

The Dangers of Modern Anaesthetics

by

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The Dangers of Modern Anaesthetics.

Introduction.

During the past fifty years there have been many changes in the practice of anaesthesia, some many years ago, others more recently. Patients used to be starved before operation and drastically purged. Premedication consisted of a small dose of atropine, and morphine, if the patient was in pain. The anaesthetic agents in use were nitrous oxide, ether, chloroform, and ethyl chloride. Local analgesia was used extensively on the Continent, but rarely in Britain. Spinal analgesia was used, but only hypobasic solutions, and little was known of the controllability of the analgesia.

The changes have been many. Pre-operative care has now become extensive, covering many fields. If necessary, an attempt is made to restore the patient's general condition as near to normal by intravenous infusions of saline, glucose solution, plasma or blood. Adequate food and drink are given, and a mild laxative or bland enema to ensure that the patient's bowel is empty when he goes to the operating theatre. Premedication is calculated for each patient, an opium derivative or barbiturate with scopolamine or atropine being the usual choice. In certain cases a basal narcotic

drug may be substituted for the opiate or barbiturate.

Induction of anaesthesia may be by the intravenous injection of one of the barbiturate drugs, and irrespective of the type of anaesthetic, oxygen is administered throughout the operation. Cyclopropane di-vinyl ether and trichlorethylene, the new inhalational anaesthetic agents, are in everyday use. The use of curare in anaesthesia is likely to mark the greatest advance in that specialty in recent years. Apparatus has developed, with mechanisation and complexity the main features. Endotracheal and endobronchial anaesthesia require dexterity in the use of the laryngoscope and bronchoscope. "Controlled" or "artificial" respiration has become a recognised technique in anaesthesia, not merely a means of resuscitation.

Advances have also been made in local, regional and spinal analgesia. New drugs, "Nupercaine" and Amethocaine, giving greater duration of effect than procaine, allow most operations to be performed under local or regional analgesia. Spinal analgesia has developed with drugs lighter, heavier and of the same specific gravity as cerebro-spinal fluid. The risk involved in the technique of sub-arachnoid analgesia has stimulated interest in the production of analgesia by the injection of similar drugs into the peridural space.

Since the advent of curare the use of regional and spinal analgesia has decreased, as sub-arachnoid block is now no longer required for the absolute relaxation of the abdominal musculature, and few are the patients too ill to tolerate curare and a "light" general anaesthesia, requiring the use of regional analgesia alone.

The status of the anaesthetist has also changed, with his share in the pre-operative and post-operative treatment of the patient gradually increasing and the care of the patient during the operation being left more and more to him.

The change has been brought about by the necessity for anaesthesia to keep pace with developments in surgery. During the two wars there have been many advances in surgical technique to cope with problems previously never encountered or thought to be insoluble. Accompanying these, there have been developments in resuscitation and blood transfusion, the supervision of which has been delegated largely by the surgeon to the anaesthetist, who, with the patient's blood-pressure, pulse and respiratory rates charted throughout the operation, is in the position of making the most accurate estimation of the patient's condition and the change likely to occur. With this information he is thus the most suitable person for the controlling of any infusions or transfusions that may be required.

The scope of the anaesthetist has increased considerably. The available drugs used to be few, and the administrators tended to concentrate their experience on the use of these drugs. It is only comparatively recently that a staff appointment at a teaching hospital was advertised as "Chloroformist to -- Hospital" (1). He is now expected to have a knowledge of the basic sciences; anatomy, in order to carry out regional analgesia; physiology, especially of the circulatory and respiratory systems; pathology, of the condition requiring the surgical operation and of any concomittant condition; pharmacology of anaesthetic, sedative and analeptic drugs, and of resuscitation. He might well be termed 'physician-anaesthetist' in contrast to the 'chloroformist' or 'etherist' of days gone by.

With better pre-operative and post-operative care, and modern methods of anaesthesia, which depend less and less on the more toxic agents, especially since the advent of the relaxant drugs, there should be fewer post-operative complications and an improvement in the immediate post-operative condition of the patient.

Unfortunately, there would not appear to be the decrease in mortality and morbidity associated with anaesthesia that one would expect when statistics published at the beginning of the twentieth century are compared with those of today. In certain cases there is apparently a

deterioration.

This investigation has been carried out in an attempt to determine whether the results of modern methods of anaesthesia are worse than those of the older and more simple techniques. One must bear in mind that the operations in many cases are much more extensive, and that many of the patients are much poorer operation and anaesthetic risks than those quoted in the earlier figures. A most important factor is the more accurate keeping of records, with the noting of minor complications which would have been regarded as the normal occurrence, or not diagnosed in the past. It is from the records of five hundred cases that it is hoped to determine the "Dangers of Modern Anaesthetics".

These cases have been recorded during the past three years at Stobhill Hospital, Glasgow, while holding the appointments of Trainee Anaesthetist, and later, Anaesthetist. Both appointments were "full-time", allowing time to be spent with the patients in the wards during the pre-operative and post-operative periods. The cases have been drawn from the surgical, paediatric, obstetrical and gynaecological wards, with a few from the unit specialising in diseases of the ear, throat and nose.

The principal reason for the investigation is dissatisfaction with the results of anaesthesia as now

practised. An attempt has been made to discover the faults in the modern technique, whether the error lies in the premedication of the patient, the anaesthesia itself, or the post-operative care of the patient, and to offer suggestions for their correction.

Five hundred cases undergoing anaesthesia in Stobhill Hospital, Glasgow have been recorded on Copeland-Chatterton Paramount Anaesthetic Record Cards. Adequate time for "follow-up" was the sole factor in the choice of each case.

PATIENT'S NAME		DATE		AGE		SEX		WEIGHT		HEIGHT		TEMP.		PULSE		B.P.		RESPIR.		O ₂		CO ₂		S.P.O ₂		ET CO ₂		ET O ₂		ET N ₂		ET F ₂		ET F ₁		ET F ₀		ET F ₋₁		ET F ₋₂		ET F ₋₃		ET F ₋₄		ET F ₋₅		ET F ₋₆		ET F ₋₇		ET F ₋₈		ET F ₋₉		ET F ₋₁₀		ET F ₋₁₁		ET F ₋₁₂		ET F ₋₁₃		ET F ₋₁₄		ET F ₋₁₅		ET F ₋₁₆		ET F ₋₁₇		ET F ₋₁₈		ET F ₋₁₉		ET F ₋₂₀		ET F ₋₂₁		ET F ₋₂₂		ET F ₋₂₃		ET F ₋₂₄		ET F ₋₂₅		ET F ₋₂₆		ET F ₋₂₇		ET F ₋₂₈		ET F ₋₂₉		ET F ₋₃₀		ET F ₋₃₁		ET F ₋₃₂		ET F ₋₃₃		ET F ₋₃₄		ET F ₋₃₅		ET F ₋₃₆		ET F ₋₃₇		ET F ₋₃₈		ET F ₋₃₉		ET F ₋₄₀		ET F ₋₄₁		ET F ₋₄₂		ET F ₋₄₃		ET F ₋₄₄		ET F ₋₄₅		ET F ₋₄₆		ET F ₋₄₇		ET F ₋₄₈		ET F ₋₄₉		ET F ₋₅₀		ET F ₋₅₁		ET F ₋₅₂		ET F ₋₅₃		ET F ₋₅₄		ET F ₋₅₅		ET F ₋₅₆		ET F ₋₅₇		ET F ₋₅₈		ET F ₋₅₉		ET F ₋₆₀		ET F ₋₆₁		ET F ₋₆₂		ET F ₋₆₃		ET F ₋₆₄		ET F ₋₆₅		ET F ₋₆₆		ET F ₋₆₇		ET F ₋₆₈		ET F ₋₆₉		ET F ₋₇₀		ET F ₋₇₁		ET F ₋₇₂		ET F ₋₇₃		ET F ₋₇₄		ET F ₋₇₅		ET F ₋₇₆		ET F ₋₇₇		ET F ₋₇₈		ET F ₋₇₉		ET F ₋₈₀		ET F ₋₈₁		ET F ₋₈₂		ET F ₋₈₃		ET F ₋₈₄		ET F ₋₈₅		ET F ₋₈₆		ET F ₋₈₇		ET F ₋₈₈		ET F ₋₈₉		ET F ₋₉₀		ET F ₋₉₁		ET F ₋₉₂		ET F ₋₉₃		ET F ₋₉₄		ET F ₋₉₅		ET F ₋₉₆		ET F ₋₉₇		ET F ₋₉₈		ET F ₋₉₉		ET F ₋₁₀₀	
1. Name		2. Date		3. Age		4. Sex		5. Weight		6. Height		7. Temp.		8. Pulse		9. B.P.		10. Respir.		11. O ₂		12. CO ₂		13. S.P.O ₂		14. ET CO ₂		15. ET O ₂		16. ET N ₂		17. ET F ₂		18. ET F ₁		19. ET F ₀		20. ET F ₋₁		21. ET F ₋₂		22. ET F ₋₃		23. ET F ₋₄		24. ET F ₋₅		25. ET F ₋₆		26. ET F ₋₇		27. ET F ₋₈		28. ET F ₋₉		29. ET F ₋₁₀		30. ET F ₋₁₁		31. ET F ₋₁₂		32. ET F ₋₁₃		33. ET F ₋₁₄		34. ET F ₋₁₅		35. ET F ₋₁₆		36. ET F ₋₁₇		37. ET F ₋₁₈		38. ET F ₋₁₉		39. ET F ₋₂₀		40. ET F ₋₂₁		41. ET F ₋₂₂		42. ET F ₋₂₃		43. ET F ₋₂₄		44. ET F ₋₂₅		45. ET F ₋₂₆		46. ET F ₋₂₇		47. ET F ₋₂₈		48. ET F ₋₂₉		49. ET F ₋₃₀		50. ET F ₋₃₁		51. ET F ₋₃₂		52. ET F ₋₃₃		53. ET F ₋₃₄		54. ET F ₋₃₅		55. ET F ₋₃₆		56. ET F ₋₃₇		57. ET F ₋₃₈		58. ET F ₋₃₉		59. ET F ₋₄₀		60. ET F ₋₄₁		61. ET F ₋₄₂		62. ET F ₋₄₃		63. ET F ₋₄₄		64. ET F ₋₄₅		65. ET F ₋₄₆		66. ET F ₋₄₇		67. ET F ₋₄₈		68. ET F ₋₄₉		69. ET F ₋₅₀		70. ET F ₋₅₁		71. ET F ₋₅₂		72. ET F ₋₅₃		73. ET F ₋₅₄		74. ET F ₋₅₅		75. ET F ₋₅₆		76. ET F ₋₅₇		77. ET F ₋₅₈		78. ET F ₋₅₉		79. ET F ₋₆₀		80. ET F ₋₆₁		81. ET F ₋₆₂		82. ET F ₋₆₃		83. ET F ₋₆₄		84. ET F ₋₆₅		85. ET F ₋₆₆		86. ET F ₋₆₇		87. ET F ₋₆₈		88. ET F ₋₆₉		89. ET F ₋₇₀		90. ET F ₋₇₁		91. ET F ₋₇₂		92. ET F ₋₇₃		93. ET F ₋₇₄		94. ET F ₋₇₅		95. ET F ₋₇₆		96. ET F ₋₇₇		97. ET F ₋₇₈		98. ET F ₋₇₉		99. ET F ₋₈₀		100. ET F ₋₈₁		101. ET F ₋₈₂		102. ET F ₋₈₃		103. ET F ₋₈₄		104. ET F ₋₈₅		105. ET F ₋₈₆		106. ET F ₋₈₇		107. ET F ₋₈₈		108. ET F ₋₈₉		109. ET F ₋₉₀		110. ET F ₋₉₁		111. ET F ₋₉₂		112. ET F ₋₉₃		113. ET F ₋₉₄		114. ET F ₋₉₅		115. ET F ₋₉₆		116. ET F ₋₉₇		117. ET F ₋₉₈		118. ET F ₋₉₉		119. ET F ₋₁₀₀	

PATIENT'S NAME		DATE		AGE		SEX		WEIGHT		HEIGHT		TEMP.		PULSE		B.P.		RESPIR.		O ₂		CO ₂		S.P.O ₂		ET CO ₂		ET O ₂		ET N ₂		ET F ₂		ET F ₁		ET F ₀		ET F ₋₁		ET F ₋₂		ET F ₋₃		ET F ₋₄		ET F ₋₅		ET F ₋₆		ET F ₋₇		ET F ₋₈		ET F ₋₉		ET F ₋₁₀		ET F ₋₁₁		ET F ₋₁₂		ET F ₋₁₃		ET F ₋₁₄		ET F ₋₁₅		ET F ₋₁₆		ET F ₋₁₇		ET F ₋₁₈		ET F ₋₁₉		ET F ₋₂₀		ET F ₋₂₁		ET F ₋₂₂		ET F ₋₂₃		ET F ₋₂₄		ET F ₋₂₅		ET F ₋₂₆		ET F ₋₂₇		ET F ₋₂₈		ET F ₋₂₉		ET F ₋₃₀		ET F ₋₃₁		ET F ₋₃₂		ET F ₋₃₃		ET F ₋₃₄		ET F ₋₃₅		ET F ₋₃₆		ET F ₋₃₇		ET F ₋₃₈		ET F ₋₃₉		ET F ₋₄₀		ET F ₋₄₁		ET F ₋₄₂		ET F ₋₄₃		ET F ₋₄₄		ET F ₋₄₅		ET F ₋₄₆		ET F ₋₄₇		ET F ₋₄₈		ET F ₋₄₉		ET F ₋₅₀		ET F ₋₅₁		ET F ₋₅₂		ET F ₋₅₃		ET F ₋₅₄		ET F ₋₅₅		ET F ₋₅₆		ET F ₋₅₇		ET F ₋₅₈		ET F ₋₅₉		ET F ₋₆₀		ET F ₋₆₁		ET F ₋₆₂		ET F ₋₆₃		ET F ₋₆₄		ET F ₋₆₅		ET F ₋₆₆		ET F ₋₆₇		ET F ₋₆₈		ET F ₋₆₉		ET F ₋₇₀		ET F ₋₇₁		ET F ₋₇₂		ET F ₋₇₃		ET F ₋₇₄		ET F ₋₇₅		ET F ₋₇₆		ET F ₋₇₇		ET F ₋₇₈		ET F ₋₇₉		ET F ₋₈₀		ET F ₋₈₁		ET F ₋₈₂		ET F ₋₈₃		ET F ₋₈₄		ET F ₋₈₅		ET F ₋₈₆		ET F ₋₈₇		ET F ₋₈₈		ET F ₋₈₉		ET F ₋₉₀		ET F ₋₉₁		ET F ₋₉₂		ET F ₋₉₃		ET F ₋₉₄		ET F ₋₉₅		ET F ₋₉₆		ET F ₋₉₇		ET F ₋₉₈		ET F ₋₉₉		ET F ₋₁₀₀	
1. Name		2. Date		3. Age		4. Sex		5. Weight		6. Height		7. Temp.		8. Pulse		9. B.P.		10. Respir.		11. O ₂		12. CO ₂		13. S.P.O ₂		14. ET CO ₂		15. ET O ₂		16. ET N ₂		17. ET F ₂		18. ET F ₁		19. ET F ₀		20. ET F ₋₁		21. ET F ₋₂		22. ET F ₋₃		23. ET F ₋₄		24. ET F ₋₅		25. ET F ₋₆		26. ET F ₋₇		27. ET F ₋₈		28. ET F ₋₉		29. ET F ₋₁₀		30. ET F ₋₁₁		31. ET F ₋₁₂		32. ET F ₋₁₃		33. ET F ₋₁₄		34. ET F ₋₁₅		35. ET F ₋₁₆		36. ET F ₋₁₇		37. ET F ₋₁₈		38. ET F ₋₁₉		39. ET F ₋₂₀		40. ET F ₋₂₁		41. ET F ₋₂₂		42. ET F ₋₂₃		43. ET F ₋₂₄		44. ET F ₋₂₅		45. ET F ₋₂₆		46. ET F ₋₂₇		47. ET F ₋₂₈		48. ET F ₋₂₉		49. ET F ₋₃₀		50. ET F ₋₃₁		51. ET F ₋₃₂		52. ET F ₋₃₃		53. ET F ₋₃₄		54. ET F ₋₃₅		55. ET F ₋₃₆		56. ET F ₋₃₇		57. ET F ₋₃₈		58. ET F ₋₃₉		59. ET F ₋₄₀		60. ET F ₋₄₁		61. ET F ₋₄₂		62. ET F ₋₄₃		63. ET F ₋₄₄		64. ET F ₋₄₅		65. ET F ₋₄₆		66. ET F ₋₄₇		67. ET F ₋₄₈		68. ET F ₋₄₉		69. ET F ₋₅₀		70. ET F ₋₅₁		71. ET F ₋₅₂		72. ET F ₋₅₃		73. ET F ₋₅₄		74. ET F ₋₅₅		75. ET F ₋₅₆		76. ET F ₋₅₇		77. ET F ₋₅₈		78. ET F ₋₅₉		79. ET F ₋₆₀		80. ET F ₋₆₁		81. ET F ₋₆₂		82. ET F ₋₆₃		83. ET F ₋₆₄		84. ET F ₋₆₅		85. ET F ₋₆₆		86. ET F ₋₆₇		87. ET F ₋₆₈		88. ET F ₋₆₉		89. ET F ₋₇₀		90. ET F ₋₇₁		91. ET F ₋₇₂		92. ET F ₋₇₃		93. ET F ₋₇₄		94. ET F ₋₇₅		95. ET F ₋₇₆		96. ET F ₋₇₇		97. ET F ₋₇₈		98. ET F ₋₇₉		99. ET F ₋₈₀		100. ET F ₋₈₁		101. ET F ₋₈₂		102. ET F ₋₈₃		103. ET F ₋₈₄		104. ET F ₋₈₅		105. ET F ₋₈₆		106. ET F ₋₈₇		107. ET F ₋₈₈		108. ET F ₋₈₉		109. ET F ₋₉₀		110. ET F ₋₉₁		111. ET F ₋₉₂		112. ET F ₋₉₃		113. ET F ₋₉₄		114. ET F ₋₉₅		115. ET F ₋₉₆		116. ET F ₋₉₇		117. ET F ₋₉₈		118. ET F ₋₉₉		119. ET F ₋₁₀₀	

The details on the front of the card are filled in during the pre-operative and post-operative visits to the patient, and those on the back during the operation.(2).

One of the criticisms of modern anaesthesia is that although the patient's condition is excellent throughout the actual administration of the anaesthetic, a deterioration sets in during the immediate post-operative period. In an attempt to evaluate this criticism, the blood-pressure has been recorded in some cases four hours after the patient has returned to the ward, and this figure compared with the pre-operative recording and that taken at the end of the operation.

It has been said that any revolutionary discovery changing the course of anaesthesia is unlikely, but that progress will be achieved by a series of comparatively small improvements in technique (3).

In this thesis it is intended to discuss the defects in anaesthesia as it is practised today as shown by the study of five hundred cases.

History.

The history of anaesthesia during the past thirty years might be described as the development of a specialised branch of medicine and the addition of a science to an art. With the description of Guedel of the signs of anaesthesia in 1920, the principles first laid down by John Snow in 1858 were reproduced in more detail.

Before this period anaesthesia had been used almost entirely for operative procedures. Some anaesthetists did employ their art in obstetric analgesia, but on the whole this was left to the obstetrician who might administer some form of 'twilight sleep' and supervise the administration of a short, light chloroform anaesthesia for the actual birth. The report of Gwathmey in 1930 concerning the administration of 20,000 anaesthetics to women in labour stimulated the interest of anaesthetists not only in America, but throughout the world in the problem of easing the pain of labour. In 1933, Minnitt developed his 'gas-and-air' analgesic apparatus for the use of midwives in domestic as well as hospital practice. In America there are many who favour a form of regional analgesia rather than that produced by inhalation of gases and vapours. In 1942 Edwards and Hingson developed the technique of continuous caudal analgesia. This has come to be used also in the treatment

of eclampsia and, in certain cases, to prevent the onset of labour. Spinal analgesia has also entered the field of therapeutics, in eclampsia, and in those conditions thought to be due to an imbalance of the autonomic nervous system such as congenital megacolon. It has also been recognised as a diagnostic agent in thrombo-angiitis obliterans.

Anaesthesia for operative procedures has made many advances with new apparatus, drugs and techniques. The apparatus has become more complicated. The Boyle anaesthetic apparatus has always been one of the most commonly used in this country. Its development, from the first model to the latest, illustrates very well the changes in anaesthesia, the greater accuracy and the greater complexity. The original 'Bubble-type' flowmeter has been superseded by the more accurate 'Bobbin-type' which has in turn given way to the 'Rotameter'. Reducing valves have replaced the original fine-adjustment valves, and some form of circle-type absorption apparatus added to allow the use of cyclopropane. The result is a complicated, expensive apparatus, in complete contrast to the simplicity and cheapness of the 'rag and bottle' method. The problem arises regarding the teaching of students to use a machine so dangerous in unskilled hands and so expensive to buy that they are unlikely

to use it in the future unless they join the ranks of the specialist anaesthetists.

Several new general anaesthetic agents have been added to the anaesthetists' pharmacopoea, several inhalational, and for the first time, an efficient intravenous general anaesthetic.

In 1928 the anaesthetic properties of cyclopropane were observed by Henderson and Lucas, who noted it as an impurity of propylene, in which they were primarily interested. They found it more efficacious than propylene and investigated its properties experimentally in animals. The drug was introduced into clinical anaesthesia by Waters and his associates in 1933 (4). The gas was investigated in Great Britain by Rowbotham and the anaesthetic staff of The Cancer Hospital, London (5). The association of the names Waters and cyclopropane might well be expected when one considers that cyclopropane requires to be administered in a closed circuit apparatus, in the development of which Waters was one of the pioneers.

Two years after the original work on cyclopropane, Leake and Chen discovered the anaesthetic properties of di-vinyl ether or "Vinesthene" (6). Three years later it was introduced into clinical anaesthesia by Gelfan and Bell, using one another as subject (7). The Medical Research

Council have reported it as "an anaesthetic of undoubted value", and it was approved by the Council on Pharmacy and Chemistry of the American Medical Association. One of the disadvantages of this drug is its extreme volatility. To avoid waste, Bourne and others recommended mixing di-vinyl ether and di-ethyl ether in the proportions of one to three (8).

The third inhalational agent, which is now generally recognised to have a definite place in anaesthesia and was introduced in the past thirty years, is trichlorethylene, or "Trilene". In 1935 Stricker and his associates (9) described a series of three hundred short administrations for minor operations. The results were inconclusive, and in the following year the Council on Pharmacy and Chemistry of the American Medical Association considered that "the case had not been completely made out" for the usefulness of the drug. In 1939 the joint Anaesthetic Committee of the Medical Research Council and the Royal Society of Medicine asked C. Langton Hewer to investigate the properties of trichlorethylene. The investigation was carried out in the department of anaesthesia of Saint Bartholomew's Hospital and the results embodied in three papers (10,11,12), when trichlorethylene was shown to be an excellent inhalant drug for producing general analgesia, and useful for light anaesthesia (13).

During this era there were outstanding developments in intravenous anaesthesia. Prior to this, many agents had been tried to produce anaesthesia by this method with no great success. During the years 1920-1930 many derivatives of barbituric acid were investigated, among them Pernostan in 1927 by Bumm in Germany, Amytal in 1929 by Serfas and his associates in America, and Nembutal in 1931 by Lundy, also in America. Intravenous anaesthesia, however, really began with the introduction of Evipan in 1932 by two Germans, Weese and Schaff. In 1933 the Medical Research Council decided that the encouraging reports from German surgeons and anaesthetists warranted the clinical trial of Evipan in this country, and accordingly a panel of anaesthetists was formed to carry out the investigation. In America, a similar trial was carried out by John S. Lundy, and in the following year he introduced "Pentothal", which was even more efficacious than Evipan. "Pentothal" was first used in Britain by Jarman who had been one of the pioneers in the use of Evipan. Since then "Pentothal", or as it is now officially named, thiopentone sodium, has become one of the anaesthetic agents most commonly used.

To match the developments in surgery, anaesthetic apparatus and drugs, there have been many developments in anaesthetic technique. For example, it is now customary, where possible, to induce general anaesthesia with one

of the ultra-short-acting barbiturates given by intravenous injection thus sparing the patient the feeling of the face-piece over the face.

Endotracheal anaesthesia is not a new development. The first time endotracheal anaesthetic was administered was by Sir William Macewen in 1880. In 1912 Kelly introduced into England insufflation endotracheal anaesthesia which was pioneered in America by Meltzer, Auer and Elsberg. As a result of their experiences in the 1914-18 war Magill and Rowbotham used, first insufflation, and later inhalational, endotracheal methods, using soft-wide-bore rubber tubes. In 1928 Magill described his method of blind, 'nasal' intubation, and Guedel and Waters re-introduced the fitting of inflatable cuffs to endotracheal tubes. In 1932 Gale and Waters demonstrated closed endobronchial anaesthesia by passing a similar cuffed tube into either main bronchus. This technique may be taken as a further example of the increasing complexity of anaesthesia. Chevalier Jackson's work on direct laryngoscopy stimulated interest in intubation. Anaesthetists gradually became laryngoscopists, and now, to be able to anaesthetise for thoracic surgery and be able to deal competently with post-operative complications, must be bronchoscopists also.

As long ago as 1850 John Snow discovered the principle

of carbon dioxide absorption and closed-circuit anaesthesia. It was rediscovered by Jackson in America in 1915 and used in anaesthesia involving animals. Until the work of Lee, it used to be thought that exhaled air contained a poisonous substance, "Anthropotoxin" (14). In the same year Waters developed the technique for clinical anaesthesia using "Soda-lime" to absorb the carbon dioxide. The simple apparatus bearing Waters' name has certain disadvantages, and in an effort to overcome these, the "circle" type of absorption unit was introduced five years later by Brian Sword. Since then, the number of "circle" units has increased till they are now legion.

In 1913 Crile of Cleveland published his Kinetic Theory of Shock (15), and seven years later with Lower he pioneered the technique of combined local analgesia and general anaesthesia in the prevention of shock from surgical operations (16). This theory of the causation of shock has been shown to be only one of the many factors involved, but Crile's Anoci Association Theory was so effective in practice that most anaesthetists made use of the theory to a greater or lesser extent until the following technique came into being and almost completely displaced the combination of 'local' and general anaesthesia.

During the past ten years a discovery has been made that has been described as "A Milestone in Anaesthesia" (17). In January 1942 Griffith of Montreal first used curare as an aid to anaesthesia. Two years later, Halton in England used tube-curarine chloride in thoracic surgery, but it was not till the following year that he realised its possibilities as a means of producing muscular relaxation in general surgery. By 1946 curare was in general use in Britain. Its advantage is the profound relaxation of spinal analgesia or deep general anaesthesia at a light plane of anaesthesia. Its undesirable action on respiration led to the introduction of "Myanesin" in 1947. In composition this is a substitution product of one of the ethers of glycerol. Like curare, this drug is given by intravenous injection. It has, however, the disadvantage of causing venous thrombosis and haemolysis of the red blood cells.

