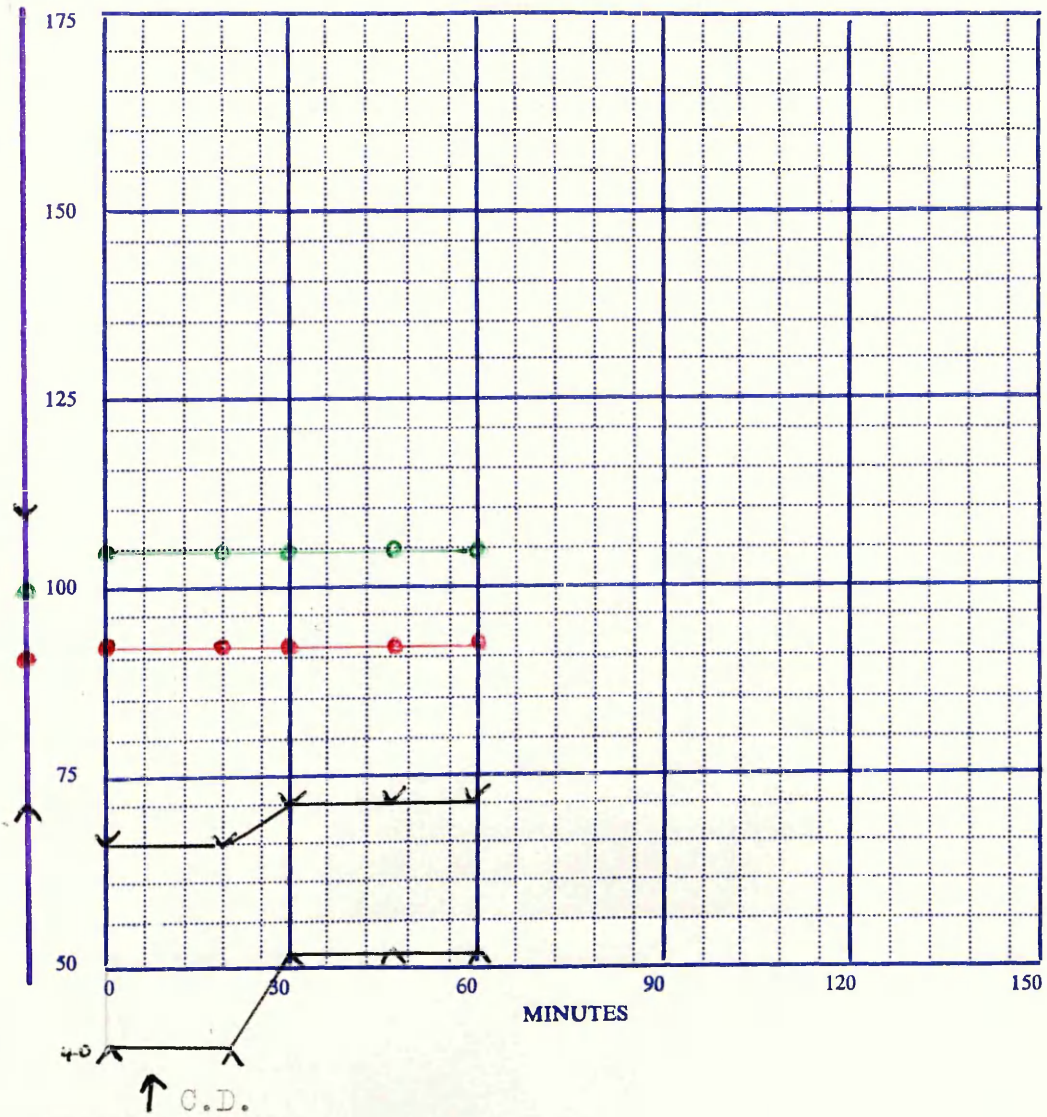
NOTES An uneventful catheterisation. The oximeter ear piece was under repair at this time.

CASE RECORD No. 6.

Name H.M. Age 3½ Diagnosis A.S.

Weight 17 Kilos. Haemoglobin 90%

Premedication Promethazine 10 mgms.
Bromethol 2 c.c.
Pethidine 35 mgms.
Atropine 0.6 mgms.



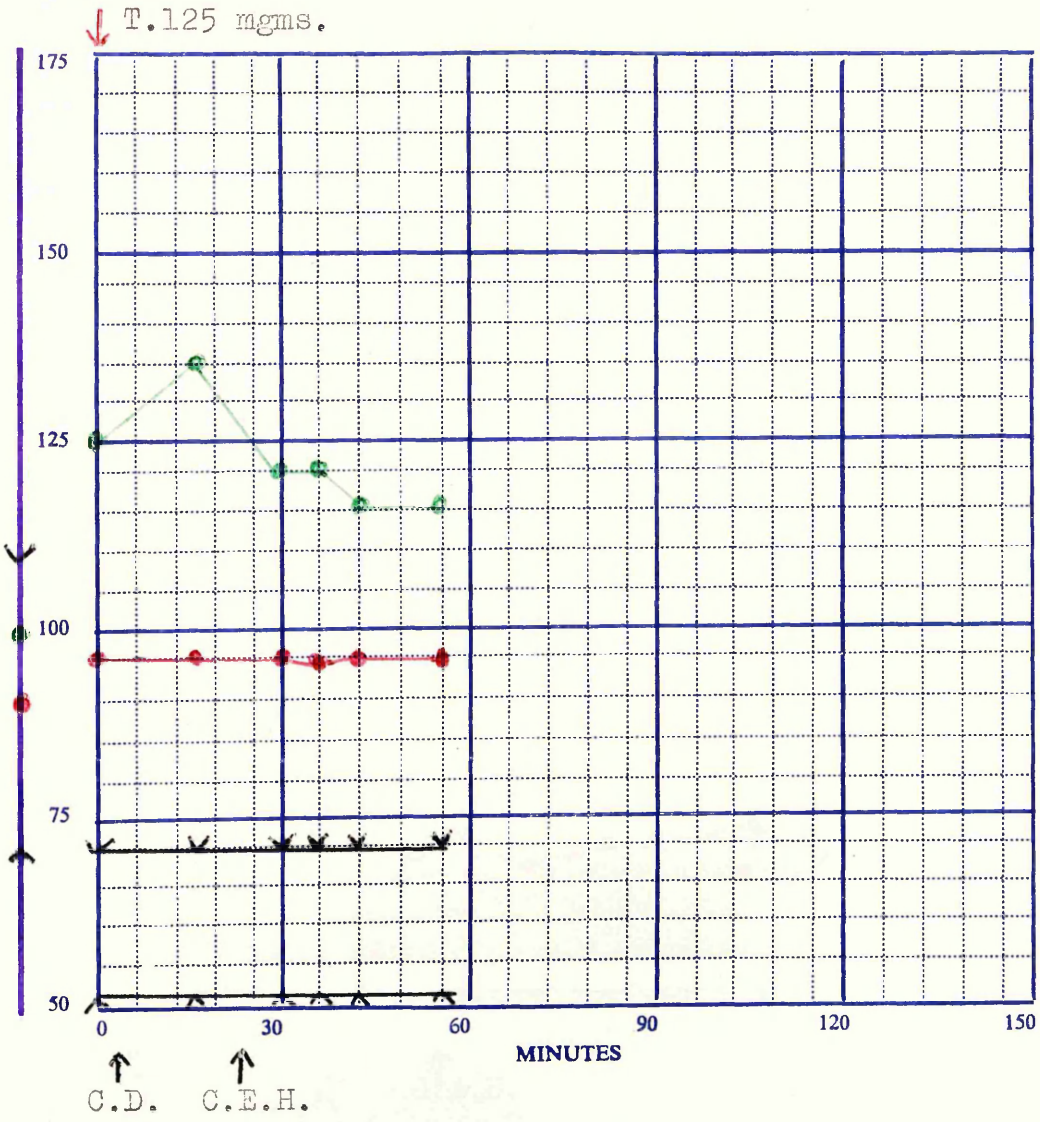
NOTES This was an unsuccessful catheterisation, the catheter passing only as far as the jugular vein.

CASE RECORD No. 7.

Name H.M. Age 3½ Diagnosis A.S.

Weight 17 Kilos. Haemoglobin 90%

Premedication
Promethazine 10 mgms.
Bromethol 1.6 c.c.
Pethidine 35 mgms.
Atropine 0.6 mgms.



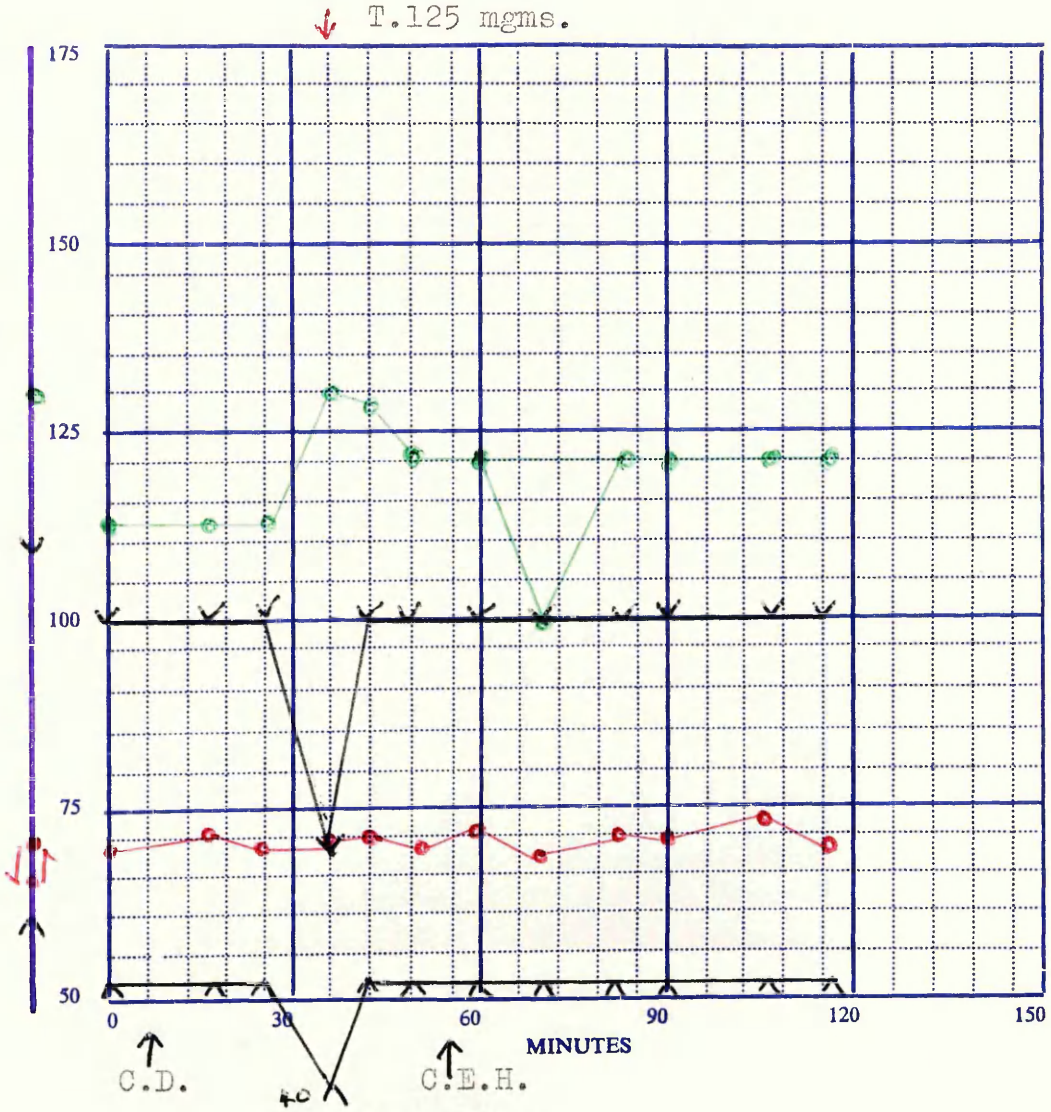
NOTES This was a repeat catheterisation on the previous case. The dose of Bromethol was reduced by 20% in view of the marked hypotension which had followed the administration of the enema. This smaller dose proved insufficient and Thiopentone was given at the beginning of the procedure.

CASE RECORD No. 8.

Name R.T. Age 3½ Diagnosis F.T.

Weight 14 Kilos. Haemoglobin 106%

Premedication Promethazine 10 mgms.
Bromethol 1.5 c.c.
Pethidine 50 mgms.
Atropine 0.6 mgms.



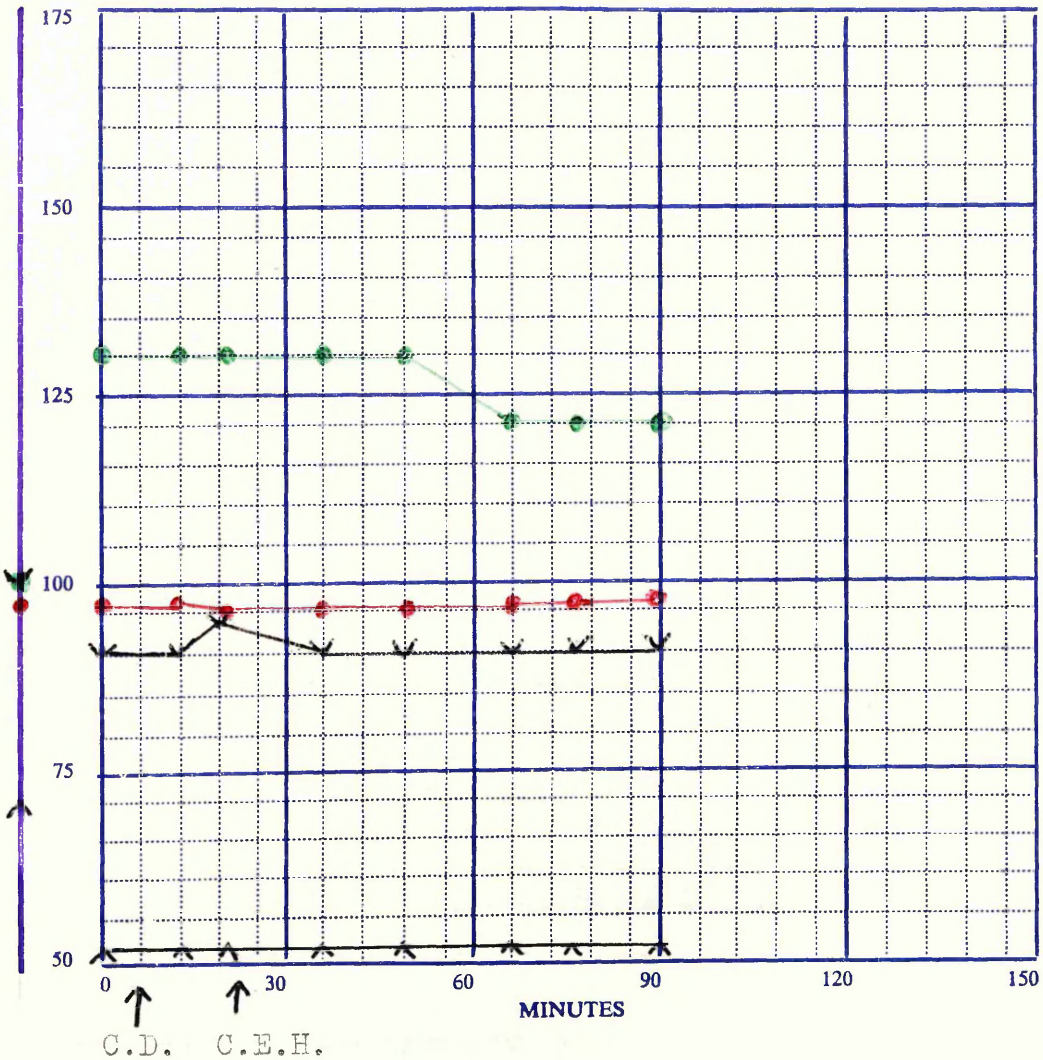
NOTES This catheterisation was accomplished after extreme technical difficulty. Marked hypotension accompanied the administration of the Thiopentone.

CASE RECORD No. 9.

Name M.D. Age 3½ Diagnosis I.A.S.D.

Weight 16 Kilos. Haemoglobin 105%

Premedication Promethazine 10 mgms.
Bromethol 1.5 c.c.
Pethidine 35 mgms.
Atropine 0.6 mgms.



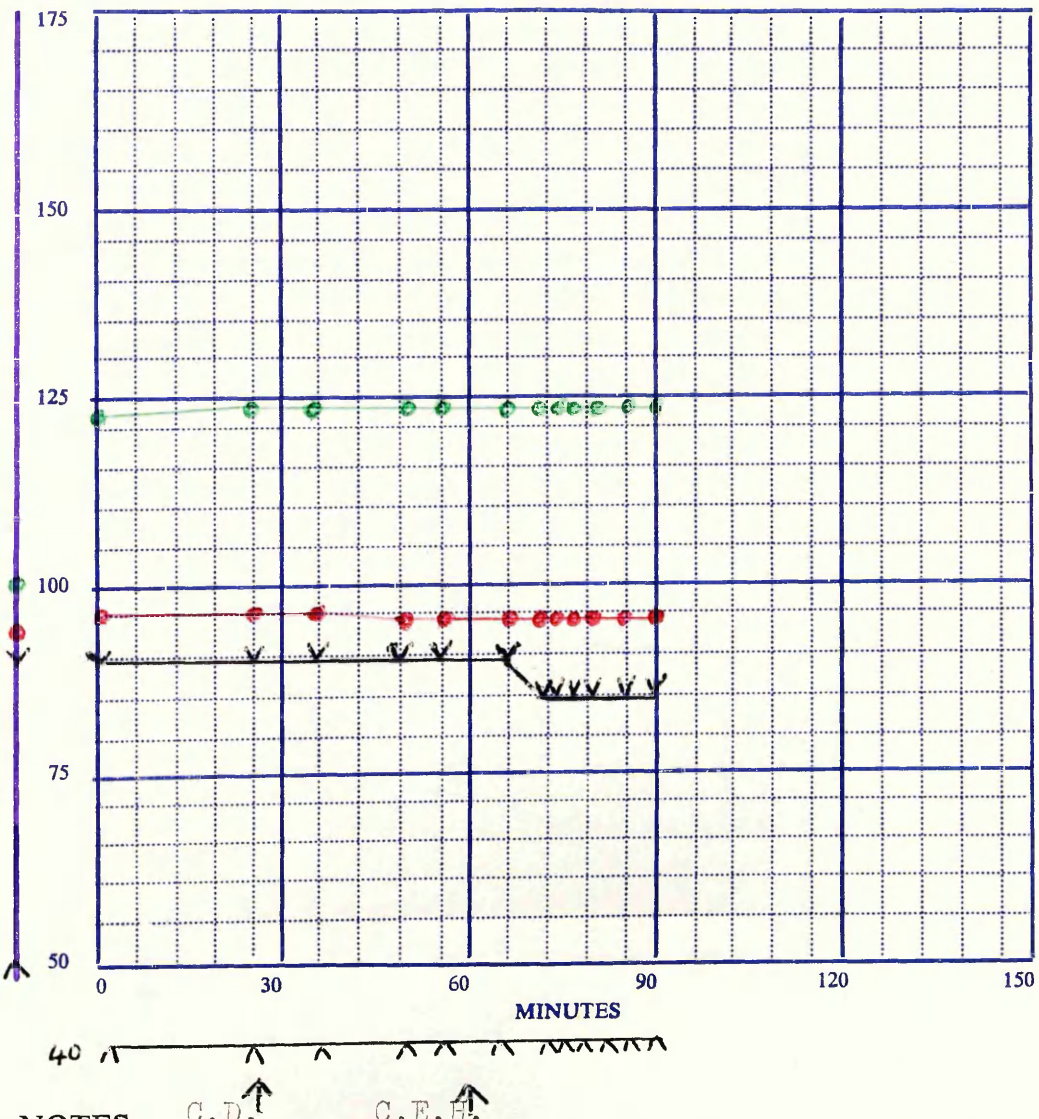
NOTES This was an uneventful catheterisation.

CASE RECORD No. 10

Name R.V. Age 3½ Diagnosis P.D.A.

Weight 12 Kilos. Haemoglobin 105%

Premedication Promethazine 20 mgms.
Bromethol 1.5 c.c.
Pethidine 25 mgms.
Atropine 0.6 mgms.



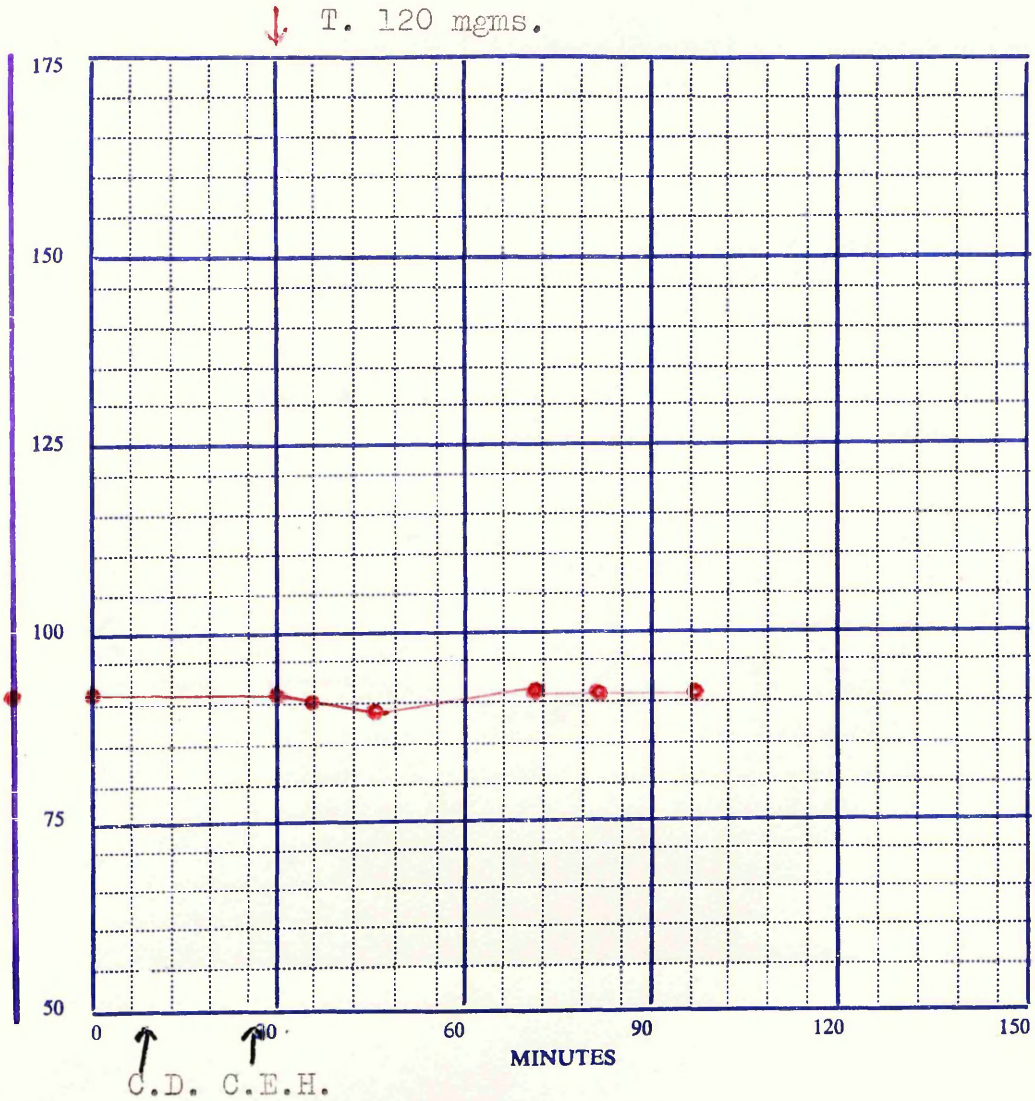
NOTES This was an uneventful catheterisation.

CASE RECORD No. 11.

Name D.F. Age 4 Diagnosis P.H.

Weight 15.5 Kilos. Haemoglobin 96%

Premedication Promethazine 10 mgms.
Paraldehyde 71.5 c.c.
Pethidine 30 mgms.
Atropine 0.6 mgms.



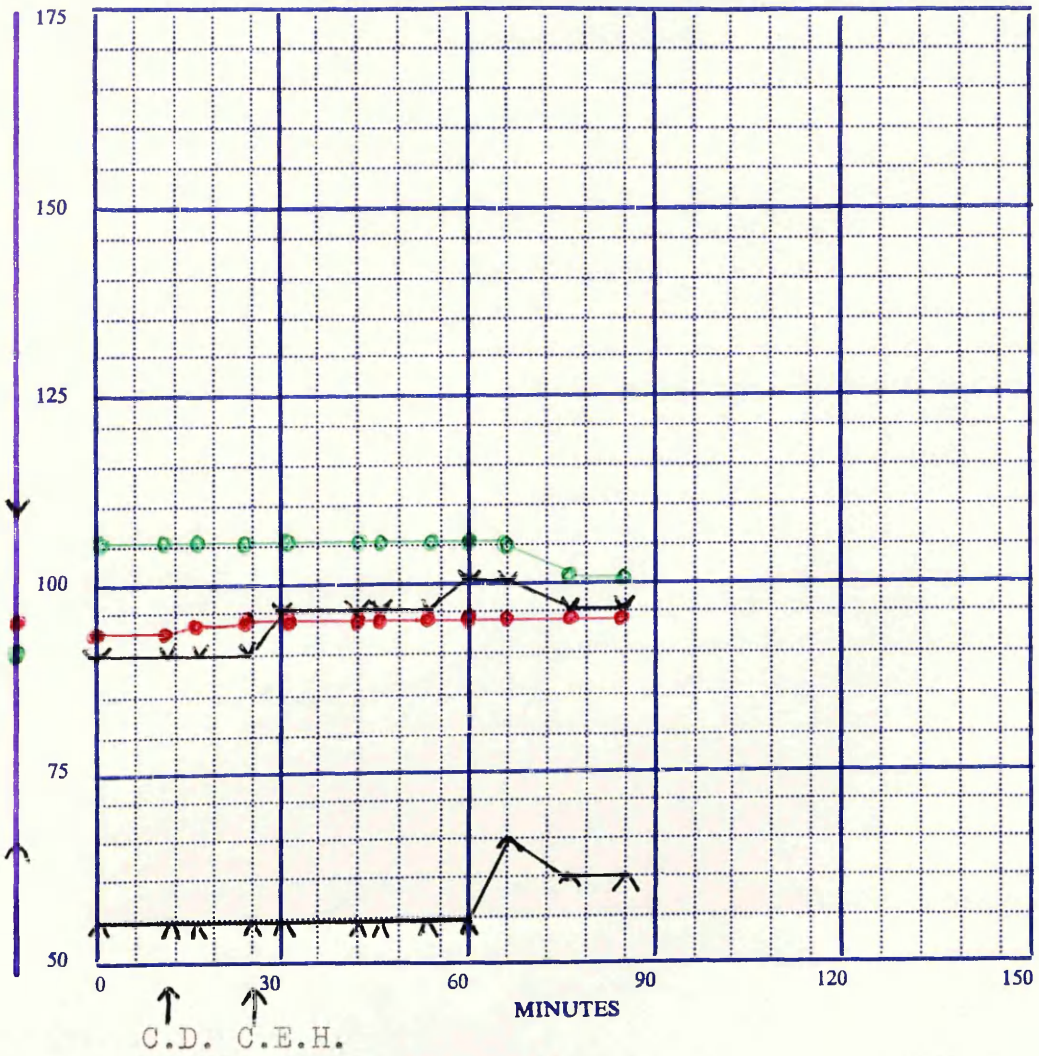
NOTES Bromethol was not available and Paraldehyde was used in its place.

CASE RECORD No.....14.....

Name R.D. Age 4 Diagnosis D.N.E.

Weight 15.5 Kilos. Haemoglobin 84%

Premedication Promethazine 10 mgms.
Bromethol 1.8 c.c.
Pethidine 25 mgms.
Atropine 0.6 mgms.



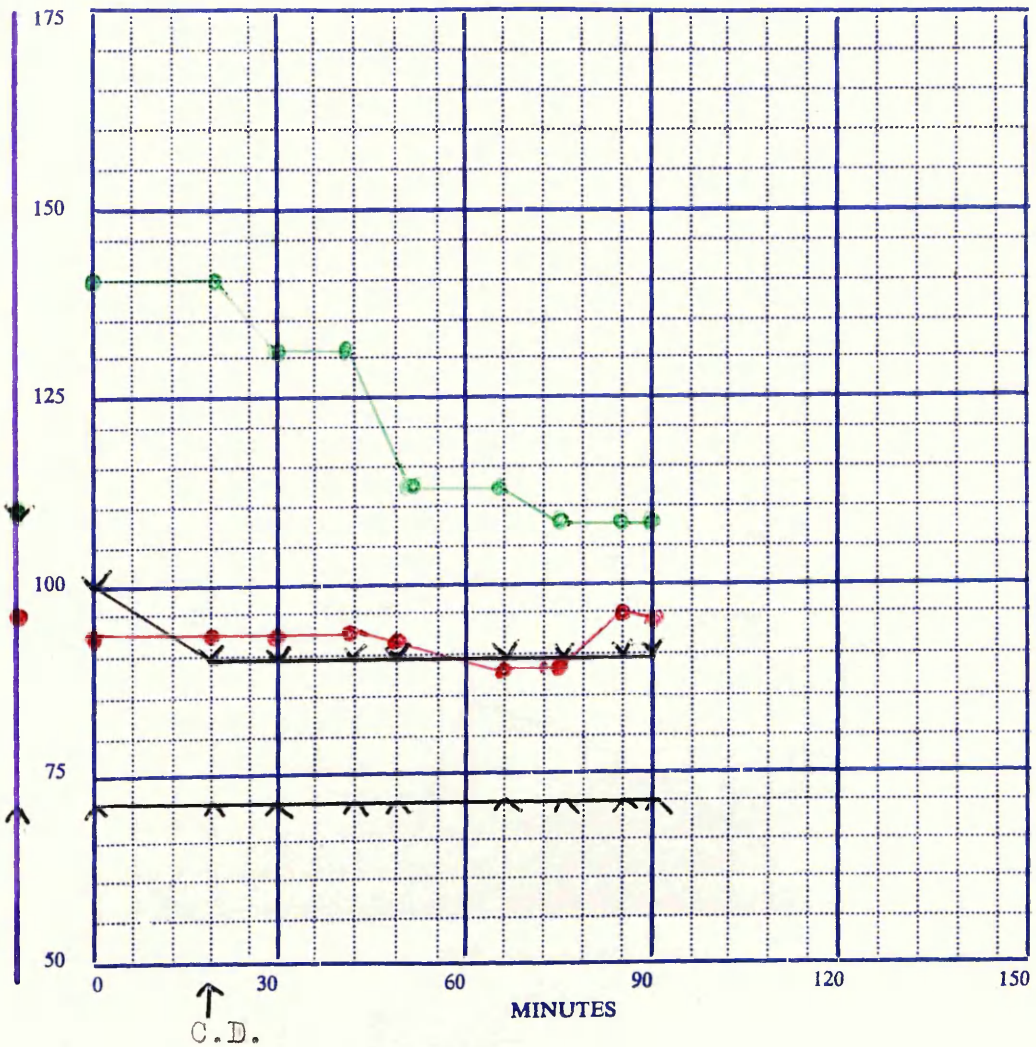
NOTES An attempt was made to perform an arterial puncture but the child woke up and would not tolerate the procedure.

CASE RECORD No. 12.

Name A.M. Age 4 Diagnosis D.N.E.

Weight 14 Kilos. Haemoglobin 102%

Premedication Promethazine 10 mgms.
Bromethol 1.75 c.c.
Pethidine 50 mgms.
Atropine 0.6 mgms.



NOTES

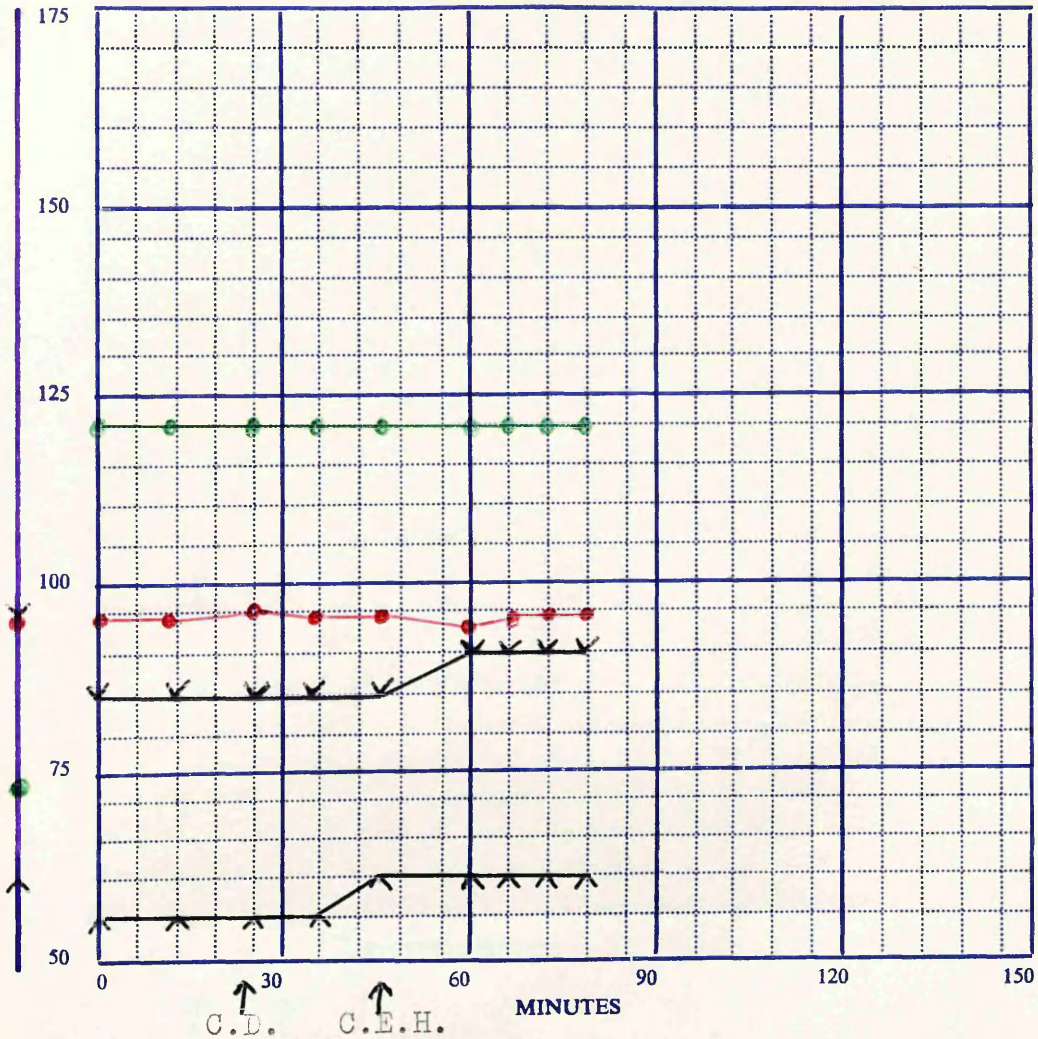
This child was in error given twice the prescribed dose of pethidine. After he fell asleep he developed a respiratory obstruction due to his tongue falling back and for a few moments was deeply cyanosed. He rapidly recovered when an oral airway was inserted.

CASE RECORD No. 15.

Name A.S. Age 5 Diagnosis D.N.E.

Weight 16 Kilos. Haemoglobin 100%

Premedication Promethazine 10 mgms.
Bromethol 2 c.c.
Pethidine 35 mgms.
Atropine 0.6 mgms.



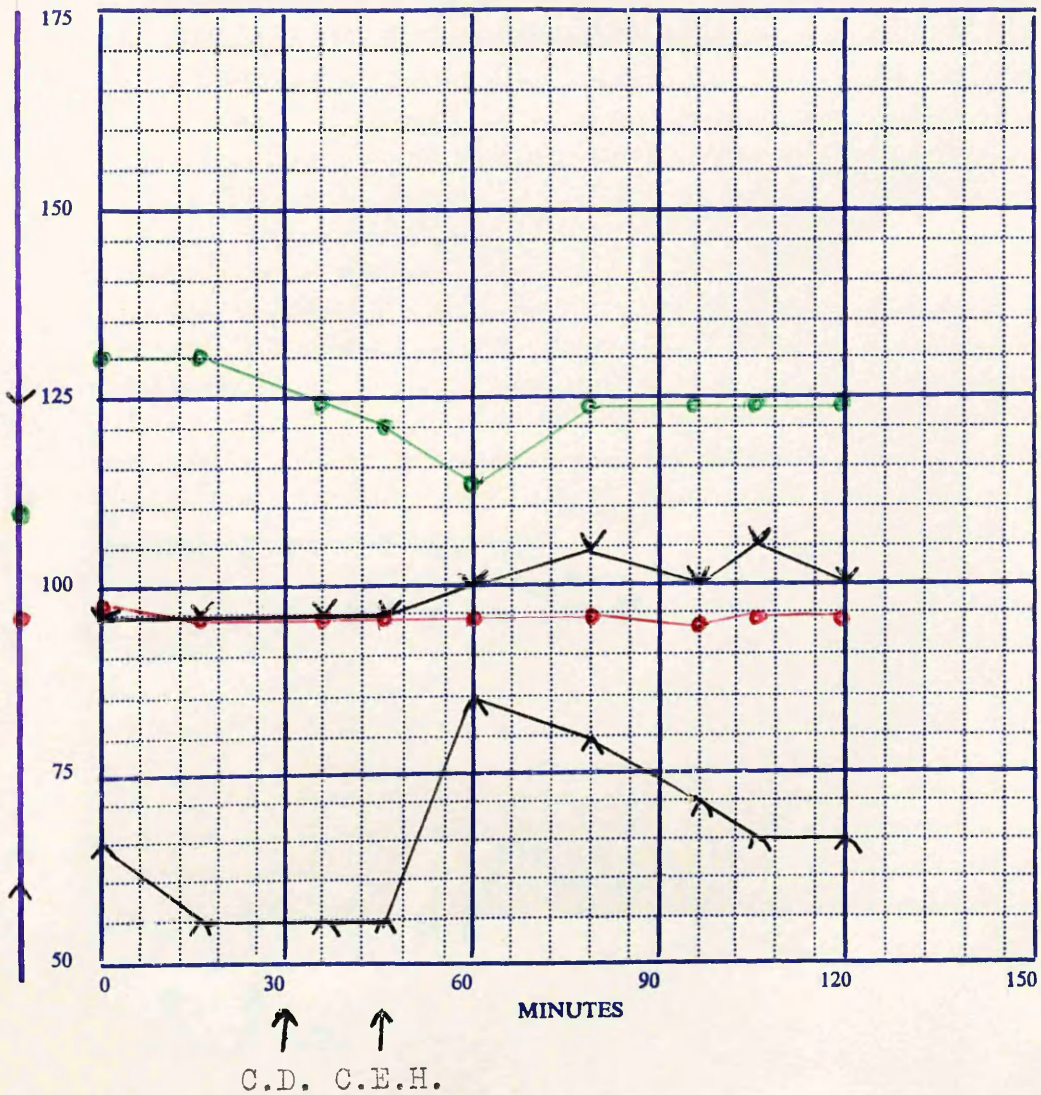
NOTES Arterial analysis - pH 7.35
pCO_2 38 mmHg.

CASE RECORD No. 13.

Name R.K. Age 4 Diagnosis I.V.S.D.

Weight 15.5 Kilos. Haemoglobin 100%

Premedication Promethazine 20 mgms.
Bromethol 1.8 c.c.
Pethidine 25 mgms.
Atropine 0.6 mgms.



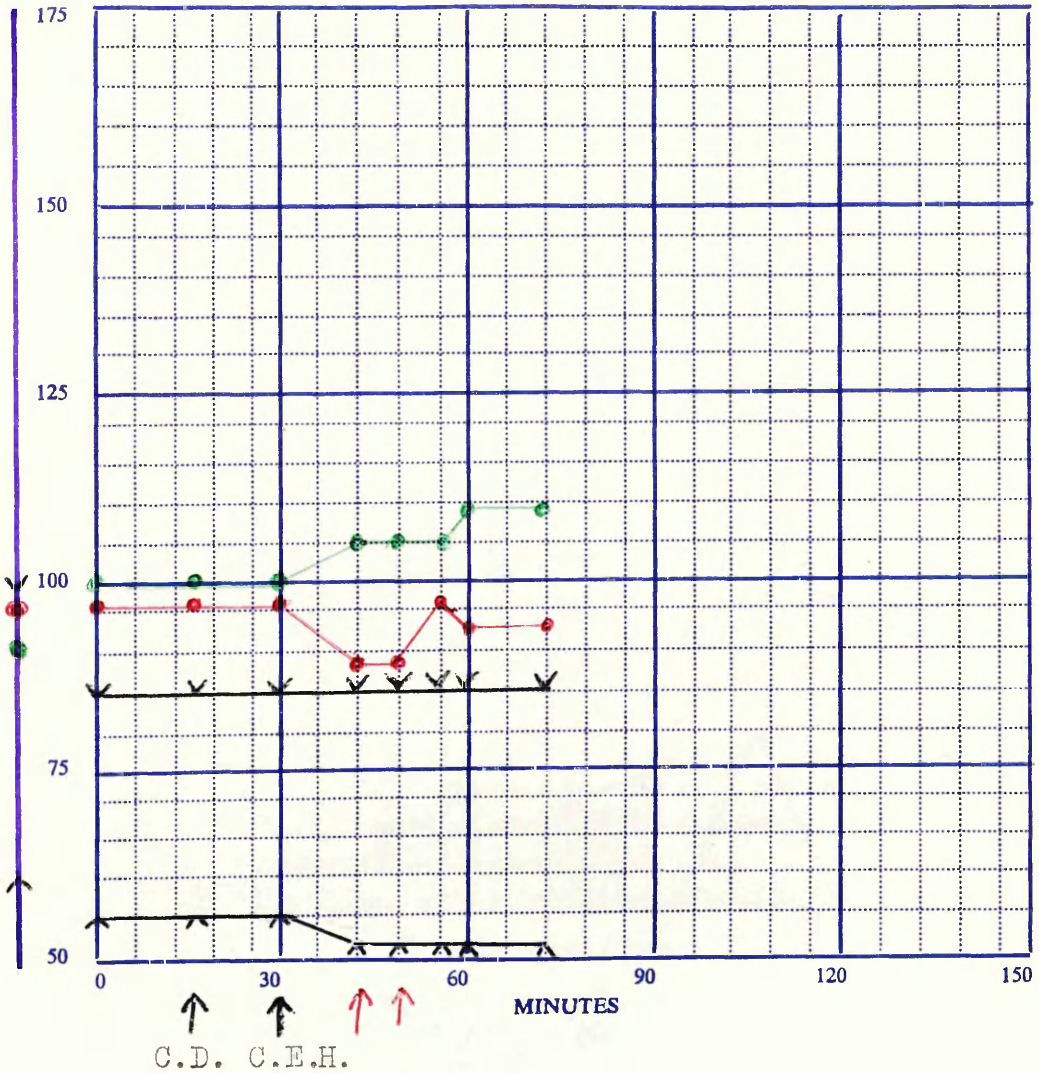
NOTES This was an uneventful catheterisation.

CASE RECORD No. 16.

Name F.McG. Age 5 Diagnosis I.V.S.D.

Weight 21 Kilos. Haemoglobin 97%

Premedication Promethazine 25 mgms.
Bromethol 2.5 c.c.
Pethidine 45 mgms.
Atropine 0.6 mgms.



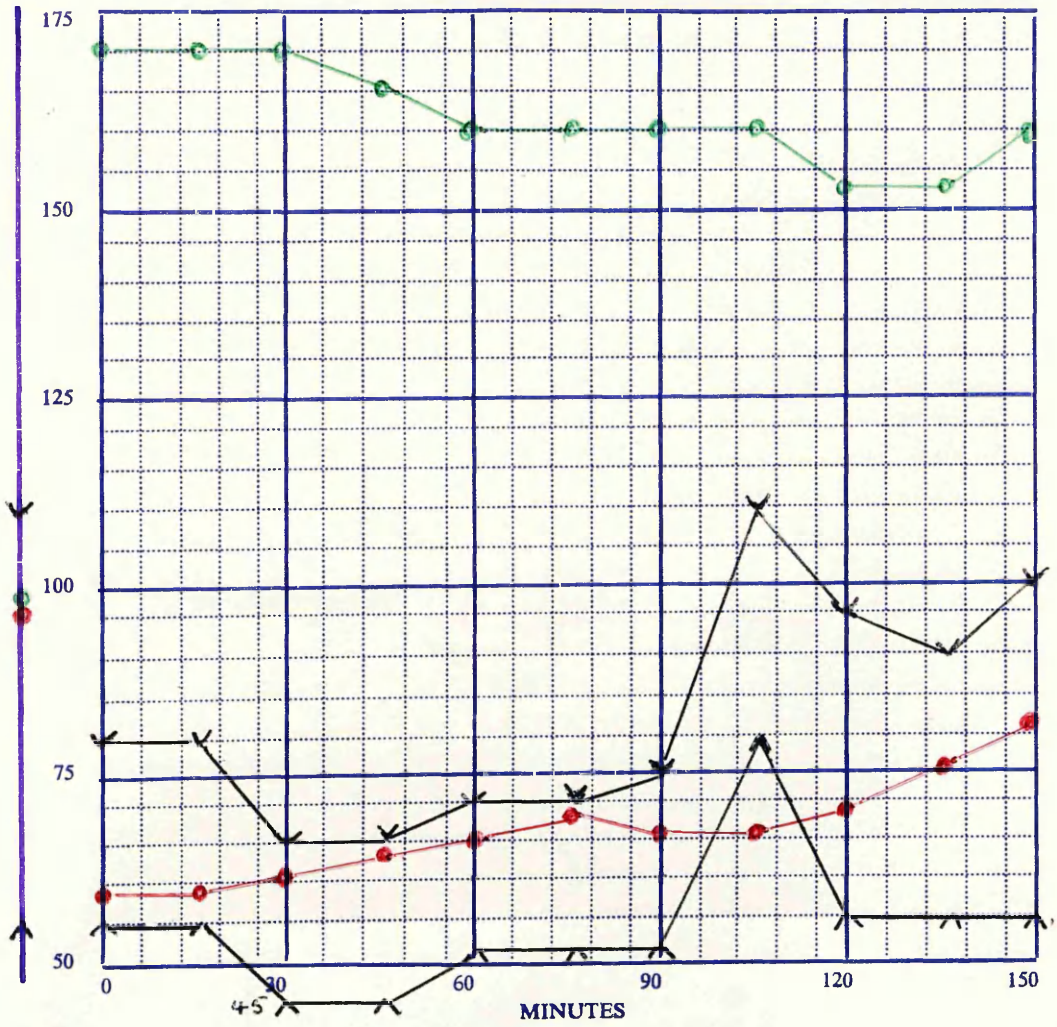
NOTES The red arrows indicate the presence of the catheter tip in the pulmonary artery. Corresponding with this there is a drop in arterial oxygen saturation.

CASE RECORD No.....17.....

Name A.M. Age 5 Diagnosis D.N.E.

Weight 22 Kilos. Haemoglobin 120%

Premedication Promethazine 10 mgms.
Bromethol 1.8 c.c.
Pethidine 25 mgms.
Atropine 0.6 mgms.



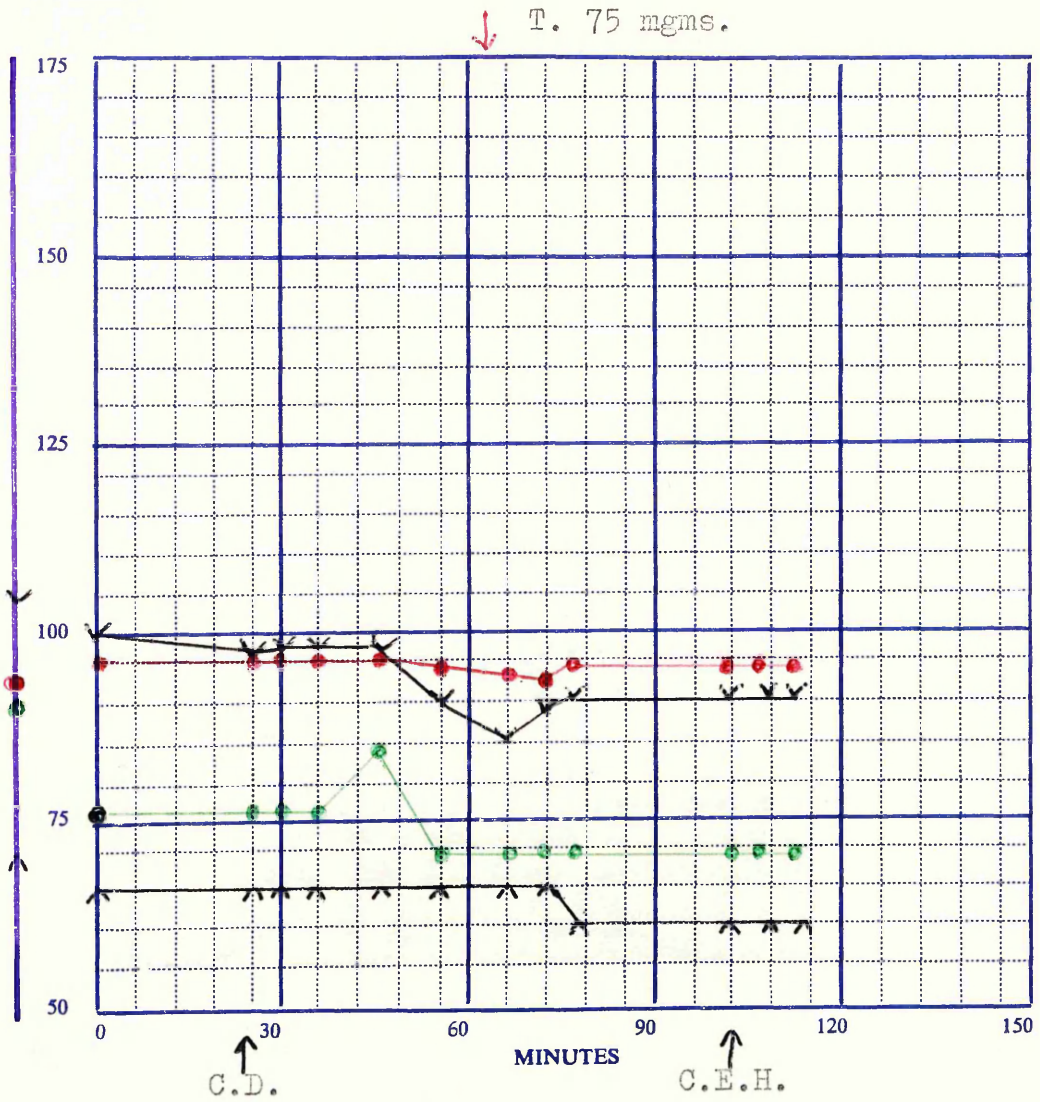
NOTES This child was previously catheterised (Case 12). In error he was allowed to develop a partial respiratory obstruction. In addition his pulse rate rose to 180 and catheterisation was not performed. This case was reported in detail and discussed in the text under "Complications of Anaesthesia".

CASE RECORD No. 18.

Name C.McD. Age 5 Diagnosis L.S.V.C.

Weight 22 Kilos. Haemoglobin 100%

Premedication Promethazine 25 mgms.
Bromethol 2 c.c.
Pethidine 25 mgms.) Half of this injection was lost.
Atropine 0.4 mgms.)



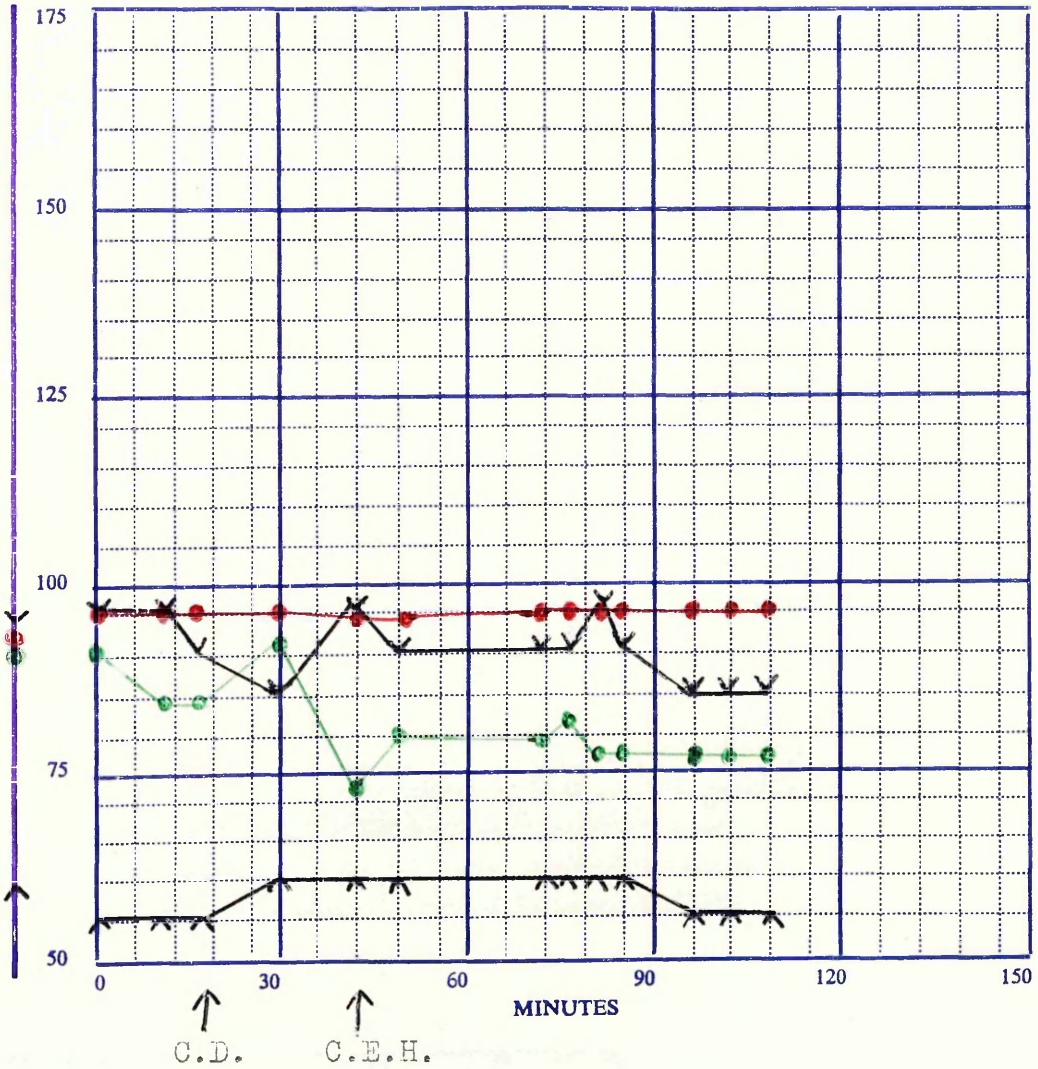
NOTES Arterial analysis - pCO_2 55 mmHg.
pH 7.27
Oxygen saturation 95%
Oximeter reading 96%

CASE RECORD No. 19.

Name C.McD. Age 5 Diagnosis L.S.V.C.

Weight 23 Kilos. Haemoglobin 94%

Premedication Promethazine 25 mgms.
Bromethol 2 c.c.
Pethidine 25 mgms.
Atropine 0.6 mgms.



NOTES Arterial analysis - pCO_2 40 mmHg.
pH 7.36

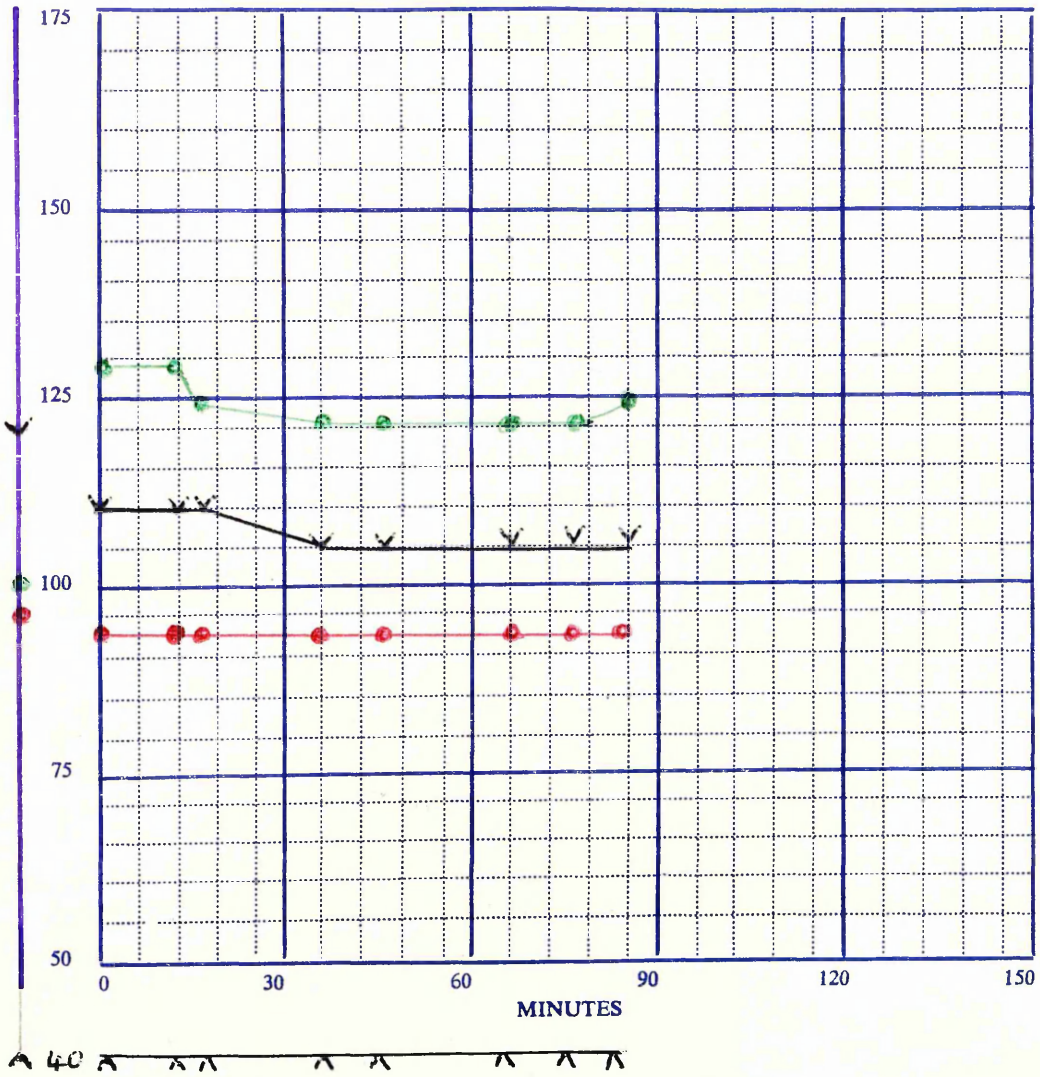
This is a repeat of the previous case.

