SPINAL ANAESTHESIA

(A critical review of the literature and a record of personal experience.)

B Y

CONWAY PHILLIPS ALLEN, M.B., Ch.B..

****

28th: February, 1949.

CONTENTS

INTRODUCTION

This thesis attempts to review and discuss the experiences recorded by others who have used spinal anaesthesia and to state my own experiences and impressions. Like many other procedures, spinal anaesthesia has been and still is the subject of much controversy. Many with vast experience have the utmost confidence in its efficiency and safety; others condemn it. Both views are understandable. The intrathecal injection of an anaesthetic agent may have disastrous results. To those who have never experienced these or who have met disaster only after a large number of successful injections the method is acceptable. Tragedy early in its use causes abandonment, often with condemnation.

It may seem strange that religious belief was what made me incline towards spinal anaesthesia. In Malaya, where I have worked for more than twenty years, a large number of the inhabitants are Moslems. Most Eastern races are not so willing to submit to operations as Westerners are; but the Moslem is the most unwilling of all - at least he was twenty years ago. They are more willing if they are promised that they will not be "put to sleep". It may be that people fear more the anaesthetic than the operation. But it is to the loss of consciousness that the Moslem objects. Many reasons have been suggested for this. I believe the real one to have a religious origin.

It is not easy to discuss religious beliefs or to understand them; for Faith is indeed "the substance of things hoped for, the evidence of things not seen". The attitude of the Moslem towards a general anaesthetic is the same as towards alcohol. Alcohol is "haram"; that is, forbidden by religion. Not only does it "demean him and altogether destroy him; it also creates the state of "mabok" (drunkenness) in which he cannot hear the word of Allah." Now a general anaesthetic produces a very deep state indeed of "mabok". Moreover, it carries with it the risk of death; and the most important time of all when the word must be heard is when dying. To the Moslem, therefore, a spinal anaesthetic attracts and a general anaesthetic repels.

As my experience of the method increased I extended its use because the second serious indication was the absence of a skilled anaesthetist. Stanley (1940), describing his experiences in 4,674 cases of spinal anaesthesia in twenty-five years, states that he started it because he had no help in the California State prison. I have help but it does not include a skilled anaesthetist.

The choice of the most suitable anaesthetic for a given patient calls for careful and not just rapid and casual consideration. It should properly be made by an experienced anaesthetist who is able to weigh up all the factors that may influence the choice and who is skilled in the technique of all forms of anaesthesia; who can foresee any difficulties that may arise and who can instantly apply remedial measures for any contingency that may arise. Even if skilled anaesthetists were everywhere available the choice in any particular patient need not necessarily be the one that all experts would adopt; there is still the factor of individual preference. But this does not mean that experienced anaesthetists do not always give the correct anaesthetic; indeed, they do, where such are available; for surely individual experience may be an important factor in determining safety.

Spinal anaesthesia is only one method of allowing the performance of an operation; it is certainly not always the correct choice. Where all methods of anaesthesia are locally available in skilled hands, spinal anaesthesia may not often be selected as the most suitable choice. It is essentially a surgeon's anaesthetic, and in the absence of a skilled anaesthetist it presents manifold advantages to the surgeon, for example in abdominal work, and especially in emergency operations. The technique, once acquired, is simple; the anaesthesia is perfect while it lasts; muscular relaxation is very marked, rendering intra-abdominal manoeuvres easy of accomplishment. The production of anaesthesia is rapid. Surgeons who have specialist anaesthetists at their service must find it hard to believe the difficulties of those of their colleagues who have to put up with unskilled attempts to obtain general anaesthesia. The anxieties of the inexperienced when called upon to give a general anaesthetic for, say, a perforated peptic ulcer, must be very great indeed. On the one hand he has to "push" the anaesthetic to obtain relaxation; on the other is his inability to achieve this, coupled with his fear in attempting it, and his consequent failure to keep peace with the surgeon. The result is a prolonged general anaesthesia, in dosage far exceeding that necessary because of the surgeon's procedures being rendered more difficult than they should be. Retraction of muscles and proper exposure are impossible, with resulting increased and prolonged shock to the patient. The post-operative phase is more stormy and complications more frequent, troublesome and dangerous. Comparison of these patients' post-operative state with that of similar patients operated upon under spinal anaesthesia would leave only the very prejudiced in any doubt about the better choice.

Where a surgeon is alone; where he has help but no skilled anaesthetist or where his help is limited and the volume of work does not allow the presence of one of his small staff to be present while he is operating, the bias towards spinal anaesthesia is inevitable. Not only this, the selection of spinal anaesthesia is

justified because it is the most suitable available choice. I make no pretence that my patients get the proper choice of anaesthetic agent; I do insist that most of them get the best available anaesthetic.

As the method became popular among the patients, so the bulk of work increased enormously. Indeed it is not going too far to state that the introduction of spinal anaesthesia did more to popularise surgery in this country than anything else.

HISTORY

Although his reasoning sounds odd to us to-day, Corning (1885) seems to have been the first to conceive the idea of injecting a drug in such a manner that it would act directly upon the spinal cord. But he did not intend to effect sub-arachnoid injection; indeed he thought this was neither possible nor necessary. It was not possible to introduce the needle beneath the membranes of the medulla spinalis without removal of the arches of the vertebrae on account of the danger of wounding the cord. It was not necessary because the effect could be achieved through the medium of the blood-vessels of the cord if the drug were introduced near them. He thought that if these vessels were removed the drug would be inert. His explanation of the action was that if cocaine was injected between the spinous processes of the lower dorsal vertebrae, to a depth near the cord, it would get into some small veins there which "joined the more considerable vessels of the plexus spinalis interna". He made two experiments.

His first subject was a young dog which received 20 minims of 2% hydrochlorate of cocaine between the lower dorsal spinous processes. Five minutes later it showed inco-ordination of the hind legs followed by paralysis, but no signs of weakness in the fore-legs. Sensibility was tested by a fine wire brush connected to a powerful Faradic battery. No response was obtained in the hind-legs unless the most powerful currents were used. When applied to the fore-legs the limb was drawn away violently and the animal "sent up the most dismal howls". These phenomena persisted for a long time and traces of inco-ordination were observed two hours after the injection had been made. Recovery seemed to be complete after four hours. Corning thought that accurate localisation was impossible "on account of the numerous blood-vessels", but said it was conceivable that "had the quantity of anaesthetic fluid injected been greater, the anterior limbs might also have been affected". This last remark forecast the later practice of obtaining greater height in the level of anaesthesia by injecting larger volumes of the drug.

Corning's second attempt was made on a man upon whom he wished to effect abolition of reflex action and annulment of sensory conduction in the cord. "To this end I injected 30 minims of a 3% solution of the hydrochlorate of cocaine into the space between the spinous processes of the 11th: and 12th: dorsal vertebrae. As there was no numbness, tingling or other evidence of modified sensibility after the lapse of six or eight minutes, I again injected 30 minims of the same solution at the same spot and in the same manner." Ten minutes later the patient complained that his legs "felt sleepy": and tests showed loss of sensation limited principally to the lower extremities, the lumbar regions, the penis and the scrotum. When the patient left the office an hour later "sensibility was still impaired to a marked degree, but otherwise he seemed none the worse

for his experience. The patellar tendon reflexes were, however, abolished." Corning ended his article with these words:- "be the destiny of the observation what it may, it has seemed to me, on the whole, worth recording." The development of spinal anaesthesia shows how well worth recording his observations were.

Corning makes it quite clear that he never intended direct subarachnoid injection which was later deliberately done by Bier (1899). But it is possible that he achieved this accidentally, in the dog at least. In a second article Corning (1888) described experiences with distribution of the anaesthetic fluid in several interspinous spaces and how he had treated four patients in this way. The first one was to inject a painful area, a practice common enough today, though cocaine is no longer used.

Bier (1899) was the first to describe the production of spinal anaesthesia for the performance of surgical operations. I propose to quote Bier's writing at considerable length because it is of great historical importance and because it is often misquoted. For example, Keys (1945) says "Bier in 1898 produced true spinal anaesthesia in animals, then on himself and an assistant Hildebrandt by injecting the spinal canal with a solution of cocaine. He was soon using it on patients with complete success", and Rowbotham (1947) who says "In 1898 Bier of Kiel, after having first submitted to an intraspinal injection of cocaine on himself, reported six operations under low spinal analgesia". Careful reading of Bier's paper shows these statements to be inaccurate. In fact, Bier performed operations on five patients under spinal anaesthesia before he submitted to an attempt at spinal anaesthesia on himself at the hands of his assistant Hildebrandt. This attempt failed; and Bier did not enjoy a personal experience of spinal anaesthesia.

In his paper, Bier refers to the local injection technique introduced by Sleich which he thought was too limited in scope to allow the performance of big operations. He refers to Quincke's (1891) method of performing lumbar puncture and says that he used this technique to inject cocaine intrathecally. His first operation under spinal anaesthesia was performed on August 16th: 1898. Using 3 cc. of a 0.5% solution of cocaine he resected the ankle-joint of a 34-year-old workman suffering from tuberculous osteo-arthritis with secondary infection. "The patient whimpered during the operation, but he did not move and later said he had had no pain but was conscious only that something important was happening in the foot." Two hours later pain recurred in the foot and he had vomiting and severe headache. The vomiting soon ceased but the headache lasted till the following day.

Bier gives details of five subsequent operations, and states not only the dates but the time of day at which each was performed. On the morning of August 24th:, he performed his fifth operation. It was on a 30-year-old "agent for beer" with a complicated infected fracture of the femur in its lower third. On the evening of that day he thought he should undergo spinal anaesthesia himself in order to get definite information about the unpleasant disagreeable symptoms such as headache, nausea and vomiting which equalled those following general narcosis. Dr. Hildebrandt did the puncture by Bier's method and injected 0.5 cc. of 1% cocaine. At the time of puncture, Bier felt pain down one leg. But the injection failed to produce anaesthesia and small cuts and pin-pricks were felt everywhere as pain. Dr. Hildebrandt then offered himself for experiment and Bier then gave a similar dose on a patient who could understand, appreciate and describe all the effects on himself. He describes poor Hildebrandt's sufferings as all sorts of tortures were inflicted upon him to determine the efficiency of the anaesthesia. He also details the subsequent phenomena in both himself and Hildebrandt. Bier says he was confined to bed for ten days afterwards. If this is true, he must have got out of bed to operate on his sixth case three days later. He concludes his article by analysing the incidence of headache and other symptoms and suggests that other substances allied or related to cocaine might not produce these disagreeable phenomena.

Tuffier (1899) quotes Quincke (1891) as showing the ease and harmlessness of lumbar puncture with or without the removal of a little cerebrospinal fluid: and Sicard as showing that drugs and sera could be introduced safely under the meninges for action on the underlying nerve centres. He gives his experiences, first upon a young man suffering from osteo-sarcoma who recovered painless movements of his legs for nearly four hours after the intrathecal injection of cocaine. Struck by this result, he performed four operations on the legs and uterus under intrathecal cocaine. It is often stated that Tuffier's work was independent of that of Bier. This may well be true; but Tuffier quotes Bier's work, of which he was therefore aware at the time of writing if not at the time when he first gave spinal cocaine. Tuffier concluded that for big operations on the lower limbs cocainisation of the cerebrospinal fluid produced a harmless and effective analgesia; but he was careful not to over-rate the method and had no intention of dethroning chloroform.

Babcock (1906) reported that certain undesirable features of chloroform and ether anaesthesia had resulted in much effort being spent in the perfection of local and spinal anaesthesia. He continued that some years earlier medullary cocainisation had had a wide vogue but had been abandoned by most of the surgeons who had used it on account of severe and prolonged headache, vertigo, nausea and vomiting, - sometimes persistent - thirst, palpitation, chills, fever, hyperhydrosis, circulatory disturbances, tremor, ataxia, rigidity of the neck or of the entire back and more rarely urinary incontinence and other palsies and even death. He estimated that

the death rate was 1 in 200. Studies of the cerebrospinal fluid had confirmed the clinical evidence that cocaine produced a distinct, though probably transient and aseptic, meningitis. It was little wonder that many people had abandoned it. But recent advances, including the introduction of less toxic substances, the addition of agents to localise or prolong its action and greater accuracy in the dural injection, had increased its effectiveness and safety. He recorded his experiences in 76 cases, 65 of whom received stovaine and 11 alypin. In 5 cases the anaesthesia was insufficient. One patient died following 64 mg. of alypin. Babcock thought that the best puncture site was the second lumbar interspace as lower punctures gave less uniform results, and he advised the rapid injection of two or more cc. of stovaine with the addition of adrenaline to prolong the action. The strictest aseptic precautions must be observed. He thought "rachistovainisation" was more efficient in preventing shock that the nerve-blocking method of Crile and says "Finally it may be mentioned that the efficiency and reliability of the method develop with experience. With greater precision in technique there comes increasing confidence in the advantages of this form of anaesthesia." How true so many of Babcock's statements are today.

Barker (1906) introduced spinal anaesthesia to England and at this time Jonnesco was reviving it on the Continent.

Pitkin (1928) described attempts at "controllable" spinal anaesthesia using a solution of novocaine to which gliadin of wheat was added. At the time, this was hailed as a great advance by such statements as "The whole of spinal anaesthesia will not have to be re-written". Pitkin is a great enthusiast and has written extensively. He makes claims for his method.

Howard-Jones (1930) made the next great advance by the introduction of a new drug, percain, in hypobaric solution. His work was modified by Etherington-Wilson (1933) who introduced a new technique with the patient in the sitting position.

The most recent innovation is that of Lemmon (1939) who described his method of continuous spinal block by fractional doses introduced through a malleable spinal needle which was left in situ. By this means, anaesthesia could be prolonged as necessary.

Tuohy (1945) continued Lemmon's work, but used a ureteral catheter instead of a metal needle.

ANATOMY

GRAY'S "ANATOMY" states "The spinal cord occupies the upper two-thirds of the vertebral canal. Its average length in the male is 45 cm.. It extends from the level of the upper border of the atlas vertebra to that of the lower border of the first lumbar vertebra or the upper border of the second as a rule, but its lower end may sometimes be found as high as the lower border of the twelfth thoracic vertebra or as low as the upper border of the third lumbar vertebra." This lower border level is of importance, because while it is possible to perform lumbar puncture at any level, the only spaces that are really safe are the fourth and fifth lumbar interspaces. The third lumbar interspace is usually also safe; that is, it carries little danger of cord injury. The end tapers off rapidly into a conical extremity.

"The position of the spinal cord varies at different stages; in early intra-uterine life the spinal cord is as long as the vertebral canal, but after the embryo has attained a length of 30 mm. the vertebral column begins to grow more rapidly than the spinal cord, and the latter gradually assumes a higher position within the vertebral canal ..... so that by the twenty-fifth week the lower end of the spinal cord has ascended from the level of the second coccygeal vertebra to that of the third lumbar vertebra, i.e., a distance of nine segments, and there remain but two segments before the adult position is reached (Streeter) ..... the nerve roots becoms more and more oblique in direction, so that in the adult the lumbar and sacral roots descend almost vertically to reach their foramina. The position of the spinal cord also varies with the movements of the vertebral column, being raised slightly when the column is flexed. It enjoys this degree of mobility because it does not occupy the whole of the vertebral canal and is ensheathed by three protective menbranes separated from each other by spaces."

The spinal dura mater is a continuation of the inner or meningeal layer of the cranial dura mater and forms a loose sheath around the spinal cord from the foramen magnum to the lower border of the back of the coccyx. It is separated from the wall of the vertebral canal by the extra-dural space which contains a quantity of areolar tissue, fat and a plexus of veins. It is important to note that these veins are more marked anteriorly, so that a bloody tap is more likely to arise when the needle is driven too far forward. Between the dura mater and the subjacent arachnoid lies the subdural space, a capillary interval containing a small quantity of fluid, probably of the nature of lymph. The subdural space extends downwards to the level of the lower border of the second sacral vertebra, where the dura and arachnoid mater blend with the filum terminale which is the continuation, downwards from the conus medullaris, of the pia mater.

The arachnoid lies below the subdural space. Separated from the pia mater by the subarachnoid space which is filled with cerebrospinal fluid, the arachnoid mater surrounds the cranial and

spinal nerves and encloses them in loose sheaths as far as their points of exit from the skull and vertebral canal. It invests the spinal cord loosely. Below, it widens out, invests the cauda equina and ends as described at the lower border of the second sacral vertebra.

The practical points that emerge from the above are that the segments of the cord corresponding to the spinal nerves are placed higher above the corresponding vertebral body or spinous process as they pass downwards until the lumbar and sacral segments are closely placed together, relatively, at the lower end of the cord; the highest space that may be safely selected for lumbar puncture with minimal risk of damaging the cord or cauda equina is the third lumbar interspace. The fourth lumbar interspace is probably the easiest one to choose for puncture; in infants, the fourth or fifth lumbar interspace should be selected.

Pease (1935) states that the posterior disc wall bulges slightly and especially if the spine is flexed, with resulting increased pressure within the disc. He thinks that in the cadaver it is difficult to know whether the disc has been struck or not and that it is easy to hit the disc, the vertebra, the interarticular facet or even the venous sinusoids in the vertebral body.

But to give satisfactory spinal anaesthesia it is necessary to know sensory levels on the surface and in the viscera, and the spinal segments that supply the muscles. For example, inguinal herniotomy is painless if the anaesthesia reaches the level of the twelfth thoracic segment, but the operation is much easier if the muscular paralysis is four or even six segments higher.

PHYSIOLOGY

Like anatomy, physiology is important to the spinal anaesthetist and it is wise for him to have a clear grasp of at least the cerebro-spinal fluid, the factors governing respiration and the control of blood-pressure, the effects of a spinal anaesthetic agent upon various bodily functions, and the nerve supply to those structures that not only provide tone to skeletal muscle, but supply the hormones concerned with the processes of living. Before one can appreciate the abnormal, one must first of all appreciate the normal.

"The cerebro-spinal fluid is clear, colourless and alkaline. This last property is of importance since it has been shown that alkalies alone can fix a spinal anaesthetic solution. The normal cell count is up to five lymphocytes per c.mm.. Its general composition is like that of protein-free plasma, but there are significant differences in the concentration of the different crystalloid constituents. The average values for its main constituents (in mg. per 100 cc.) are as follows (the figures in brackets are those for plasma): Na, 334 (330); K 10.6 (17-) Ca. 5.3 (10.3); Cl.436 (365); HCO3, 105 (150); PO4, 1.8 (3.0); SO4, 0.6 (1.9); glucose, 70 (100). The protein content is minute, i.e. 0.02% (plasma, 8.0 gm.), equally distributed between albumin and globulin. The intraventricular fluid is probably free from cells and protein; these may be added to the fluid in the subarachnoid space by exudation from the meningeal blood-vessels. The total volume of the cerebrospinal fluid is (in man) 100 to 150 cc.. When free escape is allowed to the exterior, the rate of formation is 20 cc. per hour, or about 500 cc. per day .. .. it is formed by the choroidplexuses, especially by the large plexuses which are found in the lateral ventricles ..... in view of all the (above) evidence, the formation of cerebrospinal fluid must be regarded as a process of secretion; it is affected, however, by alterations in the physico-chemical relations in the plasma and in the cerebral ventricles. The specific gravity varies from 1,004 to 1,010. The pressure is about 110 to 140 mm. of water.

Weed (1922) showed that absorption of cerebrospinal fluid takes place mainly via the arachnoid villi into the dural venous sinuses, though a little may pass into the perivascular spaces. Burford (1942) thinks this does not apply to spinal anaesthetic drugs when injected into the lumbar area because they do not reach the cisterna cerebello-medullaris in effective quantities. If absorption of these does take place, it probably does so near the site of injection. He quotes Elman as showing the presence of spinal arachnoid villi or cell clusters and at the subsracnoid angles the same morlhological scale of spinal segmental veins analogous with the cranial venous sinuses. But even Elman minimises the importance of absorption by this route. Cathelin thought fluids went along the nerves in a prolongation of the subarachnoid space. Such communication along the nerve has not

been proved though Weed thought it could occur without an open communication. As the vessels cross the subarachnoid space they carry with them into the nerve tissue a double layer of covering - the perivascular as arachnoid, the other or periarterial as pia. Between these is a cuff-like perivascular subarachnoid space containing cerebro-spinal fluid: going inward to the capillary region, it goes deep into the nerve substance and may possibly function in place of a true lymphatic system which is absent in the central nervous system. In the pre-capillary region it is believed to be confluent with the perineuronal space. Wallace and Brodie (1936) confirmed its importance.

Can spinal anaesthetic fluid enter the blood-stream? Jones (1931) thought it could and stated "The dosage of procaine is so high that the amount absorbed into the blood-stream causes a general anaesthetic state; and the itching and drowsiness and stupidity of the patient is evidence of the circulating drug acting upon the skin and cortical cells of the cerebrum. Vomiting is due to stimulation of the vomiting centre by poison." I cannot agree with Jones and feel that if spinal anaesthetics enter the blood they can do so only through the medium of a punctured vessel. Much greater quantities are given locally, and unless absorption to the blood stream occurs more readily from the cerebro-spinal fluid than from, say the subcutaneous tissues, toxic effects are more likely to follow local injection. Hill (1935) says that 10 mg. of procaine injected into the cisterna magna of a cat will result in death from respiratory paralysis in one and a half minutes; but it takes 180 mg. injected intravenously at the rate of $7\frac{1}{2}$ mg. per minute to paralyse the respiratory centre; and he concludes that respiratory paralysis is not due to action on the bulb after absorption of the anaesthetic into the blood-stream.