The name of Labat is outstanding in the recent history of regional analgesia. In 1920 in America he demonstrated local, regional or spinal analgesic techniques for almost all surgical operations, and two years later he published his book "Regional Analgesia" which was to prove the standard textbook on this subject. Also in 1920 a Spanish surgeon, Fedel Pages applied to the human subject the technique of epidural analgesia, but it has always been

considered less reliable than subarachnoid block. However, the alternative method of entering the peridural space through the sacral canal has become more popular especially in America since Edwards and Hingson developed the technique of continuous caudal analgesia for the relief of pain in childbirth in an attempt to produce perfect analgesia for the mother without causing foetal upset or disturbance of the normal uterine contractions. In the same year Allen and others (18) rediscovered the technique of amputating limbs under refrigeration analgesia. Although the decreased metabolism due to refrigeration is made use of in the treatment of gangrene of the lower limbs, it is not now used so extensively to provide the analgesia for the actual amputation. In 1943 a technique of posterior intercostal and splanchnic blocks to provide analgesia for abdominal surgery was described by James (19).

During the immediate pre-curare chapter in the history of anaesthesia there was a period of increased popularity of regional analgesia. Anaesthetists were becoming more and more expert at, and surgeons more and more inclined to delegate, regional analgesia. But with the advent of curare, the decline in popularity set in, and regional analgesia became confined to those cases who must not have a general anaesthesia, no matter how "light" it might be.

Regional analgesia was not alone in the decline in popularity due to the relaxant drugs. Spinal analgesia had been making great progress, and with the technique of 'fractional' or 'continuous' spinal, sub-arachnoid block had been placed on a rational, if not completely safe, basis.

Prior to the work of Labat (1921) and Pitkin (1927) when the former dissolved novocain in cerebro-spinal fluid and injected the solution, and the latter introduced "Spinocain", popularising spinal analgesia, the technique was not safe and was infrequently used. Also in 1927, Ockerblad and Dillon in America used ephedrine to maintain the level of the blood-pressure during spinal analgesia and a further step was made in the direction of safety in sub-arachnoid analgesia. Three and four years later Howard Jones and Etherington Wilson described their respective methods for the use of the first true hypobaric drug, "Light Nupercaine". Hyperbasic "Nupercaine" was introduced by Frankis Evans in 1934. In 1940 Lemmon introduced 'fractional' or 'continuous' spinal.

During this period various vasopressor drugs were used to maintain the blood-pressure at a level as near to the patient's normal as possible. To allow fine adjustment of this level Frankis Evans introduced the method of using a continuous intravenous drip infusion of an extremely dilute solution of adrenalin. In complete contrast to this,

Griffith and Gillies in 1948 published a description of the technique for "total spinal" for use in thoracico-lumbar splanchnic ectomy and sympathectomy (20). In order to prevent bleeding no vasopressor agent is given, and the blood-pressure is allowed to fall to levels which cannot be recorded, effective oxygenation of the patient's blood being maintained by assisted respiration.

In this chapter in the history of anaesthesia there has been an increase in the complexity of the apparatus, an increase in the number of drugs at the disposal of the anaesthetist, a tendency to discard the more toxic drugs, and an attempt not only to give the surgeon maximum relaxation, but, as shown by the 'total spinal', an attempt has been made to facilitate his task still further by minimising haemorrhage.

A change in the status of the anaesthetist has taken place as the result of these discoveries and changes in anaesthesia. Specialists in anaesthesia made their appearance in increasing numbers. In 1932 the British Medical Association formed a section devoted to the study and furtherance of knowledge of anaesthesia. In 1935, as a result of negotiations between the Association of Anaesthetists of Great Britain and Ireland and the Royal Colleges of Physicians and Surgeons, a Diploma in Anaesthetics was instituted by

these two bodies. By 1948 it had become apparent that the standard of the diploma had not kept pace with the rapid increase in the scope of anaesthesia (21) and it was decided to revise the regulations and syllabus for the examination in order to raise the highest grade of specialist anaesthetist to the same academic standing as that of other specialists. In the same year a Faculty of Anaesthesia was formed by the Royal College of Surgeons of England.

It is no longer sufficient for the anaesthetist to keep his patient quiet and relaxed on the operating table. With the drugs and equipment now at his disposal he is able to give the surgeon optimum operating conditions with the minimum of upset to the patient. To do so means careful assessment of the patient in the period before operation, the choice of drugs and method of their use at the actual operation, the ability to carry out the proposed technique and the opportunity to assess the results in the post-operative period. These conditions may only be obtained by devoting one's time solely to the practice of anaesthesia.

These improvements in anaesthesia are not reflected in the mortality and morbidity statistics associated with anaesthesia in this period. In England and Wales in 1901 there were ninety one deaths, and in 1941 there were eight

hundred and thirty five deaths associated with anaesthesia (22,23). In one American hospital pulmonary complications following abdominal surgery rose from 2.0 per cent in 1927 to 5.3 per cent in 1939 (24).

It is therefore proposed to discuss the dangers of modern anaesthetic agents and techniques in the following section.

The Specific Dangers of Modern Anaesthetic Agents.

Although many changes have taken place in anaesthesia during the twentieth century and many advances have been made, the drug giving ideal operating conditions with no untoward side-effects has yet to be discovered. Much has been achieved in making the ordeal of anaesthesia easier for the patient. The use of thiopentane has made the induction easy and pleasant, and the maintenance by nitrous oxide or cyclopropane spares the patient the distressing post-anaesthetic vomiting liable to follow ether and chloroform. Muscle relaxation required in abdominal and thoracic operations may be provided by curare. If the patient elects to remain awake during the operation most surgical procedures may now be performed under some form of regional analgesia.

Unfortunately with these new drugs and techniques new complications occur which did not accompany or follow the older methods.

Cyclopropane is capable of carrying anaesthesia through the four stages and has consequently been accorded a potency of 100 per cent by Guedel (25). It has been described as standing midway between ether and chloroform for safety and potency (26). The extreme power of the agent and the extreme rapidity with which a high concentration

of the gas may be built up in the blood and brain cells of the patient make the danger of over-dosage very real (27). The time for the anaesthesia to progress from induction to respiratory paralysis may be as short as from one half to three minutes. This rapid abolition of consciousness will allow the operation to begin before the anaesthesia has been stabilised (25,28).

Many authorities consider that cyclopropane depresses respiration. With this Waters does not agree. He considers the gas to be neither a respiratory depressant nor stimulant, and in his opinion the apparent depression of respiration results from comparison with other anaesthetic agents, all of which are active respiratory stimulants. He admits, however, that respiration is quiet and that paralysis of the intercostal muscles is apt to be complete before it is noticed (29,30,25).

As the drug does not stimulate respiration, the margin between the degree of partial paralysis of the muscles of respiration which occurs with relaxation of the muscles of the upper abdomen and complete respiratory arrest is narrow. Respiratory arrest may even supervene before complete relaxation has been obtained (27,31).

Cyclopropane may reduce the tidal respiration below the level required for the adequate exposure of the expired

gases to the soda-lime of the absorber unit (32).

With this agent, the nature and amount of the drugs used as premedication are especially important. Morphine is a powerful respiratory depressant and with cyclopropane a far greater degree of depression results than is generally appreciated. If cyclopropane is the anaesthetic of choice, then the dose of morphine employed should be much smaller than usual (30).

Cyclopropane may cause bronchoconstriction by stimulating the parasympathetic nervous system. But it also directly depresses the bronchial muscle. When the anaesthesia has been in progress for a sufficient period the bronchial muscle becomes relaxed, but if the initial concentration of the gas in the inhaled mixture has been too strong or if stimulation has occurred from the operation being begun while the anaesthesia is still incomplete, then this action will be overshadowed by the bronchial constriction (33, 27, 28).

On account of the extreme potency of the gas, anaesthesia may be maintained with a high percentage of oxygen in the inhaled mixture. Oxygen and cyclopropane are both absorbed rapidly from the pulmonary alveoli, and, owing to the quiet respiration of cyclopropane anaesthesia, a massive pulmonary collapse may occur unnoticed on the operating table (25).

The use of cyclopropane is not without ill-effects on the cardiovascular system.

It is suggested that the level of the blood-pressure remains unchanged if the respiratory efficiency is not diminished (34). But frequently the reduced tidal respiration causes an accumulation of carbon dioxide in the blood and a fall in the hydrogen ion concentration of the plasma due to an inadequate exposure of the expired gases to the soda-lime. As a result the systolic and diastolic blood pressures rise (30,32). When the respiratory depressing cyclopropane is withdrawn at the end of anaesthesia or complete rebreathing is suddenly stopped, the plasma hydrogen ion concentration and carbon dioxide content fall, and the blood pressure does likewise (35).

Waters in 1937 in discussing the toxic effects of carbon dioxide, described a syndrome simulating collapse which occurred in hypersensitive individuals exposed to an excess of carbon dioxide as follows: "A somewhat similar picture may appear directly following a period during which an individual has been subjected to an atmosphere too rich in carbon dioxide. It is then probably due to the sudden withdrawal of the excess carbon dioxide for which adjustment of the buffer mechanism of the blood and tissues has been made, perhaps the excess carbon dioxide has been blown off

more rapidly than it is possible for the buffer mechanism to keep pace". (36). This failure of the buffer mechanism may be the cause of "Cyclopropane Shock". During the anaesthesia the blood pressure has been above the pre-operative level and the patient's general condition has been satisfactory. When the mask is removed, the condition of the patient deteriorates, the systolic and diastolic blood pressures fall to a variable extent and at a variable rate. In some cases the hypotension occurs at once. Within three to five minutes the systolic blood pressure has fallen to 50-60 millimetres of mercury or lower. The hypotension, however, may take place over a period of time and may not reach such alarming proportions. Associated with a rapid fall in the blood pressure, the patient may develop a clammy skin, a pulse of poor volume, and become delirious during his emergence from the anaesthesia. The difference between this condition and surgical shock is that in the former the patient has a bradycardia. In some cases, the patients appear to be better than they are, and the level of the blood pressure suggests. They are warm, the skin is dry, the pulse slow, and the patients are orientated and alert. This hypotension may persist for several hours despite the intravenous administration of plasma (30,37).

Although tachycardia has been described in dogs, bradycardia is more frequent during clinical cyclopropane anaesthesia. This is even more frequent after morphine premedication than after cyclopropane alone. The slowing of the heart-rate offers more time for diastolic filling of the heart and consequently an increase in the stroke volume (38,29,33,30,34,31).

There is a direct relationship between the concentration of the drug in the inhaled mixture and the effects on the circulatory system. When this concentration exceeds thirty per cent or the tension of the gas in the tissue cells has been raised to an unduly high level by the cumulative effect of the prolonged administration of even a weak mixture, cardiac arrhythmias may arise. The cyclopropane excites the ventricular muscle of the heart into the production of ectopic contractions. When this concentration of cyclopropane falls, the cardiac rhythm returns to normal (31,27).

Arrhythmias are common, and, according to Adriani, include displacement of the pacemaker, vagus escape, auriculo-ventricular block and ventricular tachycardia, the latter occurring at all times during the anaesthesia, but more commonly in the lower planes (33). The arrhythmias occurring early in the anaesthesia may be of vagal origin as they may be abolished by the administration of atropine,

and they are not indicative of permanent damage to the heart (39).

Ten per cent of the cases of one series of patients under cyclopropane anaesthesia showed "multiple focus ventricular tachycardia" which invariably preceded ventricular fibrillation in the cats Levy experimented upon with chloroform. Clinically, this causes an irregular irregularity in the pulse with a pulse deficit of 30-100 beats per minute (39).

Cyclopropane reflexly sensitises the heart of the dog to injected adrenaline. The receptor nerve-endings are distributed for the most part throughout the peripheral three centimetres of the mesentery. Impulses travel by the visceral afferent fibres through the coeliac and superior mesenteric plexuses, splanchnic nerves and spinal cord to a centre in the brain above the pons. Efferent impulses then pass to the heart by way of the cardiac branches of the sympathetic nervous system and increase the irritability of the heart (40).

The administration of adrenalin to a patient under cyclopropane anaesthesia leads to marked disturbance of the cardiac rhythm particularly of the ventricular type and may result in fatal ventricular fibrillation. For this reason induction of anaesthesia by cyclopropane is not advised in

the apprehensive patient because of this risk of ventricular fibrillation (41,33,42).

In one series of 1000 cases of cyclopropane anaesthesia, two developed auricular fibrillation and two others died on the table (43).

Although the increased ooze at the skin wound does not have any serious effect by itself, the prolongation of the actual operation due to this haemorrhage may be detrimental to the patient and therefore must be included in the list of undesirable side actions of cyclopropane. The ooze may be due to an increase in the diameter of the capillaries, due to a depression of the vasomotor centre or an increase in the venous pressure (30,43,42).

The maintenance of a smooth level of anaesthesia with cyclopropane is not easy. Sudden alterations in the depth of the anaesthesia are liable to occur. Although cyclopropane is one hundred per cent potent, it produces little pupillary dilatation. With ordinary premedication as employed for other agents (Morphine gr. $\frac{1}{4}$ and Atropine gr. $\frac{1}{100}$) the fourth stage of anaesthesia is often entered without pupillary dilatation until the reserve oxygen supply of the tissues is used up, when dilatation occurs from oxygen want. The anaesthetist may be given a further

false sense of security by the inadequate pulmonary ventilation being obscured by an oxygen concentration in the inhaled mixture greater than twenty per cent (43,25,27,44).

Cyclopropane has been extensively used as the anaesthetic agent for the operation of Caesarian Section. "Pituitrin shock" appears to be accentuated when it occurs with cyclopropane (45).

This gas has been described as one of the most dangerous anaesthetic agents. The death rate is high even when the gas is used by those thought to be competent. Prolonged administration is dangerous because it conceals the true clinical condition of the patient, tending to elevate the blood pressure and reduce the pulse rate. Soon after the administration is discontinued, the patient may develop "cyclopropane shock", which may be combatted with difficulty (46).

Intravenous anaesthesia is the most pleasant for the patient, and the most time-saving for the surgeon and the anaesthetist. The latter is therefore frequently tempted or requested to administer this type of anaesthesia when it is relatively or completely contra-indicated. The danger lies in the simplicity of the apparatus required and the ease with which the induction and maintenance may be carried out as compared with inhalational anaesthesia.

One of the difficulties of intravenous anaesthesia is the estimation of the dose of the agent. The controllability of the anaesthesia is in proportion to a great extent to the rate of detoxication of the drug in the body tissues. This is subject to a number of variable influences, such as age, weight, sex, and metabolic rate of the patient. But the dose cannot be judged on these factors alone. Considerable control of the anaesthesia may be exercised by assessing hepatic and renal efficiency, metabolic rate and circulation time and the rate of administration judged in such a way as to prevent overdosage and cumulative effect. Deaths have occurred due to relative overdosage in cases suffering from shock and haemorrhage. After the Japanese attack on Pearl Harbour many of the casualties were anaesthetised with "Pentothal" for a variety of operations by men not familiar with the limitations and dangers of intravenous anaesthesia with the result that the mortality rate was one in four hundred and fifty. One has been impressed many times by the minute dose required for the induction of anaesthesia in women suffering from shock and haemorrhage due to incomplete abortions (47,31,48).

The continuous administration of thiopentone in long operations requires even greater caution on the part of the anaesthetist. A fit, healthy adult is able to detoxicate half a gram of the drug quickly, but the succeeding

doses must be given slowly because in prolonged deep anaesthesia the process of detoxication is retarded and a cumulative effect results, leading to overdosage. It has been suggested that the dose of two grams should not be exceeded in such cases (31,49).

Intravenous anaesthesia has a profound effect on respiration and consequently on the chemistry of the blood. The respiratory minute-volume exchange is decreased, and this respiratory depression may cause embarrassment if the intravenous anaesthesia is being used as an inducing agent for an inhalational anaesthesia (33,50).

In the human subject and the experimental animal, if premedication has been given, moderately deep anaesthesia with "Pentothal" does not cause a significant decrease in the level of oxygen in the blood. If pure oxygen is administered with the "Pentothal", there is a significant rise in this level (51). But in a series of animals deeply anaesthetised with "Pentothal" there was a decrease in the degree of response to oxygen lack and the period of time between the cessation of respiration and the fall of arterial blood pressure to zero was shorter than with "light" ether, "Vinesthene", chloroform, nitrous oxide and oxygen anaesthetics, but longer than with "deep" ether. The concentration of carbon dioxide in the blood is increased after

thiopentone anaesthesia (52). The failure of the patient to exhibit a rise in the arterial blood pressure due to this increased carbon dioxide tension has been attributed to the depression of the vasomotor centre by the drug (30).

Consequently it has been advised that "Pentothal" should be given only by competent physicians with facilities at hand to combat the respiratory depression and the imbalance of the oxygen and carbon dioxide in the blood (47).

Unfortunately the laryngeal and tracheal reflexes are not obtunded by this form of anaesthesia. Hyperactivity may rather result, and any stimulation lead to spasm, especially if the stimulus be an attempt to pass an endotracheal tube (50,33).

Hyperactivity of the mucous glands of the respiratory tract may also occur. A case has been described in which the patient secreted such enormous quantities of mucus as to simulate acute pulmonary oedema (53).

Should a patient under thiopentone anaesthesia suffer from anoxia due to any cause such as limited lung expansion from pleural effusion, or respiratory obstruction due to swellings in the neck, or within the pharynx or chronic lung diseases such as tuberculosis, tumour or empyema, then that anoxia will be dangerously aggravated by the anoxia caused by the depression of the medullary and

hypothalamic centres due to the drug (31). If the resulting anoxia is sufficiently great, the patient may be in great danger.

Similarly if the patient vomits during induction the resulting laryngeal spasm from the hyperactive laryngeal reflex may cause severe respiratory obstruction. This may precipitate cardiac failure or cerebral haemorrhage in patients liable to those conditions.

The cardiovascular system may be adversely affected during intravenous anaesthesia. The vasomotor centre is depressed with each injection causing a temporary fall in the blood pressure, most marked in patients suffering from hypertension and arteriosclerosis. This fall may amount to as much as forty millimetres of mercury. The decrease in blood pressure is accompanied by a sustained increase in the diameter of the arterioles and capillaries, predisposing to increased oozing at the operation site (50,33,42).

The pulse-rate is increased, and, although the cardiac rhythm is not usually disturbed if the patient is maintained free of oxygen lack, it occurs in the experimental animal if premedicated with morphine and in the human subject if elderly and arteriosclerotic with ischaemic myocardial degeneration (50,33).

Intravenous anaesthesia is not free from risk to the liver. Toxic hepatitis has occurred. In prolonged anaesthesia the liver function is severely taxed and there is an interruption of the normal glycogenic-glycogenolytic activity (54,55).

Although this form of anaesthesia is generally advocated in the treatment of convulsions occurring under other forms of anaesthesia or analgesia, cases of convulsions occurring during thiopentone anaesthesia have been reported (56,57).

The position of the patient under anaesthesia is important and it has been observed that respiratory difficulty is common in the prone position and that these patients are more liable to vomit (49).

This form of anaesthesia is especially indicated for minor surgical operations. Many such cases are treated as "out-patients", the patient being allowed to return to his home after his recovery. This practice is not without risk to the unaccompanied patient as he may fall asleep again about one hour after waking from the anaesthesia (58).

The actual injection of the drug may provoke undesirable local reactions. The vein into which the injection is made may become thrombosed. The frequency of this complication depends on the strength of the solution of

the drug. It occurs in one case in a thousand with five per cent solutions and one in three thousand with a two and a half per cent solution. If the ten per cent solution is used, then the complication is frequent. No case of embolus arising in the thrombosed vein has been reported (50,42,59). If the injection should be made into an artery, a definite risk, the patient complains at once of a burning pain shooting down the arm, which flushes scarlet and urticarial wheals may appear. Gangrene of the fingers has been recorded as a complication of intra-arterial injection. In one series of eight such cases, four developed gangrene. Another series quoted four cases in twenty-five thousand with no cases of gangrene, although in one in which an overdose was given into the artery, the excessive dose was masked by the injection not having been made into a vein, and death resulted (58,60,42). If the injection should be made extravascularly, the skin may slough, the solution causing tissue necrosis by virtue of its alkalinity, the P_H of the solutions normally used being in the region of nine or ten.

A peripheral nerve may be injured by the needle through which the injection is made into the vein (42).

There is thus ample evidence to support the view that intravenous anaesthesia must not be embarked upon

without consideration. The simplicity of administration is confined solely to the simplicity of the apparatus required. The patient may lose his hand through a faulty technique, or his life through faulty assessment of his condition by the anaesthetist. In deaths directly attributable to anaesthesia in the principal hospitals of South Africa during 1931-1935, Evipan ranks next to chloroform as the most dangerous anaesthetic with a mortality rate of 0.14 per cent. In one series of six thousand, five hundred cases there were eight deaths (61).

In few types of anaesthesia must the decision for its employment be more carefully considered than in spinal analgesia. The anaesthetist may be tempted to use the method in the mistaken idea that once the 'block' has been administered he is free to anaesthetise another case. Some surgeons may even administer the analgesia themselves and then proceed with the operation leaving the supervision of the patient to a nurse. Since the introduction of the relaxant drugs, the indications for the technique have diminished. It is still indicated where muscle relaxation is specially required where it is undesirable to have the patient asleep and where regional analgesia is contra-indicated. The contra-indications of the technique vary with the degree with which the anaesthetist favours the

method, but those listed by Hewer may be taken as reasonable (3). He states that the method should not be used in patients suffering from severe shock, advanced myocardial degeneration and an abnormally low blood pressure. Hypertension, only if accompanied by arteriosclerosis, contraindicates the technique. It should not be used in patients with disease of the central nervous system. Respiratory obstruction from any cause is a bar to high spinal block. Extensive lesions of the vertebral column, local sepsis and blood-stream infections also make the method inadvisable. It should not be used in young children or nervous adults. In his opinion the technique should not be employed in cases of intestinal obstruction due to mechanical causes.

Of great importance is the area involved in the analgesia and the 'height' of the 'block'. It has been shown that sacral and lumbar block is relatively safe, even to the handicapped patient. But where the block extends into the thoracic region the result is different. It may not be harmful to the robust patient, but it may prove so to the handicapped. There is little evidence that spinal analgesia carries a higher mortality in 'good risk' patients, but it does so in the 'poor risk' (62).

It would be reasonable therefore to restrict the use of spinal analgesia, with the possible exception of 'saddle-block', to the fit subject.

The danger of sepsis is not to be ignored: one writer describes eleven cases of meningitis following ninety-six 'spinals', one of the cases proving fatal (63). The infection may come from the anaesthetist's hands, from the skin in the lumbar region of the patient or from imperfectly sterilised syringes and needles. The needles and syringes may be contaminated by infection from imperfectly sterilised towels, from the so-called sterile water or sterile distilled water, from the local anaesthetic solution or from the spinal drug. Repeated attempts at lumbar puncture may result in the formation of haematomata which may relay infection to the meninges (64). It has been suggested that irritation of the meninges by the anaesthetic drug allows organisms to gain a hold (65), but the supposition that spinal anaesthetics in the concentrations used clinically will produce inflammatory changes in the meninges is unsupported by experimental evidence (66).

The clinical picture may be headache with meningismus, or at the other extreme, acute septic meningitis. The illness, however, may be chronic in character, with a tendency to relapse due to the formation of adhesions containing infected cerebro-spinal fluid which is liberated from time to time. These adhesions may eventually produce spinal block or even hydrocephalus. One case of

sub-arachnoid abcess has been described in a patient with a septic area in the lumbar skin who was given a spinal analgesia by the fractional technique of Lemmon (65,67).

Cranial nerve paralysis is a not unknown complication, palsies affecting all the nerves but the first, ninth and tenth have been described, with the sixth most commonly affected. Squint develops in the third day and usually disappears within a few weeks, although permanent diplopia has been recorded (3,68). The exact cause of the condition is unknown. It has been suggested that increase or decrease in the pressure of the cerebro-spinal fluid may be a factor. Alterations in the blood pressure during the spinal block have been incriminated, and also, a direct toxic effect from the spinal drug. Inflammatory changes, either a low-grade meningitis or meningismus, and pre-existing diseases, such as syphilis, have also been considered as possible aetiological factors. Increased intracranial pressure with straining, forcing the drug into the medullary area, has also been proposed as a factor (69,33).

Various theories have been advanced for the peculiar susceptibility of the sixth cranial nerve, that the long course of the nerve exposes it to injury, that it may be damaged between the pons and the occipital bone where it is crossed by the anterior inferior cerebellar artery, and that

the nerve may be injured as it bends over the angular apex of the petrous bone. A recent suggestion is "that the highest and most recently acquired faculties and attributes of the human being are the first to be lost or disturbed under stress. The varying stability of the binocular vision function makes it possible that any cerebral pathology, even slight in degree, would tend to cause a breakdown of the binocular system with resulting diplopia" (70).

Although paralysis of the sixth cranial nerve may inconvenience the patient for a short period, the occurrence of spinal nerve palsy, or the entire "Cauda Equina Syndrome" is a tragedy. In the latter, the patient fails to gain the use of his legs after the analgesia passes off. He develops loss of motor and sensory functions in the lumbo-sacral distribution, and loss of bladder and bowel function (71,72). Minor degrees of upset of nerve function in the lumbo-sacral distribution are more common, for example, foot-drop, bilateral weakness of leg muscles, paraesthesiae in the leg, sciatic nerve pain, retention of urine, incontinence or retention of faeces, sensory loss in the sacral region, and loss of sexual function (73,42,74). Retention of urine is usually transient and yields to drugs of the acetyl choline group. But in some cases the retention is severe and permanent, leaving the patient with the risk of

an ascending infection of his urinary tract. The lesion is damage to the sacral nerve roots with consequent spasticity of the internal sphincter of the bladder (75).

There are two schools of thought in the aetiology of this condition. There are those who claim that it is due to the spinal agent. In support of this view they state that the nerves of animals exposed to the highest concentration of the drug are the most affected, that trauma is unlikely, and that the rapid onset of the symptoms and the failure to cultivate organisms from the cerebro-spinal fluid seem to preclude infection (71). It may be that the patient is unduly sensitive to the drug, or that the anatomical configurations prevent the prompt distribution and dilution of the injected drug and that such concentrations of the drug may give rise to neurological sequelae (67). Other anaesthetists claim that these sequelae are due to trauma, as one may develop weakness of the leg after lumbar-puncture in which no drug has been injected, but trauma caused to a nerve (42).

Headache is probably the most common complication of spinal analgesia, but its origin is still unknown. The incidence in one series of cases was highest when a concentrated solution of the drug was used as in block of the sacral segments and it is suggested that concentrated

solutions, hyperbaric in character may predispose to headache (62). A very high incidence is recorded after continuous spinal analgesia with the catheter technique (76). The same causal factor may be involved where headache is produced by excessive attempts at lumbar puncture with no drug injected. This also produces a 'spinal' headache and leucocytosis, but no pyrexia and the symptoms are attributed to meningeal irritation from trauma and sub-arachnoid bleeding. These two theories are compatible with the less definite suggestion that the headache is due to disturbance of the intra-cranial pressure. In other cases the headache is associated with nuchal rigidity, a 'septic' type of temperature, and a positive Kernig sign. Lumbar puncture reveals blood in the cerebro-spinal fluid, but cultures of the fluid are sterile (42). Other factors, the importance of which is difficult to assess, have been suggested. There is a greater incidence in women and patients under the age of forty years. The complication is encountered twice as frequently in parturient as non-parturient women. It has been suggested that the headache is gastro-intestinal in origin (77,78,79,80).

Headaches most commonly come on between the first and fifth day after the operation. Three types have been described. There is a rare type which is defined as

'splitting'. A more common variety is a band-like oppressive ache round the head which is aggravated by movement and may be associated with vertigo (77). The headache, in a few cases, may be associated with nuchal rigidity, pyrexia, a positive Kerning sign and lumbar puncture reveals blood in the cerebro-spinal fluid. Some of these may be associated with diplopia, nausea, tinnitus, hemianopia, and difficulty in accommodation when reading. The associated diplopia may last two months or more (42).