CASE RECORD No.....20.....

Name E.S. Age 5 Diagnosis P.D.A.

Weight 20.5 Kilos. Haemoglobin 90%

Premedication Promethazine 10 mgms.
Bromethol 1.8 c.c.
Pethidine 25 mgms.
Atropine 0.6 mgms.



NOTES C.D. C.E.H.

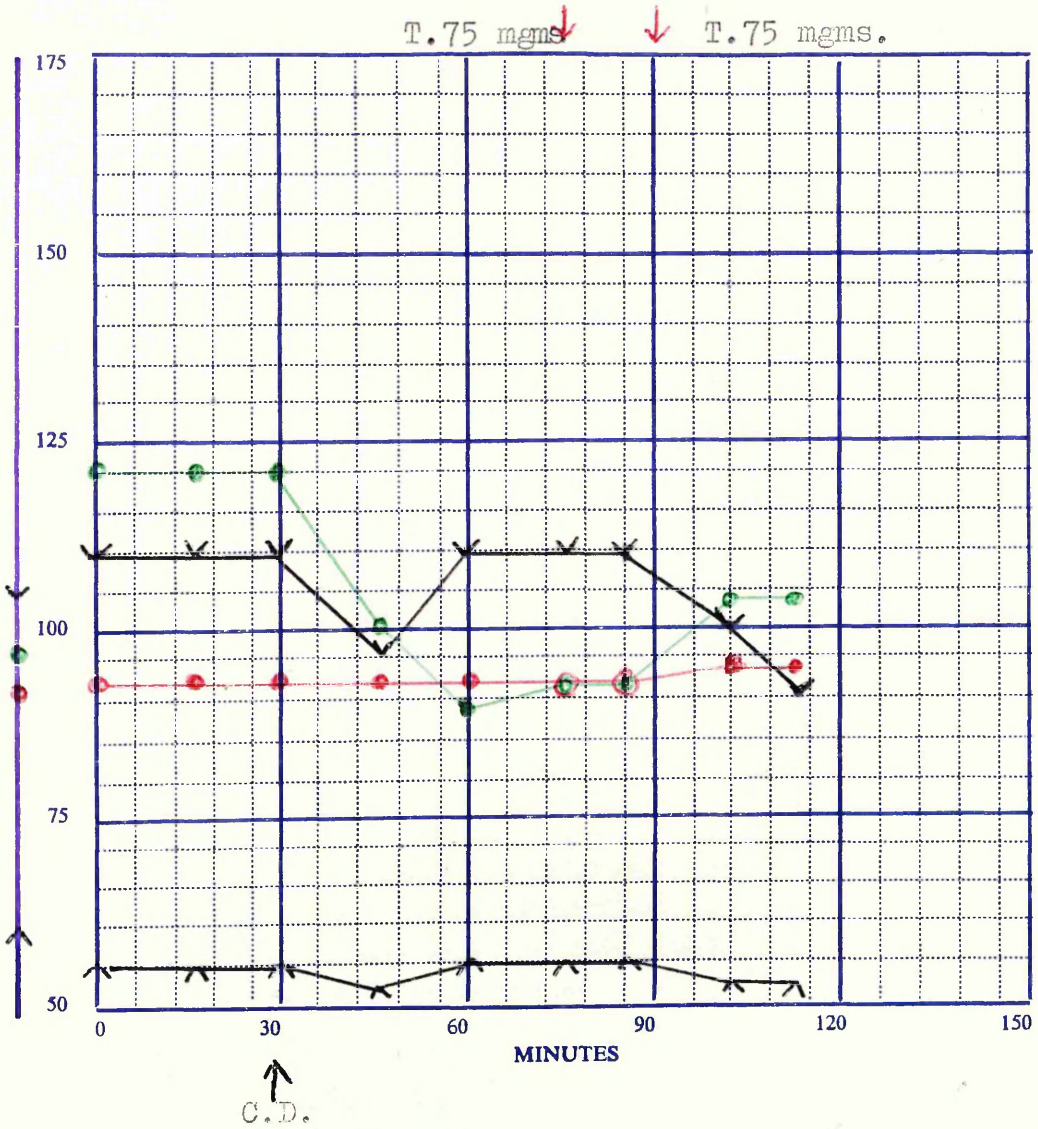
Arterial analysis - Oxygen saturation 90%
Oximeter reading 92%

CASE RECORD No. 21.

Name D.K. Age 5½ Diagnosis F.T.

Weight 23.5 Kilos. Haemoglobin 98%

Premedication Promethazine 25 mgms.
Bromethol 3 c.c.
Pethidine 50 mgms.
Atropine 0.6 mgms.



NOTES This was an unsuccessful catheterisation as the catheter would only pass a few inches into the vein. Considerable manipulation took place and was accompanied by restlessness. Despite the Thiopentone operating conditions were far from good.

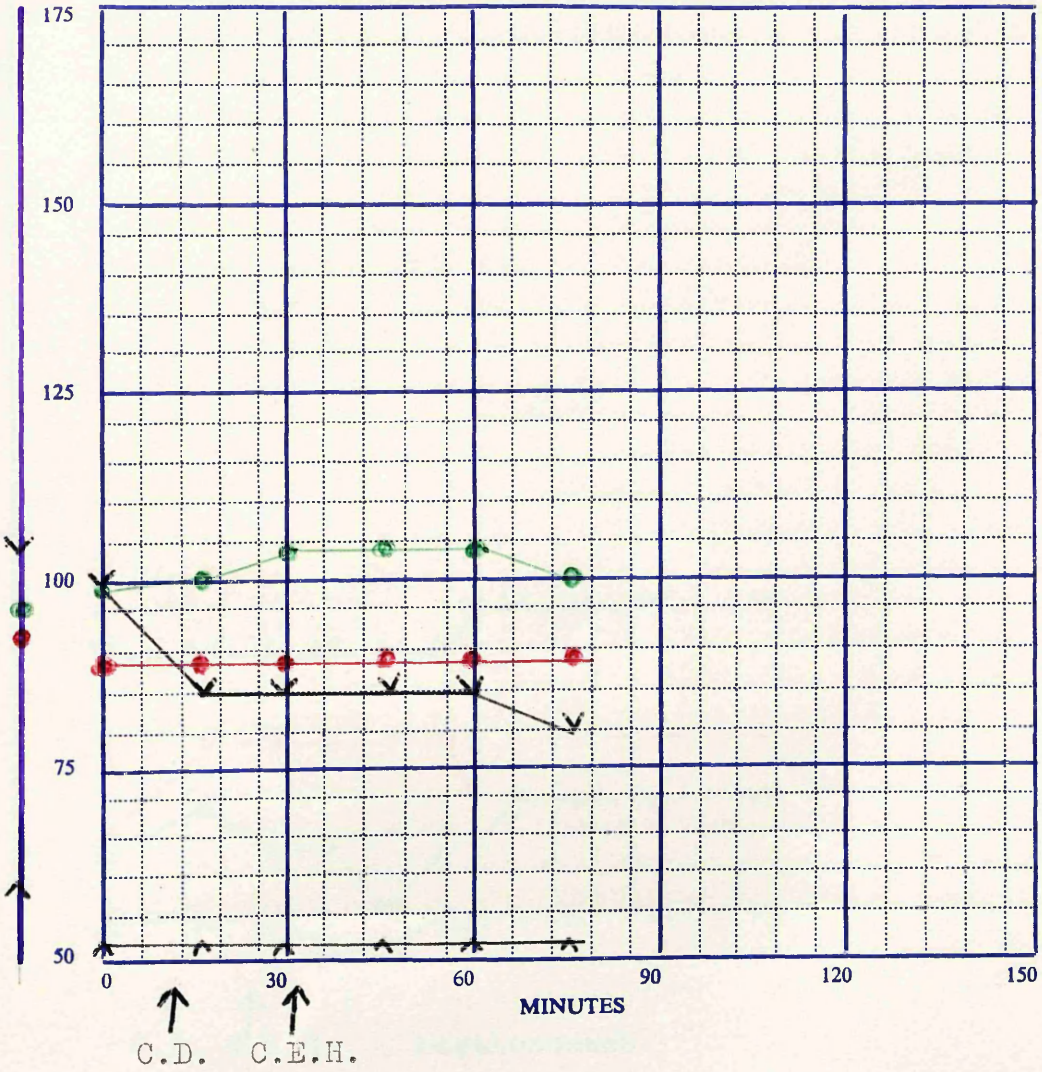
CASE RECORD No. 22.

Name D.K. **Age** 5½ **Diagnosis** F.T.

Weight 23.5 Kilos. **Haemoglobin** 98%

Premedication Promethazine 25 mgms.
Bromethol 3 c.c.
Pethidine 75 mgms.
Atropine 0.6 mgms.

↓ T.100 mgms.



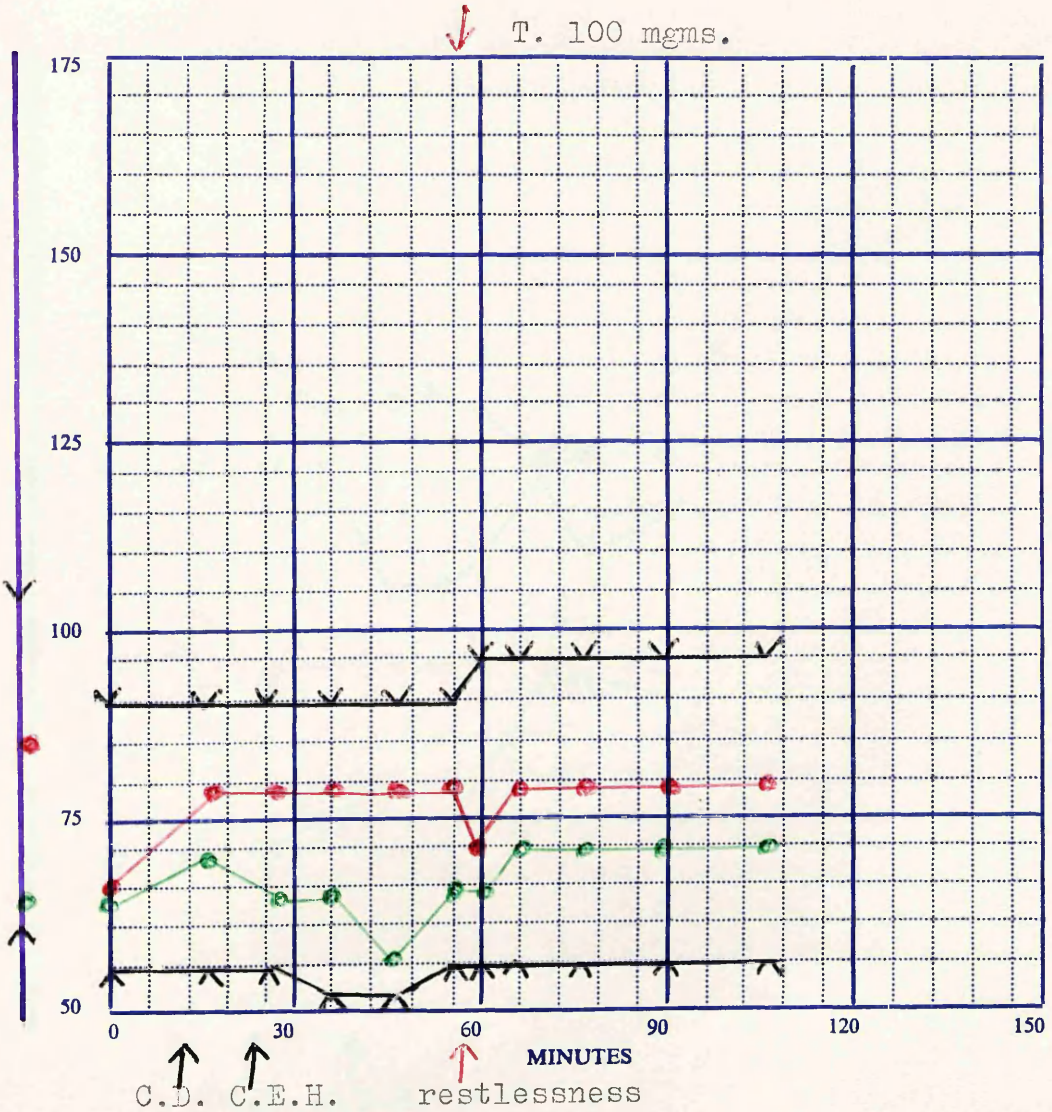
NOTES This was a repeat catheterisation on previous patient. The Pethidine was increased by 50% and operating conditions were correspondingly improved.

CASE RECORD No.....23.....

Name K.M. Age 6 Diagnosis I.V.S.D. + P.H.

Weight 15 Kilos. Haemoglobin 130%

Premedication Promethazine 10 mgms.
Paraldehyde 57 c.c.
Pethidine 30 mgms.
Atropine 0.6 mgms.



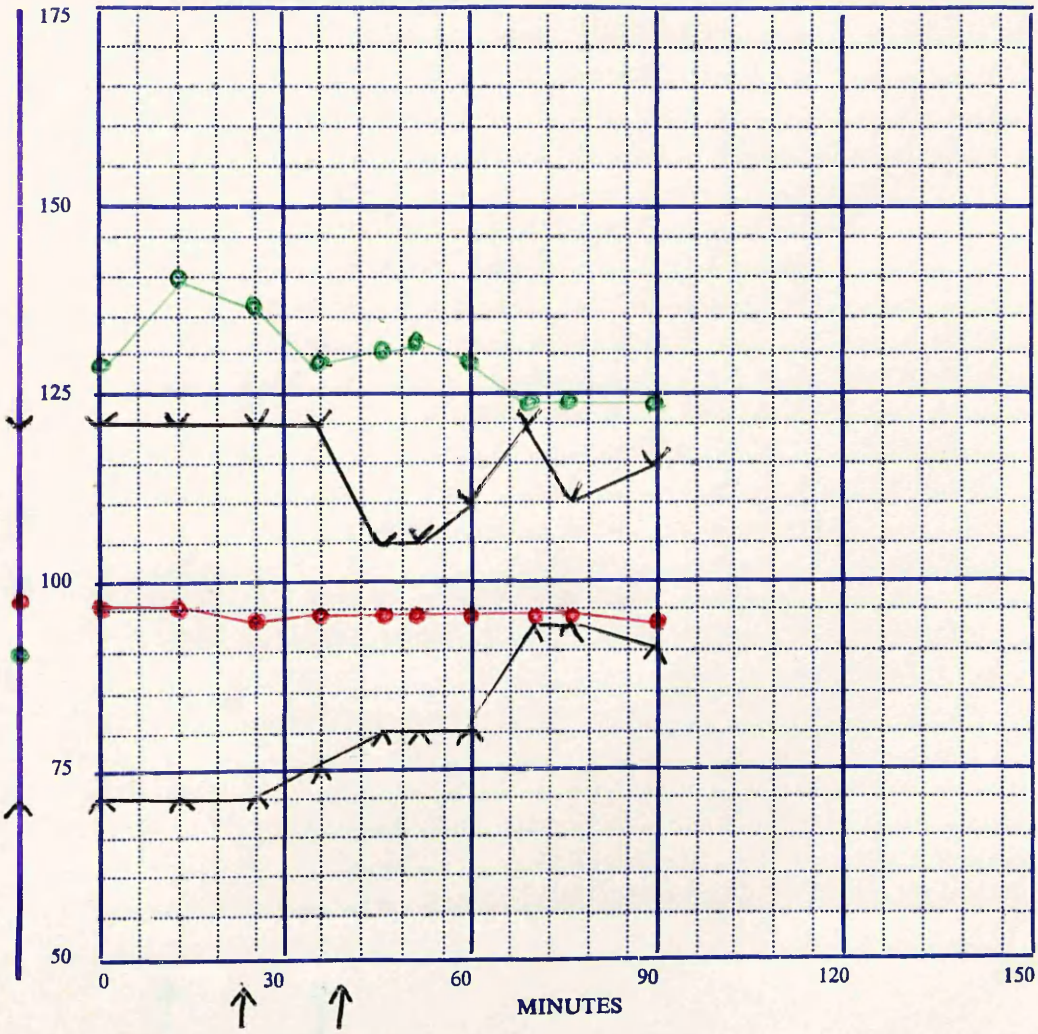
NOTES Bromethol was not available and Paraldehyde was used as a substitute. At the beginning of the catheterisation and at the point indicated the child was restless with a corresponding fall in arterial oxygen saturation.

CASE RECORD No.....24.....

Name B.M. Age 6 Diagnosis C. of A.

Weight 20.5 Kilos. Haemoglobin 93%

Premedication Promethazine 25 mgms.
Bromethol 2.5 c.c.
Pethidine 50 mgms.
Atropine 0.6 mgms.



NOTES Arterial puncture was impossible.
Venous blood analysis pH 7.26
pCO_2 57.6 mmHg.

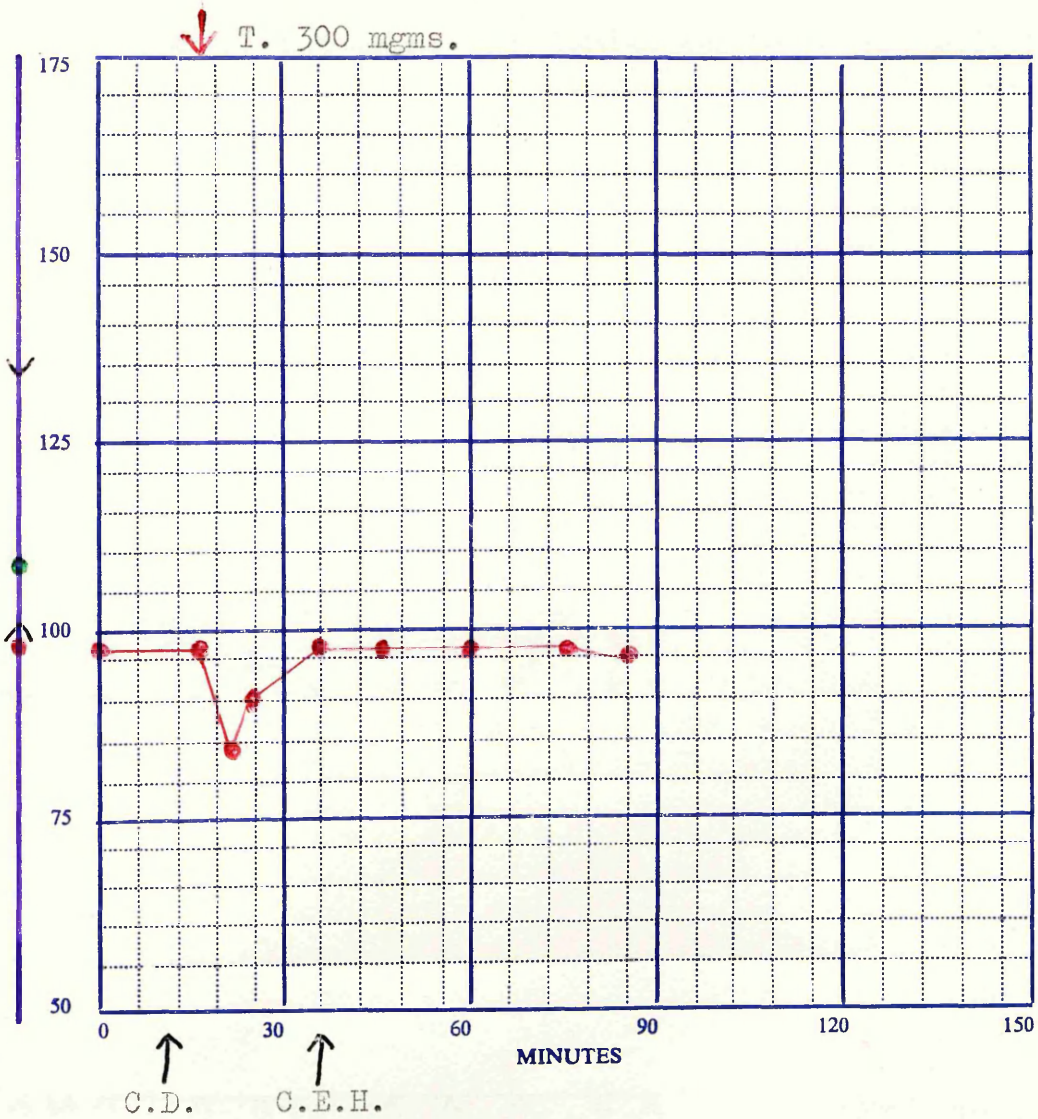
CASE RECORD No. 25

Name V.McD. Age 6 Diagnosis A.S.

Weight 21 Kilos. Haemoglobin 92%

Premedication

Promethazine	25 mgms.
Paraldehyde	85 c.c.
Pethidine	45 mgms.
Atropine	0.6 mgms.



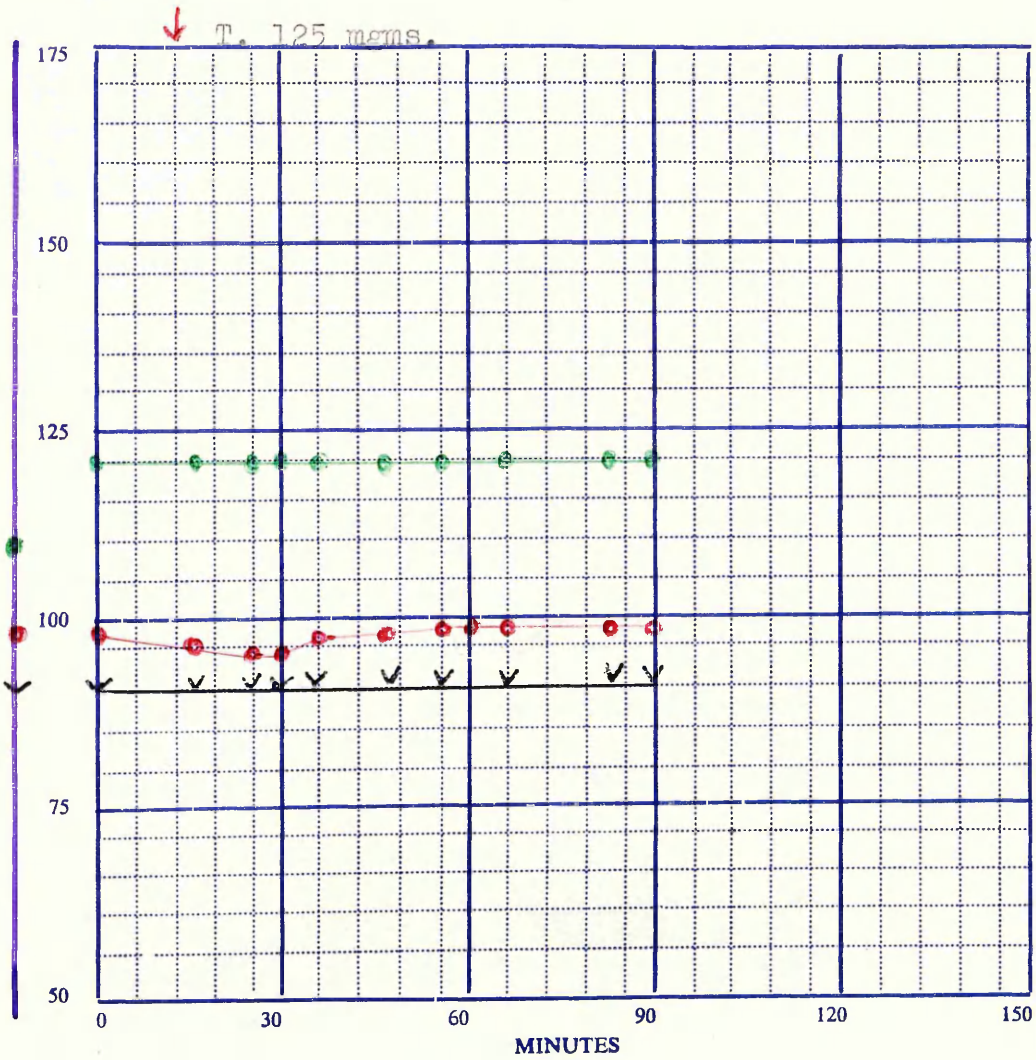
NOTES Only oximeter readings are complete for this patient. Bromethol was not available and Paraldehyde was used as a substitute. At the beginning of the catheterisation the child was restless and Thiopentone was administered to cure this. The dose was clearly excessive.

CASE RECORD No. 26.

Name M.McD. Age 6 Diagnosis D.N.E.

Weight 19 Kilos. Haemoglobin 82%

Premedication Promethazine 10 mgms.
Bromethol 1.8 c.c.
Pethidine 35 mgms.
Atropine 0.6 mgms.



NOTES

C.D. C.E.H.

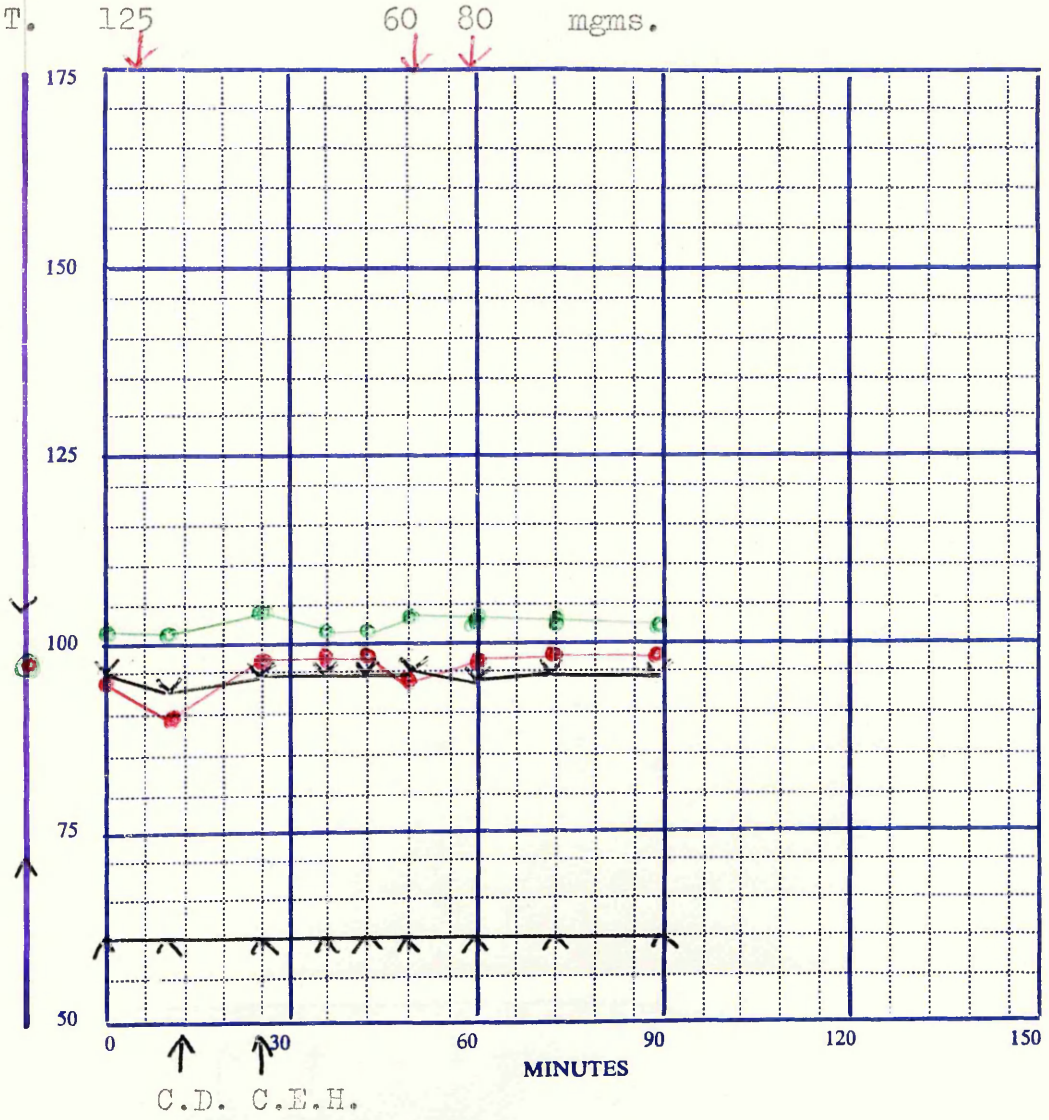
The Thiopentone was administered in response to restlessness. The catheterisation was otherwise uneventful.

CASE RECORD No. 27.

Name S.S. Age 6 Diagnosis I.A.S.D. + P.H.

Weight 15 Kilos. Haemoglobin 114%

Premedication Promethazine 10 mgms.
Bromethol 1.8 c.c.
Pethidine 35 mgms.
Atropine 0.6 mgms.



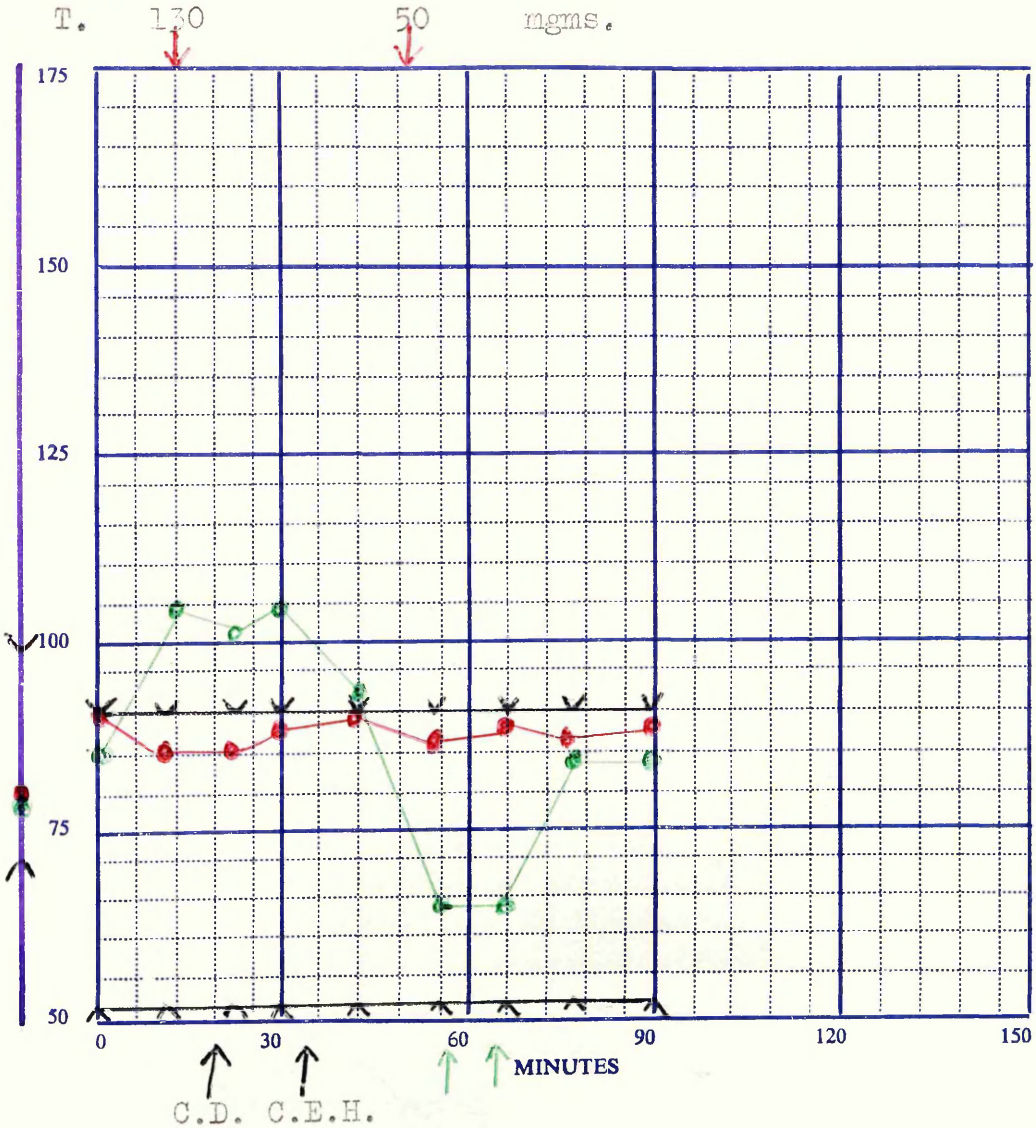
NOTES This child voided some of her enema and arrived in the X-ray Department wide awake. The Thiopentone was administered to keep her asleep.

CASE RECORD No. 28.

Name M.B. Age 6 Diagnosis F.T.

Weight 16.5 Kilos. Haemoglobin 170%

Premedication Promethazine 10 mgms.
Bromethol 1.6 c.c.
Pethidine 30 mgms.
Atropine 0.6 mgms.



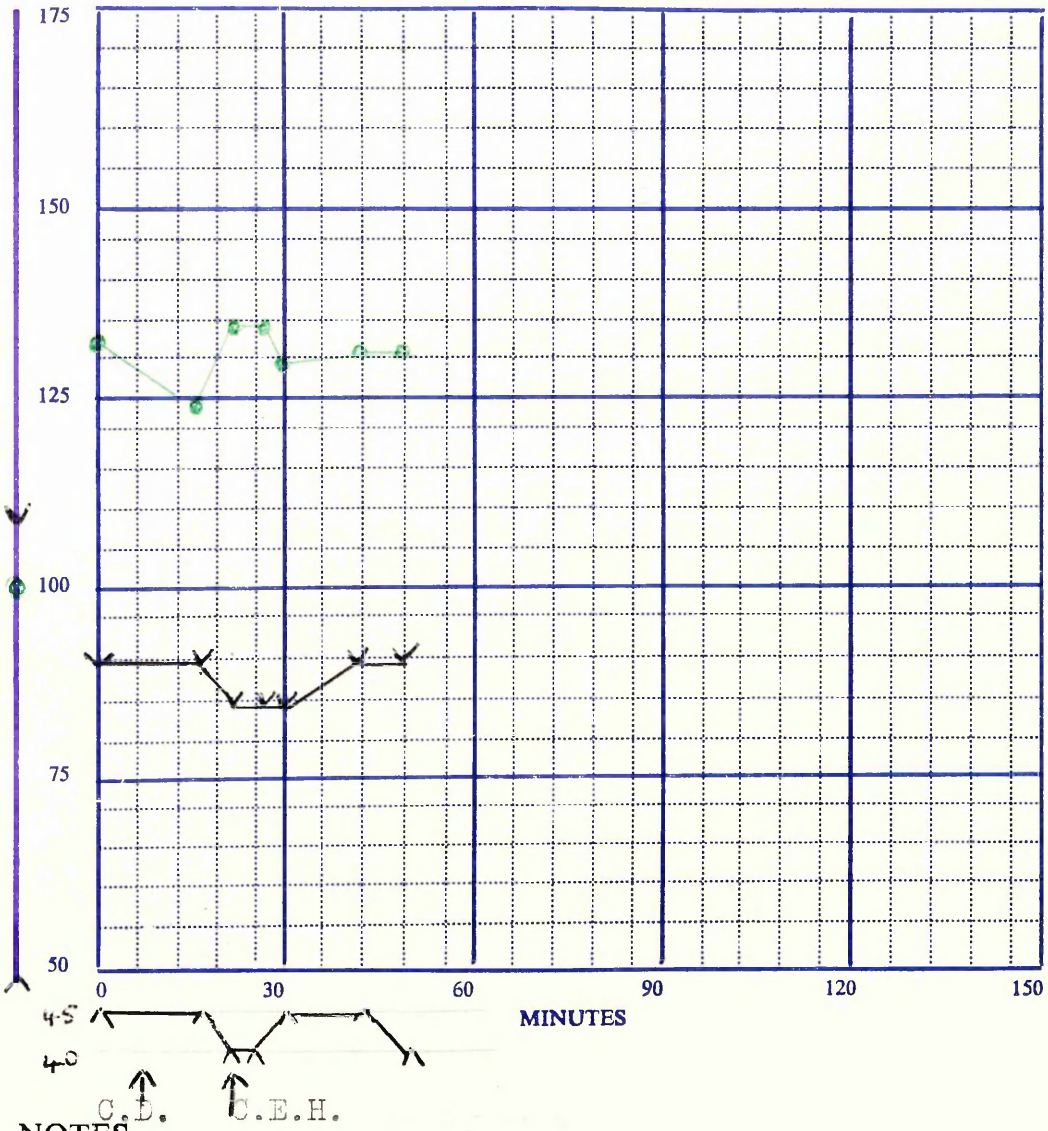
NOTES The green arrows indicate a period of 2 to 1 heart block. The child was markedly less cyanosed on arrival in the X-ray Department than when seen in the ward.
Arterial oxygen saturation 76% (Aorta).
Oximeter reading 81%

CASE RECORD No.....29.....

Name M.S. Age 6½ Diagnosis D.N.E.

Weight 22.5 Kilos. Haemoglobin 90%

Premedication Promethazine 10 mgms.
Bromethol 3 c.c.
Pethidine 50 mgms.
Atropine 0.6 mgms.



NOTES

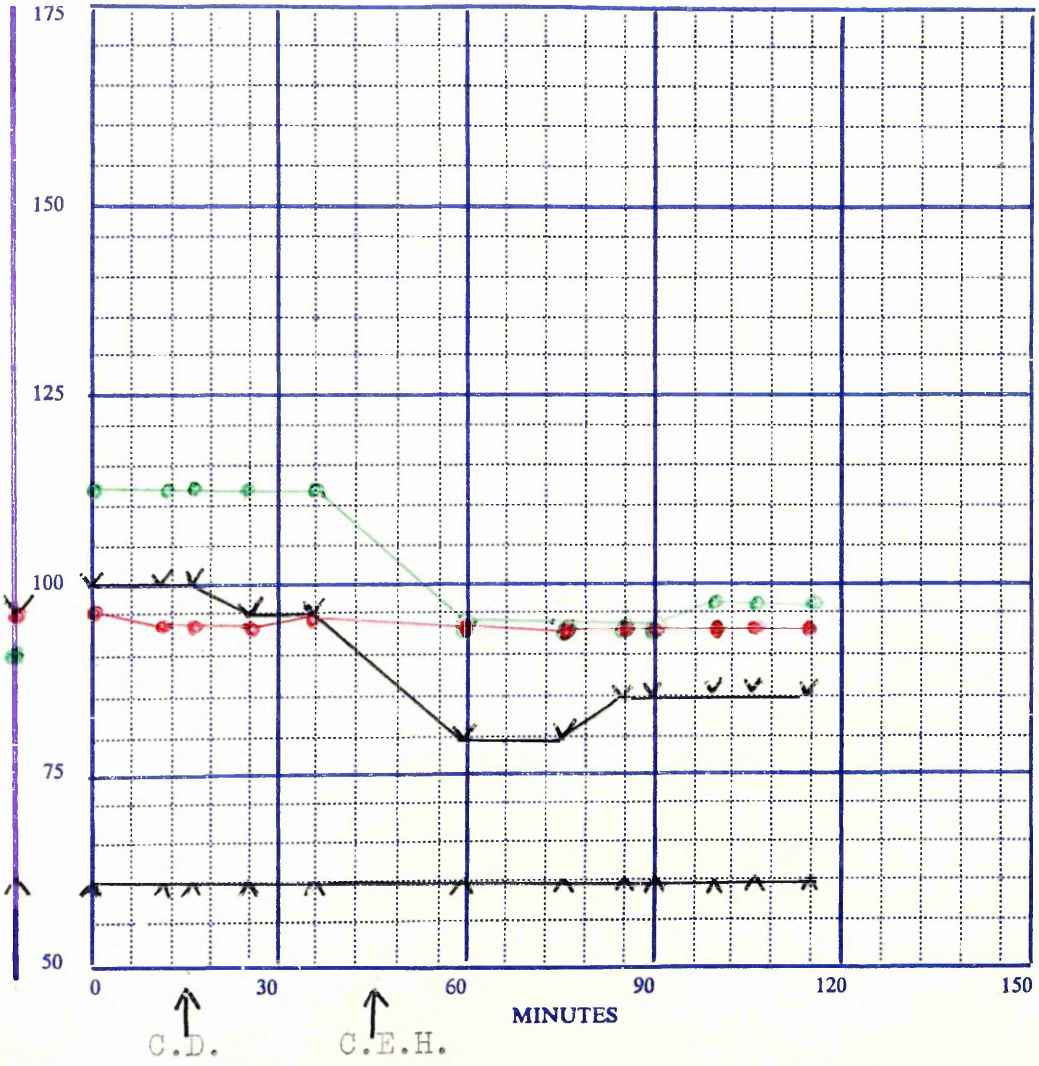
The oximeter ear piece was being repaired and was thus unavailable. The tachycardia was noted before the Bromethol was administered. Samples taken from the Superior Vena Cava had identical oxygen contents at the beginning and end of the catheterisation

CASE RECORD No. 30.

Name J.O'R. Age 7 Diagnosis I.V.S.D.

Weight 19 Kilos. Haemoglobin 100%

Premedication Promethazine 10 mgms.
Bromethol 2.2 c.c.
Pethidine 50 mgms.
Atropine 0.6 mgms.



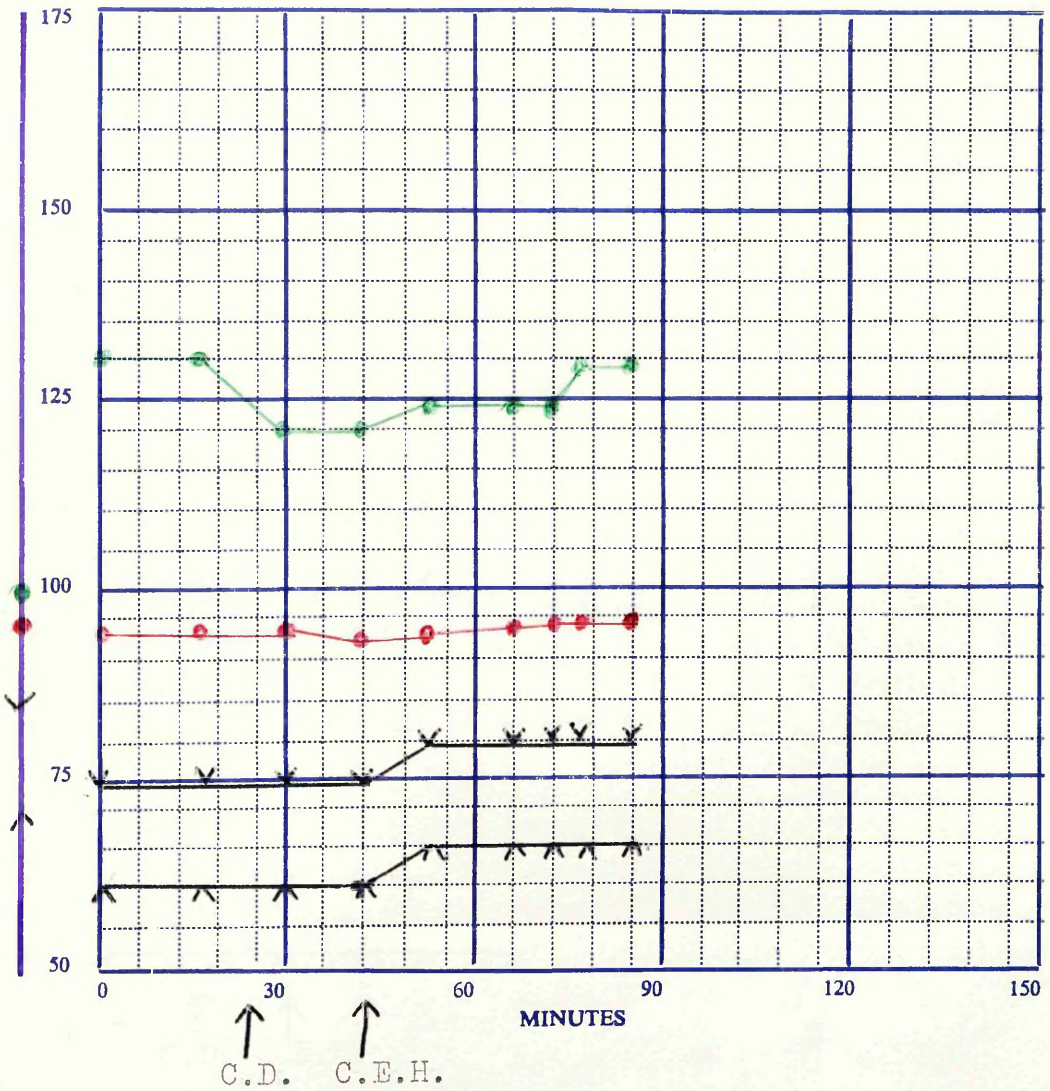
NOTES Arterial analysis - pCO_2 44.6 mmHg.
pH 7.34

CASE RECORD No.....31.....

Name M.F. Age 7 Diagnosis I.A.S.D. + P.H.

Weight 27 Kilos. Haemoglobin 90%

Premedication Promethazine 25 mgms.
Bromethol 3.3 c.c.
Pethidine 60 mgms.
Atropine 0.6 mgms.



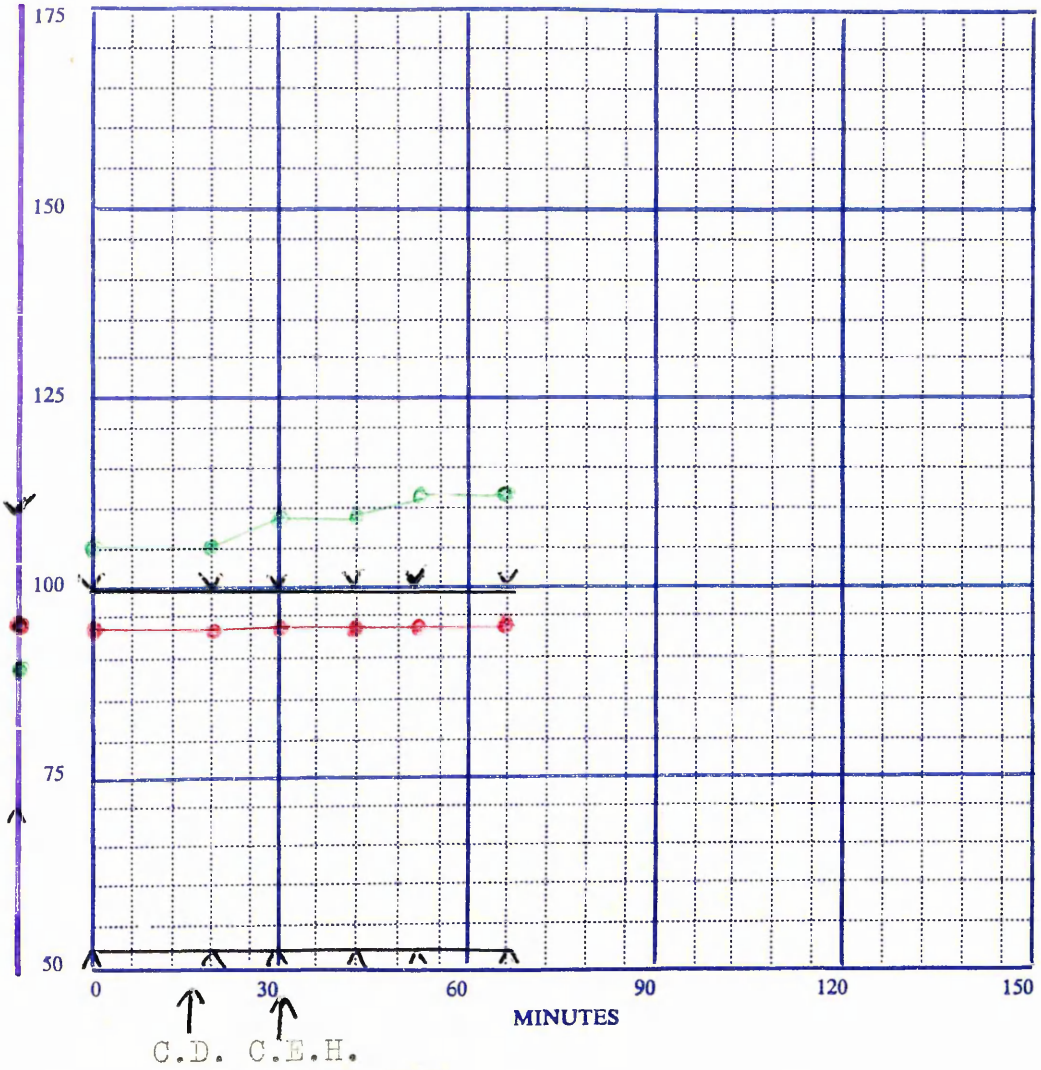
NOTES This was an uneventful catheterisation.

CASE RECORD No. 32.

Name B.B. Age 7 Diagnosis I.A.S.D.

Weight 23 Kilos. Haemoglobin 80%

Premedication Promethazine 25 mgms.
Bromethol 2.25 c.c.
Pethidine 50 mgms.
Atropine 0.6 mgms.



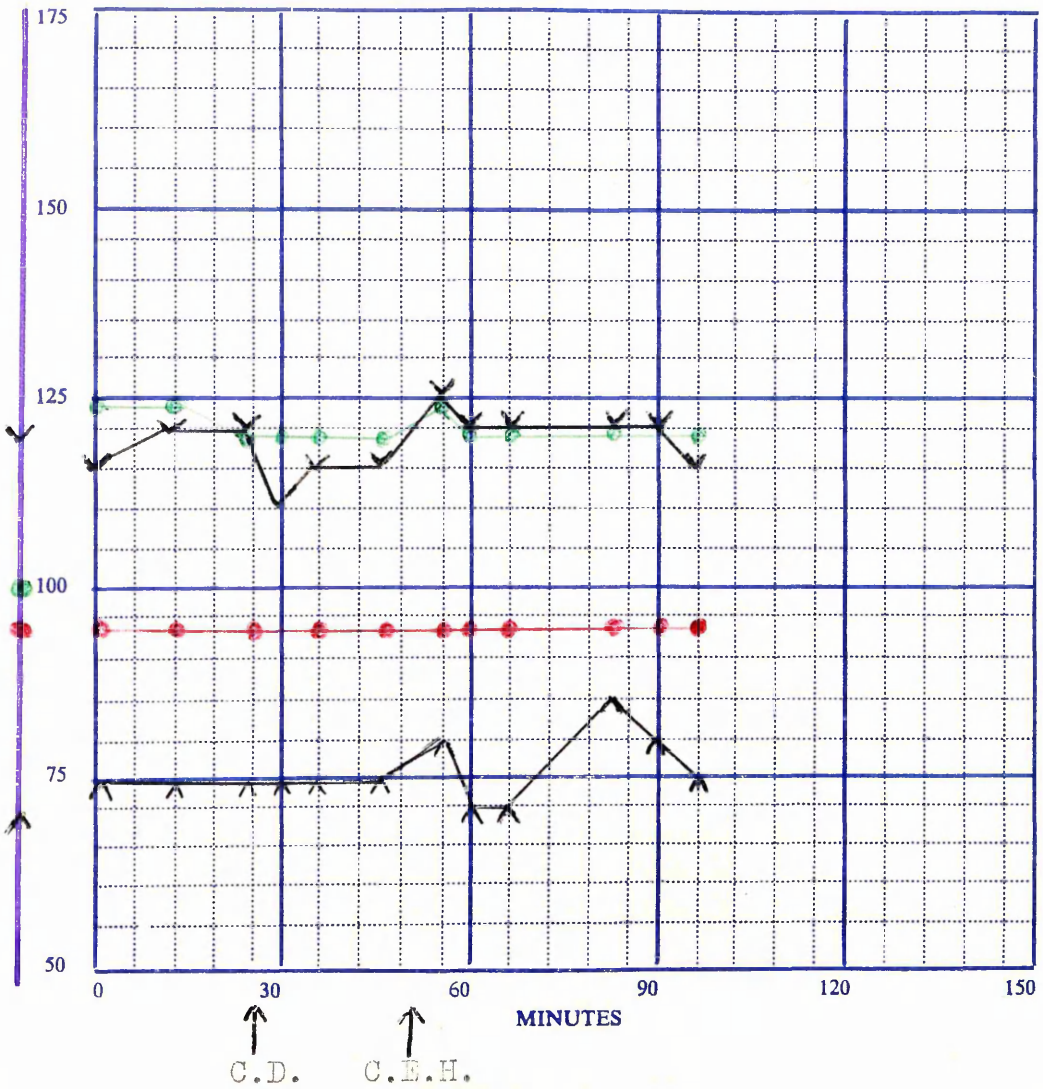
NOTES This was an uneventful catheterisation.

CASE RECORD No.....33.....

Name G.A. Age 7 Diagnosis D.N.E.

Weight 26 Kilos. Haemoglobin 92%

Premedication Promethazine 25 mgms.
Bromethol 3 c.c.
Pethidine 50 mgms.
Atropine 0.6 mgms.



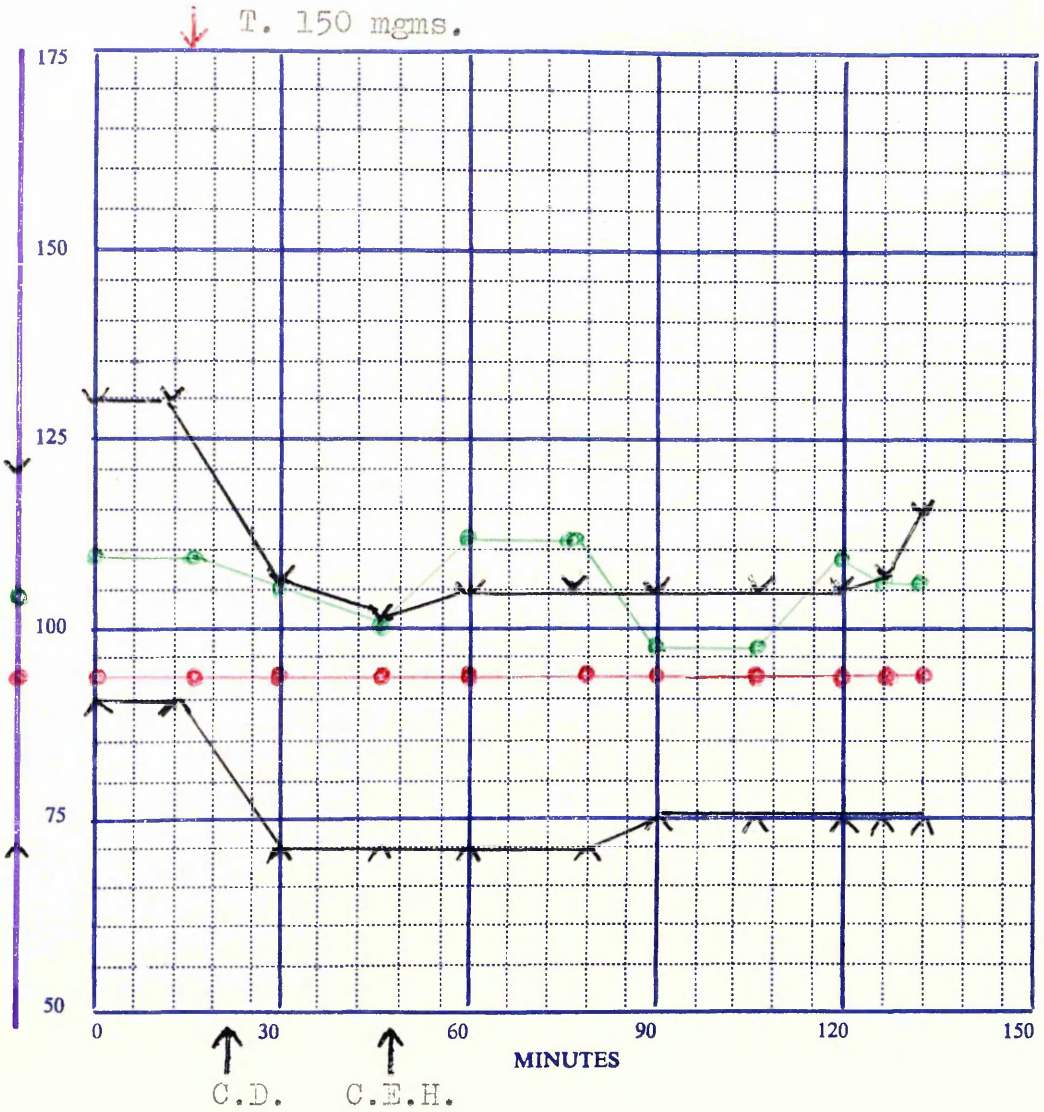
NOTES Arterial analysis - oxygen saturation 95%
(oximeter reading 93%)
pCO_2 48 mmHg.
pH 7.31

CASE RECORD No. 34.