I have frequently used as much as 900 mg. of procaine locally, for example in thoracoplasty, without ill results.

Fluid mechanics in the cerebro-spinal space

Lund and Cameron (1945) discussed the factors determining spread of the fluid after injection. These are the force of injection, the disturbance produced by barbotage and the effect of gravity. The last is checked by the absorptive affinity of nerve tissue for the drug and is governed by two processes which are easily controlled,— the difference in specific gravity and the site of introduction into the cerebro-spinal fluid. They consider that the rate of flow of cerebro-spinal fluid is so sluggish as to render it without influence. Barbotage is difficult to control. They do not favour heavy solutions as they think the head flexion then necessary for five to twenty minutes following injection is not suitable for patients who are shocked or exsanguinated or who have a low blood-pressure; and, moreover, the accentuated spinal curves necessary

when using hyperbaric solutions tends to "pool" the solution, giving unpredictable levels of analgesia. For some reason they add that much time is wasted with heavy solutions "when patients require operations on their dorsal aspects since the posterior sensory roots must be fixed before placing the patient prone".

(1936)
Vehrs thinks that the "tugging" action of the small vessels has some effect on spread. This is beyond the control of the anaesthetist.

It is, of course, a common experience of all who give spinal anaesthetics that the agent soon becomes fixed. This is easily and clearly shown by the fact that about ten minutes after injection, no change in position will alter the level of anaesthesia. Such fixation cannot occur in mock spinal anaesthesia experiments. But this does not invalidate the conclusions regarding the rate of spread arrived at by Wilson following his experiments. Indeed Wilson has applied his results in his practice, and, while he may have altered his time periods slightly, his work shows that the spread rate in the human subject approximates very closely that occurring in mock experiments using coloured solutions.

Effects of dilution

Fisher and Whitacre (1947) studied the effect in 1,124 patients, of injecting nupercaine in dilutions greater than 1:1,500. He used such dilutions for the production of spinal anaesthesia from 1:2,000 to 1:10,000 and concluded that concentration did not affect the degree or duration of sensory or motor block, or the incidence and severity of circulatory reaction. The toxicity seemed to depend more on the amount than on the concentration and there is no advantage in using weaker solutions.

Specific gravity of different solutions of anaesthetic agents

JONES (1930) quotes authoritative specific gravities of different solutions as estimated by Norman Evers, Chief Chemist to Messrs. Allen and Hanbury's. At 35.5 C., these are as follows:-

	1-1,000	1-1,500	1-2,000
In water	1.0001	1.00005	1.00005
In 0.5% NaCl.	1.0035	1.00345	1.00345
In 0.9% NaCl.	1.0062	1.0061	1.0061

and states that the variation in the specific gravity of the cerebro-spinal fluid renders it impossible to be certain whether a given solution will be isobaric.

The same writer states that 10% procaine in water is hyperbaric,

having a specific gravity of 1.017.

The first solution I had prepared for me was of procaine 1% in water in ampoules each containing 1 cc. This has a specific gravity of .

Whatever solution is being used, the administrator must be quite clear about its specific gravity. I do not agree with the use of so-called isobaric solutions and agree that such a solution for routine use is impossible of determination. Even if it were possible, I still think that the spread should be dependent upon a difference in specific gravity between the agent and the cerebro-spinal fluid. Whether the anaesthetic solution is hypo- or hyper-baric is purely a matter of individual preference and conviction. The essential is to appreciate what is happening to the solution after joining with the spinal fluid. Wilson (1934) objected to solutions containing alcohol as he thought this created uncon-trollable currents. In a later communication he (1945) states that he thinks this condemnation was wrong, though even then he still thought they were "not safe used ignorantly". Later on I propose to take Wilson's technique with hypobaric solutions and compare it with my technique using hyperbaric solutions.

SOME GENERAL EFFECTS OF SPINAL ANAESTHESIA

Boyd (1936) investigated the urea nitrogen content of the blood following anaesthesia and states that the average rise was no greater than the daily variations in controls but that a greater percentage shows this increase after general than after spinal anaesthesia.

Allen and Livingstone (1940) investigated the early post-operative reduction of prothrombin in the jaundiced and biliary-fistula patient, with special reference to the anaesthetics used. Compared with other patients, these show a fall but the anaesthetic plays no part in its causation. Their patients received spinal and various general anaesthetics.

Bellis (1944) quotes Sherrington "The postural tone of the bladder keeps the intravesical pressure normal as it fills." But the pressure increases rapidly in acute urinary retention with resulting damage due to attempts to void causing considerable rises in intravesical tension. He quotes Creevy as saying that post-operative retention of urine is due to the horizontal posture, the pain of injured tissues and the effects of drugs, for example opiates and the anaesthetic. He makes no reference to the "conditioned reflex" part in micturition. Disturbances of the central nervous system affect bladder sensation and its voluntary control. He carried out cystometry after spinal anaesthesia and says there is a residual bladder-wall insensitivity with decreased vesical tone. Cholinergic vagotropic and parasympathetico-mimetic drugs, "running water" etc. merely cause evacuation by overflow. These results suggest the desirability of passing a well-lubricated sterile catheter within at most six hours after the last emptying time for a period of twenty-four hours until normal bladder reflexes are re-established.

CIRCULATORY CHANGES

Rovenstine et alia (1942) administered 100-250 mg. of procaine in 10% solution to seven subjects who were then put in the supine position and underwent no operation or manipulation. Only three of them showed a decrease of peripheral resistance and in two of these the decrease amounted to only 12%. In the remaining four, maintenance of arterial tone was shown by lack of such decrease. Of these four, two showed a decrease in mean arterial pressure attributable to a decrease in cardiac out-put. One showed an increase in mean arterial pressure of 22% with a decrease of 16% in the cardiac out-put and one showed no change in any form. In only two cases did nausea and vomiting occur and these showed a significant fall in blood pressure. Elevation of the trunk from twenty to fifty degrees caused a rapid and marked fall in blood pressure, accompanied by faintness, which disappeared with the restoration to the supine position. They conclude that sympathetic vaso-constrictor fibres are effectively

anaethetised and that there is little if any tonic activity in the thoracic-lumbar constrictor nerves except those to the skin which they consider of relatively little importance. They believe that trauma, haemorrhage, anoxia, the weight of instruments or packs on the abdomen, combined with posture, contribute to a progressive reduction in venous pressure resulting in circulatory embarrassment.

Foster et alia (1945) investigated the peripheral circulation in anaesthesia, shock and haemorrhage, using the digital plethysograph. Spinal and regional anaesthesia produce vaso-dilatation in the anaesthetised area with concomitant vaso-constriction in the unanaesthetised area. Failure of the vaso-constrictors is accompanied by hypotension, in which ephedrine acts by restoring constriction in the unanaesthetised areas, the anaesthetised areas showing no return of tone. In some cases of reduced blood-pressure under spinal anaesthesia, it appeared that diminished cardiac output was responsible for the hypotension. They emphasise the poor tolerance towards shock and haemorrhage exhibited by the patient under spinal anaesthesia.

Gregory (1946) et alia say that in hypertensives, the fall in renal function is related to the fall in blood pressure following in spinal anaesthesia.

Gregory and Levin (1945) believed that the great fall in blood pressure following spinal anaesthesia in ~~50~~ hypertensives is due to increased vaso-motor tonus, even in patients with extremes irreversible arteriolar disease as shown by chemical and clinical evidence of uraemia. This is contrary to the thesis that the fallen blood pressure in hypertension produced by functional or anatomical interruption of the sympathetic nervous system is due to improvement in renal blood flow.

Doud and Rovenstine (1940) investigated changes in the velocity of the blood flow during spinal anaesthesia by a purely subjective test in which they told the patient what to expect and instructed him to report the feeling of warmth at each site immediately. There were 98 studies of which 59 were controls. They concluded that spinal anaesthesia above the sixth thoracic segment increases both a normal and a prolonged circulation time and say that the test is of value in selecting patients for high anaesthesia. Sensory anaesthesia below the sixth thoracic segment had little effect on the velocity of either group. They believe that the prolongation time is due principally to a decrease in cardiac output. Ephedrine is effective in restoring the circulation time prolonged by high spinal anaesthesia.

MODE OF ACTION OF LOCAL ANAESTHETICS

HEWER (1946) says that local anaesthetic agents are highly lipoid-soluble alkaloids. Their non-irritant water-soluble salts are used for injection. The slight alkalinity of the tissue fluids is believed to hydrolyse these salts and the resulting alkaloidal bases are taken up by the lipoids in nerve tissue. Another factor is probably the ionisation of the alkaloidal salt which produces an ion of the analgesic base having a positive charge which is taken up by the nerve structure which has a negative electrical charge. The effect on the nerve is complete depression without previous stimulation. But the depression is not uniform. For example, in mixed nerves the sensory fibres are affected before the motor ones, the order for loss of function being first vaso-constriction, then temperature followed by pain and touch and lastly joint and pressure sensation. In animals, this corresponds to the thickness of the myelin sheath of the various fibres. It has been shown that an early primary degeneration occurs in the nerve fibres in contact with an analgesic solution. This starts in the axis cylinder and extends to the myelin sheath. The process is normally reversible and its degree can be taken as an index of the local toxicity of the agent employed. If this is done, cocaine, procaine and stovaine show up best. The first-named agent is, however, not suitable for injection purposes on account of its high general toxicity.

WHITACRE (1944) reported on investigations to correlate the local anaesthetic potency of a compound with the concentration of the solution, with fat and lipoid solubility, with $_{p}H$ values, with oil-water distribution co-efficient, and with surface tension lowering at an oil-water interface. The results of these studies indicated that the primary effect in local anaesthesia is absorption of the anaesthetising compound by the fatty or lipoid portion of the nerve fibre. It is further proposed that, following absorption, the local anaesthetic reversibly coagulates the nerve colloids and proteins, decreases permeability of the nerve fibre to water and ions, and decreases the electrical condictivity of the nerve, resulting in localised temporary paralysis of the sensory nervous system (Hirschfelder and Bieter - 1932).

ADRIANI (1946) states that local anaesthesia agents must be present in considerably higher concentration in the nerve cells to produce anaesthesia than the general anaesthetic drugs. They are therefore safely applied only locally. The factors influencing the intensity and duration of anaesthesia are the chemical nature of the agents, the duration of its contact with the nerve cell, the concentration of the drug and the size of the fibre.

Anaesthesia varies inversely with the square root of the time the drug is in contact with the surface of the exposed nerve cell or fibre: and its intensity varies inversely with the log of the square root of concentration. Large fibres are the last to

be anaesthetised in the spinal canal. The rule is that the sensory fibres of the root are anaesthetised first, then the sympathetic fibres and lastly the motor fibres. The motor fibres are said to recover first, and the sensory second or with the sympathetic. There is some evidence that the sympathetics are anaesthetised first.

Sarnoff et alia (1948) investigated intestinal dyskinesia, colonic atony, and visceral afferent fibres by the subarachnoid injection of large quantities of procaine in weak solution (0.2%). They conclude that the visceral afferent fibres are larger and more heavily myelinated than the viscero-motor and other sympathetic efferents which appear to be about the same size and degree of myelination.

Richards (1947) states that most anaesthetics are detoxicated by the liver. Hepatic damage is known greatly to retard the destruction of procaine. He describes the results of experiments to determine the convulsive action of procaine in guinea-pigs, using normal subjects and ones deficient in vitamin-C; and also the effectiveness of giving ascorbic acid to those deficient, to reduce convulsions. He found that 100 mg. per kilo. of body weight had no effect, but 250 mg. per kilo., given three hours previously, restored normal sensitivity in depleted animals. He suggests that a liberal diet with vitamin-C and dextrose may help to diminish dangerous toxic side-effects, especially in patients in poor nutritional condition.

RESPIRATORY PHENOMENA

Schmidt (1945), discussing dyspnoea as produced intentionally in normal subjects in the laboratory or as encountered in the course of disease, points out that it is due much more to new or augmented reflex influence than to increased stimulation of the respiratory centres by the chemical products of metabolism. Anaesthetics diminish the reactivity of the centre to chemical stimuli but this is counteracted by reflex factors. If it is not, then respiratory failure is likely to be a conspicuous phenomenon of the anaesthetic process.

McCann (1947) compares Haldane's chemical control of respiration with the more recent ideas of neurogenic control as described by Heymans. With pnuemograms, he discusses the respiratory and expiratory effort and shows that there is segmental respiratory response identical in both the somatic and visceral zones in each segmental area - cephalic, cervical, dorsal, lumbar, and sacral. The response to trauma is well seen in pentothal but not under local or spinal anaesthesia. In the thoracic zone, it may arise from the skin incision or from traction on the stomach. In any given segment the response pattern is the same, whether stimuli occur in the visceral or in the somatic plane.

THE RESULTS OF EXPERIMENTAL INJECTION

BROCK et alia (1936) quote WOSSILDO (1908) as saying that in rabbits the injection of anaesthetic agents is followed by chromatolysis and swelling of cells; indeed, few normal cells are seen in the anterior horns after two hours. Return to normal was evident in six hours and was complete in twenty-four hours, which shows that the changes are readily reversible and recovery is rapid and complete. Unhappily, this is not always true, as will be shown when the complications of spinal anaesthesia are discussed.

Changes in the cerebro-spinal fluid

Iason et alia (1930) examined the spinal fluid in thirty-one cases 18 hours following spinal anaesthesia. Their results are confusing. They state that in seventeen of these no examination was carried out because there was blood in the cerebro-spinal fluid. Of the remaining fourteen, eleven showed pleocytosis, one being of polymorphonuclear type and the remaining ten of lymphocytic type. They go on to say that in twenty cases the blood sugar was raised but there was no change in the albumin, globulin or colloidal gold curves. From their figures, it is obvious that blood sugar estimations were done in at least six cases who had blood in the spinal fluid. This is an example of work that, if written, should not be afforded publication. People who cannot perform lumbar puncture without drawing blood except very rarely indeed should not give intrathecal injections until they first learn the simple technique of the puncture.

Black (1947) gives the following figures in

	No. of cases	Protein (mg.)	Cell count	Sugar (mg.)
Controls	75	31.8	normal	71
S.A. (1-12 mos. earlier)	60	38.0	normal	Not done
S.A. (two stage operations up to 30 days earlier)	40	49.0 - 63.1	normal	81
Continuous spinal	?	(before) 28.0	normal	79.8
		(after) 26.5	normal	404.0

Pontocaine and glucose were used. It seems odd to estimate the glucose after continuous spinal anaesthesia using a solution containing glucose unless a considerable time was allowed to elapse. Here the time has not been stated.

Intra-cisternal injection of anaesthetics

WOOLMER (1948) describes the effects of the inadvertent injection (there was confusion between two similar syringes) into the cisterna magna of a patient suffering from cervical cord tumour (an ependymoblastoma). The injection was given with the patient sitting upright in a chair. The dose of procaine was 60 mg. in 2% solution. In a few seconds she complained of her head feeling hot. She lost consciousness in the second minute. Respiration ceased about a minute later and remained in abeyance for forty-five minutes. All reflexes were abolished and there was profound flaccid paralysis but no marked cardio-vascular depression. There was extreme cyanosis from the onset of respiratory depression until effective artificial respiration was established "five to ten minutes later". The pulse became feeble towards the end of this time, but improved and remained satisfactory when anoxaemia was corrected, and maintained, by rhythmic insufflation of oxygen through an endotracheal tube. The patient's condition then remained good in spite of the profound flaccidity. Spontaneous respiration began to return after forty-five minutes and was fully re-established in a few minutes. The laryngeal reflex returned ten minutes later. She did not, however, regain consciousness and during the next three hours she had a series of convulsions. From then until her death six days later the clinical picture was of grave cerebral damage following anoxia, with convulsions, unconsciousness, rigidity, mask-like facies and profound dementia. The histological findings were complicated by a malignant tumour occupying much of the cord and brain stem, which may have influenced the result of the injection. The conclusion is that the dose of procaine intra-cisternally caused profound but reversible respiratory paralysis. The irreversible changes that killed the patient were due to anoxaemia.

WADE (1934) investigated the effects of procaine injected into the ventricles, the cisterna magna and the lumbar region, in large dogs under basal anaesthesia induced by barbiturates (one case) or paraldehyde. Artificial respiration was maintained in all cases by compressed air through a tracheal canula. He reports as follows:-

1) Intraventricular procaine was given in doses from 10 - 200 mg. in 1% Ringer's solution. 10 mg. failed to cause respiratory paralysis which was, however, produced by doses of 25 mg. and upwards within one and a half minutes. Respiration ceased suddenly without change and returned suddenly fifteen to thirty minutes later, becoming quite normal in about another five minutes. The cessation of respiration was associated with a slowing of the pulse rate and a fall in blood pressure starting within twenty seconds and reaching its maximum in about thirty seconds.

2) Intra-cisternal procaine. 150 mg. of procaine in 1% Ringer's solution injected into the cisterna magna caused respiratory paralysis within one minute. Artificial respiration was instituted at

once and the animal was put into a steep Trendelburg position. The pulse rate slowed and the blood pressure fell slowly in the first ten minutes to about one-third normal. After one and a half to two hours spontaneous respiration and a rise in blood pressure started and progressed so that artificial respiration could be stopped at two and a half hours. However, the dog could not be put out of the Trendelenburg position during the following half-hour without a fall in blood pressure and the need for artificial respiration. Thereafter posture ceased to have any effect.

3) Lumbar intrathecal injection of 150 mg. of procaine in 1% Ringer's solution caused the blood pressure to fall to one-third and the pulse rate to two-thirds of their former rates within twenty to sixty seconds. Respiration became purely abdominal and movement of tidal air gradually failed so that in ten minutes cyanosis was marked and respiration had failed. Cardiac failure frequently preceded respiratory failure and unless artificial respiration was started early the dog could not be saved. Under artificial respiration, in the Trendelenburg position, the colour improved and the animal remained in a condition of slow pulse and low blood pressure for one-and-a-half to two hours when a gradual rise in blood pressure and feeble respirations took place. Although it was safe to stop artificial respiration at two-and-a-half hours, the vaso-motor system was so unstable that the Trendelenburg position could not be altered for a further half hour without the occurrence of circulatory and respiratory collapse.

Cotui and Standard (1932) also showed by experiments on dogs that the intracisternal injection of procaine in sufficient dosage produced respiratory and vasomotor paralysis and that when sodium amytal with or without morphine had been given the effective dose of procaine was reduced by about forty per cent. of that required for normal dogs. When respiratory failure occurred, effective resuscitation followed the institution of artificial respiration and the injection of ephedrine.

COLLAPSE AND RESUSCITATION

HEWER (1946) points out that there are two main types of collapse: firstly, primary cardiac failure and secondly, the gradual deterioration in the patient's condition described as shock, which if untreated may lead to secondary cardiac failure. In the first, the colour suddenly changes to a grey pallor, the pupils dilate widely and become inactive, the pulse is imperceptible, the respiration sighing and finally ceasing. The causes are not clear; it often occurs with a light anaesthesia, usually maintained with chloroform or a mixture containing it. It may follow a severe surgical stimulus, adrenaline into the circulation; it is common in status lymphaticus. Intravenous local anaesthetic has caused it, as has spinal injection. In the early stages, the heart is in one of three conditions: a) beating very feebly, b) in ventricular fibrillation or, c) quiescent.

Treatment. Stop operation and adopt a steep Trendelenburg position. Inflate the lungs slowly with pure oxygen. If pressure is not available, do Silvester's method - the respiratory-pump action is a great help. Cardiac puncture: it is best to puncture the auricle and not inject unless necessary. You can use a needle electrode. If this fails, do massage without delay and continue it - a case recovered after 29 minutes.

Recovery depends on the early and efficient institution of restorative measures. The time factor is everything. The first cells to suffer are those of the cerebral cortex and permanent recovery is unlikely if these are deprived of oxygen for more than five minutes. Macleod says the periods of oxygen lack that result in death are a) small pyramidal cells of the cerebrum - eight minutes, b) Purkinje cells of the cerebellum - thirteen minutes, c) medullary centres - twenty to thirty minutes, d) spinal cord - forty-five to sixty minutes and e) sympathetic ganglia - three to three and a half hours. (Physiology and Biochemistry in Modern Medicine. 6th Edition.) The result will depend on the time that has elapsed or rather the time during which the cells of the nervous system have been deprived of oxygen. There may be complete recovery; or slight damage to the cortical cells may be evidenced by prolonged coma, by convulsions or by mental deterioration after recovery.

The cerebral cortex may be so badly damaged that consciousness may not be regained, the patient resembling a decerebrate animal and eventually dying. In rare cases, the heart does not respond and death results.

SHOCK. A difficult subject theoretically, it is easy to recognise shock clinically during an operation by pallor, sweating, coldness of the skin, rising pulse rate, falling blood-pressure (systolic more than diastolic), dilated pupil and spaced or sighing respiration. These may be modified by the anaesthesia or operation. Attempts to reduce shock to terms of blood pressure are not satisfactory. The definite signs are a rising pulse rate with a persistently falling pulse pressure. The essential pathology is a diminution in the volume of circulating blood in true shock. This differentiates it from the depressed condition of the circulation seen in spinal anaesthesia. Apart from blood loss, the loss in circulating blood is probably due to its presence in the muscle capillaries and forms a vicious circle.