The headache most commonly lasts about twelve days, but two cases have been recorded in which the duration was two years (42). In a further series of two hundred and thirty-one thousand, one hundred and seventy four this complication occurred in two cases and lasted more than two weeks (73). In contrast, in a series of five hundred cases, thirty per cent developed headache and in 13.4 per cent it was severe (81).

Spinal analgesia may be followed by unusual effects. After a 'spinal' headache has worn off the patient may complain of hyperaesthesia of the scalp, which may be due to irritation of the trigeminal nerve. In other cases the patient may be left with hyperaesthesia or paraesthesiae in the leg (42). The analgesia may persist in some parts of the body long after it has disappeared in others (82).

Occasionally in the post-operative period the function of the nerve supply to the arm is upset, although there is no such upset in the legs. Irritation of the spinal cord as the analgesia wears off may lead to clonic movements in the legs (42).

While under the influence of a spinal analgesia the patient may lapse into coma. This may resemble syncope with rapid recovery of consciousness. The patient may remain unconscious for a period up to twenty-four or thirty-six hours but recover without sequelae. Other cases may recover consciousness and live but show signs of permanent brain damage. In the most severe grade of coma, the patient dies on the table or remains comatose in the post operative period and dies in a few days. Coma during spinal analgesia is indicative of severe anoxia of the cerebral centres and if untreated for a few minutes, death ensues (83).

Changes take place in the cerebro-spinal fluid after spinal analgesia. The protein content increases slightly during the first three weeks, the greater increase being in the albumin content which is almost doubled by the eighteenth day. Thereafter there is a significant rise in the protein indicating a mild and transient irritation of the meninges. In some cases, there is still evidence of this rise ten years after the analgesia. There is a slight increase in the polymorpho-nuclear leucocytes and the

sugar content (84,85,33).

It has been shown that the concentration of the drugs used for sub-arachnoid analgesia is a large factor in the production of neurological injuries (86,87). If a toxic dose of a 'spinal' drug is injected into the subarachnoid space of a dog, ganglion cells from the site of injection show chromatolysis, dissolution of the Nissl substance, and swelling of the cell membrane. The arachnoid becomes thickened and infiltrated with plasma cells (33). Cases of ascending myelitis have been described. In a fatal case after "Spinocaine", where the whole substance of the cord in the lumbar region was found to be necrotic, there was no evidence of direct trauma to the cord, nor of the introduction of infection at the time of the lumbar puncture (88).

The effect of spinal analgesia on the respiratory system may be discussed as the effect on respiration during the analgesia, and the influence of this technique on post-operative complications.

During spinal analgesia the respiratory centre may be depressed by circulatory depression (33). There is a slight increase in the rate of respiration, and the inter-costal muscles may be paralysed. The minute-volume exchange in the lungs is decreased. Respiration is normally shallow

and regular but must be watched for the onset of respiratory paralysis. If the patient is heavily premedicated with morphine, the respiratory depression so produced, in addition to that due to the anaesthesia, is liable to produce respiratory arrest (33,3,82).

Respiratory failure may occur from anoxia of the respiratory centre due to the 'spinal' ascending so high that all the intercostal muscles, and, less likely, the diaphragm, are paralysed, or from anoxia due to the failure of the circulation (3,33,39). Diffusion of the drug into the medulla and a direct toxic effect from absorption of the drug have also been incriminated (33,67).

Death occurring immediately in spinal anaesthesia is due to respiratory failure, but when delayed, it is associated with permanent motor paralysis due to cerebral anoxia caused by respiratory and circulatory failure (39,42).

Respiratory complications occurring post-operatively are in the main those occurring after general anaesthesia, although lobar pneumonia is relatively uncommon. There is a significant increase in respiratory infection with anaesthesia extending into the upper dorsal segments (62,79, 80).

The outstanding effect of subarachnoid block on the circulatory system is the fall in the systolic blood

pressure. Many causes of this hypotension have been suggested, among them, the paralysis of the vasoconstrictor fibres in the anterior nerve roots, paralysis of the suprarenal glands, absorption of the drug causing the fall in blood pressure from depression of the vasomotor centre, the shallow respiration of 'high spinal' diminishing the pump-effect of respiration, haemorrhage, and reflexes from traction on the mesentery (89,90,91,3,33,83). This hypotension is less marked in 'continuous spinal' and may resemble that occurring after prolonged general anaesthesia (92). The most pronounced falls occur in high anaesthesia and in patients with hypertension or hypotension (33).

This hypotension may result in severe anoxia of the cerebral centres. Normally the body compensates for blood loss or diminished blood volume by compensatory vasoconstriction in less vital organs and the blood pressure is maintained despite a decreased cardiac output. In patients under 'spinal', the power of vasoconstriction is lost in a large part of the body and a comparatively small loss of blood may be fatal (83,39).

The diastolic blood pressure, on the other hand, falls only slightly due to the decreased cardiac output and decreased stroke-volume from failure of the venous circulation (33).

The effect on the heart is one of depression.

Bradycardia is prominent, caused by depression of the cardio-accelerator nerves. The cardiac output is decreased by ten per cent or more and the stroke-volume is also decreased.

The circulation time is increased by one hundred per cent (33).

While the patient is on the operating table nausea and vomiting may be troublesome. They are not uncommon even if there is no surgical operation and the 'block' has been carried out for diagnostic purposes. Any great, sudden alteration in the blood pressure may be a factor in their production. In some cases they may be due to hypoxia (42). During the operation undue traction on the stomach may cause impulses to be referred via the vagi to the vomiting centre with resulting emesis. Direct stimulation of the vomiting centre has been described as causing these effects (3,93,94). It has been claimed that the centre may be stimulated by the drug diffusing into the cisterna (33).

During the anaesthesia the patient may complain of feeling stifled or suffocated due to the head-down position in the hot operating theatre. He may also complain of feeling parched or faint for the same reason and the effect of the drugs used in premedication (3).

Nausea and vomiting follow operations under sub-arachnoid block more frequently than is supposed. In one

series of five hundred cases, vomiting occurred in over one third of the patients (95).

Abdominal distension has been observed after spinal anaesthesia even where no abdominal operation has been performed. However, if pyrexia accompanies the distension, it is doubtful whether the cause is not something other than the analgesia. In some cases, complaint has been made of muscular cramps in the abdomen when the 'spinal' was wearing off, but the condition is not encountered frequently. Ileus due to the operation is of short duration. If it persists for more than two days it is probable that the anaesthesia was not the cause, or the sole cause (42).

The action of subarachnoid block on the pregnant uterus and on pregnant women is highly controversial. It is said that it interferes with the normal sequence of contraction and relaxation of the uterus. During the first stage of labour contraction of the lower segment it hampers further dilatation, but if dilatation is complete, the hypertonus will speed up delivery (39). Macintosh, in giving his opinion on the use of this form of anaesthesia for Caesarian Section operations, observes that spinal analgesia for moderately high abdominal operations causes paralysis of the nerve supply to the lower intercostal muscles, so that the lower and mobile part of the chest-wall is

temporarily put out of action. A full-term uterus splints the diaphragm, and the effect of this is increased by the Trendelenburg position and surgical packs. "All that remains is for the surgeon's assistant to lean on the patient's chest to complete the insult to her respiratory mechanism." Even without this last, there is a probability of collapse from anoxia if the patient is breathing air only (96).

The procedure of lumbar puncture is not without risk to the patient's vertebral column. Damage caused by the needle may cause soreness of the back which may persist for a considerable time (42). Cases have been reported of persistent pain in the back and radiological changes in one intervertebral disc and arthritis limited to one intervertebral joint (97). The nucleus pulposus may become infected after a lumbar puncture (98), and so may the skin over the sacrum with resulting cellulitis and more or less extensive gangrene (99).

There is the danger that a patient who has been given one of the long-acting, hyperbaric spinal drugs may be put in the Trendelenburg position before the drug has become fixed, or the wrong solution may be injected intrathecally. Breakage of the lumbar-puncture needle in the patient's back is not a rare occurrence (42). Cases of rigors and hyperpyrexia are sometimes seen. It may be that boiling the syringes and needles in alkaline or tap-water

may have been the cause (100).

The mortality, as with any form of anaesthesia, is variable. In one series it was as high as one in two hundred, another writer quotes one in four hundred, and a third series, thirty deaths in thirty-three thousand, eight hundred and eleven (101,102,103).

The technique of caudal block, which has been greatly publicised in America for analgesia in labour, is not without serious complications to the mother.

The principal complication affecting the circulatory system, as in spinal analgesia, is hypotension which occurs secondarily to the involvement of the lumbar sympathetic nervous system (104,105). This hypotension may be associated with severe headache, faintness and dizziness (106).

Continuous caudal analgesia seriously interferes with normal labour, which may be prolonged from the administration of the block before labour is sufficiently established. The uterine contractions become less powerful and the patient is unable to make use of her abdominal muscles. The incidence of persistent occipito-posterior positions is increased to the extent of one hundred per cent. The number of deliveries, in which operative measures are required, is increased (107,105).

It is not without risk to the child. Primary intra-uterine foetal anoxia may occur from the maternal hypotension

and occasionally the foetus has developed sensitivity through the placenta to the local anaesthetic solution (105).

The central nervous system of the mother is liable to complications. Severe headache, pounding in character, and frontal in distribution, is quite common (106,108). Meningitis may occur due to infection, and the sub-arachnoid space may be entered by mistake (108,109).

The respiratory system may be involved if the block is carried too high, as may occur when it is used for Caesarian section, with the paralysis of the intercostal muscles (108).

Nausea and vomiting are frequent both before and after delivery of the child (108).

The insertion and fixation of a needle or catheter into the caudal canal is not without danger. The needle or catheter may break inside the canal. The skin over the hiatus is liable to infection. Post-partum pain in the sacral region is very common. Chilly sensations are complained of by between two and three per cent of cases. When episistomy is performed, the wound tends to bleed more than is usual (108).

The technique is not suitable for all cases, its strongest advocates admit that it is suitable for only five per cent of parturient women, and the anaesthetist or

obstetrician requires special training for its performance (110).

Di-vinyl ether has been described as occupying a place between chloroform and di-ethyl ether. It has a lower toxicity than the former and is more powerful than the latter. The depth of anaesthesia is difficult to estimate and signs such as eye-movements are unreliable. "Vinesthene" has caused burning of the patient's face. Although it is generally believed to have little or no effect on the cardiovascular system, tachycardia has been described with Vinesthene Anaesthetic Mixture (121,33,112,113).

Convulsions are apparently more frequent than with di-ethyl ether. Most of the cases occurred in children under the age of five years when a Goldman Inhaler has been in use, although one case occurred with Vinesthene Anaesthetic Mixture in a "circle-absorber", and two have been described in adults when the induction of the anaesthesia was by intravenous thiopentone. In one series of cases aberrant twitches occurred in all the patients and it is suggested that these were due to stimulation of the central nervous system below the level of the corpora quadrigemina (114,115,116,117,118).

The ill effects on the liver are the most serious complications of di-vinyl ether anaesthesia. Although liver function, in one series of experiments, was not

changed according to the dye-test, central necrosis and decreased liver function occurred with anoxia. Like chloroform this drug may cause liver necrosis, which is largely preventable by administering the anaesthetic with a high concentration of oxygen. A diet rich in carbohydrate is an even better prophylaxis. Hepatorenal syndrome has been described as a complication of Vinesthene anaesthesia (119, 33, 120, 111).

Trichlorethylene, or "Trilene" is the last of the new volatile anaesthetic agents. Induction of anaesthesia with the combination of nitrous oxide, oxygen and 'Trilene' is smooth, but prolonged. Its effect on respiration causes difficulties in evaluating the depth of anaesthesia (122).

Cranial nerve palsies have been described in patients anaesthetised by apparatus in which 'Trilene' has been, or is being used with soda-lime in closed-circuit. The palsies are disabling and may be permanent, involvement of the fifth, seventh, third, fourth, sixth, tenth and twelfth cranial nerves have occurred. Labial herpes have been described as accompanying the cranial nerve paralysis. All degrees of damage have been reported, from simple trigeminal nerve paralysis to encephalitis. The cerebro-spinal fluid in the latter cases showed a small increase in polymorphonuclear leucocyte cells and protein content. Autopsy in a fatal

case of encephalitis revealed oedema of the brain and brain-stem, with occasional localised peri-vascular collections of lymphocytes. The palsies and encephalitis are due to the inhalation of di-chloroacetylene, a toxic product formed by the inter-action of soda-lime and trichlorethylene. This reaction is speeded by the heat generated in the soda-lime (123,124,125,126).

Cases of convulsions have been recorded. These began in the legs and became generalised. The temperature of these patients remained normal and spontaneous recoveries occurred in all cases. Twitchings were observed by Hewer, but these ceased when the oxygen concentration in the anaesthetic mixture was increased, and he therefore suggested that they were due to anoxia (127,128,129,130,132).

An increase in the rate of respiration is common especially in the absence of premedication. This may be due to the action of the drug in the pulmonary stretch receptors, the sensitivity of the Hering-Breuer reflex being increased. Culbert, however, suggests that when the concentration of 'Trilene' in the blood and tissues reaches a certain level, a varying degree of histotoxic anoxia ensues leading to the rapid, shallow respiration of oxygen lack due to anoxia of the cells of the respiratory centre (131,122,132). If an attempt is made to deepen the anaesthesia below the upper

half of the second plane of the third stage, the breathing may stop suddenly or tachypnoea develops (124,131). The lung bases become filled with the heavy and relatively non-volatile vapour of trichlorethylene, thus reducing the available lung capacity and leading to anoxic anoxaemia (133).

Trichlorethylene contains three chlorine atoms, therefore upset of cardiac rhythm is to be expected. The pulse in eighty per cent of cases shows a change after ten minutes of anaesthesia. Bradycardia is more common than tachycardia. The relationship between the bradycardia and the increasing respiratory rate suggests that part of the action on the cardio-vascular system may be central in the central nervous system (134,131,122).

The blood pressure is raised between ten and thirty millimetres of mercury and when the change is made from the sequence nitrous oxide, oxygen, 'Trilene' to nitrous oxide, oxygen, ether, one writer reported that the blood pressure rose between five and eighty-eight millimetres of mercury (131).

Undesirable effects may be produced on cardiac automaticity. On the whole, the arrhythmias occur early in the anaesthesia, in about five or six per cent of cases, and generally take the form of extrasystoles. One common sequence of events is a gross irregular irregularity which

lasts some minutes, followed by pulsus bigeminus of about five minutes' duration, which reverts to normal rhythm. In contrast it has been recorded that arrhythmias occurred in sixteen per cent of cases, but none during the first ten minutes of anaesthesia (135,124,136,131). The sudden development of auricular fibrillation under 'Trilene' anaesthesia has been described (134,137).

Barnes and Ives have studied the electrocardiographic changes occurring under trichlorethylene anaesthesia. They found that arrhythmias were frequent and could be divided into groups. The first occurred early, during the first ten or twelve minutes, often during induction, were transient and not significant. They suggested that they might be due to an increase in vagal tone. The second group occurred later, in the first or upper second plane of the third stage of anaesthesia. They found that ectopic foci initiating premature contractions develop sometimes in the auricles, but more often in the ventricles. At first, ectopic beats occur haphazardly and from a single focus, but as anaesthesia proceeds, they tend to give way to alternating ventricular premature contractions causing pulsus bigeminus. They may be followed in about fifteen per cent by multiple ventricular contractions arising from several different foci and a few of these progress to multiple

ventricular tachycardia, giving a pulse indistinguishable from auricular fibrillation.

Both types of changes may occur in the same case. Ventricular premature contractions have been noted during induction. No significant changes in the R-T interval or T waves, and no late changes, for which the anaesthetic could be held responsible, have been seen in electrocardiogrammes taken at the end of operations or in those recorded several days later.

Multifocal ventricular tachycardia has been described in ten per cent of one series. This is only seen with chloroform, cyclopropane and 'Trilene'. If this occurs when the heart rate is rapid, it is much more likely to be associated with myocardial damage. It is potentially dangerous, as adrenalin has the power of converting this rhythm into ventricular fibrillation. In industry, sudden death has occurred in workers with pure trichlorethylene and at autopsy no significant cause for death was found (138). Death has been recorded during 'Trilene' anaesthesia and been attributed to 'vagal inhibition' (139).

Hepatic impairment is caused by 'Trilene' anaesthesia in the majority of patients subjected to it. This is slight in degree and is less than that due to di-ethyl ether. Fatal cases of liver necrosis, however, have been recorded (140,141,142).

Recent experimental work in animals has shown that 'Trilene' may not be as safe an analgesic agent in obstetrics as was first supposed. In sheep, trichlorethylene appears in the foetal circulation almost immediately after it is administered to the mother. The concentration in the foetal circulation becomes higher than in the maternal arterial circulation. In goats, the drug appeared in the foetal circulation equally quickly, but in this species the higher concentration in the foetal blood did not take place (143). *(more for goats)*

Other undesirable side-effects of 'Trilene' anaesthesia are the development of giant urticaria and generalised sweating (131).

Trichlorethylene is a good example of further investigation proving that the drug is not as safe as originally supposed. Several deaths occurred before it was realised that the drug should not be used in closed-circuit. The risk of the cessation of respiration at a relatively high level of anaesthesia makes the drug relatively unsafe in unskilled hands. The recent experimental work on blood concentrations in pregnant animals shows that the pharmacology of the drug is not yet fully understood.

Although the introduction of Curare into anaesthetic practice has been of great benefit to the patient,

surgeon and anaesthetist, its use is not without danger and difficulties.

The effect on the blood pressure may be to cause a rise or a fall. A slight rise may occur which may be due to a quixotic action on the autonomic nervous system or to the inadequate ventilation of the lungs with the subsequent accumulation of carbon dioxide in the blood (144). This hypertension may increase any tendency to haemorrhage (145).

An immediate and transient fall in the blood pressure may occur following the rapid injection of a large dose of curare. This may be due to a loss of vasomotor tone caused by interference with the transmission between preganglionic and postganglionic fibres of the sympathetic nervous system, or merely to the widespread and complete peripheral muscle relaxation with resulting lack of tonal aid to the venous return (144,146,147).

Death has been known to occur from circulatory failure due to the curare and an effect on the heart has been postulated (148,149).

The danger in the use of curare is the paralysis of the muscles of respiration. This paralysis may be complete, or, when present to a lesser extent, result in the dangers of incomplete pulmonary ventilation with oxygen lack and the building up of carbon dioxide in the blood. This depression

is increased with the patient in the Trendelenburg position because the weight of the liver and abdominal viscera press on the partially paralysed diaphragm (150,145).

In certain cases, the injection of curare may result in bronchospasm, which may be due to a histamine-like action by the drug (151).

Hiccups, which may prove embarrassing during abdominal and thoracic surgery, may occur in about five per cent of cases (152).

In doses sufficient to produce paresis of the intercostal muscles, curare causes a reduction in the tone and peristalsis of the intestine and the dilatation of the bowel may inconvenience the surgeon in abdominal surgery (153,147).

The relaxation of the cardia produced by curare may be accompanied by regurgitation of stomach contents in the unprepared patient, especially if that organ be chronically distended, or pressed on by the surgeon, with the risk of an aspiration pneumonia (154,145,155).

Bloody diarrhoea has been produced in dogs with large doses of the drug and it has been suggested that curare in large doses is contra-indicated in diseases of the intestinal tract (156).

Experimental work on patients receiving large doses

of curare has shown that the liver function may be impaired (146).

The margin between the effective and over-doses of the drug is small, and cases of idiosyncrasy have been described (144,157).

With curare, the anaesthetist is deprived of the ordinary, classical signs for estimating the depth of anaesthesia, and consequently, while aiding the anaesthetist in the production of muscular relaxation, the maintenance of anaesthesia is made more difficult (144).

Relaxation with "Myanesin" is less predictable than with curare. Although it has been stated that respiratory depression occurs as commonly as with curare, the consensus of opinion is that "Myanesin" has little effect on respiration. But in a few cases there is a temporary depression of respiration following the injection of the drug, and the respiratory depression caused by a given dose of thiopentone appears to be increased when mixed with "Myanesin" (158,159, 160).

The principal complication affecting the cardiovascular system is thrombophlebitis of the vein used for the injection. Local venous thrombosis has been recorded as occurring in five per cent of cases, inflammatory reaction in fourteen per cent, and in a third series, thrombophlebitis

occurred in three per cent of cases (158,161,160).

Surgical shock has been produced by the administration of "Myanesin" to the unanaesthetised patient (158).

Many cases have been described of transient haemoglobinuria and albuminuria. These usually disappear spontaneously within forty-eight hours. But, more serious effects on the kidney may result, as death has occurred from renal anoxia (158,161,162).

"Myanesin" appears to cause a definite haemolysis and increase the fragility of the erythrocytes, although in some cases the haemolysis does not evidence itself as haemoglobinuria (163).

An authoritative American comment on "Myanesin" states that: "the complicating factors of venous irritation and haemoglobinuria are sufficiently serious to bring us to the conclusion that "Myanesin" in its present form is not a satisfactory substitute for curare." (158).

The prolonged respiratory depression as a result of the administration of the muscle-relaxant drugs may predispose to post-operative respiratory complications. The reduced expansion of the lungs during the anaesthesia may result in areas of lobular atelectasis or even collapse of a lobe of a lung. The impaired pulmonary ventilation may result in the biochemical changes associated with carbon dioxide retention in the blood. Pinkerton is of the

opinion that the maintenance of muscle tone will prevent the incidence of the post-operative shock, and Hewer includes surgical shock among the causes of post-operative pulmonary complications. It is reasonable to suppose that prolonged reduction in muscle tone may be a factor in the causation of respiratory complications (164,3).

Explosions form a relatively frequent and often fatal danger in modern anaesthesia. At least one hundred cases of burns of eyebrows, lips, and pharynx occur in Great Britain every year from ether explosions (165). In a recent American series, compiled from the records of eighty seven anaesthetists, the explosion rates for ether, ethylene, and cyclopropane were all in the neighbourhood of two to four per hundred thousand anaesthesias and the explosion mortality was one in one million, one hundred and fifty thousand cases (166).

Anaesthetic explosions may be discussed as anaesthetic explosions proper, oxygen fires, explosions in body cavities and bursting of reducing valves.

Anaesthetic explosions occur because most drugs used in inhalational anaesthesia burn readily. The anaesthetic drugs in use in Britain which are explosive are diethyl ether, di-vinyl ether, cyclopropane, and ethyl chloride. Halogens tend to decrease inflammability, chloroform is non-

explosive, and trichlorethylene almost so. In the case of the former group, the concentration of each in the inspired mixture required for anaesthesia falls within their explosive limits. Dilution of the anaesthetic agent with inert gases, such as carbon dioxide as nitrogen, lowers the limits of inflammability (33).

The practice of anaesthetising deeply with ether for laryngoscopy, bronchoscopy and aesophagoscopy, the source of the anaesthetic being removed when the patient is at an adequate depth of anaesthesia, raises the question of the time that must pass after the withdrawal of the anaesthetic before the patient's exhalations are no longer liable to ignite or explode. It has been determined that ten or fifteen minutes are sufficient for the reduction of the tension of the inflammable anaesthetic vapour in the exhalations to a sub-inflammable level (25).

In as much as cyclopropane is administered in closed circuit, there is almost never an explosive concentration outside the breathing bag. Divinyl ether and ethyl chloride may be considered to behave in a similar manner to di-ethyl ether (25).

Before the universal use of oxygen in anaesthesia any fires which occurred were usually not fatal, as fatalities depend mainly on the propagation of the flame into the lungs.

Ether in air burns so slowly that the flame is not likely to be propagated into the lungs. But the flame about the mouth and nose will be drawn into the upper air passages or lungs with inspiration. "Open ether" is relatively safe. Samples of air have been taken at a distance of two inches from the mask after the operation has been in progress for an hour and ten ounces of ether used. It was found that the percentage of ether vapour in the samples was too small to ignite (25,167).

The introduction of oxygen increases the dangers of explosion. The limits of oxygen concentration required for combustion with the anaesthetic agent are less than the physiological requirement of the patient. If an explosion occurs when ether and oxygen are being administered, the flame will travel along the tube of the apparatus and blow up the ether bottle. If the ether is administered with oxygen alone or with nitrous oxide and oxygen, the rapidity of propagation of the flame is so great that it will pass down the patient's trachea and burst the lungs (33,167,25).

The ignition temperature of an anaesthetic mixture varies with the mixture and some mixtures require a longer period of contact with the source of ignition to self-sustain combustion than others. The higher the 'flash-point' of an agent, the safer it is. The source of ignition in

anaesthesia may be provided by flames, electricity or static electricity (33). Open flames, such as pipes, cigarettes, gas and spirit lamps, and open fires, are not often the cause of explosions because they are usually evident and therefore avoided (25).

Electric sparks may be caused by stopping and starting an electric motor, and from electric heaters. Although modern X-ray apparatus is said to be spark-proof, it is unwise to use an inflammable vapour while the plant is in operation. The lowest temperature at which a cautery is effective is sufficient to flash the mixture. One of the most recently reported cases of explosion in England occurred when diathermy was used in a case undergoing pneumonectomy under cyclopropane anaesthesia with fatal results. Faulty electrical switches in the operating theatre or imperfect connections in laryngoscopes, bronchoscopes or oesophagoscopes are other electrical sources of ignition (168,25,169,33)

Until recently no explosions due to static sparks had been reported in Britain, but there are now several incidents which seem to have been due to this cause. In dry climates such as is found in certain parts of America there is a definite risk and an elaborate system of earthing has been adopted. The static spark is not obvious. It may originate between the mask and the patient's face, from the

opening and closing of valves in the apparatus, from friction between the inner surfaces of the rubber rebreathing bag, from the friction caused by the passage of dry gases through the apparatus and the hose, from blankets, operating tables, and the removal of adhesive. The modern tendency to use dry flowmetres instead of the old water sight-feed bottle, which provided a certain amount of water vapour for the inside of the anaesthetic machine, constitutes a danger especially if air-conditioning of operating theatres increases (170,171,33,25,167).

The source of ignition causing an explosion may not be determined. In a recent incident a leak between the rebreathing bag and the bagmount was found, but what ignited the escaping gases was not discovered (169).

The effect on the lungs depends on whether an explosion takes place, or whether flame is inhaled. If an explosion takes place the lung is ruptured, if the explosion is less powerful or a flame about the mouth and nose is inhaled, the alveolar membrane is so burned that it cannot maintain its function. In the first instance death occurs at once, but in the latter it may be slightly delayed (25).

If oil or grease comes into contact with highly compressed oxygen, as is used in anaesthesia, a spontaneous fire may start which is difficult to extinguish. This fire

may act as the source of ignition for a true anaesthetic explosion (25,3).

There is the risk of an explosive concentration of such an anaesthetic agent forming in body cavities other than the lungs. After an hour's anaesthesia with ether, the vapour is present in explosive concentrations in body cavities. The same danger is present with cyclopropane, 'Vinesthene' and ethyl chloride. It may happen in an operation such as gastrectomy, where the surgeon makes use of diathermy to excise the stomach and incise the intestinal wall, that an explosive agent has been safely used in a rigid closed circuit with complete safety until the vapour accumulates in the stomach and intestine with disastrous results when the diathermy knife is used (25).