Name M.K. Age 8 Diagnosis I.A.S.D.

Weight 23 Kilos. Haemoglobin 105%

Premedication Promethazine 25 mgms.
Bromethol 2.7 c.c.
Pethidine 50 mgms.
Atropine 0.6 mgms.



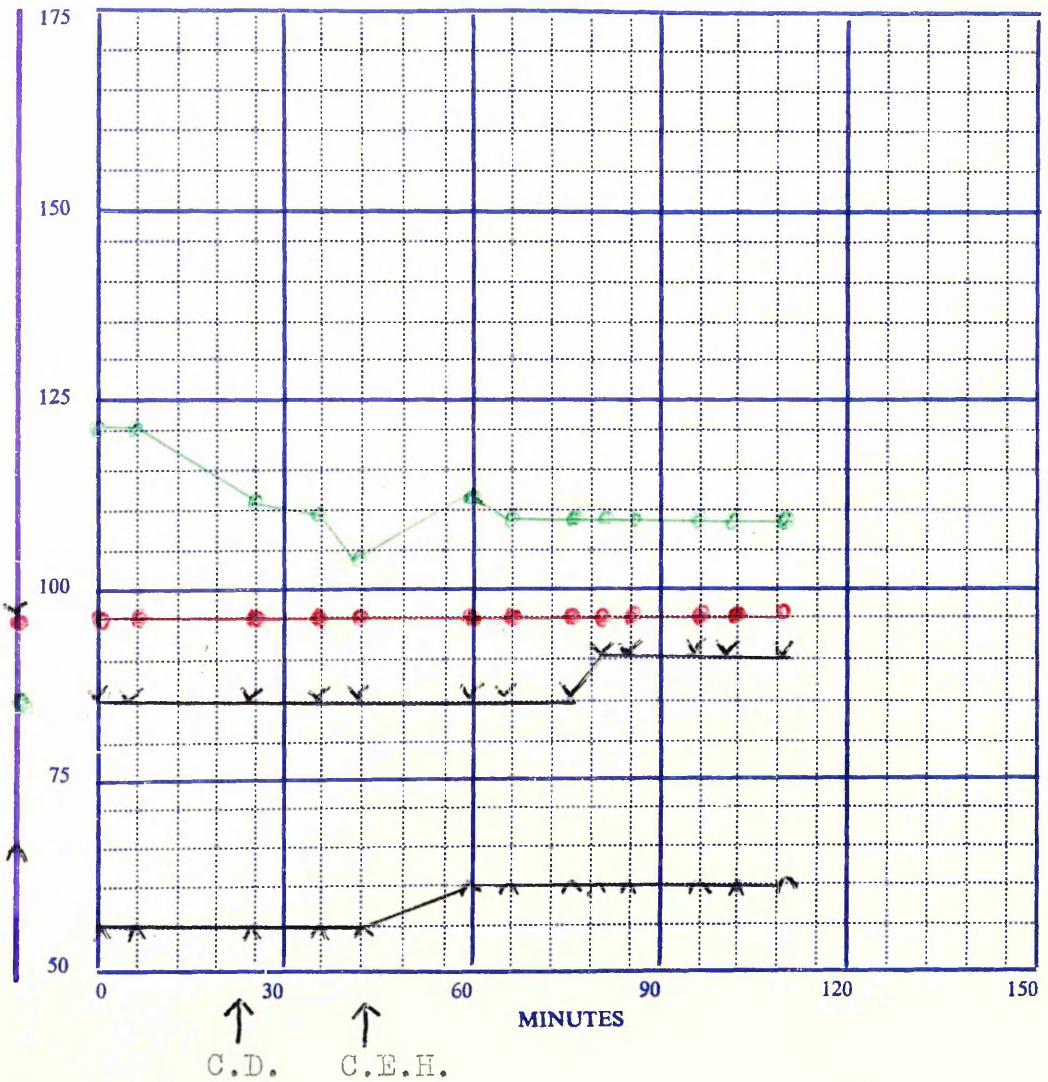
NOTES The child voided about 20% of her Bromethol and though drowsy whimpered on arrival in the X-ray room. She settled after administration of the Thiopentone. The change in blood pressure from the waking to the sleeping state is notable.

CASE RECORD No.....35.....

Name R.H. Age 8 Diagnosis I.A.S.D.

Weight 25.5 Kilos. Haemoglobin 105%

Premedication Promethazine 25 mgms.
Bromethol 3 c.c.
Pethidine 50 mgms.
Atropine 0.6 mgms.



NOTES Arterial analysis - oxygen saturation 92.5%
(oximeter reading 95%)
pCO_2 49.5 mmHg.
pH 7.27

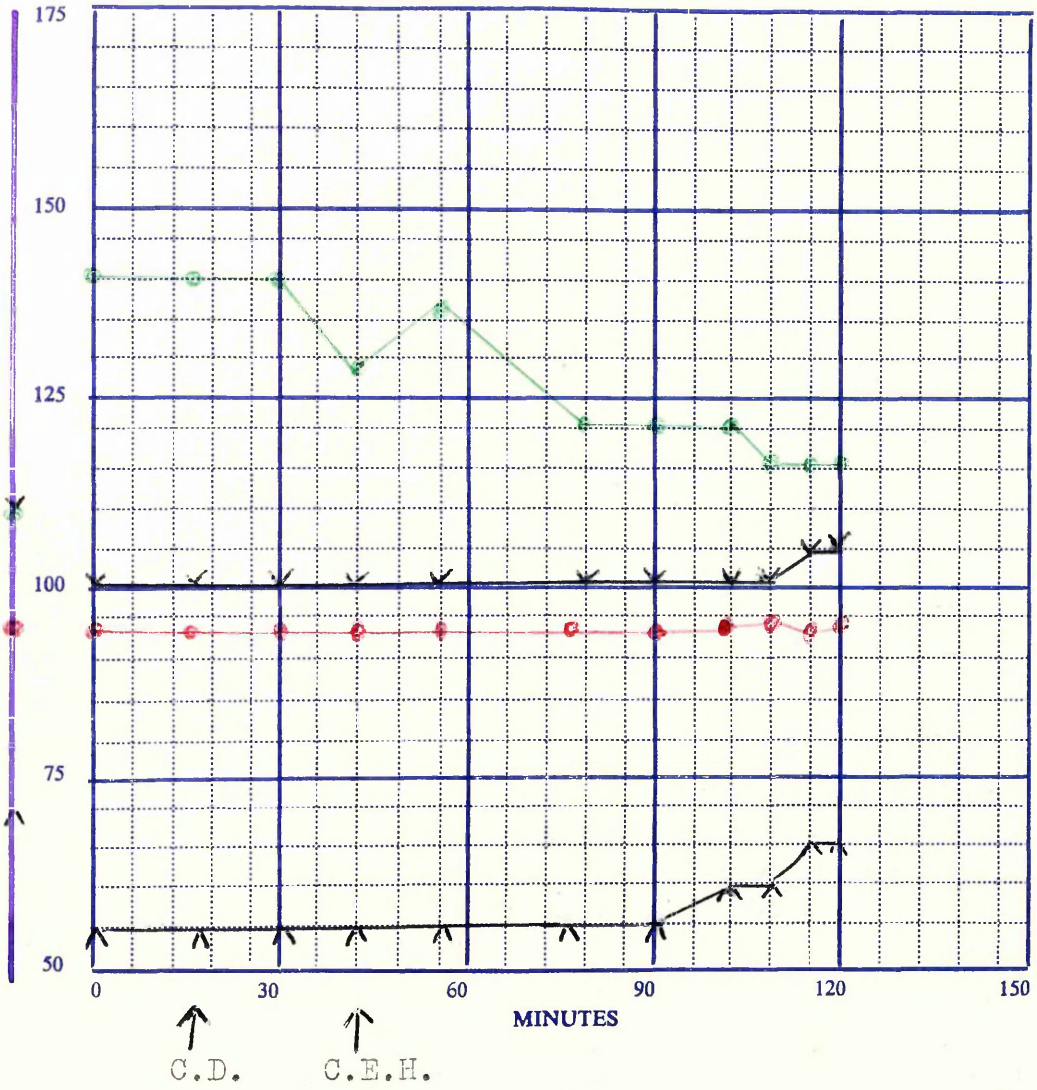
This child had chronic nasal catarrh and an oral airway was used.

CASE RECORD No. 36.

Name M.K. Age 8 Diagnosis P.D.A.

Weight 25.5 Kilos. Haemoglobin 96%

Premedication Promethazine 25 mgms.
Bromethol 3 c.c.
Pethidine 50 mgms.
Atropine 0.6 mgms.



NOTES Arterial analysis - oxygen saturation 90.5%
(oximeter reading 93%)

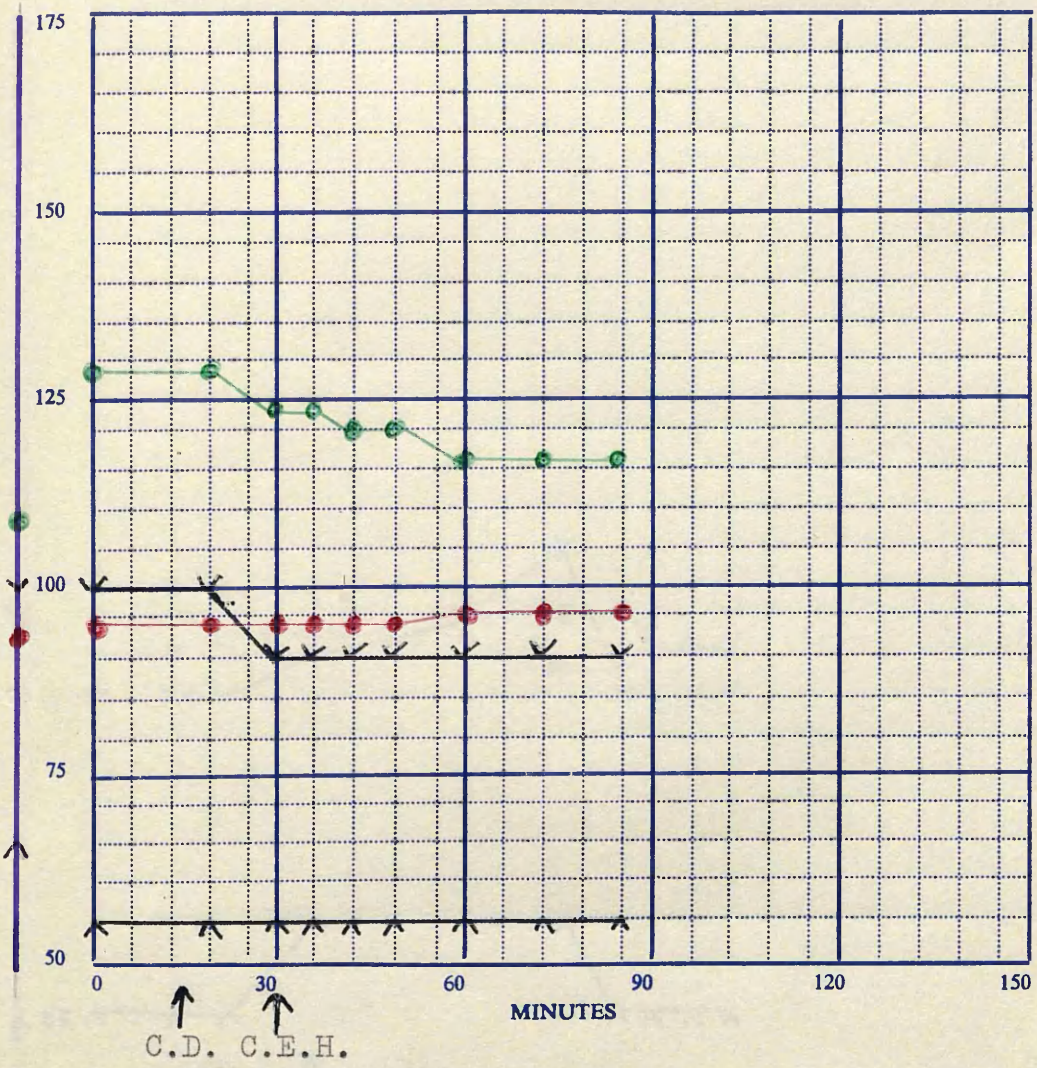
This child is a mongol and subject to chronic nasal catarrh. An oral airway was used.

CASE RECORD No.....37.....

Name W.McN. Age 8 Diagnosis I.V.S.D.

Weight 22.5 Kilos. Haemoglobin 102%

Premedication Promethazine 25 mgms.
Bromethol 2.5 c.c.
Pethidine 50 mgms.
Atropine 0.6 mgms.



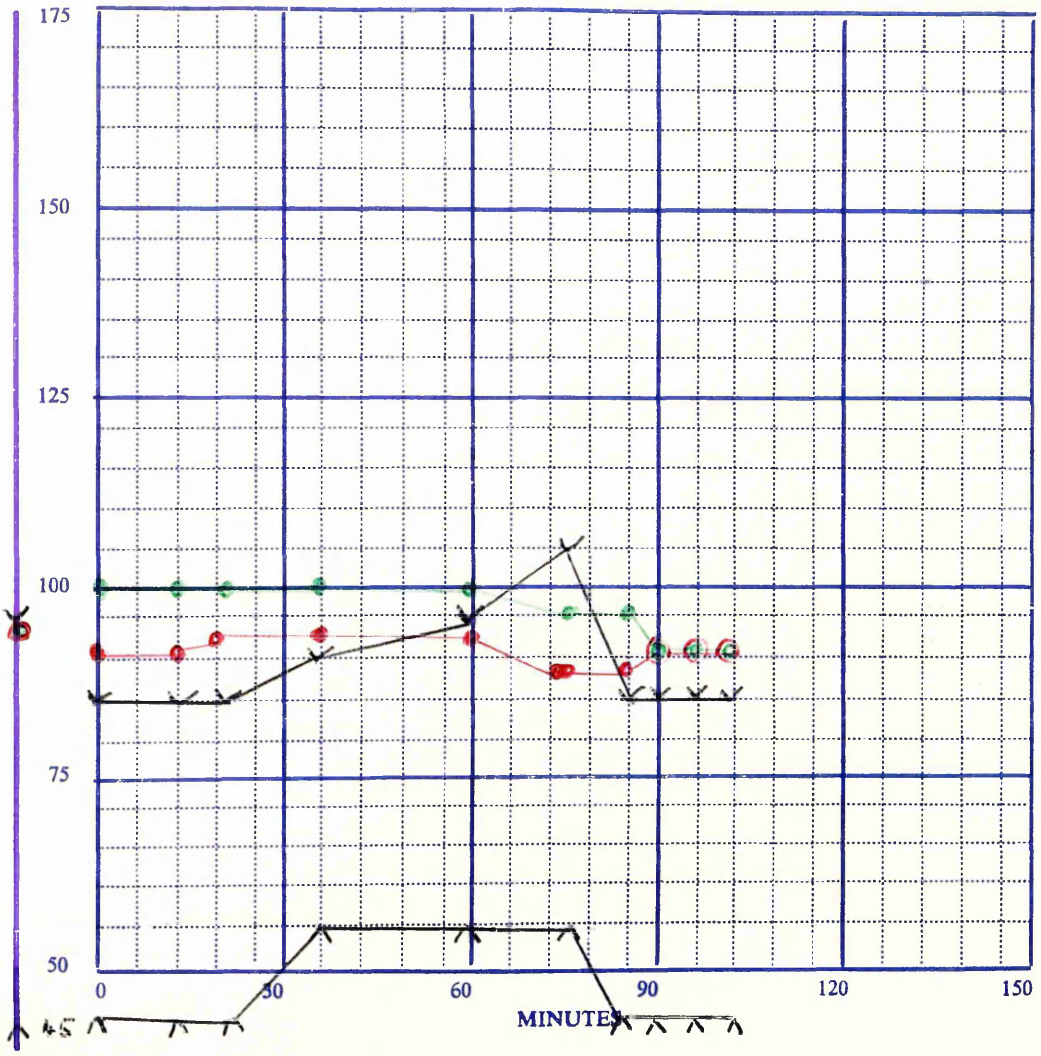
NOTES This child had residual nasal congestion following a cold and required an oral airway.

CASE RECORD No.....38.....

Name L.McS. Age 8 Diagnosis P.S.

Weight 25 Kilos. Haemoglobin 92%

Premedication Promethazine 10 mgms.
Bromethol 3 c.c.
Pethidine 50 mgms.
Atropine 0.6 mgms.



NOTES The drop in saturation in the latter part of the examination was associated with the presence of the catheter in the pulmonary valve.

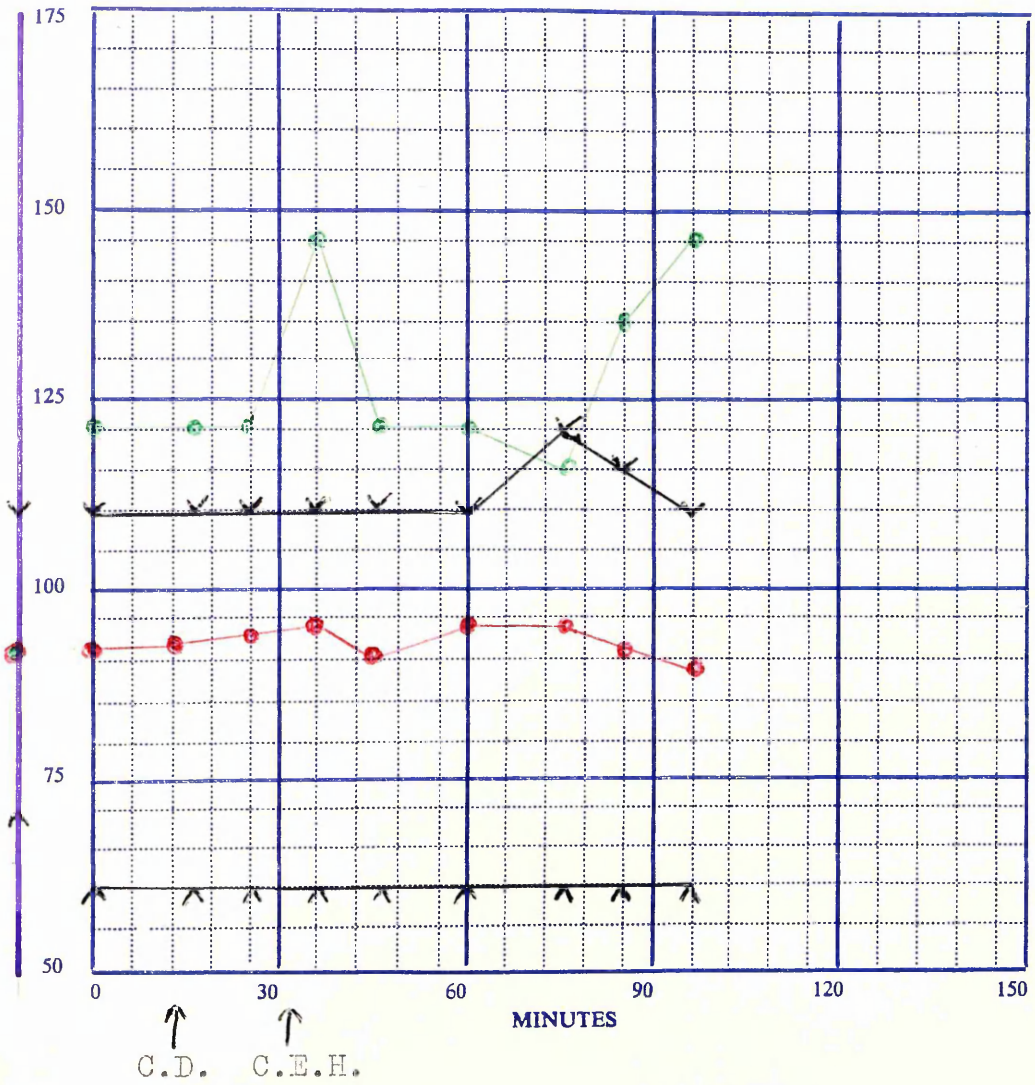
CASE RECORD No. 39.

Name E.McL. Age 8 Diagnosis P.S.

Weight 24 Kilos. Haemoglobin 105%

Premedication Promethazine 25 mgms.
Bromethol 2.5 c.c.
Pethidine 20 mgms.
Atropine 0.6 mgms.

T. 75 75 125 75 mgms.



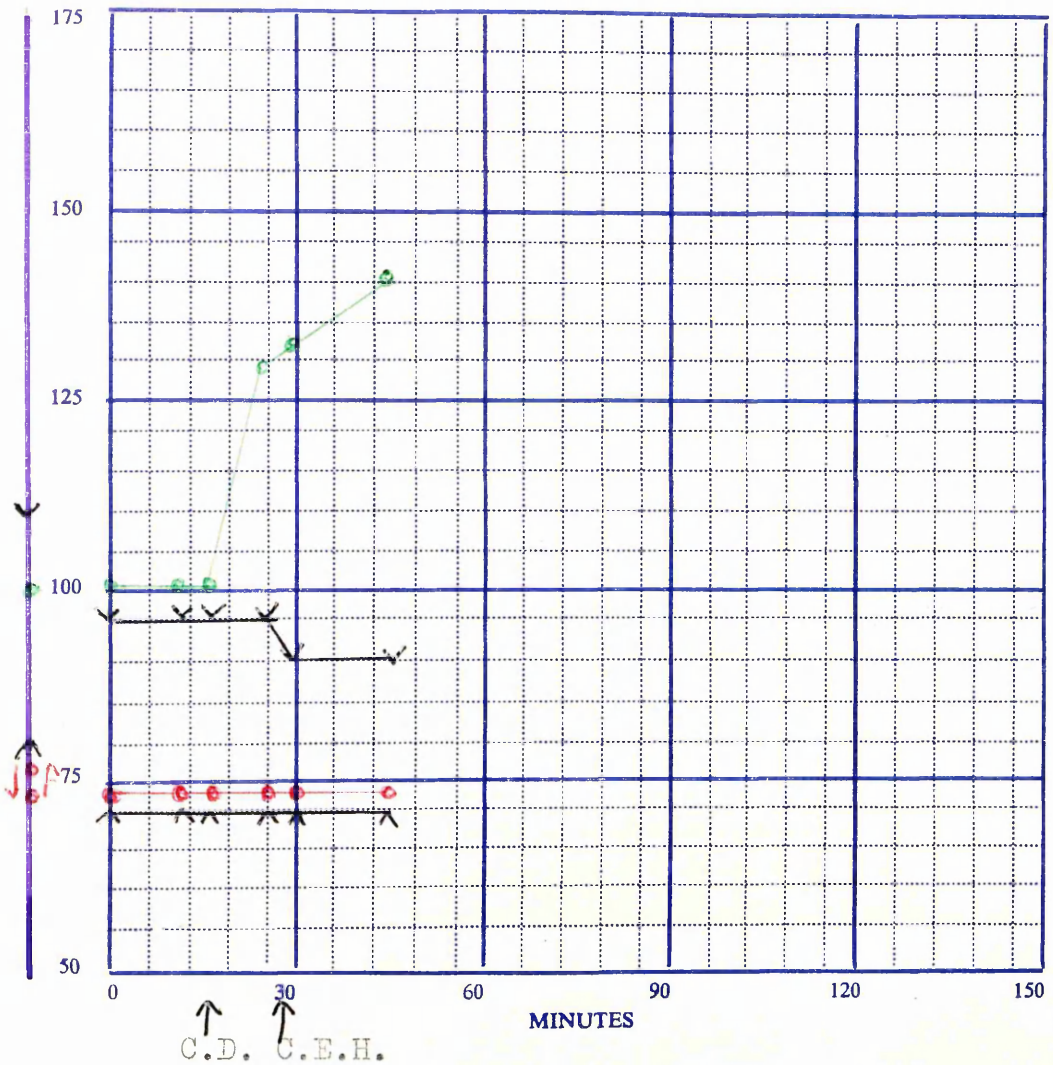
NOTES This child was inadequately sedated on a reduced dose of Bromethol. She was given the Thiopentone to combat restlessness.

CASE RECORD No. 40.

Name M.B. Age 8 Diagnosis F.T.

Weight 21 Kilos. Haemoglobin 135%

Premedication Promethazine 25 mgms.
Bromethol 2.5 c.c.
Pethidine 45 mgms.
Atropine 0.6 mgms.



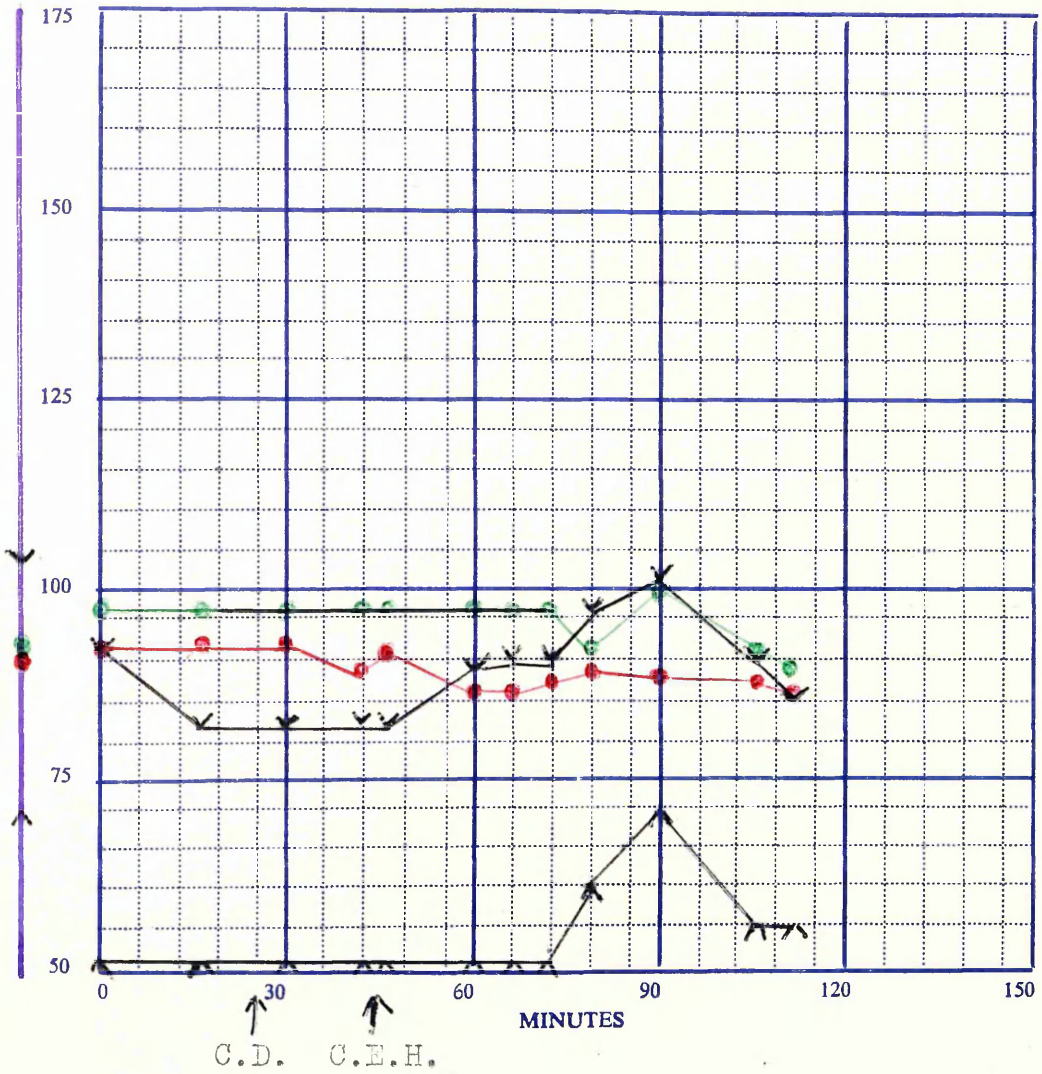
NOTES This is a repeat catheterisation of case 28. Despite an increase in the dose of Bromethol the child remained only pleasantly sleepy but gave no trouble during the catheterisation. The rise in pulse rate was associated with a great deal of Manipulation.

CASE RECORD No. 41.

Name M.M. Age 8½ Diagnosis I.A.S.D.

Weight 23.5 Kilos. Haemoglobin 100%

Premedication Promethazine 25 mgms.
Bromethol 2.8 c.c.
Pethidine 50 mgms.
Atropine 0.6 mgms.



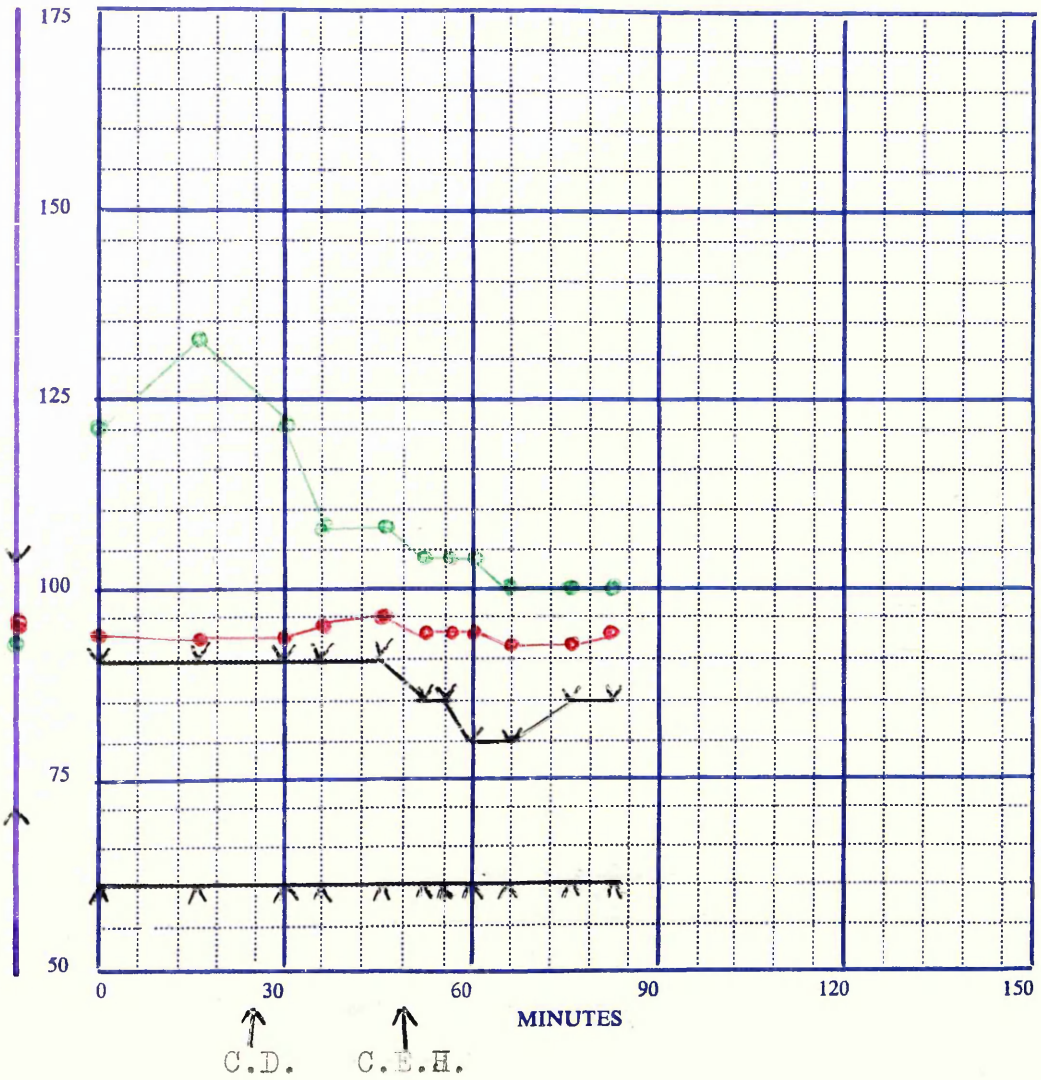
NOTES There was no obvious explanation for the fluctuation in oxygen saturation other than a change in haemodynamics resulting from the passage of the catheter into the left side of the heart.

CASE RECORD No.....42.....

Name D.H. Age 9 Diagnosis I.V.S.D.

Weight 23 Kilos. Haemoglobin 90%

Premedication Promethazine 25 mgms.
Bromethol 3.5 c.c.
Pethidine 50 mgms.
Atropine 0.6 mgms.



NOTES Arterial analysis - pCO_2 46.5 mmHg
pH 7.34

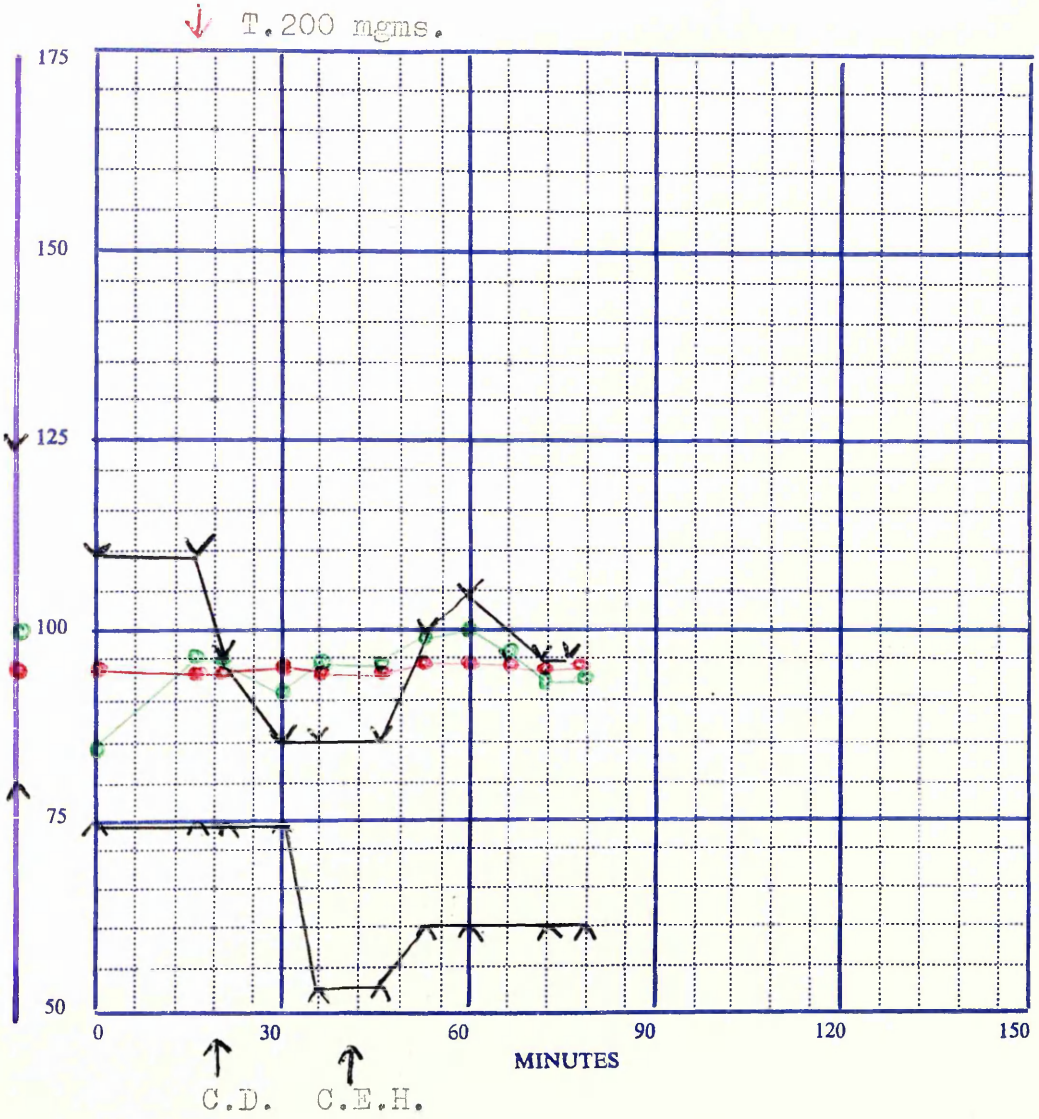
This child was deeply sedated and required an oral airway.

CASE RECORD No. 43.

Name G.G. Age 9 Diagnosis M.S.

Weight 34 Kilos. Haemoglobin 92%

Premedication Promethazine 25 mgms.
Bromethol 3.5 c.c.
Pethidine 75 mgms.
Atropine 0.6 mgms.



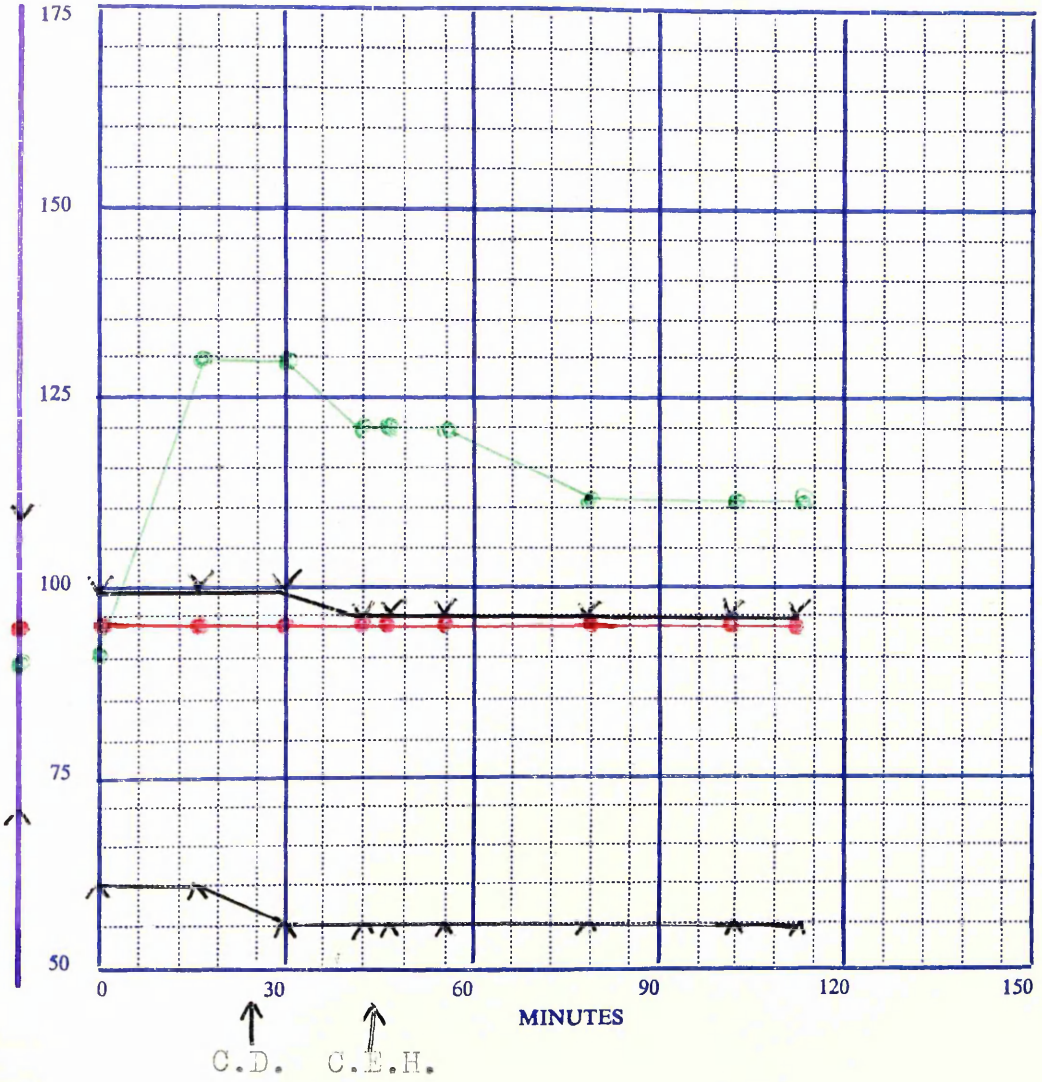
NOTES The child arrived at the the X-ray department partly awake and it was suggested that some of the Bromethol had been voided. This may be so but she had been given a smaller dose of Bromethol in view of her fixed cardiac output. Samples taken from the right atrium before and after the Thiopentone tallied to within 0.2 vols. oxygen.

CASE RECORD No. 44.

Name H.F. Age 9 Diagnosis V.S.D. + P.H.

Weight 19.5 Kilos. Haemoglobin 98%

Premedication Promethazine 25 mgms.
Bromethol 2.5 c.c.
Pethidine 50 mgms.
Atropine 0.6 mgms.



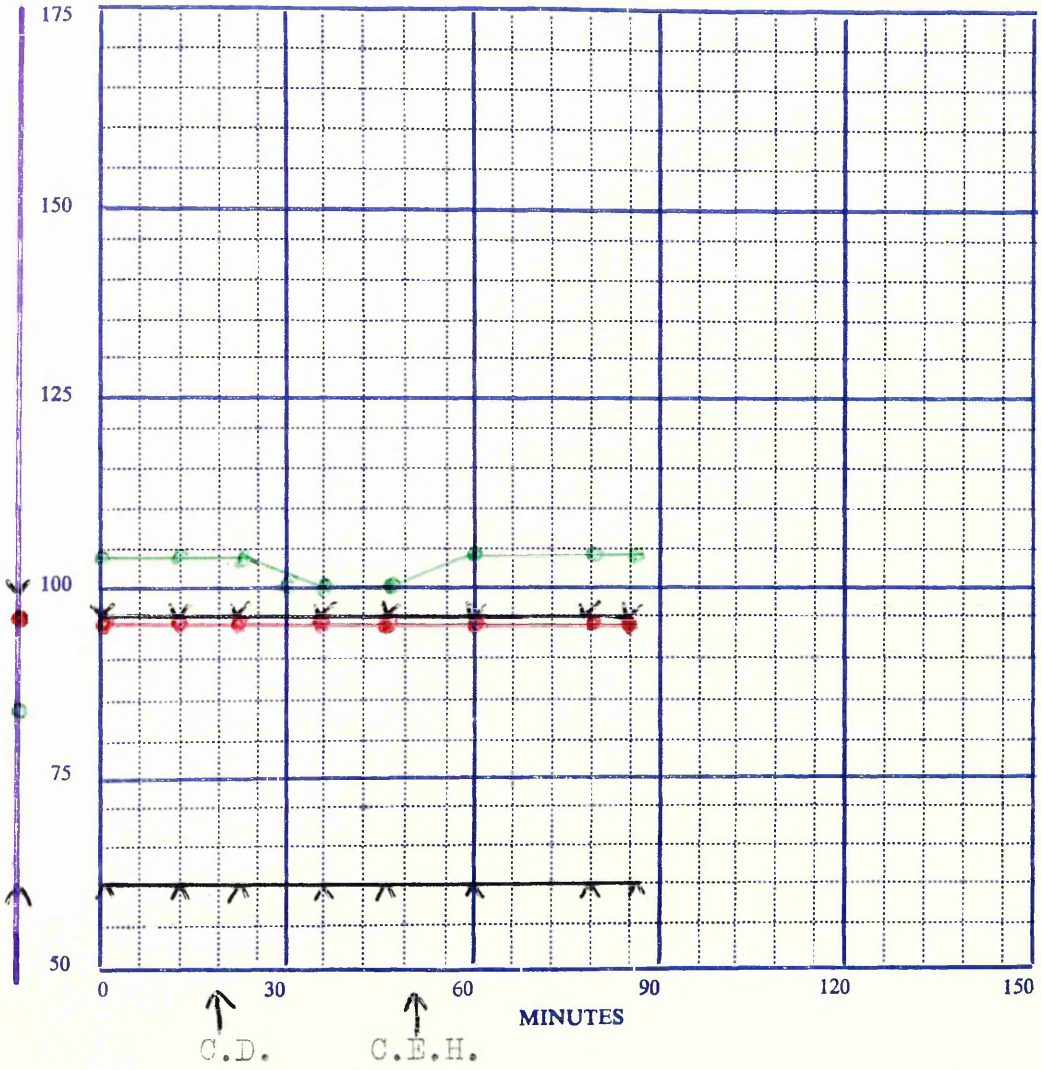
NOTES Arterial analysis - pCO_2 47.8 mmHg.
pH 7.28

CASE RECORD No.....45.....

Name T.McC. Age 9 Diagnosis I.A.S.D.

Weight 25.5 Kilos. Haemoglobin 95%

Premedication Promethazine 25 mgms.
Bromethol 3 c.c.
Pethidine 50 mgms.
Atropine 0.6 mgms.



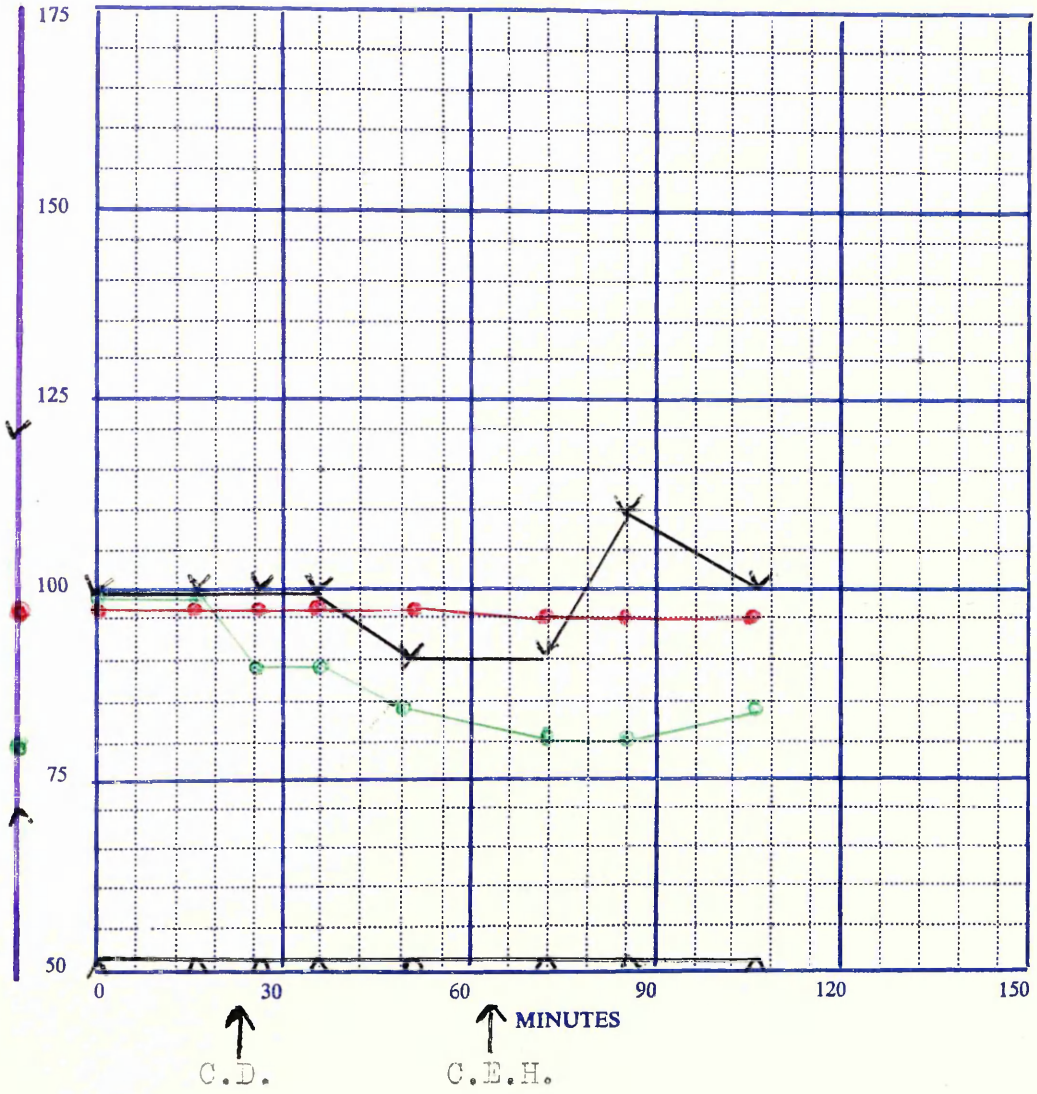
NOTES This was an uneventful catheterisation.

CASE RECORD No.....46.....

Name R.J. Age 9 Diagnosis P.S.

Weight 24 Kilos. Haemoglobin 100%

Premedication Promethazine 25 mgms.
Brompthol 3 c.c.
Pethidine 50 mgms.
Atropine 0.6 mgms.



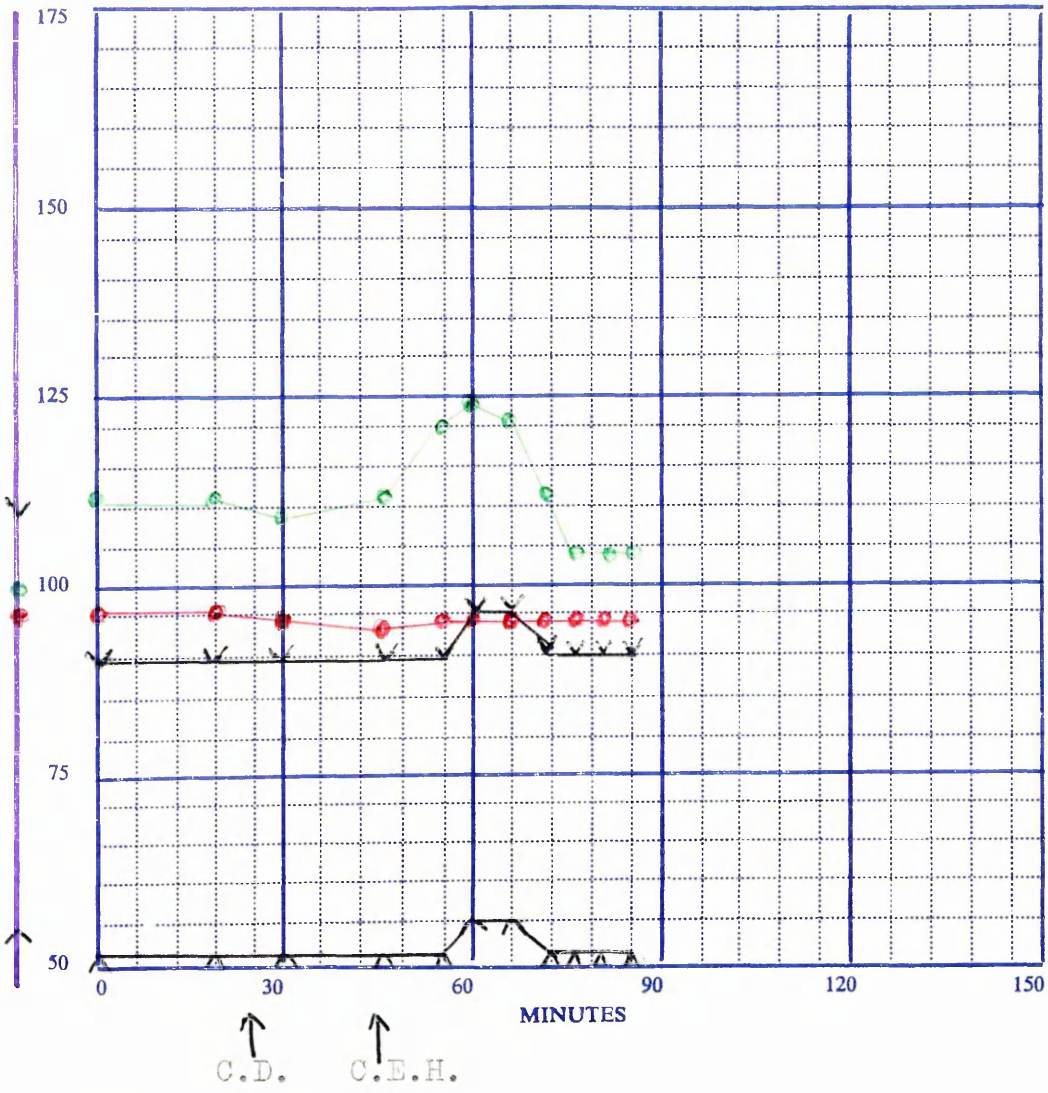
NOTES This catheterisation was unsuccessful and patient moved during the investigation and was awake at the end.

CASE RECORD No. 47.

Name A.C. Age 10 Diagnosis P.D.A.

Weight 40 Kilos. Haemoglobin 95%

Premedication Promethazine 25 mgms.
Bromethol 5 c.c.
Pethidine 50 mgms.
Atropine 0.6 mgms.



NOTES Arterial analysis - oxygen saturation 95.5%
(oximeter reading 94%

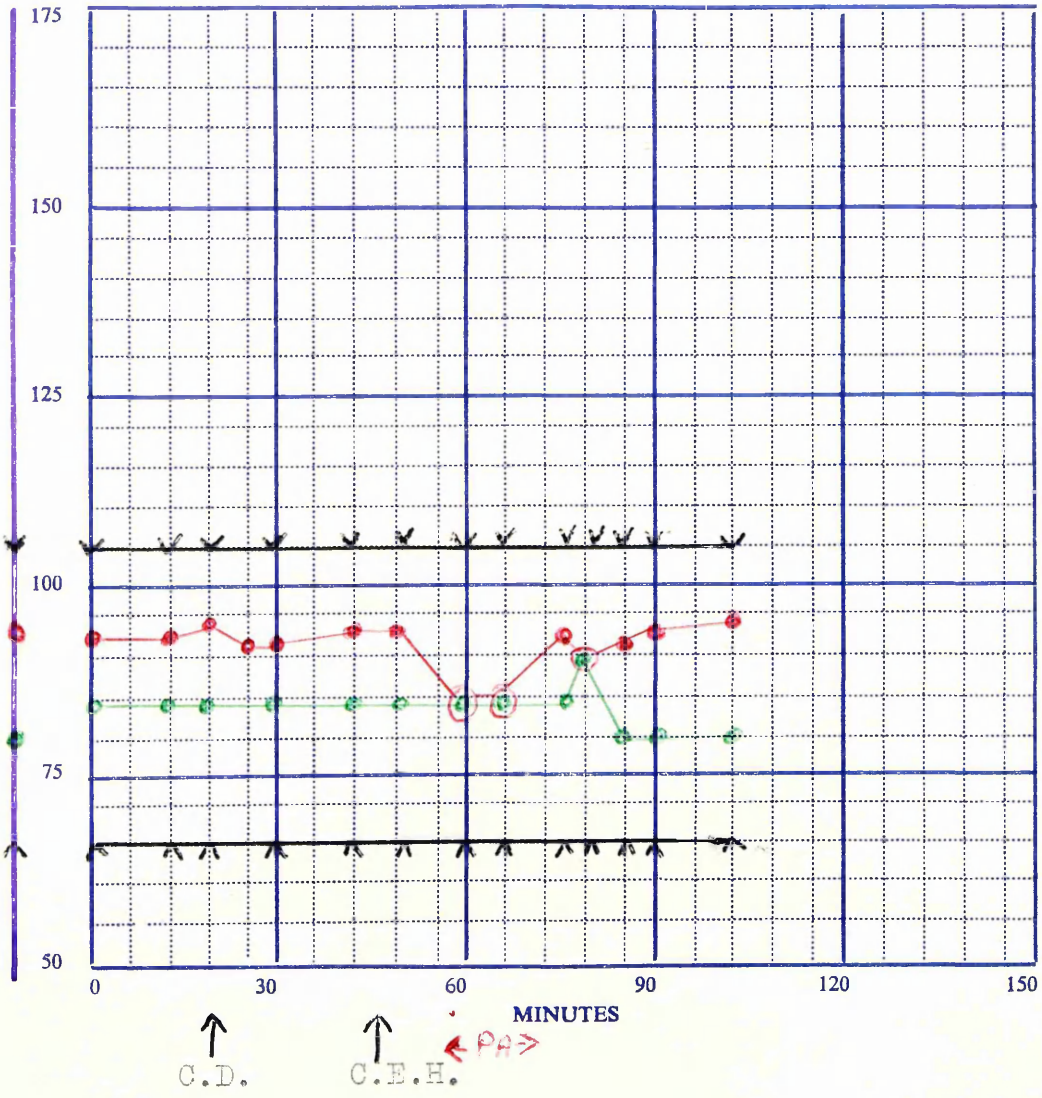
pCO_2 51.2 mmHg.
pH 7.27

CASE RECORD No. 48.

Name W.C. Age 10 Diagnosis P.S.

Weight 25.5 Kilos. Haemoglobin 98%

Premedication Promethazine 25 mgms.
Bromethol 2.8 c.c.
Pethidine 55 mgms.
Atropine 0.6 mgms.