CAUSES AND PREVENTION. Emotion (psychic shock); loss of fluids - water by visible and invisible sweating and respiratory loss: blood by haemorrhage and extravasation; trauma, by nerves in reflex shock and muscles in "histamine" shock; loss of heat, by exposed viscera or cold anaesthetic inhaled vapours; overdose of anaesthetic, relative or absolute; oxygen deficiency from obstructed airway, etc.; excessive duration of operation.
Prevention: Hypnotics and basal narcotics and suggestion help. Reduce the nocio ceptive stimuli - this DOES occur in light anaesthesia. Chloroform sensitises the capillaries to the action of histamine. Do not use anything but N_2O and O_2 in shocked patients. In the crush syndrome, its renal deficiency may be due to anoxia of the kidneys. It can be helped by 4.28% sodium sulphate at 100 drops per minute, intravenously. Remember the law of diminishing resistance. Keep the theatre warm. Watch on the way back to the ward. It has been said that a patient cannot tolerate a systolic blood-pressure of 80 mm. Hg. for more than twenty minutes. If this is materially exceeded he will die within forty-eight hours. This is not always true but should be borne in mind.

TREATMENT OF SHOCK

MORGAN (1943), discusses the treatment of shock inasmuch as it is related to preparation for operation and anaesthesia under a) general and b) intravenous measures. Under "general", he advises removal of the cause, rest of body and mind, the relief of pain, the promotion of warmth, or rather the reduction of heat loss, in a room to control the heat and humidity (70% to 80% and 65%) and the administration of oxygen. He points out that the administration of 100% oxygen raises the total in the blood by only 10% to 15%. Oxygen want may be extreme in the absence of cyanosis, and must be given in high concentration. In blast injury he thinks it should even be given subcutaneously or even intravenously. Sweet warm drinks and the institution of a rectal drip tend to mobilise the plasma proteins from the liver. Intravenous measures are necessary in severe shock; here speed is necessary and the patients need blood, plasma or serum. A falling blood-pressure and a rising pulse-

rate are of bad omen. In acute blood loss transfusion should be carried out till at least 60% is obtained (each 500 cc. is estimated to give a rise of 10%), and the first 500 cc. should be run in as rapidly as possible. In non-urgent cases, care should be taken not to overload the right heart. In burns, peritonitis and other cases where there is loss of fluids rich in proteins, plasma or serum are necessary in large amounts, for example four litres in four hours. If plasma is not available, salines should not be given, and blood should be given instead. In acute water loss (dehydration), saline, with or without glucose should be given and you should not be afraid to give plenty. Blood is contra-indicated here. Where dehydration is accompanied by anaemia, as in congenital hypertrophic pyloric stenosis, diarrhoea and malignant disease of the colon, you must give blood. In haemo-concentration shock it is important to give plasma and not blood. Whatever the cause of shock, its end-result is anoxia; and he urges therefore the use of an anaesthetic agent allowing the maximum of oxygen. He states that cyclopropane is the anaesthetic of choice and that chloroform or spinal anaesthesia is contra-indicated in shock.

Anoxia under spinal anaesthesia is of the ischeamic or stagnant type. "The tension of oxygen in the blood is normal, but the amount reaching the tissues is inadequate. The rate of tissue oxidation is normal, because oxygen is supplied at a high pressure level head. As the blood circulates more slowly in the tissues there is more time available for the reduction of oxy-haemoglobin. Furthermore, the impaired circulation causes CO_2 accumulation in the tissues, which facilitates the giving-off of oxygen. Thus the tissues make the more effective use of what oxygen does reach them in the blood." (Samson Wright: 1945). This type of anoxia is that found in haemorrhage, impaired venous return, heart failure and shock. It is obvious that to combat it, measures must be taken to prevent or ameliorate these. In addition, however, there is respiratory depression to some extent. It seems that high-tension oxygen (100%) is indicated, and I am convinced on purely practical experience that this is one of the most valuable therapeutic means we have of reducing the effects of tissue anoxia. Too often is reliance placed upon the absence of cyanosis; too rarely is it realised that tissue anoxia can be present in the absence of cyanosis.

It is important that all restorative measures should be ready and that no delay should take place in applying them.

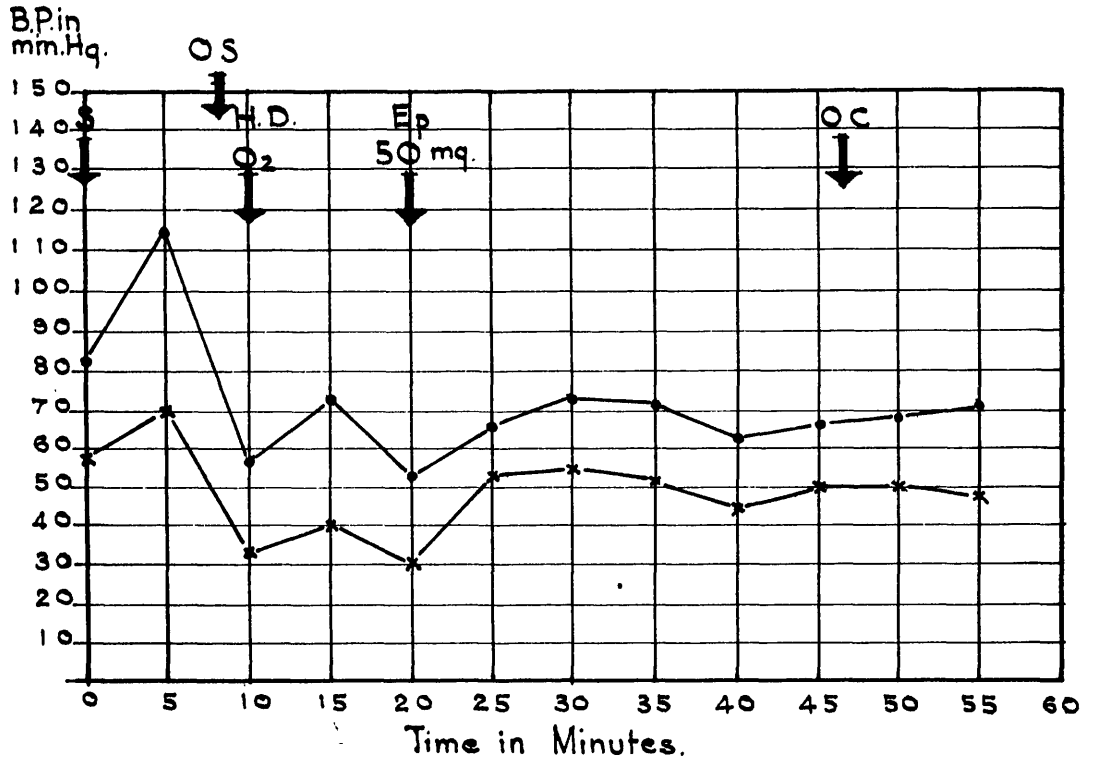
Spinal anaesthesia and Shock

O'Shaughnessy and Slome (1935) gave spinal anaesthesia to nine cats and then inflicted trauma only after the knee-jerks were abolished. In almost every case the blood-pressure at the end of the first hour fell only a little while in the control group a fall to shock level (60 mm. Hg.) is usual in this period. They say "Even if we are wrong in the emphasis we lay upon the discharge of nociceptive impulses, the beneficial effect of spinal anaesthesia remains a fact which seems worthy of note and further investigation".

Slome and O'Shaugnessy (1938) discussing the nervous factor in traumatic shock state "If transfusion of blood is accompanied by spinal anaesthesia, the rise in blood-pressure produced by the transfusion is maintained; otherwise blood transfusion is not efficacious."

SACHS (1941) subscribing to the theory that the essence of shock is loss of plasma proteins, investigated this under ether, avertin and ether, spinal, nitrous-oxide and caudal anaesthesias and finding no change in plasma protein, haemoglobin or blood hydrogen-ion concentration under any of these, concludes that anaesthetics play no part in the production of shock.

"The essential pathological change in shock is now thought to be such a diminution in the total volume of circulating blood that the compensatory mechanisms (such as arterial contraction and the discharge of blood from the spleen) are insufficient and the cascular system is no longer adequately filled. This leads to such effects as a fall in blood-pressure, tissue anoxia, etc.. This conception differentiates true shock from depressed conditions of the circulation such as that seen during spinal analgesia". (Hewer - 1944). "The fundamental difference between the low blood-pressure in spinal shock and true shock is that in the former condition there is no change in the volume of the circulating blood. It is interesting to observe that if a spinal block be given to a patient in acute pain from an abdominal lesion, an immediate fall in blood-pressure accompanies the relief from pain. The suggestion has been made that this is due to the cessation of pressor impulses which have been passing via the posterior roots to the vaso-motor centre and which are the cause of the high blood-pressure often noted in the initial stages of shock (as first pointed out by Parsons and Gray in 1912)." (Hewer - 1944).



Blood-pressure chart. Female aged 28 years.
Anaesthetic. 4 cc. 0.2% percaine in 6% dextrose
Operation. Sub-total hysterectomy.

S. = spinal injection	O.S. = operation started
Ep. = ephredrine	O.C. = operation completed
H.D. = head-down position	O. = 100% oxygen started

Note. a) The rise in blood-pressure following intrathecal injection without preliminary analeptic.
b) The response to adoption of the head-down position and 100% oxygen.
c) The response to ephedrine- the effect is more marked on the diastolic pressure.

The fall in blood-pressure during spinal anaesthesia.

Almost every one who gives a series of spinal anaesthetics is impressed by the fact that a fall in blood-pressure is very common. Few people have criticised the method without using this as a strong reason for not using it. The blood-pressure with spinal anaesthesia is certainly capricious; but it is not true that a fall in pressure is inevitable. It is even less true that it is disastrous and that the patient is even dangerously ill. There are many theories about the causes of the fall, - peripheral vasodilatation, diminished supply to the adrenal glands with resulting fall in circulating adrenaline, diminished cardiac output, loss of respiratory pump action from depressed circulation, traction upon mesenteries. But whatever the cause, the result is tissue asphyxia from depressed circulation. The treatment therefore is to diminish or abolish this tissue asphyxia. This can be done by giving analeptic drugs, and indeed many people give these prior to the spinal injection in cases of low blood pressure. But blood pressure is an individual pressure and varies greatly in apparently healthy people. The normal patient with a blood pressure of 100 mm. mercury will tolerate a fall in pressure much better than one with continued hypertension. My own belief is that the best way to tide the patient over the period of low pressure is to give continuous 100% oxygen inhalations: and, while I often see great falls, I believe that these are always temporary and that the patient can be safely tided over them. A low blood pressure before operation will not prevent me from giving a spinal anaesthetic - see chart opposite, where the patient underwent hysterectomy under intrathecal percaine. Her blood pressure prior to operation was 82/58. Following the spinal injection the pressure rose to 112/77. The chart explains itself. The patient did not cause me any alarm. High tension oxygen was given during the operation and the patient made an uneventful recovery. Since analeptics are commonly used and are very helpful, I propose to discuss them in the following pages.

THE CHEMISTRY OF ANAESTHESIA

The following remarks are taken from Adriani (1946):

Nuper caine (percaine) is a white neutral salt which forms salts with the mineral acids. The hydrochloride is very soluble in water, with a $_{p}H$ of 6.2 to 6.5, and its free base is readily precipitated by alkalies and alkaline substances. It is therefore prepared in distilled water and kept in alkali-free glass. The salts can be heated and are compatible with ephedrine hydrochloride. It gives prolonged anaesthesia lasting three to four hours.

Metycaine (also called neothesin) has somewhat similar properties. Solutions are self-sterilising, though the salt can be sterilised by heat without decomposition. Prepared solutions are destroyed by moulds and bacteria, which can be prevented by adding chlorbutanol. Ephedrine can be added and thiourea will prevent the decomposition of metycaine-ephedrine mixtures.

Procaine was synthesised by Einhorn in 1905. It has many other names, the commonest being ethocaine, novocaine, neocaine, and planocaine. A base, its salts are readily soluble in water, but such solutions are readily decomposed by bacteria. Solutions of the nitrate and hydrochloride are acid, the latter being the commonly used salt whose solutions have a $_{p}H$ of 6.0, and which must therefore be stored in alkali-free glass. Solutions of the borate are alkaline ($_{p}H$ 8.1).

Pontocaine (also called pantocaine and tetracaine) is usually used as the hydrochloride whose aqueous solutions are almost neutral, but the free base separates on the addition of alkali.

Parasympathetic depressants. Atropine and hyoscine produce their effects by decreasing the amount of acetylcholine formed, by increasing its rate of destruction or by inhibiting its action on end-organs. Parasympathetic stimulation can be produced by the inhibition of the activity of cholesterinase (as by morphia) or by increasing the production of acetylcholine.

SYMPATHETICO-MIMETIC DRUGS. A substance called "sympathin" is liberated at the nerve-endings of sympathetic fibres. It is believed not to be a simple substance but a combination of two types, sympathin E and sympathin I. "E" is an excitor and gives the effects of sympathetic stimulation. "I" is an inhibitor. The existence of these two types explains the different responses to sympathetic stimulation. In addition to sympathin, the hormone epinephrine produces effects of sympathetic stimulation. The precise nature of sympathin is not known. It is believed to be similar to though not identical with epinephrine. Epinephrine is destroyed by an enzyme, amino-oxydase, believed to be widely distributed in living tissues. The sympatheticomimetic drugs are all amines, deriverative of phenylethylamine, which can be varied to give such drugs as ephedrine, synephrine, neosynephrine, cobefrin and paradrine.

Epinephrine is the hormone from the medullary portion of the adrenal gland. It is quickly destroyed by the liver.

Epredrine is either laevo-ephedrine or dextro-pseudo-ephedrine. The laeve- form alone (the offocial substance) exists in the plant Ma Huang, while other plants contain mixtures. The synthetic form ephitonin has both forms and has less action than the pure laevo-form. It is now possible to get synthetic laevo-ephedrine. Ephedrine has the same physiological properties as epinephrin but lasts over a longer period of time. It is also stable to air, light and pH changes and can be boiled without inactivation.

Neosynephrine is also widely used in spinal anaesthesia. All of these substances are chiefly detoxicated in the liver.

These sympathetico-mimetic drugs are extensively used in spinal anaesthesia for the prevention or correction of the fall in blood-pressure which is such a common accompanying phenomenon, and much has been written about them. Thus Babcock (1906) quotes Klopp as having shown the value of adrenalin and epinephrine in delaying the absorption of local anaesthetic agents.

Pitkin (1929) claimed that "Ephedrine employed in an amount proportionate to the height of anaesthesia will stabilise the blood-pressure." "For anaesthesia reaching the seventh thoracic segment, 66 mg. of ephedrine will at all times stabilise the blood-pressure." But Pitkin is in a class by himself; no one else can achieve his remarkable results, few will support his claims and I think that many of his statements are frankly absurd, for example his statement that his preparations give "no shock or fall in blood-pressure". He further claims that the rate of absorption depends not on the presence of vaso-constrictors but on the viscosity of the solution. He agrees that ephedrine prolongs the effect of the anaesthetic, when added to it.

Bittrick (1939) records his results with neosynephrin in two hundred and twenty cases to whom it was given as a prophylactic routine, in doses of $7\frac{1}{2}$ mg. for high spinals and 5 mg. for low. His results are:

Result	No. of Cases	%	
Satisfactory	204	92%	(B.P. + or maintained)
Excessive	13	5.9%	(B.P. raised 50 mg. or more)
Intermediate	3	1.3%	(B.P. maintained at or above 80 mm. Hg.).

No blood-pressure below 80 mm. Hg. was recorded in the whole group.

CHAIKOFF (1940) reviews the attempts made to prevent or overcome the fall in blood-pressure. He says Jonnesco and Pitkin tried intrathecal strychnine for many years and found it useless. Babcock gave intravenous saline with enough adrenalin to maintain the blood-pressure level at 100 mm. Hg. and found that he had used 80 to 90 minims at the end of the operation. The effects were so variable and so short-lived and often so alarming that he abandoned the method. He says pitressin may be bad on account if its effect in constriction the coronary arteries, and quotes Essex, Herrick, Mann, and Wegria as saying that experimentally, in dogs, pitressin decreased the flow through the heart by eighty per cent. Chen (1924) extracted ephedrine from the plant Ma Huang. It is a powerful vaso-constrictor, and like adrenalin, dilates the coronaries; but one dose does not seem to be enough and subsequent doses are not so effective in time or height. Melville and Stehle (1931) showed that the combination of pitressin and ephredrin had a synergistic effect. The chief effect was widening of the pulse pressure. Chaikoff says there is no harm in overdose and recommends that the combination be used also for the prevention or relief of post-operative distension.

The same writer (1947) analyses 1,440 administrations and concludes that ephredrine alone or ephedrine and pitressin (gr. ½ and 0.5 cc.) will give adequate maintenance in lower abdominal operations but that for high spinals the combination should be used twice, before the spinal injection and before the incision. If the drop is severe, he gives the second dose intravenously. A third dose of the mixture may be given if necessary.

Many others have written on the effects of different pressor substances. Thus, Rochberg and Apgar (1942) found the combination of ephedrine and epinephrine satisfactory in high spinals. Melville (1946) prefers epredrine and pitressin and states that the best combination is in the ratio of 4.8 mg. ephredrine to each unit of pressor pituitary extract (postlobin-V). Dodd and Prescott (1943) and Anderson (1946) favour methredrine; Roman-Vega and Adriani (1946) like oenethyl; Bittrick (1939) and Thomas and Sica (1946) used neo-synephrine. Altschule and Gilman (1939) and Nathanson, Engelberg and Hersh (1942) report on the success of paredrine: Lorhan and Mosser (1947) are impressed with propadrine; and Jackson (1947) got good results with "EA-83" which he thinks acts best in small doses (10 mg.) repeated as necessary. Most of these writers discuss the undesirable side-effects produced by different vaso-pressors in making their choice.

THE USE OF PRESSOR SUBSTANCES ADDED TO THE SPINAL ANAESTHETIC SOLUTION

Various authors have described the effects of combining pressors with different anaesthetic solutions. Potter and Whitacre (1946) and Campbell, Crane and Sankey (1947) used pontocaine-dextrose-ephedrine with satisfaction, and Romberger (1943) in a not very modest article ultimately preferred ephredrine and procaine. Pitkin (1940) added suprarenin 0.36 mg. and ephredrine 50 mg. to his heavy and light solutions, with great satisfaction. All these writers emphasise that the addition of the pressor substance to the anaesthetic agent enhances and prolongs the effect of the latter, making a reduction in its dosage possible.

I use ephedrine in doses of 25-50 mg. when I think it is indicated. My first ampoules were made up of 50 mg. in 2.5% solution using the active principle extracted from the plant Ma Huang, bought in the local market in Kuala Lumpur, by Mr. Millard, Superintending Pharmaceutical Chemist of the Malayan Medical Service.

EPHEDRINE AS A SPINAL ANAESTHETIC AGENT

Schultz (1940) conducted some experiments with ephedrine hydrochloride dissolved in 0.7% saline. Small quantities (0.4 c.c.) of different strength solutions of ephedrine were injected intracutaneously into the flexor surface of the forearm and the response to pin pricks compared with the adjoining area. He found that the minimal effective concentration of ephedrine was 0.1%. The solution was very irritating while being injected; but this soon disappeared. Anaesthesia lasted five to eight minutes where 0.1% and 0.5% solution respectively were injected. Schultz then tried to block nerves using the sciatic of the frog. He then injected 0.03 c.c. to 0.15 c.c. intraspinally in frogs. He concluded that 5% solutions can block the sciatic nerve in frogs. The minimal dose for spinal anaesthesia in frogs was 0.1 milligrams per gram of body weight. Intracutaneous injection of 0.1% solution produces anaesthesia in humans. All these effects are completely reversible. Concentration of 5% and 10% produced no anaesthesia when applied to the cornea of rabbits.

Ruben et alia (1948) prior to administering spinal anaesthesia injected fifty milligrams of ephedrine sulphate in triple distilled water mixed with one c.c. of cerebro-spinal fluid. For a period of twenty minutes after injection the patients were tested for loss of pin prick sensation and of anal sphincter tone. The effects were usually noted after ten minutes or more and were most frequently seen in the area supplied by the second, third and fourth sacral nerves. Some loss of sensation was noted as high as the eighth dorsal nerve. All of fifteen patients showed some diminition of pin prick sensation which varied from slight hypaesthesia to anaesthesia. Anal sphincter tone was diminished enough to allow the passage of two or more fingers without discomfort in the six patients tested for this. No other paresis was seen. None showed sympathetic effects, for example elevation of pulse or blood pressure. Many showed warming of legs with lumbar sympathetic paralysis and a few showed the fall in blood pressure seen in spinal anaesthesia. In some cases the ephedrine anaesthesia was enough for operation without the addition of a standard agent: as an example they quote a forty-years old diabetic female who underwent amputation of two toes and extensive incision and drainage of the foot, done under fifty milligrams of ephedrine only. This patient was not excessively pre-medicated, could answer questions and co-operated well. She later said she could feel the operation but had had no pain. They conclude, "The anaesthetic effect of ephedrine should be considered when adding this drug to spinal anaesthetic mixtures."

SOME SYNONYMS

For the following remarks I am indebted to Langton Hewer (1946) and Adriani (1946):

The literature on spinal anaesthesia is often confusing because of the large number of names used for the same anaesthetic agent or analeptic. Procaine is known as allocaine, ethocaine, kerocaine, neocaine, novocaine, planocaine, scurocaine, sevicaine and syncaine. It is also the analgesic principle in spinocaine, gravocaine, and duracaine.

Nupercaine is also called percaine and, in Russia, sovkain.

Pontocaine is also known as pantocaine, tetracaine, amethocaine, anethaine, butethanol, decicaine, dicaine.

Stovaine (amlyocaine) is known as Barker's solution which contains stovaine 5% and glucose 5%. S.G. 1.023. Chaput's formula contains stovaine in 10% solution, S.G. 1.080.