Disintegration of reducing valves, although not true anaesthetic explosions, may have similar though less violent effects. If the main cylinder pressure is turned on suddenly, especially with an outlet valve on the reducing-valve closed, the sudden strain may rupture the valve diaphragm or otherwise cause a leak from the high pressure to the low pressure side with disastrous consequences (3).

The prevention of anaesthetic explosions is simple in the majority of cases. If an anaesthetic is to be administered where there is a potential ignition source, there

is now no reason why a non-explosive agent should not be used. With a relative atmospheric humidity greater than sixty per cent, an explosion due to static electricity is unlikely, if not impossible. With complete rebreathing, the humidity within the apparatus becomes so high that the production of a static spark is impossible under ordinary circumstances. Where atmospheric conditions predispose to the formation of static electricity, an efficient coupling system will provide almost complete safety.

The inhalation of vomitus or foreign bodies is one of the dangers of anaesthesia still to be overcome. The modern tendency to use intravenous anaesthesia and muscle-relaxant drugs has increased this danger in certain cases, for example, the use of thiopentone in dentistry and curare in intestinal obstructions.

The patient may vomit during the induction of anaesthesia, because the induction is difficult or prolonged on account of improper premedication, incorrect selection of anaesthetic agent, or the wrong technique. If the patient comes to theatre with a full stomach, or a pharyngeal airway is inserted too soon, vomiting may occur. In certain cases the use of morphine in premedication may increase the liability of the patient to vomit. Vomiting may also occur during the maintenance of anaesthesia. If the depth of

anaesthesia becomes insufficient, mucus or an artificial airway may initiate emesis (44).

Certain cases are more prone to vomit than others. In patients who are about to have the operation of Caesarian Section there is an increase in the basal metabolic rate, and nervousness which causes hyperactivity of the mucous glands in the respiratory tract. This results in an out-pouring of mucus during induction which seriously interferes with the anaesthesia. Deep anaesthesia is not required and therefore the anaesthesia may become too light, with resulting vomiting, if the stomach is full, as is likely when such cases have to be treated as emergencies. Pre-operative gastric lavage is not a complete answer to the problem. It may give a false sense of security to the anaesthetist as large portions of food may be left in the stomach. These may be vomited and block the larynx or a main bronchus (25).

The anaesthetist may have some warning that vomiting is about to occur during the maintenance of anaesthesia. Its advent is indicated by shallow respiration, pallor, a 'small' pulse, and an active pupil, the last showing that the previous signs are not due to overdosage (31).

Regurgitation of stomach and intestinal contents into the pharynx may occur when the cardia relaxes and the stomach is full. Manipulation of bowel in cases of intestinal obstruction may result in an obvious regurgitation, but

in that condition there is also the risk of a slow drainage of stomach and intestinal contents into the pharynx throughout the operation with partial aspiration into the trachea and lungs (25,44).

The acid gastric contents may irritate the laryngeal mucous membrane and set up severe laryngeal spasm and obstruction. Solid particles inhaled into the respiratory tract may cause acute asphyxia and death, bronchopneumonia, atelectasis of one or more lobes, or peripheral circulatory failure. Asphyxia due to aspiration is a frequent cause of death on the operating table. If the stomach is empty when the patient vomits, there may be no ill effects, but he may injure his tongue or lips in the great activity of the muscles of mastication which may accompany vomiting. Vomiting or regurgitation may occur with little or no evidence at the time that it has taken place, but the patient develops a severe post-operative respiratory complication of the type already described (44,25).

In the surgery of the nose, mouth and throat there is a special risk of aspiration. In tonsillectomy there is always some aspiration into the trachea, the amount of blood aspirated depends on the techniques of operator and anaesthetist. In addition there is the risk of a clot forming in the posterior pharynx which may be aspirated and cause

complete respiratory obstruction (25).

Sponges and packs used in anaesthesia for dental surgery may cause a fatal obstruction in the larynx or trachea. The obstruction may be 'ball-valve', allowing expiration, but not inspiration. Respiratory effort increases as the level of carbon dioxide rises in the blood and the oxygen reserve is consumed. The resultant increased respiratory effort pulls the sponge into the larynx or trachea.

During the insertion of laryngoscopes, airways, or gags, or during extraction, teeth may be broken and aspirated. This does not lead to immediate respiratory difficulty, but it does lead to small or large areas of atelectasis which may progress to a lung abscess.(25)

Intubation is a procedure in everyday use and although apparently easy to perform, is not without risks. It has been suggested that the technique may be of value in operations in which obstruction to the airway may be expected either from the nature of the disease, the surgical technique, the position of the patient, or the presence of fluids in the air-passages. Most operations on the head and neck, except those for toxic goitre, should be carried out under endotracheal anaesthesia. The procedure may be an advantage in most prolonged upper abdominal operations

where shallow respiration is desirable and in which spinal, splanchnic or other local blocks are not employed (3).

In some cases of acute infection of the upper part of the respiratory tract, use of an endotracheal tube is probably not advisable. There is the type of patient who cannot tolerate any trauma to the mucous membranes. When patients have foul lesions in the mouth or on the face, and have difficulty in coughing and expectorating to rid themselves of material from the respiratory passages, the use of an endotracheal tube is likely to increase the incidence of post-operative pneumonia, or if pneumonia would have developed, to increase its severity (172).

Several factors influence the degree of damage to the larynx. On the degree of proficiency in laryngoscopy of the anaesthetist not only depends the damage to the larynx, but also the degree of damage to the other tissues of the upper respiratory tract. The individual anatomical characteristics of the patient, such as a short neck, affect considerably the ease or difficulty of intubation. The degree of relaxation and the rapidity with which the intubation must be attempted differentiate between an easy intubation with the patient at a reasonable depth of anaesthesia and an emergency intubation with the patient lightly anaesthetised. The diameter, shape and consistency of the

endotracheal tube, and the time during which it is in position affect the issue whether or not the patient will suffer any after-effects. The age of the patient is also important. A tube that fits tightly the glottis and trachea of an infant may cause oedema which may prove fatal. Epistaxis or bleeding from the throat of a child or infant is easily produced. Intubation is therefore usually contraindicated for young children, and a tube that can be inserted into a tiny trachea does not have a large enough lumen to provide the easy respiration necessary for an infant (173,42).

In the adult, complications of endotracheal anaesthesia are due to compression of the tube during anaesthesia and trauma in introducing the tube. If the tube becomes compressed or kinked during anaesthesia, the patient will develop acute asphyxia if the compression is complete, but if the obstruction is of a lesser degree, then oxygen lack and carbon dioxide retention will develop. Similar effects result from the presence of mucus in the endotracheal tube (42).

Laryngoscopy in expert hands should be atraumatic, but in the hands of a novice, or where the laryngoscope must be introduced in unfavourable circumstances, considerable damage may be done. The lips and gums may be lacerated. The teeth may be loosened, fractured, or extracted. The

hard and soft palates and the posterior wall of the pharynx may be abraded or lacerated. The tongue may be injured if caught between the teeth and the laryngoscope (173).

Granuloma of the larynx has been reported on several occasions. The cause may be excoriation from the laryngoscope or the endotracheal tube. This becomes secondarily infected and covered with granulations and exudate. The wound edges become everted, healing is delayed and a granuloma is formed. It has been suggested that all the cases reported had factors in common which may have a bearing on the aetiology. They were all intubated for long periods. The almost invariable location of the tumour was near the posterior commissure, very often attached to the vocal process of the arytenoid cartilage. The time of onset of the outstanding symptom, hoarseness, varies from the immediate post-operative period to one month after intubation. In all cases the granulomas were eventually removed (173,175,176,177,178, 179, 180, 181, 182).

A series of five hundred and eighteen cases of endotracheal anaesthesia was analysed for the frequency of laryngeal sequelae. The findings were based solely on post-anaesthetic subjective complaints and not on objective laryngeal examination. One case developed hoarseness without pain, six patients manifested definite laryngitis or

tracheitis, and four, pharyngeal ulcerations (183).

The pathology of these complications may be considered as soft-tissue injuries, cartilaginous and perichondral injuries.

The most common reactions observed in the first twenty four to seventy two hours are a loss of lustre, and a pinkish infiltration of the vocal cords which lack their flat appearance and sharpness. This is attributed to contact with the tube, and depends on the calibre of the tube, its chemical composition, pre-operative preparations, and the duration of the intubation. Recovery is usually prompt after minor injuries to the mucous membrane, but more severe trauma may cause lacerations, tears and avulsions, and these, or minor injuries which have become infected, may lead to laryngeal obstruction and require tracheotomy. Oedema of the larynx is not rare, and benign papillomata of the vocal cords have been described (174, 184, 185, 173).

Injuries to the cartilage and perichondrium tend to be more serious. Repair is poor, and perichondritis, prolonged suppuration and cartilage necrosis may result. The resultant scar tissue formation causes deformity and laryngeal stenosis (173).

Intubation by the nasal route has its own additional complications, most commonly nasal mucosa abrasions and

lacerations, but a tube has been passed into the sub-mucosa of the nasopharynx (173, 186). A case has been reported in which a blood-cast of a bronchus was coughed up. This had been formed following the intermittent epistaxis during a previous nasal intubation (187).

Death may occur from tracheitis due to the anti-septic used for sterilising the tube and from the rupture of an inflatable cuff on the tube (188, 189).

One cannot, therefore, but agree with Hewer that the routine use of endotracheal anaesthesia for every type of operation and patient cannot be too strongly deprecated (3).

Discussion of Mortality and Morbidity Statistics.

A comparison of the statistics of mortality associated with anaesthesia shows that a great increase has occurred in the number of these deaths during the twentieth century. In Scotland the increase has been from 48 such deaths in 1913 to 106 in 1921 and 112 in 1945 (190, 191, 192). In England the increase has been from 91 deaths in 1901 to 296 in 1913, 337 in 1921 and 843 in 1939 (193, 194, 195).

Many factors may be suggested as having influenced this increase. The scope of surgical treatment has been greatly enlarged, the treatment of many diseases previously thought to be purely medical has become surgical, for example in the case of bronchiectasis. Patients who would previously not have been considered fit to survive the necessary surgical operation are now able to benefit from such treatment. There has been an increase in the number of road accidents and similar cases requiring treatment. The use of anaesthesia in childbirth has become more common, more examinations, inductions of labour, and perineal repairs are being performed under anaesthesia, and the operation of Caesarian Section is being carried out with much greater frequency. The use of anaesthesia as an aid in diagnosis

and as a method of treatment has also increased.

In order to compare the scope of surgical treatment of fifty years ago with that of the present day, an examination of the records of the Western Infirmary of Glasgow was carried out and the types of operation performed during the year 1899-1900 are shown in Table 1 (196).

Table 1. Operations performed in the Western Infirmary of Glasgow in the year 1899-1900.

<u>Operation</u>	<u>Number of Cases.</u>
Abdominal Section	108
Caesarian Section	1
Removal of Ovarian Cyst	33
Opening of Abscess	100
Amputations	115
Aspiration or Tapping	8
Gastration	6
Catheterisation	5
Cauterisation	205
Cholecystotomy	8
Circumcision	15
Colotomy	9
Colperrhaphy	1
Curetting	32
Craniectomy	1

Cystotomy	1
Dilatation	5
Emmett's Operation	5
Excisions of bones, joints and varicose veins	77
Excision of tumours	365
Forcible movements	3
Gastro-enterostomy	1
Gastrostomy	4
Herniotomy	10
Radical cure of hernia	78
Hysterectomy	5
Incisions	79
Induction of labour	1
Laminectomy	2
Lithotomy	4
Lithotripsy	1
Mastoidectomy	17
Nephrectomy	1
Nephrorrhaphy	4
Nephrotomy	5
Nerve Stretching	3
Nephrorrhaphy	6
Operation for Hydrocele	13
Osteotomy	13
Perineal Section	6

Perineorrhaphy	19
Plastic Operations	67
Radical Cure of Bubonocoele	2
Reduction of Dislocation	9
Removal of Disease by Scraping or Gouging	122
Removal of foreign body or piece of bone	20
Removal of haemorrhoids	27
Removal of tonsils	114
Removal of tumour	25
Resection of rib	15
Scarification	1
Sequestrectomy	20
Skin grafting	7
Staphylorrhaphy	1
Suturing	7
Tarsectomy	1
Teeth Extraction	12
Tendon Grafting	4
Tenotomy	8
Thyroidectomy	1
Tracheotomy	2
Trephining	20
Turbinectomy	74
Urethrectomy	2
Wiring or Pinning	42
Wounds dressed	48

Of these cases, 25 per cent might be said to have undergone major operations. 105 of the cases died in hospital, that is, the mortality rate was 5 per cent.

The operations performed on the cases investigated for this thesis are classified in Table 2. The cases were selected solely by the criterion of time available for adequate follow-up, and are representative of modern surgery.

Table 2. Operations performed on 500 patients in Stebbill Hospital.

<u>Operation</u>	<u>Number of Cases.</u>
Partial Gastrectomy	21
Hernia, Repair of	
Inguinal and Femoral	25
Ventral	6
Lumbar	1
Gastro-enterostomy	9
Partial Colectomy	8
Thyroidectomy	5
Nephrectomy	6
Cholecystectomy	8
Prostatectomy	
"Punch"	2
Suprapubic	16
Miscellaneous Urological	15

Mastectomy	3
Lumbar Sympathectomy	5
Orthopaedic	
Insection of Smith-Peterson Nail	6
Other	30
Key-hole Operation for	
Hernated Nucleus Pulposus	7
Nar, Nose and Throat Operations	5
Appendicectomy	24
Laparotomy	18
Colostomy	6
Cholecystectomy	6
Closure of Gastric or Duodenal Perforation	9
Perineal Repair	73
Oophorectomy	6
Hysterectomy	15
Sterilisation	12
Caesarian Section and Sterilisation	8
Hysterotomy and Sterilisation	5
Myomectomy	2
Hysterotomy	2
Caesarian Section	12
Operation Cancelled	4
Dilatation and Curettage	49

Salphingo-Oophorectomy	4
Unclassified	61
Diagnostic and Therapeutic Anaesthetics	1.

17% of the operations were regarded as "minors" and 83 per cent major in severity. 37 of the patients died in hospital, the mortality rate being 7.4 per cent. 21 per cent of the patients were classified as poor operative risks, and the pre-operative condition of 3 per cent was serious, that is, almost 25 per cent of the cases were not good surgical risks. 36 per cent of the patients were between the ages of 40 and 60 years, and 17 per cent were older than 61 years.

The number of deaths associated with anaesthesia in England and Wales during this century has increased ninefold. Various causes have been suggested and this series of cases has been compared with the operations performed in one of the large teaching hospitals during the last year of the last century to show that the severity of operations has increased. In many of the operations in the earlier series described as "Removal of tumour", the tumour was described as a sarcoma. Many of these patients must have died shortly after returning to their homes, but they were not included in the numbers of the patients who had died.

It is reasonable to suggest that the results of the

two series compare favourably.

Many pleas have been made that anaesthetists record their failures and accidents as well as their successes, and it would appear that some are responding, with the result that the science of anaesthesia has progressed. The improvement in the reporting of deaths associated with anaesthesia is evidenced by the fact that deaths have been notified where the patient had been given morphine and atropine without any actual anaesthetic having been administered.

Although figures for deaths associated with anaesthesia show such an increase over the past forty years, these figures may, in themselves, be misleading. The number of fatalities depends on the extent to which various agents have been used as well as on the risk attaching to them. The deaths associated with each type of anaesthetic cannot be collated with the number of administrations. It is impossible to say whether, or to what extent, the increase in the number of deaths implies increasing mortality under anaesthesia. The number of administrations is known to be increasing, but it cannot be estimated, and the figures for the number of deaths can only be those associated with, and not those caused by anaesthesia. It is impossible from certification to distinguish between deaths from operation under anaesthesia and deaths due to the anaesthesia itself.

There is no definite legislation regarding the notification of deaths under anaesthesia in England or Scotland. It is stated that an anaesthetic may not necessarily render a death unnatural and therefore notifiable. As there is only a legal duty to report a death which is unnatural to the Coroner or Procurator-Fiscal, there might be differences of opinion as to whether or not a certain death need be notified and whether the fact that the death under anaesthesia be included in the death certificate (197, 198, 199).

A study of statistics of mortality associated with anaesthesia as reported in published articles gives a more exact, but not complete picture, as the total number of cases in each series may be ascertained as well as the number of deaths and consequently comparisons may be made. During the years 1884-1901 one writer quotes 32 deaths from chloroform anaesthesia in 35,612 cases, equal to a percentage of 0.089, and 54 deaths in 20,012 cases of spinal analgesia in 1932, a mortality rate of 0.26 per cent. Two recent figures for deaths under spinal analgesia were 3.3 per cent and 1.04 per cent. Regional analgesia for abdominal surgery and curare in 1946 and 1947 respectively have resulted in mortality rates of 6 per cent and 5.8 per cent (200, 201, 202, 203, 204).

A study of the statistics for morbidity associated with anaesthesia reveals an increase similar to that of mortality. In 1918 Whipple reported that respiratory complications occurred in 2.8 per cent of his cases. In 1946 the respiratory complication rate with curare, nitrous oxide and oxygen was 18 per cent, and with other types of anaesthesia, 19 per cent. The reported incidence of post-operative vomiting has increased from 5 per cent in 1935 to 9.3 per cent for non-abdominal operations, and 19.5 per cent for abdominal operations (205, 206, 207, 208).

This increase may be due to the same factors as suggested for the apparent increase in the mortality rate. In addition there is the very important factor that as anaesthesia has become a specialised branch of medicine so has the interest of the anaesthetists in their patients increased. The result is that minor post-operative complications are diagnosed whose incidence would have been unknown without special inquiry. This is well demonstrated by King who showed that the incidence of post-operative pulmonary complications during the two years that he investigated the problem was double that of the years before and after (209).

In the present investigation 37 (7.4 per cent) patients died following operation. In 2 cases (0.4 per cent) death was due to the effects of anaesthesia. In order to

substantiate the statement that death was due to the anaesthesia in two cases only, a synopsis of the cases which ended fatally is included.

1. Perforated peptic ulcer: female, aged 43 years: operation - repair of perforation: anaesthesia - Pentothal, curare, gas, oxygen and Trilene of 35 minutes' duration: induction and maintenance of anaesthesia were satisfactory and patient had recovered reflexes before leaving theatre: developed severe chest complication and oliguria: died on 3rd day: autopsy revealed sub-phrenic abscess.
2. Inoperable carcinoma of stomach: male, aged 51 years: operation - jejunostomy: anaesthesia - Pentothal, curare, nitrous oxide, oxygen and Trilene of 75 minutes' duration: induction and maintenance of anaesthesia were satisfactory and the patient had recovered reflexes in theatre: died on 3rd day of haematemesis.
3. Intestinal obstruction: male, aged 37 years: operation - division of band of omentum: anaesthesia - spinal - Light Eupercaine 12 cc. (Etherington Wilson) and Pentothal, nitrous oxide, oxygen and Trilene of 55 minutes' duration: induction and maintenance of anaesthesia were satisfactory, and reflexes were recovered in theatre: died on 8th day of existing disease: he had been given food and drink by a mentally deficient patient in the next bed.

4. Pyloric stenosis: male, aged 55 years: operation - gastro-enterostomy: anaesthesia - fractional spinal with 1 per cent procaine by Lemmon technique: case misjudged by surgeon and anaesthetist: induction of anaesthesia followed by severe respiratory depression and hypotension, neither responding to treatment: patient died on second day.

Death due to anaesthesia.

5. Colostomy for closure: male, aged 64 years: operation - laparotomy and closure of colostomy: anaesthesia - Light Nupercaine (Etherington-Wilson Spinal Technique) of 50 minutes' duration: induction and maintenance of anaesthesia were satisfactory: developed paralytic ileus: died on 10th day: autopsy - perforation of colon proximal to colostomy.

6. Carcinoma of gall-bladder: male, aged 47 years: operation - laparotomy: anaesthesia - Light Nupercaine (Howard-Jones Spinal Technique) and nitrous oxide, oxygen and Tri-lene of 45 minutes' duration: induction and maintenance of anaesthesia were satisfactory and the reflexes were recovered in theatre: condition gradually deteriorated: died on 5th day of malignant cachexia.

7. Prostatic enlargement: male, aged 77 years: operation - suprapubic prostatectomy: anaesthesia - spinal Procaine of 40 minutes' duration: induction and maintenance of

anaesthesia satisfactory: condition deteriorated post-operatively: died on 2nd day from cardiac failure, old age and inability to stand up to strain of operation.

8. Multiple urethral strictures and perineal fistula, and stone in urethra: male, aged 56 years: operation - transplantation of ureters: anaesthesia - spinal Nupercaine, of 120 minutes' duration: induction and maintenance of anaesthesia were satisfactory: died on 26th day from uraemia.

9. Prostatic Enlargement: male, aged 62 years: operation - supra-pubic prostatectomy: anaesthesia - light Nupercaine Spinal of 60 minutes' duration: induction of anaesthesia was satisfactory but emoliation of the prostate was followed by sudden and severe fall in the blood pressure: haemorrhage occurred after return to the ward, followed by gradual cardiac failure: died of uraemia on 20th day.

10. Gangrene of leg: female, aged 52 years: operation - mid-thigh amputation: anaesthesia - unilateral spinal with Heavy Nupercaine and nitrous oxide, oxygen and Trilene of 45 minutes' duration: induction of anaesthesia was satisfactory but there was a marked fall in the blood pressure before the operation was begun: reflexes were recovered in theatre: developed rigors 8 hours later, became cyanosed and died: autopsy revealed multiple recent infarcts in brain, liver and kidneys.

11. Small bowel obstruction of 3 days' duration: female aged 50 years: operation - laparotomy and resection of large portion of small bowel: anaesthesia - Pentothal, curare, nitrous oxide, oxygen, Trilene and ether of 90 minutes' duration: induction and maintenance of anaesthesia were satisfactory and reflexes were recovered in theatre: died two days later.
12. Carcinoma of Ovary (Carcinomatosis): female, aged 46 years: operation - removal of ovarian cyst and salpingo-oophorectomy: anaesthesia - Pentothal, curare, nitrous oxide, oxygen and Trilene of 25 minutes' duration: induction of anaesthesia was satisfactory and reflexes were recovered in theatre: developed brady cardia followed by tachycardia during anaesthesia: died on 12th day from malignant cachexia.
13. Popliteal Embolism: female, aged 54 years: operation - exploration of popliteal artery: anaesthesia - sciatic nerve block of 95 minutes' duration: induction and maintenance of anaesthesia were satisfactory: patient died of effects of multiple emboli on 19th day.
14. Pyloric stenosis: male, aged 44 years: operation - gastro-enterostomy: anaesthesia - posterior intercostal nerve block of 100 minutes' duration: induction of anaesthesia was satisfactory but severe fall in blood pressure

took place during maintenance. died on 3rd day from effects of pyloric stenosis.

15. Carcinoma of stomach: female, aged 70 years: operation - gastro-enterostomy: anaesthesia - sub-costal block of 60 minutes' duration: induction and maintenance of anaesthesia were satisfactory: developed broncho-pneumonia and died on 2nd day.

16. Carcinoma of stomach: male, aged 44 years: operation - jejunostomy: anaesthesia - posterior intercostal and anterior splanchnic nerve blocks of 90 minutes' duration: induction of anaesthesia was satisfactory: maintenance was marred by severe fall in blood pressure: died on 3rd day of malignant cachexia.

17. Carcinoma of rectum: female, aged 74 years: operation - perineal excision of rectum: anaesthesia - Light Nupercaine spinal and Pentothal, nitrous oxide, oxygen and Trilene of 110 minutes' duration: induction and maintenance of anaesthesia were satisfactory and reflexes were recovered in theatre: developed broncho-pneumonia and died on 2nd day.

18. Carcinoma of Colon: male, aged 61 years: operation - transverse-sigmoid colectomy: anaesthesia - Pentothal, nitrous oxide, oxygen, Trilene and ether of 95 minutes' duration: induction and maintenance of anaesthesia were satisfactory and reflexes were recovered in theatre:

developed massive pulmonary collapse which did not respond to suction under direct vision with bronchoscope; patient died on 3rd day.

19. Primary Carcinoma of Head of Pancreas with Hepatic metastases: female, aged 63 years: operation - laparotomy: anaesthesia - Pentothal, curare, nitrous oxide, oxygen and ether of 20 minutes' duration: induction of anaesthesia marred by respiratory depression on injection of curare: reflexes were not recovered in theatre: developed intractable vomiting and died on 3rd day.

20. Prostatic hypertrophy: male, aged 80 years: operation supra-pubic prostatectomy: anaesthesia - Pentothal and cyclopropane and oxygen of 25 minutes' duration: induction and maintenance of anaesthesia were satisfactory: reflexes were recovered in theatre: developed severe oliguria and died of uraemia on 3rd day.

21. Carcinoma of Colon: female, aged 50 years: operation - resection of tumour: anaesthesia - Cyclopropane, ether oxygen of 45 minutes' duration: induction of anaesthesia was unsatisfactory owing to difficulty in intubation: maintenance of anaesthesia was satisfactory and the reflexes were recovered in theatre: developed bronchopneumonia: died on 2nd day.

22. Gangrene of leg: male, aged 77 years: operation -

lumbar sympathectomy: anaesthesia - Pentothal, curare, nitrous oxide, oxygen and Trilene of 60 minutes' duration: induction and maintenance of anaesthesia were satisfactory: reflexes were recovered in theatre: became confused, incontinent and died of cardiac failure on 12th day.

23. Strangulated femoral hernia: male, aged 44 years: operation - relief of obstruction and repair of hernia: anaesthesia - Pentothal, curare, nitrous oxide, oxygen and Trilene of 105 minutes' duration: induction and maintenance of anaesthesia satisfactory: recovered reflexes in theatre: died of peritonitis on 11th day.

24. Carcinoma of bile-duct: male, aged 63 years: operation - cholecyst-jejunostomy: anaesthesia - Pentothal, curare, nitrous oxide, oxygen and Trilene of 105 minutes' duration: induction of anaesthesia unsatisfactory due to difficulty in intubation: reflexes were recovered in theatre: was well for first 36 hours, condition then deteriorated and patient died from existing disease and cardiac failure on 3rd day.

25. Pyloric stenosis: female, aged 74 years: operation - partial gastrectomy: anaesthesia - Pentothal, curare, nitrous oxide, oxygen and Trilene of 105 minutes' duration: induction and maintenance of anaesthesia satisfactory, and reflexes were recovered in theatre: developed broncho-

pneumonia and died on 3rd day.

26. Renal tumour: female, aged 73 years: operation - laparotomy and removal of tumour: anaesthesia - Pentothal, nitrous oxide, oxygen and Trilene: induction of anaesthesia was satisfactory, during maintenance, the inferior vena cava was damaged by the surgeon and the patient died one hour after leaving theatre.

27. Intestinal obstruction of 5 days' duration: male, aged 65 years: operation - laparotomy and reduction of internal hernia: anaesthesia - nitrous oxide, oxygen, V.A.M. and curare: induction of anaesthesia was satisfactory but maintenance was unsatisfactory and the reflexes were not recovered in theatre: patient vomited, when peritoneum opened, to such an extent that pack round endotracheal tube was moved and mouth filled with vomitus; pack was removed and mouth and pharynx aspirated; endotracheal tube with inflatable cuff was inserted and anaesthesia and operation continued; bronchoscopy was performed at end of operation and main bronchi aspirated under direct vision: patient vomited continuously on return to ward and died suddenly 3 hours after the end of the operation: death probably was due to the anaesthesia.