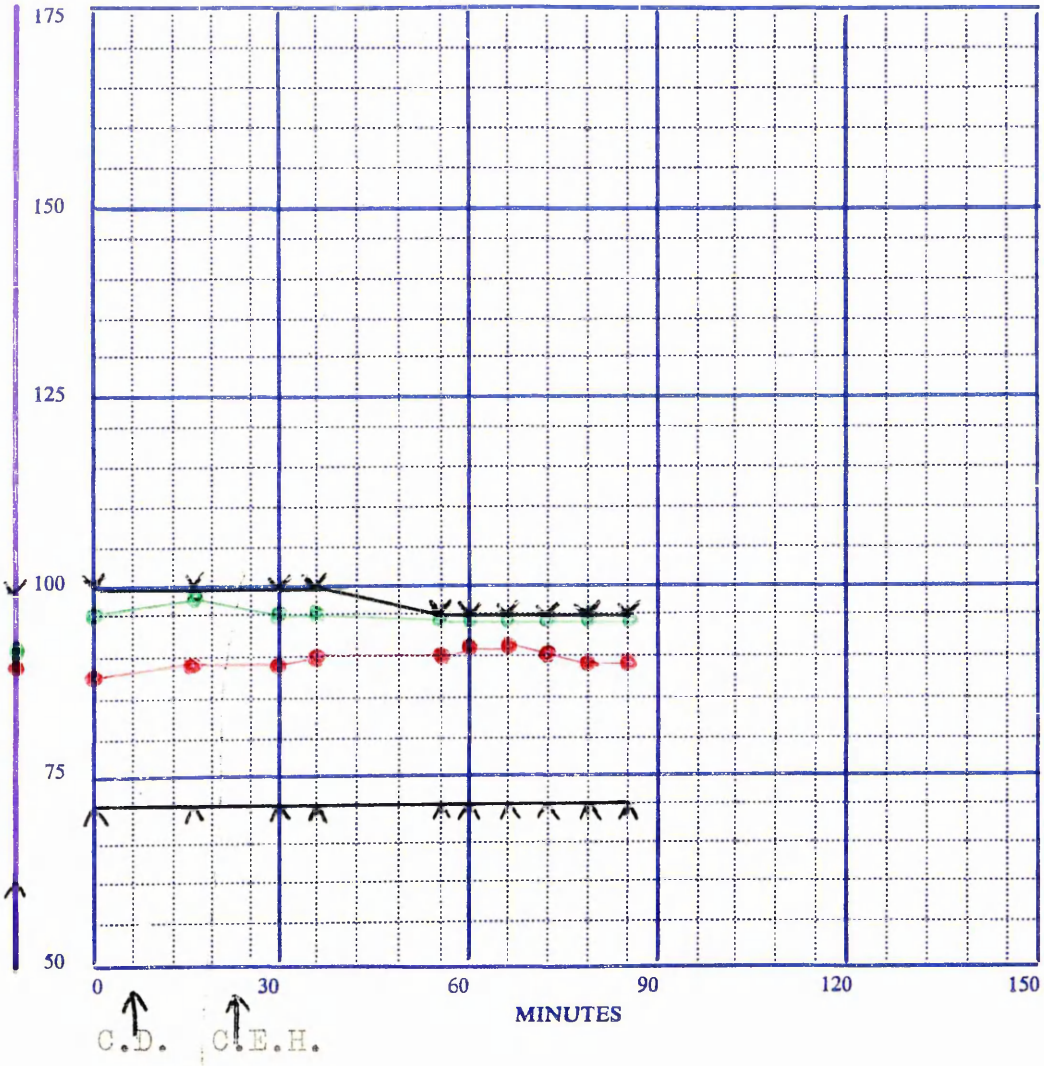
NOTES At the point indicated by the red arrows the catheter was in the main pulmonary artery. The fall in oxygen saturation accompanying the blocking of the pulmonary valve can be clearly seen.

CASE RECORD No. 49.

Name J.M.H. Age 10 Diagnosis F.T.+ L.S.V.C.

Weight 25.5 Kilos. Haemoglobin 128%

Premedication Promethazine 25 mgms.
Bromethol 2.6 c.c.
Pethidine 55 mgms.
Atropine 0.6 mgms.



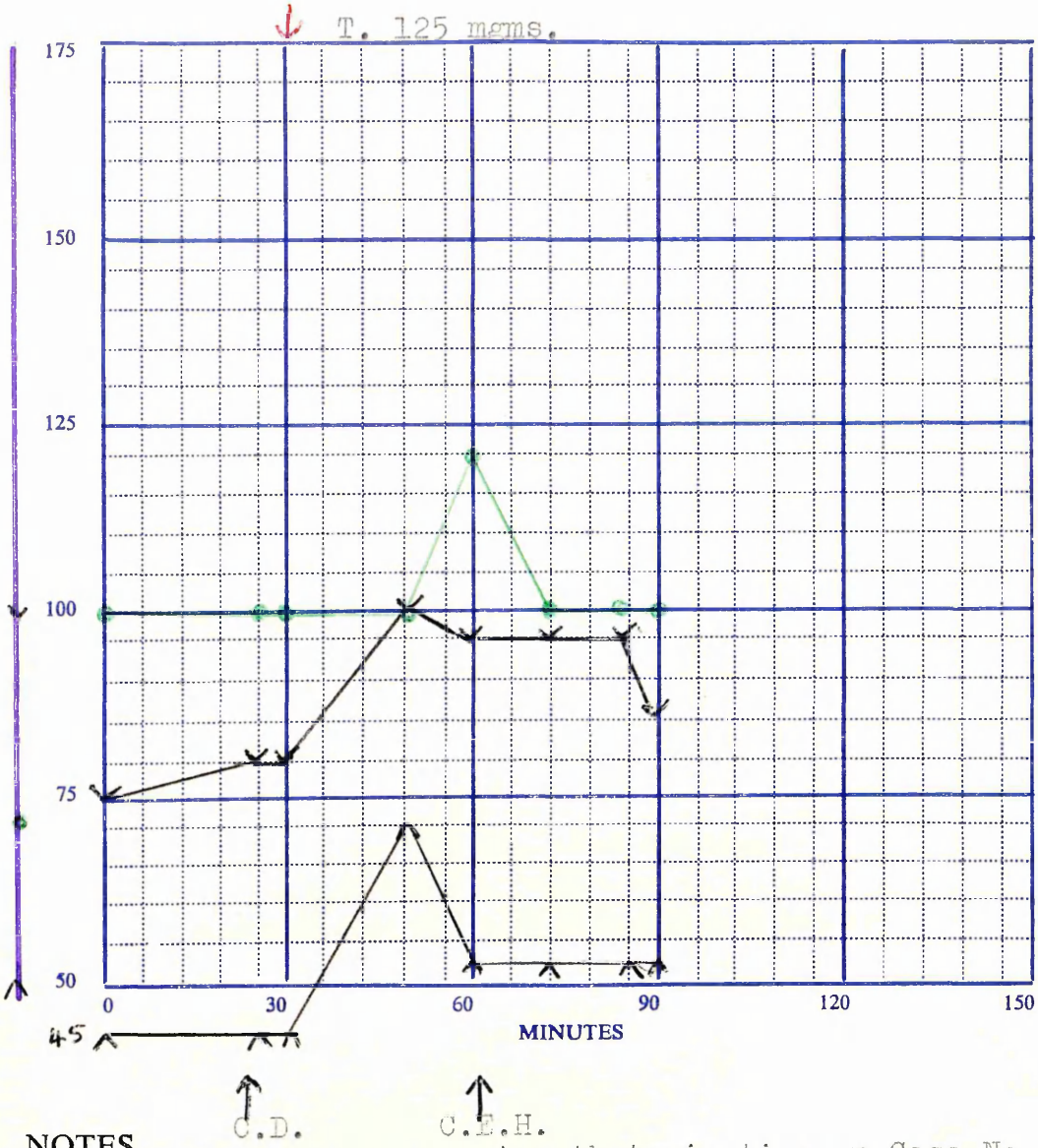
NOTES This was an uneventful catheterisation in a potentially cyanosed patient.

CASE RECORD No. 50.

Name R.J. Age 10 Diagnosis P.S.

Weight 27 Kilos. Haemoglobin 90%

Premedication Promethazine 25 mgms.
Bromethol 3 c.c.
Pethidine 50 mgms.
Atropine 0.6 mgms.



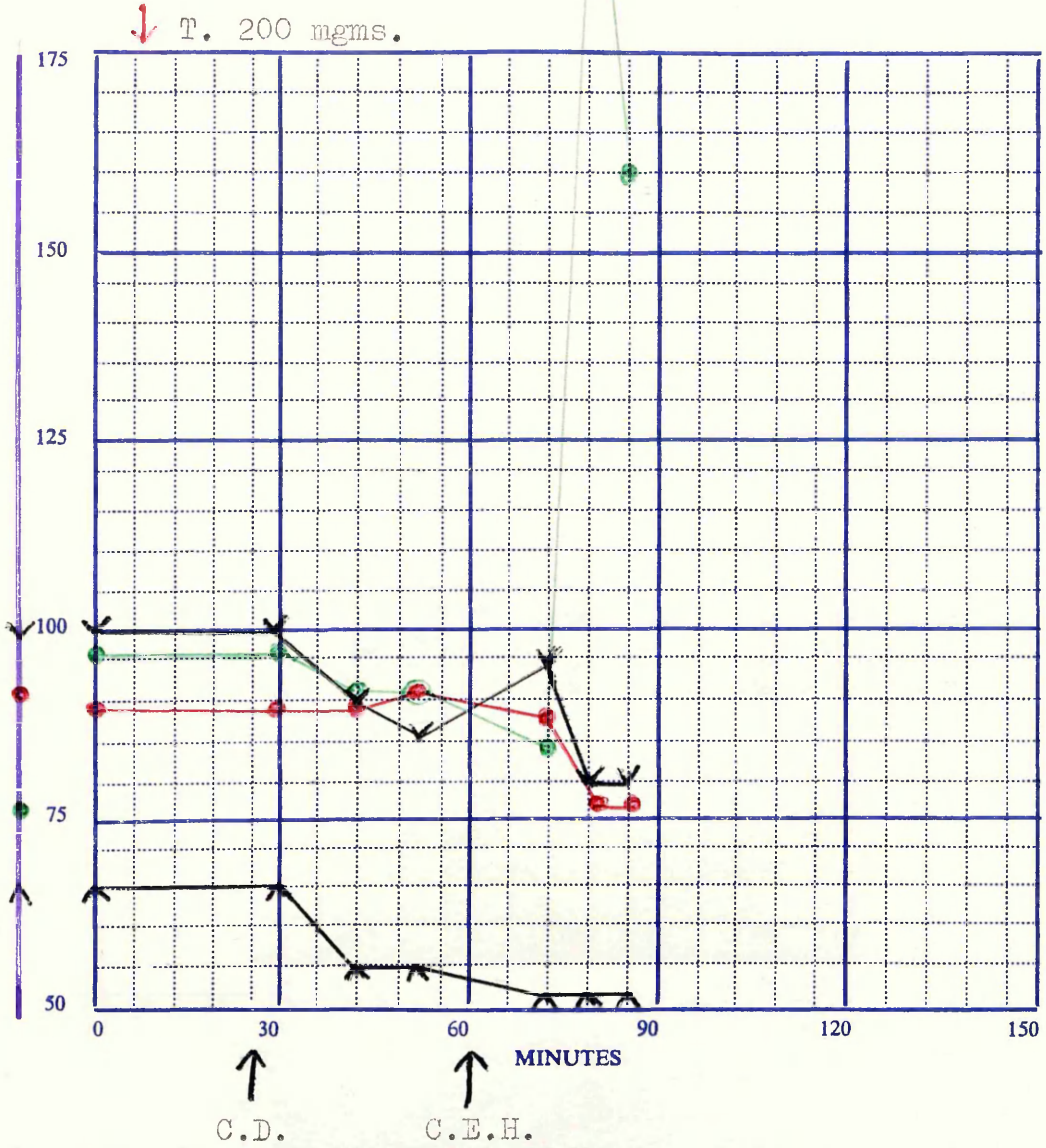
NOTES This was a repeat catheterisation on Case No. 59, some of the Bromethol was voided and the Thiopentone was given to supress restlessness. The oximeter ear piece was under repair at the time of the catheterisation but on the previous occasion no changes in oxygen saturation were noted.

CASE RECORD No.....51.....

Name R.A. Age 10 Diagnosis I.A.S.D.

Weight 30 Kilos. Haemoglobin 110%

Premedication Promethazine 25 mgms.
Bromethol 3.6 c.c.
Pethidine 60 mgms.
Atropine 0.6 mgms.



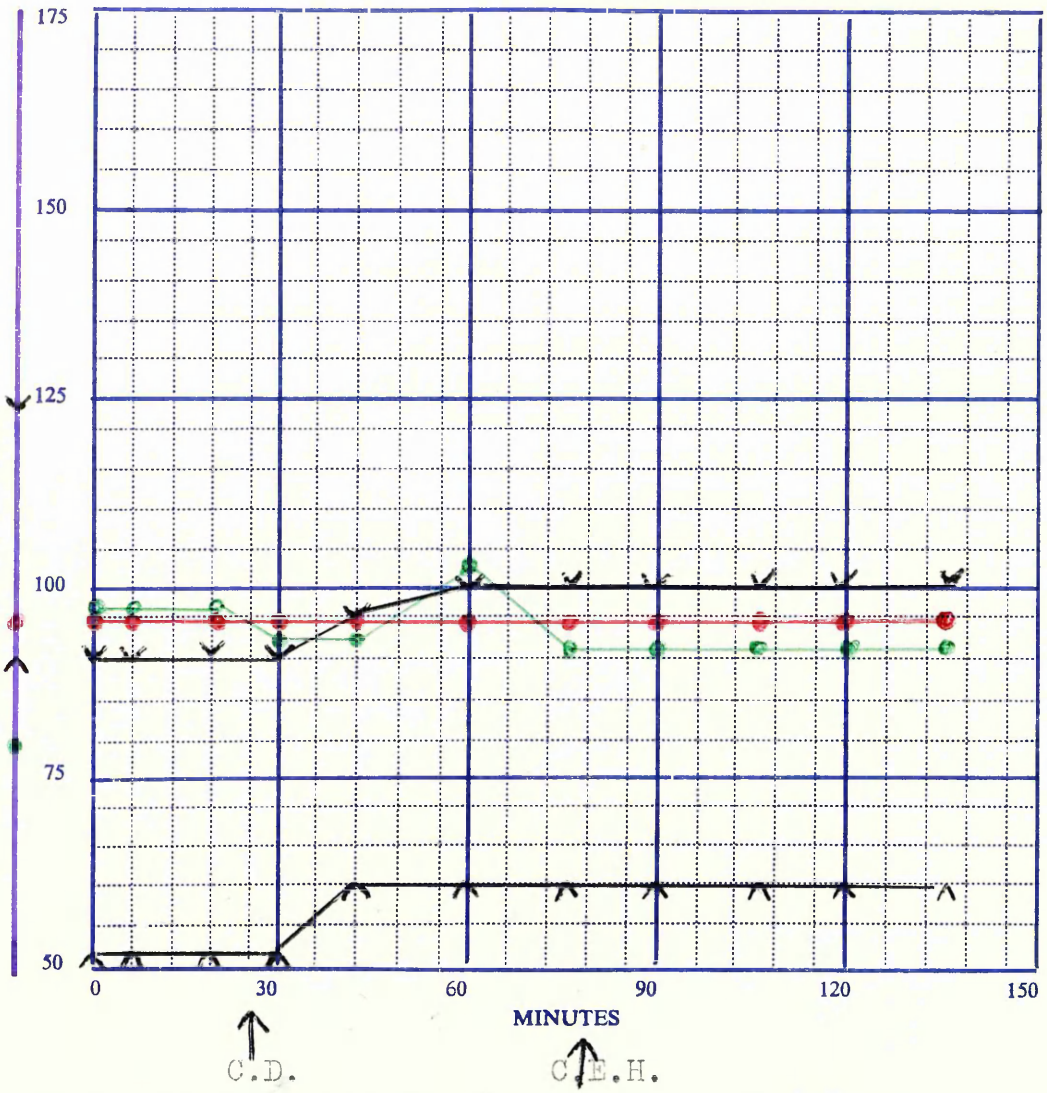
NOTES This child was extremely restless on arrival in the X-ray department. His Bromethol had been given late and probably not very well. The Thiopentone proved sufficient to settle him. While attempts were made to pass the catheter through the pulmonary valve a supraventricular tachycardia developed. The drop in arterial saturation which accompanied this can be clearly seen.

CASE RECORD No.....52.....

Name I.S. Age 10 Diagnosis P.S.

Weight 31.5 Kilos. Haemoglobin 105%

Premedication Promethazine 25 mgms.
Brometh0l 3.6 c.c.
Pethidine 70 mgms.
Atropine 0.6 mgms.



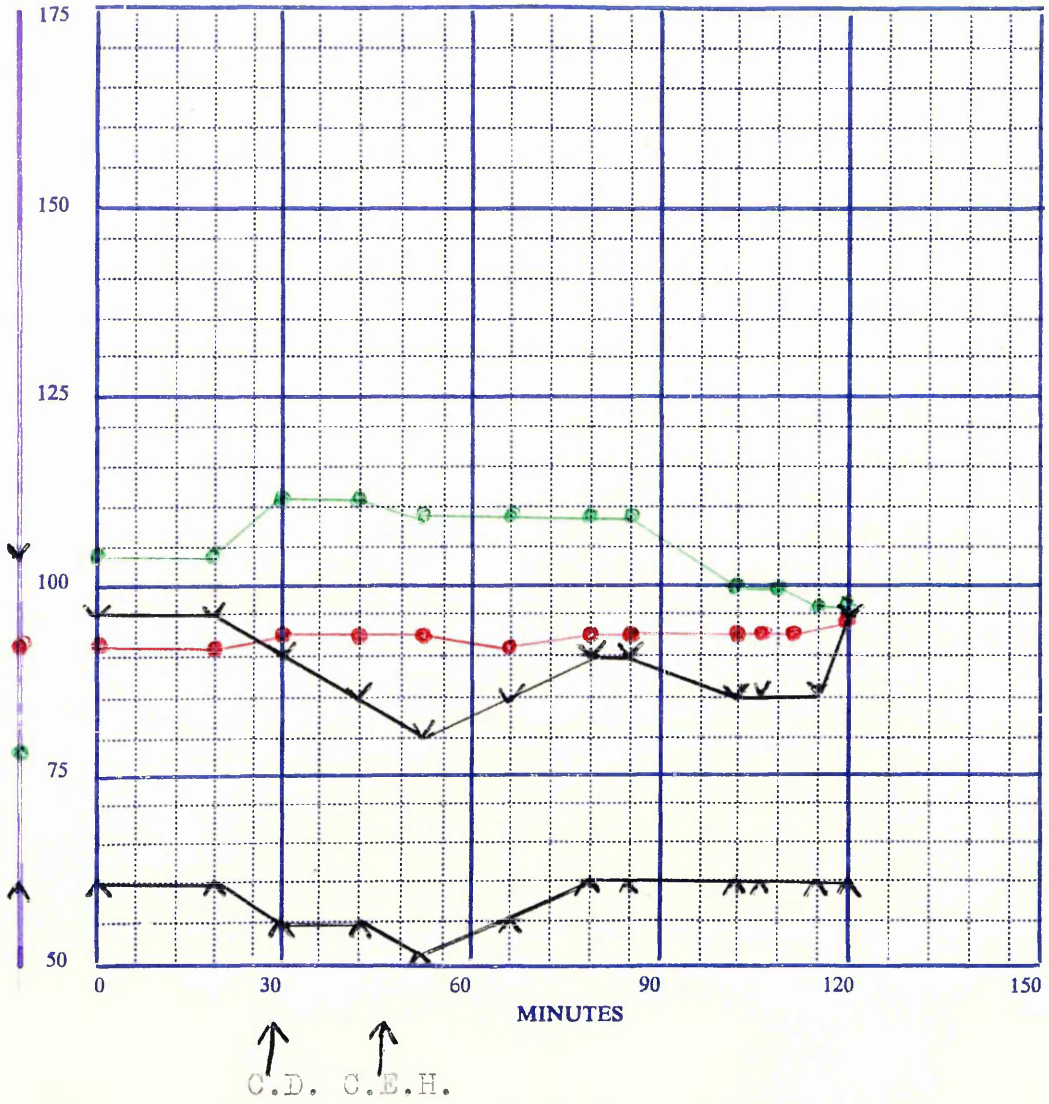
NOTES This child was sensitive to Quinidine and was given Procaine Amide instead. This may partly explain the drop in blood pressure. The catheterisation was a technical failure. The child awakened just after completion of the investigation.

CASE RECORD No.....53.....

Name J.L. Age 11 Diagnosis I.V.S.D.

Weight 34 Kilos. Haemoglobin 92%

Premedication Promethazine 25 mgms.
Bromethol 4 c.c.
Pethidine 50 mgms. } Intravenously
Atropine 0.6 mgms. }



NOTES Arterial analysis - oxygen saturation 97%
(oximeter reading 94%)
pCO_2 52.5 mmHg.
pH 7.35

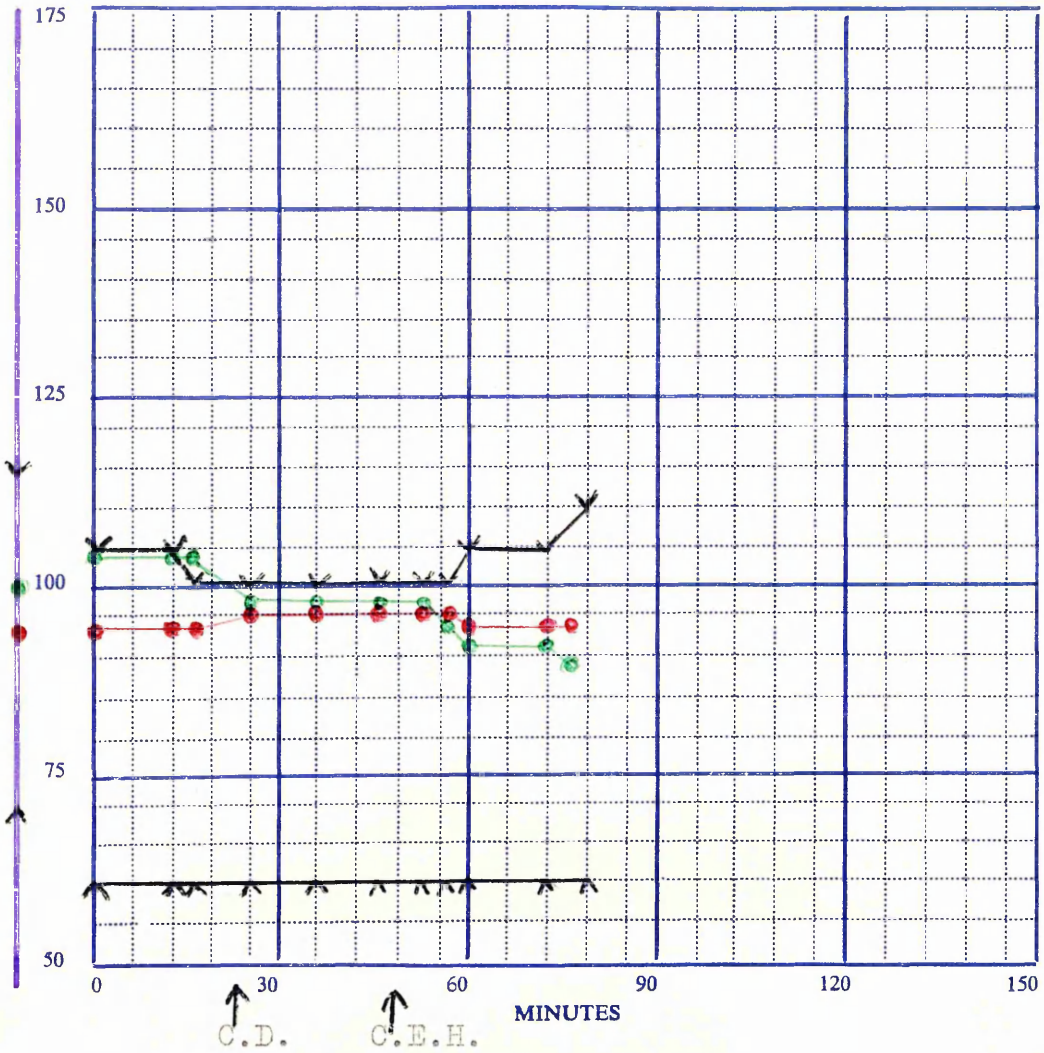
This child was given his Bromethol on a previous occasion and slept soundly all day. On this occasion he required the Pethidine before settling.

CASE RECORD No.....54.....

Name B.D. Age 11 Diagnosis A.S.D.

Weight 35.5 Kilos. Haemoglobin 125%

Premedication Promethazine 25 mgms.
Bromethol 4 c.c.
Pethidine 75 mgms.
Atropine 0.6 mgms.



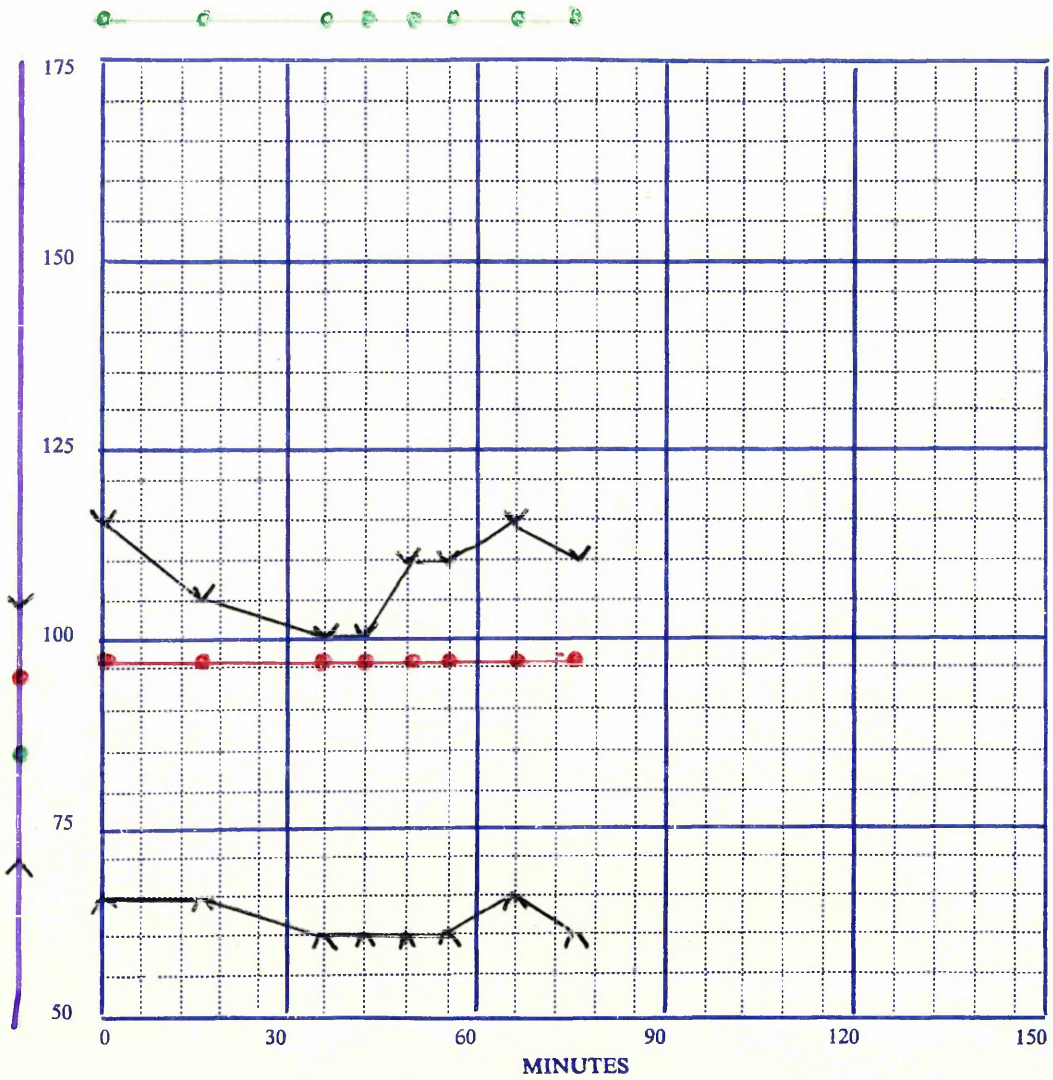
NOTES This was an uneventful catheterisation.
Arterial analysis - pCO_2 37.1 mmHg.
pH 7.4

CASE RECORD No.....55.....

Name E.McL. Age 11 Diagnosis P.S.

Weight 22.5 Kilos. Haemoglobin 90%

Premedication Promethazine 10 mgms.
Bromethol 4 c.c.
Pethidine 75 mgms.
Atropine 0.6 mgms.



NOTES This child developed a supraventricular tachycardia before arrival in the X-ray department. She had done this on previous occasions for no apparent reason. No attempt was made to perform the catheterisation and she was merely observed during the period charted above.

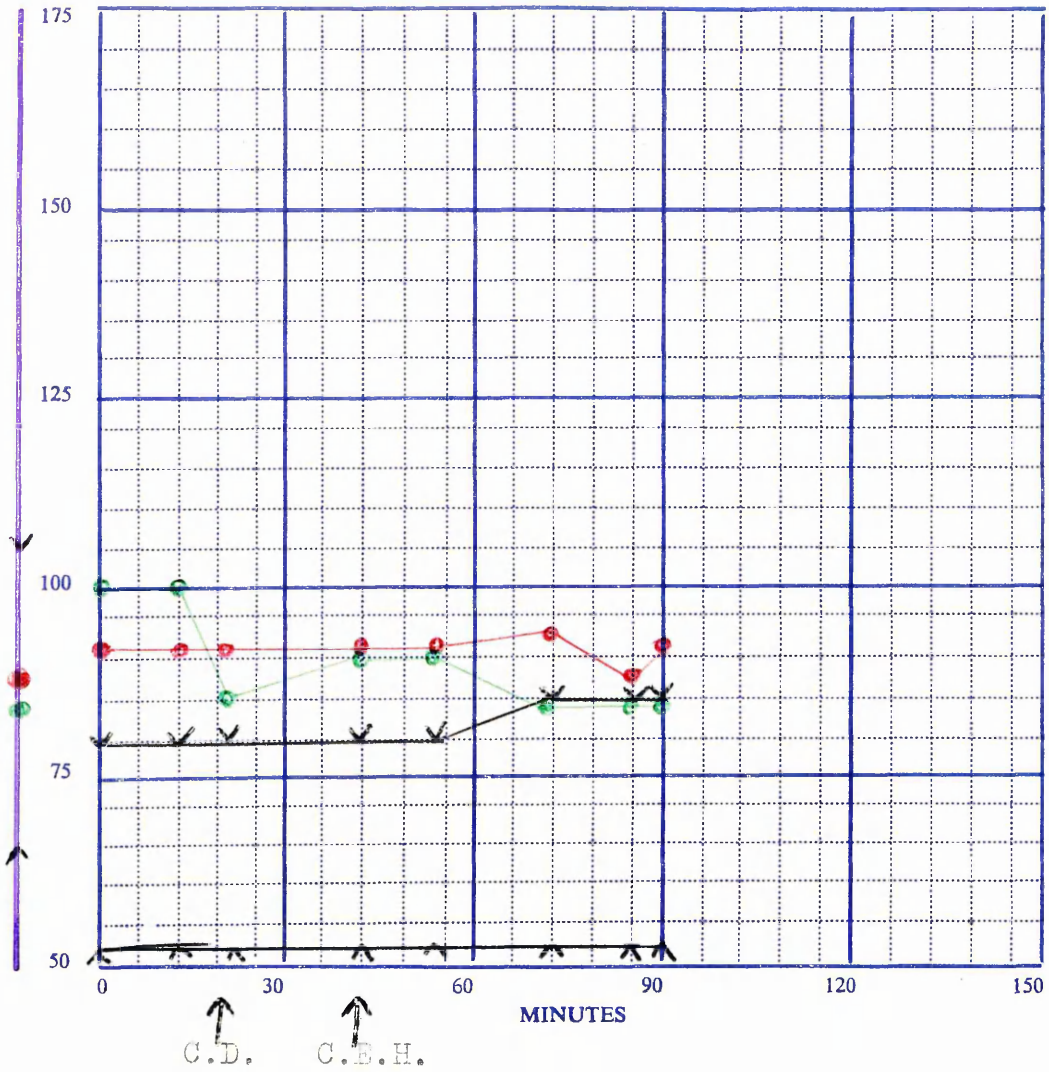
ARTERIAL: O_2 sat. 96%, pCO_2 30.5 mmHg., pH 7.37.

CASE RECORD No.....56.....

Name M.A. Age 11 Diagnosis V.S.D. + P.H.

Weight 26.5 Kilos. Haemoglobin 100%

Premedication Promethazine 25 mgms.
Bromethol 3.3 c.c.
Pethidine 60 mgms.
Atropine 0.6 mgms.



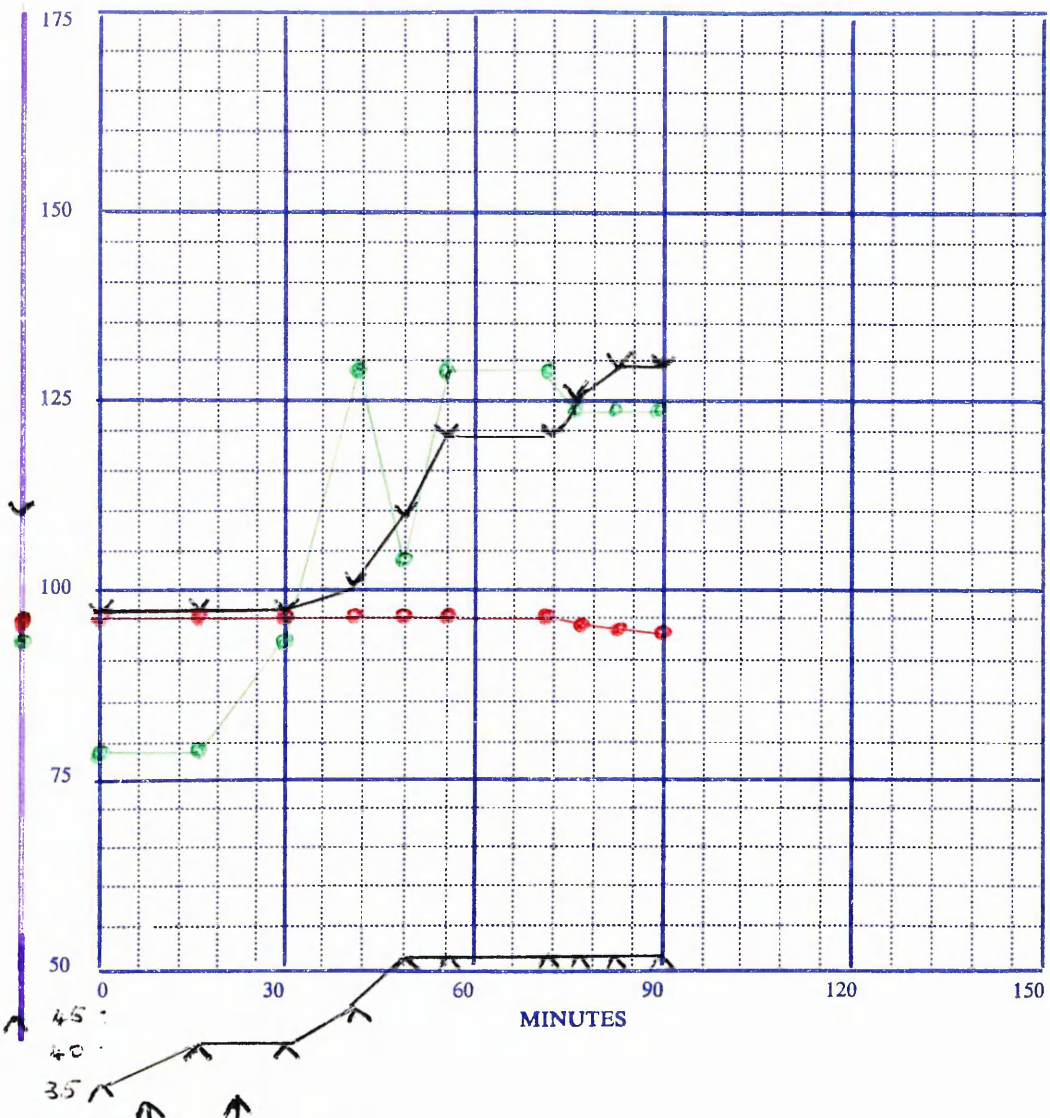
NOTES The drop in arterial oxygen saturation towards the end of the catheterisation was associated with the presence of the catheter tip in the pulmonary artery.

CASE RECORD No.....57.....

Name J.W. Age 12 Diagnosis P.S.

Weight 31.5 Kilos. Haemoglobin 84%

Premedication Promethazine 25 mgms.
Bromethol 3.8 c.c.
Pethidine 70 mgms.
Atropine 0.6 mgms.



NOTES ↑C.D. ↑C.E.H.

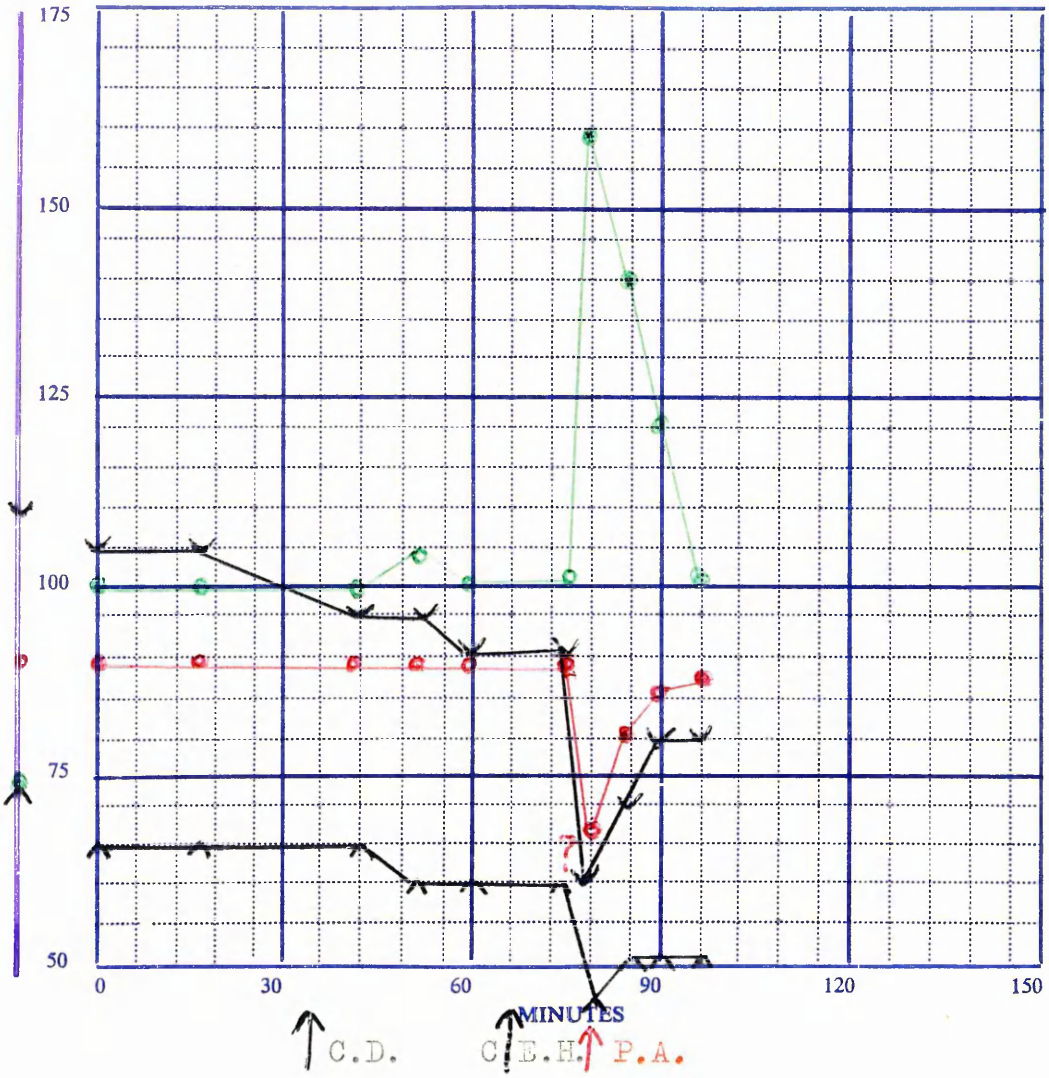
The dose of Bromethol in this case was reduced to 110 mgms/Kilos. The response was poor but the catheterisation was carried out without further anaesthesia. At the end of the investigation the child was moving, talking and actively sick.

CASE RECORD No. 58.

Name A.M. Age 13. Diagnosis P.S.

Weight 42 Kilos. Haemoglobin 98%

Premedication Promethazine 25 mgms.
Bromethol 5 c.c.
Pethidine 75 mgms.
Atropine 0.6 mgms.



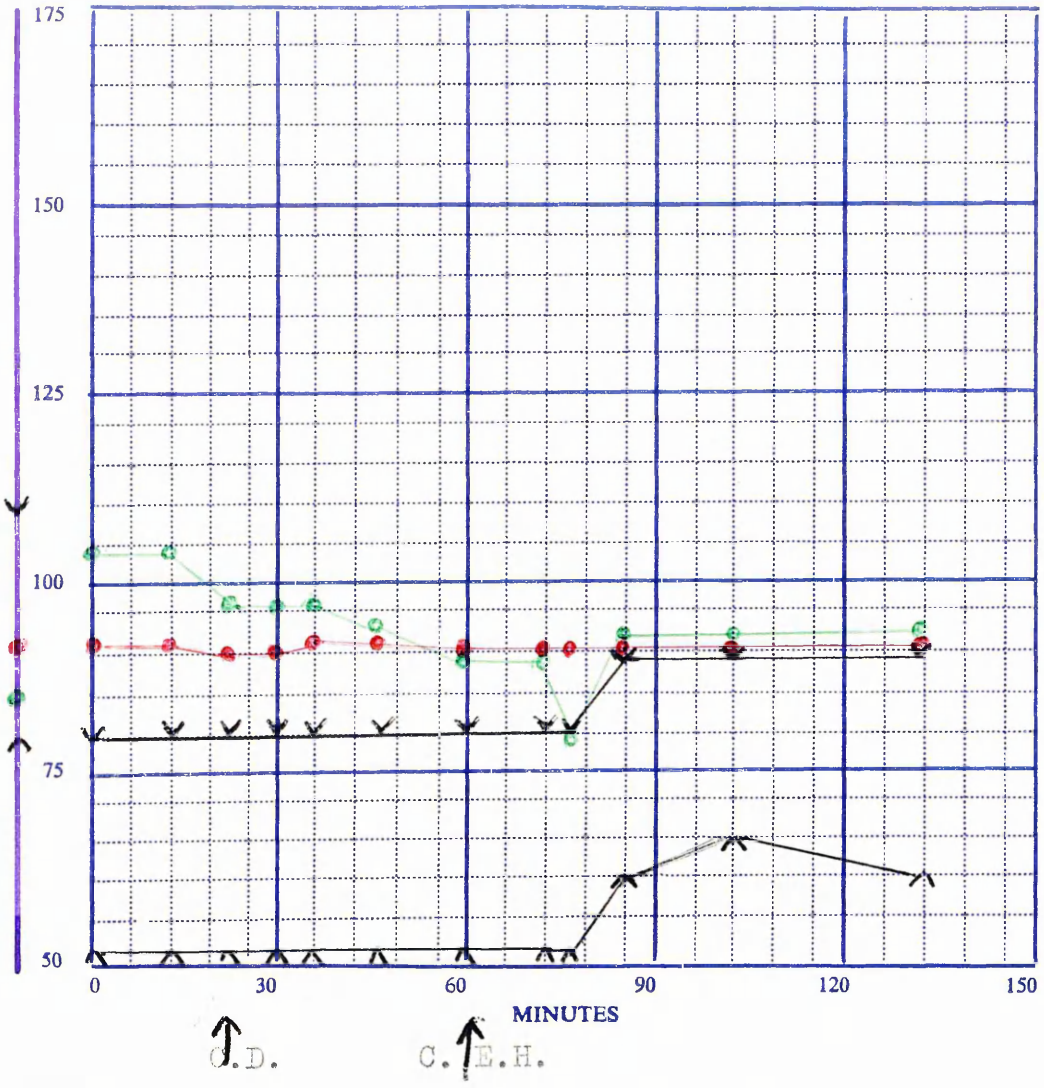
NOTES This child had been catheterised unsuccessfully in another hospital on two occasions. When the catheter passed through the pulmonary valve this time, a gradient of 170 mmHg. was revealed. The profound changes which resulted can be seen and they almost caused the patient's death.

CASE RECORD No.....58.....

Name MA. Age 13 Diagnosis V.S.D. + P.H.

Weight 31 Kilos. Haemoglobin 100%

Premedication Promethazine 25 mgms.
Bromethol 3.5 c.c.
Pethidine 15 mgms.
Atropine 0.6 mgms.



NOTES This was an uneventful catheterisation and the results are almost identical to those obtained on a previous occasion (Case 56).

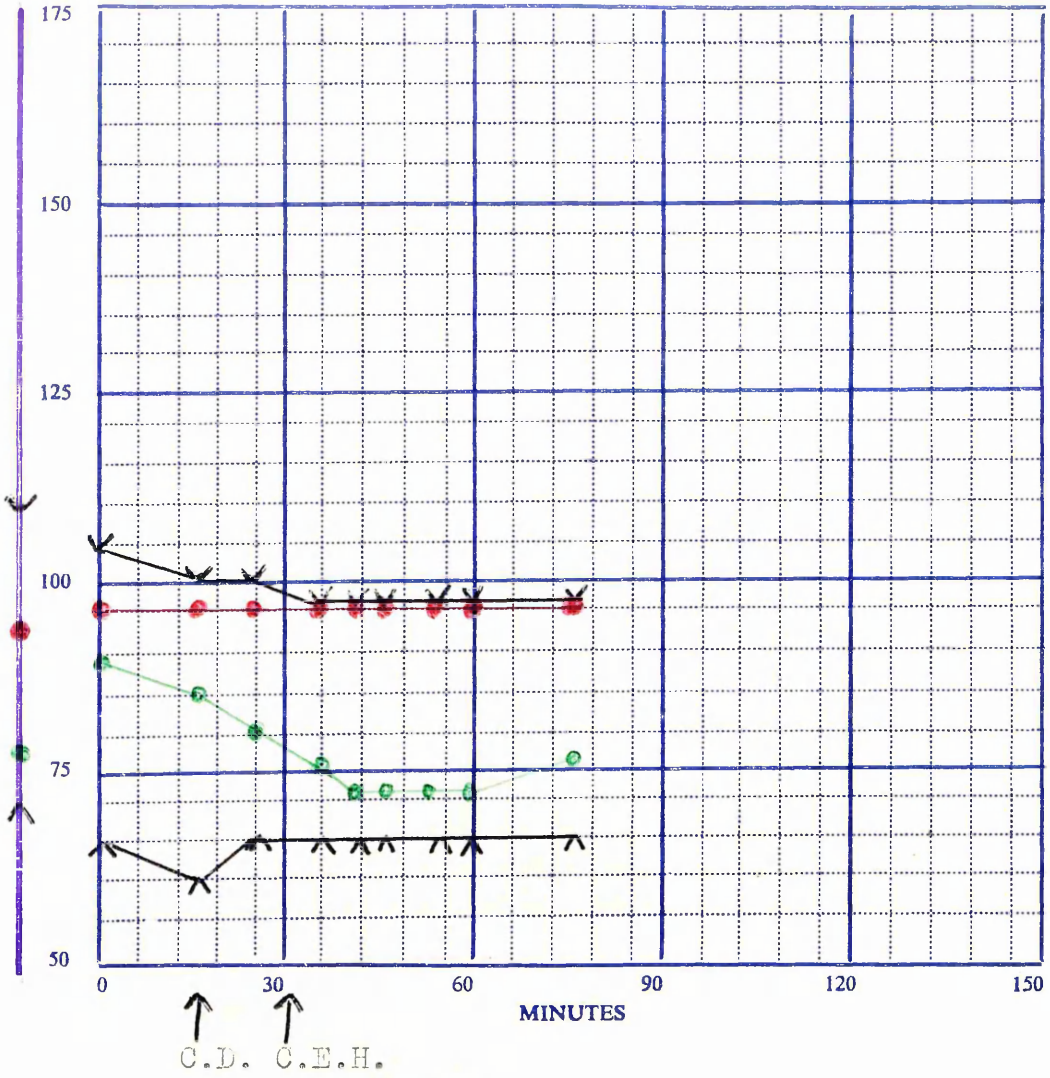
Venous analysis - pCO2 54.2 mmHg.
pH 7.26

CASE RECORD No.....60.....

Name M.C. Age 15 Diagnosis V.S.D.

Weight 50 Kilos. Haemoglobin 92%

Premedication Promethazine 25 mgms.
Bromethol 6 c.c.
Pethidine 100 mgms.
Atropine 0.6 mgm.



NOTES Arterial analysis - oxygen saturation 97%
(oximeter reading 95%)
pCO_2 49 mmHg.
pH 7.29

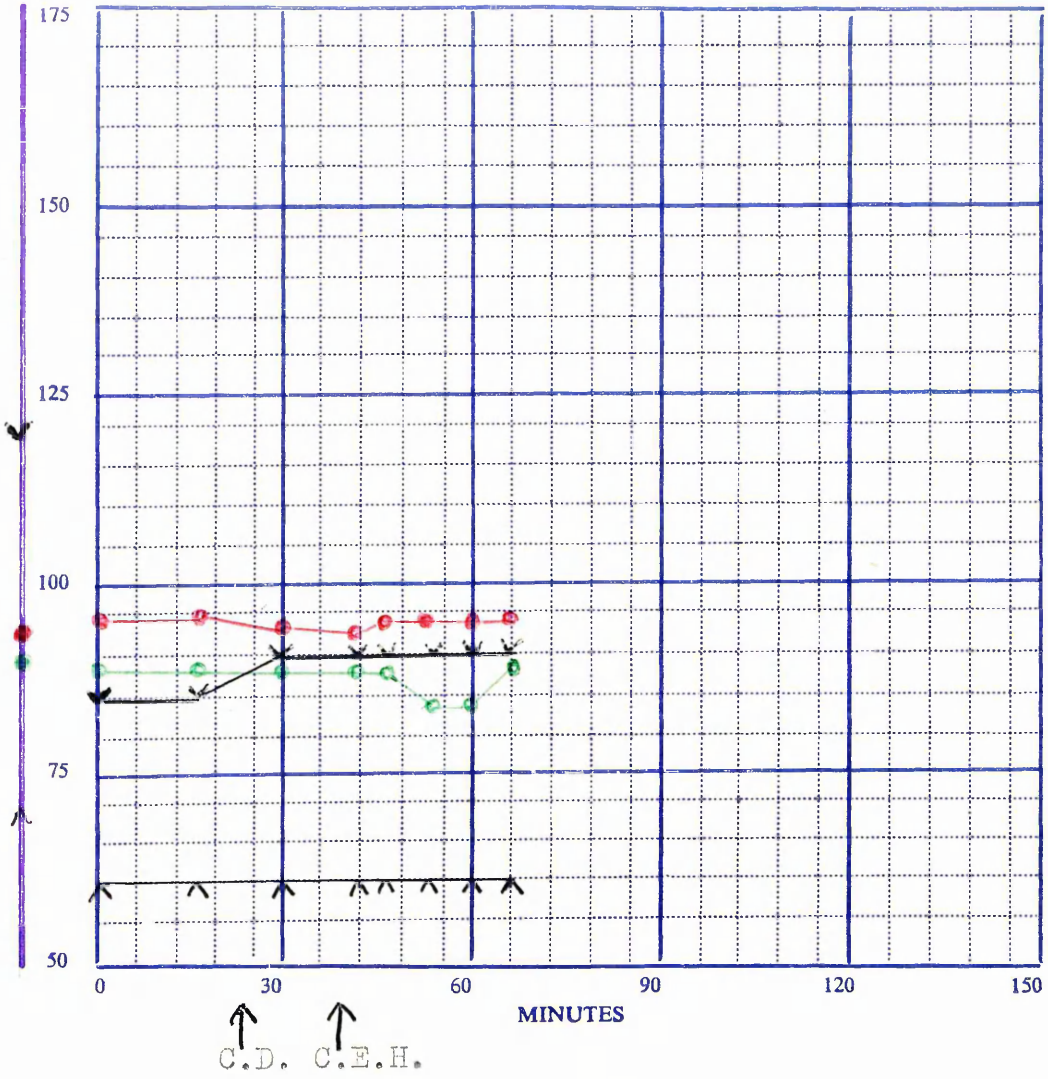
This patient had been previously catheterised under sedation (Case 78).

CASE RECORD No.....61.....

Name A.D. Age 16 Diagnosis I.A.S.D.

Weight 50 Kilos. Haemoglobin 100%

Premedication Promethazine 25 mgms.
Bromethol 6 c.c.
Pethidine 100 mgms.
Atropine 0.6 mgms.



NOTES Arterial analysis - pCO_2 44.2 mmHg.
pH 7.39

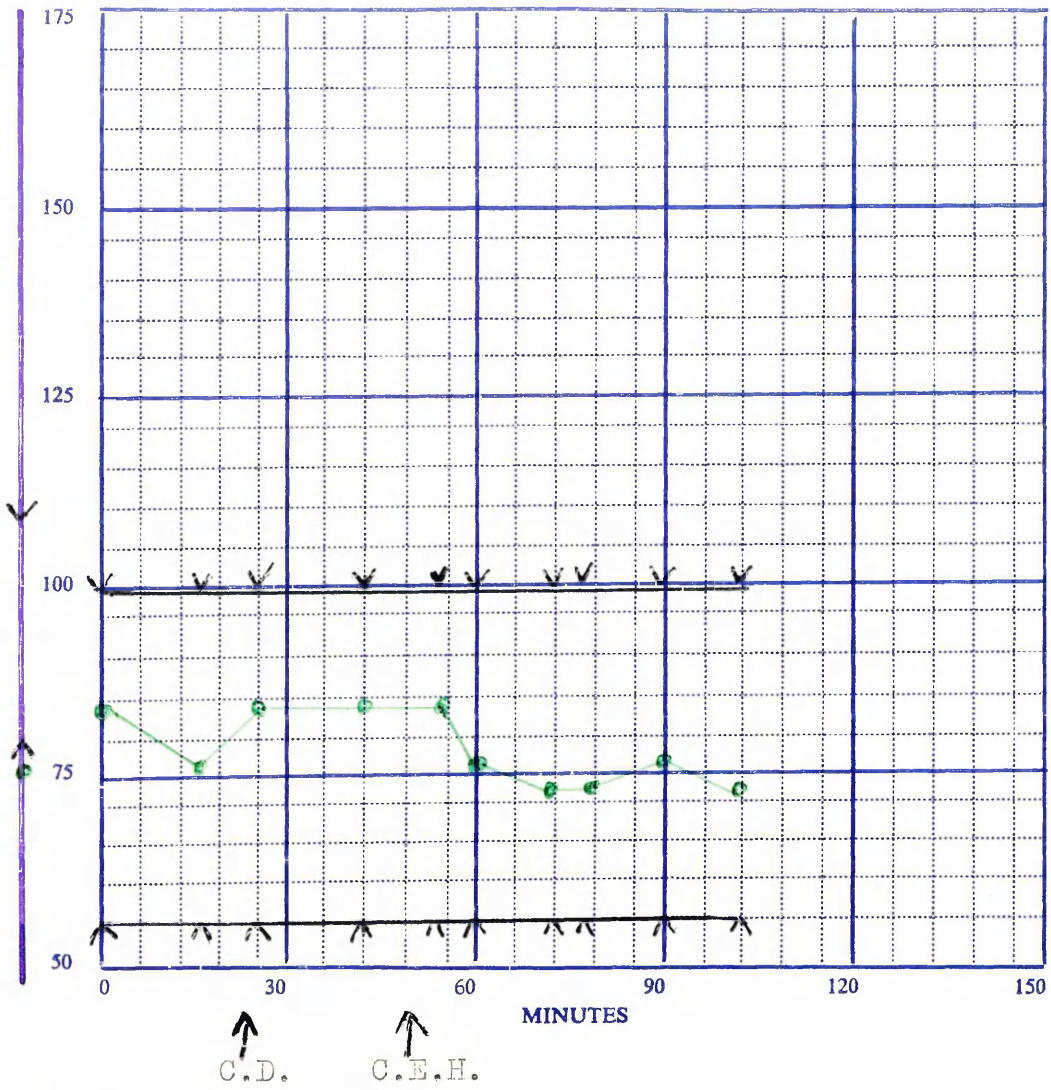
This was an uneventful catheterisation.

CASE RECORD No.....62.....

Name E.D. Age 17½ Diagnosis M.S.

Weight 36.4 Kilos. Haemoglobin –

Premedication Promethazine 25 mgms.
Brometol 4.5 cc.
Pethidine 75 mgms.
Atropine 0.6 mgms.