Other analgesics quoted in this thesis are intracaine and monocaine (formate). Metycaine is the same as neothesine, and is used in 10% solution for spinal anaesthesia.

Alypin, eucaine and B-eucaine are rarely used now.

Adrenaline is known as auprarenin, surrenin and epinephrin.

Spinine is a synthetic compound with an action similar to that of adrenaline. It is more stable and is used in about ten times the strength.

Cobefrin is the same as corbasil and dipheprolamin.

One would have to be a research chemist to keep track of all these.

The B.P. 1948 has not made things any easier by making cinchocain the official name for percaine and nupercaine.

FAILURE OF SPINAL ANAESTHESIA

A spinal anaesthesia may fail absolutely; that is, no anaesthesia or insufficient anaesthesia may result. Or the level may be inadequate. Or the anaesthesia is satisfactory while it lasts but passes off before the operation can be completed. It is for these reasons that so many reports of supplementary general anaesthesia have been reported.

Of course, a patient may express a wish to be put to sleep. I have often had this request, and have replied by asking him to accept the spinal injection first of all, assuring him that if he wanted to be put to sleep at any time I would arrange this. No patient, however nervous, has so far asked me to give a general anaesthetic after I had started the operation. But I have had to supplement with a general anaesthetic because of failure to produce adequate anaesthesia and now and then because I had not time to complete the operation. Since I started using percaine, I have never failed to get sufficient anaesthesia in degree, level or time and have not had to use a supplement.

Nitikman reported failure in four per cent of one hundred and fifty cases of spinal percaine anaesthesia for operations of the spinal column lasting one hour and ten minutes to five hours and twenty-five minutes. The everage duration was two and a half hours. Fifty operations lasted more than three hours.

Watter (1941) describes his experiences of two thousand, one hundred and seventy-four spinal anaesthetics in ten years. There were sixty-six administrators; the author gave one thousand and seventy-one. In addition there were fifty-four cases in which anaesthesia was not obtained in spite of successful lumbar puncture. He thinks that experience has nothing to do with failures where puncture is successful. The cause of failure lies in the agent itself. Attempts at lumbar puncture failed in only two patients.

Wilson (1934) on the other hand states that the percentage of failures is in proportion to the skill and knowledge of the anaesthetist.

Sebrechts (1934) insists that rachi-sensitive and rachi-resistant patients exist, and that these characteristics are familiar, saying that he has operated on a man and his three sons, all of whom proved refractory to spinal anaesthesia. He tests his patients one hour before operation by giving them 1 cc. of "sedol" (morphia 0.01 gm. and scopolamine 0.0004 gm. and determines their likely reaction to the spinal anaesthetic by their reaction to this-bold-and awake, normal or very sleepy.

Wilson (1945) records complete failures in 2%, but only 0.5% in his last 400 cases. He believes failure must be due to

entire extra-dural injection, and imperfect results to the incomplete injection of the drug. Blunt needles, blood drips and slow drips should be rectified before injection and this is often helped by corkscrewing the needle a little further in. In contrast to Sebrechts he states that the operator and not the patient is at fault. "It is easier to draw off cerebro-spinal fluid than to inject a solution, through the same needle, into the subarachnoid space." I agree with Wilson; the incidence of failures is in direct proportion to the ability and experience of the administrator; and I have never failed to get anaesthesia following successful puncture. Sometimes the anaesthesia has not been satisfactory, but this has always been when the agent had been put up in ordinary glass whose alkali had partly fixed the solution. This difficulty troubled me a little in my earlier years and it recurred in the immediate post-war period when I could not get proper ampoules. At that time I was using the only solution available, 1% novocaine put up in glass bottles. Now and then the resulting anaesthesia was very poor.

Mr. E.G.R., an obese plethoric European aged 29 years was admitted with acute appendicitis. Injection of 1 cc. of 1% novocaine solution failed to give anaesthesia and was followed by general anaesthesia - open ether. With much difficulty, appendicectomy was carried out through a right paramedian incision and the would closed. He developed a hernia through the scar which was successfully repaired by fascial suture three months later. This is the sort of case that argues ill for spinal anaesthesia.

PREMEDICATION

There is general agreement that a patient about to have an operation under general or spinal anaesthesia should be given some form of premedication. This serves two purposes; it allays apprehension and reduces the patient to a more neutral state - when he is neither a coward nor a hero. It also tends to reduce the amount of anaesthetic required or to protect the patient against its side-effects.

My early cases got morphia and atropine. Quite frankly I gave the morphia to calm the patient and the atropine in case I had to give a general anaesthetic. For a time I omitted the atropine and gave morphia alone; but I soon returned to the combination and am convinced that the addition of atropine reduces the incidence of nausea and vomiting.

Practice varies from only very minimal doses of a sedative to complete basal anaesthesia. Hill (1931) thought that atropine should be given to every patient undergoing spinal anaesthesia; that morphia was a help but depressed the respiratory centre and that a nervous patient should get muscular relaxation with spinal anaesthesia and unconsciousness with ether.

de Caux (1932) asks and answers the question "Why is premedication given?" Because it places the patient in a tranquil frame of mind, not only on humane grounds although the few hours preceding an operation are often the worst from the patient's point of view; but on physiological grounds for if tranquil he requires less anaesthetic and therefore makes a better recovery. His preference is for pantopon and scopolamine. The latter is the German name for preparations containing only laevo-rotatory hyoscine. Excitatory symptoms are due to the dextro-rotatory form the British Pharmacopia allows. He is chiefly concerned with premedication for general anaesthesia.

Sellman (1940) says that preoperative medication relieves anxiety and inhibits psychic shock; it aids the course of anaesthesia, insulates the patient from sound and the rattle of instruments and the noise of suction and lets the surgeon talk about the operation. It raises the threshold of local stimuli that cause retching and vomiting. The patient should be calm and drowsy but not stuporous. She quotes SISE as describing three stages:

a) where the patient is conscious but quiet and not apprehensive;
b) where central control is inhibited, making him restless, fearful and unco-operative. This stage should be avoided.
c) where he is relaxed and usually unconscious and amnesic.

Either a) or c) is good. Stage c) is best for spinal or regional anaesthesia. Lethargic patients respond to small doses better than the highly nervous ones. She uses larger doses for spinal than for

general anaesthesia, giving pantopon gr. 1/3 and scopolamine gr. 1/150 two hours before spinal anaesthesia. She thinks morphia causes retching, nausea and vomiting, especially when operating in the upper abdomen, and quotes her analysis of two hundred hysterectomies and two hundred cholecystectomies under spinal anaesthesia in support of this contention.

Wangeman and Hawk (1942) investigated the effects of morphia, atropine and scopolamine in human subjects and found that there was a slight effect on the blood-pressure with lowered pulse rate. After three to five minutes there was hunger pain and a sense of motility in the epigastrium, with a desire to defaecate, which subsided in ten minutes. For a period of twelve to eighteen hours attempts to change position or to get up produced nausea, which ceased when recumbency was resumed. Scopolamine produced vertigo, nausea, unsteadiness in gait, inability to remember what one had to say and inability to accommodate visually for from ten to twenty hours.

Erratum.
For "circular," read "circulatory"

ANAESTHESIA AT THE EXTREMES OF LIFE

My youngest subject for spinal anaesthesia was a twenty-three day old male Chinese suffering from strangulated inguinal hernia. He recovered. Other infants upon whom I have operated under spinal anaesthesia are two at three months (for intussception and strangulated hernia); one at eight months for intussusception and one at eleven months for hernia. Anaesthesia was perfect in all of them and they all recovered.

At the other end of life, I have given spinal anaesthetics on 45 occasions to patients aged 65 and over in this series. Of these 27 were given at the age of 70 or over. There have been three deaths, a stab wound of the abdomen at 65, a strangulated hernia at 70, and a cancer of the stomach at 74.

Baird (1943) analyses 461 patients over 70 years of age to whom 673 anaesthetics were administered for acute and chronic illnesses. These patients varied from good risks (if that is possible at the age of 70) to almost moribund cases. Discussing fatal complications, he states that the largest percentage involving the circular system followed when spinal anaesthesia was given. On the other hand the mortality rate with spinals (17) out of 181) was the lowest in the series apart from pentothal which was used only for minor procedures. In poor risks, cyclopropane contributed to a mortality of 13.4% compared with 6.7% where spinal anaesthesia was used. In fair risks the figures were 6.7% and 3.04%; in good risks 3.1% of deaths followed cyclo-propane and no deaths occurred where spinal anaesthesia was used. He insists on getting the senile patient out of bed so that he can support his failing systems. Shock manifests itself early and tends to be severe in the aged. The judicious use of fluids, blood, plasma and oxygen is often the key-note of success, combined if necessary with vitamin- and chemo-therapy.

Dillon (1947) reports 909 anaesthetics in 905 patients. Spinal anaesthesia was used in 507 (55%) but was not employed if the blood pressure was over 180 millimetres of mercury. If the blood pressure fell 20 millimetres after premedication, if the haemoglobin was less than 12 grams, if disease of the central nervous system existed, or in cases of recent shock, the dosage was kept small; 100 mgm. of procain being the maximum and infrequently used. Small doses of ephedrine were given preventively. The anaesthesia was kept below the 12th dorsal segment, and unilateral where possible. He insists on the importance of supportive therapy, replacement and oxygen, the avoidance of bleeding, and, should bleeding occur, its replacement

at the time of loss. Early mobilisation is important. The death rate following spinal anaesthesia was low, being 4.3% compared with 14.9%, 14.5% and 10% where inhalation, regional and refrigeration anaesthesia was used. One death occurred on the table, in a patient who had a spinal anaesthetic. The patient showed no signs of blood pressure fall or distress but suddenly complained of pain in the chest and died. No autopsy was performed and this was probably a "coronary episode".

SPINAL ANAESTHESIA IN HEART DISEASE

Belinkoff (1946) discusses the choice of anaesthesia in coronary disease in decompensation and hypertension. In coronary arterial disease, he recommends pre-operative sedation, - erring slightly of the side of too much, - the use of plenty of oxygen and the avoidance of overloading with infusions. He states that spinal anaesthesia is usually contra-indicated on account of the liability to sudden fall in blood pressure. If spinal anaesthesia is chosen he insists upon the use of pressors and 100% oxygen. In decompensation he says the choice is spinal anaesthesia and quotes, Sarnoff and Farr (1944) who used spinal anaesthesia in the treatment of severe cardiac failure with pulmonary oedema where it acts as a bloodless phlebotomy. He describes hypertensive disease under two types - where the systolic pressure alone is raised and where both systolic and diastolic pressure are raised. The diastolic rise is more significant since the coronary arteries depend on it for their blood supply. In those with a high diastolic pressure spinal anaesthesia is contra-indicated for any intra-abdominal operation, where a sharp fall in blood pressure might be anticipated but it is safe for other procedures. He considers it wise to administer 100% oxygen, insists upon urine analysis and blood chemistry and advises spinal anaesthesia where there is much kidney damage.

Reid (1941) advises administering vitamin B complex in cardiac cases, and the avoidance of irritation to the respiratory tract. At the menopause adequate oestrin should be given. Grant White (1937) is of opinion that patients with cardio-vascular disease do well if plenty of oxygen is administered and he regards spinal anaesthesia as being suitable for these cases, and also in cases of respiratory and renal disease, toxic goitre and acidoctic states. On the other hand Morrison (1948) reviews 701 operations on 478 patients in a period of ten years and concludes, "No single anaesthetic agent is definitely superior for patients with heart disease. However, it seems that local anaesthesia should be used if feasible, and spinal anaesthesia avoided if possible."

SPINAL ANALGESIA FOR THERAPY

PULMONARY OEDEMA.

Sarnoff and Farr (1944) quote Koster (1928) as saying "spinal anaesthesia has the effect of a bloodless phlebotomy," and after discussing the mechanism of the production of pulmonary oedema say that spinal anaesthesia diminishes venous return to a greater extent than any other known means, and without reducing the cell count or haemoglobin percentage. The effect can be achieved by continuous spinal anaesthesia or injection could be repeated as indicated. They report three cases who improved rapidly under spinal analgesia.

THYROID DISEASE.

Although it is possible to perform the operation of thyroidectomy under spinal anaesthesia, few people would consider this: the analgesia would have to include segments two to four of the cervical cord. Spinal anaesthesia has been used with benefit in cases of hyperthyroidism. Arguing that adrenal hyperactivity plays a conspicuous part in the syndrome of hyperthyroidism: and that if adrenal activity and output can be reduced by spinal anaesthesia up to the fourth dorsal segment, cases have been described of its use (a) to treat post-operative thyroid storm and (b) to forestall the occurrence of severe immediate post-operative reactions.

Thus Rea (1944) used it to inhibit adrenal medullary releases during operation, and describes its use in "twenty cases, with real satisfaction". He describes his first case in detail and points out that operation had been attempted in the usual way and abandoned after the platysma was cut. Operation later on with spinal anaesthesia resulted in a smooth post-operative course where other measures had failed to get the basal metabolic rate below plus-sixty at any time. Spinal anaesthesia was induced by procain in doses of eighty milligrams to one hundred and twenty milligrams. No pressors were used and if the blood pressure fell the patient was put into Trendelenburg's position and given intravenous fluids.

Knight (1945) describes a similar case where the first attempt was abandoned, the patient having a rise in blood pressure. A second attempt three months later was successful, the patient being given 75 milligrams of procaine in the second lumbar inter-space, with anaesthesia up to the third intercostal space.

Campbell, Crane and Sankey (1947) discuss these cases. While most cases of toxic goitre can be managed without resort to

spinal anaesthesia, there are cases which show too much hyperactivity in spite of heavy sedation and basal anaesthesia, and for these specially active cases they use heavy sedation and if necessary, spinal analgesia.

Their plan is to give phenobarbital 4½ gr. morphia gr. ¼ and scopolamine gr. 1/100 pre-operatively. If the pulse is above 120 on arrival in the operation theatre, pentothal and oxygen are given. If there is little or no tendency for the pulse to slow down and it is decided to proceed with the operation, spinal anaesthesia is considered. They do not quote any cases or say what agent they use, remarking that "The longer-acting drugs are best."

PERIPHERAL ARTERIAL EMBOLISM.

Raymond Daley (1943) reports two cases in which the embolus passed on following the production of spinal anaesthesia, and he suggests that you should give spinal anaesthesia in the ward, leave the patient for half an hour and then determine if the embolus has passed on to a fresh site where gangrene will not follow and operation becomes unnecessary.

Agar (1943) describes five cases, four of whom had spinal anaesthesia. The third case had two emboli removed but the anaesthetic is not stated. If the embolism does not pass on to a "safe" area, embolectomy can be carried out under the spinal analgesia.

GASTRO-INTESTINAL ADRALASIA. (Cardio-spasm: congenital megacolon).

Margaret Hawksley (1944) points out that spinal anaesthesia was first advocated in cases of Megacolon by Stabins, Morton and Scott in America and says the literature is strangely silent on the important matter of high spinal anaesthesia. The analgesia must reach the sixth thoracic anterior roots. (Gray's anatomy suggests that you should anaesthetise from Thoracic five to lumbar three inclusive). She says the test should be not by anaesthesia but by motor paralysis, as it is the anterior roots that must be affected. She describes its use in twelve cases of whom six got one injection, five had two injections and one had three injections. She classifies six as "cured" five as "improving" and one as "no better". I recently treated a case on four occasions at intervals of ten to twenty-three days. The immediate effect is dramatic, especially if you pass a rectal tube, the abdomen rapidly deflating before the eyes. Although I tried to get this child brought to hospital for follow-up, I lost sight of it and do not know the ultimate result. Aird (1948) states that

in many centres of children's surgery recurrence of symptoms after sympathectomy and lumbar puncture has been so universal that there is already a trend towards colectomy; and he reports a case of such failure followed by successful colectomy.

Telford (1939) claims remarkably good results from spinal anaesthesia in four cases (three boys and one girl, whose ages varied from nine to thirteen years). His anaesthesia reached the third dorsal segment in every case. He states that "the temporary paralysis has caused the two halves of the autonomic nervous system to come again into step".

Hewer (1946) says the rationale in megacolon and mega-oesophagus is the possibility of renewed "step" in the autonomic nervous system.

Sarnoff, Arrowood and Chapman (1948) describe their technique for investigation of intestinal dyskinesia, colonic atony and visceral afferent fibres by the subarachnoid administration of a large volume of a dilute solution of procaine hydrochloride (0.2%). This is delivered from an elevated levelling bulb through a calibrated dripper to an inlying needle in the third lumbar interspace. The optimal initial dose is sixteen c.cs. in about four minutes. The drip is then continued at the rate of sixteen drops per minute until the desired block is accomplished and then set at that rate which will maintain the block until it is no longer required. Such a solution can produce a sympathetic block and a block of the fibres concerned with pin-prick sensation without grossly affecting touch, position sense, vibratory sense, or motor power. They use a four - balloon intestinal tube and describe seven cases in which their findings suggested that sympathectomy would be effective - repeated peristalsis and evacuation of gas, with a fall in the abdominal girth. Case six seems to have shown that fibres carrying impulses concerned with the sensations of intestinal distension are not influenced by that concentration of procaine which is capable of blocking sympathetic efferent and pin-prick fibres. This was confirmed in case seven. The response is believed to be more specific than that of conventional spinal block. They suggest that viscero-motor fibres are of about the same size and degree of myelenisation as other sympathetic efferents. Visceral afferents would seem to be larger and more heavily myelinated. Relief is not due to blocking of afferents concerned with the sensation of distension. It is important not to give morphia and scopolamine - indeed you should withhold all medication when attempting to augment intestinal transport by means of spinal analgesia.

SPINAL ANAESTHESIA IN CERTAIN SPECIAL SURGICAL PROCEDURES

There are many reports of the use of spinal anaesthesia in the surgical specialties, for example in thoracic surgery, obstetrics and orthopaedics. In an extraordinary paper, Koster and Kasman (1929) discussed spinal anaesthesia for the head, neck and thorax, and its relation to respiratory paralysis. In the previous one and a half years they had extended their use of this method to include ALL operations above the diaphragm except short trivial ones in ambulant cases; and they now report their experience of over seven hundred and fifty complete body anaesthesias! They encountered no serious respiratory or cardiac embarrassment and think the anaesthesia should be made to rise to, and not be prevented from rising to, the medulla. They give their dosages from the age of under two and upwards. They say the method is "fool-proof" and that they use it as routine for operations on the mastoid process. "In over two hundred and fifty out of four thousand five hundred cases the anaesthetist, whose only duty is to record the blood-pressure and respiration during the entire surgical procedure, notified us that it was impossible to secure a blood-pressure reading with the manometer. Of late, when we receive such information, nothing is advised. Our experience has taught us that ultimately the pressure will return and no untoward effect be occasioned as long as the patient remains in the Trendelenburg position." I think that their courage is equalled only by their foolhardiness. I would be very worried indeed if I were informed that no blood-pressure could be recorded; and the method certainly cannot possibly be "fool-proof". There is just no form of anaesthesia to which this term can be applied.

Lewis (1936) says that spinal anaesthesia is the ideal in bilateral lobectomy for bronchiectasis and describes a case in which he carried this out after an interval of eight weeks.

Magill (1936) agrees that spinal anaesthesia "is a safe and practical procedure for lower thoracoplasty and lobectomy" and quotes twenty-three cases, using Jones' technique. He states that you need the patient's co-operation and should keep this intact. It upsets previous conceptions about positive pressure in the presence of open pneumothorax. The patient is usually able to breathe quite well and oxygen is rarely needed for cyanosis. The cough reflex remains active throughout and sputum is voided with reasonable ease. There is usually a fall in blood-pressure but this causes no anxiety and it is significant that the fall is not so high as in upper abdominal operations. Retching is almost absent.

Edwards (1939) in a critical review of tumours of the lung, states "The great majority of pneumonectomies have been done under general anaesthesia, using either positive pharyngeal or intratracheal pressure ..... In this country, a considerable number of lobectomies and pneumonectomies have been carried out under spinal anaesthesia, with most satisfactory results." Percaine in 1: 1,500 solution was the rule, with consistent anaesthesia to the third thoracic segment.

Gowar (1941) in a clinical and experimental study of the post-operative complications of pulmonary lobectomy, concluded that collapse was more common when spinal anaesthesia was used.

Bourne et alia (1942) describe their results in thoracoplasty. One hundred patients had two hundred and eighty-two operation under spinal anaesthesia with nine deaths, four of which were due to the anaesthetic. Twenty-nine had sixty-five operations under other anaesthesias with eight deaths, none of which was due to the anaesthetic. There were fewer post-operative complications resulting in death following spinal anaesthesia.

Hewer (1946) says that the opinion is now generally held that coughing is nearly always harmful in thoracic surgery. It can never be completely effective with the patient lying on his sound side and with a mobile chest wall or open pnemothorax. It tends to spread infection and if prolonged leads to a marked deterioration in the patient's condition.

SPINAL ANAESTHESIA FOR GENITO-URINARY SURGERY

Harris (1939) says that for cystoscopy the choice at Saint Peter's Hospital for Stone is low spinal, because the patient is still conscious of over-distension and this is of value to the surgeon to avoid bleeding. The sensation is abolished if anaesthesia reaches the eleventh thoracic segment.

Andre (1942) quotes Woodbridge (1940) as saying that spinal anaesthesia has no harmful effect upon the kidney. For operations on the kidney, spinal anaesthesia is usually best. For suprapubic prostatectomy, she advises low spinal with small dosage in youngish and good-risk older subjects. In the more aged, she recommends local anaesthesia with a supplement. For short procedures, for example cystoscopy, she advises intravenous barbiturates.