28. Ovarian cyst: female, aged 62 years: operation - removal of cyst: anaesthesia - Pentothal, cyclopropane,

oxygen and curare of 40 minutes' duration: induction and maintenance of anaesthesia were satisfactory and the reflexes were recovered in theatre: died one month later from intestinal obstruction.

29. Carcinoma of Rectum: female, aged 67 years: operation - laparotomy: anaesthesia - Pentothal, cyclopropane, oxygen and curare of 30 minutes' duration: induction and maintenance of anaesthesia were satisfactory, and the reflexes were recovered in theatre: died on 15th day: autopsy revealed fatty degeneration of the heart and carcinoma spreading throughout the peritoneal cavity.

30. Malignant ovarian cyst: female, aged 38 years: operation - laparotomy: anaesthesia - Pentothal, cyclopropane, ether and oxygen of 15 minutes' duration: induction and maintenance of anaesthesia were satisfactory: died 3 months later from malignant cachexia.

31. Gangrene of leg: male, aged 75 years: operation - amputation: anaesthesia - Pentothal, nitrous oxide and oxygen of 45 minutes' duration: induction and maintenance of anaesthesia were satisfactory, and the reflexes were recovered in theatre: developed massive pulmonary collapse and died on 10th day.

32. Embolic gangrene of leg: female, aged 39 years: operation - amputation: anaesthesia - Kemithal, nitrous

oxide, oxygen and Trilene of 35 minutes' duration: induction and maintenance of anaesthesia were satisfactory and the reflexes were recovered in theatre: died from cerebral embolism on 11th day.

33. Gangrene of leg: female, aged 73 years: operation - re-amputation: anaesthesia - Pentothal, nitrous oxide, oxygen and Trilene of 15 minutes' duration: induction and maintenance of anaesthesia were satisfactory and the reflexes were recovered in theatre: patient later lapsed into coma and died on the following day.

34. Fracture of neck of femur: female, aged 84 years: operation - manipulation and insertion of Smith-Peterson nail: anaesthesia - Pentothal, nitrous oxide, oxygen and Trilene of 75 minutes' duration: induction and maintenance of anaesthesia were satisfactory and the reflexes were recovered in theatre: patient made satisfactory progress until the 3rd day when she had a fatal haematemesis after an injection of carbachal for retention of urine.

35. Gangrene of foot: male, aged 78 years: operation - amputation: anaesthesia - Pentothal, nitrous oxide, oxygen and Trilene of 35 minutes' duration: induction and maintenance of anaesthesia were satisfactory and the reflexes were recovered in theatre: died from cerebral thrombosis on 10th day.

36. Fracture of neck of femur: female, aged 67 years: operation - manipulation and insertion of Smith-Peterson nail: anaesthesia - nitrous oxide, oxygen, Trilene and ether of 75 minutes' duration: induction and maintenance of anaesthesia were satisfactory: reflexes were not recovered in theatre: suddenly collapsed at end of operation: patient recovered consciousness, but died 7 hours later: autopsy revealed a sub-sternal thyroid gland.

37. Thyrotoxicosis: female, aged 25 years: operation - subtotal thyroidectomy: anaesthesia - Avertin, nitrous oxide and oxygen of 195 minutes' duration: induction of anaesthesia was unsatisfactory because of difficulty of inserting endotracheal tube due to tracheal deviation: maintenance of anaesthesia was satisfactory and the reflexes were recovered in theatre: death was due to cardiac failure on second day.

The incidence of post-operative complications and vomiting in the present series of cases is shown in Table 3. This forms the basis of the comparison between the anaesthetic agents and techniques which have been long established in general use, and those more recently introduced. The influence of the site of operation, the age and physical state of the patient on the incidence of the post-operative complications and vomiting, as shown in the succeeding

tables will be taken into consideration in the assessment of the two groups. Ether, nitrous oxide and regional analgesia have been taken as representative of the older drugs and techniques, and spinal analgesia, intravenous anaesthesia and thiopentone sodium cyclopropane, Trilene, Vinesthene Anaesthetic Mixture or "V.A.M", intravenous anaesthesia with curare, nitrous oxide with curare, and cyclopropane with curare as representative of the more recently introduced drugs and techniques.

The post-operative complications classified as "Technical", "burst" abdomen, wound sepsis, intestinal obstruction and pyloric stenosis, leakage of intestinal contents into the peritoneal cavity, recurrent laryngeal nerve palsy following sub-total thyroidectomy, dysphagia after excision of a thyroglossal cyst, pain in the shoulders due to pressure of the shoulder rests, haemorrhage, and oedema of the stoma following gastro-enterostomy, cannot be said to influence the merits of the anaesthetic agents in question, and consequently require no further consideration. Included in the classification of major respiratory complications are conditions such as broncho-pneumonia, lobar pneumonia, collapse of one or more lobes of a lung, severe attacks of acute bronchitis, and pulmonary embolism. Minor respiratory complications included were threats, cough with

no physical signs to be made out on clinical examination, exacerbations of bronchitis, and were assumed to be present in these with pre-operative minor respiratory complications such as chronic bronchitis, in whom a slight exacerbation was difficult to diagnose. As a result the incidence was probably less than the figure of twenty-one per cent. The most common complication affecting the central nervous system was headache. One case developed herpes following trigeminal root section, one case of facial nerve palsy following the same operation. There were also two cases of mental confusion, one of cerebral embolism, one fatal cerebral anoxia, one median and radial nerve palsy due to the pressure of the shoulder rests with the patient in a steep Trendelenburg position. The relatively high incidence of headache may be due in part to the fact that almost three per cent of all the cases gave histories of being subject to headaches. The complications classified as "Other" were those which could not be included in any of the previous categories, such as dysentery, cellulitis of arm following intravenous infusion, local sepsis, dermatitis due to "Elastoplast", and back-pain following the operation of dilatation and curettage. Such complications could not be related to the anaesthetic, and therefore no further comment is required.

**Table 3. The Incidence of Post-operative Complications
and Vomiting.**

Complications	Number of Cases	Percentage
Technical	17	3.4
Respiratory Major	25	5.0
Respiratory Minor	105	21.0
Cardio-Vascular System during 1st 24 hours	25	5.0
Cardio-Vascular System later	12	2.4
Central Nervous System	74	15
Genito-Urinary	57	11.4
Other	14	2.8
None	246	49
No Vomiting	330	66
Less than 12 hours Vomiting	116	23
Less than 24 hours Vomiting	22	4
More than 24 hours Vomiting	32	7

Table 4. The Sites of Operation.

Site of Operation	Number of Cases
Extremities	37
Perineal	142
Genito-Urinary	37
Inguinal	26
Lower Laparotomy	139
Upper Laparotomy	74
Body Wall	19
Other Head and Neck	6
Thyroid	6
Mouth, Nose and Throat	3
Brain and Spinal Cord	10
No operation	1

Table 5. The Age Distribution.

Age in Years	Number of Cases	Percentage
0 - 10	9	2
10 - 20	27	5
20 - 40	200	40
40 - 60	178	36
61 and over	86	17

Table 6. The Distribution of Cases According to
Pre-operative Physical State.

Physical State	Number of Cases	Percentage
Good	146	29
Fair	237	47
Poor	104	21
Serious	13	3

Table 7. The Distribution of the Principal Anaesthetic Agents.

Agents	Number of Cases	Percentage
Regional	11	2.2
Spinal	127	24.2
Intravenous	12	2.4
Intravenous + Curare	28	5.6
Nitrous Oxide	151	30.2
Nitrous Oxide + Curare	43	8.6
Cyclopropane	36	7.2
Cyclopropane + Curare	7	1.4
Ether	64	12.8
Ether + Curare	2	0.4
Trilene	11	2.2
Trilene + Curare	3	0.6
"V.A.M."	10	2.0
"V.A.M." + Curare	1	0.2
All Curare Cases	84	16.8

Table 8. The Distribution of Drugs used in Premedication.

Drug	Number of Cases	Percentage
Opiate	349	70
Opiate + Oral Barbiturate (1)	19	4
Opiate + Avertin	11	2
Oral barbiturate (2)	45	9
Rectal Paraldehyde	4	1
Atropine or Hyoscine only	48	9
Pethedine	23	5
None	1	0

(1) Nembutal, Seconal or Carbrital.

(2) Nembutal, Seconal, Sodium Senexyl or Carbrital.

Table 9. The Age Distribution of Post-Operative Complications

Age (Years)	Post-Operative Complications													
	Resp. Major		Resp. Minor		C.V.S. 1st 24 hrs.		C.V.S. later		C.N.S.		G.U.		None	
	No.	%	No.	%	No.	%	No.	%	No.	%	No.	%	No.	%
0-10	0	0	3	33	0	0	0	0	1	11	0	0	5	55
10-20	1	3.6	8	29	0	0	0	0	2	7.3	1	3.6	13	48
20-40	9	4.5	43	21	2	1.0	4	2	36	18	16	8.0	105	52
40-60	9	5.0	34	19	10	5.4	5	2.8	29	16	25	14.0	85	47
61+	6	7.0	17	20	13	15	3	3.5	4	4.6	15	17.0	38	44

Table 10. The Distribution of the Principal Anaesthetic

Age (Years)	Regional		Spinal		Intravenous		Intravenous + Curare		Nitrous Oxide		Nitrous Oxide + Curare		Cyclopropane	
	No	%	No	%	No	%	No	%	No	%	No	%	No	%
0-10 *	0	0	1	12.5	0	0	0	0	2	25.0	0	0	0	0
10-20	0	0	5	18	0	0	1	0	9	33	5	18	1	0
20-40	3	1.5	41	20.5	6	3	4	2	65	32.5	17	8.5	16	8
40-60	5	2.8	56	32	4	2.2	12	6.7	48	22.7	14	7.8	11	6.1
61+	3	3.8	18	20	2	2.3	11	12.3	27	34.5	7	8	8	9.2

* Too few cases to be of statistical significance

Agents throughout each age group.

Cyclopropane + Curare	Ether	Ether + Curare*	Trillene	Trillene + Curare*	V.A.M.	V.A.M. + Curare*	All Curare Cases.
No %	No %	No %	No %	No %	No %	No %	No %
0 0	3 37.5	0 0	0 0	0 0	2 25	0 0	0 0
0 0	3 11	0 0	2 7	0 0	1 3	0 0	6 18
1 0.5	35 17.5	1 0.5	4 2	1 0.5	6 3	0 0	24 12
3 1.6	19 10	0 0	3 1.6	2 1.1	1 0.5	1 0.5	31 17.2
3 3.8	4 4.6	1 1.6	2 2.3	0 0	0 0	1 1.6	23 27.6

Table 11. The Occurrence of Post-Operative Complications
in each grade of Pre-Operative Physical State.

Physical State	Resp. Major		Resp. Minor		Card. Vasc. Syst. during 1st 24 hrs.		Card. Vasc. Syst. later		Cent. Nerv. System		Genito Urin. Syst.		None	
	No	%	No	%	No	%	No	%	No	%	No	%	No	%
Good	7	4.8	19	13	3	2.1	1	0.7	15	10.2	12	8.2	94	64.4
Fair	9	3.8	59	25	7	3.0	6	2.5	43	18	32	13	107	45.1
Poor	7	7	24	23	11	10.6	4	4.0	13	11	12	11	42	40.4
Serious	2	15	3	23	4	30.7	1	7.0	1	7	1	7	3	23.1

Table 12. The Occurrence of Post-Operative Complications
according to Site of Operation.

Site of Operation	Resp. Major		Resp. Minor		Cardio Vasc. Syst. during 1st 24 hours		Cardio Vasc. Syst. Later		Central Nervous System		Genito Urinary System		None	
	No	%	No	%	No	%	No	%	No	%	No	%	No	%
Extremities	1	2.7	6	16.2	4	10.8	2	5.4	6	16.2	3	8.1	19	51
Peroneal	2	1.4	23	16.4	4	3	1	0.7	21	15	25	18	82	58.5
Genito-Urin.	1	2.6	9	23	5	12.8	1	2.5	3	7.6	4	10.3	19	48
Inguinal	0	0	7	27	0	0	0	0	3	11.5	3	11.5	15	57.7
Low.Lap.	11	8	33	23.5	2	1.4	4	3	21	15	11	8	65	46.4
Upp.Lap.	8	11	16	22.2	10	14	4	5.5	11	15.3	6	8.3	26	36
Body Wall	1	5.2	3	15.2	0	0	0	0	2	10.5	1	5.2	12	53
Other Head & N.	0	0	4	-	0	0	0	0	1	-	1	-	0	0
Thyroid	1	-	3	-	0	0	0	0	0	0	1	-	0	0
Mouth, Nose & T.	0	0	0	-	0	0	0	0	1	-	0	0	2	-
Brain & Cord.	0	0	1	10	0	0	0	0	3	30	2	20	5	50

**Table 13. The Incidence of Post-Operative Complications
with the Principal Anaesthetic Agents.**

Complication	No.	%	No.	%	No.	%	No.	%	No.	%	No.	%	No.	%	No.	%
Ether	2	3	5	3	2	18	6	5	0	0	1	0	0	0	1	10
Nitrous Oxide	14	22	26	17	1	10	20	15	4	33	8	22	3	27	4	40
Regional	0	0	4	25	1	9	11	85	1	85	1	25	1	9	0	0
Spinal	0	0	3	2	1	9	3	23	0	0	2	55	0	0	0	0
Intravenous	0	0	3	2	1	9	3	23	0	0	2	55	0	0	0	0
Cyclopropane	0	0	4	25	1	9	11	85	1	85	1	25	1	9	0	0
Trilene	0	0	3	2	1	9	3	23	0	0	2	55	0	0	0	0
"V.A.M."	0	0	3	2	1	9	3	23	0	0	2	55	0	0	0	0
Intravenous + Curare	0	0	3	2	1	9	3	23	0	0	2	55	0	0	0	0
Nitrous Oxide + Curare	0	0	3	2	1	9	3	23	0	0	2	55	0	0	0	0
Cyclopropane + Curare	0	0	3	2	1	9	3	23	0	0	2	55	0	0	0	0
All Curare Cases	0	0	3	2	1	9	3	23	0	0	2	55	0	0	0	0

Table 15. The Principal Anaesthetic according to the

Site of Operation	Ether		Nitrous Oxide		Regional		Spinal	
	No	%	No	%	No	%	No	%
Extremities	1	1.5	26	17	1	9	3	2.4
Perineal	22	34	92	61	0	0	4	3.0
Genito-Urinary	0	0	3	2	0	0	24	20
Inguinal	2	3	1	0.7	0	0	15	12
Lower Laparotomy	36	57	2	1.4	0	0	36	30
Upper Laparotomy	0	0	0	0	10	91	33	27
Body Wall	0	0	9	6	0	0	5	4
Other Head & Neck	2	3	2	1.4	0	0	0	0
Thyroid	0	0	5	3.3	0	0	0	0
Mouth, Nose & Throat	1	1.5	2	1.4	0	0	0	0
Brain & Spinal Cord	0	0	9	6	0	0	0	0
No Operation	0	0	0	0	0	0	1	1

Table 16. Sex Incidence of Post-Operative Complications.

Sex	Resp.Major		Resp.Minor		C.V.S. during 1st 24 hours		C.V.S. later		C.N.S.		G.U.		None	
	No.	%	No.	%	No.	%	No.	%	No.	%	No.	%	No.	%
Male	12	8.0	43	29	14	9.3	6	4	21	14	17	11.3	84	56
Female	13	3.7	62	17.7	11	3.1	6	1.7	51	14.6	40	11.4	162	46.3

Table 9, the age distribution of the post-operative complications, showed that with the exception of minor respiratory complications, the incidence of post-operative complications increased with the age of the patient, but between the ages of twenty and sixty years, the only significant increase was in the incidence of complications affecting the cardio-vascular system during the first twenty-four hours after the operation and complications affecting the genito-urinary system.

Table 10, the distribution of the principal anaesthetic agents throughout the age-groups, showed that with the exception of intravenous anaesthesia, nitrous oxide, cyclopropane and Trilene, the greatest number of administrations of any of the drugs in any one age group was associated with

the age-group forty to sixty years. In the case of the exceptions, the comparative age group was that of twenty to forty years. Consequently, for these drugs, the age of the patients was a factor in favour of a low incidence of post-operative complications, as it has been shown that the incidence of these complications increased with the age of the patient.

Table 11, the occurrence of post-operative complications in each grade of pre-operative physical state, showed that major respiratory, and complications affecting the cardio-vascular system increased in frequency with the decline in the pre-operative physical state of the patient. The incidence of complications affecting the central nervous and genito-urinary systems tended to decrease with the decline in the pre-operative physical state.

Table 12, the occurrence of complications according to the site of operation, showed that major respiratory complications occurred most frequently with lower and upper laparotomies, and operations on the body wall. Minor respiratory complications were associated most frequently with upper and lower laparotomies, operations on the genito-urinary system, and in the inguinal region. Complications affecting the cardio-vascular system during the first twenty-four hours after the operation were most frequently associated with upper laparotomies, operations on the genito-

urinary system and on the extremities. Complications affecting the cardio-vascular system after the first twenty-four hours were most frequent following upper laparotomies, and operations on the extremities. Central nervous system complications occurred most often after operations on the brain and spinal cord, perineum, and after upper and lower laparotomies. Genito-urinary complications were most frequently associated with operations on the brain, spinal cord, and perineum.

Table 13, the incidence of post-operative complications with the principal anaesthetic agents, showed that major respiratory complications were most frequently associated with regional analgesia, "V.A.M." anaesthesia, the combination of intravenous anaesthesia and curare, and cyclopropane and curare; respiratory minor complications with cyclopropane and curare, "V.A.M.", intravenous anaesthesia, nitrous oxide and curare; cardio-vascular complications during the first twenty-four hours after the operation with regional and spinal analgesia, Trilene, intravenous anaesthesia with, or without curare, and cyclopropane and curare; cardio-vascular complications after the first twenty-four hours with regional analgesia, cyclopropane, and intravenous anaesthesia and curare; central nervous system complications with spinal analgesia, ether,

and nitrous oxide anaesthesia; genito-urinary complications with intravenous anaesthesia, cyclopropane, nitrous oxide, intravenous anaesthesia and curare, and cyclopropane and curare.

Table 14, the principal anaesthetic agents in each grade of pre-operative physical state, showed that eighty-nine per cent of the cases given ether, eighty-three per cent of cases given nitrous oxide, none of the cases given regional analgesia, eighty-six per cent of those given spinal analgesia, sixty-seven per cent of those given intravenous anaesthesia, sixty-one per cent of those given cyclopropane, seventy-three per cent of those given Trilene, ninety per cent of those given "V.A.M.", sixty-one per cent of those given intravenous anaesthesia and curare, sixty-five per cent of those given nitrous oxide and curare, and forty-three per cent of those given cyclopropane and curare were classified as good or fair anaesthetic risks. It might be suggested that this factor favoured a decrease in the incidence of major respiratory complications, complications affecting the cardio-vascular system, and an increase in the incidence of complications affecting the central nervous and genito-urinary systems with ether, nitrous oxide, spinal analgesia, Trilene, and "V.A.M." anaesthesia, and the opposite effect with regional analgesia, and the

combination of cyclopropane and curare.

Table 15, the principal anaesthetic agent according to the site of operation, showed that ether was most frequently administered for operations on the perineum and the extremities, nitrous oxide for operations on the perineum, regional analgesia for upper laparotomies, spinal analgesia for operations on the genito-urinary system and upper and lower laparotomies, intravenous anaesthesia for operations on the perineum and extremities, cyclopropane for operations on the perineum and lower laparotomies; Trilene for operations on the perineum, "V.A.M." for operations on the inguinal region and for lower laparotomies, and the combination of general anaesthesia and curare for upper and lower laparotomies.

Table 16, the sex incidence of post-operative complications, shows that in this series of cases post-operative complications were more liable to occur in women than in men. Major and minor respiratory and circulatory system post-operative complications were more frequent in men than in women. *final column is for women*

Ether was associated with a relatively high incidence of complications affecting the central nervous system. It was employed in the majority of cases for operations associated with complications affecting the cardiovascular, central nervous, and genito-urinary systems. The physical state of

the patients favoured a decrease in the incidence of major respiratory, and circulatory complications, and an increase in the incidence of complications involving the central nervous and genito-urinary systems.

Nitrous oxide was associated with a relatively high incidence of complications affecting the central nervous and genito-urinary systems. It was employed in the majority of cases for operations associated with complications involving the same two systems. The physical state of the patients favoured an increase in these two groups of complications and a decrease in the incidence of major respiratory and circulatory system upsets.

Regional analgesia was associated with a relatively high incidence of complications affecting the respiratory system to a major degree, and the cardio-vascular system during, and after, the first twenty-four hours following the operation. It was employed almost entirely for operations associated with major respiratory, minor respiratory, and central nervous system complications, and upset of the cardio-vascular system during, and after, the first twenty four hours following the operation. The physical state of the patients favoured a rise in the incidence of complications affecting the cardio-vascular system, and the respiratory system to a major degree, and a fall in the

incidence of those affecting the central nervous and genito-urinary systems.

Spinal analgesia was associated with a relatively high incidence of complications affecting the central nervous and cardio-vascular systems during the first twenty-four hours following the operation. It was employed largely for operations associated with major and minor respiratory, central nervous system and cardio-vascular system complications during and after the first twenty four hours following the operation. The physical state of the patient favoured a decrease in major respiratory and all cardio-vascular system complications, and an increase in those affecting the central nervous and genito-urinary systems.

Intravenous anaesthesia was associated with a relatively high incidence of minor respiratory, genito-urinary and cardio-vascular system complications during the first twenty four hours following the operation. It was employed on the whole for operations associated with central nervous, genito-urinary, and all cardio-vascular system complications. The physical state of the patients was not a factor of importance, but the age of the patients favoured a decrease in the complications affecting the genito-urinary system, and the cardio-vascular system during the first

twenty four hours following the operation.

Cyclopropane was associated with a relatively high incidence of complications affecting the genito-urinary system and the cardio-vascular system after the first twenty four hours following the operation. It was employed in operations associated with major and minor respiratory, central nervous system, and genito-urinary complications. The physical state of the patients was not a factor of importance, but the age of the patients favoured a decrease in genito-urinary complications and those affecting the cardio-vascular system during the first twenty four hours following the operation.

Trilene was associated with a high incidence of complications affecting the cardio-vascular system during the first twenty four hours following the operation. It was administered for operations associated with a high incidence of central nervous and genito-urinary system complications. The physical state of the patients favoured a decrease in major respiratory and all cardio-vascular system complications, and an increase in those affecting the central nervous and genito-urinary system. The age of the patients favoured a decrease in complications affecting the genito urinary system, and the cardio-vascular system during the first twenty four hours following the operation.

"V.A.M." was associated with a relatively high incidence of major and minor respiratory complications. It was given for operations associated with a high incidence of major and minor respiratory complications, and central nervous system complications. The physical state of the patients favoured a decrease in major respiratory and cardio-vascular system complications, and an increase in those affecting the central nervous and genito-urinary systems.

The combination of intravenous anaesthesia and curare was associated with a high incidence of major respiratory, genito-urinary, and all cardio-vascular system complications. The combination was administered for operations associated with a high incidence of major and minor respiratory, early and later cardio-vascular, and central nervous system complications. The physical state of the patients was not a factor of importance.

The combination of nitrous oxide anaesthesia and curare was associated with a relatively high incidence of major respiratory complications. It was employed for operations associated with a high incidence of major and minor respiratory, early and later cardio-vascular, and central nervous system complications. The physical state of the patients was not a factor of importance. The age of the patients favoured a decrease in the incidence of

complications affecting the genito-urinary system, and the cardio-vascular system during the first twenty four hours following the operation.

The combination of cyclopropane anaesthesia and curare was associated with a relatively high incidence of major and minor respiratory, genito-urinary, and early cardio-vascular system complications. It was administered in the majority of cases for operations associated with a high incidence of complications affecting the respiratory system to a major and minor degree, the central nervous system, and the cardio-vascular system during and after, the first twenty four hours following the operation. The physical state of the patients favoured a rise in respiratory major and early and late cardio-vascular system complications and a fall in the incidence of central nervous and genito-urinary system complications.

The incidence of no post-operative complications has not been discussed, as the absence of all post-operative complications would include the absence of complications classified as 'Technical' and 'Other' which have been shown to be uninfluenced by the anaesthetic agent or technique in use.

Having considered the influence of the factors, site of operation, pre-operative physical state and the age

of the patient, on the incidence of post-operative complications associated with the anaesthetic agents and techniques employed in this series of cases, it is suggested that the modern drugs and techniques were not more liable to be associated with post-operative complications than the older drugs and techniques.

The relationship between the drugs used in pre-medication and post-operative complications must now be discussed. The actual drugs and the number of cases to which each was administered has already been noted (Table 8). The combination of opiate and a drug administered per rectum, and rectally administered drugs were used almost entirely for the premedication of cases requiring neuro-surgical operations, or operations on the thyroid gland. The other drugs and combinations of drugs were administered prior to all types of operations with the exception of the two mentioned above, and of these, pethidine would appear to be the most satisfactory in respect of association with post-operative complications (Table 17).

Post-operative vomiting is important with regard to the comfort of the patient, and also because prolonged vomiting may result in harmful biochemical changes. Later, an attempt will be made to demonstrate the benefits of slight vomiting with regard to complications affecting the respiratory system.

The incidence of vomiting is shown in Table 3. In the following tables (Tables 18-22) the relationships between vomiting and age, sex, and pre-operative physical state of the patient, the site of operation and the principal anaesthetic agents are shown.

Table 18. The Incidence of Vomiting in Each Age Group.

Age (Years)	Vomiting (Hours)							
	None		Less than 12		Less than 24		More than 24	
	No	%	No	%	No	%	No	%
0 - 10	7	78	2	22	0	0	0	0
11 - 20	17	63	6	22	1	4	3	11
21 - 40	140	70	41	20	7	4	12	6
41 - 60	105	60	48	27	13	7	12	6
61+	61	71	19	22	1	1	5	6

**Table 19. The Incidence of Vomiting on each grade of
Pre-Operative Physical State.**

Physical State			Vomiting (Hours)					
	None		Less than 12		Less than 24		More than 24	
	No	%	No	%	No	%	No	%
Good	97	69	32	23	8	4	9	4
Fair	151	63	62	26	10	4	14	7
Poor	71	68	21	20	4	4	8	8
Serious	11	84	1	8	0	0	1	8

Table 20. The Incidence of Vomiting for each Site of
Operation.

Site of Operation	Vomiting (Hours)							
	None		Less than 12		Less than 24		More than 24	
	No	%	No	%	No	%	No	%
Extremities	28	76	6	16	2	6	1	2
Perineal	90	63	43	30	7	5	2	2
Genito- urinary	26	70	5	14	1	2	5	14
Inguinal	20	77	6	23	0	0	0	0
Lower Laparotomy	85	61	33	24	10	7	11	8
Upper Laparotomy	51	70	10	13	1	1	12	16
Body Wall	16	84	3	16	0	0	0	0
Other Head and Neck	4	66	2	33	0	0	0	0
Thyroid	4	66	1	17	0	0	1	17
Mouth, Nose and Throat	1	33	2	66	0	0	0	0
Brain and Spinal Cord	4	40	5	50	1	10	0	0

Table 21.Sex Incidence of Vomiting.