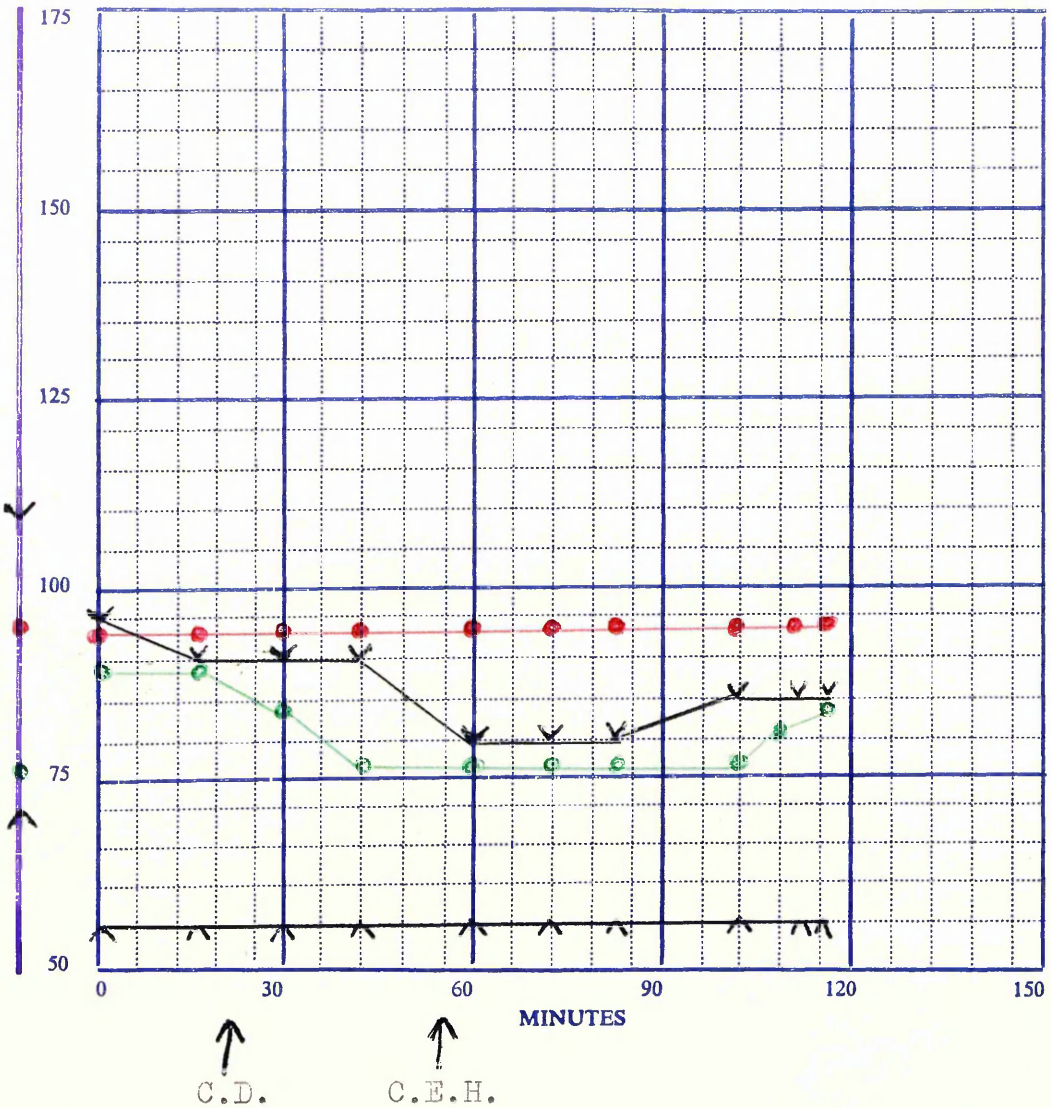
NOTES This boy was a mental defective and resisted all investigations. It was impossible to perform oximetry in the ward or to obtain blood to estimate haemoglobin. The catheterisation itself was smooth and uneventful.

CASE RECORD No.....63.....

Name H.C. Age 21 Diagnosis I.A.S.D.

Weight 57 Kilos. Haemoglobin 70%

Premedication Promethazine 25 mgms.
Bromethol 6 c.c.
Pethidine 100 mgms.
Atropine 0.6 mgms.



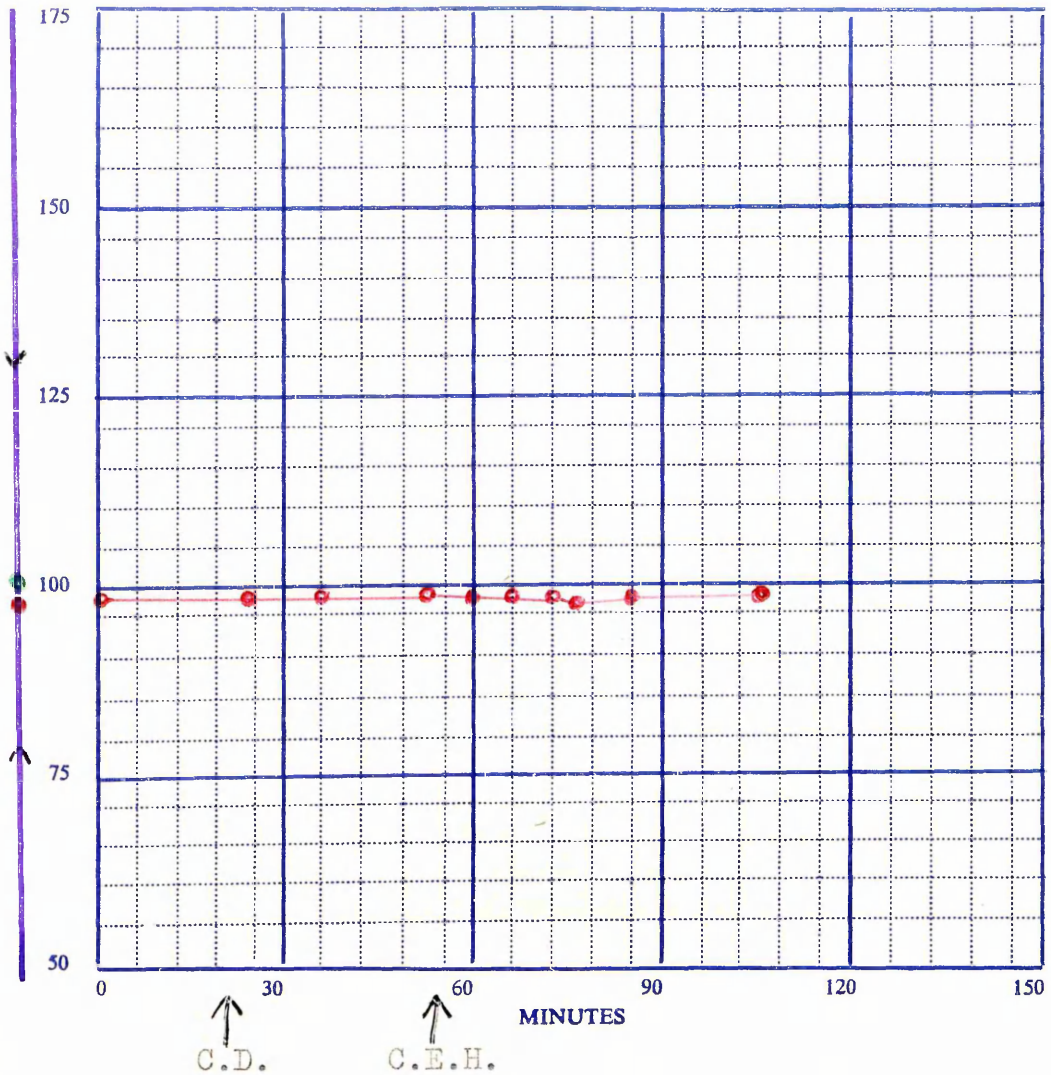
NOTES An attempt had been made five years previously to catheterise this girl under heavy sedation but had to be abandoned when she became hysterical. She was later given Thiopentone, developed a convulsion and E.E.Gs. showed an epileptiform pattern for some time afterwards. This catheterisation was uneventful.

CASE RECORD No.....6.....

Name J.C. Age 10 Diagnosis I.A.S.D.

Weight 29 Kilos. Haemoglobin 80%

Premedication Pentobarbitone 90 mgms.
Pethidine 50 mgms.



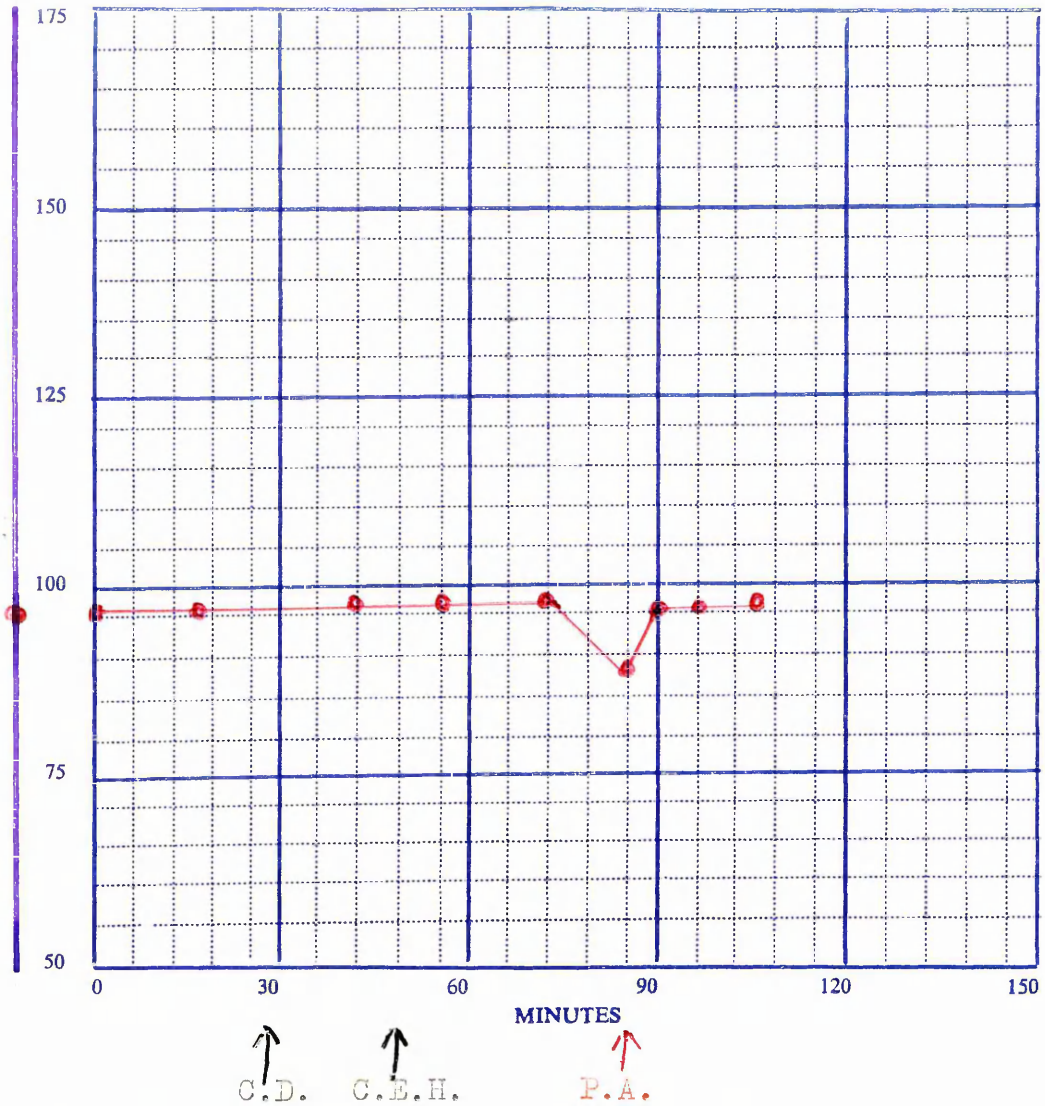
NOTES The patient complained of slight abdominal pain as the catheter entered the inferior vena cava.

CASE RECORD No. 65.

Name J.M. Age 10 Diagnosis P.S.

Weight 27 Kilos. Haemoglobin 108%

Premedication Pentobarbitone 90 mgms.



NOTES

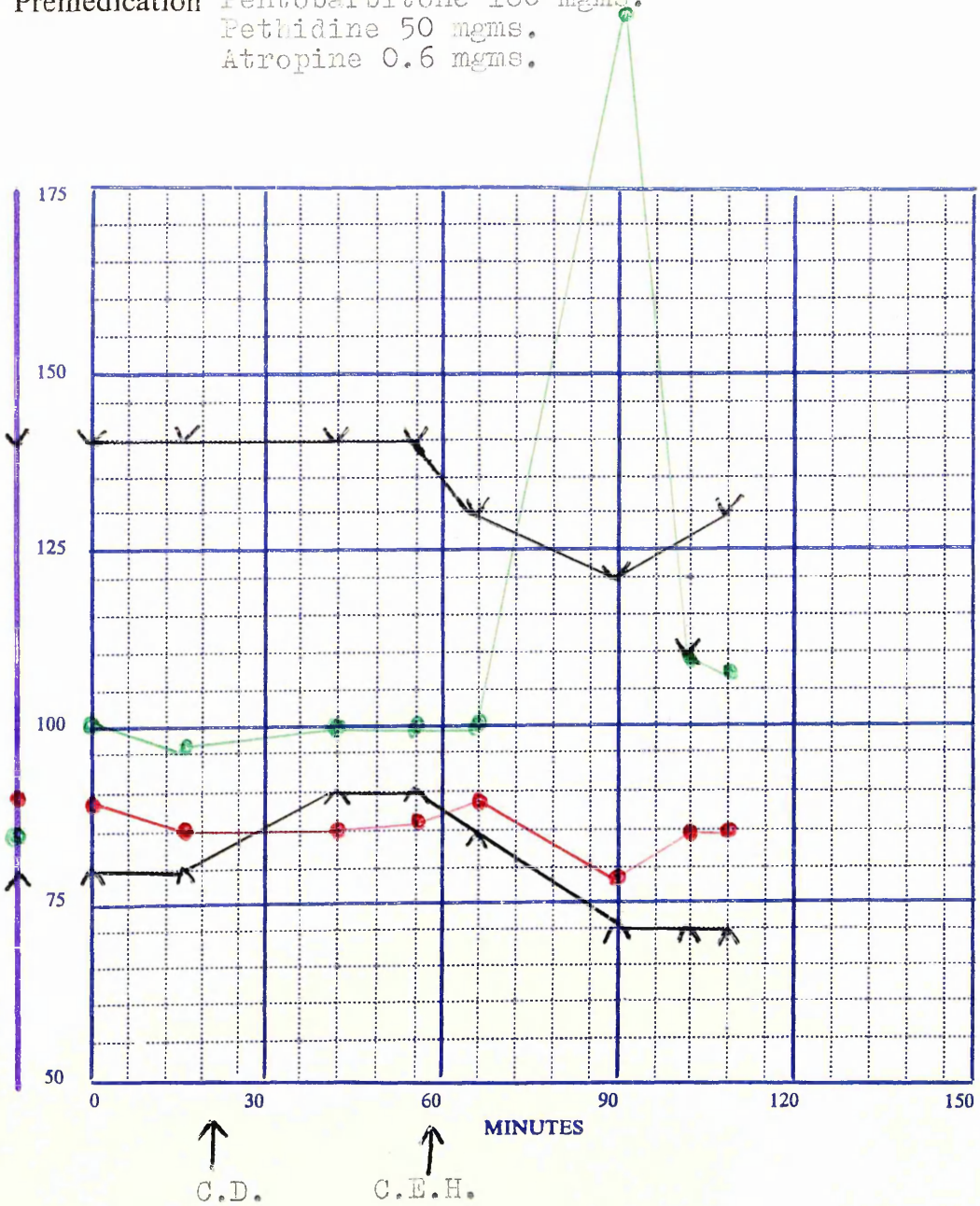
This patient had a tight pulmonary stenosis and the drop in arterial oxygen saturation occurred as the catheter blocked the pulmonary valve.

CASE RECORD No.....66.....

Name B.McG. Age 11 Diagnosis P.S.

Weight 34 Kilos. Haemoglobin 116%

Premedication Pentobarbitone 180 mgms.
Pethidine 50 mgms.
Atropine 0.6 mgms.



NOTES

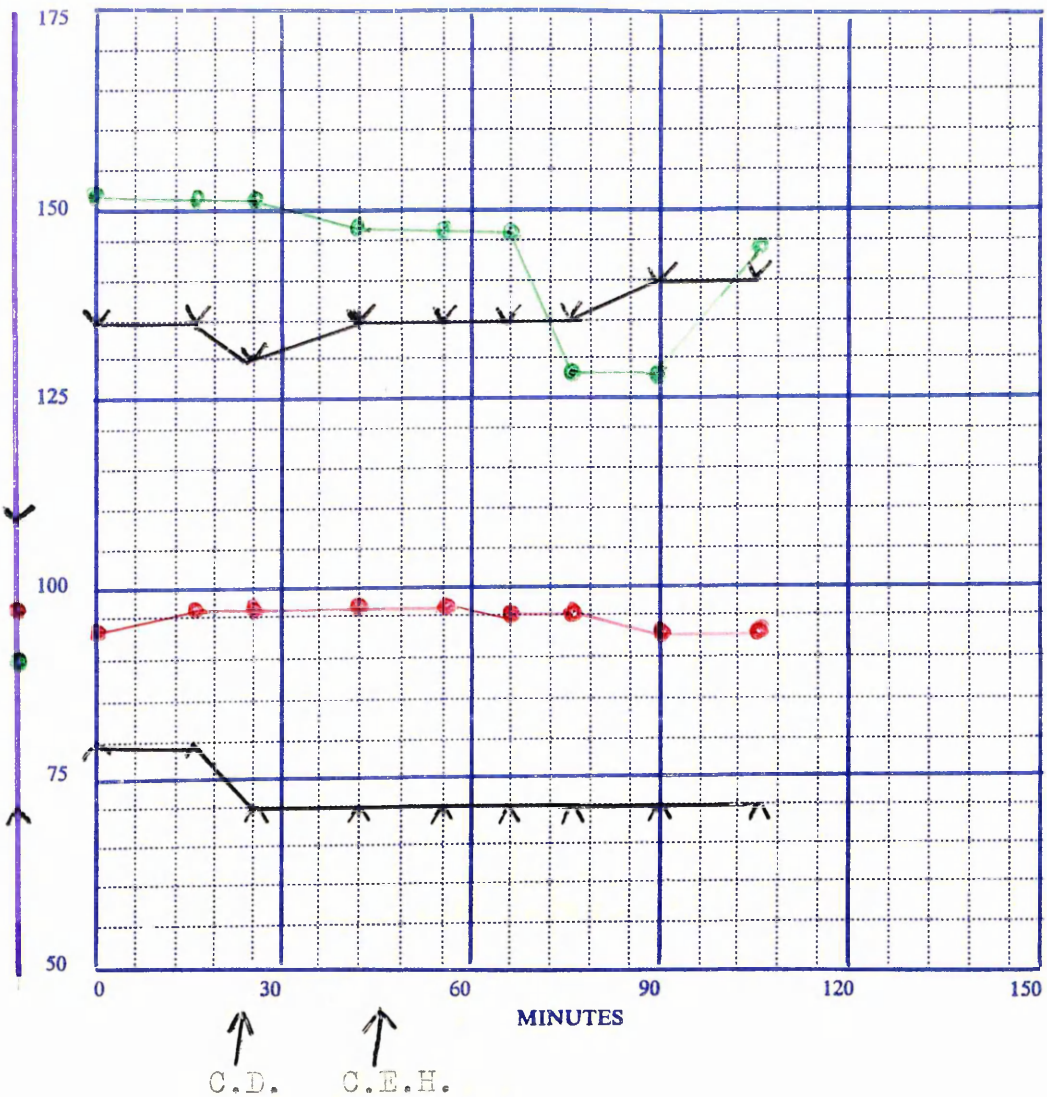
This patient developed a supraventricular tachycardia after considerable manipulatiom of the catheter. The drop in arterial oxygen saturation which resulted is clearly seen. Procaine Amide was administered and the investigation abandoned.

CASE RECORD No.....67.....

Name P.J. Age 12 Diagnosis P.S.

Weight 34 Kilos. Haemoglobin 110%

Premedication Promethazine 25 mgms.
Amylobarbitone 180 mgms.
Pethidine 75 mgms.
Atropine 0.6 mgms.



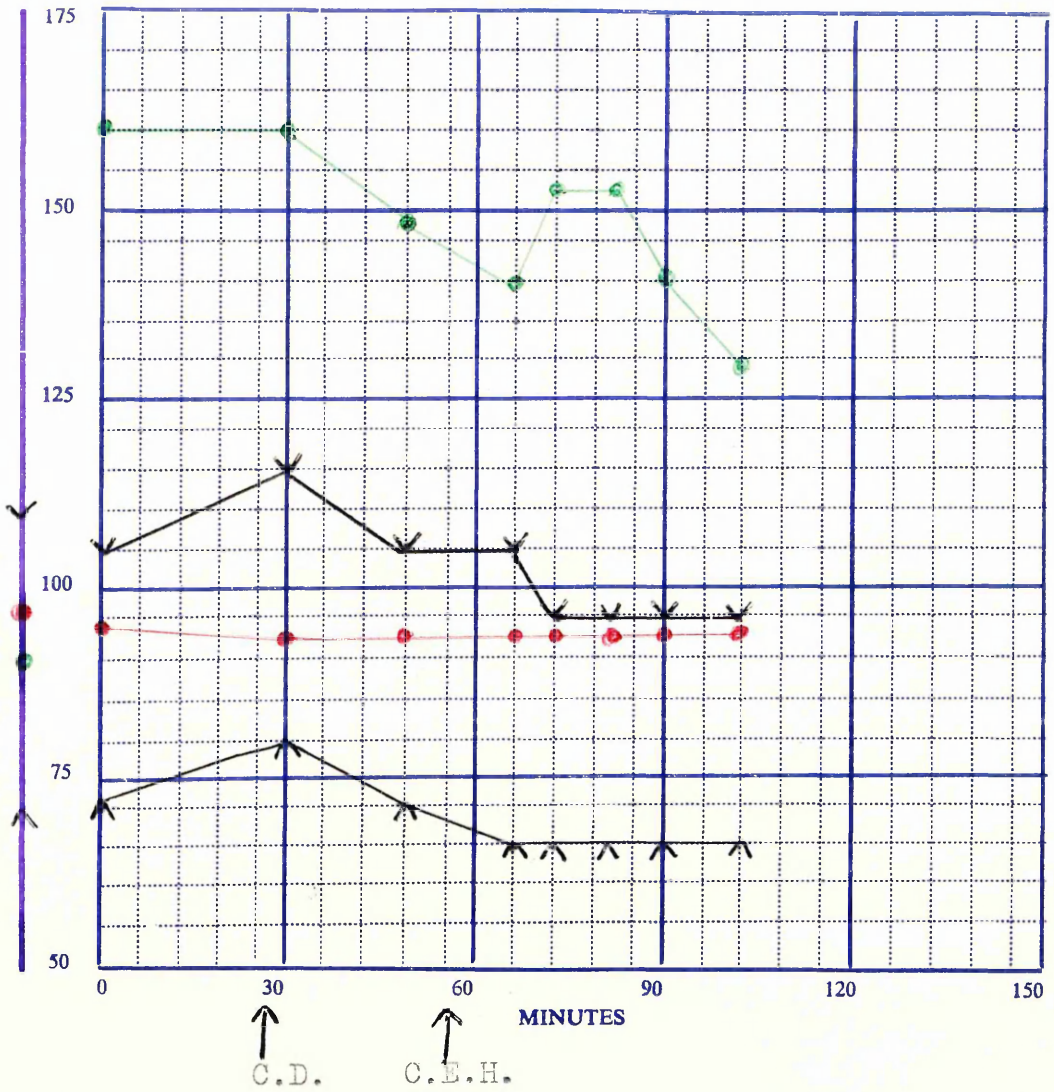
NOTES The child was restless and latterly complained of pain in the axilla. For the last half hour he was sobbing continuously.

CASE RECORD No.....68.....

Name P.J. Age 12 Diagnosis P.S.

Weight 36 Kilos. Haemoglobin 95%

Premedication Promethazine 25 mgms.
Amylobarbitone 180 mgms.
Pethidine 75 mgms.
Atropine 0.6 mgms.



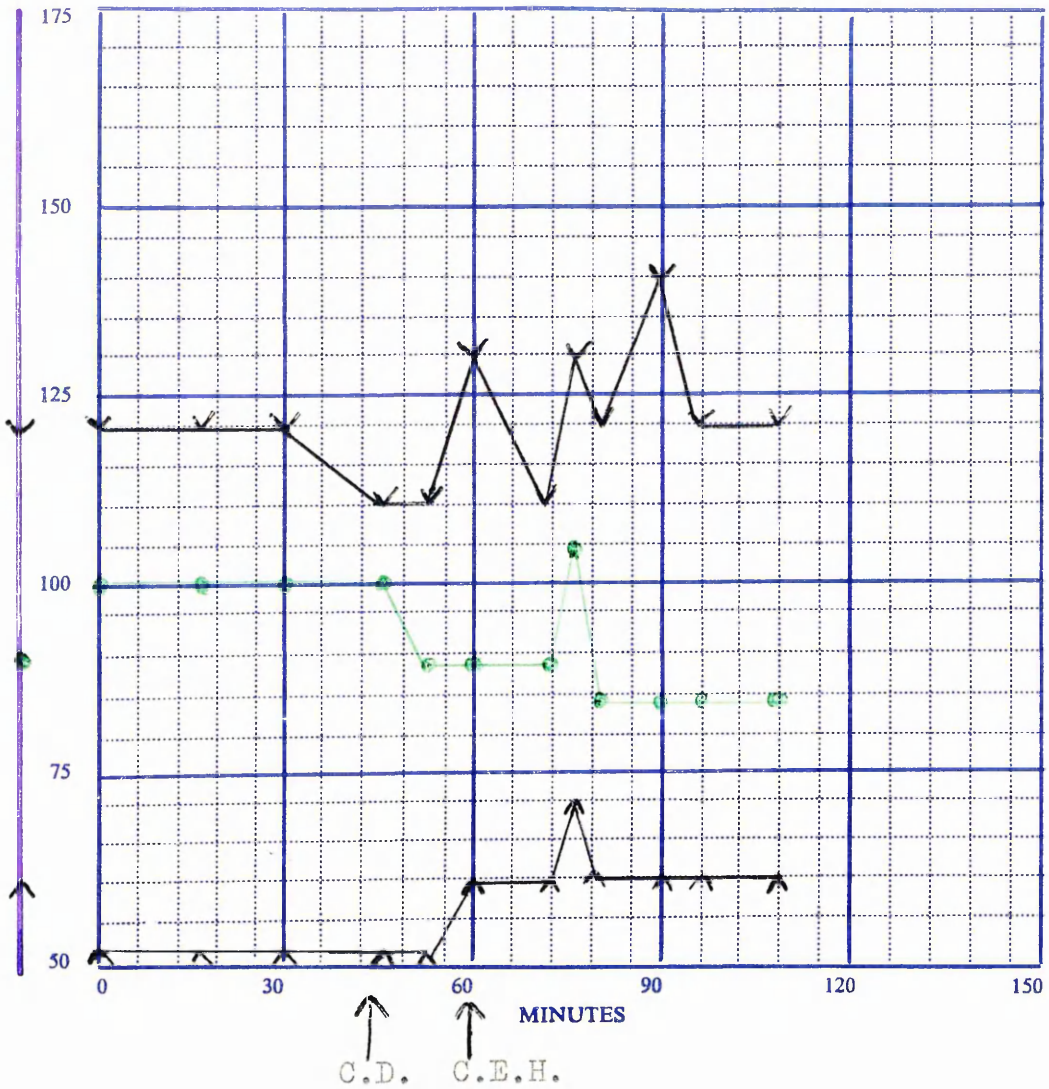
NOTES The child in error was not given Quinidine. Towards the end of the catheterisation he was actively sick. Arterial analysis - Oxygen sat. 90.5%
Oximeter reading 92%

CASE RECORD No. 69

Name J.G. Age 13 Diagnosis I.V.S.D.

Weight 41 Kilos. Haemoglobin 100%

Premedication Papaveratum 20 mgms.
Scopolamine 0.4 mgms.



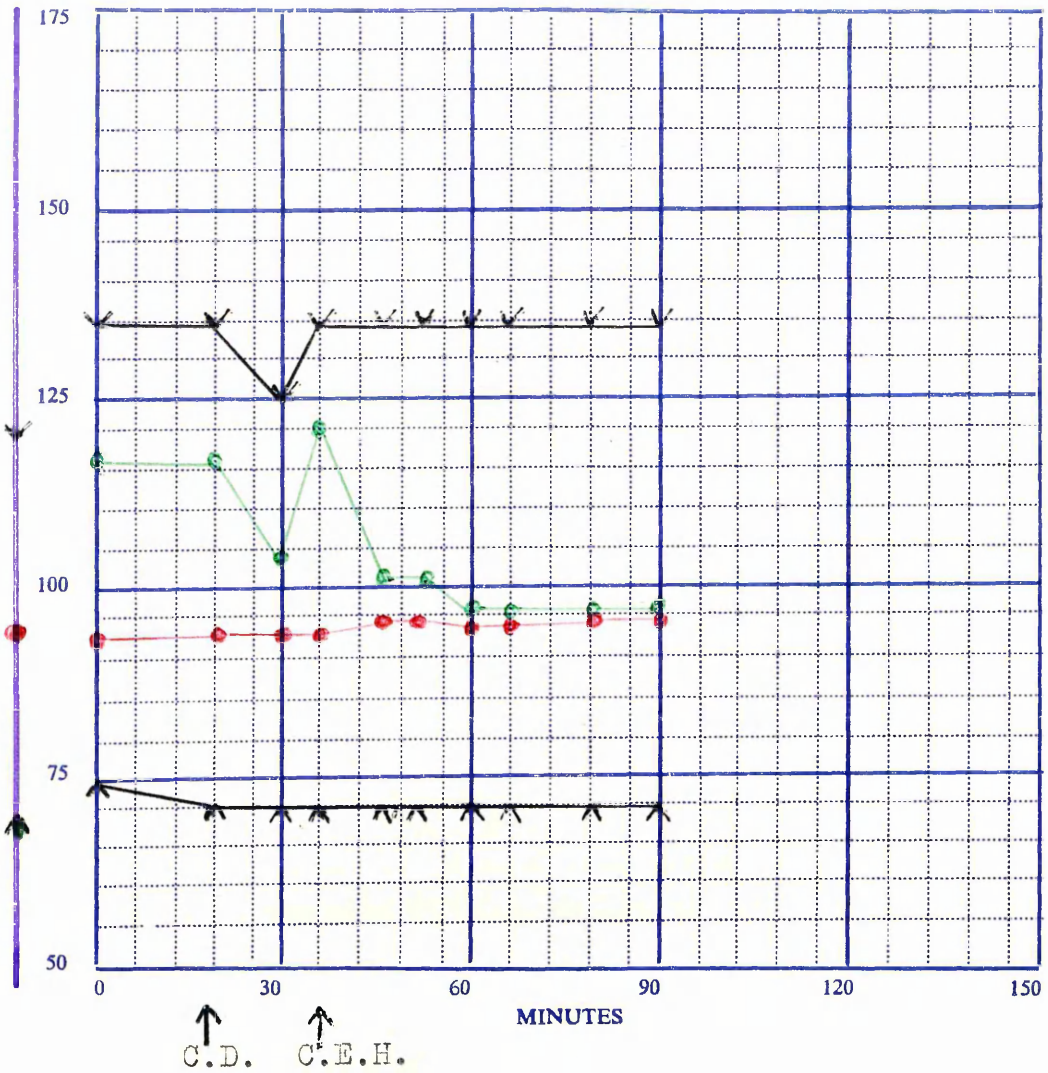
NOTES This patient complained of pain as the catheter passed up her arm and discomfort as it passed the pulmonary valve. The fluctuations in blood pressure took place as the patient alternately dosed and talked to the ward nurse.

CASE RECORD No.....70.....

Name D.McN. Age 13 Diagnosis P.S.

Weight 55 Kilos. Haemoglobin 112%

Premedication Promethazine 25 mgms.
Amylobarbitone 200 mgms.
Pethidine 100 mgms.
Atropine 0.6 mgms.



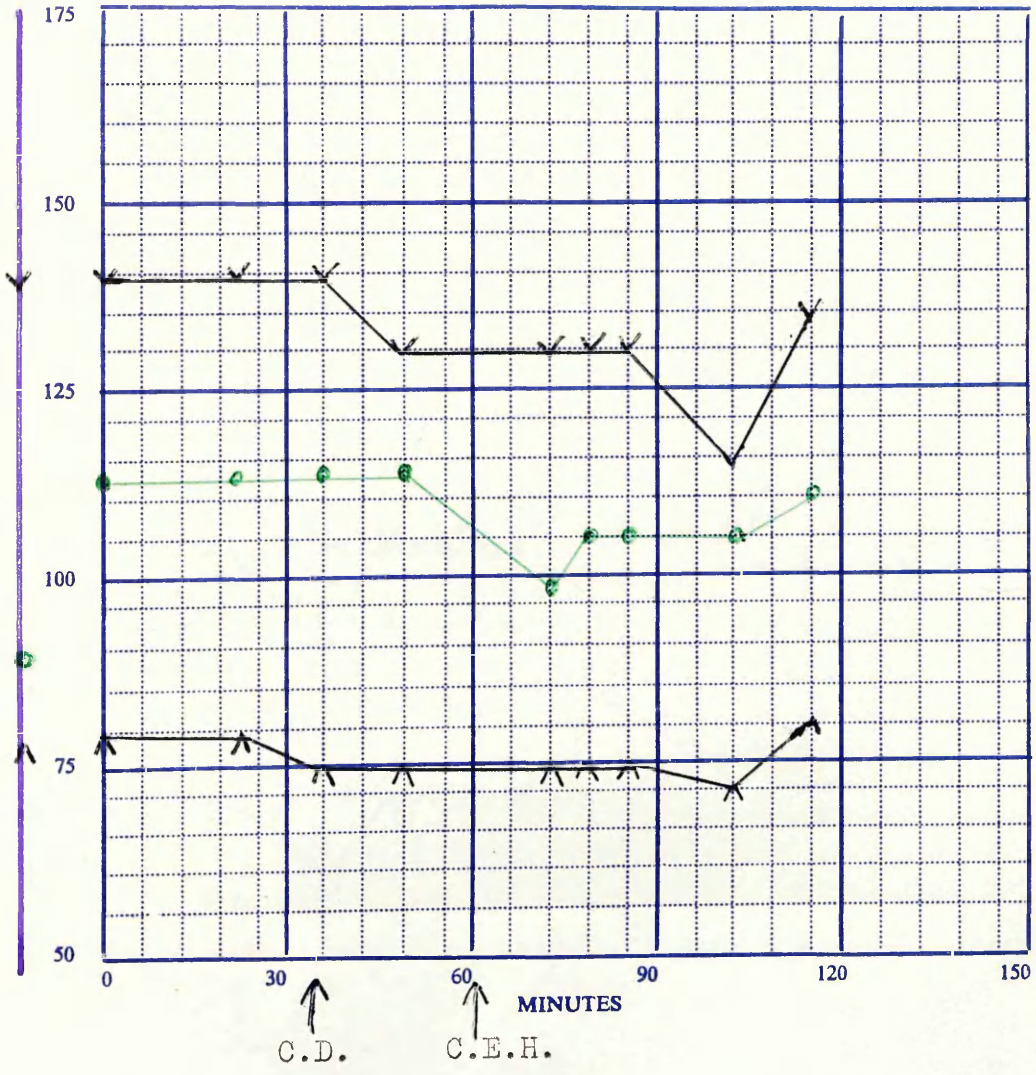
NOTES This child was mentally dull and was premedicated with a view to giving a general anaesthetic if necessary. On two occasions during the catheterisation he complained of a pain in his arm and a feeling of nausea.

CASE RECORD No.....71.....

Name B.MCG. Age 13 Diagnosis P.S.

Weight 46 Kilos. Haemoglobin 115%

Premedication Amylobarbitone 200 mgms.



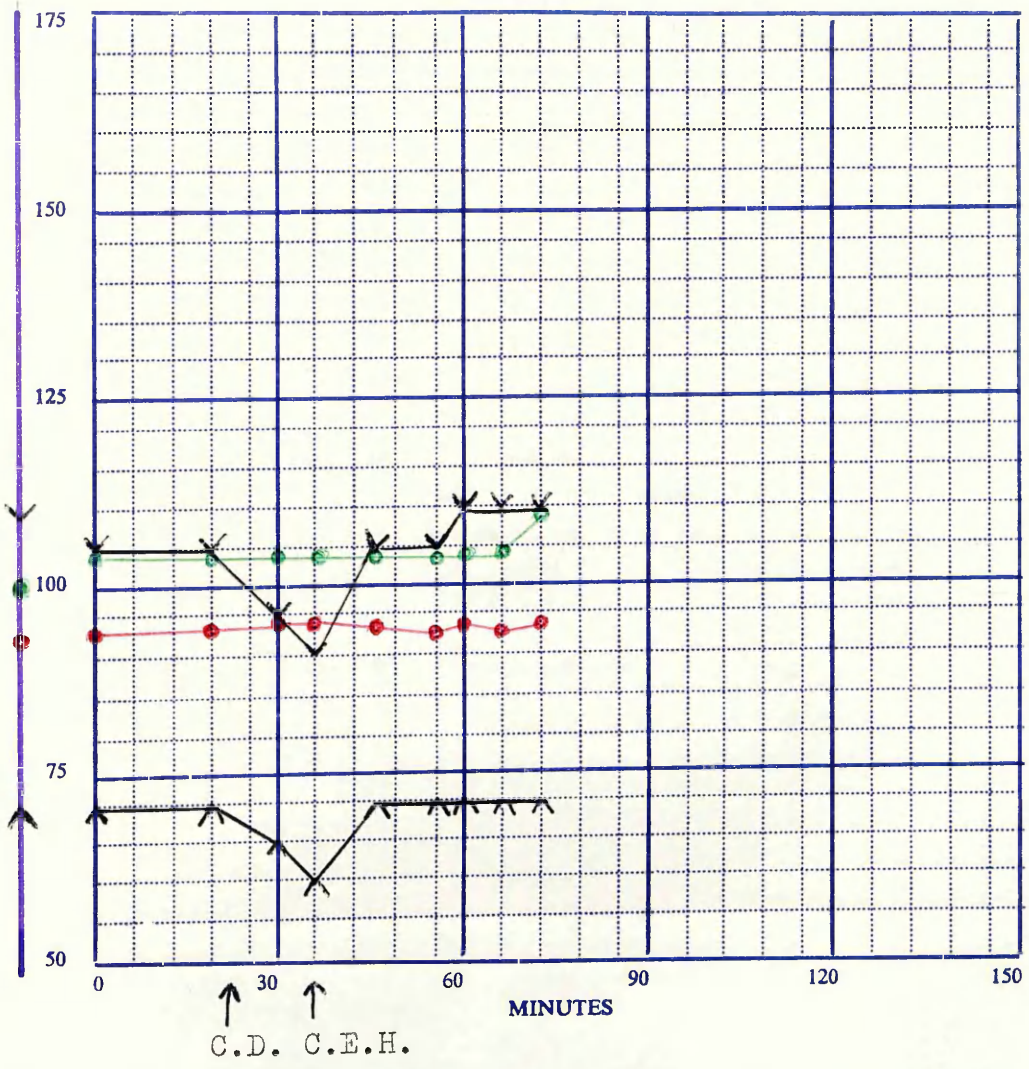
NOTES This is a repeat catheterisation on Case 66. The oximeter was unfortunately out of commission at this time.

CASE RECORD No.....72.....

Name J.McG. Age 14 Diagnosis P.S.

Weight 40 Kilos. Haemoglobin 105%

Premedication Promethazine 25 mgms.
Amylobarbitone 200 mgms.



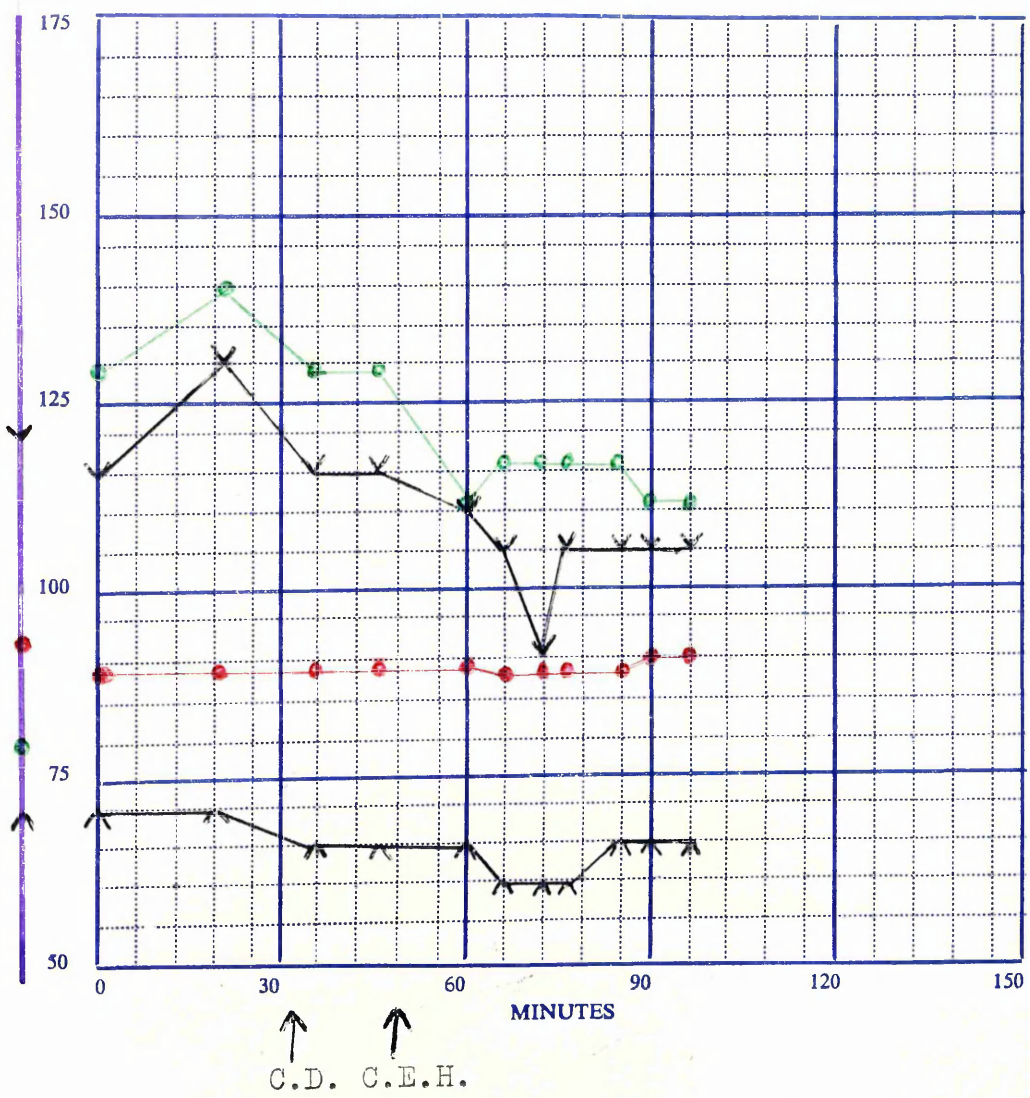
NOTES This child though apparently placid before the investigation complained of pain in the arm axilla and shoulder and despite reassurance was sobbing on occasion. At the periods of hypotension she appeared more relaxed.

CASE RECORD No.....73.....

Name A.H. Age 14. Diagnosis I.A.S.D.

Weight 37 Kilos. Haemoglobin 95%

Premedication Promethazine 25 mgms.
Amylobarbitone 200 mgms.



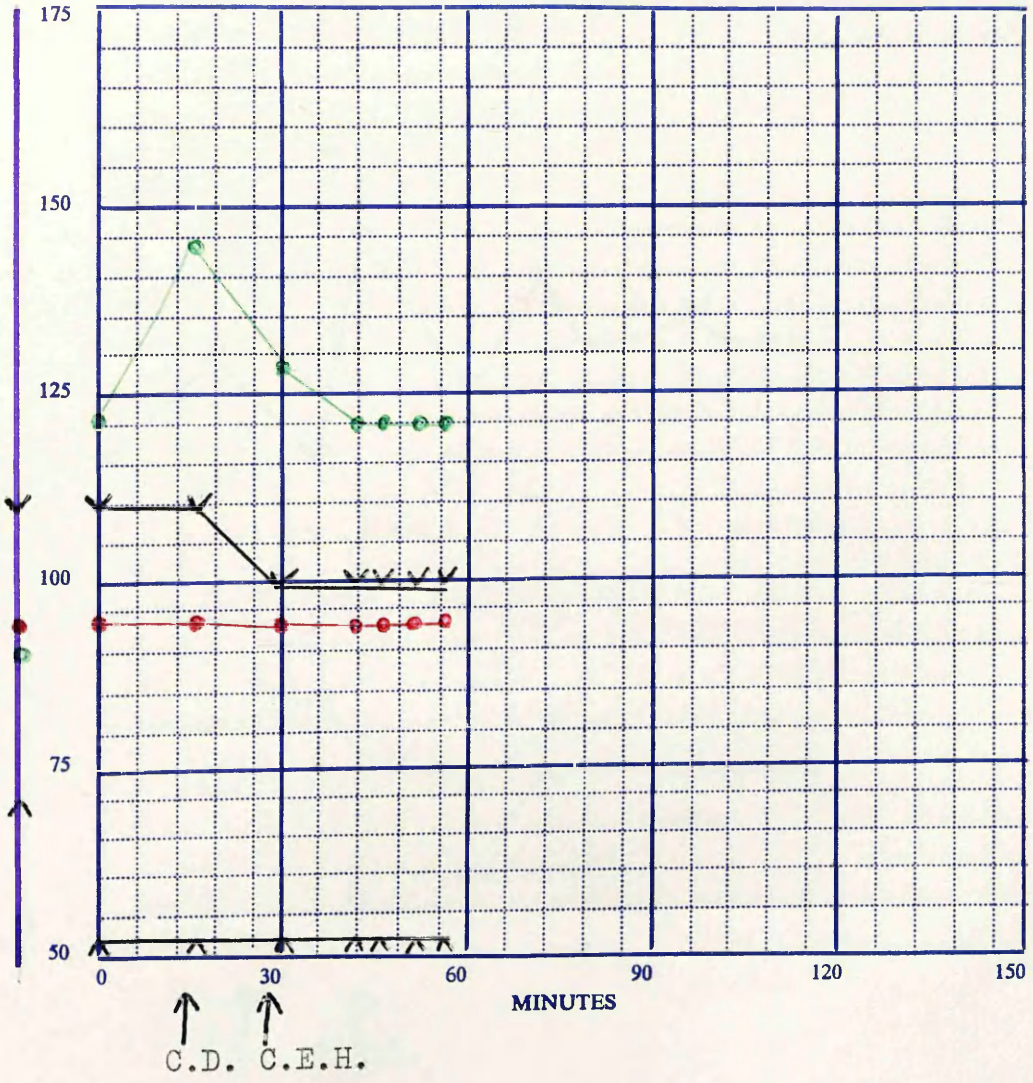
NOTES The patient was quite comfortable and it was noticeable that as he became more accustomed to his surroundings, he became drowsy. This was accompanied by the hypotension noticed in the later part of the investigation.

CASE RECORD No.....74.....

Name W.T. Age 14 Diagnosis A.S.

Weight 40.3 Kilos. Haemoglobin 90%

Premedication Promethazine 25 mgms.
Meprobamate 800 mgms.



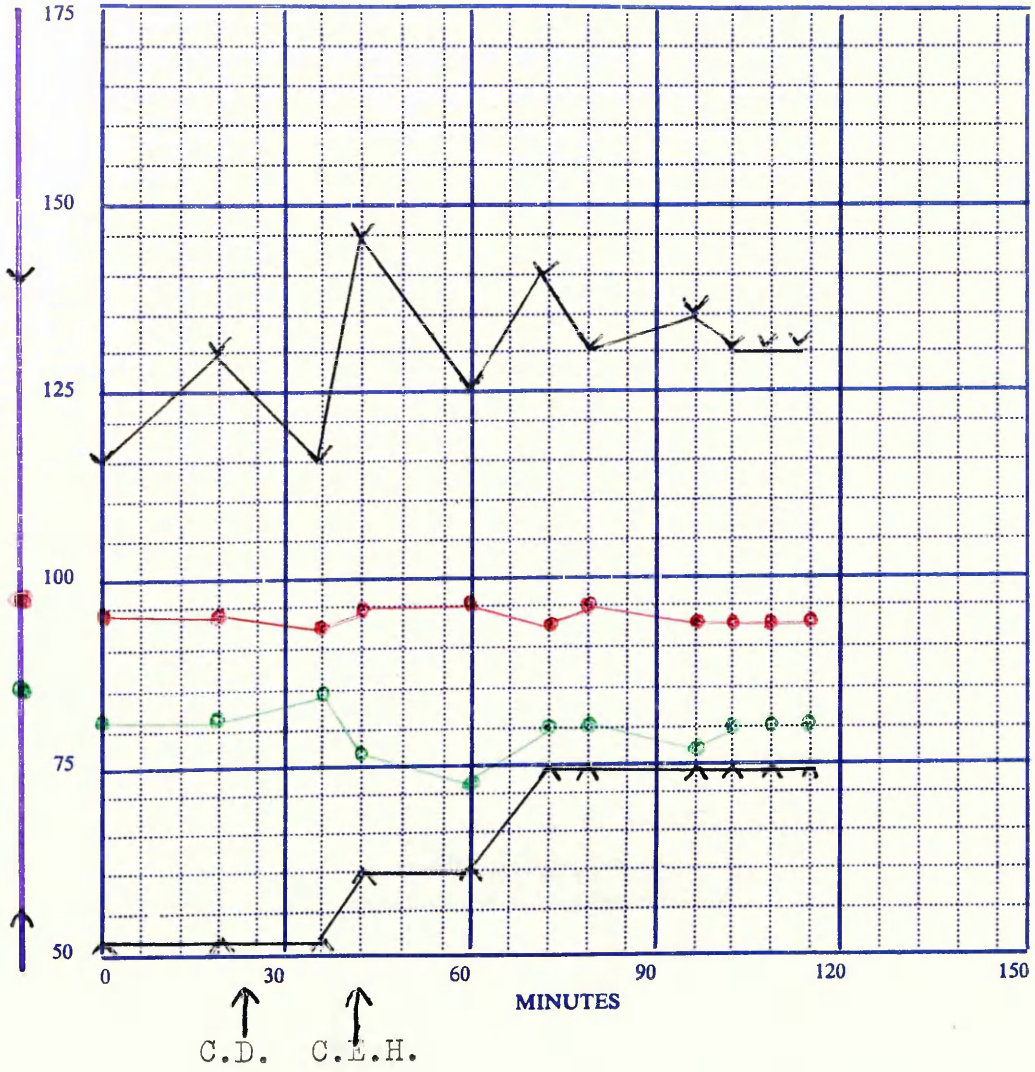
NOTES This was an uneventful catheterisation.

CASE RECORD No.....75.....

Name A.S. Age 15. Diagnosis I.V.S.D.

Weight 75 Kilos. Haemoglobin 100%

Premedication Promethazine 25 mgms.
Papaveratum 20 mgms.
Scopolamine 0.4 mgms.



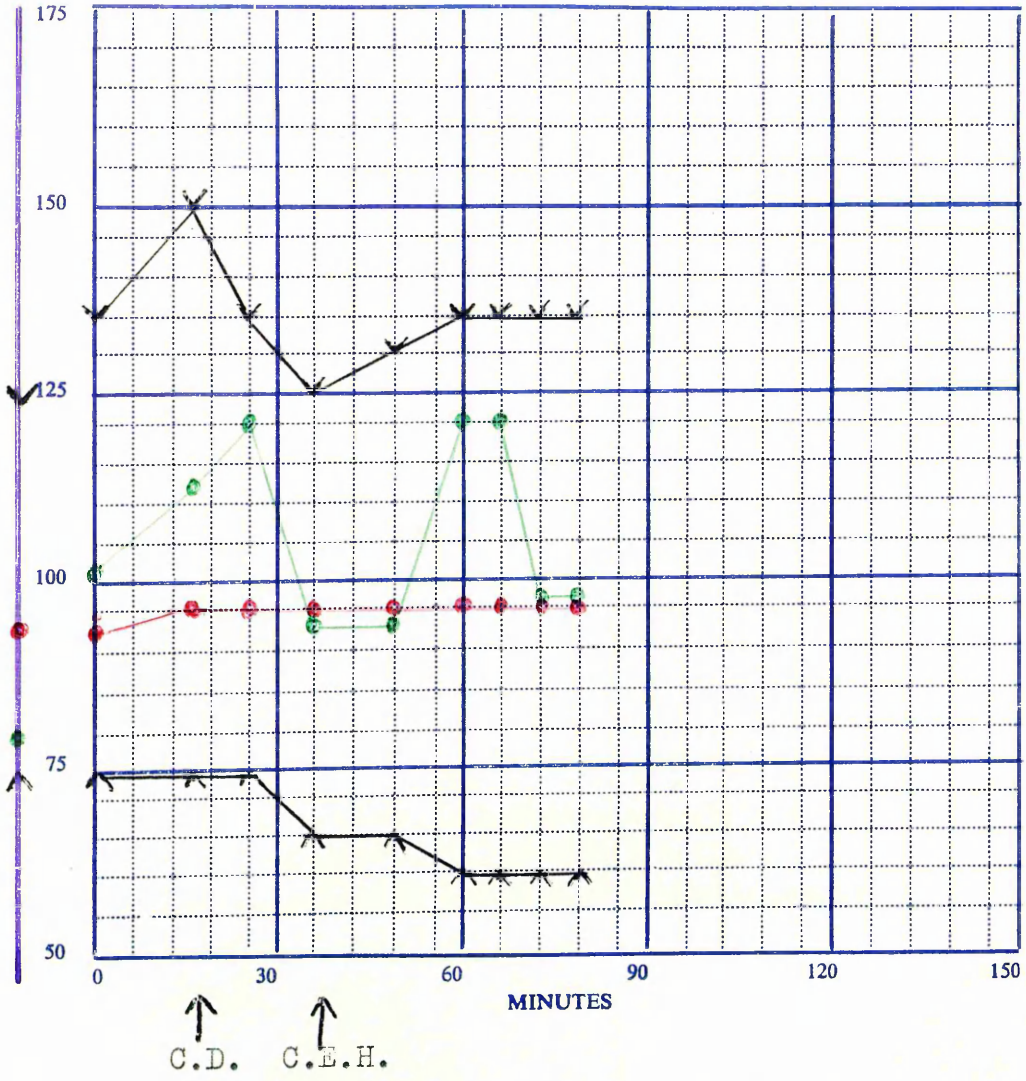
NOTES This boy when first brought to the ward ran away and was not seen for two days, hence the rather heavy premedication.
Arterial Analysis - O_2 saturation 93%
(Oximeter reading 92%)
pH 7.42
pCO_2 42.9 mmHg.

CASE RECORD No.....76.....

Name R.M. Age 15 Diagnosis P.S.

Weight 53 Kilos. Haemoglobin 105%

Premedication Papaveratum 20 mgms.
Scopolamine 0.4 mgms.



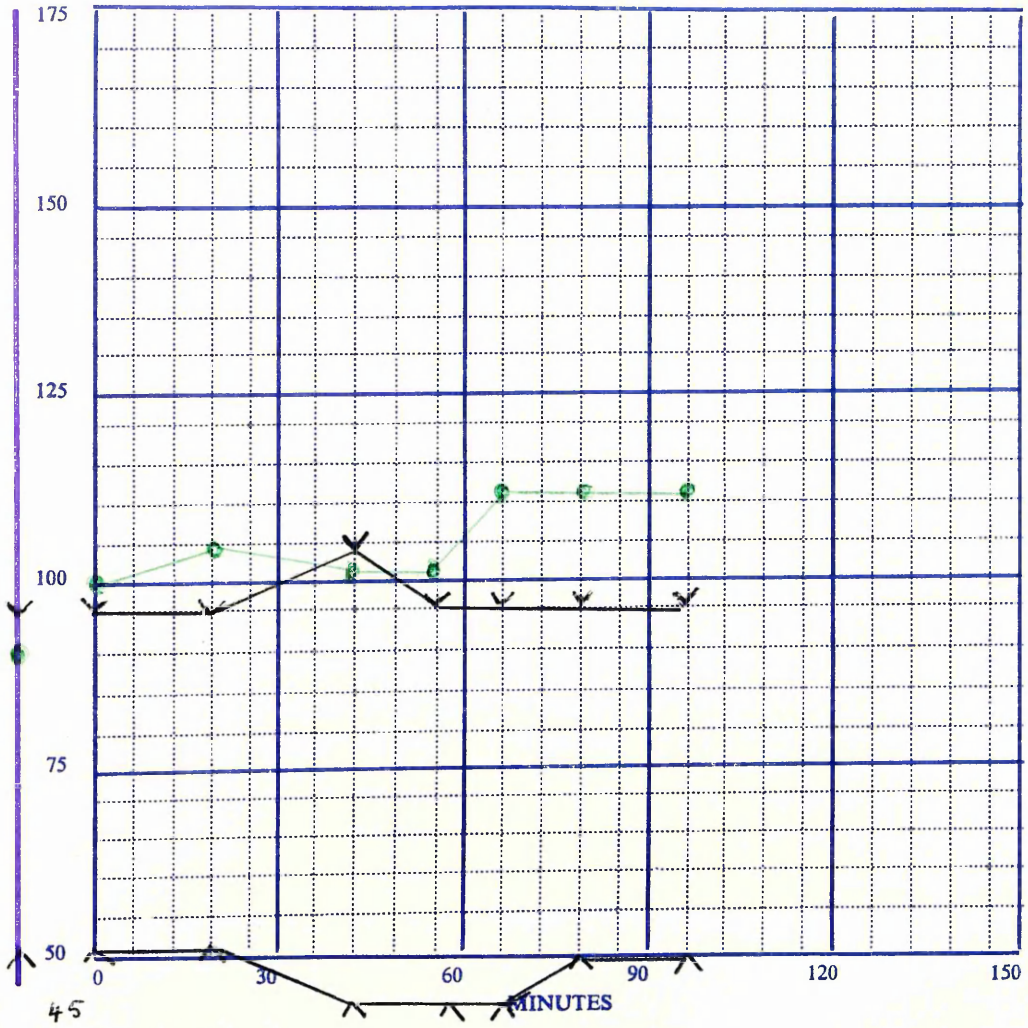
NOTES This was an uneventful catheterisation.

CASE RECORD No. 77.

Name W.S. Age 15 Diagnosis F.T.

Weight 44.5 Kilos. Haemoglobin 108%

Premedication Day before catheterisation - Meprobamate 400 mgms. at 4 p.m., 800 mgms. at 9 p.m.
Day of catheterisation - Meprobamate 400 mgms. at 6 a.m., 400 mgms. at 9 a.m.