Harbord (1943) reports ninety-four cases of prostatectomy under intrathecal novocaine, of which seventy eight were personally conducted. The average age was sixty-four years. Seventy-seven were admitted with acute retention. Six per cent were unsatisfactory and required supplement. Forty-three per cent had a first-stage operation, usually under local anaesthesia. The dosage of novocaine was usually one hundred milligrams. These were all poor-risk cases.

Bitterick (1945), in a not very convincing paper, advises spinal anaesthesia for urological procedures below the umbilicus; and that, especially in the aged, it is not safe above this level.

All my genito-urinary operations and most of my diagnostic cystocopies are carried out under spinal anaesthesia. A word of warning is necessary when doing retrograde pyelography with this method not to inject much opaque fluid, otherwise there is a danger of dilatation of the renal pelvis and calyces, with subsequent serious haemorrhage and the painful passage of ureteral casts of blood-clot.

I have performed prostatectomy eleven times under spinal anaesthesia. All eleven recovered. The oldest was 76 years of age.

SPINAL ANAESTHESIA IN ORTHOPAEDIC SURGERY

Rapoport (1941) says that most orthopaedists prefer general anaesthesia because the operations are often long, and hammering, chiselling and sawing disturb the patient. The orthopaedic table is not usually suitable for alterations in posture and besides, changes in position are not good for patients under spinal anaesthesia. He states that in his series, only three hundred and sixty-two out of four thousand three hundred and forty cases received spinal anaesthesia, but thinks a much larger number would be benefited by it.

Wardle (1945) says that for fixation of fractures of the femoral neck by pin and graft, you require an anaesthetic for aged patients which is safe, lasts an hour or more, produces no shock and leaves no serious after-effects, particularly in the chest. The ideal has been found in hypobaric nupercaine (1:1500) following premedication with omnopon and scopolamine. "If the blood non-protein nitrogen is not over 45 mg. % and the blood pressure is steady over two or three days, it can be given with safety whatever the patient's age."

Nitikman ( ) describes the use of nupercaine, in 1:1500 solution in one hundred and fifty cases who underwent operations on the vertebral column. The injection was performed in the prone position, which is the operating position. The operations lasted from one hour and ten minutes to five hours and twenty-five minutes, the average being two and a half hours. In fifty patients the operation lasted more than three hours. There were six failures and in addition an additional injection was given in seven per cent of cases. She was satisfied with the effects.

In the few cases of pinning I have done and in a larger number of intertrochanteric osteotomies I have performed on aged patients with fractures of the femoral neck, I have been well satisfied with spinal anaesthesia. By my method, the Trendelenburg position is unnecessary in these cases, and the procedure and subsequent plaster application are easily carried out on a Hawley table.

SPINAL ANAESTHESIA FOR OPERATIONS ON THE RECTUM

Bacon (1941) favours "lumbar" anaesthesia (by intrathecal injection) in ano-rectal conditions for operations lasting up to one and a-half hours.

Wilkinson (1942) used combined procaine and nupercaine spinal anaesthesia for abdomino-perineal resection of the rectum, the former preceding the latter by a period of two minutes. There were no deaths or respiratory embarrassment. Success was complete in twenty-eight cases. Four required supplementary nitrous-oxide-oxygen towards the end. One failure required cyclo-propane and ether.

Bowman (1946) is satisfied with spinal anaesthesia using novocaine crystals in the patient's own cerebro-spinal fluid, the injection being given in the sitting position, for haemorrhoids, fissures, fistulae et cetera. He uses only 25 mg.. I regard these conditions as an absolute indication for spinal anaesthesia in small dosage, irrespective of the age or condition of the patient. Involving, as they do, only the lowest (sacral) segments of the cord, they are ideal for a heavy solution which can be limited to this area.

SPINAL ANAESTHESIA IN OBSTETRICS.

Spinal anaesthesia in obstetrics can be used for vaginal delivery or for Caesarean section. De-lee and Greenhill state that spinal anaesthesia is the most dangerous form of anaesthesia for pregnant women, the theory being that in the bearing down stage, the anaesthesia fluid is forced up to the medulla. This view is supported by F.B. Mallinson (1938) who describes a fatality in an obese patient "with six weeks' amenorrhoea and slight bleeding for the last ten days". The pre-operative diagnosis was ruptured ectopic gestation. The patient showed depression, pallor and cyanosis after 45 minutes. The anaesthesia used was 14 c.c. of 1 in 1,500 percaine in the third lumbar space, the patient sitting by Etherington-Wilson's technique for 40 seconds. She received ephedrine gr. $1\frac{1}{2}$. No mention is made of the preparation itself, of the level of anaesthesia obtained or of blood-pressure records. He suggests that pregnancy makes a patient rachi-sensitive to spinal anaesthesia. The patient was put from the Trendelenburg position to the head up position for resuscitation, in spite of the fact that a light solution was used. The article suggests that lack of familiarity with the method is more to be blamed for the fatality than the method itself.

Cullen and Griffith (1947) discuss a series of 200 deliveries under spinal anaesthesia; and 200 under gas and oxygen. The spinal anaesthesia technique is to give 5 to 6 c.c. of 1% procain solution with the patient in the lateral position. The patient is lifted to the spinal position for 5 minutes and then put into the lithotomy position for delivery. Those showing signs of nervousness (42%) got cyclo-propane. The other results showed 20% of headaches in the mother in each series. Few cases of severe headache occurred; there were some nausea and vomiting in 20% of the gas cases. Only slight nausea and vomiting rarely occurred in spinal anaesthesia. There were no lung complications or other effects, except in case number 84, who complained of headache and blurred vision six hours later. Vision was almost normal after 24 hours and complete recovery took place in 48 hours. The child always showed better spontaneous respiration when spinal anaesthesia was used. The obstetrician noted a minimal amount of bleeding in the spinal anaesthesia series and said the low loss was "phenomenal", the average loss being 170 c.c. compared with 280 c.c. in the gas series. In a few cases the uterine contractions seemed diminished and required the higher application of forceps. They concluded that spinal anaesthesia offers safe relief from pain in vaginal deliveries, that the results are best if the dose is small and the solution dilute, that spontaneous respiration and oxygenation of the child are increased, that bleeding is less, and that headache is no more frequent, but may be more distressing.

Salb and Mueller (1946) tried spinal anaesthesia (procain 50 mgm.) in 250 cases of vaginal delivery. 11 were multipara, the rest were primipara. 206 were free from pain. Contractions continued but there was no bearing down or use of the accessory powers of labour. The anaesthesia lasted 50 to 70 minutes and gave enough time for draping etc., the application of forceps and repair of episiotomy. The loss of blood was estimated to be less, (it was not measured). They found difficulty in performing intra-uterine manoevres, for example in the three cases in which they had to effect manual removal of the placenta. Two infants were still born. The first was a 3 lbs 7 oz twin of a syphilitic mother; the second weighed 5 lbs 3 oz but was also syphilitic. They state that eclamptics, pre-eclamptics and nephritics are more safely delivered under spinal anaesthesia than under any other form of anaesthesia. They also state that it is safer where other diseases, for example diabetes, complicate pregnancy.

Cosgrove et Alia (1937) describe 2,789 cases in 3½ years under spinal anaesthesia, using novocaine in a maximum dose of 150 mgm. 2% were completely non-effectual and 6.4% were supplemented on account of the duration of labour. They had to revive two, one almost immediately and one after 33 minutes. Headache occurred in 17% of the cases which they compared with 3.3% under general anaesthesia and 2.7% where no anaesthetic was used. In half of the cases the headache was severe but did not last beyond one week. They record spinal anaesthesia as the most prompt and least time consuming, the simplest to apply, and the most efficient form of anaesthesia in obstetrics; there is less post-partum trouble, less bleeding and uterine tone remains good. It is safest for the complications of pregnancy, including diabetes which alone earns it its place in obstetrics. In the series 244 sections for delivery were performed under spinal anaesthesia, without immediate or remote mortality attributable to the anaesthesia and say that spinal anaesthesia is the best form of anaesthesia for Caesarean section.

Spinal Anaesthesia for Caesarean section.

K.M. Heard (1946) reviews 360 cases of Caesarean section in 11 years by different surgeons under anaesthetists of varying experience and skill. The anaesthetics used were spinal 185 (51.4%). cyclo-propane or gas oxygen in 102 (28.3%) and ether, alone or in combination, in 73 (20.3%). In 19 only was ether used from the start, the remaining cases being gas failures. There were only two complete failures with spinal anaesthesia. In 23 more cases gas was given after the delivery of the baby.

In these series many agents were used, percain crystals, procain solution, pontocain, pontocain-niphanoid, pontocain-glucose and nupercain 1 in 1500. He states that the safest and most satisfactory agent was procain crystals dissolved in the patient's own cerebro-spinal fluid. He aims at obtaining anaesthesia to the xiphoid process, and says you should avoid higher spread. He suggests that relief to the mother by sedatives is impossible without affecting the baby. He therefore advises as little pre-operative sedation as possible, (but he makes no recommendations regarding drug or dosage); and advises the use of a prophylactic pressor, (ephedrine 30 mgm with neosymephrine 0.2 c.c.) 10 minutes before the insertion of 120 to 150 mgm procain crystals dissolved in the patient's cerebro-spinal fluid, through the third lumbar interspace. Referring to the effect on the baby, he points out that in 56 gas cases only 45 babies breathed spontaneously, 11 (20%) required resuscitative measures; with ether 20% required resuscitation (4 for prolonged periods and of these one died of atelectasis in five hours); with spinal anaesthesia only one in a 106 was depressed and all were normally developed babies.

Regarding the mother's condition, he says a fall in blood pressure does not always mean shock. A "harmless" fall of 10 to 40 millimetres of mercury occurs in 6.4% of spinal cases and only 1.9% of gas cases. A rise of 10 to 60 millimetres of mercury occurs in 15.1% of spinal cases and 20.5% of gas cases. The blood pressure under ether was more stable - 2.7% fell and 8.2% rose. In 23 cases marked shock accompanied a fall of more than 40 millimetres of mercury; in 9.7% under spinal anaesthesia, 3.9% under gas and only 1.3% with ether. He states "The effect on the baby is not judged by the first ten minutes, but by the first ten days. The operation is not a success till the mother is safely at home attending to the needs of her healthy child." He refers to spinal anaesthesia as "the protector of the baby in Caesarean section".

Batten (1943) reviews 96 cases of Caesarean section under spinal anaesthesia in 11 years using a procain mixture, with morphine and scopolamine in the ratio of 25 to 1. He gives 50 mgm of ephedrine unless there is hypertension. He discusses 25 more cases and says he urges spinal anaesthesia for Caesarean section.

Stanley (1943) states "intraspinal anaesthesia with or without 'balanced' anaesthesia stands near the top of the list of choices for Caesarean section operations".

Torrie (1945) describes 120 cases of Caesarean section under spinal anaesthesia. There were no maternal deaths or untoward sequelae. There were 7 foetal deaths not related to the anaesthesia, (5 were premature with central placenta praevia as the indication for Caesarean section. One had erythroblastosis foetalis. In the 7th case the cause of death was unknown.). He concludes that spinal

anaesthesia is good for Caesarean section but it tends to rise to higher levels than in other cases especially if there are good uterine contractions, the blood-pressure being more likely then to drop. Respiration is easily depressed if the anaesthesia goes too high.

Van Der Post (1938) describes three "disturbing" cases using Etherington-Wilson's technique. The first case was 29 years old (and looked rather ill). 1½ hours prior to Caesarean section she was given Omnopon grs. 1/3, scopolamine grs. 1/150. She received 10 c.c. 1 in 1500 procain and then lay for 20 seconds. She was then allowed to sit for 20 seconds, at the end of which she was given ephedrine grs. 1½. The operation was started after 10 minutes. At thirty minutes she had air-hunger and poor pulse. They put her into the sitting-up position and gave 5 c.c. of coramine and 3/20 of a grain of lobeline. After 10 minutes of anxiety she recovered. The second case, age 33 years, who had been in labour for 15 hours, received spinal anaesthesia exactly as the first case. The operation (Caesarean section) started at 5.30 p.m.. At 5.57 p.m., when the surgeon was sewing up, the anaesthetist "asked to be excused". The operation was complete at 6.10 p.m. at which time the house-surgeon, convinced that the patient was quite well, accompanied the surgeon to his car. When he got back, the patient was dead on the Trendelenburg trolley. In the third case, a 35 year old female had spinal anaesthesia at 2.20 p.m. and operation (for ectopic pregnancy) started 10 minutes later. Soon after 2.40 p.m. breathing ceased and the pulse became imperceptible. Administration of carbon dioxide, and the institution of cardiac massage etc. were of no avail. He criticises Etherington-Wilson's periods and ideas of diffusion but he gives no blood-pressure records and does not state the levels of anaesthesia obtained. His paper has been criticised by Etherington-Wilson himself, and by H. Brennan: also by Falkner Hill who says you should use a heavy solution.

"At the discussion on anaesthesia for Caesarean section at the Section of Obstetrics and Gynaecology of the Royal Society of Medicine, on March 31st: 1947, the preponderance seemed in favour of spinal anaesthesia, in an unusually interested meeting." (B.M.J. April 5th: Volume 1 .. 463 - 464.)

THE COMPLICATIONS OF SPINAL ANAESTHESIA

The injection of a spinal anaesthetic has in many cases been followed by some complication or sequela. These vary in incidence and in seriousness. Some, like nausea and vomiting, headache and backache, are very common indeed. These may be severe but they are temporary. A needle may break at the time of lumbar puncture. But the really serious ones are those involving the meninges, cord and cauda equina, caused by the introduction of infection or by injury to the intervertebral discs, cord or cauda equina at the time of injection.

Breaking of needles can occur but its incidence is lessened by using rustless steel needles tested before use and by making sure that the patient is quite still. The latter is more easily achieved if the puncture track is anaesthetised before the lumbar puncture needle is passed. This series has been free from this accident; but I have had two cases in which the needle broke in my pre-war cases. Immediate removal was effected in both. The removal is not easy.

Headache has been a bugbear since the time of Bier, three of whose six cases had severe headache and one of whose cases had slight headache. Bier, who as I have pointed out did not experience spinal anaesthesia, nevertheless developed headache as did his assistant Hildebrandt. It is worth noting that Bier says that after these experimental injections he and Hildebrandt "went out to dinner and drank wine and smoked several cigars". This may have contributed to their headaches.

The cause of post-spinal headache is not clear, and many theories have been put forward. Most people think it is due to seepage of cerebro-spinal fluid at the site of puncture. Wilson (1934) thinks this is unlikely to occur, as it is improbable that the puncture remains patent in the dura or that the puncture in the arachnoid remains open and opposite that in the dura; extra-dural fat is likely to act as a cork when the dura is pressed back by the cerebro-spinal fluid. Hewer (1941) says that in the true spinal headache the spinal pressure is always low. Wilson has further pointed out that in laminectomy a great deal of cerebro-spinal fluid is lost, yet headache is not a feature of these cases. Watson (1943) discusses the causes, includes aseptic meningitis with INCREASED intracranial pressure and psychoneurosis. He says the latter theory is supported by the fact that it is commoner after minor than after major operations, though the latter keep more quiet. He describes a case of psychoneurotic origin.

Headache is a very common symptom in all medical and surgical patients, and has many causes. It is a common accompaniment of disease and of anxiety states. Drugs and worry can cause it. I remember in my early cases how irritated I became when, as frequently happened, a patient told me that the ward Sister had told him he

would be certain to have headache if he had a spinal injection.

Headache is a common post-spinal symptom. In my own experience, it has been much more common among the more highly educated and "civilised" races. Though a common symptom in all members of the "coolie" class in the wards, it is rarely complained of as a post-spinal acquisition among them. It may be that, scared almost out of their wits, they are so surprised at their survival after an operation, and so conscious, perhaps, of their pain, that a headache is of little significance to them. I have a great admiration for the courage of these people who come to us like frightened children and yet place their lives in our "white Devil" hands with such confidence and a degree of gratitude that is beyond complaint. Undoubtedly, in the tropics at least, the "white" patient is much more likely to be the one who is introspective and enlarges upon all his symptoms.

Backache is a post-spinal symptom that has worried me, not only as a surgeon and anaesthetist, but as a patient. Once more, it is a symptom of the European patient. My own conviction, for what it is worth, is that it arises from paralysis of the postural musculature, the normal dorsi-lumbar curve being flattened out and strain being put upon the ligaments while the patient is lying on a hard flat unyielding table. It is rare to see an Asian, who cannot put his phalanges flat on a table and then extend the hand at the metacarpo-phalangeal joint to a right angle or beyond this. This degree of joint movement manifests itself everywhere in these people and is equally applicable to the joints of the spine. Moreover, they are accustomed to sleeping on a flat, hard bed. People who, accustomed to the luxury of a soft mattress and during the war years were guests of an enemy who failed to provide this luxury, found the art of sleeping to be an elusive one till they had acquired the habit of lying on their sides. In a personal experience of spinal anaesthesia at my own request, I confess that I was tortured by backache night and day for five days afterwards. The post-operative pain of a femoral herniotomy was nothing compared to this incessant racking backache. For many years, I have put a small pillow, about one and a half inches thick, under the backs of all patients undergoing spinal anaesthesia, and have insisted that this pillow remain under the back in the ward for at least twenty-four hours. I have been struck by the marked lowering in the incidence of backache. Where this precaution has been omitted, backache has been a constant and serious complaint, at least among my European patients. My conviction is that post-spinal backache is due to ligamentous strain, and is largely, if not totally, preventable. I think I should add that this type of backache is felt quite deep in the back, and is different from, for example, the pain of lumbago, crippling though this itself is. It is even different from the deep, racking pain of dengue fever. I have suffered all three and hope I appreciate their differences.

Nausea with or without vomiting is a frequent accompaniment of spinal anaesthesia. At one time its occurrence was disregarded as the "twenty minutes vomit" which could be expected. It is

extremely common in the early part, say from five to twenty minutes after the injection. The fact that it is often prevented and usually minimised by "putting the patient to sleep" with some form of general anaesthesia (evipan, penthothal, etc.) suggests that the spinal anaesthetic is not the only factor in its causation. If sleep ameliorates its incidence and intensity, there is reason to believe that psychological factors play a part. Unless profoundly drugged, a patient undergoing an operation is in an apprehensive state of mind and he reacts accordingly. Too, nausea and vomiting are certainly more common in vago-tonic subjects. Most of us are surely familiar with the reaction of the big, burly vago-tonic with some trifling injury, say a fracture of a phalanx, and are wise enough to see that he is at least sitting before we touch the painful area, otherwise we shall have difficulty in preventing him from falling on the floor when he faints, as he is almost certain to do. This fainting is accompanied by a feeling of nausea and a tendency to vomit. These are less suitable subjects for spinal anaesthesia than the apparently highly-strung sympathatetico-tonic subjects.

Nausea and sometimes vomiting are commoner with high spinals; but this is not necessarily purely because of the high level. There is no doubt that they are the rule where traction is exerted upon mesenteries. In my experience they have not been a disturbing feature after return to the ward.

Mc Carthy (1947) says that these early complications are associated with the physiological disturbance caused by the anaesthetic and the delayed ones are the late results of these disturbances.

Immediate one include vasomotor depression, respiratory depression, toxic reactions and nausea and vomiting. The cause of vaso motor depression is still controversial, but the writer says it is undoubtedly associated cephalad spread of the agent as with low blocks it is rare. Spinal anaesthesia produces intercostal paralysis, loss of muscular tone over the affected area, sympathetic block, and, if the level is to the fifth thoracic segment or higher, the sympathetic supply to the heart is blocked. Intercostal paralysis reduces the sucking action of the chest wall and therefore lessens the venous return. Medullary hypoxia may result and oxygen should always be given in high blocks. Loss of muscle tone reduces the venous return with consequent diminution in cardiac filling and therefore in cardiac output. Block of sympathetics causes vaso-dilatation in the affected area with reduction in the volume of circulation blood. If the sympathetic supply to the heart is cut off, there results a slowing of the pulse and a drop in blood-pressure from uninhibited vagus activity. Preliminary atropin to reduce vagal activity is beneficial. Most of the sympathetic depression is probably due to block of the nerve supply to the adrenals and all its manifestations may be alleviated by sympathomimetic drugs. All these disturbances may be due to direct action on the medulla from the cerebro-spinal fluid or through the

blood-stream? Neither is proved and you can give relatively larger doses locally without causing them.

Respiratory depression usually follows from the hypoxia caused by the vasomotor depression. But the agent may involve the third, fourth and fifth cervical segments with phrenic paralysis. Artificial respiration is here indicated as the accessory niscles are inadequate. He says that phrenic paralysis will seldom last more than twenty minutes.

The incidence of these various post-spinal symptoms has varied enormously . Pitkin has claimed that his methods have almost abolished them, but for most of us they are a constant source of anxiety and a search for means of prevention and amelioration. Headache varies from about two per cent to well over twenty per cent. Nausea and/or vomiting, common enough on the table, are often continued in the ward, as is backache. Many are the methods of treatment suggested.

PULMONARY COMPLICATIONS

Although these are not so common in this part of the world as they are in temperate climates, I have had my share of them as will be seen from the analysis of my cases. Among them have been three cases of pulmonary embolish, one of whom died. This was a 29-year old Dane who, ten days after an emergency appendicectomy, developed a warning haemorrhage which was repeated four days later. After the first of these I arranged everything in the ward so that if he got a massive one I could operate on him. On his 17th: post-operative day I was operating on another patient in my theatre when I got word that the young Dane had died. Asking my assistant to complete the operation I was engaged upon I hurried to the ward and carried out pulmonary embolectomy, removing long ribbon-shaped organised clots from both main branches of the pulmonary artery. The heart beat was restored by massage and kept up for 47 minutes after which it ceased to respond. In spite of continuous artificial respiration with 100% oxygen, automatic respiration was never re-established, and life was not restored.