Sex	Vomiting (Hours)							
	None		Less than 12		Less than 24		More than 24	
	No	%	No	%	No	%	No	%
Male	137	75	29	16	2	2	13	7
Female	193	60	87	27	20	7	19	6

**Table 22. The Incidence of Vomiting with each Principal
Anaesthetic Agent.**

Anaesthetic Agent	Vomiting (Hours)							
	None		Less than 12		Less than 24		More than 24	
	No	%	No	%	No	%	No	%
Ether	31	48	25	39	4	6	4	6
Nitrous Oxide	100	66	40	26	7	4	4	4
Regional	7	64	2	18	0	0	2	18
Spinal	86	72	20	16	5	4	10	8
Intra-venous	11	91	1	9	0	0	0	0
Cyclopropane	22	61	11	30	2	6	1	3
Trilene	7	63	3	27	1	10	0	0
V.A.M.	5	50	5	50	0	0	0	0
Intravenous + Curare	21	75	3	11	1	3	3	11
Nitrous oxide + Curare	31	72	3	7	1	3	8	18
Cyclopropane + Curare	4	57	2	29	1	14	0	0
All Curare Cases	61	72	9	10	3	5	11	13

In this series of cases post-operative vomiting bore no relationship to the age of the patient or the pre-operative physical state. The absence of vomiting was greatest in cases where the operation was performed on the body wall, extremities, or in the inguinal region, and excessive vomiting occurred most frequently following operations on the thyroid gland, on the genito-urinary system and following upper laparotomies. The modern anaesthetic agents were associated with less vomiting than the older drugs and techniques. Slight vomiting occurred more often with the modern, but excessive vomiting least with these drugs and techniques.

The incidence of post-operative vomiting with the various drugs used in premedication was then investigated. It would appear from Table 23 that the absence of such vomiting was most frequent after oral premedication, that is, one of the following barbiturates administered by mouth, "Nembutal", "Seconal", "Sodium Seneryl" or "Carbrital". Vomiting was more frequent when pethedine was used.

Table 23. The Incidence of Post-Operative Vomiting with the various Types of Premedication.

Drug	Percentage of cases having the Drug with			
	No Vomiting	Under 12 hours Vomiting	Under 24 hours Vomiting	More than 24 hours Vomiting
Opiate	68	19	4	9
Opiate + Oral	58	32	5	5
Opiate + Rectal	55	36	9	0
Oral	75	15	3	7
Oral + Rectal	No statistical significance			
Atropine or Hyascine	63	33	2	2
None	No statistical significance			
Pethedine	35	56	9	0

It has been suggested that one of the factors in the increase in post-operative respiratory complications with modern anaesthesia is the decrease in post-operative vomiting. Table 24 shows that, in this series of cases, the statistical evidence was inconclusive.

Table 24. The Incidence of Post-Operative Respiratory Complications according to the Duration of Vomiting in the Post-Operative Period.

Respiratory Complication	No Vomiting Percentage of Cases	Less than 12 Vomiting. Percentage of Cases	12-14 hours Vomiting. Percentage of Cases	Over 24 hours Vomiting. Percentage of Cases.
Respiratory Major	5.2	5	9	19
Respiratory Minor	21.2	18	18	22
None	73.6	77	73	59

It has been suggested that one of the undesirable results of the modern technique of light general anaesthesia and curare is the tendency for a fall in blood-pressure to occur when the patient has returned to bed. In order to test the veracity of this statement, blood-pressure recordings were taken four hours after the end of the operation in a series of cases who had undergone the operation of partial gastrectomy. Thirty-six cases were studied, twelve were given light general anaesthesia and curare, eleven were given spinal analgesia by the fractional technique of Lemmon, and thirteen were given spinal analgesia by the single dose technique of Howard Jones, and the blood-pressure alterations noted in Tables 25, 26 and 27.

Table 25. The Alterations in Blood-Pressure Associated with the Operation of Partial Gastrectomy, Performed under Light General Anaesthesia and Curare, during the operation, at the end of the first four hours following the operation when compared with the pre-operative level and with the level at the end of the operation.

	Change in blood-pressure during operation. (Millimetres of Mercury).			Change in blood-pressure at end of first four hours compared with pre-operative.			Change in blood-pressure at end of first four hours compared with end of operation.		
	Syst- olic	Dias- tolic	Pulse Press.	Syst- olic	Dias- tolic	Pulse Press.	Syst- olic	Dias- tolic	Pulse Press.
Case 1	+10	+15	-5	-10	-15	+5	-20	-30	+10
" 2	+30	+15	+15	+15	-15	+30	-25	-45	+20
" 3	+30	+20	+10	+10	+10	0	-20	-10	-10
" 4	0	+10	-10	-30	-20	-10	+10	-15	+25
" 5	+30	+20	+10	-20	0	-20	-20	-10	-10
" 6	0	+10	-10	-35	-10	-25	-35	-20	-15
" 7	+10	0	+10	0	0	0	+40	+25	+15
" 8	+10	+10	0	+20	0	+20	0	0	0
" 9	+10	0	+10	-30	-15	-15	-30	-15	-15
" 10	-10	-5	-5	+10	+20	-10	0	-5	+5
" 11	0	-10	+10	+2	+10	-8	0	+10	-10
" 12	0	+10	-10	-10	0	-10	-10	-10	0
Average	+10	+7.9	+2.0	-6.5	-2.9	-3.5	-9.1	-10.4	+1.2
Range	-10 to +30	-10 to +20	-10 to +15	-35 to +20	-25 to +20	-25 to +30	-35 to +40	-45 to +25	-15 to +25
Standard Deviation	12.9	9.27	9.23	18.13	11.8	15.3	19.9	16.64	13.2

Table 26. The Alterations in Blood Pressure Associated with the Operation of Partial Gastrectomy, Performed under Fractional Spinal Analgesia, during the operation, at the end of the first four hours after the operation when compared with the pre-operative level, and after the first four hours post-operatively compared with the level at the end of the operation.

	Change in blood-pressure during operation (millimetres of mercury)			Change in blood-pressure at the end of the first four hours compared with pre-operative (millimetres of mercury)			Change in Blood-pressure at end of first four hours compared with end of operation (millimetres of mercury)		
	Syst- olic	Dias- tolic	Pulse Press.	Syst- olic	Diast- olic	Pulse Press.	Syst- olic	Diast- olic	Pulse Press.
Case 1	0	-5	+5	+35	+20	+15	+35	+35	0
" 2	-50	-30	-20	-10	+10	-20	+10	+30	-20
" 3	-20	0	-20	-20	+10	-30	-30	-20	-10
" 4	-50	-15	-35	+15	+15	0	+45	+20	+25
" 5	-5	0	-5	-10	-10	0	-25	-15	-10
" 6	-5	-15	+10	+35	+35	0	+45	+40	+5
" 7	-20	0	-20	+25	+15	+10	+45	+15	+30
" 8	-30	-5	-25	-20	+15	-35	+10	+20	-10
" 9	-10	0	-10	0	-10	+10	+20	-10	+30
" 10	-40	-10	-30	+20	+5	+15	+40	+25	+15
" 11	-40	-10	-30	+35	+20	+15	+35	+20	+15
Average	+23.6	-8.1	-16.3	+9.5	+11.3	-1.8	+20.9	+14.5	+6.3
Range	-50 to 0	-30 to 0	-35 to +10	-20 to +35	-10 to +35	-35 to +15	-30 to +45	-20 to +40	-20 to +30
Stand. Deviation	18.89	8.9	14.1	20.07	7.54	18	25.9	19.4	16.9

Table 27. The Alterations in Blood Pressure Associated with the Operation of Partial Gastrectomy, Performed under Spinal Analgesia, during the operation, at the end of the first four hours after the operation when compared with the pre-operative level, and after the first four hours post-operatively compared with the level at the end of the operation.

	Change in Blood-pressure during operation (mms. of mercury).			Change in Blood-pressure at end of first four hours compared with pre-operative. (mms. of mercury).			Change in Blood-pressure at end of first four hours compared with end of operation (mms. of mercury).		
	Syst-olic	Diast-olic	Pulse Press.	Syst-olic	Diast-olic	Pulse Press.	Syst-olic	Diast-olic	Pulse Press.
Case 1	+5	+15	-10	+15	+10	+5	+10	0	+10
" 2	+5	+20	-15	+40	0	+40	+10	-25	+35
" 3	+5	+20	-15	+5	+15	-10	0	0	0
" 4	+40	+20	+20	-10	-5	-5	-20	-15	-5
" 5	+10	0	+10	+50	+25	+25	+40	+25	+15
" 6	+10	+10	0	+30	+40	-10	+25	+10	+15
" 7	-10	0	-10	-15	+15	-30	-5	+5	+10
" 8	-40	0	-40	-20	+10	-30	+20	+10	+10
" 9	+10	+15	-5	-20	0	-20	-10	-10	0
" 10	+30	+30	0	+10	+10	0	0	-15	+15
" 11	-20	0	+20	0	-10	+10	0	-10	+10
" 12	+20	+10	+10	+25	+20	+5	-5	+5	-10
" 13	0	+5	-5	0	+20	-20	-10	+5	-15
Average	+5.0	+11.1	-3.07	+8.4	+11.5	-3.0	+4.3	-1.1	+5.4
Range	-40 to +40	0 to +30	-40 to +20	-20 to +50	-10 to +40	-30 to +40	-20 to +40	-25 to +25	-15 to +35
Stand. Deviation	19.69	9.48	15.36	21.56	12.96	19.77	15.7	12.68	13.22

The following conclusions are suggested as a result of study of Tables 25, 26 and 27. In this series of thirty-six cases of partial gastrectomy, a light general anaesthesia and curare was associated with the least change in the systolic blood pressure and in the pulse pressure during the operation, and at the end of the first four hours after the operation when compared with the respective levels before operation. This combination was also associated with least alteration in the pulse pressure at the end of the first four hours after the operation when compared with the pulse pressure at the end of the operation. Fractional spinal analgesia was associated with the least alteration in the diastolic blood pressure during the operation, and the least change when the diastolic blood pressure taken at the end of the first four hours after the operation was compared with the diastolic blood pressure before operation. Single-dose spinal analgesia was associated with least change in the systolic and diastolic blood pressures and the pulse pressure when the values at the end of the first four hours after the operation were compared with those at the end of the operation.

It is concluded that the statement that an undesirable fall in blood pressure is liable to occur in the first four hours after the end of an operation which has been

performed under light general anaesthesia and curare is incorrect in respect of this series of cases.

The statement, that the new anaesthetic agents and techniques have no appreciably greater mortality and morbidity rates associated with their use than the old and more simple methods, is not without risk, as it may tempt the unskilled or "occasional" anaesthetist into their administration without his realising the danger involved in the use of these drugs by such a person. These dangers are very real and may arise in a number of ways. There is the "occasional" anaesthetist who receives samples and literature concerning new drugs, and who attempts their administration. There is the house-surgeon left by the specialist anaesthetist with a patient anaesthetised by such drugs, or, who attempts their use when the anaesthetist is not present. There are the dangers due to the untoward side-effects of these drugs. Such dangers have been described in detail. The unskilled anaesthetist is not acquainted with these effects, the conditions liable to be followed by them, their premonitory signs, and he is unable to deal with the condition when it has developed. He has not the experience necessary for the selection of cases suitable for these drugs, and he may be influenced by the surgeon or the patient into administering an anaesthetic quite unsuited to the case. Although

the use of the older anaesthetic agents is not free from risk, for example, anoxia in nitrous oxide anaesthesia, and primary cardiac failure with chlroform, the changes in the anaesthesia develop more slowly, allowing the inexperienced anaesthetist longer in which to appreciate the state of the anaesthesia and to take any action that may be required.

It is suggested that the use of the modern anaesthetic drugs and techniques be restricted to those who have been trained adequately in their administration.

Suggestions for Improving the Present Position.

As a result of this investigation showing little difference between the mortality and morbidity associated with the old and new methods of anaesthesia, to improve the position search must be made for small faults in the present technique. The scope and training of anaesthetists must be widened especially with regard to clinical medicine, and the resident medical and nursing staffs must be educated to play their parts. As all anaesthetics at present cannot be administered by specialist anaesthetists, general practitioners must be trained in simple and relatively safe anaesthetic techniques for cases which they may be called upon to anaesthetise. As a result of the present investigation, suggestions will be given regarding the avoidance of certain anaesthetics for certain patients.

The previous section shows that it is impossible to advocate the old or the new methods of anaesthesia to the exclusion of the other. If a patient has certain pre-operative complications, it would be better to choose the agent or method least likely to aggravate them. Similarly with drugs used in premedication, the choice should be made of those best suited for the patient's pre-operative state, the operation, and also in an attempt to avoid post-operative complications which are specially undesirable in this case.

In as much as post-operative complications increase in frequency when specially sought as shown by King, so do pre-operative complications. A liability to headaches on the part of the patient may not interest the house-surgeon when writing the case-record, but it does interest the anaesthetist considering administering a spinal analgesia. An early head-cold may not be diagnosed by the resident medical officer preparing a case for partial gastrectomy although the patient may have been fully investigated in all other ways. It is essential, therefore, for an anaesthetist to be able to see every case before and after operation (209).

It must be realised, however, that the anaesthetic is not the sole factor in the causation of the post-operative complications, but that the patient and the operation are at least equally important. While anaesthetists attempt to improve the position, surgeons are making it worse every day by more and more extensive operations on less and less fit patients.

As there is little difference between the results of the old and new anaesthetic agents and methods as a whole, any improvement must come from improvement in the choice of anaesthetic and the technique of the anaesthesia. For the individual anaesthetist the solution lies in experience and paying attention to detail. For future anaesthetists, much

depends on the teaching given to students, "residents", and specialist anaesthetists in training.

In the case of students, the question arises whether they should be taught modern methods of anaesthesia, or the simpler, older methods only. There are two schools of thought in this matter. The first believes that modern anaesthesia is not for undergraduates, because of the complexity of the methods, and the scope of the basic sciences. The second would teach the student everything about anaesthesia, from "open" ether at one extreme, to curare at the other. The ideal lies between the two, the production of general practitioners capable of administering reasonable anaesthesia, but who realise their own limitations. In future fewer anaesthetics will be given by general practitioners. But, for many reasons, a considerable time must elapse before a specialist anaesthetist will be available for every case. If the student is taught nothing about anaesthesia, he will go into practice unfitted to administer such anaesthetics as he may be called upon to give, such as in obstetrics, for dental extractions, and minor surgical procedures.

If he is to be taught anaesthesia, it may be either by watching the specialist anaesthetist use modern agents and techniques as might be required for a transthoracic

gastrectomy, or, certain cases have to be subjected to an "open" ether or chloroform anaesthesia for the benefit of the student. It might be argued that the first alternative is the correct choice because it is better for the patient and easier for the surgeon, the anaesthesia may be conducted by the specialist as though the student were not present, an "open" induction of anaesthesia is more time-consuming than the modern intravenous induction, there is less post-operative upset, and consequently less nursing care required, and the patient may demand the easy induction of intravenous thiopentone. The arguments against this choice are four in number. Modern anaesthesia is not the type the student will be called upon to administer in practice as he will not have the necessary apparatus, and it is unlikely that he will be asked to anaesthetise for major surgery. He does not have the necessary knowledge of the basic sciences and the applied pharmacology of the drugs used. Accidents may occur so quickly with the more rapidly acting modern agents that the supervising specialist may not be able to act sufficiently quickly to avoid disaster. In the short time at his disposal, the student cannot assimilate sufficient knowledge of complicated techniques to be of any practical value.

If it is decided to teach only the old "rag and bottle" anaesthesia, the number of cases available for teaching pur-

poses is reduced, the case chosen has to suffer a less pleasant induction of anaesthesia and post-operative period, the speed of working in theatre is reduced, and the old types of anaesthesia are not without risk, for example, the primary cardiac failure liable to occur with chloroform.

It has been suggested that the student be taught these simpler and more time-consuming anaesthetic techniques in the out-patient department and fracture clinic. But such patients have been incompletely investigated, the risk of vomiting is greater than with "in-patients", and few have received premedication. Out patients must be fit to return to their homes after a shorter interval than is possible after ether or chloroform anaesthesia. Such large numbers of patients are treated in these departments that speed is essential under the circumstances suggested. It may be difficult to justify subjecting a patient with a small abscess to ether or chloroform, when a very short gas and oxygen anaesthesia is all that is required.

Therefore, it would appear reasonable to suggest that the ideal cases would be in-patients having minor surgical operations, fully investigated, prepared and premedicated. The anaesthetist would choose those suitable for teaching purposes. Such cases are usually dealt with at the end of the session when the theatre staff are tired and anxious to

finish as soon as possible. In order to avoid undue delay and at the same time increase his experience, the student should be taught intravenous anaesthesia for induction purposes and as the sole anaesthetic for minor operations. In many small hospitals staffed by general practitioner anaesthetists there are Boyle's anaesthetic apparatus, therefore in order to give patients the benefit of an intravenous induction and the safety of a gas, oxygen and ether anaesthesia, the student should be taught this sequence, with the dangers and limitations of intravenous anaesthesia stressed, and the methods of resuscitation explained. If this is combined with a working knowledge of "open" ether, chloroform and ethyl chloride, he will have a knowledge of anaesthesia sufficient to meet the requirements of general practice. If there should be a dental unit in the hospital he might be taught gas and oxygen anaesthesia for dental extractions. The value of this is debatable as dental anaesthesia may be very difficult, and is beyond the scope of the general practitioner in the opinion of many, dentists and anaesthetists.

The student should receive instruction in the signs and dangers of anaesthesia, and how to deal with these complications as they arise. The value and use of the analeptic drugs should be explained. He should accompany the anaesthetist to the wards to see the special examination of patients

to determine fitness for anaesthesia, and be instructed in pre-operative care, premedication. In visits to the patient after the operation, the rudiments of post-operative care might be taught.

During major operations the student might assist with the charting, and learn the elements of venipuncture and blood transfusions. He should not be taught cyclopropane, endotracheal or closed circuit anaesthesia. Although spinal analgesia should not be included in the curriculum, local analgesia, which may be of great value in general practice, should be demonstrated, and if possible, carried out by the student. Students should live in hospital during their final surgical term and give anaesthetics to the emergency cases admitted to the hospital.

Anaesthesia in obstetrics should be taught when the student is receiving his training in midwifery and should include analgesia, and instruction in the use of the common types of apparatus used to obtain this state. It is better that the future general practitioner should receive this knowledge from a specialist anaesthetist than from the advertisements and instructions of the manufacturer of the analgesic machine. The instruction of anaesthesia in obstetrics is not without risk, the danger of vomiting is great and the beds in most labour wards cannot be tilted. The

student under instruction has only to make a slight error to cause severe spasm during induction or allow some obstruction to the airway during maintenance of anaesthesia to cause the foetus to gasp while in utero, filling its lungs with amniotic fluid. Too deep an anaesthesia to escape the danger of vomiting will result in impaired uterine contractions. Resuscitation of the baby should also be taught. The instruction should include open ether and chloroform, gas, oxygen and ether, and gas, oxygen and Trilene anaesthesia, the proper use of morphine and pethedine, gas and air, gas and oxygen, and Trilene analgesia.

Such a scheme of instruction for the undergraduate may appear over ambitious, but the present instruction is so inadequate that a curriculum must be drawn up and enforced by those in authority (210, 211, 212, 213, 214).

The training of the specialist anaesthetist may be discussed with regard to theoretical and practical teaching. There are two schools of thought regarding the former, one against, and one in favour of lectures. The extent of the training depends on the duration of the traineeship, which should be at least two years. This time might be divided into terms of three months, each to be spent with a different anaesthetist, and, if possible, changes of hospital might be arranged. Early in the training period he should have

lectures on the basic sciences in as far as they pertain to anaesthesia. These lectures should be given by members of the staffs of the respective departments of the university. Time might be spent in the anatomy laboratory where anatomy of special interest to the anaesthetist might be taught in a series of lecture demonstrations. Lectures on the physiology of the central nervous, respiratory and cardio-vascular systems might be given at the same time as the anatomy demonstrations. An anaesthetist should be responsible for the teaching of the pharmacology of the anaesthetic agents and the drugs used in anaesthesia. Lectures might also be given on blood-transfusion by the director of the Blood Transfusion Service, on the examination and diseases of the blood by a haematologist, and on the chemistry of the blood by a bio-chemist. One or two lectures might be given by a suitable psychiatrist on personality types, suggestion and hypnosis, and the treatment of the various psychological problems which may be met in patients before operation.

This scheme might have to be modified on economic grounds, cost to the trainee or the state depending on which was paying the lecturers' fees. It involves taking the lecturers from their usual work, and university staffs, especially medical, with inflated classes, are already over-worked. Also, the scheme results in the trainee spending

considerable periods neither in theatre, nor the wards.

The success or failure of practical training depends to a great extent on the good-will of the surgeons. The actual theatre training might be divided into three periods, during the first of which the trainee acts as the keeper of the anaesthetist's records, and in so doing learns, and is taught by the anaesthetist, the effect of the drugs used and the techniques of anaesthesia. During the second period he might carry out these techniques under supervision; during the third he might work almost independently of his teacher. For example, if there were two operating tables in use in the theatre, he might anaesthetise the cases on the second table. As time passed and his experience increased, the degree of supervision by the teacher would decrease.

For instruction in pre-operative care, premedication, and post-operative care, the trainee might merely accompany the anaesthetist in his visits to the wards during the first period of training. In the second phase the trainee might give the instructions under the immediate supervision of his instructor, and in the third carry out the visits to the wards by himself.

Each trainee should not be appointed to one teaching hospital, but to a group of hospitals and remain for only a certain period with each anaesthetist, and then pass to another

so as to learn different methods, each anaesthetist making a special study of one particular technique, and of these, one should be regional analgesia. During the basic training in anaesthesia for general surgery, he should be given instruction in intravenous infusions and blood transfusions, and, at the end of operations, in tracheo-bronchial toilet and bronchoscopy. During the post-operative period the prophylaxis of respiratory and circulatory system complications should be demonstrated. When not occupied in theatre or ward, the trainee should be in the out-patient department or fracture-clinic where his services will be of value and where he will gain experience in anaesthesia for out-patients.

After this basic training in anaesthesia he should move to the units dealing with diseases of the ear, nose and throat, the eyes, and the gynaecological department. If possible, anaesthesia for thoracic and neurological surgery should be included in the curriculum. A longer period should be spent with an obstetrical unit than with the other specialised branches of surgery, because although a basic training in anaesthesia for neurosurgery or thoracic surgery is all that is required unless becoming permanently attached to one of these units, any anaesthetist may be called upon for obstetrical anaesthesia or analgesia. The training of midwives in analgesia may interfere with his instruction by

occupying the time of the anaesthetist, and by taking their quota of cases reduce the number available for him, and therefore he should accompany the anaesthetist during his teaching of the midwives. He should be taught to use methods of analgesia other than the Minnitt apparatus, and also the resuscitation of infants. The difficulties of teaching anaesthesia for obstetrics have already been described, but it is essential that the future specialist should have an adequate training in this subject.

In order to make possible the suggested scheme of training, the pupil would have to be appointed by some central authority such as a regional hospital board. The appointment should extend to all the hospitals in the region, and not, as at present, to one particular hospital or anaesthetist (214, 215).

Statistics show that the mortality associated with anaesthesia rises each time new house-surgeons are appointed to a hospital. The fault does not lie with the resident medical officers, as, with large classes and insufficient time for students to have anaesthetic teaching, and the policy of more and more hospitals of demanding that all anaesthetics be administered by anaesthetists, students cannot be taught adequately.

The principal anaesthetics given by house-surgeons

are at the end of the morning operating list when the anaesthetist has departed, on "receiving" nights when no anaesthetist is present, and in the out-patient and casualty departments. In the first and third instances the cases are usually requiring minor surgical operations, but in the second, the anaesthetics may be for major surgical operations. In the first case, the "houseman" may be left with an anaesthetic for a major operation which had been commenced by the anaesthetist, and in anaesthetics in the out-patient department there are the risks of anaesthetising incompletely examined, unpremedicated patients, with full stomachs due to recently digested meals.

As soon after his appointment as possible each house-surgeon should be taught by an anaesthetist the signs of anaesthesia, simple anaesthesia such as 'open' ether and chloroform, the administration of intravenous thiopentone, as the sole anaesthetic, and as an induction agent to be followed by nitrous oxide and oxygen, with or without ether. Simple premedication, the accidents that may occur during anaesthesia and their treatment should also be taught. Perhaps most important of all, he should be made to realise when a case is beyond his capabilities and ask for a specialist anaesthetist for such cases as those undergoing upper abdominal operations, those suffering from surgical shock,

and those suffering from intestinal obstruction, the very young, and the very old.

During the main operating sessions the house-surgeon may learn about modern methods of anaesthesia, and how a particular case is standing up to the operation and anaesthetic, so that if the anaesthetist has to depart before the operation is completed, he will be able to continue the administration.

The anaesthetist should be dogmatic regarding the anaesthetics the house-surgeon may administer, and the drugs and apparatus he may use. The result is that if the 'resident' becomes a general practitioner, he will be able to administer any anaesthetics he may be called upon to give. If he should become a trainee anaesthetist, he has a useful basis on which to build his knowledge of anaesthesia. If he specialises in surgery or any of its branches, medicine or obstetrics, the knowledge cannot but be of value.

The problem of general practitioner anaesthetists is what anaesthetics should he give?-any which he could reasonably be expected to administer such as those for minor surgical operations and some major operations such as appendicectomy, and in obstetrics. The question arises as to whether he should attempt modern anaesthesia or have one or two techniques and drugs with which he is reasonably

proficient. He may favour the first alternative, but he will not administer sufficient anaesthetics to become efficient in their use, sufficiently experienced to choose the correct agent and technique for each case, and the apparatus is too expensive for occasional use. Consequently his technique should be limited to 'open' ether and chloroform or simple apparatus for the administration of nitrous oxide, oxygen and ether. He should use thiopentone only if he has a source of oxygen and some means of inflating the patient's lungs, which limits the use of that drug to practitioners with an anaesthetic apparatus. Spinal analgesia, the relaxant drugs, endotracheal anaesthesia and cyclopropane are beyond the scope of the general practitioner anaesthetist, and their use should be restricted to the specialist.

The position of analgesia in obstetrics as administered by the general practitioner is unsatisfactory. If he uses one of the many apparatus designed for this purpose, the manufacturer's instructions, in most cases, are his sole guidance in its use. It should be possible for him to take his apparatus to the maternity hospital in his area, and receive instruction in its use from one of the anaesthetists. For such an ideal state of affairs to exist the liaison between general practitioners and hospital staffs would require to be close, and much would depend on the individual

practitioner and anaesthetist.

Future general practitioners are the students of today and as such their training has been discussed previously.

In order that the anaesthetist may be able to give his cases adequate pre-operative and post-operative supervision, certain conditions must be satisfied. The cooperation of the surgeons and gynaecologists is essential, to allow the anaesthetist to enter their wards and order treatment. The relations between the anaesthetist and the junior members of the surgical staff, registrar and house-surgeons, must be good. The entry of the anaesthetist into the wards means yet another person from whom the ward sister receives instructions, and her co-operation is essential. The anaesthetist's commitments in theatre must allow adequate time for him to see cases before and after operation. If his appointment is full-time, for economic reasons he must spend every morning in theatre, and afternoons when required. Consequently he undertakes the pre-operative and post-operative supervision when his services are not required in the operating theatre. If both surgeons and anaesthetist have full-time appointments, the actual time spent in the operating theatre will increase, so that the anaesthetist may be in theatre morning and afternoon, and have no

opportunity to supervise pre-operative and post-operative care. A quarter of an hour is required for the adequate examination of a case requiring a major surgical operation and the writing of the anaesthetic record card and five minutes for each minor case. During an operating session three major and four minor surgical operations may be performed, requiring one hour and five minutes to be spent by the anaesthetist in pre-operative examinations. If a physician has to be consulted with regard to some co-existing pathology, the time allocated will require to be extended.