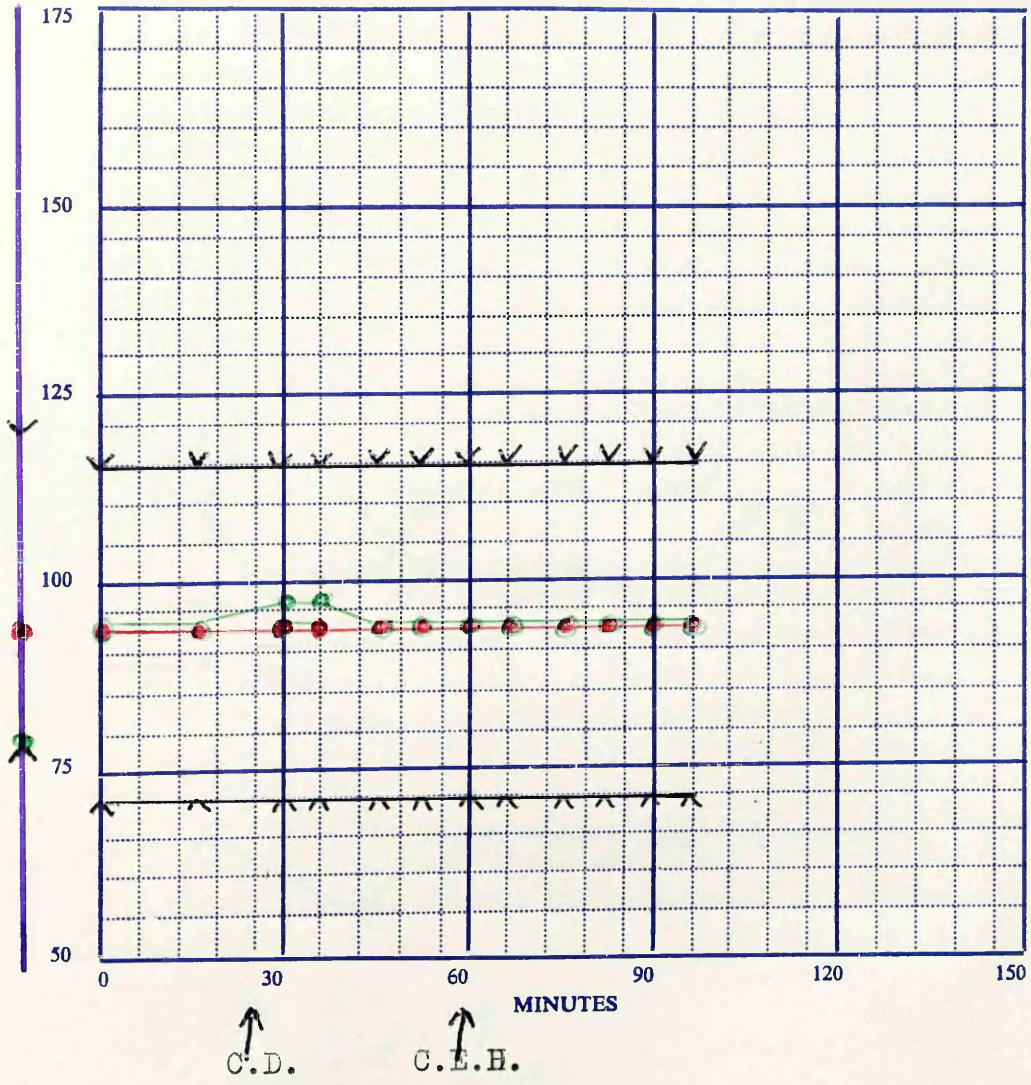
NOTES The child was quite alert but contented and co-operative. The oximeter earpiece was under repair.

CASE RECORD No.....78.....

Name M.C. Age 15 Diagnosis I.V.S.D.

Weight 58 Kilos. Haemoglobin 95%

Premedication Amylobarbitone 300 mgms.



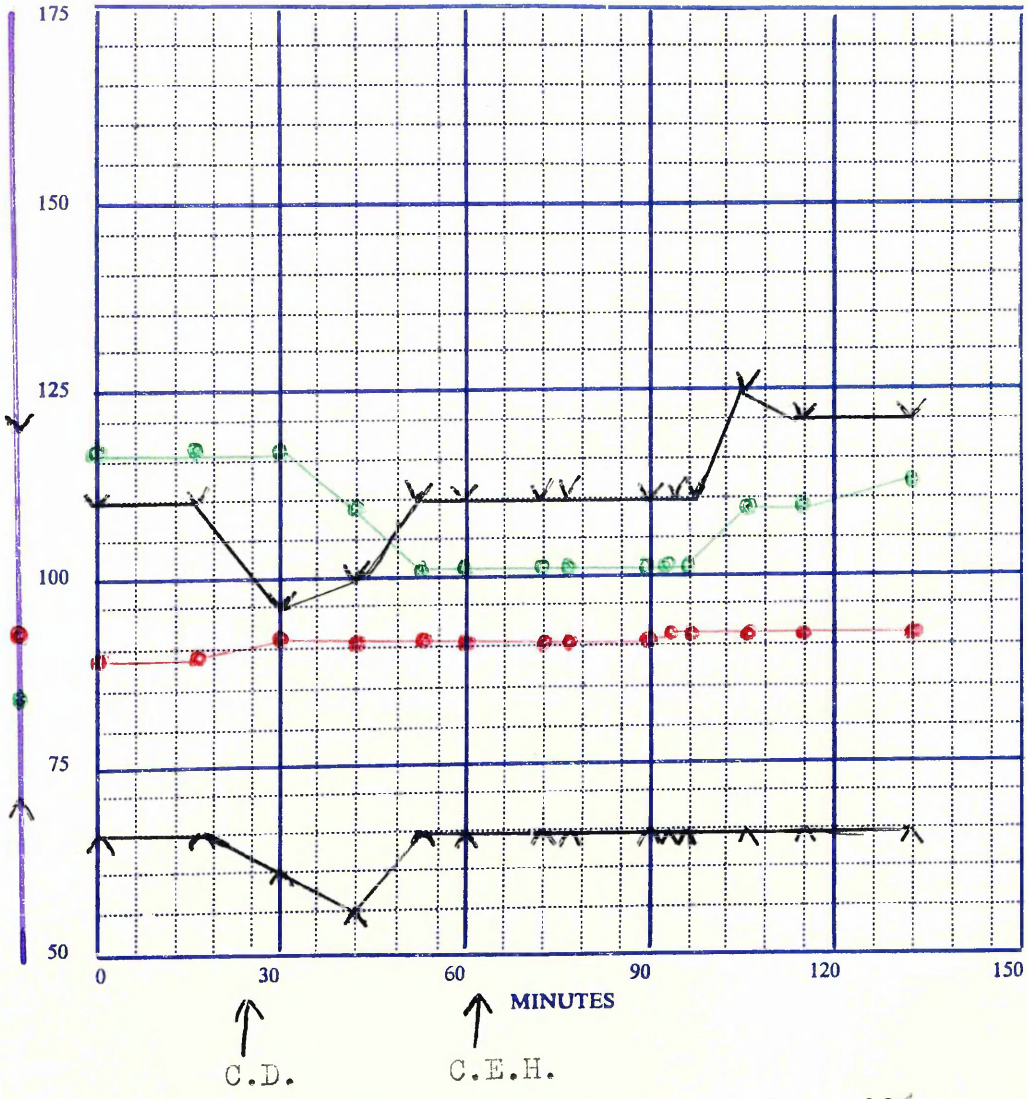
NOTES This girl was very nervous and reacted violently to attempts to take off blood in the ward. This accounts for the rather heavy premedication.

CASE RECORD No.....79.....

Name H.W. Age 16 Diagnosis P.S. + I.V.S.D.

Weight 53 Kilos. Haemoglobin 103%

Premedication Papaveratum 20 mgms.
Scopolamine 0.4 mgms.



NOTES Arterial analysis - O_2 saturation 92%
(Oximeter reading 90%)
pH 7.33
pCO_2 31.8 mmHg.

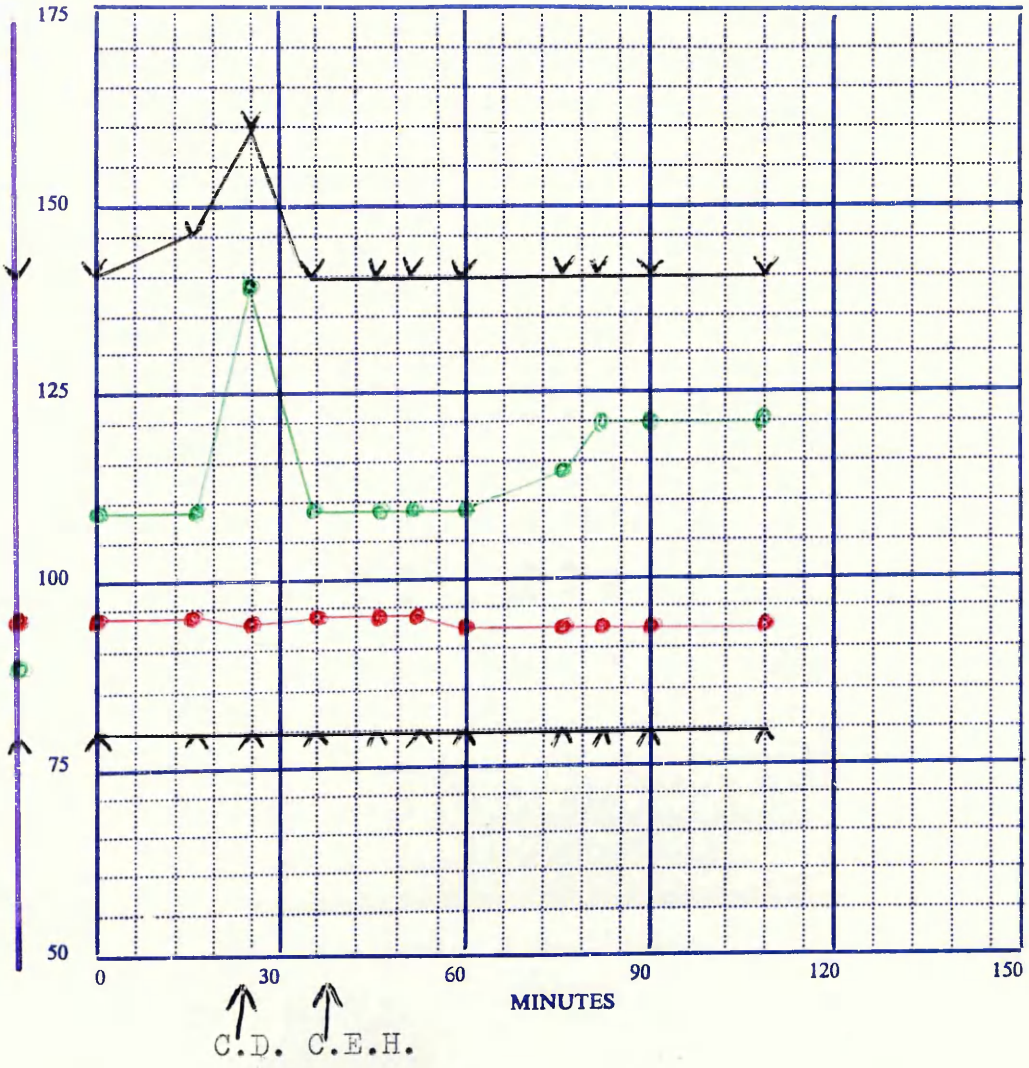
The patient on arrival in the X-ray room complained of nausea and would have preferred to be asleep.

CASE RECORD No.....80.....

Name J.T. Age 16 Diagnosis A.S.

Weight 52.5 Kilos. Haemoglobin 105%

Premedication Pentobarbitone 100 mgms.
Promethazine 25 mgms.
Pethidine 50 mgms.



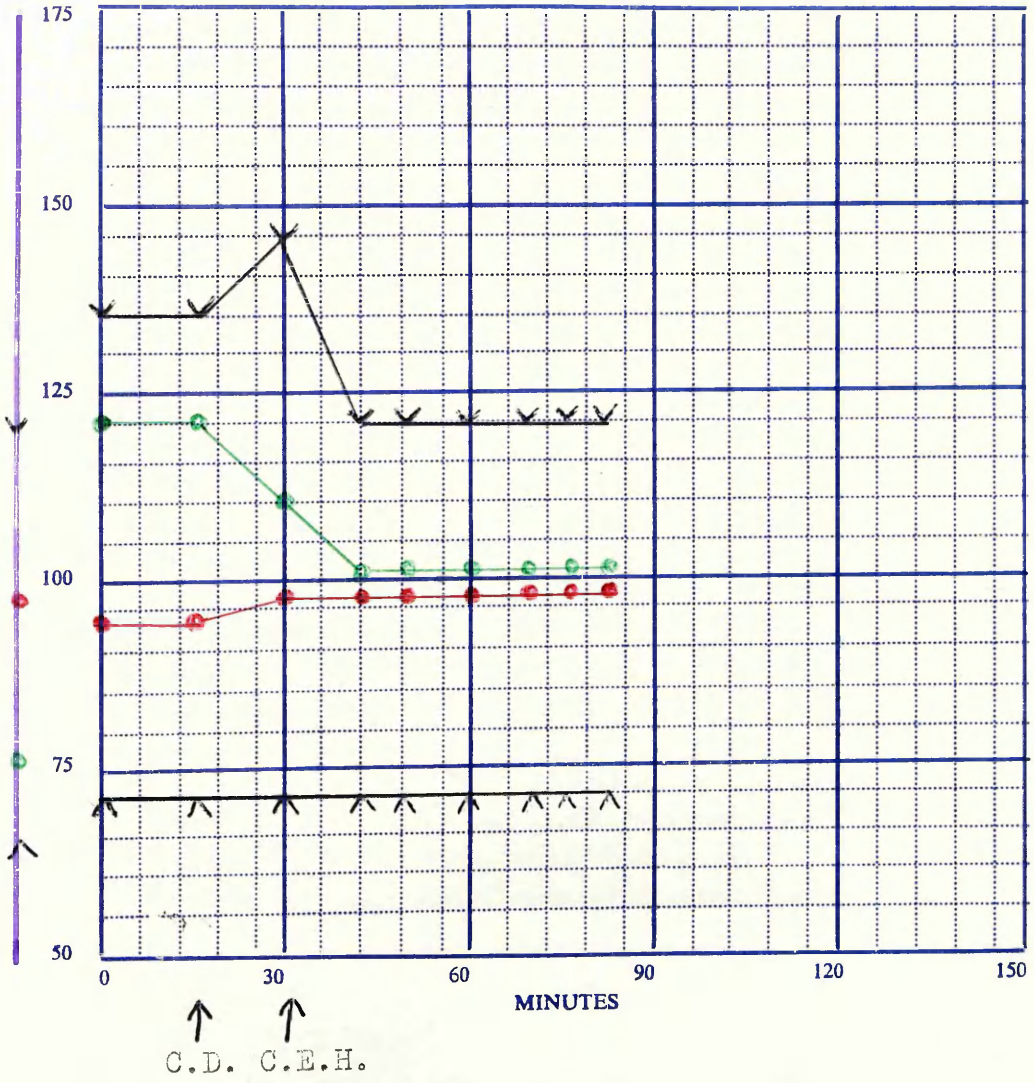
NOTES During the proceedings the girl was on occasion tearful despite reassurance.

CASE RECORD No. 81.

Name P.McG. Age 16 Diagnosis P.D.A.

Weight 48.5 Kilos. Haemoglobin 78%

Premedication Pentobarbitone 200 mgms.



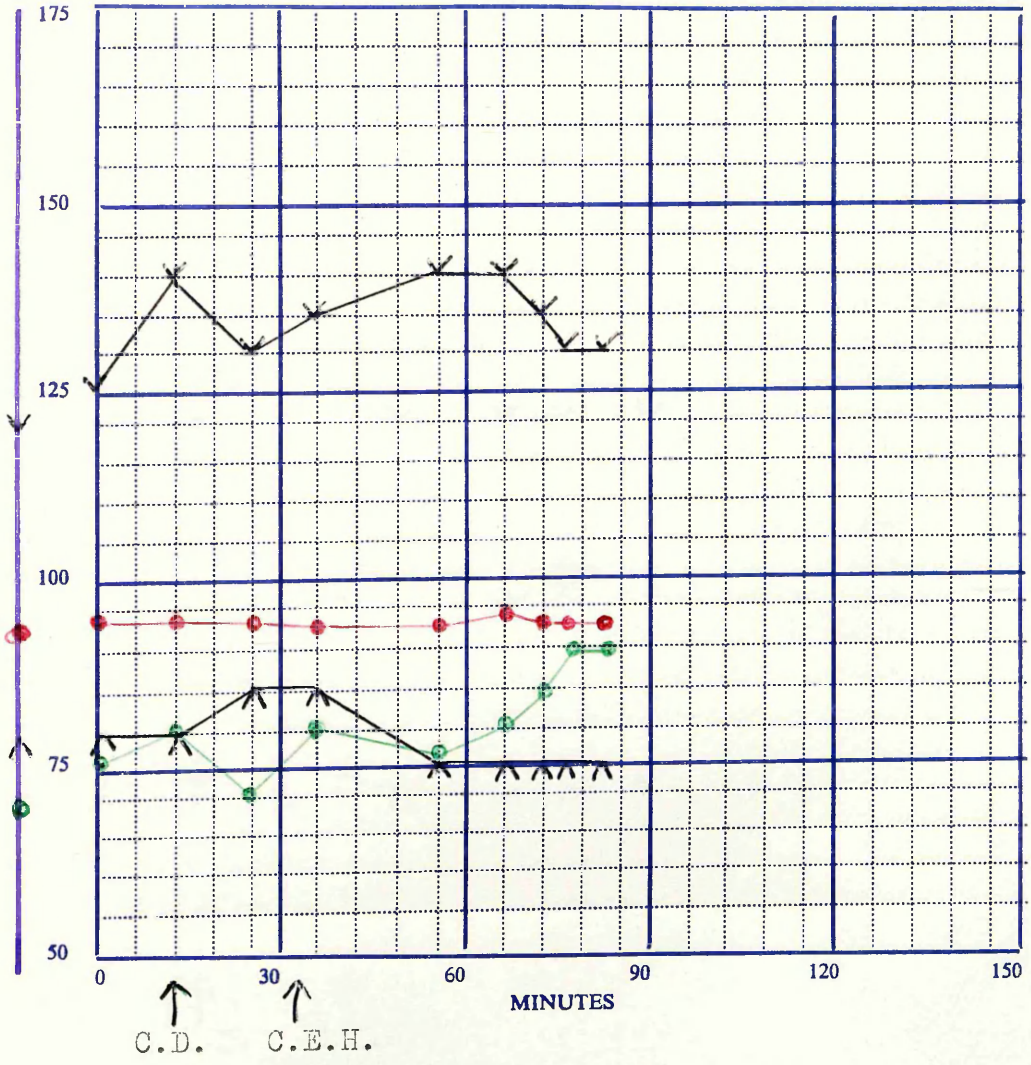
NOTES After the Pentobarbitone, the patient became quite excited and on arrival in the X-ray Department she was still apprehensive. As time passed she settled down.

CASE RECORD No. 82.

Name P.McS. Age 16 Diagnosis I.V.S.D.

Weight 66 Kilos. Haemoglobin 120%

Premedication Amylobarbitone 200 mgms.



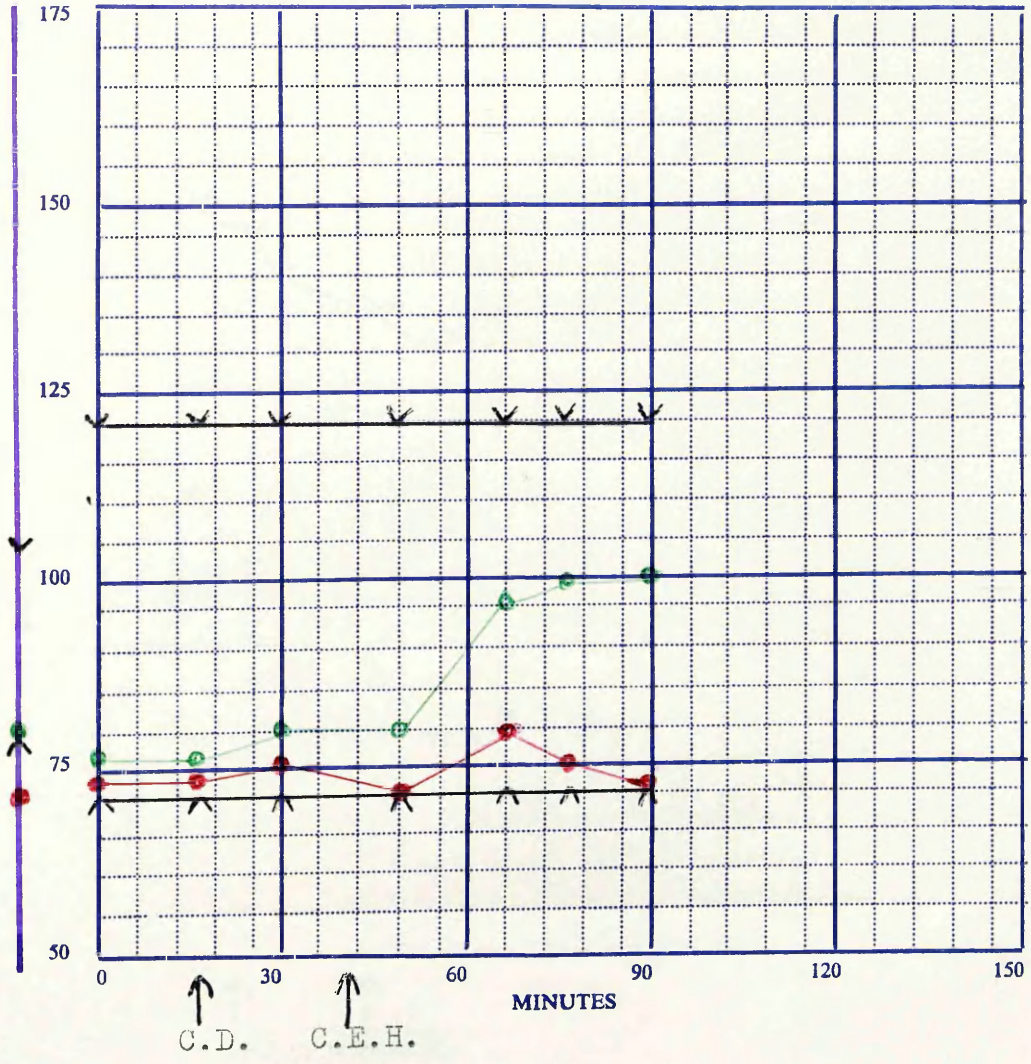
NOTES During the catheterisation the patient complained of pain in the chest whenever the catheter impinged on the pulmonary valve.

CASE RECORD No.....83.....

Name P.D. Age 16 Diagnosis F.T.

Weight 64 Kilos. Haemoglobin 170%

Premedication Pentobarbitone 200 mgms.



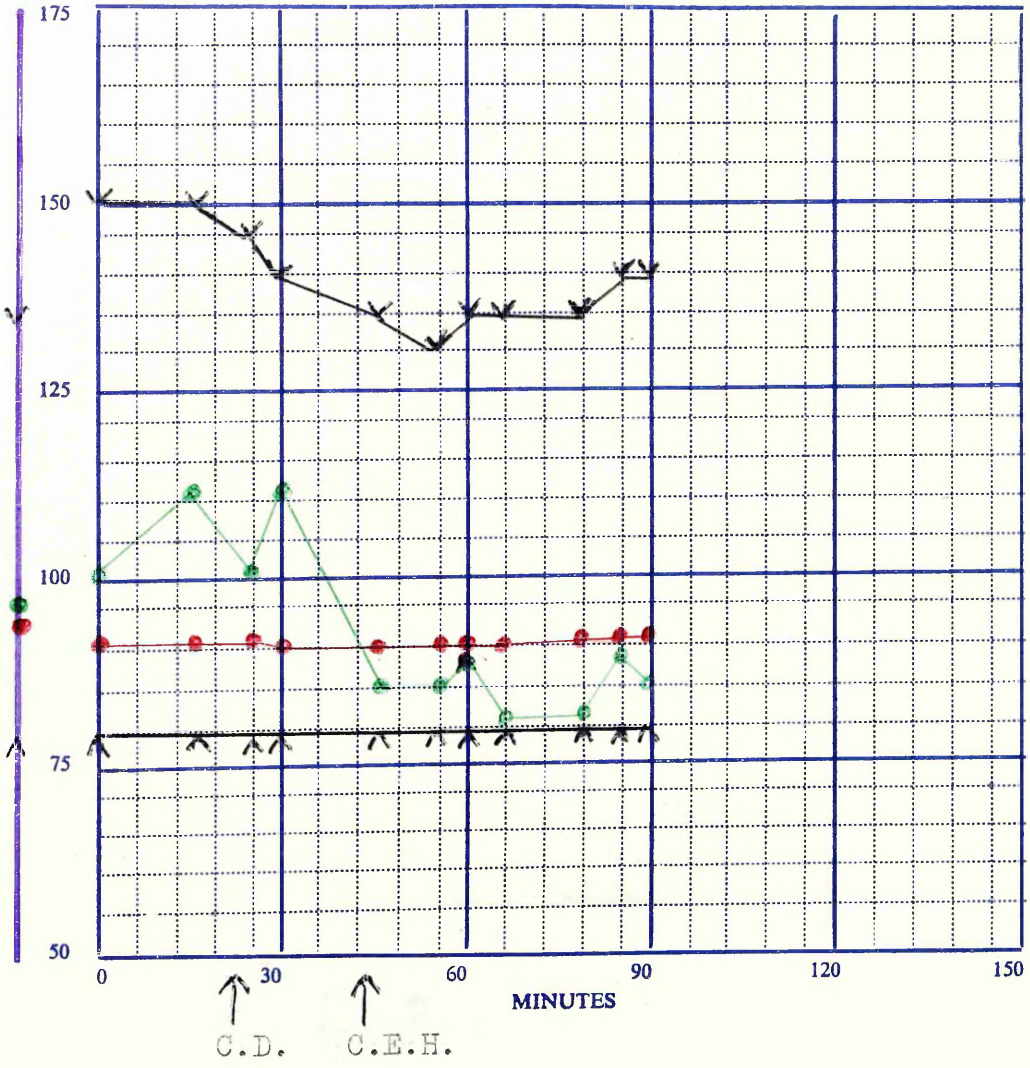
NOTES This was an uneventful catheterisation.

CASE RECORD No.....84.....

Name J.B. Age 17 Diagnosis MS.

Weight 51 Kilos. Haemoglobin 92%

Premedication Promethazine 25 mgms.
Papaveratum 20 mgms.
Scopolamine 0.4 mgms.



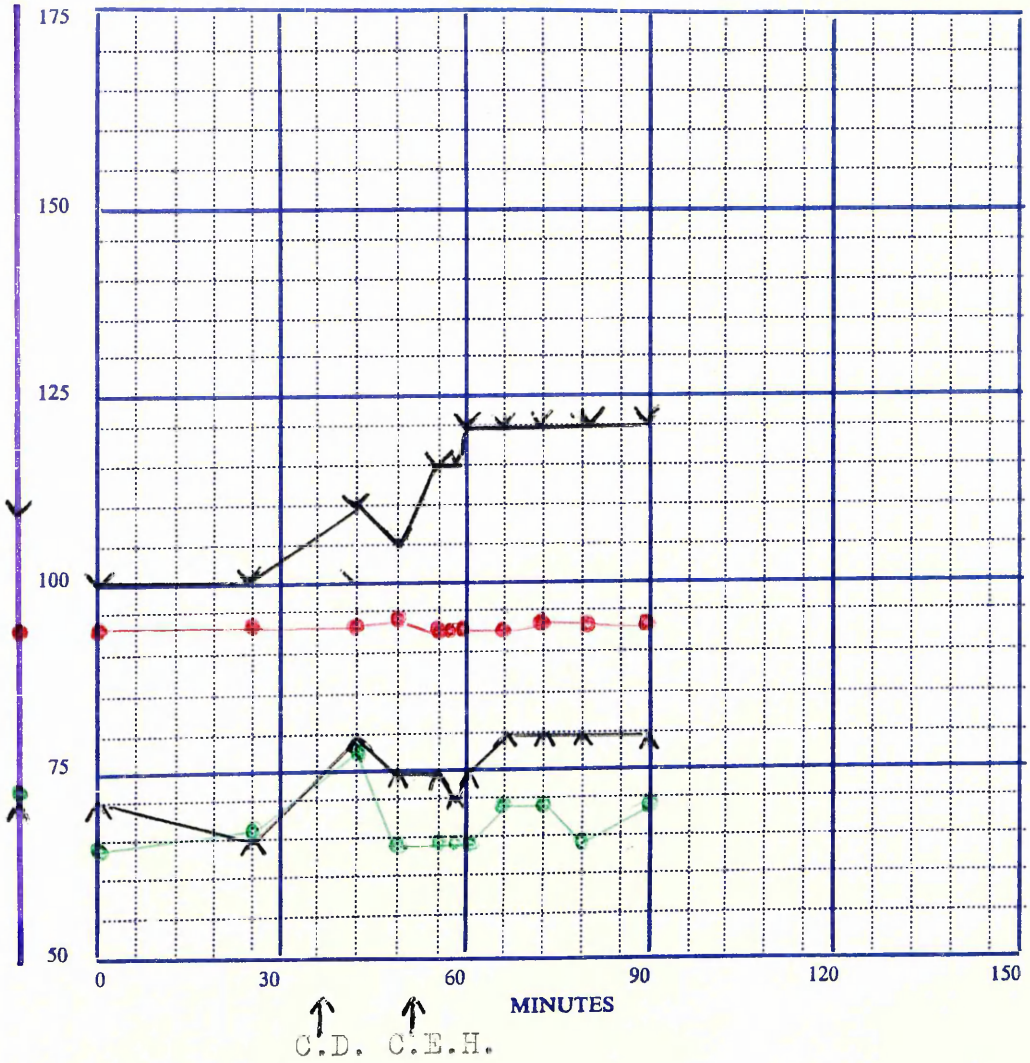
NOTES Arterial analysis - pCO_2 60 mmHg.

CASE RECORD No.....85.....

Name E.M. Age 17 Diagnosis I.A.S.D.

Weight 48 Kilos. Haemoglobin 104%

Premedication Papaveratum 25 mgms.
Scopolamine 0.4 mgms.



NOTES This was an uneventful catheterisation.

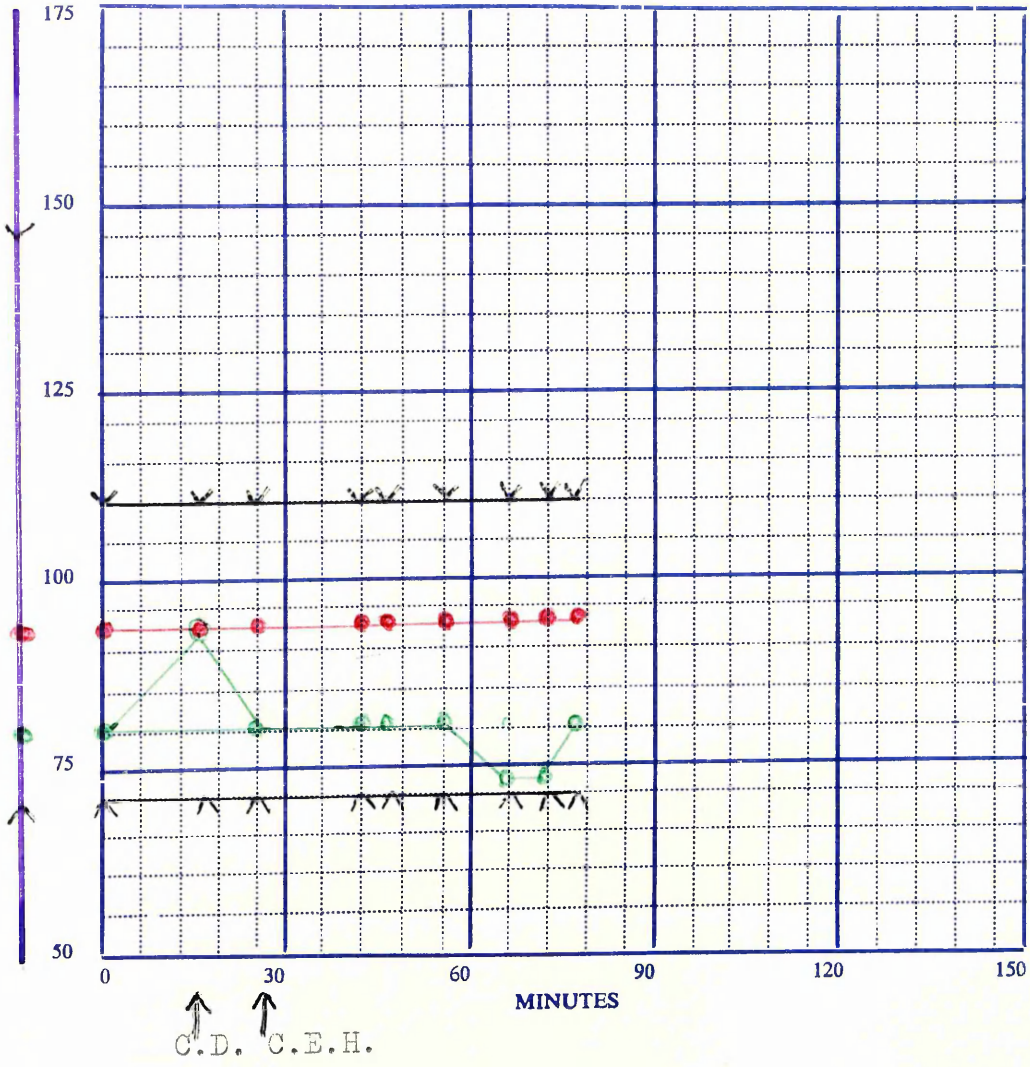
Arterial analysis - pH 7.32
pCO_2 57 mmHg.

CASE RECORD No. 86.

Name J.S. Age 17 Diagnosis I.V.S.D.

Weight 68 Kilos. Haemoglobin 108%

Premedication Promethazine 25 mgms.
Amylobarbitone 200 mgms.



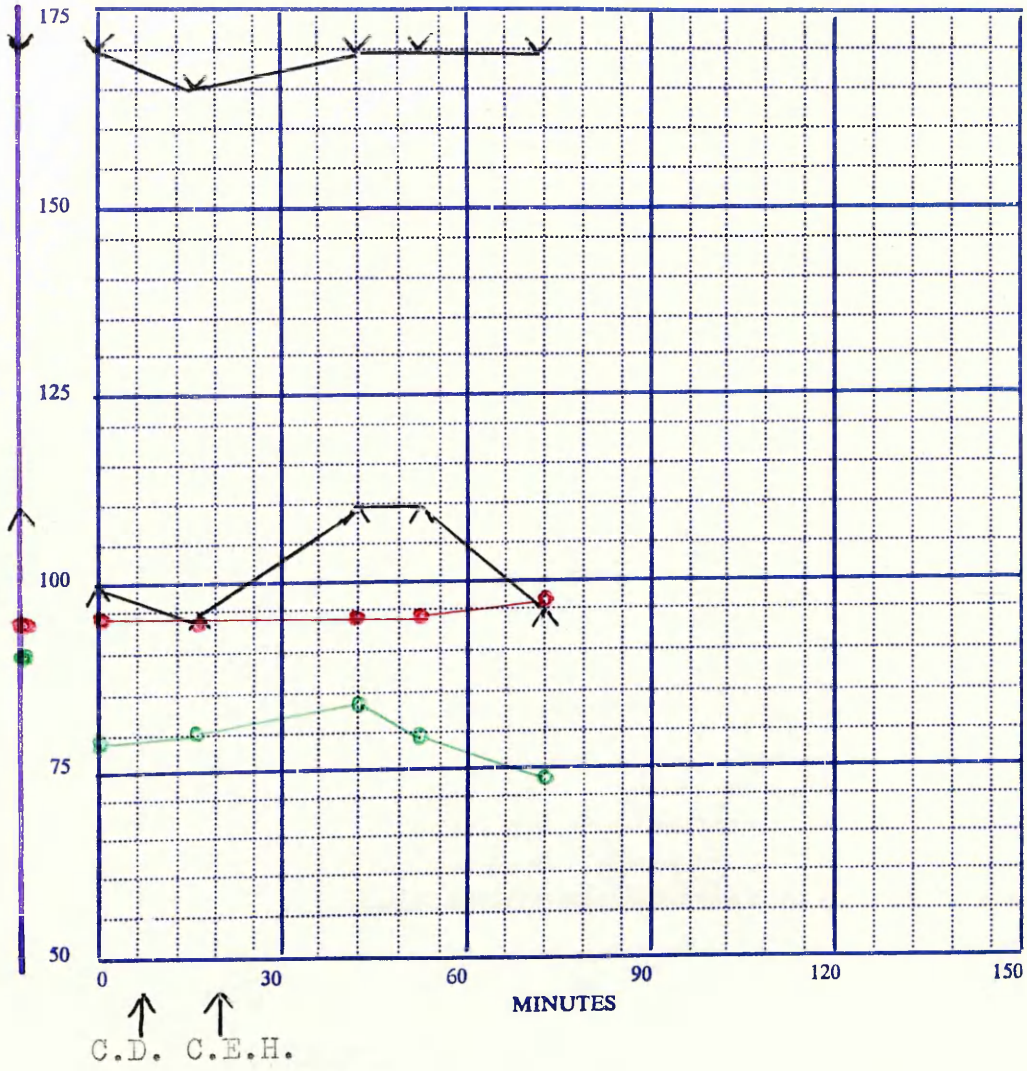
NOTES As the catheter entered the patient's chest, he complained of retrosternal pain.

CASE RECORD No.....87.....

Name H.S. Age 17 Diagnosis C. of A.

Weight 42 Kilos. Haemoglobin 100%

Premedication Meprobamate 400 mgms.



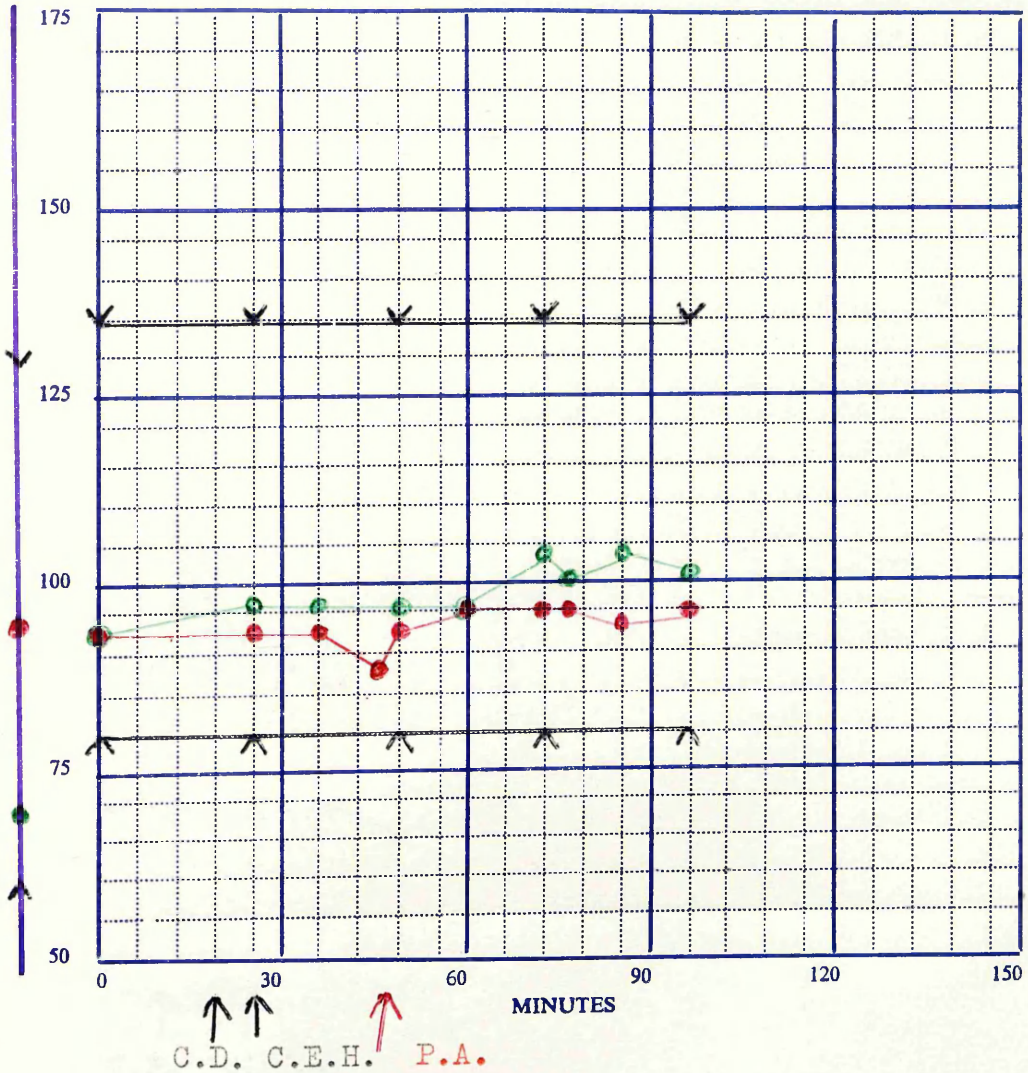
NOTES The patient on this occasion was alert and co-operative.

CASE RECORD No. 88.

Name J.W. Age 17 Diagnosis P.S.

Weight 54 Kilos. Haemoglobin 98%

Premedication Promethazine 25 mgms.
Pentobarbitone 200 mgms.



NOTES

During the catheterisation the patient complained bitterly of pain in his left shoulder and chest and sobbed for the first half hour. The drop in arterial oxygen saturation when the catheter tip was in the pulmonary artery can be seen.

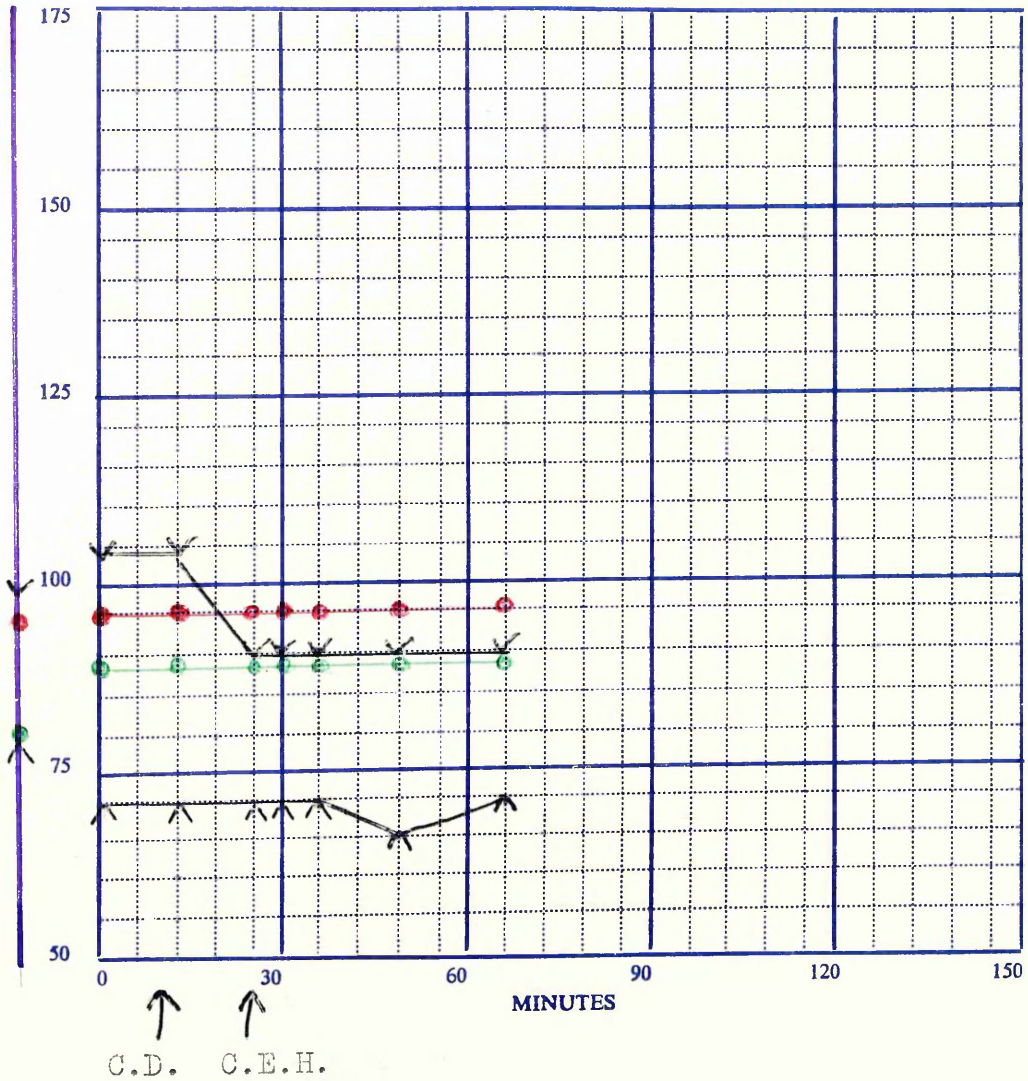
Arterial analysis - O_2 sat. 90% (oximeter 92%)

CASE RECORD No. 89.

Name G.McD. Age 18 Diagnosis A.S.

Weight 46 Kilos. Haemoglobin 88%

Premedication Pentobarbitone 200 mgms.



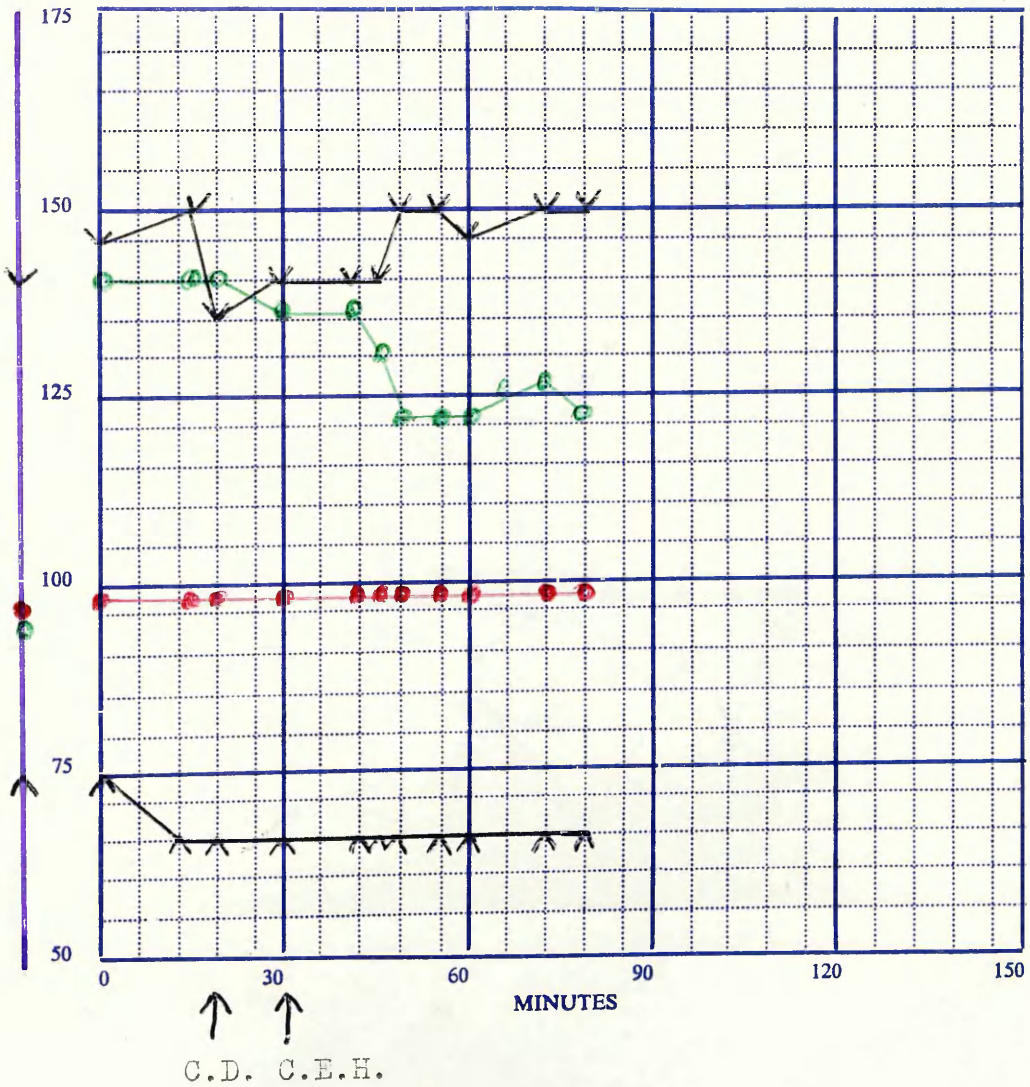
NOTES The patient exhibited a certain degree of spontaneous restlessness throughout the procedure. How much this could be attributed to Pentobarbitone and how much to mental deficiency is difficult to estimate.

CASE RECORD No. 90.

Name M.McC. Age 18 Diagnosis I.V.S.D.

Weight 59 Kilos. Haemoglobin 96%

Premedication Papaveratum 20 mgms.
Scopolamine 0.4 mgms.



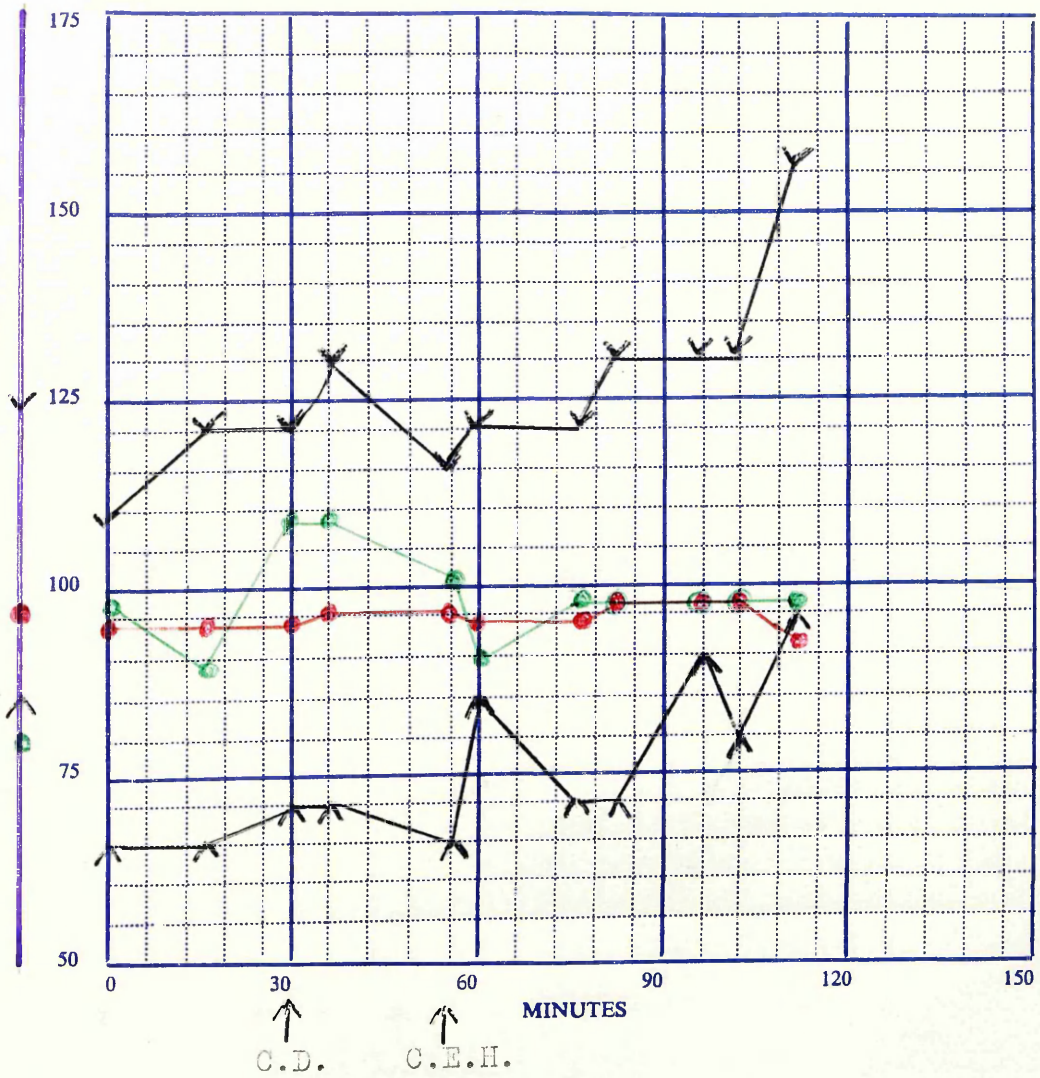
NOTES Arterial analysis - Oxygen saturation 96%
(oximeter reading 97%)
pCO_2 30.9 mmHg.

CASE RECORD No. 91.

Name R.H. Age 18 Diagnosis I.V.S.D.

Weight 59 Kilos. Haemoglobin 112%

Premedication Promethazine 25 mgms.
Papaveratum 20 mgms.
Scopolamine 0.4 mgms.



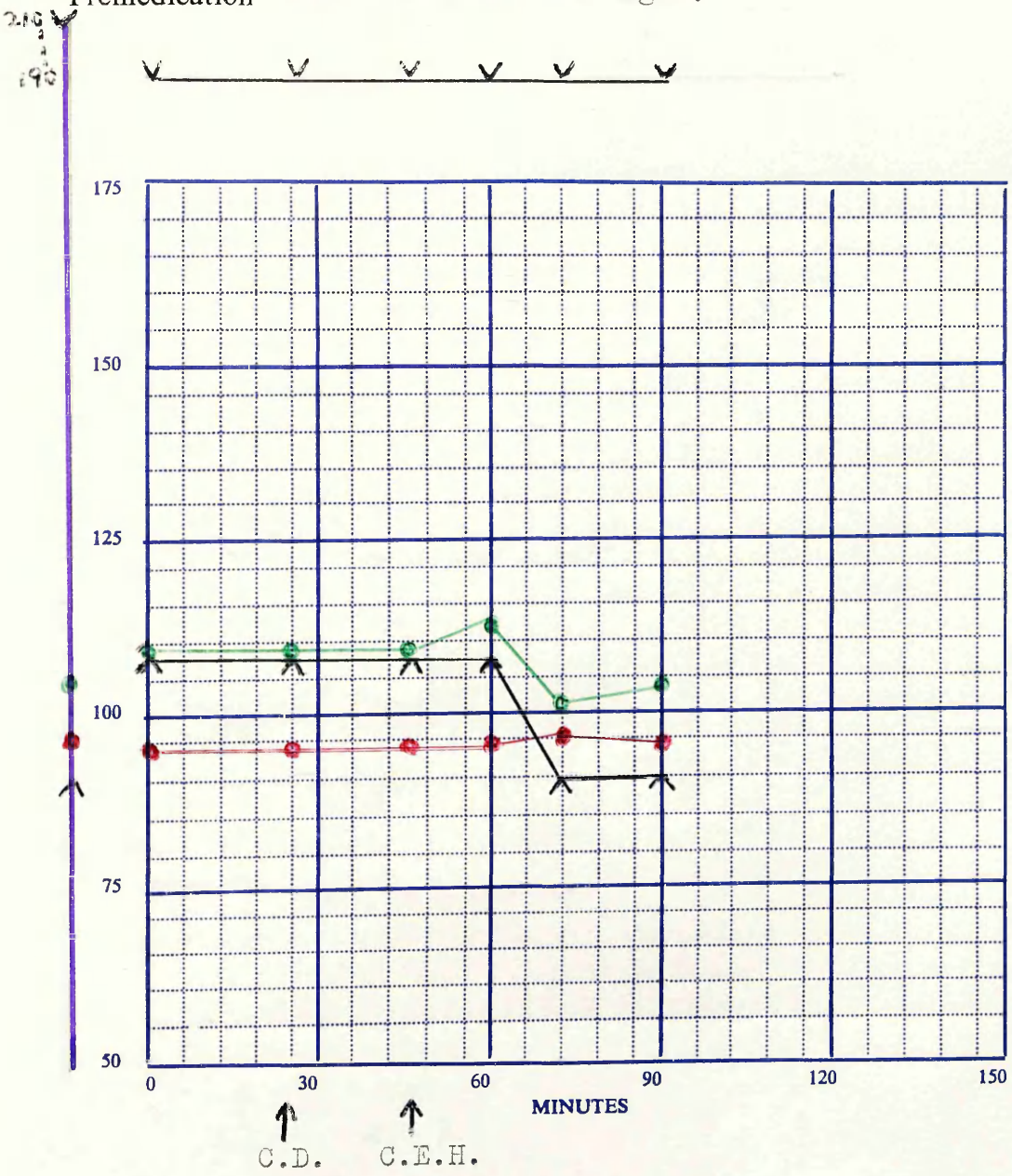
NOTES Arterial analysis - pH 7.43
pCO_2 39.6 mmHg.
Oxygen saturation 92%
(oximeter reading 92%)

CASE RECORD No.....92.....

Name M.M. Age 18 Diagnosis C. of A.

Weight 49 Kilos. Haemoglobin 95%

Premedication Pentobarbitone 200 mgms.



NOTES The patient was adequately sedated throughout the procedure.