Sise (1932) analyses the statistics of various clinics and concludes that the type of anaesthetic has little effect upon the incidence of post-operative pulmonary complications, though he thinks ether slightly increases their incidence. The type of operation is the dominant factor. Respiratory infection, acute or chronic, is an important cause, and inhalations of carbon dioxide after operation appears to lessen the incidence of atelectasis.

Griffiths (1934) says they are just as common after regional or spinal as after general anaesthesia. They are commonest in winter and after abdominal operations where the abdomen is opened. They are not so common if the peritoneum is not involved.

Taylor et alia (1937) studied the chest complications in 12,349 anaesthesias of all types. The cases in which procaine was used amount to 1,209 (9.8%) including local and spinal cases. In the spinal series, they were commonest when intercostal paralysis was produced, showing a rate of 15.5%. Most of these had upper abdominal operations and the figure compares with that of 20.8% for all upper abdominal cases. The chief factors were the degree of narcosis, the duration of operation, the type of operation and the surgical risk.

Apgar (1939) thinks respiratory depression the chief hazard and it was severe in 4% of her cases. All these had complete intercostal paralysis and varying degrees of phrenic paralysis. One of her patients suffered permanent mental damage. She thinks they are preventable with oxygen and advises against the use of carbon dioxide.

Lemmon (1944) had 45 pulmonary complications in 2,000 cases of serial spinal anaesthesia.

Harris (1942) says that spinal and local anaesthesia on the trunk produce intense pulmonary stasis of fixed duration, for anaesthesia to be efficient must be complete. Flexible control of inhalation anaesthesia permits this intensity and duration to be reduced.

Holmes (1948) compares the pulmonary complications in 2,064 consecutive cases of general and spinal anaesthesia. His figures are - 4.6% in 710 spinals to the 12th: dorsal segment or lower: Just under 2% in 52 cases up to the 3rd: dorsal segment: 1.85% in 1,290 cases of general anaesthesia. Most cases occurred in women in early middle life, and in the operations of appendicectomy and herniotomy. He concludes that spinal anaesthesia does not reduce the incidence of post-operative pulmonary complications.

It seems reasonable to assume that attempts should be made to increase lung ventilation during spinal anaesthesia by asking the patient to take deep breaths now and then during the operation and to continue deep breathing exercises after return to the ward.

CARDIOVASCULAR COMPLICATIONS

Hill (1931) quotes Labat as saying "The heart having light work to perform takes a rest during spinal anaesthesia and for this reason it is the anaesthetic of choice for all cardiovascular cases."

Taylor et alia (1937) found the cardiovascular complications in spinal anaesthesia to be 10.5%, which was less than with cyclopropane and equal to the incidence with ether., ethylene and gas-oxygen. Of these the fall in blood-pressure was greatest in their spinal cases and shock was six times commoner than with general anaesthesia. Arrythmias occurred in 3% of spinals, compared with 1.3% and 4.6% when ethylene and cyclopropane respectively were used.

Apgar (1939), in the very complete analysis of her 1076 cases of pontocaine spinal anaesthesia, had 12 auricular fibrillations not present pre-operatively, 4 bradycardias, 4 coronary occlusions, 15 pulmonary emboli, 17 cases of thrombophlebitis of a lower limb, 2 "cerebral accidents", and 14 cases of post-operative shock.

Uncommon complications and sequelae.

Fairlie (1932) and Latham (1942) have each described two cases of hiccough following spinal anaesthesia. Latham also reported two cases of urticaria. Apgar (1939) had a patient who developed paralysis agitans two weeks afterwards. Lemmon and Hager (1944) found anaesthesia of the lower lip appearing in a patient undergoing cystoscopy. It soon passed off. Snyder and Snyder quote three cases, from the literature, of the acute onset of syphilis following spinal anaesthesia and report a personal case which cleared up after two weeks anti-syphilitic therapy.

CEREBROSPINAL COMPLICATIONS AND SEQUELAE

It is possible that many of the phenomena of spinal anaesthesia have a nervous origin. Headache, backache, nausea and vomiting may be due to excitation caused by absorption, though it is noteworthy that they are unusual with much larger doses used locally.

Davies and others (1931) showed experimentally that the injection of a spinal anaesthetic may cause changes in the meninges, the cells and the fibres. The meninges show a sterile reaction with lymphocytes and subsequent scarring which is more marked with larger doses. The cells show swelling and oedema of the nuclear membrane and granularisation of the Nissl substance which may go on to complete cell destruction. The fibres show swelling and fragmentation of the axons and demyelinisation of the sheaths. These findings are confirmed by post-mortem examination on cases with persistent clinical evidence of neurological damage. Such changes are usually reversible, if indeed they always occur. So called aseptic meningitis may result from the use of strong solutions or from the unintended introduction of an irritant such as phenol or iodine. An ampoule may develop an invisible crack through which such irritant may enter it. Blood in the cerebrospinal fluid following puncture may act as an irritant and cause headache and backache. It is impressive to note the high incidence of septic meningitis. Surely this must be due to errors in technique.

Somberg and Goldberg (1945) say that spinal anaesthesia has been reported to have been followed by disturbances of sensation, stiff neck, involvement of the second, third, sixth, seventh, eighth and twelfth cranial nerves, impaired bladder and rectal function, hemiplegia, paraplegia, pyramidal tract degeneration, meningoencephalitis, polioencephalitis, myelitis, myelopathy, radiculitis, severe neuritis, septic and aseptic meningitis, aracnoiditis and cauda equina neuritis. They describe three cases, the first two of which show how easily these complications may not be thought by the patient to be related to the intrathecal injection. Neither of these patients informed either the surgeon or the anaesthetist of the appearance of paralytic symptoms. Both subsequently underwent laminectomy at which adhesive arachnoidal changes were found. The separation of these adhesions was followed by improvement though the second case was still greatly disabled. Their third case underwent extensive laminectomy and separation or arachnoidal adhesions, following which his condition was described only as "improved". In the first two cases the symptoms developed slowly over a period of several months. It is interesting to speculate how many similar cases there are whose symptoms are not thought to be due to a forgotten spinal anaesthesia.

MENINGITIS

Apart from the terrible tragedy of sudden death, surely there can be no more frightful complication of spinal anaesthesia than meningitis. I have had one case and I feel I must record it at once. It was preventable as I am sure it always is. The following is a description of the case and the circumstances. Unfortunately the case records were lost during the Japanese occupation of Malaya, but the facts are engraved in my memory. In the year 1938, a lady doctor in our service offered to help me by giving anaesthetics and I soon found that she was a good and careful anaesthetist. After some time she asked me if she could assist me at my operations when I was giving the patient a spinal anaesthetic. Keen as I was on her help, I had to refuse her, because she went in for the modern fashion in finger-nails- long claws, painted a vivid red, which on account of their length are incapable of being adequately rendered free of organisms by any amount of scrubbing up, however prolonged. I explained this to her as pleasantly as I could and forbade her from giving a spinal anaesthetic for this very reason. One day she did give, unknown to me, a spinal injection. The following day the patient had a temperature and it was soon evident that she had meningitis from which she died. This was a very salutary lesson to me and since then I have been particular to a point of fussiness about the need for adequate preparation of the proposed site of injection and the need for proper scrubbing up on the part of anyone proposing to give an intrathecal injection. In a long experience of spinal anaesthesia, this is the only case of meningitis I have had and I hope it will be my last. I could not be more convinced of anything than I am that meningitis is preventable, always.

Perhaps the most disastrous series recorded is that of Barrie (1941) who reported 11 cases of meningitis in 96 cases of spinal anaesthesia in a period of three months. Fortunately all except one recovered. The clinical signs and symptoms were similar in all cases. The temperature rose to 100 degrees or over in the first three days without noticeable meningeal irritation at that stage. All developed typical meningitis with abrupt onset on the seventh to the tenth day, mostly with drowsiness, photophobia, neck rigidity and a positive Kernig's sign. The blood showed a polymorphic leucocytosis. All except the fatal case were free from fever on the eighteenth day. Nine, examined four weeks afterwards, had no resisual signs or symptoms except slight lateral nystagmus. The fatal case became comatose and died after seven days. Smith and Smith (1941) undertook the investigation of this series and found that the cause lay in the water from a filter which was being used to wash the syringes. The same writer found that 89 (39.9%) of 223 unselected specimens of cerebro-spinal fluid were contaminated with non-pathogenic organisms by cultivation in simple peptone water and that many sterilised waters used for washing syringes were contaminated. Of ten specimens of C.S.F. collected "with reasonable aseptic precautions" nine were sterile and one contained a diptheroid.

It is astounding that as late as not a decade ago, anyone should put trust in water from a filter. It is even more astonishing that the same procedure should be carried out over a period of three months with more and more cases developing meningitis. The appearance of a "stitch abscess" should prompt a surgeon to undertake an immediate and thorough overhaul of the whole process of sterilisation in his theatre. Barrie is to be commended for his courage in publishing this series of disasters and it should serve as a warning to all that meningitis can so easily be caused. It is, too, a reminder that it is preventable.

Records of aseptic meningitis are fairly common. Reynolds and Wilson (1934) recorded three cases following diagnostic lumbar puncture and think that temporary meningitic symptoms may arise from an outpouring of cells, especially if blood appears; for example blood in the C.S.F. following spontaneous subarachnoid haemorrhage will produce stiffness of the neck and the appearance of Kernig's sign. Their three cases had also stupor, delirium and a pronounced increase in the cell content of the C.S.F.. The first case was suffering from cerebral syphilis and developed meningitic symptoms within twelve hours of the lumbar puncture. The following day his cell count was 2,900, all being polymorphs. The white cell count was only 6,800. He recovered in four days. The second case was suffering from chronic encephalitis, recovered from the aseptic meningitis within one week and died from chronic encephalitis three weeks later. Once more, the cell output was of polymorphs. The third case developed symptoms within eight hours and a polymorph outpouring of cells but recovered in three days. They add an interesting post-script of a case who had his lumbar puncture postponed for twenty-four hours and in the meantime died of an acute subarachnoid haemorrhage which they say would have been blamed on the spinal anaesthetic, with the chance of subsequent litigation.

Brock et alia (1936) quote the work of Wossildo (1908) and say that following the work of experimentalists one would hesitate to use spinal anaesthesia; yet Sullivan (1932) records a case who underwent spinal anaesthesia five times within a period of thirty-eight hours. They record seven instances of meningitis following different forms of spinal anaesthesia. The first two were presumably benign "aseptic" cases who developed symptoms slowly and recovered completely. The third case developed signs and symptoms following an automobile ride on the eighteenth post-operative day. She improved a little on the thirtieth day but still had ocular weakness and defective speech after a period of nineteen months. She is regarded as a case of polio-encephalitis. In the fourth case radiculitis and signs of mild cord involvement were still present after five months. The fifth case recovered from a severe cauda equina neuritis and an apparently independent form of myelopathy in the thoracic cord after a period of five months. In the sixth case, which seems to have had a neurological element from the beginning, there developed a cauda-equina syndrome. Chordotomy was performed three years later but he developed a transverse myelitis from which he

died. Great clinical and post-mortem details are given of the seventh case which showed extensive cord involvement leading to death three months after the intrathecal injection of 1 in 500 percaine.

These authors, discussing the aetiology, say there may be a direct chemo-toxic effect produced by the drug, and that there may be a tissue sensitivity in a few cases, but there is so far no way of testing for this. They do not state the total number of their spinal anaesthesia cases and so their incidence is undetermined. They recommend that spinal anaesthesia should "be restricted to a special group of individuals unable to withstand general anaesthesia".

Other reports of aseptic meningitis come from Adelman and Irwin (1946) who mention two cases that followed para-vertebral lumbar sympathetic blocks, in which they think that the subarachnoid space must have been entered and that the procaine had chemotoxic properties. Hurxthal (1932) describes one case who underwent lumbar puncture for some neurological complaint and developed meningeal symptoms of short duration. Six months later there were no signs and the patient was said to be greatly improved but whether from the meningeal attack or the neurological complaint is not made clear. Livingstone et alia (1943) quote Orkin (1936) who summarises 45,966 cases by twenty authors with an aseptic meningitis incidence of 0.26%. They themselves then report two cases of "chemical" meningitis following the intrathecal injection of novocain in doses of 150 and 140 mg., which cleared up after four and five days respectively.

The more serious and often fatal septic meningitis has been recorded frequently. Kremer (1945) thinks it would appear more frequently still if there was not reluctance to publish all cases. After quoting others, he describes seven cases in his own experience. One recovered after many months but "tired easily". The second case died following the intrathecal injection of infected penicillin containing calcium and the seventh had the same infection from the same batch of penicillin. The third one showed paralysis of pyramidal type with the apathetic appearance and character change seen after the effects of a severe closed head injury. The fourth case developed internal hydrocephalus as shown by ventricular tap and injection. His acute hydrocephalic attacks were relieved by ventricular tap, but the relief was very temporary. After subtemporal decompression had failed to cure his headaches, he was submitted to a posterior fossa decompression and a fistula was created between the cisterna magna and the deep cervical muscles. There was considerable distension of the ventricles but "natural cure" is said to have occurred in this case. Complete recovery took place in case 5, and the remaining two cases showed relapses. He believes that these relapses were possibly due to loculation and subsequent natural freeing of the loculated fluid. He discusses the theories of causation, and concludes that there is a low-grade meningitis due to the introduction of organisms at time of the lumbar puncture causing usually a chronic illness with a tendency to relapse. Adhesions may cause spinal block or hydrocephalus.

Ps. pyocyanea meningitis has been described apart from Kremer. Davidson (1947) records the occurrence in two successive patients in one day. Three patients underwent operation for inguinal hernia on April 16, 1945. All had spinal anaesthetisa. The first was uneventful; the second got meningitis and recovered. The third died of meningitis 16 weeks later. The source of the Ps. pyocyanea was not traced. Vuylsteke (1947) records four cases of meningitis following spinal anaesthesia, all with positive cultures. The first three were due to Ps. melanogenes and the fourth to Ps. pyocyanea. Three died, after 9, 76 and 114 days. The fourth recovered on the 92nd: day. They proved very resistant to treatment, the third getting 1,172 gm. sulphonamide in 114 days, without avail.

Symonds (1925) reports a case of septic meningitis due to Staphylococcus aureus who developed symptoms three days after lumbar puncture for suspected syphilis. There was thick pus in the C.S.F. and recovery followed forcible washing through a cisternal puncture with a needle in the lumbar area.

Evans (1945) records two cases of Ps. pyocyanea meningitis following spinal anaesthesia.

Ps. pyocyanea seems to cause a meningitis of very prolonged duration and a high death rate. Botterell and Magner (1945) suggest treatment by intravenous iodides. For B. coli infection Esker (1945) suggests a combination of sulphadiazene and urea.

Meningitis is an avoidable complication of spinal anaesthesia if proper care is exercised over every step in the procedure. The sources of infection are the skin of the operator's hands, the skin on the patient's back, the needles and syringes, the local anaesthetic used for the puncture, the spinal agent itself and any lotion used for rinsing the ampoules, needles or syringes. Where time permits, the area of puncture is prepared widely on the previous day just as it were going to be the site of operation. The administrator should similarly scrub-up, not casually but properly. I insist on a minimum of ten minutes for this. The intrathecal needle should be grasped by the butt end and the needle part be kept out of touch. The use of gloves is unnecessary and the unmistakeable sensation of the needle passing intrathecally is more clearly obtained if gloves are not used. This is, however, a matter of personal preference. A sterile towel is draped so that it curves and covers the area around the site of proposed puncture. This square is tuck under the patient's back as he is placed supine. It is elementary that no injection shall be given if there is any sepsis near the site of puncture. With regard to secondary meningitis occurring following lumbar puncture in the presence of a heavy bacteriaemia, Weed et alia (1920) said it was possible experimentally and Remson (1936) confirms this. It is a not unreasonable supposition.

Further steps to prevent infection lie in the care of ampoules. It is well known that labels are difficult to sterilise. Moreover, tiny invisible cracks may develop in an ampoule which has been sterilised and infection can get in through such cracks. The ideal is for all ampoules to be of a definite size for each solution to prevent confusion after the labels have been removed. Each different set of ampoules is kept in a labelled glass jar containing a coloured antiseptic, for example, methylene blue which is what I use. If a tiny crack develops, the coloured solution will find its way into the ampoule and colour the contents and if this happens, the ampoule is regarded as contaminated and discarded. That such can happen I have proved by personal experience. Recently (in October 1948) an ampoule containing ephedrine was found to be discoloured on being removed from the storage solution. The blue colour of the contained ephedrine solution was very evident. It is quite easy to see that this might well have occurred in an ampoule containing the solution for intrathecal injection and a septic meningitis could easily have resulted. I have been very struck on a number of occasions by the casual manner in which lumbar puncture is sometimes carried out, especially, if I may say so, on the Medical side of the House, and far from being surprised that infection can occur, I am surprised that it is not more frequent. Even to-day, when I decide to give a spinal anaesthetic in a ward, as I occasionally do to put a traction pin or wire through a bone or manipulate a fracture with the patient in his bed, I do so with considerable trepidation in spite of all the care that I exercise to avoid infection. When I do decide on this, I will allow no bed-making or dressings to be done in the ward until I have given the spinal in the ward, and of course I arrange to do it at an appropriate time to make this possible. Perhaps all these procedures are better carried out in the theatre; but I confess to a preference for doing such manipulations where the chance of later disturbance following the manipulation will be minimised. In many parts of this country, the patient has a long journey from the ward to the theatre and back. Indeed there is only one theatre in the whole country which is not a long way away from the nearest ward.

THE CAUDA EQUINA SYNDROME FOLLOWING SPINAL ANAESTHESIA

Apart from retention of urine, there have been no neurological disturbances in this series of cases, up to the time of discharge from hospital, No patient has reported since discharge with nerve symptoms. But remembering the cases described by Somberg and Goldberg it is possible that they may have occurred. The only case I have seen in consultation was K.S.K., a Chinese male aged 47 years, who underwent appendicectomy with drainage under heavy spinal percaine on 10.11.48. There was no retention of urine afterwards, nor did he complain of pain, anaesthesia or paralysis up to the time of his discharge from hospital on 4-12-48. On 17-1-49 he was admitted suffering from low back pain and inability to raise his legs, which he said had gradually developed over a period of five weeks. When I saw him on 22.1.49, he was confined to bed and could stand only with difficulty on account of severe pain in the lumbosacral area. Defaecation and micturition were normal and there was no residual urine. Wasting was present in the calf muscles. The left knee-jerk was elicited with difficulty, but there was no detectable anaesthesia. X-ray showed narrowing of the fourth lumbar interspace. I thought he had a disc lesion from injury at the time of lumbar puncture. As his pain and stiffness were marked, I manipulated his spine under pentothal anaesthesia in the ward. He discharged himself two days later saying he had no longer any pain could walk normally. He is now back at work and symptomless.

Ferguson and Watkins (1938) believe that these cases are much commoner than reports indicate and they report fourteen cases of which 13 occurred in a period of 13 months, although they could find only 16 cases reported in the literature up to that time. Their cases usually showed retention of urine which progressed to incontinence, incontinence of faeces with loss of sphincter tone, impairment of sensation in the saddle area or down the back of the thigh, and absent or reduced reflexes in one or both legs. They believe the basic lesion is the same in all. The fact that the rectal symptoms were often delayed suggests that the lesion is progressive. Three patients died and all were submitted to autopsy at which no definite abnormalities were found in the central nervous system. Discussing the causes, they think that trauma would not cause lesions so extensive. Inflammation would not cause their very early appearance. The drug was considered the likely factor. Most of their cases had heavy duracaine.

Nicholson and Eversole (1946) say that any neurological sign following spinal anaesthesia is ominous. They believe that the toxicity lies in the drug, though direct injury, inflammatory reaction or precipitation of latent neurological disease by injection of the drug may all play a part. They list some series of cases in which the incidence varied from 0 in 15,000 to Ferguson And Watkins' incidence of 14 in 1,710. They describe five cases from the Lahey Clinic. Three were cauda equina lesions, two of which were later

shown to have had spinal block resulting from metastatic carcinoma. Two had peroneal neuropathy shown by foot-drop. Two other cases were shown to have had cord tumours. They describe two further suspected cases, one of which recovered completely, and four cases seen in consultation.

They emphasise the importance of avoiding the presence of irritants in the solution, give some contraindications and say that if the level of anaesthesia is less than expected, a spinal block should be suspected.

Dinsdale (1947) says that Ferguson has pointed out that lumbar puncture is carried out in thousands of patients without these symptoms developing; there is no evidence that bleeding is the cause; it is too extensive to be caused by injury to one, two, or even three nerves, and distant ones occur. He says symptoms always date from the time of injection; but Somberg and Goldberg have pointed out that this is not always true. Alcohol has been injected without any permanent analgesia or motor paralysis. Lesions follow solutions that contained neither alcohol nor glycerine. He points out that MacDonald and Watkins failed to produce lesions in cats with 15% ethyl alcohol and 20% glycerine. He himself had no cases with 5% stovaine in glucose (Barker's solution) but had three following the 10% solution of Chaput. He is convinced that the solution should be as isotonic as possible.

I have said that there were no neurological disturbances in the present series. But I can remember two cases whose records were lost during the Japanese occupation of Malaya.