Cases which have undergone major surgical procedures should be seen by the anaesthetist daily for the first seven days and then every third day during the second week after the operation, and finally before their dismissal from the hospital. Minor surgical cases should be seen on the following day and before dismissal.

The anaesthetist must spend one half of each working day in the wards in order to carry out this programme. While he will administer fewer anaesthetics, he will become a more valuable member of the surgical team by the experience so obtained. It should be realised that the work of a good anaesthetist, like that of the surgeon, is not carried out solely in the operating theatre, but if his records and care of the patient are to be adequate, then much of his time should be spent in the wards looking after patients to whom

he will administer, or has administered an anaesthetic.

If the anaesthetist's appointment is in a part-time capacity, his afternoons will be occupied with work undertaken outside the hospital. It is unlikely that the authorities will pay for sessions which are not occupied in administering anaesthetics, and consequently if he is to undertake the supervision of cases before and after operations, it must be done in a voluntary capacity during any morning that is not considered one of his sessions at the hospital. It is unreasonable that the part-time anaesthetist should be penalised for attempting to make himself more efficient and more useful to the hospital.

As the number of full-time specialists increases, the number of operating sessions in each hospital will also increase. As theatre accommodation is limited, operations will have to be performed in the mornings and afternoons to the limit of the number of beds in the hospital and the extent to which the nursing staff is able to cope with acutely ill patients. At present a full-time anaesthetist is occupied in theatre during every morning and four afternoons of the week. If his appointment involves several of a group of hospitals, the greater will be the time actually spent in the operating theatre and the less available for work in the wards. This is further curtailed by one afternoon of each week

being set apart by the hospital authorities for the patients to be visited by their relatives and friends. The anaesthetist's night on duty for the treatment of emergency cases presents a special problem. If he is to see all the cases before operation, then accommodation must be available for him to spend such nights in the hospital. If, on such occasions he may be called to other hospitals for the same reason, it becomes impossible for him to see every case before operation, and reliance must be placed on the resident medical officer and the premedication ordered according to the picture of the case given by the latter during a telephone conversation. Other factors interfering with satisfactory pre-operative and post-operative care of the patients in the wards are the teaching of trainee specialists and medical students, lectures to the nursing staff, and if the hospital has an obstetrical unit, the instruction of midwives in analgesia.

There is, therefore a good reason to suggest that the appointment of anaesthetists should be in a full-time capacity, in their interest and for the sake of an efficient anaesthetic service in the hospitals. Each hospital group should have a pool of anaesthetists, with a minimum of one anaesthetist additional to the number required for routine operating sessions. His time might be fully employed in coping

with emergencies occurring during routine sessions, and replacing colleagues on vacation, study leave, or who are ill.

The question arises as to what should be done at the anaesthetist's visit to the patient before the operation. The case-record should be examined for facts relevant to the anaesthetic and operation, and additional information obtained by questioning the patient. That auscultation of the patient's heart reveals the presence of an uncondacted apical systolic murmur may be of interest, but the patient's response to exercise and breath-holding are of paramount importance. The state of the mouth should be examined and the presence or absence of an upper respiratory tract infection looked for as these examinations are usually performed in a perfunctory manner by the house-surgeon. The presence of veins in the patient's arm suitable for intravenous infusions and the use of intravenous thiopentone for the induction of the anaesthesia. The importance of any concurrent pathology of special interest to the anaesthetist should be assessed, and also the patient's mental make-up and reaction to the operation. At the same time an attempt should be made to gain the confidence of the patient. The drugs to be used in premedication, and the dose to be employed should be noted on the case-record, and the ward-sister informed of this fact.

The choice of the agents and technique of anaesthesia should also be decided and noted on the anaesthetic record card. If necessary, the aid of a physician or the surgeon should be obtained for the treatment or controlling before operation of any outstanding pathology such as diabetes mellitus. The ward-sister should be informed of any additions or alterations to the routine post-operative treatment. In emergency cases, especially in the obstetrical unit, the fact that the patient's stomach is empty should be confirmed, and if otherwise, steps taken to have the organ emptied. Arrangements should be made for physiotherapy. In the present series of cases pre-operative breathing exercises were only given when specially ordered, or for cases undergoing extensive operations such as partial gastrectomy. Post-operative physiotherapy was given to all surgical cases who had had major operations from the first day after the operation to the fourteenth, and in the obstetrical and gynaecological wards from the first day until the patient was discharged. The time required for this may be relatively short in cases such as appendicectomy, but of a considerable extent in the case of patients undergoing the operation of thymectomy where pre-operative preparations and post-operative care are of supreme importance.

From the point of view of the anaesthetist the

problem of adequate post-operative care depends on the time available and the extent to which the anaesthetist is permitted to take part by the surgeon. It should begin while the patient is still on the operating table after the completion of the operation with tracheo-bronchial suction or bronchoscopy as required. The anaesthetist should have slips of paper in the container for his record cards on which instructions may be sent to the ward regarding inhalational therapy, intravenous therapy, and position of the patient in bed. For convenience, a note may be made on this paper of the fluid administered in theatre for entry on the ward-sister's fluid-balance charts. The last blood-pressure, pulse and respiration rates are also included so that any sudden deterioration in the patient's general condition during the journey back to bed may be recognised.

Modern hospital architecture with central blocks of operating theatres has the disadvantage that patients have a longer journey back to bed with the risk of becoming chilled, vomiting, or displacing needles in position for intravenous therapy. It is impossible for the anaesthetist to accompany each case on the journey, and the responsibility should be delegated to some responsible person, and not to a hospital porter. The question arises as to whether a patient may be permitted to leave the operating theatre without

recovering the activity of the pharyngeal reflex, or whether he should be induced to vomit while relatively safe in the care of the anaesthetist with suction apparatus at hand.

After the patient's return to the ward post-operative care passes into the hands of the nursing staff. In these times most wards are staffed by an insufficient number of nurses, and consequently the post-operative care should be routine in as many cases as possible with additions in special cases. Contact must be made with the physiotherapist if her treatment is to be other than routine.

The role of the house-surgeon in post-operative care will depend on the experience he has acquired. The control of the blood chemistry requires considerable experience and must evolve on the surgical registrar or the anaesthetist. Similarly the examination of the patient's respiratory system requires experience to correlate the pre-operative and post-operative findings and decide when active treatment as opposed to routine prophylaxis is required. This is a further reason why the anaesthetist should be qualified to act as physician-anaesthetist. Similarly the control of pre-operative blood-transfusions in cases of haemorrhage might be delegated to the anaesthetist, who, in co-operation with the surgeon, will decide when to operate, and the former will then take over control of the infusion

or transfusion during the operation and regulate it according to his recording of pulse and respiration rates and the blood pressure. For continuity of control, he should continue this supervision in the post-operative period.

All this requires a considerable knowledge on the part of the anaesthetist which is not strictly anaesthesia. Previous generations of anaesthetists took no great part in this supervision and the present are gaining the necessary experience, which can only be done by spending much of their time in the wards. The anaesthetist might see cases requiring advice from a physician along with the latter in order to improve his knowledge of general medicine. The preparation of cases like diabetes mellitus should be left to the physician, and the anaesthetist take over when the patient is ready for the operation.

It is suggested that in order to improve the pre-operative and post-operative supervision of the patients, the anaesthetist must play his part. He must have a greater knowledge of clinical medicine than customary at present, he must be prepared to spend part of his working day in the wards and he should be encouraged to do so by the hospital authorities.

The training of nurses in anaesthesia is a recent development in Britain. It is difficult to know how much

to teach them, and their curriculum is already extensive. The training might be part theoretical and part practical and include pre-operative care, premedication and her part in preparing the patient for operation, her duties as anaesthetic nurse in the operating theatre, post-operative care, and a little about anaesthesia.

It is much more important that a nurse should know how to prepare a case for operation, both physically and mentally, than that she should know the properties of the drugs used in anaesthesia. Although a sympathetic approach to a patient is inherent in a good nurse and cannot be taught to a poor one, the lectures might include brief notes on the common attitudes of patients to operation and anaesthesia. She should be fully conversant with the physical preparation of patients for operation and the reasons for such care. The reason for "premedication" should be explained, and the doses and methods of administration of the drugs most frequently used, and the danger of leaving drinking water within the reach of a patient feeling the effects of the scopolamine or atropine stressed to her. The necessity for the signing of the slip giving permission for the operation and anaesthetic by the patient or guardian as soon as possible should be explained. She should be given the picture of what might result from leaving a denture in the

patient's mouth especially a small one in a position that may not be seen by the anaesthetist.

Whenever the anaesthetic nurse is replaced, the newcomer should be shown the apparatus on the anaesthetic trolley, airways, gags, forceps, and laryngoscope, which she may be ordered to bring in an emergency. The information she received in the lectures on anaesthesia on the cleaning of face-pieces, airways, endotracheal tubes, their connections, and laryngoscopes should be repeated. She should be shown the equipment required for intravenous anaesthesia, for basal narcosis by rectal instillation of bromethol, paraldehyde and thiopentone, and for local, regional and spinal analgesia. The necessity for absolute sterility of the instruments and drugs used in spinal analgesia should be stressed as strongly as possible. She should be shown the drugs usually used for surface analgesia, regional and spinal analgesia respectively in an attempt to reduce the possibility of her supplying the anaesthetist with the wrong drug. The modern method of autoclaving syringes and needles should be demonstrated by members of the nursing staff permanently attached to theatre. In the anaesthetic room the anaesthetist should explain her duties, and also what she must not do, the importance of the correct transfer of patient from trolley to, and, from the actual

operating table. The correct positioning of the patient on the table, the fixing of his hands and the insertion of an arm board and preparation of apparatus for intravenous infusion might be included in her training and responsibility.

Post-operative care is the most important section of the nurse's training in anaesthesia. The nurse should be taught how to maintain the patient's airway and the methods of treating obstruction due to the tongue falling back. She should learn to differentiate between efforts at respiration and the actual occurrence of free ingress and egress of air in the patient's respiratory passages. She should be taught the correct position of the patient in bed while recovering from the anaesthetic, and that it is her duty to remain at the bedside till consciousness has returned; modern methods have reduced this recovery period considerably. The importance of frequent changing of the patient's position should be stressed. Oxygen therapy is, on the whole, carried out inefficiently in the wards, and the responsibility for this lies with the medical staff. The reasons for oxygen therapy might be described briefly, and the various apparatus demonstrated, especially the rate of flow of the oxygen. The use of carbon dioxide inhalations in the prophylaxis of respiratory and circulatory system complications should be explained. She should be

instructed in the administration of fluid and in particular the care of intravenous infusions and blood transfusions, and the necessity for noting the fluid intake and output. The reason for the charting of the blood-pressure, temperature, pulse and respiration rates should be explained, also the method of estimating the blood-pressure. Many nurses do not appreciate the reason for these charts, but if this is made clear to them their interest is aroused, and the recording improved. They should be informed of the reason for the special care required after spinal analgesia, and the fact stressed that although the patient may be conscious he is liable to severe burns if hot-water bottles are left in his bed. The importance of the nursing care in the post-operative period is so great that no effort should be spared in order to improve its efficiency.

The lecture or lectures on anaesthesia proper should be brief. A short account might be given of the anaesthetic agents in general use, and the benefits of modern anaesthetic techniques such as the use of a light general anaesthesia and curare, and endotracheal anaesthesia explained. The complications of anaesthesia, in the induction maintenance, recovery, and post-operative periods might be taught in slightly more detail, and the part played by the nurse in their prevention and treatment given in detail. This

lecture should be given with the purpose of rousing the nurse's interest as it is of relatively little importance to the nurse in comparison with the remainder of her training in anaesthesia which has been given in order to increase the standard of nursing in the hospital, and so decrease the morbidity and mortality associated with anaesthesia.

Research and investigation of new methods of anaesthesia must now be considered. The search for the perfect anaesthetic continues, but in the meantime anaesthetists must become more efficient in the use of the drugs and techniques at their disposal. Few would claim that they are as expert with all the types of modern anaesthesia as the older anaesthetist was with his chloroform and ether. Consequently, one of the most important factors in improving the results of anaesthesia would be an increase in the expertness of anaesthetists with the modern drugs and techniques.

The question arises as to who is to carry on the research. The Faculty of Anaesthesia and the Medical Research Council should have a list of all anaesthetists who are willing and have the time to give potential anaesthetic agents a clinical trial. This list should be comprised of the names of anaesthetists who are members or fellows of the Faculty, with approximately ten years' experience of

anaesthesia. It should include the conditions under which the anaesthetist would employ the drug, in abdominal, neurological, thoracic or gynaecological surgery, in obstetrics, paediatrics or geriatrics. When the experimental pharmacologists have decided that a new drug is suitable for clinical trial, a committee set up by the two bodies should draw up a scheme covering the use of the drug in all types of anaesthesia. The list of anaesthetists is then consulted, and a certain number, all in different parts of the country, are chosen to investigate the drug clinically. They are then sent detailed instructions of the investigation they are to perform, including the types of cases to be used, abdominal, thoracic, obstetrical, or in the out-patient department, and a time limit by which their results must be returned. When all the results of the clinical trials have been examined, the committee must make two decisions, whether the drug has any advantage over another already in use, and whether the drug is safe for general use. The investigators would be permitted to embody their results in published articles. If any anaesthetist fails to carry out his instructions to the satisfaction of the committee, his name should be erased from the list. If the investigation was carried out in a satisfactory manner, the anaesthetist's name should be retained, but at the end of the

list, so that eventually he would be invited to perform a further investigation. Such a scheme would have two advantages, the Faculty of Anaesthetists and the Medical Research Council would have a panel of anaesthetists willing to take part in their investigations, and the individual anaesthetist would have the benefit of doing some research work under the direction of experts, and obtaining the basis of a valuable publication. The patients would also benefit, for if a definitely unfavourable report was published, it would not be economic for the manufacturers to continue its manufacture.

At present thousands of anaesthetics each year are wasted for research purposes. No two anaesthetists work by identical methods. The statistics of all the anaesthetists in the country accurately recorded would provide much useful information regarding the drugs and techniques used in modern anaesthesia. Good records would be absolutely essential. Holerith and similar systems are too complicated and require special apparatus for "sorting". A card on the Cope-Chatterton "key-sort" principle, similar to that designed by Nosworthy without its obvious statistical errors, would be ideal.

The present investigation has shown a high incidence of post-operative complications involving the respiratory

system associated with modern anaesthesia in spite of physiotherapy, post-operative inhalational therapy and tracheo-bronchial toilet when required, and therefore some effective prophylaxis must be found. A relation between post-operative vomiting and respiratory complications has not been proved statistically, but there is enough evidence to warrant further investigation. The present series of cases is relatively small, but the results obtained from a very large series might show whether the decrease in vomiting associated with modern anaesthesia is a factor in post-operative respiratory complications. The use of pethedine in premedication was found to be accompanied by a high rate of less than twelve hours' vomiting in the post-operative period, and by a relatively low rate of respiratory complications. It is therefore suggested that the use of this drug for this purpose be considered.

Summary and Conclusions.

The improvement in the preparation of the patient before operation with a view to restoring his general condition to normal, the use of less toxic anaesthetic drugs requiring more complex techniques and apparatus, and the development of post-operative care have been described. The alteration in the position of the anaesthetist in the surgical team and in his work has been discussed. The reasons for the investigation and a description of the manner in which it has been carried out have been given.

A brief history of anaesthesia during the past fifty years shows such an increase in the scope of the subject that it now justifies the title of specialty. Great interest has been taken in the relief of the pain of labour, culminating, on the one hand, in a simple analgesic apparatus for use by midwives, and, on the other, the highly specialised continuous caudal analgesia for hospital use only. The use of anaesthesia in diagnosis and therapeutics has increased. Anaesthetic apparatus has become more complicated, more efficient, and with the advent of the closed circuit technique, less wasteful. New anaesthetic drugs have been discovered, some for inhalation anaesthesia, others for local and spinal analgesia, and for the first time, an efficient means of producing anaesthesia by intravenous injection.

The techniques employed have become more complicated. Endotracheal anaesthesia has become firmly established in everyday use. The advent of curare has resulted in a decrease in the use of regional and spinal analgesia, the employment of which had been increasing up to that time. Statistics associated with anaesthesia have shown that there has been no apparent improvement in the results of anaesthesia during this century.

The third section of the thesis has been concerned with the specific dangers of modern anaesthetics. The tendency has been to make anaesthesia more pleasant for the patient, but the use of the new agents and techniques has brought new complications and increased liability to others. The use of cyclopropane, with its depression of respiration, carbon dioxide retention, and blood pressure maintenance, has been associated with difficulty in estimating the condition of the patient under its influence. It has been shown to upset, in some cases gravely, the automaticity of the heart. The pleasant and time-saving induction of anaesthesia by the intravenous injection of thiopentone has been counteracted by the difficulty of estimating the dose of this drug so fatally easy to administer, especially to the patient with hypertension and arteriosclerosis, due to its aggravation of any pre-existing anoxia and rendering hyperactive the laryngeal and tracheal reflexes. The

perfect relaxation of spinal analgesia has been shown to be obtained at the cost of terrible results if any fault be present in the aseptic technique of the anaesthetist.

Palsies affecting the cranial and lumbo-sacral spinal nerves have been shown to occur following spinal analgesia with no apparent reason for doing so. Alterations in the composition of the cerebro-spinal fluid, and pathological changes in the meninges and spinal cord indicated that the injection of the analgesic drug was not to be undertaken needlessly, and trauma to the spinal column that lumbar puncture was not free from risk. The paralysis of the peripheral circulatory system in the area of analgesia was shown to have a profound effect on the blood pressure which in turn was influenced by the paresis of the muscles of respiration. The combined effect was shown to increase the danger from haemorrhage, especially in the operation of Caesarian Section. Caudal block, with special reference to analgesia in child-birth, was discussed in view of the risk of sepsis, the alteration in the mechanics of labour and hypoxia affecting the infant secondary to hypotension in the mother. The administration of di-vinyl ether has been shown to result in an anaesthesia, the depth of which is difficult to estimate, and in which there is an increased tendency to convulsions and to liver necrosis. The danger of administering Trilene in a closed-

circuit apparatus has been discussed. Its action on the nervous control of respiration and the cardiac rhythm has been described. The value of the drug as an analgesic in labour has been discussed in view of recent experiments on pregnant animals. Paresis of the muscles of respiration and difficulty in estimating the depth of the associated anaesthesia have been shown to follow the use of curare. Relaxation of the cardia and reduction of intestinal tone have been found to occur after the injection of the drug. The advantage of the relative absence of paresis of the muscles of respiration with Myanesin has been annulled by the haemolysis of the erythrocytes, the haemoglobinuria and albuminuria liable to follow the injection of the drug, and the thrombophlebitis of the vein into which the injection has been made. The relationship between post-operative respiratory complications and the profound relaxation produced by these drugs has been discussed. The causation and prevention of anaesthetic explosions have been discussed and the dangers of performing endoscopic examinations of the upper respiratory tract under anaesthesia with explosive volatile drugs have been emphasised. The effect of oxygen in increasing the risk and force of the explosion and the new danger in Britain of static electricity in air-conditioned theatres have been described. It has been emphasised that

almost all explosions are preventable. It has been shown that the danger of the patient inhaling vomited material has been increased by modern anaesthesia, especially during induction, resulting in laryngeal spasm, death by drowning, or a septic pneumonia. The special liability of certain cases to this complication during anaesthesia, and the fact that gastric lavage and suction during the anaesthesia do not give complete immunity have been discussed. The liability of laryngoscopy and intubation of the larynx to carry infection and foreign matter into the trachea, and to traumatise the mouth, pharynx, larynx and trachea was described. The use of too large or too small endotracheal tubes was shown to be of great importance, especially in young patients.

The fourth section has been comprised of a discussion of the mortality and morbidity associated with anaesthesia. A study has been made of the statistics of mortality associated with anaesthesia. It was found that there has been an increase in the number of deaths associated with anaesthesia. Several reasons for this have been suggested, especially the increase in the scope of surgery, and the fact that many more anaesthetics are administered today than fifty years ago. A comparison has been made between the surgery of the beginning of the century and that of today, and errors in the

statistics associated with the former have been noted. The statistics of the Registrar General of deaths associated with anaesthesia have been shown to be incomplete. The mortality rates given in various published articles have been shown to be more exact and to allow of comparison to a certain extent. Statistics of the morbidity associated with anaesthesia have been studied, and an increase has been found to have occurred during the twentieth century. Reasons for this have been suggested, especially the follow-up of their cases by anaesthetists during recent years. A synopsis has been made of all the cases in the present series which ended fatally. A comparison has been made between the drugs and techniques long established in the practice of anaesthesia and those recently introduced with regard to post-operative complications and vomiting. A similar comparison has been made between the various drugs used for premedication. The incidence of post-operative respiratory complications has been compared with the duration of post-operative vomiting. Comparisons have been made of the alterations in blood-pressure during and after the operation of partial gastrectomy performed under light general anaesthesia and curare, fractional spinal analgesia, and single dose spinal analgesia. The dangers of the administration of the new potent anaesthetic drugs by the unskilled anaesthetist have been discussed.

This investigation has shown that there is little difference between the morbidity associated with the older and the new anaesthetic drugs and techniques, and that any improvement must come from greater efficiency on the part of the anaesthetist. It has been stressed that the anaesthesia is not the sole factor in the causation of post-operative complications. It has been suggested that in order to improve the practice of anaesthesia, teaching must be improved. With regard to the teaching of students, the scope of the training has been discussed. A scheme of instruction to fit the student for general practice or the duties of a house-surgeon has been described. The teaching of the future specialist anaesthetist has been shown to depend on the co-operation of the surgeon, and to involve the departments of anaesthesia, anatomy, physiology, haematology, biochemistry and psychiatry. It has been suggested that trainees be appointed to a group of hospitals to enable the instruction to include anaesthesia for obstetrics, thoracic and neurological surgery. The responsibility of newly-appointed house-surgeons for deaths associated with anaesthesia has been noted. The anaesthetics that the house-surgeon may be called upon to administer have been described, and a scheme of instruction has been suggested to enable him to fulfil these requirements. It has been

stressed that the house-surgeon be taught to realise when an anaesthetic is beyond his capabilities and that strict limitations be imposed on him regarding the anaesthetics that he may administer. The anaesthetics which the general practitioner may be expected to administer have been described; the problem of analgesia in labour in domestic midwifery has been discussed, and suggestions made for its improvement. The problem of adequate pre-operative and post-operative care on the part of the anaesthetist has been shown to depend on the co-operation of the surgeon, and on the time available for the anaesthetist to carry out this care. The value of an anaesthetist who is also a good clinician has been stressed. The difficulties involved in adequate pre-operative supervision, especially of emergency cases, have been described. Suggestions have been made regarding the form of this pre-operative supervision. The extent of the post-operative care has been shown to depend on the surgeon. It has been suggested that it commence while the patient is still on the operating table, and that accurate messages regarding the condition of the patient be sent back to the ward. The problem of transporting the patient back to his bed, and the influence of the present shortage of nurses on post-operative treatment have been described. The position of the house-surgeon in the post-operative care has been

discussed and reasons given for the anaesthetist taking part. The instruction of nurses in anaesthesia has been discussed with reference to pre-operative nursing care and premedication, the duties of the anaesthetics nurse, and the post-operative nursing care of the patient. In the duties of the anaesthetic's nurse have been included the knowledge of the instruments that may be required in an emergency, the cleaning of apparatus such as face-pieces and artificial airways, the autoclaving of syringes and needles, and the positioning of the patient on the operating table. It has been suggested that in the training in post-operative nursing care emphasis should be placed on the maintenance of the patient's airway, the position of the patient in bed, the prophylaxis of complications affecting the respiratory and cardiovascular systems, and the care and maintenance of inhalation and intravenous therapy. It has been stressed that no attempt should be made in any lectures to teach nurses anaesthesia proper. A scheme for the clinical investigation of new drugs, its advantages for anaesthetists and patients, and the greater use of anaesthetic records have been suggested. This investigation has shown a high rate of post-operative complications affecting the respiratory system despite the use of modern prophylactic measures. Suggestions have been made for the utilisation of post-operative vomiting as prophylaxis in view of the possible relationship between

slight post-operative vomiting and respiratory complications.

Conclusions.

1. The new anaesthetic drugs and techniques are no more liable to be followed by post-operative complications than the older drugs and techniques.
2. Vomiting is less liable to follow the new drugs and techniques.
3. The older drugs and techniques are associated with a higher rate of excessive vomiting.
4. The use of Pethedine in premedication is associated with the lowest rate of post-operative complications.
5. The absence of post-operative vomiting is most frequent after the use of barbiturates given by mouth as premedication.
6. Post-operative vomiting of under twelve hours' duration may have some value in the prophylaxis of post-operative respiratory complications.
7. There is no special liability to a marked fall in the level of the blood-pressure during the first few hours after the operation following the use of curare.

Bibliography.

1. Editorial - "Anaesthesia", July 1947.
2. Copeland - Chatterton Paramount Card-Anaesthetic Record
Explanatory Leaflet.
3. Hewer - Recent Advances in Anaesthesia and Analgesia -
Vth Edition (Churchill), London.
4. Adriani, J. - Chemistry of Anaesthesia - 1946 (Oxford),
page 125.
5. Lancet, 16th November, 1935, page 1110.
6. The Anaesthetic Properties of Certain Unsaturated Ethers -
Leake, C.D., and Chen, Mei-Yu, Proceedings of the Society
for Experimental Biology and Medicine, November 1930, p.151.
7. The Anaesthetic Action of Di-vinyl Oxide on Humans:
Gelfan, S., and Bell, I.R. The Journal of Pharmacology
and Experimental Therapeutics: Vol.XLVII, January 1933,
No.1, page 1.
8. Bourne, W., McDowell, J.F., and White, J.C. (1937),
"Further Studies in Vinyl Ether (Vinesthene)
Obstetrical Anaesthesia: Mixtures with Ethyl Ether:
Effect on Coagulation Time of Blood": Curr. Researches
Anaesth. 16: 46.
9. Stricker, Goldblatt, Warm and Jackson: Curr. Researches
in Anaesth: 14: 68.

10. Hewer, C.Langton and Hadfield, C.F. - Trichlorethylene as an Inhalation Anaesthetic, British Medical Journal (1941), 1; 924.
11. Hewer, Langton C. - Trichlorethylene as a General Analgesic and Anaesthetic. Proc.Royal Society Med. (1942), 35: 463.
12. Hewer, C.Langton - Further Observations on Trichlorethylene. Proc.Royal Soc. Med. (1943), 36: 463.
13. Hewer, C.Langton - Brit.Med.Bulletin: Vol.4; No.2. 1946: page 108.
14. Lee, F.S. - Rep. New York State Comm. on Ventilation, 1923.
15. Kinetic theory of Shock - Crile, G.: Lancet 5th July 1943
16. Surgical Shock: Crile & Lower: Philadelphia, 1920.
17. A milestone in Anaesthesia ? - Gray, T.C. & Halton, John: Proc.Roy.Soc.Med.: (Section on Anaesthetics) - 34: 400-410: (May) 1946).
18. Allen, F.M. et al.: Jour.Internat.Coll.Surg., 1942, March - April, p.125.
19. James, N.R. - Regional Analgesia for Intra-abdominal Surgery, 1943 (London).
20. Griffiths, H.W.C. & Gillies, John - Thoraco-lumbar Splanchnicectomy and Sympathectomy : Anaesthetic Procedure Anaesthesia - Vol.3, p.134.
21. Editorial: Anaesthesia, Vol.3, No.1.