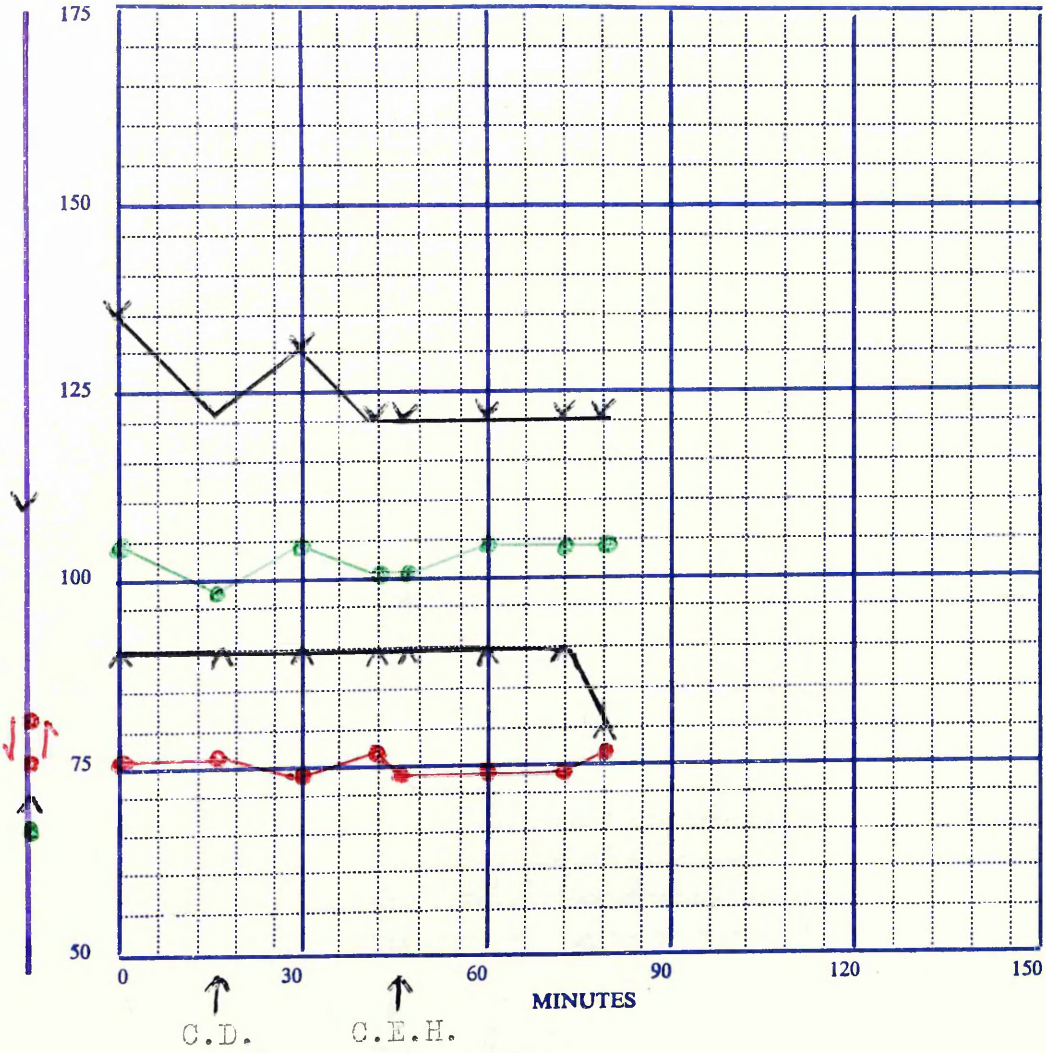
Arterial analysis - Oxygen saturation 95.2%
(oximeter reading 94%)

CASE RECORD No. 93.

Name G.C. Age 18 Diagnosis F.T.

Weight 50.2 Kilos. Haemoglobin 105%

Premedication Pethidine 100 mgms.
Promethazine 50 mgms.
Atropine 0.6 mgms.



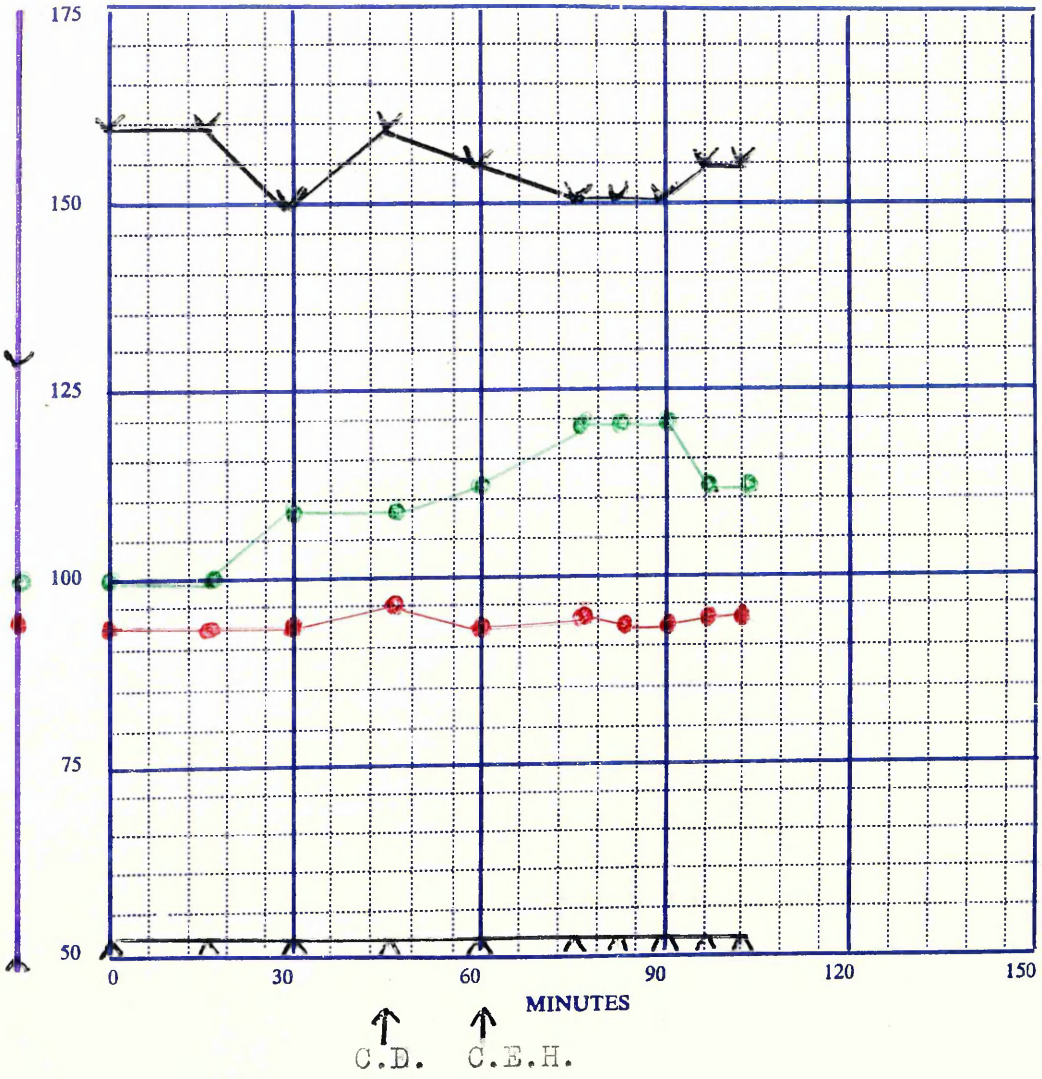
NOTES Apart from technical difficulties in passing the catheter, the procedure passed off smoothly.

CASE RECORD No.....94.....

Name K.McG. Age 18 Diagnosis A.S. and M.S.

Weight 53.3 Kilos. Haemoglobin 82%

Premedication Amylobarbitone 200 mgms.



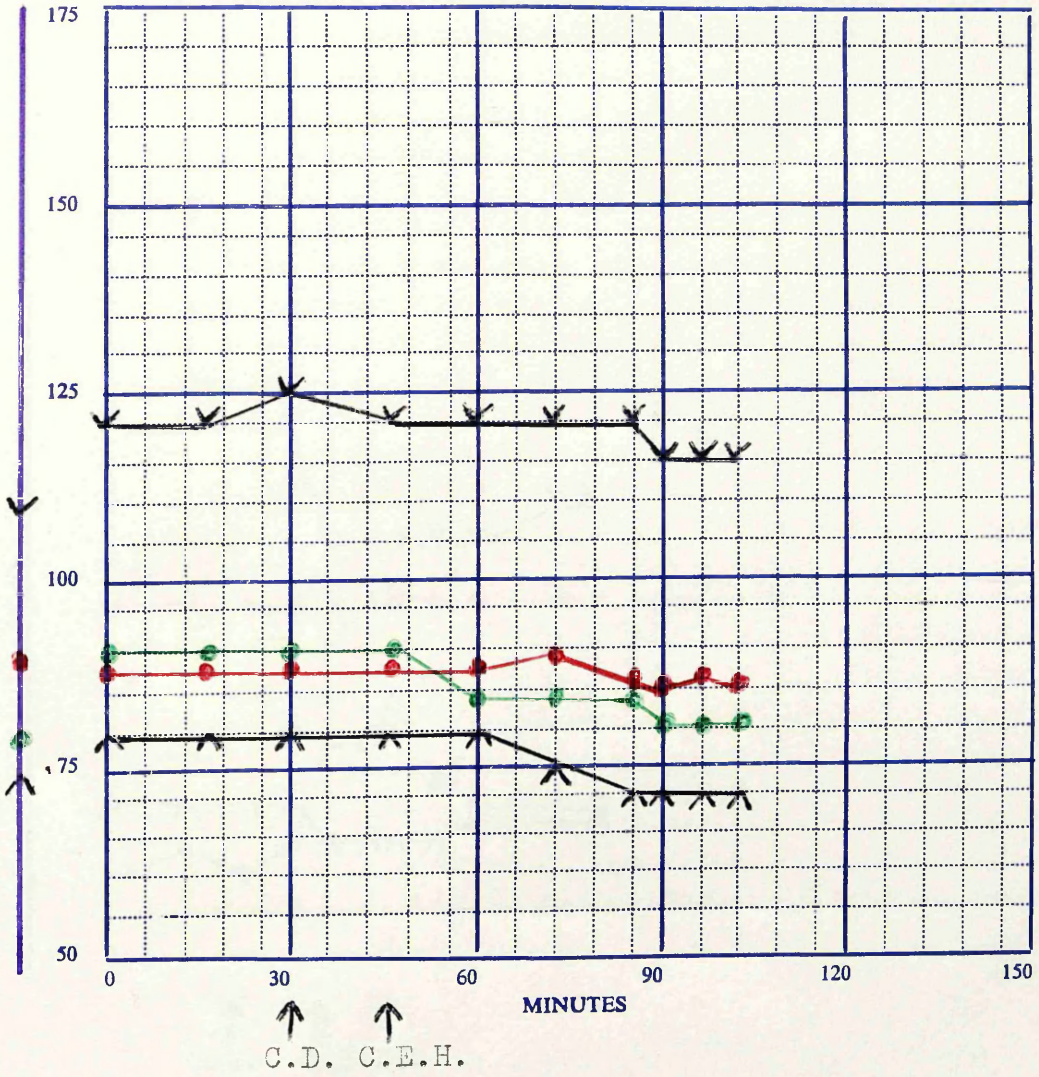
NOTES This was an uneventful catheterisation.

CASE RECORD No.....95.....

Name E.R. Age 18 Diagnosis P.H.

Weight 42.5 Kilos. Haemoglobin 108%

Premedication Pentobarbitone 200 mgms.



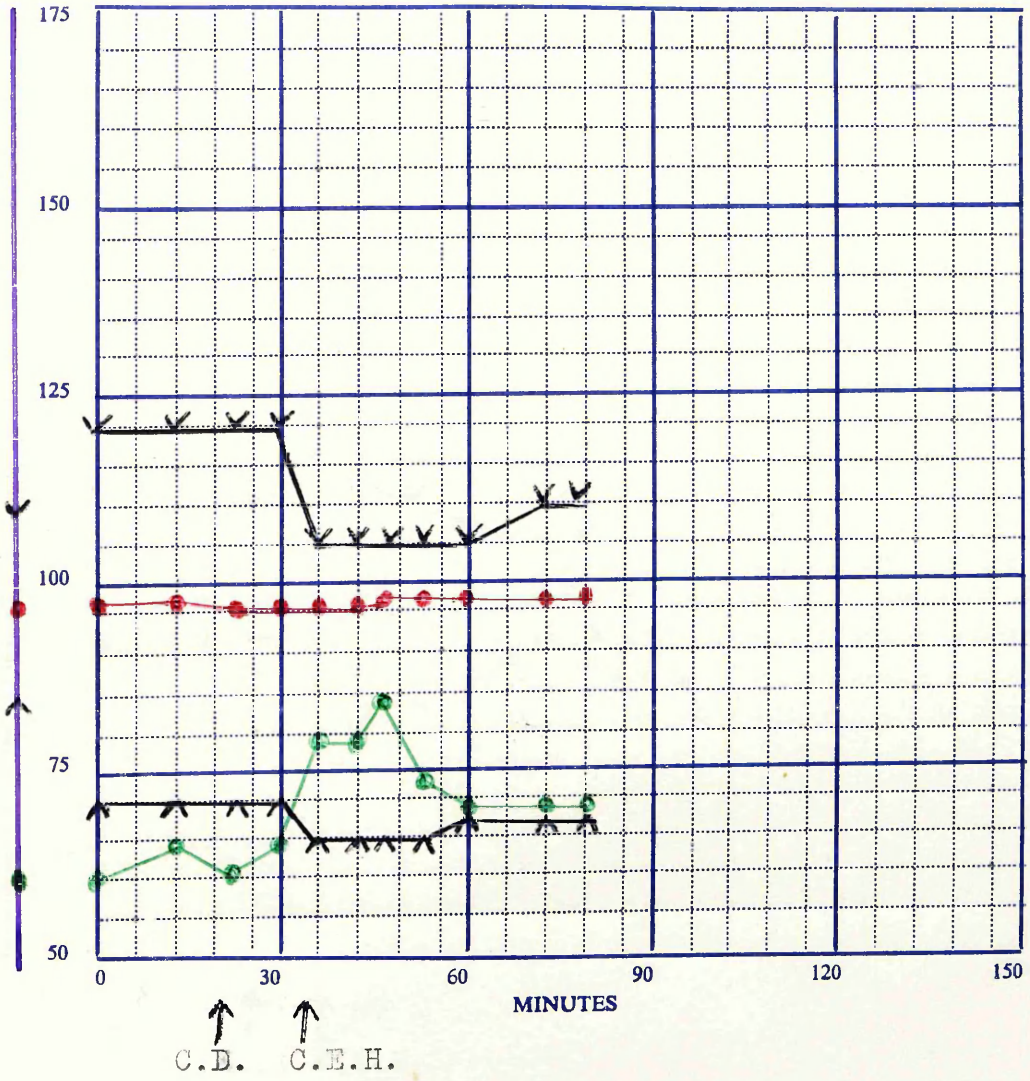
NOTES This was an uneventful catheterisation.

CASE RECORD No.....96.....

Name H.B. Age 19 Diagnosis Dextrocardia.

Weight 56 Kilos. Haemoglobin 80%

Premedication Promethazine 25 mgms.
Amylobarbitone 200 mgms.



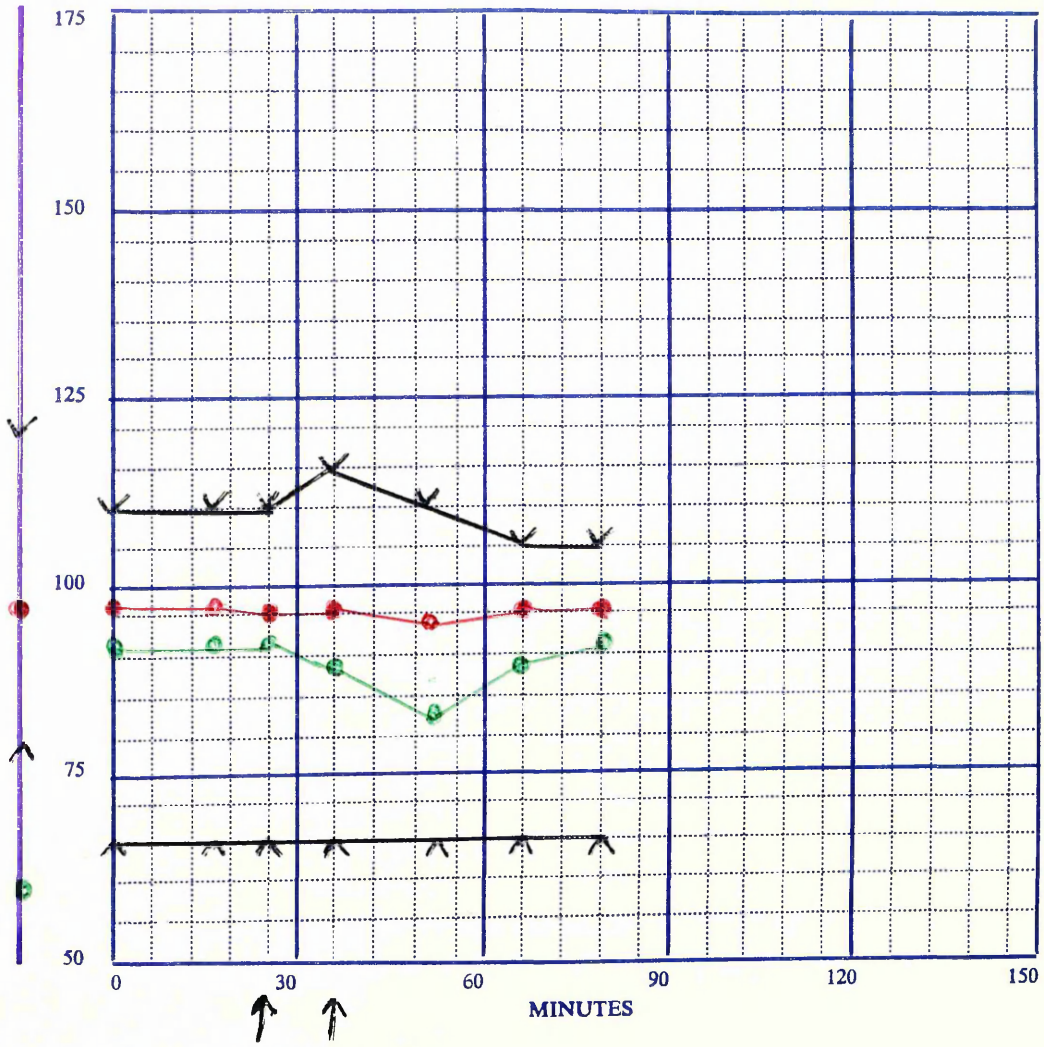
NOTES The patient complained of slight pain in the arm as the catheter was passing.

CASE RECORD No. 97.

Name S.M. Age 19 Diagnosis MS.

Weight 71 Kilos. Haemoglobin 93%

Premedication Amylobarbitone 200 mgms.



C.D. C.E.H.

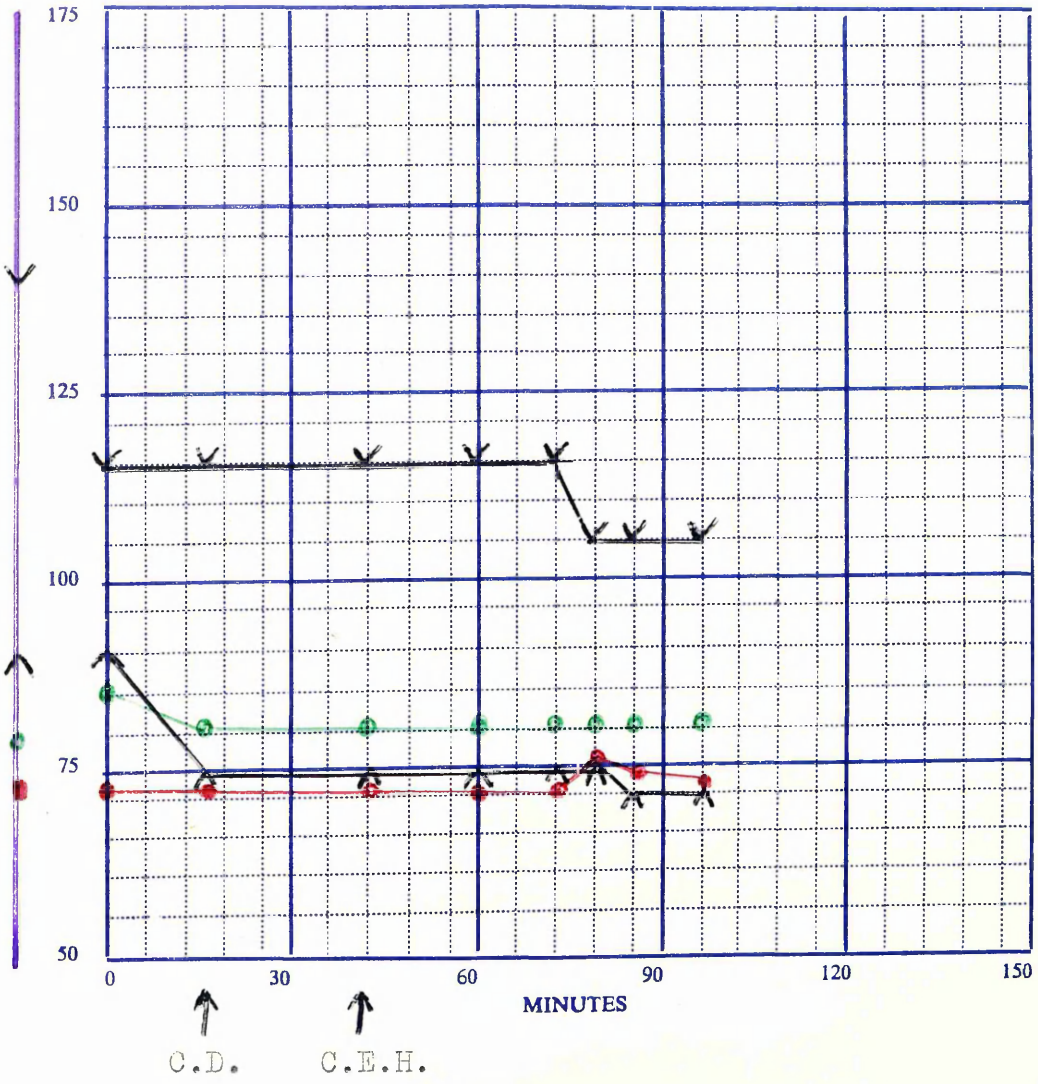
NOTES This was an uneventful catheterisation.

CASE RECORD No. 98.

Name W.C. Age 20. Diagnosis F.T.

Weight 52.3 Kilos. Haemoglobin 150%

Premedication Pentobarbitone 200 mgms.



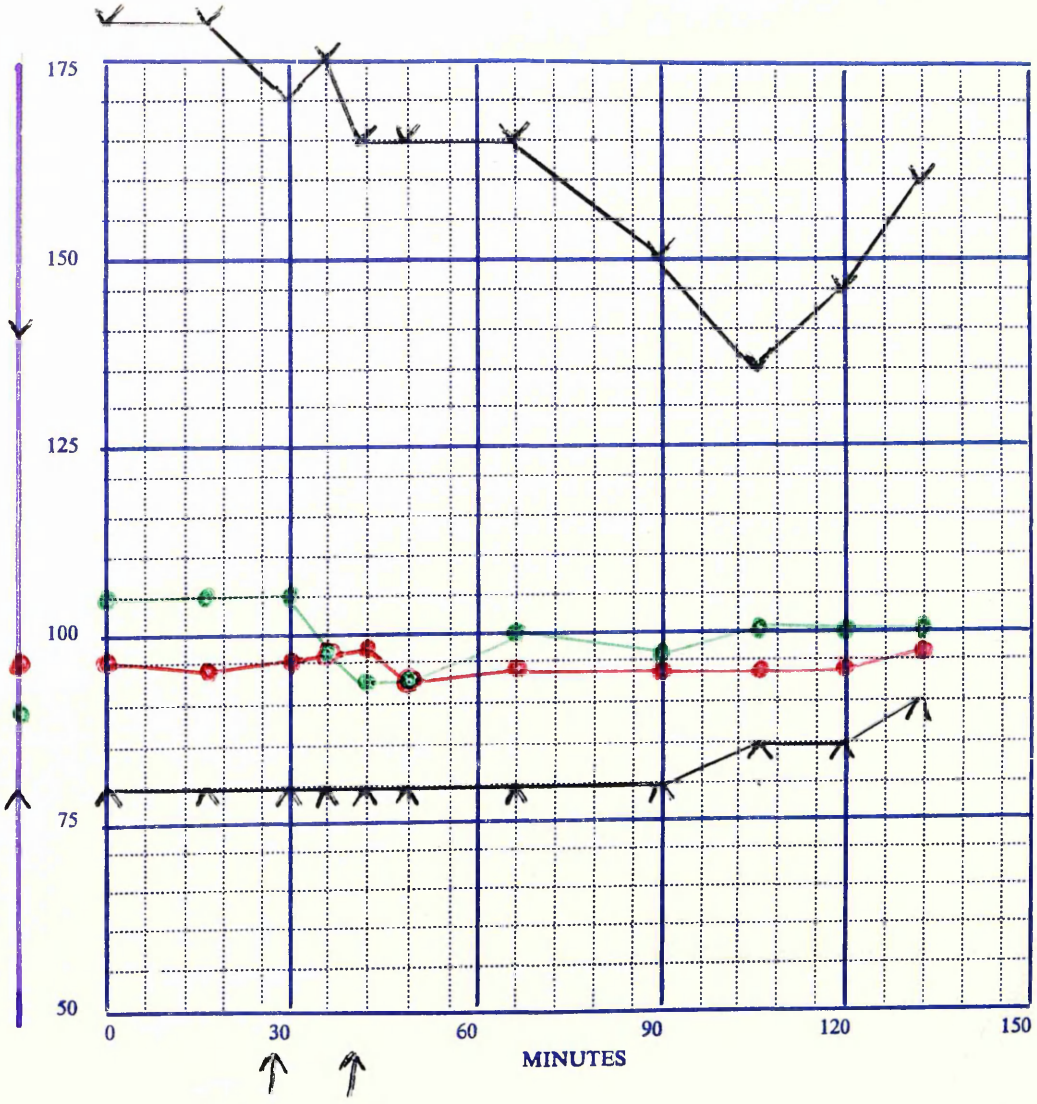
NOTES The patient was a little restless but otherwise co-operative. During the early part of the catheterisation he complained of pain in chest and epigastrium.

CASE RECORD No. 99.

Name R.G. Age 20. Diagnosis D.N.E.

Weight 56 Kilos. Haemoglobin 120%

Premedication Promethazine 25 mgms.
Amylobarbitone 200 mgms.



C.D. C.E.H.

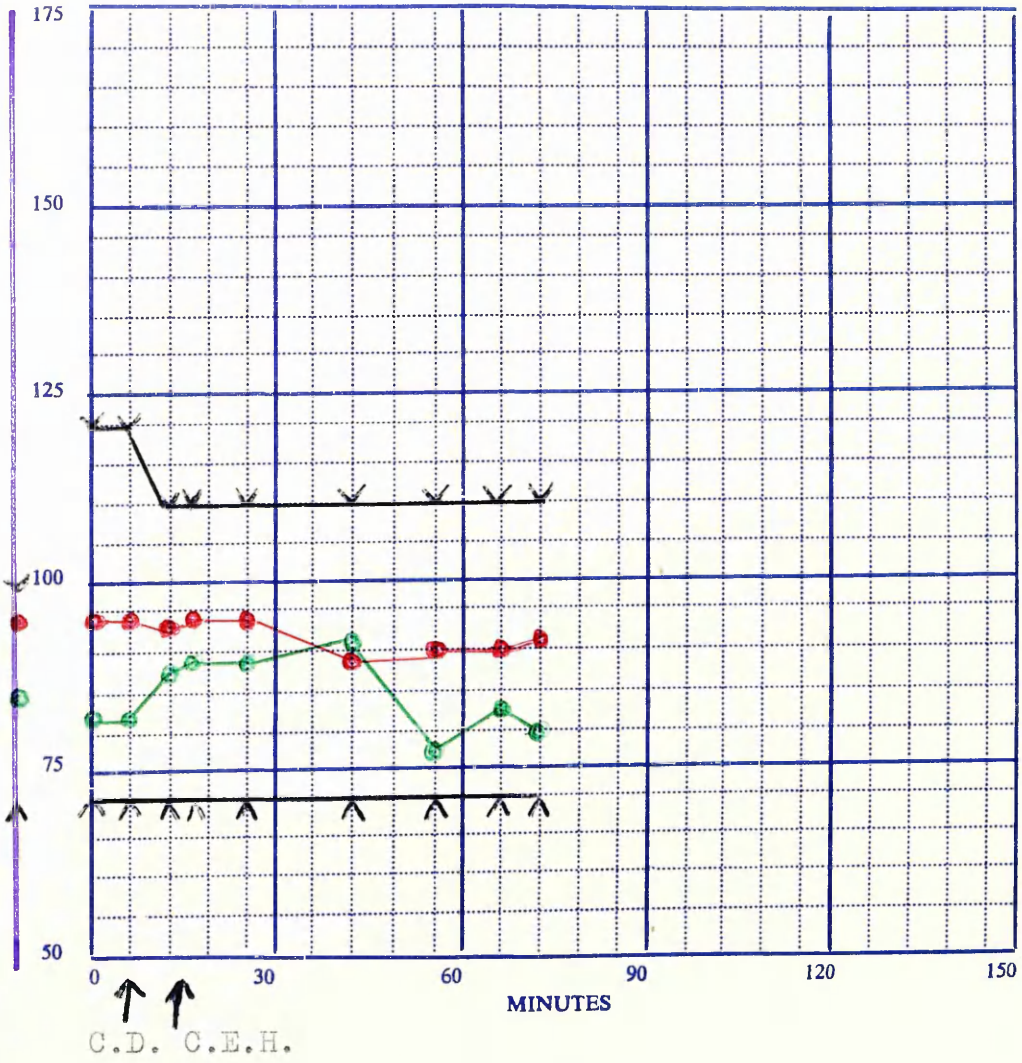
NOTES The patient complained of pain in the arm as the catheter passed and abdominal pain as it entered the atrium.

CASE RECORD No. 100.

Name J.L. Age 21 Diagnosis I.A.S.D.

Weight 45 Kilos. Haemoglobin 96%

Premedication Pentobarbitone 200 mgms.



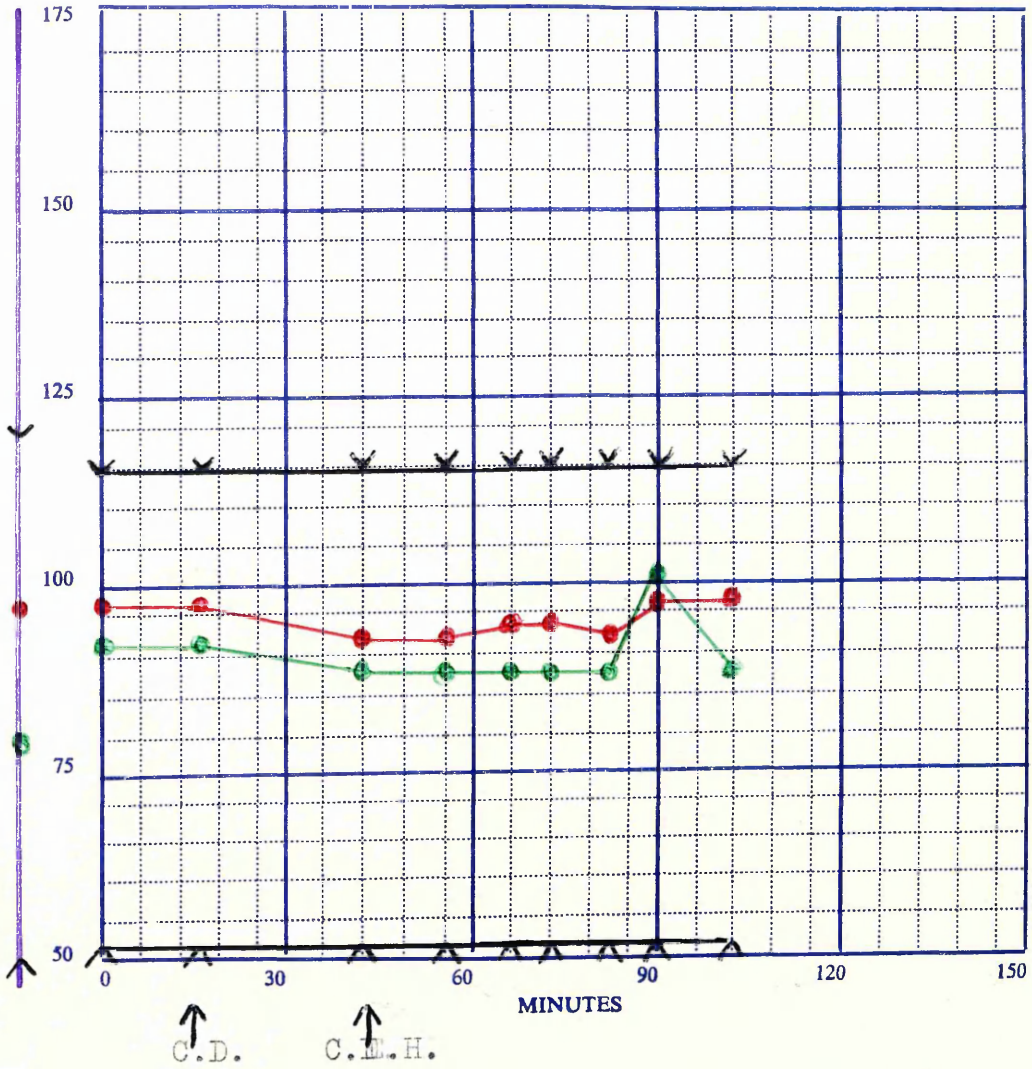
NOTES The patient was fairly well sedated but she complained of pain in the chest as the catheter was lying in the superior vena cava.

CASE RECORD No.....101.....

Name E.C. Age 23 Diagnosis P.D.A.

Weight 46.5 Kilos. Haemoglobin 97%

Premedication Promethazine 25 mgms.
Pentobarbitone 200 mgms.



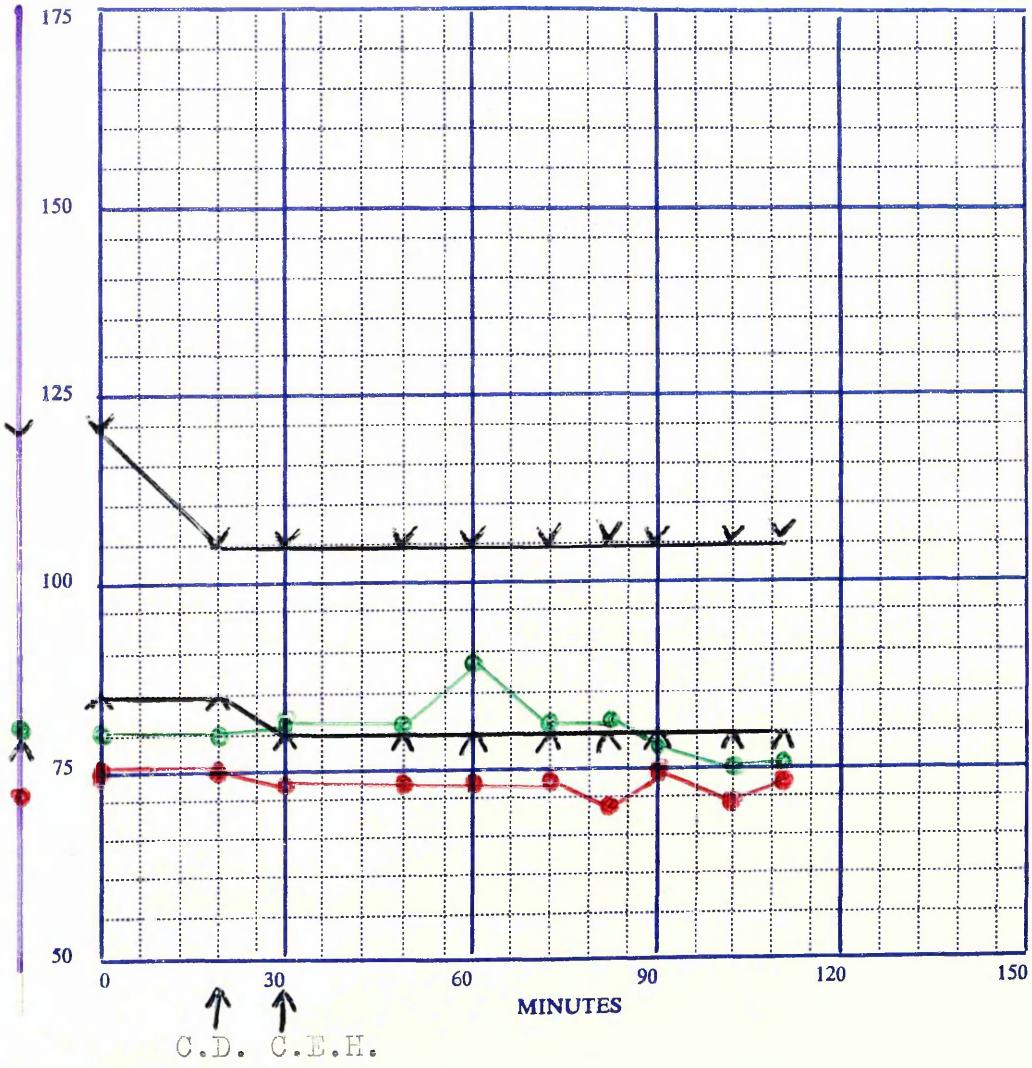
NOTES This patient was of poor intelligence and was restless during the catheterisation. At times she wept openly and re-acted markedly to minor stimuli.

CASE RECORD No. 102.

Name E.E. Age 23. Diagnosis F.T.

Weight 48 Kilos. Haemoglobin 138%

Premedication Promethazine 25 mgms.
Meprobamate 400 mgms.



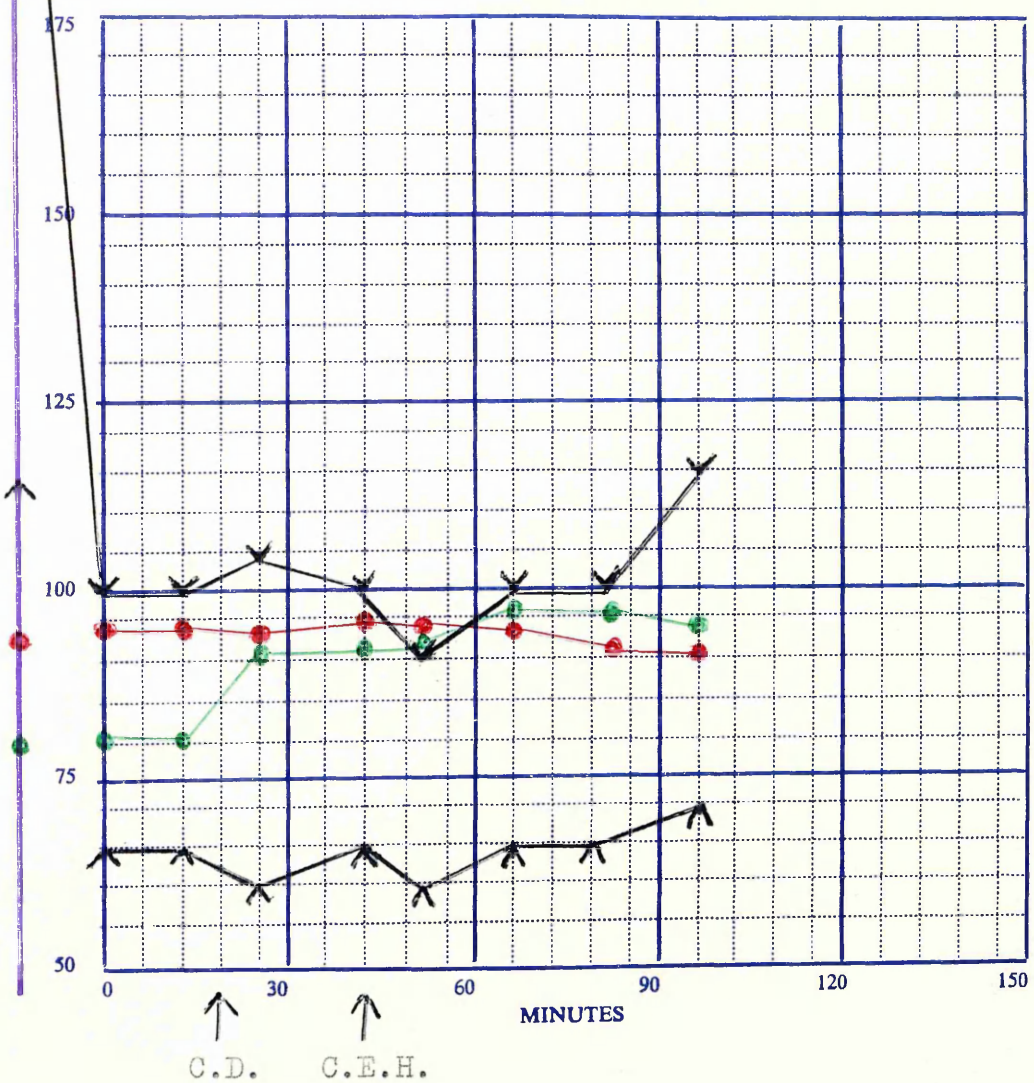
NOTES During the latter part of the examination the patient complained of pain in her arm and was actively sobbing at times. This was reflected by the fall in arterial oxygen saturation.

CASE RECORD No. 103.

Name A.B. Age 23 Diagnosis C. of A.

Weight 85 Kilos. Haemoglobin 100%

Premedication Promethazine 25 mgms.
Papaveratum 20 mgms.
Scopolamine 0.4 mgms.



NOTES Venous analysis - pH 7.35
pCO_2 51.2 mmHg.

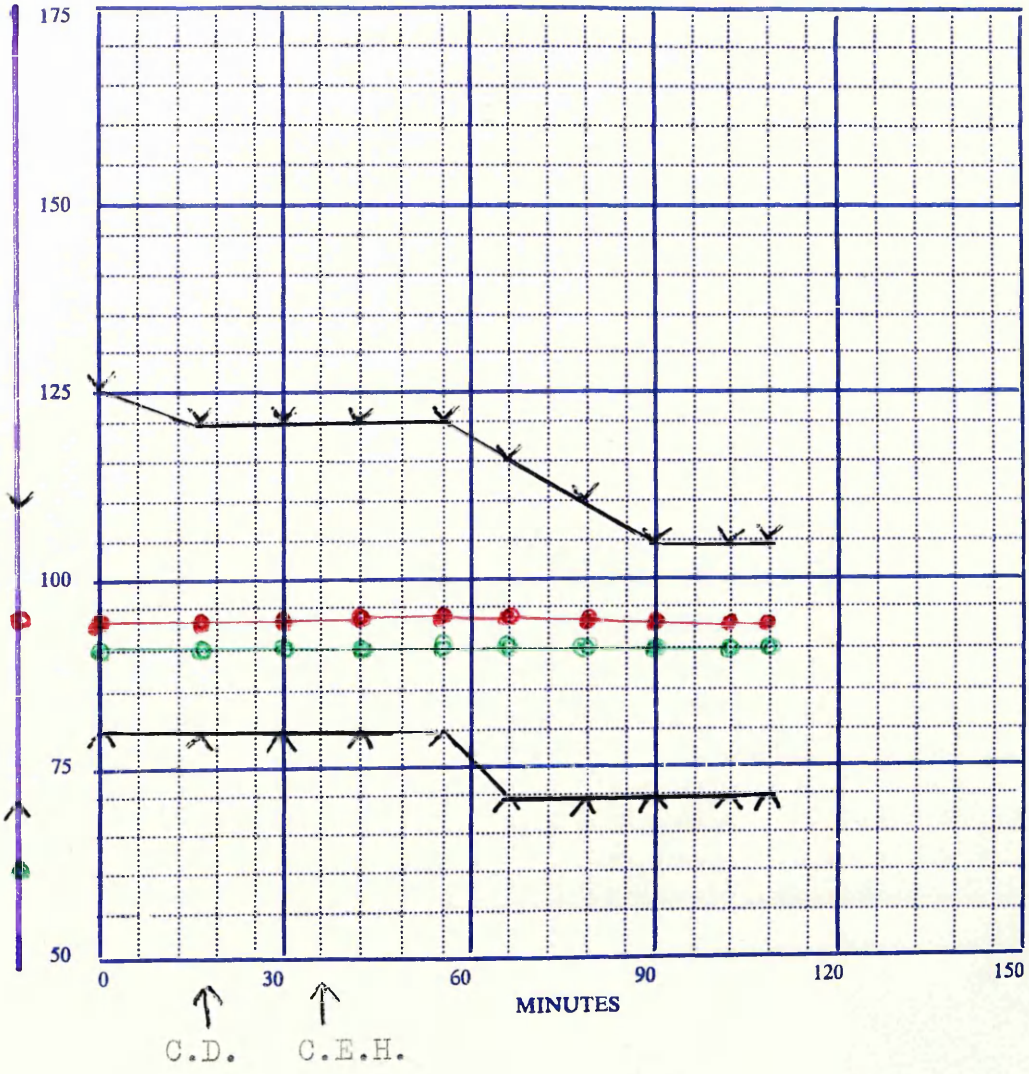
Despite the spectacular drop in blood pressure there were no apparent ill effects and the catheterisation and angiocardiography were accomplished quite easily.

CASE RECORD No. 104

Name E.McC. Age 24 Diagnosis I.A.S.D.

Weight 43 Kilos. Haemoglobin 100%

Premedication Amylobarbitone 200 mgms.



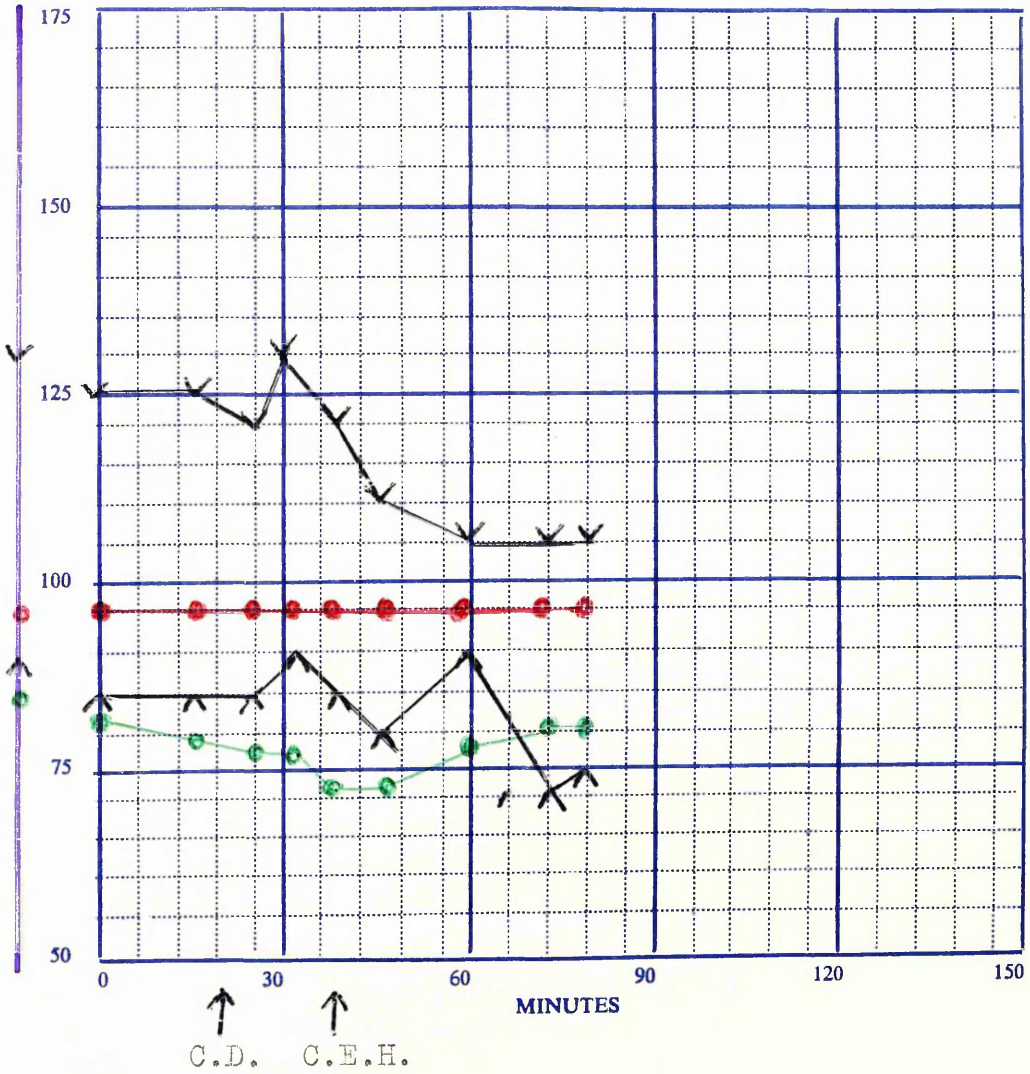
NOTES Arterial analysis - pH 7.46
pCO_2 37.7 mmHg.

CASE RECORD No. 105.

Name M.D. Age 24 Diagnosis I.V.S.D.

Weight 58.5 Kilos. Haemoglobin 116%

Premedication Papaveratum 20 mgms.
Scopolamine 0.4 mgms.



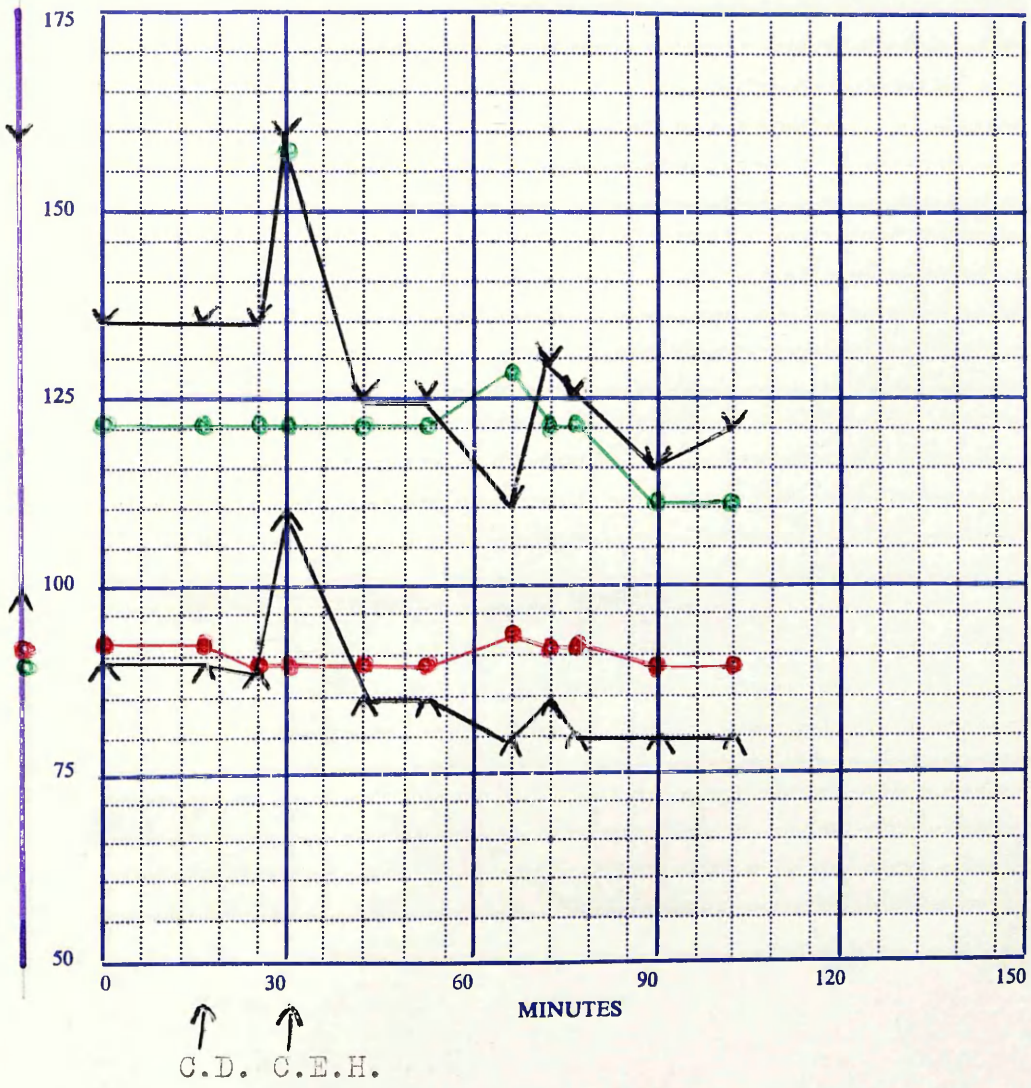
NOTES Arterial analysis - pH 7.42
pCO_2 40.4 mmHg.

CASE RECORD No.....106.....

Name J.McC. Age 24 Diagnosis I.V.S.D.

Weight 58.5 Kilos. Haemoglobin 110%

Premedication Promethazine 25 mgms.
Papaveratum 20 mgms.
Scopolamine 0.4 mgms.



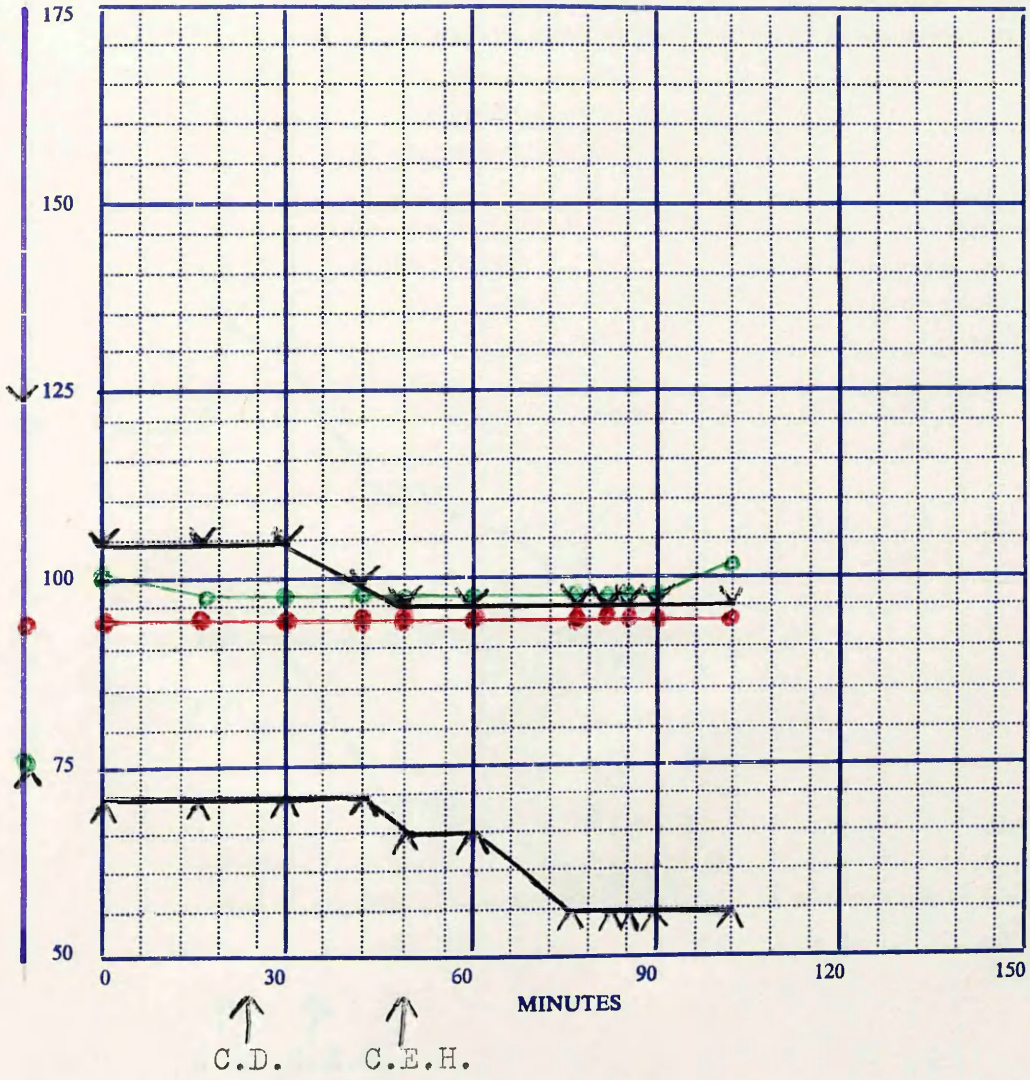
NOTES Venous analysis - pH 7.36
pCO_2 45.8 mmHg.

CASE RECORD No.....107.....

Name M.I. Age 25 Diagnosis M.S.

Weight 49 Kilos. Haemoglobin 92%

Premedication Pentobarbitone 200 mgms.