The first was in the year 1932 when a married woman (European) in the early thirties who had undergone hysterectomy under spinal procaine developed weakness and tingling and vascular disturbances of the hands soon afterwards. She was found to be suffering from bilateral cervical ribs. The operation had been carried out with the patient in a steep Trendelenburg position and it is probable that the resulting downward thrust exerted by the shoulder supports was the causal factor. The second was an unmarried Chinese girl in the middle twenties who had an ovarian cyst, removed also under spinal procaine, the dose being 1 cc. of 15% solution diluted with 3 cc. of cerebro-spinal fluid. She was discharged apparently well but three months later reported with gross wasting of the muscles of the right arm and hand and shoulder girdle. It is too long ago now for me to remember all the affected muscles but the intrinsic muscles of the hand were all wasted and she had a main en griffe. The deltoid and both pectoral muscles were very wasted. There was no sensory disturbance. I asked my Physician colleague to see her and he reported to me that she was a typical progressive muscular atrophy. This was a very striking and remarkable case because at the time I looked up her old notes and in them was a remark that two days after operation she had complained of tingling, numbness and

weakness in the right hand. Examination revealed no sensory loss. Is this just the coincidence of a case of progressive muscular atrophy developing symptoms at that particular time ? That is the easy explanation. Yet I cannot help feeling that the intrathecal injection had something to do with her symptoms, though I am at a loss to explain just how. This patient was not put in Trendelenburg's position.

LUMBAR PUNCTURE INJURIES: INJURIES TO THE INTERVERTEBRAL DISCS

Billington (1924) describes thirty-five cases with persistent back symptoms dating from an attack of acute meningitis for which all had had numerous lumbar punctures and injection of serum. Fourteen showed spinal osteo-athritis with positive X-ray findings. In twelve, the changes were limited to the third, fourth and fifth lumbar vertebrae and discs; and, of these, seven showed destruction of the fourth and fifth discs. Five patients had signs of spondylitis with negative X-rays. Sixteen had symptoms but no definite signs of spondylitis, and these were undoubted disc lesions, perhaps due to lumbar puncture.

Pease (1935) states that the posterior disc wall bulges slightly,- especially when the back is flexed, with resulting increased pressure within the disc. He thinks that in the cadaver it is difficult to know whether the disc has been struck or not and that it is easy to hit the disc, the vertebra, the inter-articular facet or the venous sinusoids in the vertebral body. He quotes three instances of disc injury, two of them following single and one following repeated lumbar puncture.

Milward and Grout (1936) describe five cases of disc injury following spinal anaesthesia, and think they all happened at the time of lumbar puncture.

Gellman (1940) says that so-called dry and bloody taps are due to ineptness, and promptly forgotten. He describes a case in which the second and fourth lumbar discs were injured during lumbar puncture on a fourteen-year old syphilitic girl. Her first puncture was on June 20th: 1926 and was symptomless. On September 4th:, three further lumbar punctures were attempted at different levels with immediate local pain which became continuous. She later developed the typical Schmorl's nodules regarded as diagnostic of disc disturbance. He is convinced that injury to the second and fourth lumbar discs occurred during lumbar puncture. His X-rays show injuries to the first and third lumbar discs and not the second and fourth, as he says.

Everett (1942) reports three cases of disc injury, the first following lumbar puncture, the second following repeated lumbar punctures, the third following spinal anaesthesia.

Baker (1947) describes a disc injury in a four year old child who had repeated lumbar punctures which was successful only after several attempts. Nine days later the child complained of back-ache. Three weeks later X'rays showed narrowing of the space between the second and third lumbar vertebrae. The child developed the typically disc lesion, which recovered. I consider this child was lucky not to have a nerve injury, as the second lumbar interspace is much too high to choose in a four year old child.

McCarthy (1947) thinks that extreme flexion alone during lumbar puncture may cause disc rupture with resulting nuclear prolapse.

CRANIAL NERVE PALSY

The chief example of this complication is that affecting the abducens nerve. A number of people have reported it, either alone or with some associated paralysis. Steinberg and Bishop (1946) report a case who complained of headache and diplopia which started on the fourth day, the patient showing paralysis of the right external rectus with decrease of sensibility and widening of the palpebral fissure. The headache lasted until the twentieth day and the diplopia ceased on the thirtieth day.

Milowsky and Betancourt (1945) describe a case of bilateral paralysis of the hypo-glossal and abducens nerves following spinal anaesthesia in a twenty-one year old patient who under-went an operation for urinary calculi. Anaesthesia reached the fifth dorsal segment following two hundred milligrams. of procaine intrathecally. Fifteen days later he developed double vision and difficulty in moving his tongue. The cerebro-spinal fluid was examined that day and found to be normal. Two weeks later, the tongue movements and speech were better and the eyes recovered one month after the onset. This patient showed pro-dromal symptoms of malaise and headache for two days prior to the onset of symptoms of paralysis.

Etherington Wilson (1945) reports ten cases in which diplopia followed spinal anaesthesia. It passed off rapidly, as a rule; more slowly in a few cases.

WOLTMAN (1936) cites a left abducens nerve palsy following ether anaesthesia. The patient recovered in seven days.

It is very difficult to explain the occurrence of abducens paralysis. Fairclough (1945) discusses the theories for susceptibility of the sixth cranial nerve. One reason given is that its long course exposes it more to injury. Actually, the fourth is the longest (75 millimetres) and the slenderest cranial nerve. Cushing thought that the most likely site of injury was between the pons and the occipital bone where the anterior inferior cerebellar artery crosses the nerve anteriorly at right angles, as it does in eighty per cent of people. The internal auditory nerve is also credited with injuring the sixth nerve in the same way. It is possible for the nerve to be injured where it bends over the angular apex of the petrous bone,— for example in cases of increased intra-cranial pressure, when the bone stem may be forced down the foramen magnum, and the nerve, being practically fixed at its two ends, is stretched and damaged as it bends over the petrous angle. Fairclough has a theory which, while not providing the basal cause of sixth nerve paralysis, accounts for it showing signs of injury more readily than any other cranial nerve, even if anatomical disadvantages did not exist.

In all intra-cranial conditions, toxaemias and mechanical disturbances, all the cranial contents are disturbed by the same factor: and frequently there comes a time when the sixth cranial nerve is first to show evidence of trauma. Phylogenetically, stereoscopic binocular vision is a lately acquired attribute, and may be the first to break down. This theory explains the double vision of the intoxicated. He reports ten cases. All had spinal percain 1 in 200. Seven had 2 c.c., two had 1.5 c.c., and one had 2.5 c.c.. They all said their headache and diplopia were the worst part of their experiences, and some said the headache was intolerable. One also had "double hearing" for a time and one had slight ptosis and anterior polar cataract which were congenital. These ten cases occurred in two thousand and twenty one cases of spinal anaesthesia.

MENTAL CHANGES FOLLOWING SPINAL ANAESTHESIA

Apgar (1938) reported one case of permanent mental damage in 1,076 pantocaine administrations.

Noble (1946) reports a case of asphyxia under spinal pontocaine, with subsequent clinical manifestations indicative of cerebral damage and later evidence of cerebral regeneration and recovery, the details of which are worth recording, for it is an example of an avoidable accident. An 18-year old male received morphine gr. ¼ and hyoscine gr. 1/100 forty-five minutes prior to operation for acute appendicitis. Following the intrathecal injection of 20 mg. pontocaine, in 1% solution with 2 cc. 10% dextrose, in the second lumbar interspace without barbotage, he was placed in a 10 degree Trendelenburg slope for ONE MINUTE, by which time the anaesthesia had reached the level of the third thoracic segment, and the table was levelled. A minute later he became pale, then cyanosed and respiration had ceased by the end of the third minute. Artificial respiration was carried out by the Sylvester method, using oxygen. After five minutes, the pulse ceased, with a rapid fall in blood-pressure. He was then given intracardiac adrenaline, endo-tracheal insufflation with direct administration of oxygen by manual pressure in the bag, and intravenous 5% glucose-saline to which was added neosynephrine. The cardiac arrest was estimated to have lasted five minutes, and respiratory arrest thirty minutes. During this time the unconscious patient had his appendix removed. After operation, the tube was removed; but cyanosis recurred. He had three clonic siezures in the ward. After twenty-four hours he regained consciousness but was still stuperous. He was still confused and euphoric on the fourth day, was improved by the seventh day and apparently quite normal by the end of the second week.

The points about this case are the large dose of premedication and the placing of the patient in a ten degree Trendelenburg slope for a period of one minute. A study of the behavious of fluids of different specific gravities, as shown by Wilson, makes it quite clear that the GREATER the slope, the SLOWER is the flow. Here, the anaesthetic undoubtedly reached the cisterna magna. The period of circulatory arrest is interesting, because it has been shown that the Betz cells cannot survive oxygen lack for more than about eight minutes, and the medullary centres for more than twenty minutes. It emphasises the importance of the avoidance of anoxia, and the value of oxygen in high concentration.

DIFFERENT TECHNIQUES OF SPINAL ANAESTHESIA

There is great variation in the methods used to achieve anaesthesia. These depend upon the choice of agent, whether an analeptic is added to it or used separately as indicated, and whether the solution is hypobaric, isobaric or hyperbaric. This last is the most important aspect to consider, since spread is chiefly dependent upon differences in specific gravity between the solution and the cerebro-spinal fluid. Few people use isobaric solutions - indeed it is impossible to say whether any given solution is isobaric, on account of the variation in specific gravity of the cerebro-spinal fluid. It used to be said that for high anaesthesia it was necessary to inject the fluid at a higher level; but this is no longer widely practised, though many techniques increase the dose, or the volume, to obtain a higher level. This is reasonable.

Procaine has the disadvantage that its effects pass off after some forty-five minutes, though it often gives very prolonged anaesthesia. Percaine can be depended upon to give complete analgesia and relaxation lasting for at least three hours and often much longer.

The favoured technique is not in itself as important as the necessity to appreciate what is going to happen to the fluid after injection and to have a clear grasp of all the details of method and management. It would be difficult to find two methods that differed more than Wilson's and mine. Wilson premedicates heavily, so that his patients have to be supported in the sitting position while the injection is given. He uses a light solution of percaine, allows the sitting position for a given number of seconds depending upon the level of anaesthesia he desires, and then puts the patient in a slight Trendelenburg position for the operation. I premedicate mildly, use a hyperbaric solution, give the injection with the patient in the left lateral position and then turn him on his back with the head acutely flexed, the trunk being in Trendelenburg's position except for very low anaesthesias. Yet I would not claim that my method or results are superior to Wilson's. During the early part of 1940 I had the privilege of seeing Wilson's mock spinal experiments which he kindly performed for me. I also saw his technique in practice. Both experiences impressed me very much. If I still use my old method, it is not because I am not certain that his way is not as satisfactory; it is only because I have been satisfied with my method and prefer to continue to tread a familiar path.

Serial spinal anaesthesia

Lemmon (1939) was not satisfied with the single dose method because of failure in level and degree, because it did not last long enough, and because of the difficulty of removing the anaesthetic agent if toxic symptoms appeared. In 1940 he reported two hundred cases without failure. With Paschal (1941) he presented observations on the first five hundred cases. With Hager (1944) he gave

observations on the first two thousand cases. He uses a special mattress and a malleable metal needle. The dosage has varied greatly, from 50 mg. procaine for prostatectomy in a patient aged eighty-five years to 2,200 mg. for a hysterectomy that lasted only thirty-five minutes. He gave as much as 250 mg. to a child to get anaesthesia satisfactory for appendicectomy. In the last two there must have been a slip-up or a slip-out.

Tuohy (1945) described a new method utilising a ureteral catheter. He maintains that any agent is satisfactory and that there is no need to use the longer-acting ones, and agrees that you can "de-anaesthetise" by washing out with normal saline.

Lee (1943, 1944 and 1945) states his experiences with this method, discusses the problem of turning the patient without disturbing the needle, describes an "obstetric helper" and explains its use in the removal of a patient from the trolley to the operation table and until the patient is postured.

Other series have been described by Haugen et alia (1942), Apgar (1942) and Martin et alia (1945). I have no experience of this method, since I have been able to carry out all my operative procedures without recourse to it (that is, with a single injection) since I have adopted percaine in place of procaine. I rarely perform operations occupying more than two hours and have only once performed an operation lasting more than two and a half hours. This last was a left hemi-colectomy for cancer and took me two hours and forty minutes to perform. It was satisfactorily completed with a single injection of 1 cc. of 1% novocaine. Let me admit that this was an usually long time for anaesthesia to last with this dose and I expected to have recourse to supplementary general anaesthesia to complete the operation. Most of the operations that I am called upon to perform can be completed within one hour; but it seems to me that serial spinal anaesthesia is a very distinct advance. As Tainter (1946) remarks, there is a great advantage in using a short-acting compound by continuous drip, if its effect can be terminated at will.

Fractional spinal anaesthesia appears to have great advantages. It has recently been postulated that very weak solutions do not paralyse the phrenic nerve. A continuous weak solution, otherwise effective, should therefore be safer than a strong one.

Why use a heavy solution ?

My own use of a heavy solution started long before Jones described his technique with hypobaric percaine. After three years of experience with my 1% solution of procaine, I returned to Britain in the year 1931 and saw Jones' technique through the courtesy of the senior anaesthetist at one of the teaching Hospitals. Of the first three cases, two were returned to the ward on account

of the collapse that had developed in the anaesthetic room following the injection and subsequent manipulation. I confess that I was not impressed. With a small degree of Trendelenburg tilt, there is not likely to be much difference in the level of anaesthetisation of the anterior and posterior roots. In these cases, the patient was turned on his face following the injection and then turned supine. If there is one thing I have learned about spinal anaesthesia it is the fact that patients under its influence do not tolerate alterations in position or early return to the ward. This was very clearly impressed upon me later on when I gave a large number of spinals to the air-raid casualties in Penang. It may well be that these had more serious injuries than we could realise in the great rush of work we had at that time (we admitted six hundred and six serious casualties to my wards on the first day of the raids). Jones' method involves moving the patient and turning him. Even Wilson's technique forces the patient to be put flat after the desired level of anaesthesia has been attained. With a heavy solution, the patient is postured to attain the desired level and can usually remain there until the end of the operation. If it is necessary to move him this can be done after the anaesthetic is fixed.

THE INDICATIONS FOR SPINAL ANAESTHESIA

Opinions of anaesthetistsvary: opinions of surgeons are even more divergent. Some will not allow its use while others use it on every possible occasion. Most regard the blood-pressure as a guide and will withhold spinal anaesthesia if this is at what they regard as being low. Few people will use it in the presence of shock, though the same people will prefer it if the proposed operation is one in which shock is likely to develop.

In my preface I have pointed out that a great indication for its use is the lack of a skilled general anaesthetist, especially where there is someone skilled in spinal anaesthesia. For many years now I have given spinal anaesthesia as a routine, whatever the condition of the patient. A patient is treated for shock and when he is considered fit for a general anaesthetic he is considered fit for a spinal anaesthetic if the operation demands it. I use it for everything whenever I can except for such trivial procedures as the opening of abscesses. On an earlier page I have given the blood-pressure chart of a recent case which shows that a preliminary low blood-pressure will not deter me from using it. So far I have had no reason to regret this practice.

Many contra-indications have been put forward. Examples of these are unlimited - a common one is in prostatectomy, because of the fear of reactionary haemorrhage of pre-renal anuria from circulatory depression. The small number of prostatectomies in this series have all been carried out without mishap, and they include all the cases during the whole period; no prostatectomy has been done under any other form of anaesthesia. If the number is small, I can only say that senile hypertrophy of the prostate was at one time believed not to occur in Orientals. This is certainly untrue; but the condition is uncommon in them.

If I have not said much on what is usually regarded as a most important aspect of spinal anaesthesia it is only because, in my practice, a patient fit for a general anaesthetic is fit for a spinal one.

DEATHS UNDER SPINAL ANAESTHESIA

Following the injection of a spinal anaesthetic, death may take place during the operation; or death may be delayed and take place in the ward. In either case one should endeavour to assess the part played by the anaesthetic in causing death. Adequate pre-operative investigation of the patients' general condition and fitness for operation, a consideration of all the facts during the anaesthesia and the post-mortem findings provide the data. The patients present two extremes, the apparently fit and young undergoing an operation of not great severity, and the desperately ill subject who even if not suffering from some surgical disaster is in such a state of reduced fitness on account of intercurrent disease (for example of the cardio-vascular system) that he would be a bad risk even for a planned operation allowing time for all possible preparation.

The only case in this series which occurred on the table in March 1948 was that of a young Chinese female aged 21 years, the mother of four children, who had not produced a child for two years and had a gross chronic cervicitis upon which I proposed to operate. She was given 3 cc. of 0.2% percaine in 6% glucose. She was injected, postured and prepared and draped in the usual way and I was just about to start the operation when she said she could not breathe (this in a voice that I did not hear), suddenly collapsed and died, all efforts at restoration failing to revive her. This is the sort of death that is so difficult to explain.

One goes on year after year giving a large number of spinal anaesthetics with success and satisfaction, never relaxing efforts to ensure the safety of the patient; and then, suddenly, an inexplicable and totally unexpected death occurs. Perhaps the most maddening feature of this case was that I was at a complete loss to understand the cause, and can only seek refuge in the possibility that a few - a very few - patients do have an idiosyncracy to the drug. No post-mortem examination was carried out in this case, the reason being that she was Chinese and of a very wealthy family. These people have very definite ideas about the sanctity of the dead body, and a post-mortem examination adds a degree of insult upon injury that we are at a loss to understand. No purpose would be served if I here gave a chapter upon their attitude towards death and a corpse, interesting as such is.

Many of us wish that some test of sensitivity could be found that would be workable in practice; I have had two deaths under local procaine anaesthesia; one in a thoracoplasty to whom I certainly gave, purely inadvertently, more than the recognised maximum dose: and another for a simple thyroid adenoma in which I observed the usual precautions of local injection. In the latter case, I am convinced that idiosyncracy existed. She certainly did not have an intravenous injection; yet she died, and no efforts could resuscitate her. How often does the general surgeon apply cocaine to a mucous surface without mishap, and yet occasionally a sudden death occurs. Surely

these are due to an abnormal, if not yet understood, idiosyncracy.

Perhaps some test for sensitivity will be developed. So far a skin test has not been proved valuable. It has been suggested that the fluid to be injected should be mixed with a few ccs. of cerebro-spinal fluid and the resulting mixture inspected. If precipitation occurs, the injection should not be given. Most of us will agree with Tainter (1946) that an antidote is urgently required.

MY PRESENT TECHNIQUE

Convinced of the immense importance of attention to detail, I now present my own technique in every particular.

Ampoules: Three sizes of ampoule are used -

a) one containing 5 cc. of 1-in-500 percaine in 6% dextrose.
b) one containing 2 cc. of 0.5% novocaine.
c) one containing 1 cc. of 5% ephedrine.

It will be noted that these ampoules are all of different sizes. There can therefore be no risk of confusing them. All are prepared by the Superintending Pharmaceutical Chemist of the Federation of Malaya Medical Department in Kuala Lumpur. He prepares them personally and does not leave any part of the work to an assistant. I know to what lengths he goes to ensure that all apparatus is really clean and free from grease and that the triple distilled water is pyrogen-free. When sterilised and sealed, samples from each batch are sent to the Institute for Medical Research in Kuala Lumpur to be tested for sterility. When this has been certified, they are packed in dozens and sent to me. They arrive labelled. I remove the labels and put each into a solution of methylene blue in alcohol which will show the presence of cracks at the time of using if the contents are seen to be discoloured.

Syringes: These are three in number, 5 cc., 2 cc., and 1 cc., for the contents of each ampoule. Each syringe is used for its own solution and for no other purpose.

Needles: Fine rustless steel hypodermic needles are used for the local anaesthetic and the ephedrine. The lumbar puncture needles are 22 gauge and made of rustless steel. A spinal awl is kept in readiness but is rarely used.

The patient: A sedative, usually luminal gr. 1, is given on the previous night. The lumbar area of the back is widely prepared as though it were to be the seat of operation and a sterile square is bandaged round the patient. On arrival in the theatre, the dressings are removed, the position is adjusted so that the patient's cervico-dorsal junction is placed at the part of the table where the head-piece joins the rest of the table. He is then turned on his left side.

The anaesthetist scrubs up as for an operation. I am particular to a point of fussiness about this and insist upon a minimum of ten minutes under running water. The syringes and needles and ampoules are washed in freshly sterilised distilled water and the syringes are filled with their appropriate solutions. Each filled syringe is then inspected for colour. The slightest bluish tinge indicates possible contamination and the contents are discarded.

Attention is then directed to the patient. The back is again widely prepared (I use 2½% iodine) and draped. The fingers of the left hand are placed on the crest of the ilium and the thumb then defines the fourth lumbar spine. A dermal wheal is raised with local anaesthetic over the fourth lumbar interspinous space and the tissues beneath this are similarly injected. A spinal needle is selected, inspected to make certain that its stilyette is fitting accurately, and lumbar puncture is carried out. This is done with the ungloved hand, care being taken that only the butt end of the needle is touched. I do not use gloves because the sensation of entering the intrathecal space is unmistakeable to the ungloved hand. A few drops of cerebro-spinal fluid are withdrawn into the syringe and the contents then rapidly injected. The back is covered with a sterile square and the patient turned supine with a small 1½ inch thick rubber pillow placed under the lumbar area. He is then postured. The head is acutely flexed. The remainder of the table is in a slight Trendelenburg position depending upon the level of anaesthesia it is wished to attain (except in low spinals, where the table is raised slightly). I confess that I always see that the level of anaesthesia is high enough and do not worry if it is slightly higher than absolutely necessary - the essential is to have it of at least adequate level.