22. Elam, John: Present Position of Ether Anaesthesia:
Post-Graduate Medical Journal, October 1946.
23. Report by Registrar General to Parliament (1901) for
England and Wales.
24. King, Donald S.: Post-Operative Pulmonary Complications:
Pre-Operative and Post-Operative Care, edited by Mason,
R.L. & Zintel, H.A. (1947) : Philadelphia.
25. Guedel, A.E. : Inhalation Anaesthesia: 1937 (New York).
26. Boyd, John : Cyclopropane Anaesthesia : Medical Press :
216: 469-470 (December 18) 1946.
27. Waters, R.M.: Cyclopropane - A personal evaluation :
Surgery : 18 : 26: (July) 1945.
28. Nosworthy, M.: Some Respiratory Disturbances during
General Anaesthesia : Anaesthesia: 3: 86 (July) 1948.
29. Enderby, G.E.H.: Choice of Anaesthetic - A comparison of
Anaesthetic Agents: Anaesthesia: 2: 138 (October) 1947.
30. Dripps, R.: The Immediate Decrease in Blood Pressure seen
at the conclusion of Cyclopropane Anaesthesia: Cyclopropane
Shock: Anesthesiology: 8: 15-34: 1947.
31. Minnit, R.J. & Gillies, John: Textbook of Anaesthetics :
(Edinburgh) 1944.
32. Stormont, R.T., Hathaway, H.R., Shideman, F.E. and Seevers,
M.H. : The Acid-Base Balance during Cyclopropane Anaes-
thesia: Anesthesiology: 3: 369-378 (July) 1942.

33. Adriani, John: The Pharmacology of Anaesthetic Drugs: (Illinois) 1942.
34. Volpitto, P.P., Woodbury, R.A. & Hamilton, W.F.: Direct Arterial and Venous Pressure Measurements in Man as Affected by Anaesthesia, Operation and Shock: American Journal of Physiology: 128: 238-246 (January) 1940.
35. Goldstein, J.D. & Dubois, E.L.: The Effect on the Circulation of Man of Rebreathing Different Concentrations of Carbon Dioxide: American Journal of Physiology: 81: 650-660: (August) 1927.
36. Waters, R.M.: Toxic Effects of Carbon Dioxide: New Orleans Medical and Surgical Journal: 90: 219-224: (October) 1937.
37. Waters, R.M.: Newer Viewpoints on Clinical Anesthesia: Federation Proceedings: 1: 213-218 (June) 1942.
38. Robins, B.H. & Baxter, J.H.: Studies of Cyclopropane IV, The Cardiac Output in Dogs under Cyclopropane: Journal of Pharmacology and Experimental Therapeutics: 62: 179-188 (August) 1938.
39. Beecher, H.K.: Physiology of Anaesthesia: (New York and London) 1938.
40. Stutzman, J.W., Quill Murphy, Allen, C.R. & Meek, W.J.: Further Studies on the Production of Cyclo-Epinephrine Tachycardia: Anesthesiology: 8: 579-583 (November) 1947.
41. Adriani, John: The Chemistry of Anaesthesia (Oxford),

1946, p.269.

42. Lundy, John S.: Clinical Anaesthesia (Philadelphia)
1943: pp.433-439.
43. Boyd, John: Cyclopropane Anaesthesia: Ulster Medical
Journal: 15: 58-77: (May) 1946..
44. Adriani, John: Techniques and Procedures of Anaesthesia:
(Illinois) 1947, p.146.
45. Anderson, A.W.: Caesarian Section: Canadian Medical
Association Journal: 56: 170-177: (February) 1947.
46. Mousel, L.W. & Weiss, A.W.: Comments on Anesthesia:
The S.Clin.North America, Nationwide Number: 1072-1082
(October) 1945.
47. Pratt, T.M., Tatum, A.L., Hathaway, H.R. & Waters, R.M.:
Sodium Ethyl (1-Methyl Butyl) Thiobarbiturate, Prelimin-
ary Experimental and Clinical Study: American Journal
of Surgery: 31: 464: (March) 1936.
48. McCarthy, K.C.: War Advances Anaesthesia: Ohio State
Medical Journal: 42: 1150-1153: (November) 1946.
49. Wainwright, C.A.: Experiences with Pentothal during the
first hundred days following the Normandy invasion:
Canadian Medical Association Journal: 52: 484-488:
(May) 1945.
50. Hunter, A.R.: Pentothal Sodium Anaesthesia: Anaesthesia:
3: 116 (July) 1948.

51. Barton, G.D., Wicks, W.R., and Livingstone, H.M.: Effects of Pentothal on Arterial Blood Gases: *Anesthesiology*: 7: 505-517: (September) 1946.
52. Allen, C.R., Echols, R.S., Hoeflick, E.A., O'Neal, K.C., & Slocum, H.C.: Variations in the Signs of Acute Oxygen Want during Anesthesia: *Anesthesiology*: 8: 601-614: (November) 1947.
53. Parry-Price, H.: Practical Anaesthetics: (Bristol) 1946.
54. Booker, W.M.: Observations on the Carbohydrate Metabolism During Prolonged Pentothal Anaesthesia in Dogs.
I. The Blood Sugar and Liver Glycogen: *Anesthesiology*: 7: 405-415: (July) 1946.
55. Heard: Canadian Medical Association Journal: 1936: August: p.628.
56. Bodman, R.I. & Farr, A.: Convulsions under Thiopentone: British Medical Association: 2nd August: 1947: p.175.
57. Wynne, R.L.: Convulsions under Thiopentone: British Medical Journal: 10th January 1948, p.48.
58. Jarman, R.: History of Intravenous Anaesthesia with Ten Years Experience of the Use of Pentothal Sodium: Post-Graduate Medical Journal: October 1946, 311-318.
59. Lundy, John S.: *Annals of Surgery*: 1939: November 5th: p.878.
60. Macintosh, R.R. & Heyworth, P.S.A.: *Lancet*: 2: 571: 1943.

61. South African Medical Journal 1936; November 14th.
62. Crankshaw, T.P. & Kaye, G.: Spinal Analgesia in a Metropolitan Public Hospital 1937-1946: Anaesthesia: 2, 127: (October) 1947.
63. Barrie, H.J.: Lancet, 1941, February 22nd, p.242.
64. Evans, F.T.: Sepsis and Asepsis in Spinal Analgesia: Proceedings of the Royal Society of Medicine: 39: 181: (February) 1946.
65. Kremer, M.: Meningitis after Spinal Analgesia: British Medical Journal: 2: 309-313 (September) 1945.
66. Garrod, L.P.: The Nature of Meningitis following Spinal Anaesthesia and its Consequences: British Medical Bulletin: 4: 106-108: 1946.
67. Arrowood, J.G. & Foldes, F.F.: The Management of Sub-arachnoid analgesia by the continuous drop method: British Journal of Anaesthesia: 20: 60-71: (July) 1946.
68. Heard, K.M.: Anesthesia and Analgesia: 1938: May-June, p.124.
69. Yaskin, J.C.: Archives of Ophthalmology 21: 1010: 1939.
70. Fairclough, W.A.: Sixth-nerve paralysis after Spinal Analgesia: British Medical Journal: 801-803 (December 8) 1945.
71. Nicholson, M.J. & Eversole, U.H.: Neurological Complications of Spinal Anesthesia: Journal of American Medical Association: 132: 679-685 (November 23) 1946.

72. Yaskine, H.E. & Alpers, B.J.: Neuropsychiatric Complications following Spinal Anesthesia: *Annals of Internal Medicine*: 23: 184-200 (August) 1945.
73. Kellum, J.M.: Spinal Anesthesia, Survey for 1945: *Journal of the Medical Association of Georgia* 36: 165-168: (April) 1947.
74. Ferguson, F.R.: Proceedings of the Royal Society of Medicine: 1937: March 18.
75. Rieser, C.: *Journal of the American Medical Association*: 1941: July 12th, p.98.
76. Conn, J.E. & Wycoff, C.C.: Continuous Spinal Anesthesia: A modification of the Ureteral Catheter Technique: *Anesthesiology*: 91: 288-295 (May) 1948.
77. Frankson, C. & Gordk, T.: Headache after Spinal Anaesthesia and a Technique for Lessening its Frequency: *Acta. Chir.Scandinav.*: 94: 443-454: (September 10th) 1941.
78. Grondahl, N.B.: *Acta.Chir.Scandinav.*: 1932: December, p.51.
79. Resnick, Louis: Heavy Nupercaine Spinal Analgesia in Operative Obstetrics with a report on 394 cases: *British Medical Journal*: November 24th: 1945: p.24.
80. Lund, P.C. & Rumball, A.C.: Hypobaric Pontocaine Spinal Anesthesia; 1640 Consecutive Cases: *Anesthesiology*: 8: 188-199 (March) 1947.
81. Lewis, D.L. & Palser, E.G.M.: *British Medical Journal*: June 4th: 1938: p.1202.

82. Smith, F.A.: Unusual Incidents following Spinal Anesthesia, a report of two cases: *Anesthesiology*: 7: 419: (July) 1946.
83. Belinkoff, S.: Coma during and following Spinal Anesthesia: *Annals of Surgery*: 122: 278-286 (August) 1945.
84. Black, M.G.: Spinal Fluid Findings in Spinal Anesthesia: *Anesthesiology*: 8: 382-389: (July) 1947.
85. Lienhoof, F: Schmerz, Larkose v. Anaes., 1936, December: p.145.
86. MacDonald, A.D. & Watkins, K.H.: *British Journal of Surgery*: 25: 879: 1938.
87. Lundy, J.S., Essex, H.E. & Kernochan, J.W.: *Journal of the American Medical Association*: 101: 1546: 1933.
88. Brain, W.R. & Russell, D.: *Proceedings of the Royal Society of Medicine*: March 18th: 1937.
89. Harris, T.A.B.: *British Journal of Anaesthesia*: 1939: (July): p.131.
90. Pitkin, G.: *Anesthesia and Analgesia*: 1940 (September - October), p.241.
91. Harris, T.A.B. & Rink, E.H.: *Guy's Hospital Report* 1937; January.
92. Cooper, W.G. & Zumwalt, W. & Sugarbaker, E.D.: A Limited Comparison of Continuous Spinal and General Ether Anesthesia: *Surgery*: 16: 886-895: (December) 1944.

93. Howard-Jones, W.: Medical Press and Circular: 1934:
October 3rd.
94. Cotui, F.W.: Anesthesia and Analgesia: 1934: July -
August: p.144.
95. Jennings, W.K. & Karabim, J.E.: American Journal of Sur-
gery: 1939: November: p.317.
96. Macintosh, R.R.: British Medical Journal: 5th March 1949.
97. Milward, F.J. & Crant, J.L.A.: Lancet: 1936: July 25th:
p.133.
98. Bromley, L.L., Donaldson, C.J. & Lipman Kessel, A.W.:
Infected Intervertebral Disc after Lumbar Puncture;
British Medical Journal: 22nd January: 1949: pp.132-133.
99. Contempre, J. - Le Scalpel: 1934: June 16th, p.859.
100. Jost, T.A.: Anesthesia and Analgesia: 1935: July -
August: p.191.
101. Saunders, E.W.: Annals of Surgery: 1931: November: p.931.
102. Babcock, M.E.: Anesthesia and Analgesia: 1932: July: p.187
103. Veal, J.R. et al.: American Journal of Surgery: 1936:
December: p.106.
104. Newton, C.W. & Andros, G.J.: Continuous Caudal Analgesia
in Curettage for Abortion: American Journal of Obstetrics
and Gynecology: 50: 430 - 433: October: 1945.
105. Hingson, R.A., Edwards, W.B., Lull, C.B., Whitacre, F.E.,
and Franklin, H.C.: Newborn Mortality and Morbidity with

Continuous Caudal Analgesia; an Analysis of Cases in New York, Philadelphia and Memphis, with Controls: Journal of the American Medical Association: 136: 221-229: (January 24th) 1948.

106. August, R.V.: Continuous Caudal Analgesia: Journal of the Michigan State Medical Society: 44: 1341 - 1346: (December) 1945.
107. Nicodemus, R.E., Ritmiller Le R.F., & Ledden, L.J.: Continuous Caudal Analgesia in Obstetrics on Trial: American Journal of Obstetrics and Gynecology: 50: 312-318: (September) 1945.
108. Brown, H.O., Thomson, J.M. & Fitzgerald, J.E.: An Analysis of 500 Obstetrical Cases with Continuous Caudal Anesthesia, using Pentocaine: Anesthesiology: 7: 355-373: July: 1946.
109. Brown, W.W.: Meningitis following Continuous Caudal Anesthesia: American Journal of Obstetrics and Gynecology: 53: 682-683: April: 1947.
110. Hingson, R.A.: Contra-Indications and Cautions in the Use of Continuous Caudal Analgesia: American Journal of Obstetrics and Gynecology: 47: 718-721: November 5th: 1944.
111. Hawk, M.H., Orth, O.S. & Pohle, F.E.: Hepatorenal Syndrome following administration of Vinesthene; A case report: Anesthesiology, 2: No.4: July: 1941: p.388.

112. Lyons, S.E.: Vinesthene Burn: Journal of the American Medical Association: 1st October: 1938: p.1284.
113. Cartwright, F.F.: Vinesthene Anaesthetic Mixture: British Medical Journal: 1: 1081: 1939.
114. Dawkins, C.J.M.: Divinyl Ether Anaesthesia: British Medical Journal: 4th June 1938: p.1236.
115. Guerrier, S.M. & Dafoe, C.S.: Convulsions under Vinesthene Anaesthesia: British Medical Journal: 2nd March: 1940: p.366.
116. Boston, F.K.: Convulsions after Vinesthene Anaesthesia: British Medical Journal: 1: 929: 8th June: 1940.
117. Durrans, S.F.: Convulsions after Vinesthene Anaesthesia: British Medical Journal: 2: 228: 16th August: 1941.
118. Orth, O.S., Slocum, H.C., Stutzman, J.W. & Meek, W.J.: Studies of Vinesthene as an anaesthetic agent: Anesthesiology: 1: 246-260: November: 1940.
119. Goodman and Gillman: The Pharmacological Basis of Therapeutics: 1941: New York.
120. Goldschmidt, S., Ravdin, I.S. & Lucke, B.: Anesthesia and Liver Damage; Protective Action of Oxygen against Necrotising Effect of Certain Anesthetics on the Liver: Journal of Pharmacology: 59: 1: 1937.
121. Draper, W.B. & Whitehead, R.W.: Chances of Resuscitation after Overdose of Ether, Divinyl Ether and Chloroform: Lancet: 1: 442: 1942.

122. Gordon, R.A. & Shackleton, R.P.W.: Trichlorethylene in Plastic Surgery: British Medical Journal; 1: 380: 1943.
123. McClelland, M.: Trilene Toxicity, Some Toxic Effects following Trilene Decomposition Products: Proceedings of the Royal Society of Medicine: 37: 526: 1944.
124. Enderby, G.E.H.: Use and Abuse of Trichlorethylene: British Medical Journal; 2: 300: 1944.
125. Humphrey, J.H. & McClelland, M.: Cranial Nerve Palsies with Herpes following General Anaesthesia: British Medical Journal; 1: 315: 1944.
126. Carden, S.: Hazards in the use of the Closed-Circuit Technique for Trilene Anaesthesia: British Medical Journal; March 4th: 1944: p.319.
127. Culbert, T.D.: Convulsions under Trilene Anaesthesia: British Medical Journal; 2: 679: 1942.
128. Garland, Y.: Convulsions under Trilene Anaesthesia: British Medical Journal; 2: 607: 1942.
129. Condon, H.A.: Convulsions under Trilene Anaesthesia: British Medical Journal; 14th August 1948.
130. Hower, C.L.: Further Observations on Trilene: Proceedings of the Royal Society of Medicine: 36: 463: 1943.
131. Johnson, E.E.: Experience with 500 Trichlorethylene (Trilene) Anaesthetics at an E.M.S. Hospital: British Journal of Anaesthesia: 19: 71: 1944.

132. Culbert, T.D.: Trichlorethylene Anaesthesia: British Medical Journal: 1: 598: 1943.
133. Millar, R.A.: Trichlorethylene in General Anaesthesia: British Medical Journal: March 13th: 1948 (Correspondence) p.524.
134. Lloyd-Williams, K.G. & Hewspear, D.: Trichlorethylene as a General Anaesthetic: British Medical Journal: 2: 170: 1942.
135. Wagner, F.W.E.: Trichlorethylene Anaesthesia: Irish Journal of Medical Science: 6 series: 717-723: October: 1946.
136. Ostlere, G.: The Role of Trichlorethylene in General Anaesthesia: British Medical Journal: January 31st: 1948: p.195.
137. Ewing, J.B. & Britain, G.J.C.: Auricular Fibrillation after Trichlorethylene Anaesthesia: British Medical Journal: 20th November: 1948 (Correspondence).
138. Barnes, C.G. & Ives, J.: Electrocardiographic Changes during Trilene Anaesthesia: Proceedings of the Royal Society of Medicine: 37: 528: July: 1944.
139. Haworth, J. & Duff, A.: A Note of Trilene Anaesthesia: British Medical Journal: 1: 381: 1943.
140. Herdman, K.N.: Acute Yellow Atrophy of Liver following Trilene Anaesthesia: British Medical Journal: 2: 689-690: November 17: 1945.

141. Dodds, G.: British Medical Journal; 1: 769: 1945.
142. Armstrong, D.M.: The Assessment of Liver Damage following Trichlorethylene and Di-ethyl Ether Anaesthesia: Anaesthesia: 2: No.2: 45-50: April: 1947.
143. Helliwell, P.J. & Hutton, A.M.: Analgesia in Obstetrics: Anaesthesia: 4: No.1: 18-21: January: 1949.
144. Gray, T.C. & Halton, J.: A Milestone in Anaesthesia: Proceedings of the Royal Society of Medicine: 39: 400: (May) 1946.
145. Prescott, F., Organe, G. & Rowbotham, S.: Tubocurarine Chloride as an Adjunct in Anaesthesia: Lancet: 2: 80: 1946.
146. Harroun, P., Beckert, F.E. & Fisher, C.W.: The Physiologic Effects of Curare and its Use as an Adjunct to Anaesthesia: Surgery, Gynecology and Obstetrics: 84: 491-498: 1947.
147. Gross, E.G. & Cullen, S.C.: The Action of Curare on the Smooth Muscle of the Small Intestine and on the Blood-Pressure: Anesthesiology: 6: 231-238: 1945.
148. Adriani, J. & Ochsner, A.: Some Observations on the Use of Curare in the Treatment of Tetanus: Surgery: 22: 509-515: 1947.
149. Ostlere, G.: Use of Curare in Poor-Risk Patients: British Medical Journal: April 5th: 1947: p.448.
150. Anonymous: Curare in Anaesthesia: Lancet: 2: 81-82: 1945.
151. Comroe, J.H. & Dripps, R.D.: The Histamine-like Action

- of Curare and Tubocurarine Injected Intracutaneously and Intra-arterially in Man: *Anesthesiology*: 7: 260-262; 1946.
152. Knight, R.T.: Combined Use of Sodium Pentothal, Intocostrin, (Curare), Nitrous Oxide and Oxygen: *Canadian Medical Association Journal*: 55: 356-360: 1946.
153. Harroun, P., Beckert, F.E. & Hathaway, H.R.: Curare and Nitrous Oxide Anesthesia for Lengthy Operations: *Anesthesiology*: 7: 24: 1946.
154. Holaday, D.A.: Nitrous-Oxide-Cyclopropane-Curare Anesthesia, A Review of 200 cases: *Anesthesiology*: 7: 426-439: 1946.
155. Gray, T.C.: d-Tubocurarine in Caesarian Section: *British Medical Journal*: April 5th 1947.
156. Cole, Frank: A New Lethal Dose of Curare, with some Observations on the Pathology produced by Large Doses: *Anesthesiology*: 7: 190-197: 1946.
157. Gray, T.C. & Halton, J.: Idiosyncrasy to d-Tubocurarine Chloride: *British Medical Journal*: April 24th: 1948: p.784.
158. Griffith, H.R., Stephen, C.R., Cullen, W.G. & Bourne, W.: Myanesin as a Muscle-Relaxant: *Anesthesiology*: 10: 61-65: 1949.
159. Ballantine, R.I.W.: Myanesin - A Report of 76 Cases: *Anaesthesia*: 3: 20: 1948.

160. Musgrove, H.: Myanesin: Anaesthesia: 3: 168-172: 1948.
161. Stephen, C.R. & Chandy J.: Clinical and Experimental Studies with Myanesin: A preliminary report: Canadian Medical Association Journal: 57: 463-469 (November) 1947.
162. Hewer, T.F. & Woolmer, R.F.: Death from Renal Anoxia after Myanesin: Lancet 2: 909-910: 1947.
163. Wilson, H.B. & Gordon, H.E.: Myanesin as an Aid to Anaesthesia in Children: Lancet: 1: 367-368: (March) 1948
164. Pinkerton, H.H.: Anaesthesia for Urological Surgery: British Journal of Anaesthesia: 21: 78-94: (July) 1948.
165. Pinson, K.B.: British Medical Journal: 2: 312: 1930.
166. Woodbridge, P.D.: Journal of the American Medical Association: 1939: December 23rd: p.2308.
167. Hasler, J.K.: Risks of Explosion in Anaesthesia: Modern Anaesthetic Practice: 1941 (London).
168. Hewer, C.L.: Proceedings of the Royal Society of Medicine: 1930: April.
169. Medico-Legal: British Medical Journal: January 15th: 1949: p.117.
170. Ironside, R.: Proceedings of the Royal Society of Medicine: 1935: March 1st.
171. Chivers, E.H.: Lancet: 1943: April 24th: p.527.
172. Lundy, J.S.: Endotracheal or Intratracheal Anaesthesia: S.Clin. North America, Mayo Clinic Number: 795-833: August: 1945.

173. Donnelly, W.A., Grossman, A.A., & Green, F.M.: Local Sequelae of Endotracheal Anaesthesia, as Observed by Examination of One Hundred Patients: *Anesthesiology*: 9: 490-497: (September) 1948.
174. Shaw, W.M.: Oedema of the Larynx, A Rare Complication of Endotracheal Anesthesia: *Anesthesiology*: 7: 416-418: (July) 1946.
175. Barton, L.W.: Granuloma of the Larynx; A late complication of Endotracheal Anesthesia: *Annals of Otology, Rhinology and Laryngology*: 56: 191-193: (March) 1947.
176. Clausen, R.J.: Unusual Sequelae of Tracheal Intubation: *Proceedings of the Royal Society of Medicine*: 25: 1507: (May) 1932.
177. Gould, R.B.: Laryngeal Granuloma following Intratracheal Intubation: *British Medical Journal*: 2: 499-500: (September) 1935.
178. Cohen, M.: Tumour of Vocal Cord following Nasal Endotracheal Anaesthesia: *British Medical Journal*: 1: 283-284: (February) 1938.
179. Smiley, W.A.: Polypoid Granuloma of the Larynx following Endotracheal Anaesthesia: *Annals of Otology, Rhinology, and Laryngology*: 49: 556-558: (June) 1940.
180. Kearney, H.L.: Bilateral Granuloma of the Larynx, following Intratracheal Anaesthesia: *Annals of Otology, Rhinology and Laryngology*: 55: 185-187: (March) 1946.

181. Tuft, H.S. & Ratner, S.H.: Laryngeal Polypoid Granuloma following Intratracheal Anaesthesia: *Annals of Otology, Rhinology, and Laryngology*: 56: 187-189: (March) 1947.
182. Finer, G.H.: Granuloma of Larynx following Intubation: *Anesthesiology*: 9: 554-555: (September) 1948.
183. Kaye, G.: Sequelae of Anaesthesia: *British Journal of Anaesthesia*: 13: 157-176: (July) 1936.
184. Griffith, H.R.: Further Experiences with Endotracheal Gas-Oxygen Anaesthesia: *Anaesthesia and Analgesia*: 11: 206-209: (September-October) 1932.
185. Farriar, J.B.: Contact Ulcer of Larynx Developing after Intratracheal Anaesthesia: *Archives of Otolaryngology*: 36: 238-239: (August) 1942.
186. Barnard, J.: An Unusual Accident During Intubation: *Anaesthesia*: 3: 126: (July) 1948.
187. Mark, L.C. & Burstein, C.L.: Anaesthetic Management for Reconstructive Orthopaedic Surgery; A Case Report: *Anesthesiology*: 9: 656-657: (November) 1948.
188. Ransom, S.G.: Fatal Tracheitis after Endotracheal Anaesthesia: *British Medical Journal*: February 8th: 1946: p.222.
189. Lennan, B.B. & Rovenstine, E.A.: Fatality following Rupture of an Inflatable Cuff on Endotracheal Airway: *Anaesthesia and Analgesia*: 18: 217-220: (July-August) 1939.

190. Registrar General for Scotland Detailed Annual Report for 1913.
191. Registrar General for Scotland Detailed Annual Report for 1921.
192. Registrar General for Scotland Statistical Review 1945.
193. Report by Registrar General to Parliament 1901 for England and Wales.
194. 76 Annual Report of Registrar General of Births and Marriages in England and Wales 1913.
195. Registrar General's Statistical Review for England and Wales for the years 1938 and 1939.
196. Western Infirmary of Glasgow Reports, 1895-1904.
197. Vaines, J.C.: Textbook of Anaesthetics: (Minnitt & Gillies) 1948: p.547.
198. Purchase, W.B.: Pye's Surgical Handicraft: 1942: p.509.
199. Smith, Sidney: Pye's Surgical Handicraft: 1942, p.511.
200. Corlette, C.E.: Medical Journal of Australia: 1946: April 20th: p.545.
201. Sircar, B.N.: Indian Journal of Surgery: 8: 181-183: (December) 1946.
202. Barrie, H.T.: Lancet: February 22nd: p.242: 1941.
203. Ostlere, G.: British Medical Journal: April 5th: 1947, p.448.
204. Chivers, E.H.: British Journal of Anaesthesia: 20: 55-59 (July) 1946.

205. Whipple, A.O.: Surgery, Gynecology and Obstetrics:
January 1918.
206. Harroun, P., Beckert, F.E. & Hathaway, H.R.: Anes-
thesiology: 7: 24-28: (January) 1946.
207. Shipway, F.: Lancet: 1: 82: January 1935.
208. Organe, G. & Broad, R.J.B.: Lancet: November 19th:
1938: p.1170.
209. King, D.S.: Pre-operative and Post-operative Treatment:
Mason, R.L. & Zintel, H.A. (Philadelphia) 1947.
210. Harboard, R.P.: British Journal of Anaesthesia: 21:
49-51: (July) 1948.
211. Gray, T.C.: British Journal of Anaesthesia: 21: 49-51:
(July) 1948.
212. Woolmer, R.: British Journal of Anaesthesia: 21: 49-51:
(July) 1948.
213. Challis, J.H.T.: British Journal of Anaesthesia: 21:
52-60: (July) 1948.
214. Rawdon Smith, G.F.: Anaesthesia: 3: 110-112: (July) 1948.
215. Bourne, W.: Anesthesiology: 10: 157-163 (March) 1949.
216. Dilling, W.J.: (1943): The Pharmacology and Therapeutics
of the Materia Medica (London), p.178.
217. Roberts, H.: British Medical Journal: September 25: 1948.
218. Hellijas, C.S., Tovell, R.M. & Holt, K.R.: Anesthesiology:
8: 115: 1947.