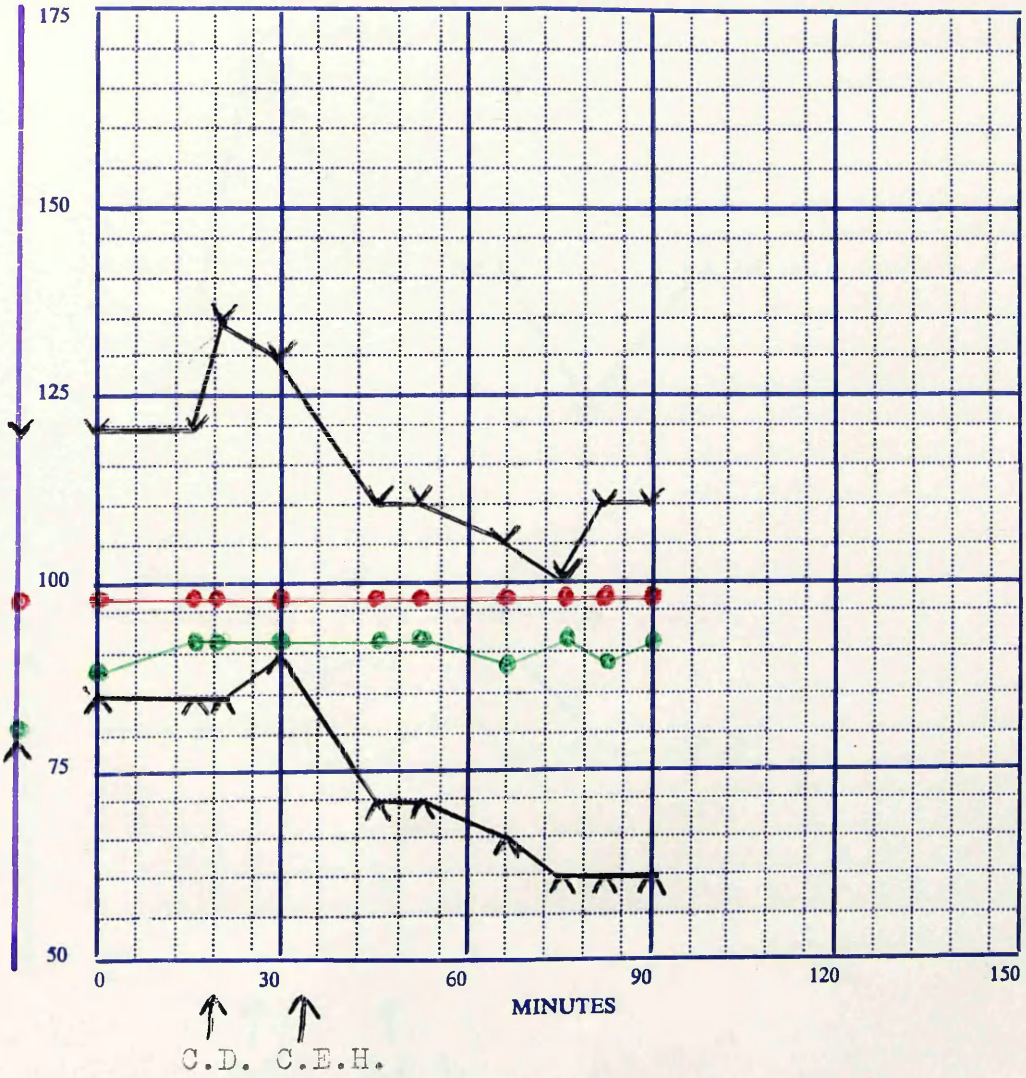
NOTES This was an uneventful catheterisation.

CASE RECORD No.....108.....

Name H.M. Age 25 Diagnosis I.V.S.D.

Weight 75 Kilos. Haemoglobin 110%

Premedication Promethazine 25 mgms.
Papaveratum 20 mgms.
Scopolamine 0.4 mgms.



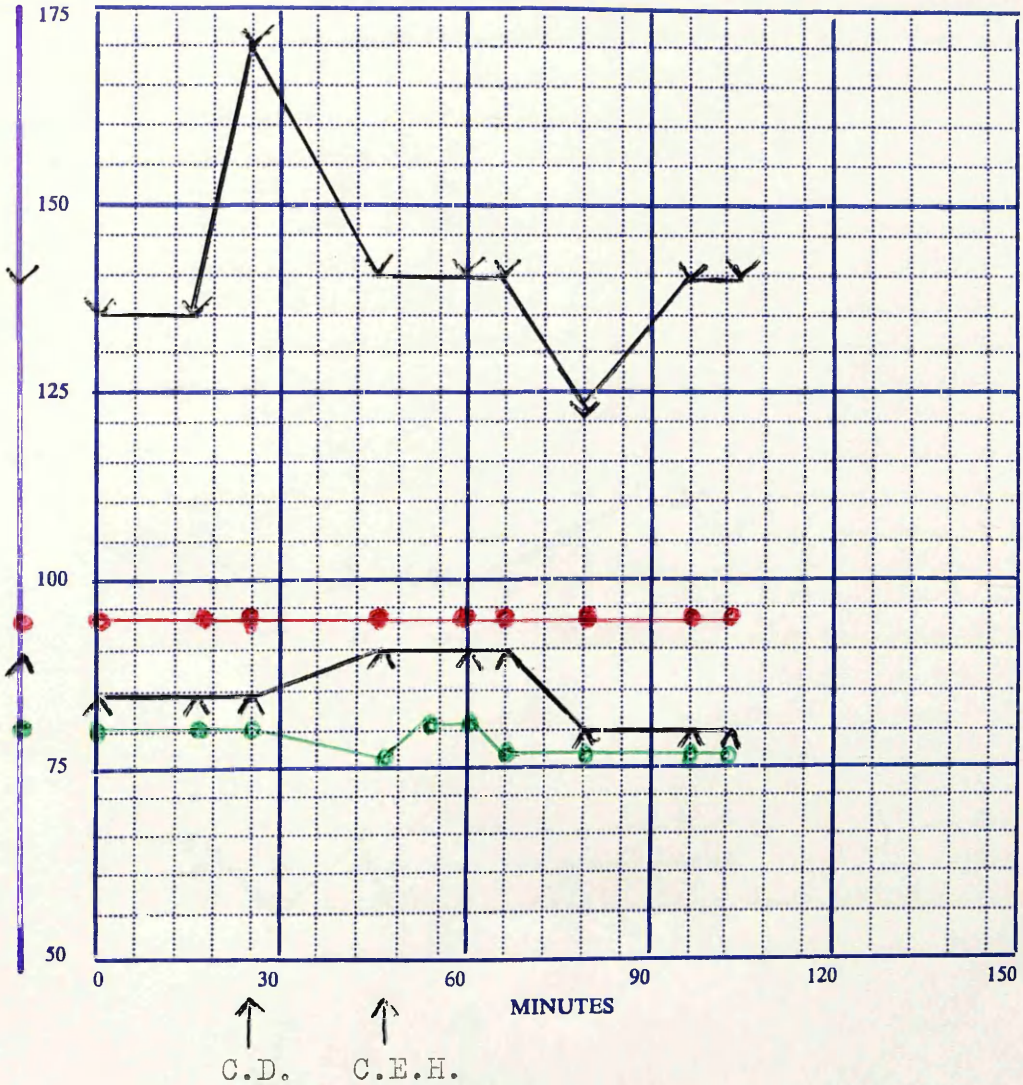
NOTES Arterial analysis - pH 7.48
pCO_2 36.4 mmHg.

CASE RECORD No. 109.

Name M.M. Age 27 Diagnosis P.S.

Weight 87 Kilos. Haemoglobin 89%

Premedication Amylobarbitone 200 mgms.



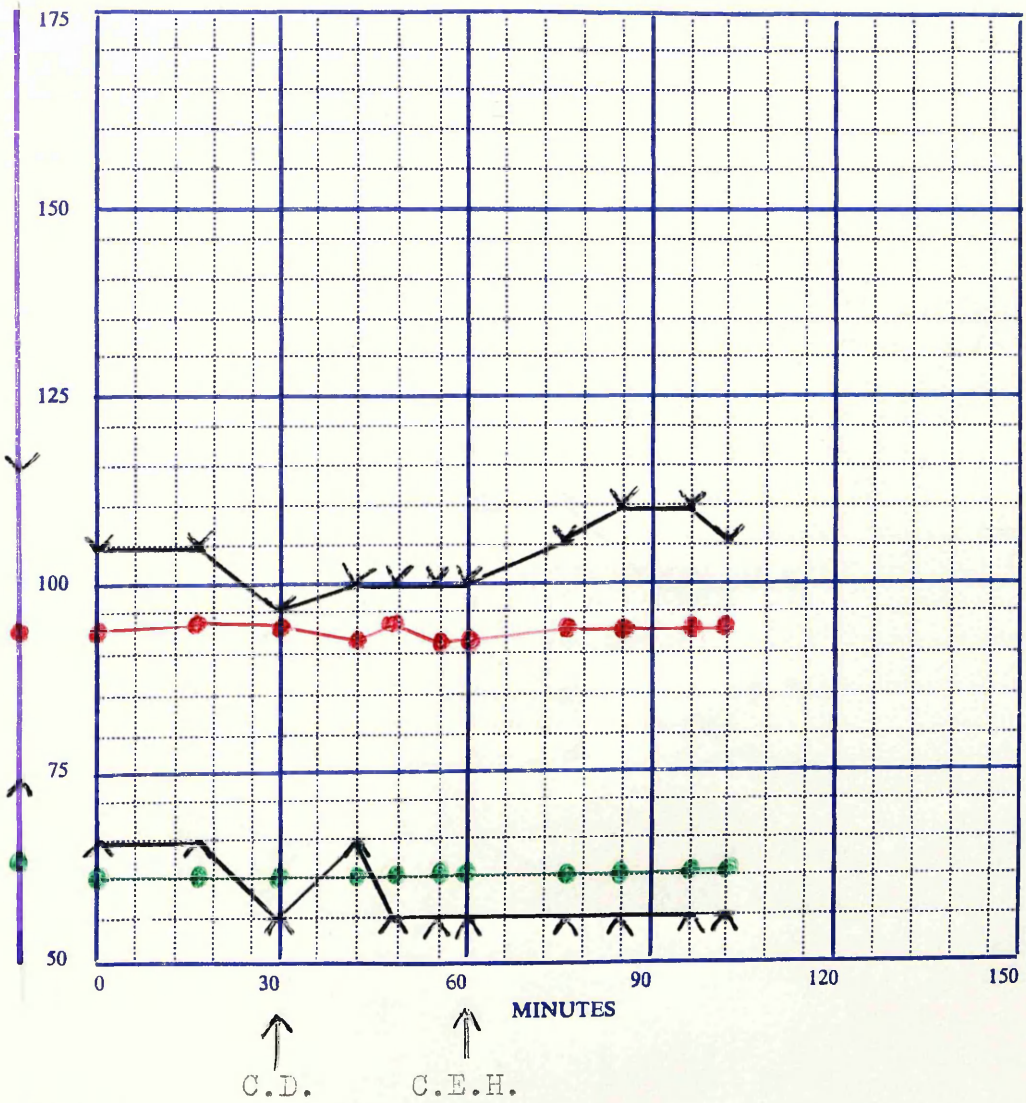
NOTES A marked rise in blood pressure was associated with cutting down. While the catheter was being manipulated in the ventricle the patient complained of abdominal and chest pain.

CASE RECORD No.....110.....

Name E.C. Age 28 Diagnosis P.S.

Weight 53 Kilos. Haemoglobin 120%

Premedication Promethazine 25 mgms.
Papaveratum 20 mgms.
Scopolamine 0.4 mgms.



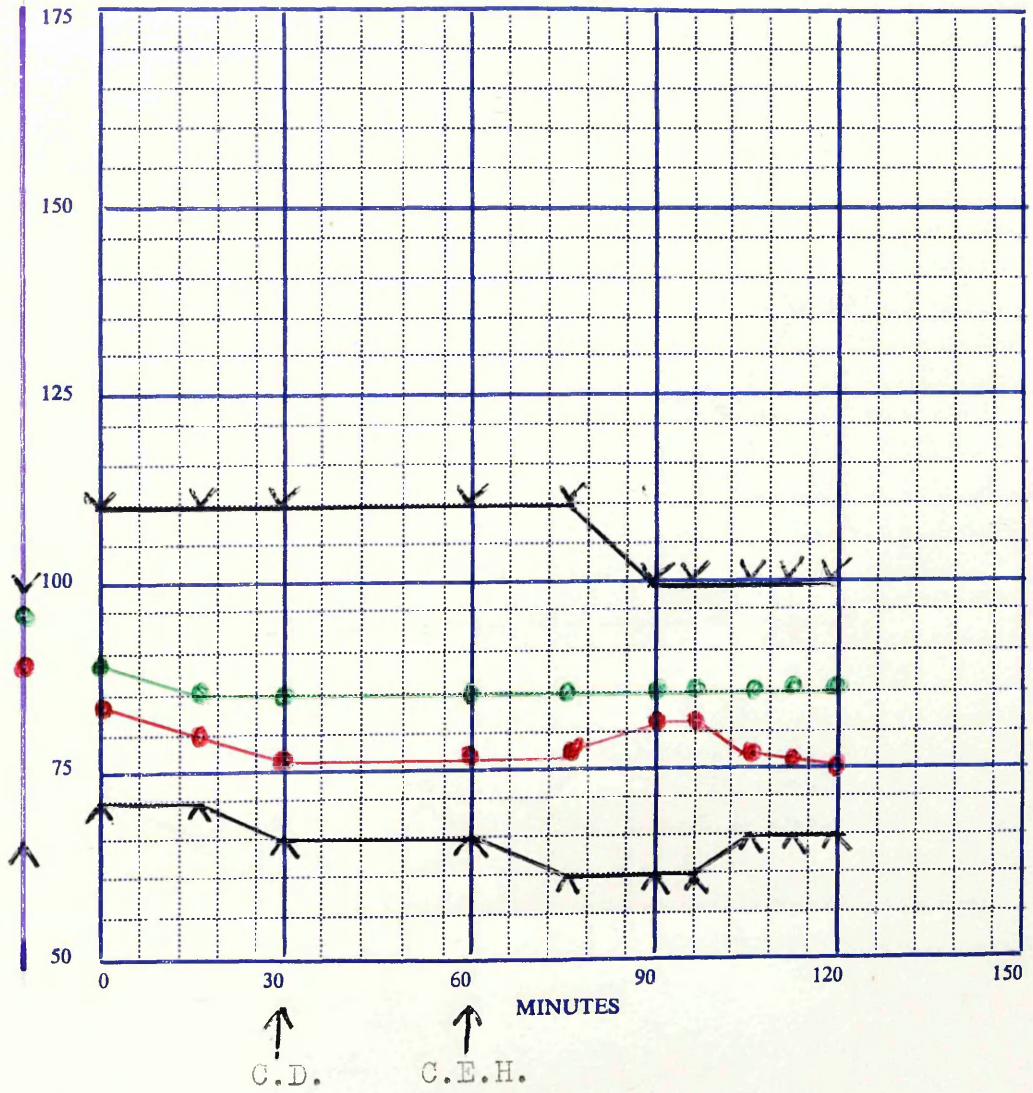
NOTES This was an uneventful catheterisation.

CASE RECORD No.....111.....

Name E.G. Age 29 Diagnosis Ebstein's Disease.

Weight 54 Kilos. Haemoglobin 100%

Premedication Promethazine 25 mgms.
Amylobarbitone 200 mgms.



NOTES

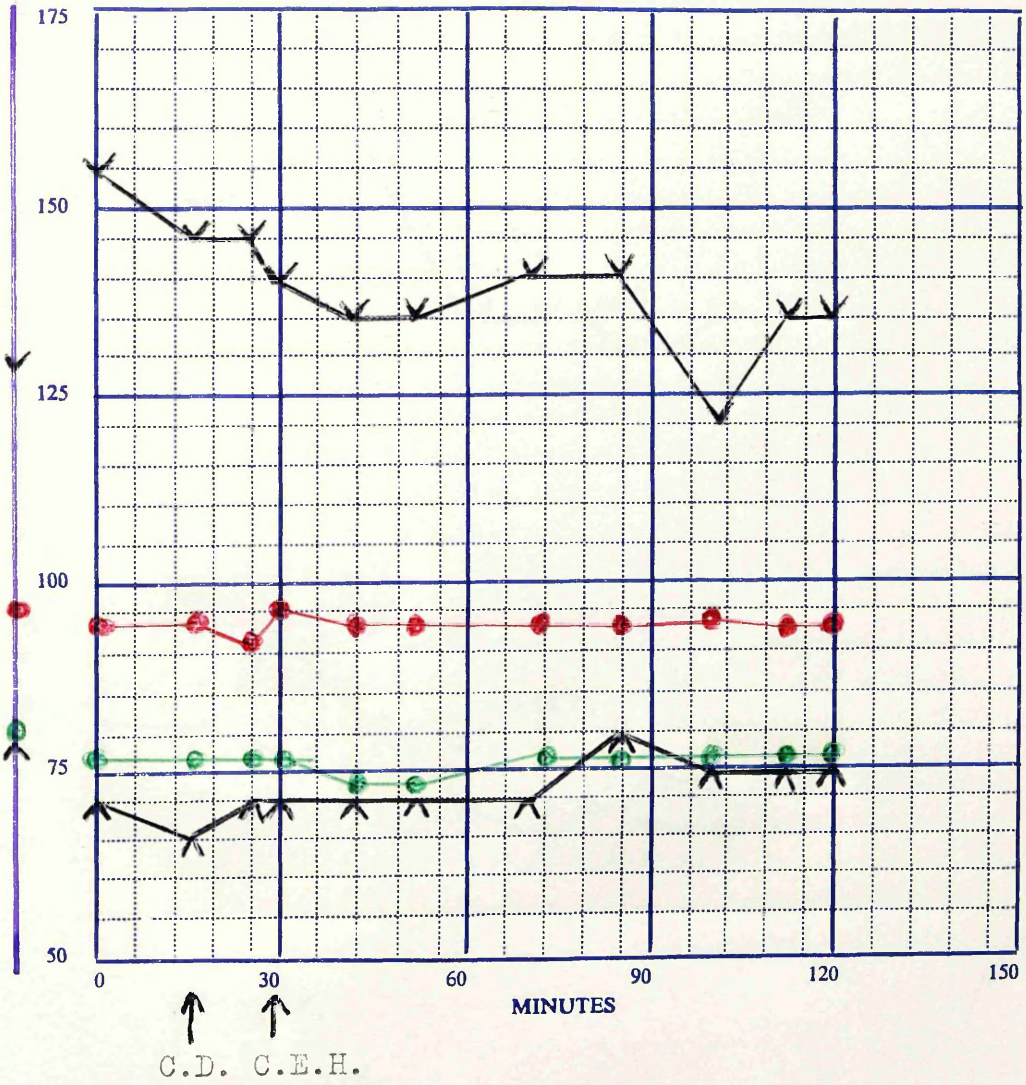
It was noted during the catheterisation that pressures recorded in the vena cava and atrium on the way in were higher than those taken on withdrawal.

CASE RECORD No. 112.

Name T.McI. Age 31 Diagnosis I.V.S.D.

Weight 54.9 Kilos. Haemoglobin 105%

Premedication Amylobarbitone 200 mgms.



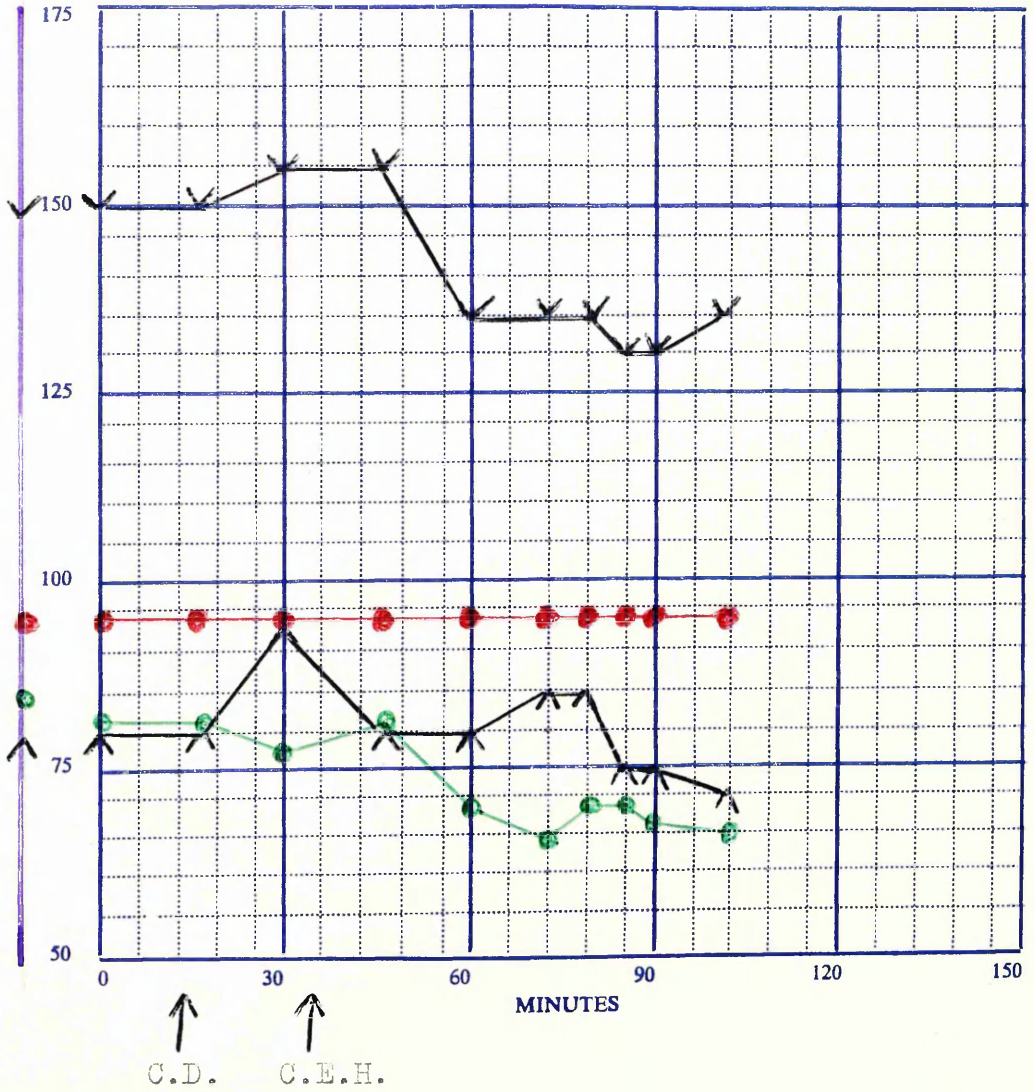
NOTES This was an uneventful catheterisation.

CASE RECORD No. 113.

Name E.M. Age 32 Diagnosis E.D.A.

Weight 70 Kilos. Haemoglobin 98%

Premedication Promethazine 25 mgms.
Amylobarbitone 200 mgms.



NOTES Arterial analysis - Oxygen saturation 91%
(oximeter reading 94%)

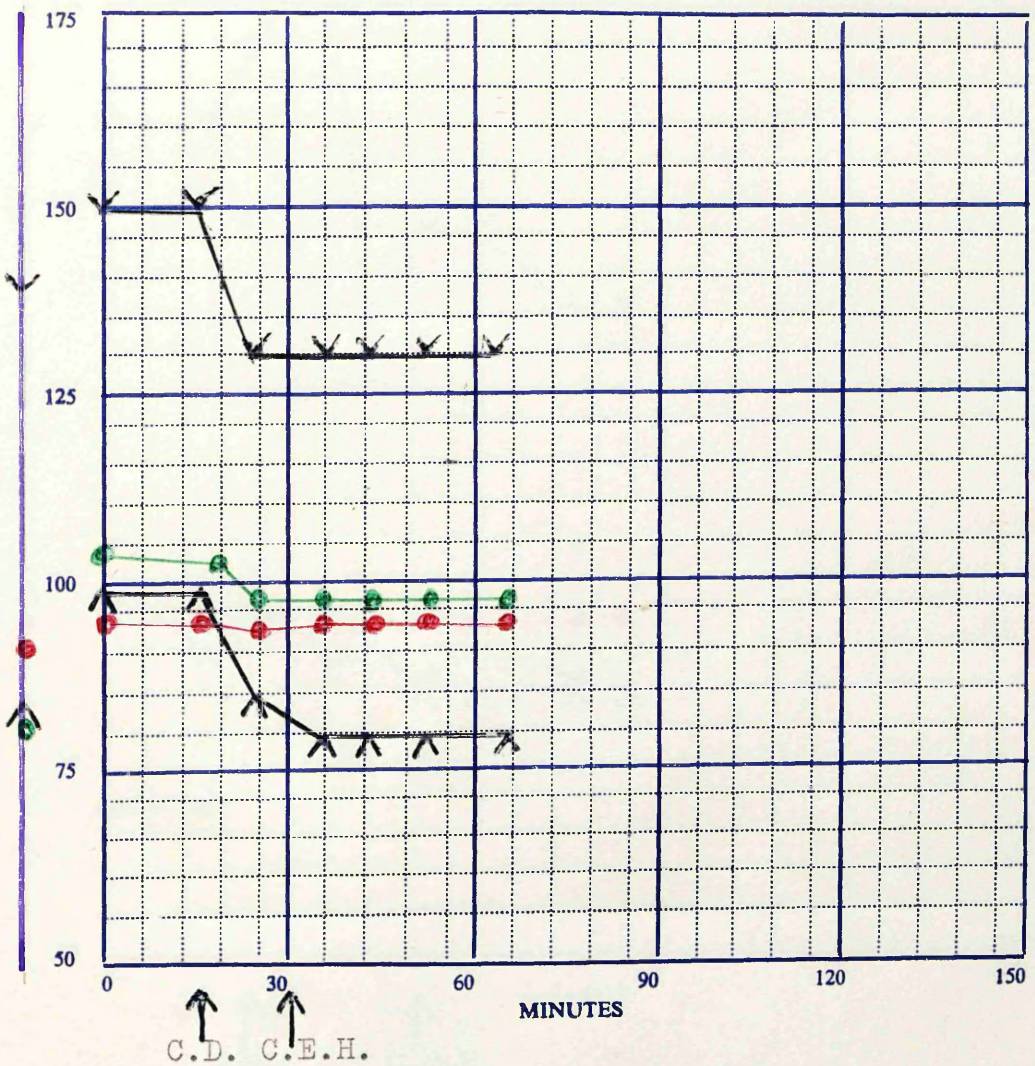
During the early part of the procedure the patient complained of pain as the catheter entered his chest.

CASE RECORD No.....114.....

Name M.S. Age 32 Diagnosis Ebstein's Disease.

Weight 53 Kilos. Haemoglobin 120%

Premedication Promethazine 25 mgms.
Papaveratum 20 mgms.
Scopolamine 0.4 mgms.



NOTES Arterial analysis - pCO_2 45.8 mmHg.

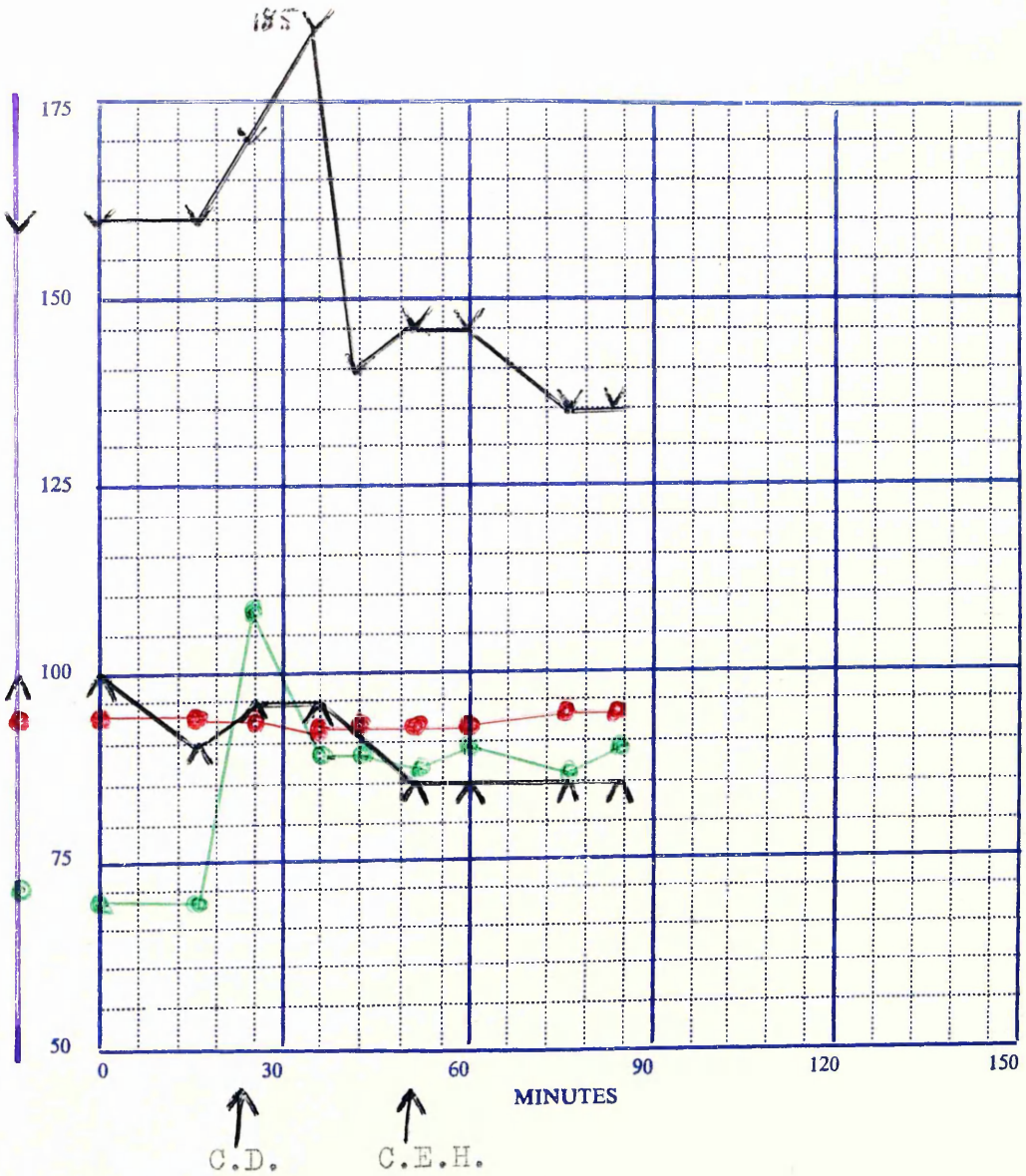
Although this patient was very nervous before the procedure, she settled well and slept throughout most of it.

CASE RECORD No. 115.

Name B.C. Age 33 Diagnosis C. of A.

Weight 55 Kilos. Haemoglobin 120%

Premedication Papaveratum 20 mgms.
Scopolamine 0.4 mgms.



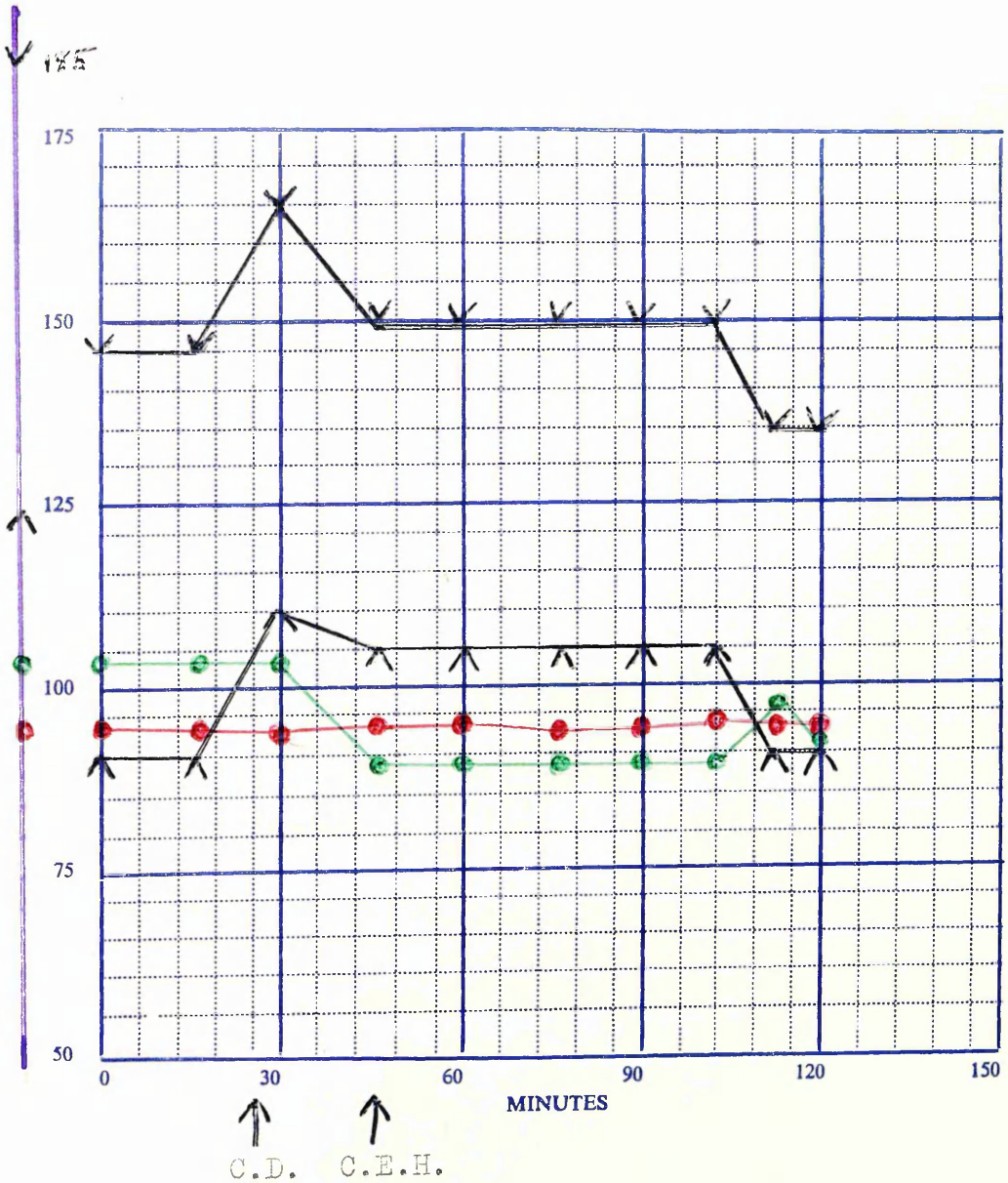
NOTES Arterial analysis - pH 7.39
pCO_2 46 mmHg.

CASE RECORD No. 116.

Name P.R. Age 35. Diagnosis I.A.S.D.

Weight 58.5 Kilos. Haemoglobin 104%

Premedication Promethazine 25 mgms.
Amylobarbitone 200 mgms.



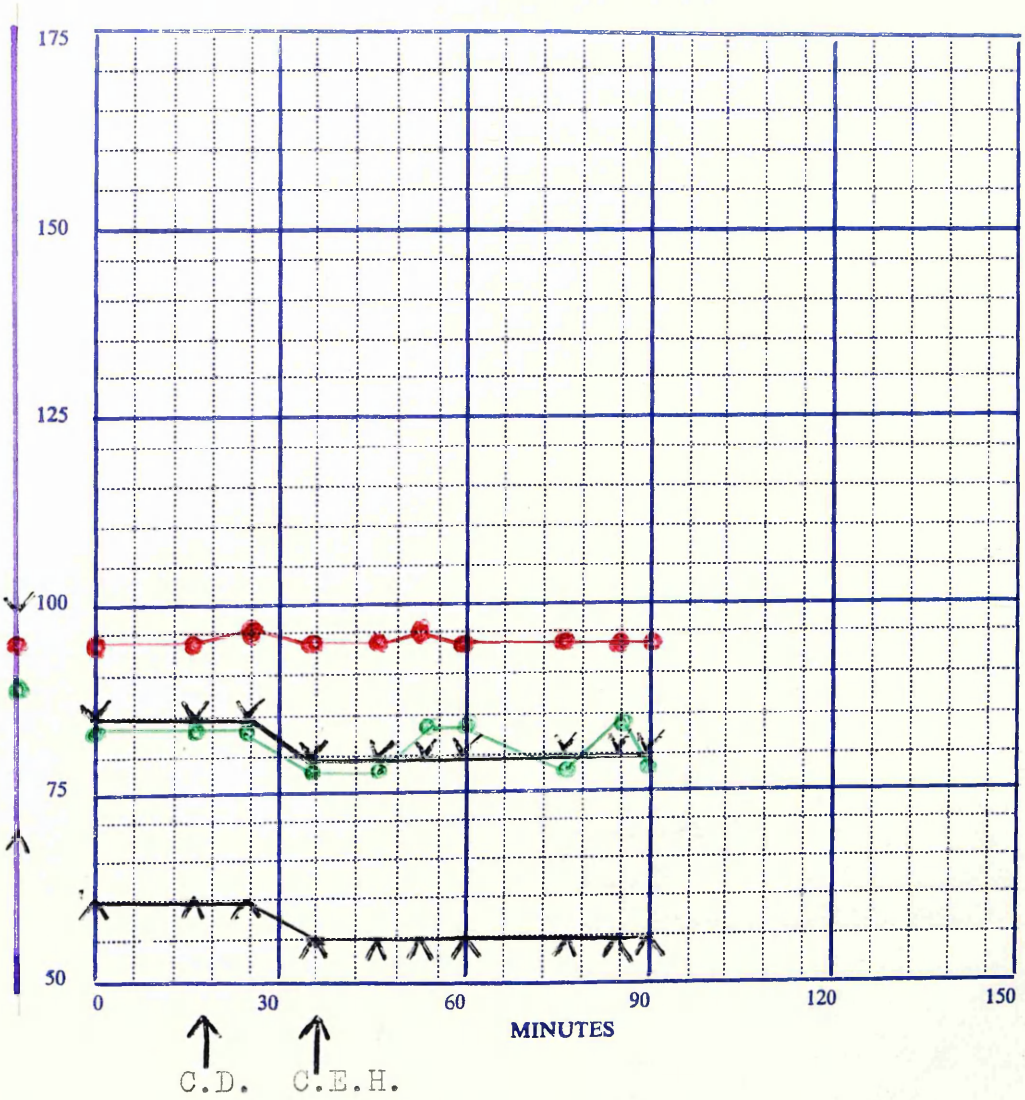
NOTES At intervals throughout the catheterisation, the patient complained of pain in the chest and abdominal discomfort also occurred when the catheter entered the liver.

CASE RECORD No.....117.....

Name M.C. Age 36 Diagnosis M.S.

Weight 62 Kilos. Haemoglobin 101%

Premedication Promethazine 25 mgms.
Amylobarbitone 200 mgms.



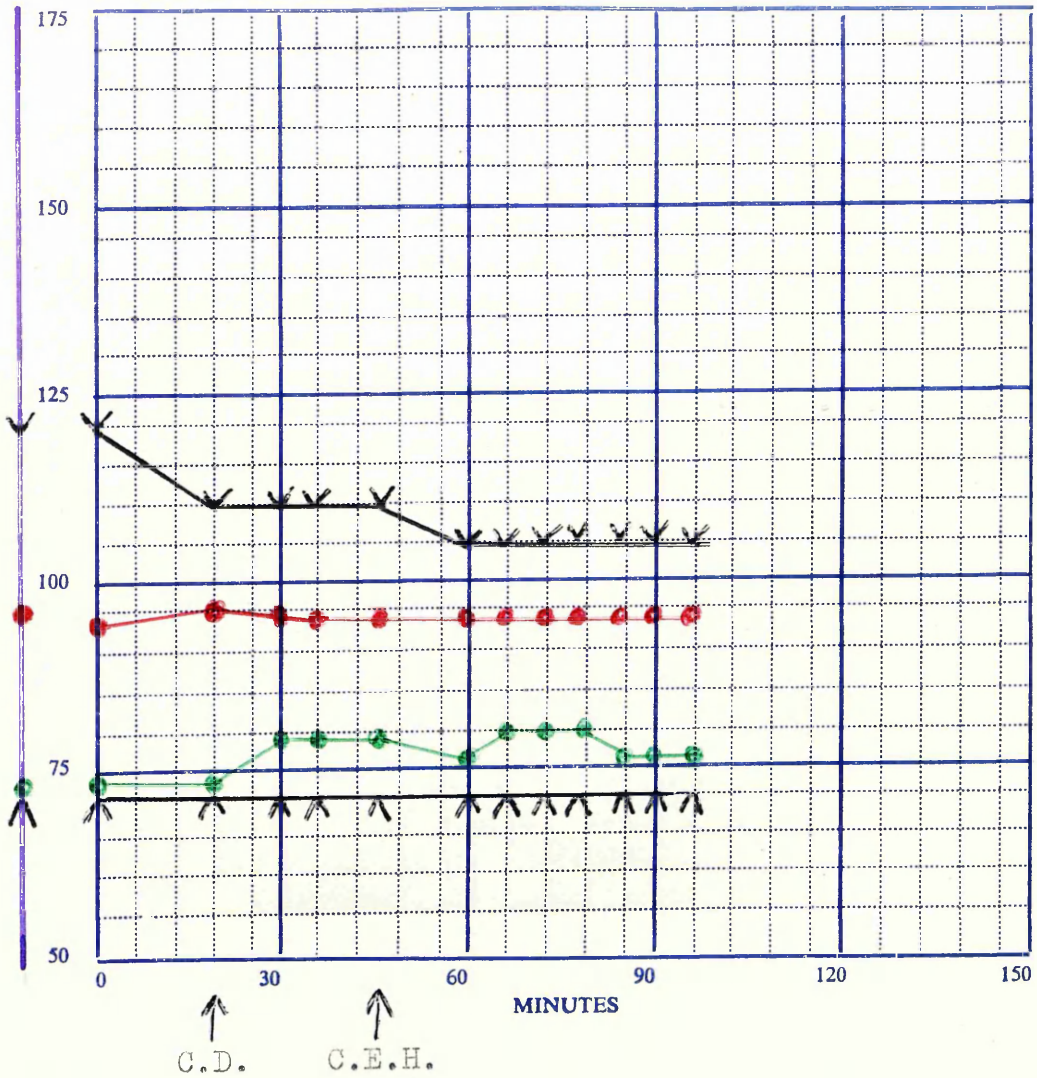
NOTES The patient was adequately sedated but complained of pain when the catheter being changed.

CASE RECORD No. 118.

Name H.S. Age 37 Diagnosis P.S. + I.V.S.D.

Weight 48 Kilos. Haemoglobin 78%

Premedication Amylobarbitone 200 mgms.



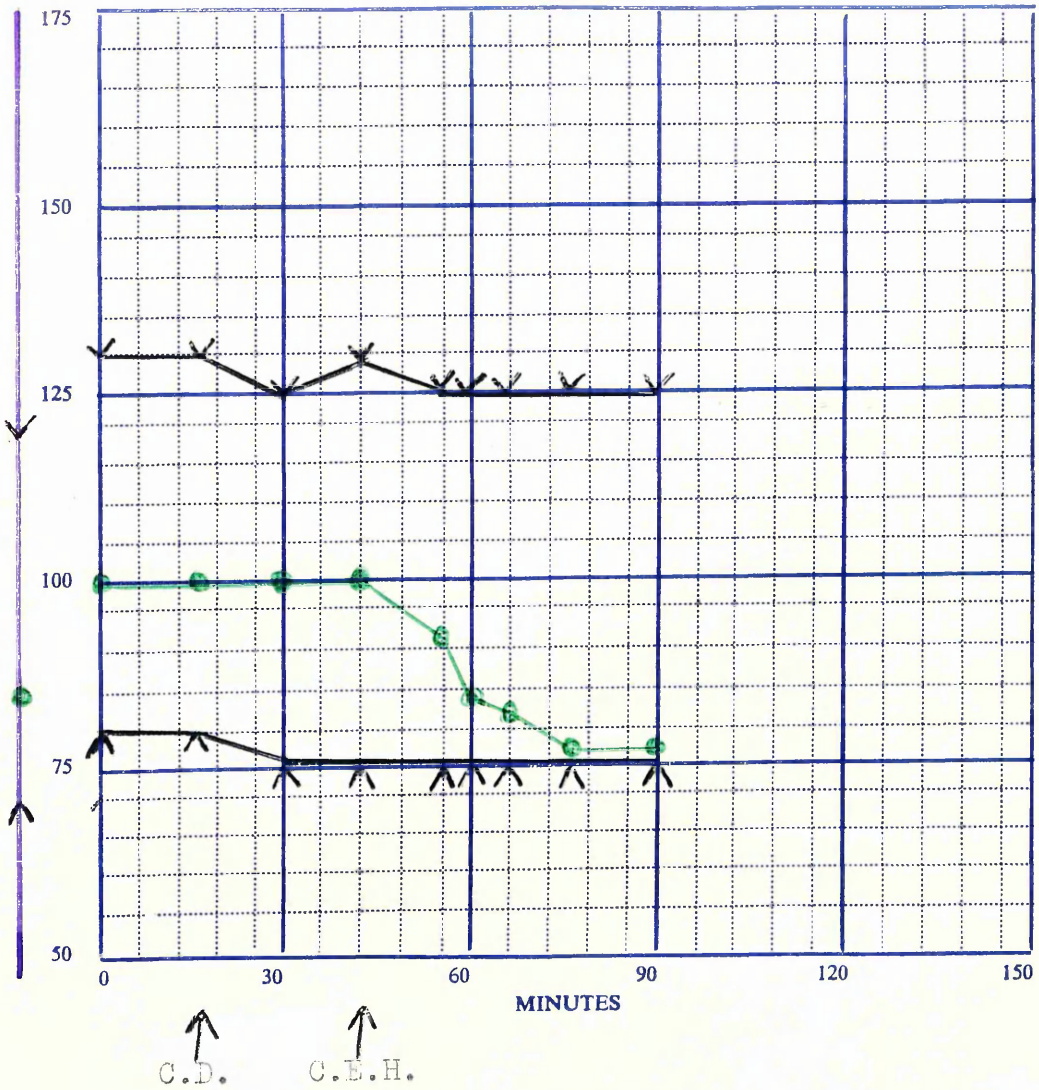
NOTES This was an uneventful catheterisation.

CASE RECORD No. 119.

Name H.S. Age 37 Diagnosis P.S. + V.S.D.

Weight 50 Kilos. Haemoglobin 100%

Premedication Papaveratum 20 mgms.
Scopolamine 0.4 mgms.



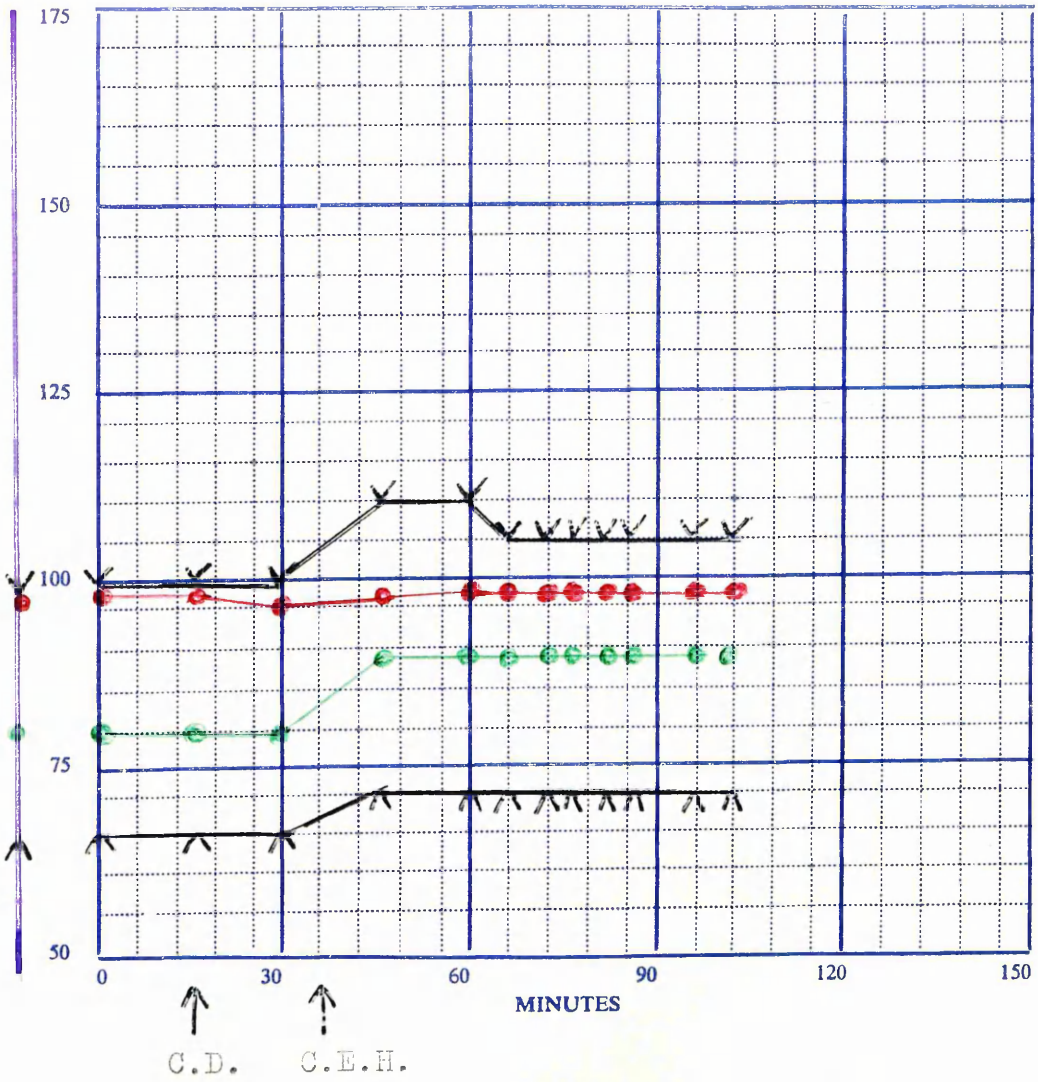
NOTES This is a repeat catheterisation on the previous case. On this occasion considerable pain was experience during the cut down as the physician had difficulty in finding the vein. The oximeter was out of commission at this time.

CASE RECORD No.....120.....

Name E.McI. Age 41 Diagnosis I.A.S.D.

Weight 42 Kilos. Haemoglobin 96%

Premedication Promethazine 25 mgms.
Amylobarbitone 200 mgms.



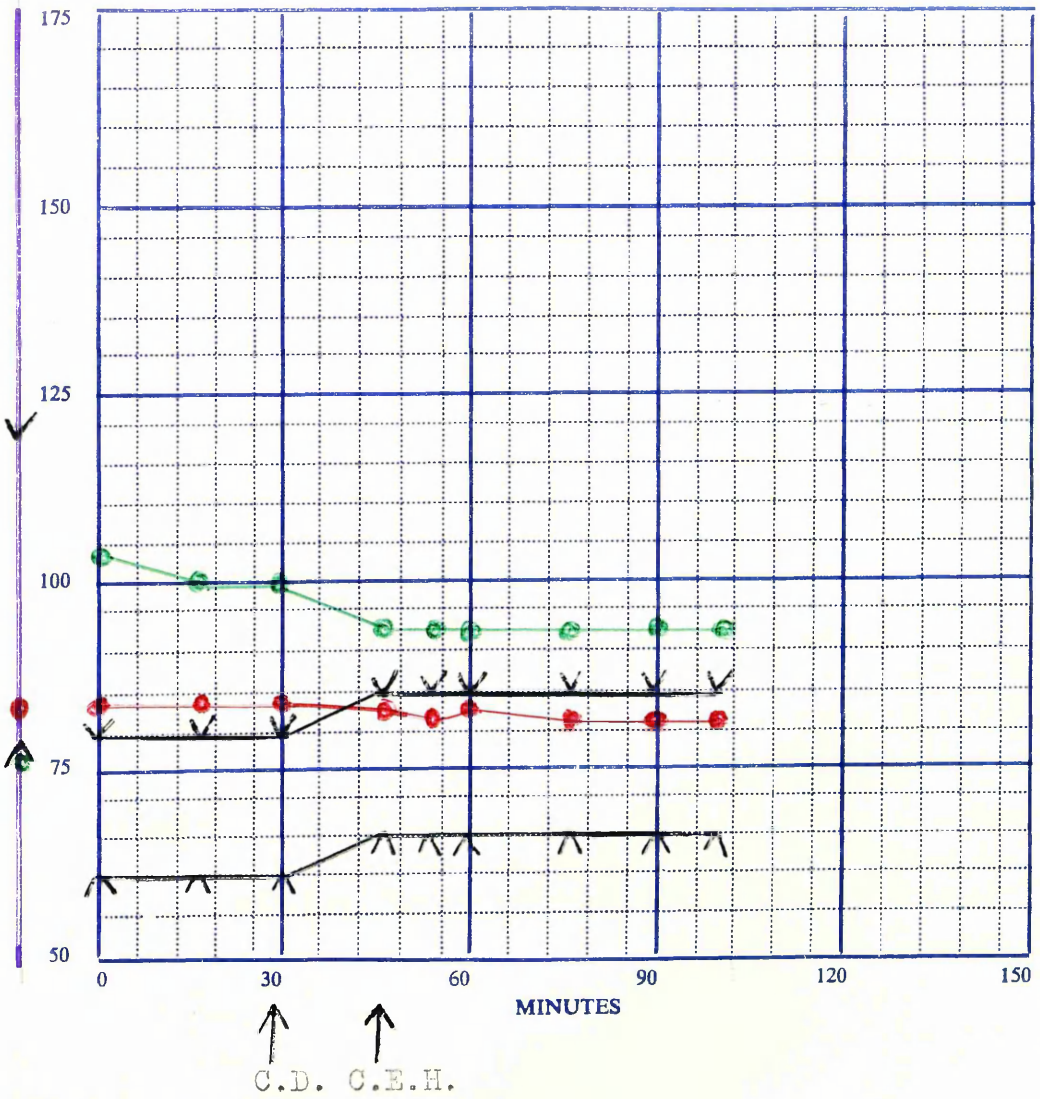
NOTES During the early part of the catheterisation the patient complained of pain and nausea. This pain was associated with stimulation of the ventricular septum.

CASE RECORD No. 121

Name M.M. Age 42 Diagnosis Pulmonary Fibrosis.

Weight 60 Kilos. Haemoglobin 100%

Premedication Papaveratum 20 mgms.
Scopolamine 0.4 mgms.



NOTES

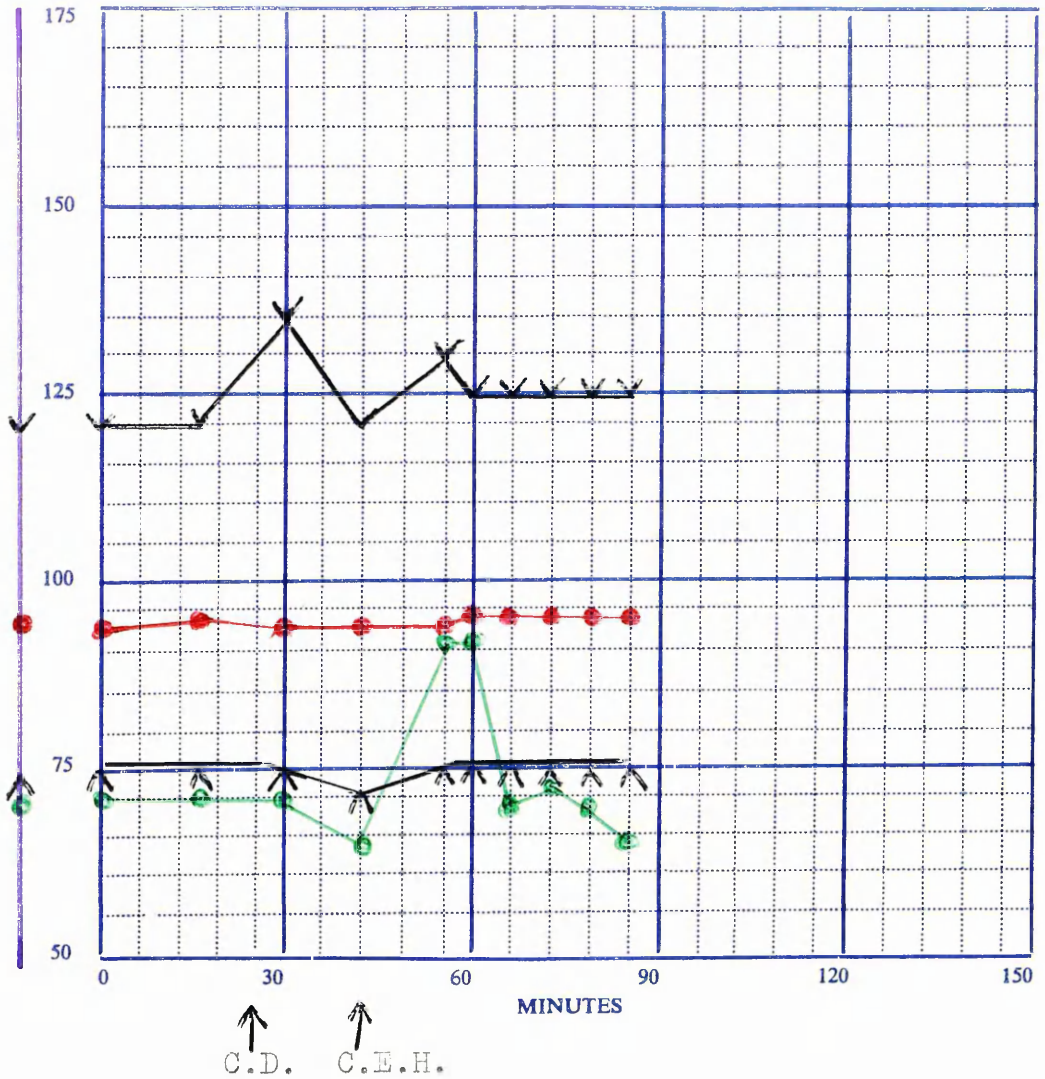
Arterial analysis - Oxygen saturation 75%
(oximeter reading 82%)
pH 7.45
pCO_2 30.7 mmHg.

CASE RECORD No. 122.

Name A.McN. Age 47 Diagnosis I.A.S.D.

Weight 26 Kilos. Haemoglobin 95%

Premedication Amylobarbitone 200 mgms.



NOTES Arterial analysis - pH 7.45
pCO_2 35 mmHg.