Meanwhile everything else necessary is ready. The anaesthetist's table has a syringe and needle and an ampoule of ephredrine, a stomach tube and an endotracheal catheter, gag and prop. The gas-oxygen apparatus is at hand with the cylinders turned on at their mains so that high-pressure oxygen can be administered without delay.

At any time after ten minutes the patient can be put into any position desired as the anaesthetic solution is then fixed and no change in posture will alter the level of anaesthesia. By the time the operation area is sterilised and draped the patient is ready for operation.

An odd thing that has struck me about many theatres I have been privileged to visit has been the fact that while every member of the theatre staff has been gowned and masked, it is quite common for the patient under spinal anaesthesia to be without a mask. Surely this is an example of the carelessness that can happen in a theatre suite or surgical unit that is not properly organised. It is just as important for the patient to wear a mask as it is for those attending upon him. A mask in no way interferes with the administration of such accessory treatments as for example the administration of oxygen.

The patient's eyes are covered. Sometimes I put cotton-wool in the ears. Although I always use a goitre screen, I still cover the eyes; otherwise the patient can see what is going on in the reflecting surface of the scialytic lamp, in spite of the goitre screen.

Nowadays I rarely keep a regular chart of the blood-pressure, except in high spinals. There is no doubt that frequent readings disturb the patient. They often disturb the anaesthetist too. If the blood-pressure drops to 80 mm. Hg., high pressure oxygen is administered by the closed method. I used to give ephedrine as a routine but have stopped this practice for some years now. It is a useful stimulant if the patient's condition causes anxiety.

On return to the ward, the patient is put flat in bed without a pillow and he remains thus for twenty-four hours, with the small pillow under his back. If, after 24 hours, he has headache on sitting up, he is at once put flat again. Most headaches respond to posture, and the few severe ones respond to aspirin gr. 5, repeated as necessary. If he has not passed urine within eight hours, he is catheterised and an antiseptic dressing is bandaged over the penis and urethral orifice in the male and kept there till he passes urine himself. Until then he is catheterised eight-hourly. In females, an antiseptic swab is applied to the vulva, after swabbing the area, and is held in position by a sterile pad and T-bandage.

ANALYSIS OF THE SERIES OF CASES PRESENTED

The cases analysed in the following pages number 1,921, and are divided into four sections, as follows:

Period	Anaesthetic	Number
11.8.1940 to 8.12.1941	15% procaine	583
1.7.1946 to 21.10.1946	15% procaine	182
22.10.1946 to 18.3.1948	0.2% percaine in 6% dextrose	904
14.9.1948 to 31.1.1949	0.2% percaine in 6% dextrose	252
	Total	1,921

When the Japanese occupied Malaya they destroyed all the hospital records, and the only information I have of pre-war cases is from the theatre records in the general hospital in Penang. All these cases were given a maximum of 1 cc. of 15% procaine, usually mixed with a few cc. of cerebro-spinal fluid, up to a total injection not exceeding 5 cc.. They are included because they can be used for the total deaths and the failure incidence.

The second series was also a 15% procaine, the only difference being that instead of being in mica glass ampoules, it was put up in 25 cc. bottles of ordinary glass. Failures were much more common in this series. They were also carried out in Penang.

The third and fourth series are the same except that the third was done in Penang and the fourth at Ipoh, the intervening period being due to my absence on furlough in Britain. All these were given up to a maximum of 5 cc. of 0.2% percaine in 6% dextrose put up in ampoules made of mica glass.

Failures: 17 of the first 583 failed. None failed absolutely. In 2 the level of anaesthesia was just not quite high enough and were supplemented by local procaine. In 15, the anaesthesia did not last long enough and these were completed under general anaesthesia by gas-oxygen-ether.

In the second series, anaesthesia was effected in the same way, but from ordinary glass bottles, which were boiled every morning before use. This was at a time when I could get neither percaine nor proper ampoules. Anaesthesia was most unsatisfactory and operation could not be attempted in 24 out of 182 cases. In all these, operation was carried out under general anaesthesia with gas-oxygen-ether, or with open ether. Pentothal sodium was not used because at that time there had been a number of deaths resulting from its use in Malaya. Many batches were found to have deteriorated and in the end an order came from the Director of Medical Services forbiding its use. I may add that I had three deaths at that time following pentothal sodium.

The cause of the failures in so many cases was almost certainly

the method of storage of the procaine and the need for frequent re-sterilising. Doubtless the glass gave off more and more alkali which fixed the procaine.

In the third and fourth series, a total of 1,156 injections of percaine from ampoules was given without any sign of failure in level, degree, or length of anaesthesia.

Deaths: There has been one death on the table, to which I have already referred. It took place towards the end of the third series and followed the injection of 3 cc. of 0.2% percaine in 6% dextrose. The Coroner did not order an autopsy. After the inquiry the finding was merely "Death under an anaesthetic. No blame could be attached to anyone."

Deaths in the series: Out of a total of 1,921 spinal injections there were 26 deaths. These are summarised in Appendix A. It is always difficult to assess the part played by the anaesthetic. Unfortunately it is difficult to get permission for post-mortem examinations in this country except on a Police order.

Appendix A shows the cause of death, the age and the period after operation at which death took place. The only significant ones are the two cases of ruptured ectopic gestation and the caesarean section. All were young women. All had lost a lot of blood and were very shocked on admission. All received blood before and during operation. Yet they died at 16, 8 and 8 hours, after operation respectively. Did spinal anaesthesia contribute to their deaths ? I wish I could be quite certain that it did not. My experience is not large enough. The death rate in ruptured ectopic gestation was 11.1%; in Caesarean section it was 7.54%. Is it true that pregnant women are not suitable subjects for spinal anaesthesia ? On the whole I think it more probable that the shock and haemorrhage were the factors rather than the fact of pregnancy. It may well be that one or more of these patients could have been saved by some other form of anaesthesia in skilled hands.

The remaining deaths call for little comment, except that two gun-shot wounds and two stab-wounds of the abdomen died in 13 hours and two days. The stab wound that died after two days had injuries to the liver and gall-bladder. He died of peritonitis due to B. welchii. These four cases also suffered from haemorrhage with shock to which trauma contributed. Perhaps the cases that do worst under spinal anaesthesia are those with shock due to trauma and blood loss.

The death rate in this series has been what could be expected in a large number of operations that were not of great hazard.

Complications: These refer only to the last 1,338 cases.

Headache & Backache: The incidence in Europeans and Asians has been so different that I put them separately. There are no records of the first 583 cases.

	Europeans	Asians
Total cases ...	113	1,225
Headache ...	9	13
Backache ...	3	Nil.

No patient was asked if he had headache or backache. The figures given are of those who actually informed us that they had those symptoms. Only two had severe headache. Both were Europeans. One, a young man of 29 years, had severe headache for five days, hardly relieved by aspirin gr. 5, or even by posture. He told me he often suffered headache since he had had an air-crash three years earlier. The other was a European married woman who had severe headache for three days following an operation for haemorrhoids. She was 39 years of age, and had menopausal symptoms.

Nausea and vomiting: This has been by far the commonest complication. Some degree of nausea occurred on the table in 382 out of 1,125, that is, in 31.02%. Of these 26 vomited after the premedication and before operation. All had morphia gr. ¼ and atropin gr. 1/100, one hour before operation. The incidence was highest in the high anaesthesias; but this may have been due to the operative procedure.

The following shows the incidence in three zones:

	Number	Nausea	%	Vomited	%
High ..	212	175	83	141	66
Medium ..	1322	167	12.65	53	4.2
Low ..	387	3	0.77	Nil	Nil.

After return to the ward, only 27 cases vomited. None vomited, as a result of the anaesthetic, after twenty-four hours.

Respiratory:	Number	Died
Massive collapse of lung	4	1
Bronchopneumonia	7	1
"Bronchitis"	83	Nil.

	Number	Died
Cardio-vascular:		
Peripheral phlebitis, leg	3	Nil
Pulmonary embolism, certain	3	1
probable	2	2
Circulatory failure	3	3
Cerebro-spinal:		
Paralysis abducens nerve	7	Nil.

These were all in people who complained of double vision. Three were in Europeans. A very large number of my Asian patients are illiterate; many of these may have had an unnoticed paralysis. All cleared up in four days.

No case of meningitis or cauda equina involvement occurred.

SUMMARY

A review of the literature on spinal anaesthesia is presented. The recorded experiences and opinions of others following experimental and clinical injection have been criticised and commented upon. It has been noted that all writers have observed contraindications. Spinal anaesthesia has been used for almost every operation in Surgery - some under total body anaesthesia. The dangers and difficulties and complications are explained. A personal experience of 1,921 cases of spinal anaesthesia is presented. Hyperbaric solutions were used in all. Everyone considered fit for a general anaesthetic was submitted to spinal anaesthesia. The technique and care of the patient are described. The results are recorded, with details of failures, complications and deaths.

CONCLUSION

A review of a large number of papers on spinal anaesthesia, written mostly in English, shows the enormous interest that has been taken in this subject since the time of Bier. Most of these show that it is a useful and valuable method, giving excellent muscular relaxation and results comparable with those of general anaesthesia, at least where the services of a specialist anaesthetist are not available. A few writers have been opposed to it, and some alarming experiences have been recorded. The method is not without danger, especially in unskilled hands and where these risks are not appreciated.

Perhaps the chief drawback to spinal anaesthesia is the fall in blood-pressure that is such a common accompanying phenomenon. The important question to decide is whether this is a cause for alarm. A long personal experience persuades me that this low blood-pressure does not imperil the life of the patient, so long as measures are taken to prevent and combat its effects. These measures that are effective are putting the patient in the Trendelenburg position, injection of analeptic drugs and the administration of 100% oxygen under pressure. The Trendelenburg position is part of the technique of light spinal anaesthesia and can safely be adopted some ten minutes after injection of a hyperbaric solution. Drugs like ephedrine will certainly help to reduce the fall in blood-pressure and correct it. Their effect tends to pass off and injection may have to be repeated once or even twice. In emergency, they can be used intravenously. It appears that whatever the cause of this fall in blood-pressure, its most important effect is to cause tissue anoxia, and this effect is best counteracted by oxygen in high concentration.

Spinal anaesthesia has its place in the practice of the specialist anaesthetist. But its greatest appeal and value will be to the surgeon who does not enjoy this skilled assistance. The introduction of curare to produce muscular relaxation will tend to replace spinal anaesthesia where this effect is the chief indication for its use. At the moment curare has not been shown to be without

danger. Its administration calls for the service of an anaesthetist, and so it cannot oust spinal anaesthesia completely.

Controversy has always existed in Medicine and Surgery. It has certainly raged about anaesthesia since the days of chloroform and ether. As a medical student, I was brought up in a chloroform school. This was in the years 1918 to 1923. As late as 1936 I had great difficulty in persuading a senior surgeon in my own teaching hospital to allow me to give an ether anaesthesia. He was a believer in chloroform. To him chloroform was the only safe anaesthetic agent. I spent much time in his anaesthetic room and was appalled at the number of patients to whom artificial respiration was given before transfer to the theatre. The house surgeon, who was the anaesthetist, had very much more anxiety in one month of chloroform than I have had in twenty years with spinal anaesthesia.

The important thing about spinal anaesthesia is not prejudice in favour of a certain method. What matters is thorough understanding of the technique it is proposed to adopt. Every detail matters; none is trivial. Few indeed of the accidents and serious complications and sequelae are unavoidable.

Failure is due to improper technique. Meningitis is avoidable by careful asepsis.

Where the services of a specialist anaesthetist are not available, patients cannot receive the most suitable anaesthetic. They must be submitted to the best form of anaesthesia locally obtainable, and the surgeon must share part at least of the anaesthetic responsibility. The adoption of spinal anaesthesia relieves the surgeon of the anxieties that accompany general anaesthesia in unskilled hands.

Some at least of the deaths following spinal anaesthesia may be due to idiosyncracy. A test for sensitivity would be invaluable.

Apart from the obvious one of local sepsis at the site of puncture, the chief contraindication to spinal anaesthesia appears to be shock resulting from haemorrhage, especially when aggravated by trauma.

Although total body anaesthesia can be obtained, it is safer not to go higher than the second dorsal segment.

In a tropical climate of high humidity where coughs and colds and chronic respiratory disease (apart from tuberculous) are uncommon and rheumatic fever is unusual, respiratory and cardiovascular complications are minimal.

APPENDIX "A"

List of Operations under Spinal Anaesthesia

A. Abdominal	Number	Deaths
Cholecystectomy	7	-
Cholelithotomy	3	-
Choledocholithotomy	1	1
Gastrectomy (Cancer)	3	1
Gastrectomy (Ulcer)	6	-
Gastrostomy	5	-
Gastro-jujenostomy	19	-
Cholecyst-gastrostomy	1	-
Perforated Gastric Ulcer ..	9	1
Perforated Duodenal Ulcer ..	6	-
Splenectomy	4	--
Laparotomy	6	-
Laparotomy (G.S.W.)	5	2
Laparotomy (Stab Wound) ..	12	2
Laparotomy (Ruptured intestine) ..	4	-
Colectomy (right Hemi.) ..	-	-
~~Colectomy~~ (Cancer)	2	-
~~Colectomy~~ (Tuberculosis) ..	6	-
~~Colectomy~~ (Chrohn's Disease) ..	2	-
~~Colectomy~~ (persistent fistula) ..	1	-
Colectomy, spenic flexure ..	1	-
Colectomy, sigmoid	1	-
Colectomy, abdomino-perineal ..	1	1
Closure of Colostomy	1	-
Colostomy	5	-
Exteriorisation of Colon ..	2	1
Megacolon (Therapy)	7	-
Acute Intussusception (reduction)	3	-
Appendicitis (acute)	211	5
Appendicitis (interval) ..	59	-
Appendicitis (abscess)	10	-
Lumbar Sympathectomy	2	-
Persacral neurectomy	2	-

B. Genito-urinary	Number	Deaths
Operations on the kidneys, ureters and bladder:		
Nephrectomy (Cancer) ...	1	-
Nephrectomy (calculous pyonephrosis)	3	1
Nephrectomy (rupture) ...	3	-
Nephrolithotomy ...	4	-
Pyelolithothoy ...	17	-
Renal Sinus ...	4	-
Uretero-lithotomy ...	6	-
Transplantation of Ureters ...	6 (3 cases)	-
Suprepubic lithotomy ...	27	-
Rupture of Bladder ...	1	-
Bladder Fistula ...	1	-
Cystoscopy ...	97	-
Prostatectomy ...	11	-
Operations on the penis, scrotum, testes:		
Amputation (partial) ...	2	-
Amputation (complete, with glands)	7	-
Lacerated penis ...	2	-
Lacerated Scrotum ...	1	-
Urethrolithotomy ...	5	-
Ruptured Urethra ...	7	-
Urethral Fistula ...	17	-
Stricture ...	26	-
F.B. in Urethra ...	1	-
Phimosis (all adults) ...	6	-
Paraphimosis (adult) ...	1	-
Varicocoele ...	7	-
Haematocoele ...	4	-
Hydrocoele ...	72	-
Hydrocoele (of cord) ...	1	-
Orchidectomy (Tuberculosis) ...	2	-
Orchidectomy (Tumours) ...	4	-

C. Gynaecological		
Pelvic floor repair ...	29	-
Pelvic floor repair (with abdominal section)	7	-

	Number	Deaths
Perineal Tumours (Cancer)	2	-
Perineal Tumours (Simple)	2	-
Dilatation and Curretage (diagnostic)	7	-
Dilatation and Curretage (retained products)	62	-
Dilatation and Curretage (Hydatidiform mole)	2	-
Oophorectomy	59	-
Salpingo-oophorectomy	7	-
Salpingectomy	14	-
Urethral Caruncle	2	-
Hysterectomy, subtotal	64	-
total	8	-
pan-	14	1
Wertheim's	11	1
Myomectomy	3	-
Broad ligament tumours	3	-
cysts	1	-
Ruptured ectopic gestation	26	2
Vesico-vaginal fistula	6	-
Amputation of Cervix	6	1
Excision Bartholin's Cyst	1	-
Excision Cyst of vaginal wall ...	1	-
Caesarean Section	13	1

D. Legs and Buttocks

Amputations, Syme's	3	-
leg	1	-
supracondylar	1	-
through thigh		
Fracture of patella, excision ...	3	-
suture ...	2	-
Removal of f.b. (bullets, etc.) ...	13	-

	Number	Deaths
Repair of lacerations ...	9	-
Cut tendo achilles (repair)	3	-
Excision of Tumours ...	12	-
Varicose Veins ...	4	-
Internal derangements knee-joint	6	-
Compound Fractures ...	37	-
Simple Fractures ...	48	-
Dislocation, Hip ...	2	-
Semimenbranous bursa ...	3	-
Flexion contrature knee ...	4	-
Osteotomies ...	16	1
Smith-petersen pin ...	4	-
Sequestrectomies ...	27	-

E. Operations on the rectum and anus, etc.:

Haemorrhoids ...	113	-
Fissure in ano ...	14	-
Fistula in ano ...	57	-
Removal of foreign body ...	1	-
Prolapse ...	3	-
Pilonidal sinus ...	1	-

F. Operations for hernia:

Inguinal ...	321	2
Inguinal (bilateral) ...	8	-
Inguinal (strangulated) ...	75	3
Inguinal (recurring) ...	3	-
Femoral ...	11	-
Femoral (strangulated) ...	4	-
Umbilical ...	5	-
Umbilical (obstructed) ...	2	-
Gluteal ...	1	-
Incisional ...	1	-

APPENDIX A (Contd.)

AGE-GROUPS:

Age	Number	Age	Number	Age	Number
Under 1 year:		10-14 years ..	49	70 years	7
23 days	1	15-19 years ..	119	71 years	6
3 months	2	20-29 years ..	490	72 years	3
5 months	1	30-39 years ..	551	73 years	2
8 months	1	40-49 years ..	388	74 years	2
11 months	1	50-59 years ..	168	75 years	1
1-2 years	2	60-64 years ..	61	76 years	5
3-4 years	9	65-69 years ..	17	80 years	1
5-9 years	33			85 years	1

ANALYSIS OF DEATHS:

Age at death	Operation	Survived	Cause of death
62	choledocholithotomy ...	3½ hours	hepatic failure.
74	subtotal gastrectomy ...	3 days	heart failure.
36	closure perforated ulcer - gastric ..	2 days	peritonitis.
25	G.S.W. abdomen ...	13 days	peritonitis.
61	G.S.W. abdomen ...	17 hours	multiple wounds.
65	stab wound abdomen ...	2 days	B. welchii peritonitis
23	stab wound abdomen ...	14 hours	multiple injuries.
53	abdomino-perineal excision of rectum ...	33 hours	shock, operative.
7	acute appendicitis ...	24 hours	peritonitis
29	acute appendicitis ...	17 days	pulmonary embolism.
33	acute appendicitis ...	3 days	peritonitis.
46	acute appendicitis ...	2 days	peritonitis.
61	acute appendicitis ...	2 days	peritonitis.
59	exteriorisation of colon .	9 days	heart failure.
43	nephrectomy (calculous pyonephrosis) ...	4 days	broncho pneumonia.
37	panhysterectomy ...	3 days	massive atelectasis.
39	Wertheim's operation ...	24 hours	shock, operative.
23	Ruptured ectopic gestation	16 hours	shock due to blood-loss, operation and possibly anaesthetic.
27	Ruptured ectopic gestation	8 hours	-- do --
19	Caesarean section ...	8 hours	-- do --
62	osteotomy (fracture neck of femur) ...	23 days	? pulmonary embolism.
41	hernia ...	20 days	? pulmonary embolism.
63	hernia ...	10 days	heart failure.
43	strangulated hernia (resection) ...	19 hours	peritonitis.
26	strangulated hernia (resection) ...	2 days	peritonitis.
31	strangulated hernia (resection) ...	3 days	peritonitis.

APPENDIX "B"

REFERENCES

- A -

Adelman, M.H. & Irwin, C.I. (1946) - Anaesthesiology 7: 422-425.

Adriani, J. (1946) - Chemistry of Anaesthesia: Oxford: Blackwell Scientific Publications.

Adriani, J. & Rovenstine, E.A. (1940) - Proc. Soc. Exp. Biol. Med. 45: 415-417.

Agar, H. (1943) - British Medical Journal 2: 101-103.

Aikenhead, D.C. (1945) - Can. Med. Ass. J. 52: 162-165.

Allen, J.G. & Livingstone, H. (1940) - Anaesthesiology 1: 89-93.

Altschule, M.D. & Gilman, S. (1939) - New Eng. J. Med. 221: 600-601.

Anderson, B.M. (1946) - Anaesthesiology 7: 1-9.

Andre, R.H. (1942) - An. & Analg. 21: 54-59.

Apgar, V. (1939) - An. & Analg. 18: 241-245.

Apgar, V. (1942) - Anaesthesiology 3: 522-529.

Armstrong, D. (1946) - Brit. Medical J. 2: 264-265.

Ashworth, H.K. (1933) - Brit. J. An. 10: 127-132.

- B -

Babcock, M.E. (1932) - An. & Analg. 11: 184-188.

Babcock, W.W. (1906) - Therap. Gaz. 30: 239-244

Babcock, W.W. (1928) - Amer. J. Surg. 5: 571-576.

Bacon, H.E. (1941) - An. & Analg. 20: 333-337.

Baird, J.W. (1943) - Anaesthesiology 4: 17-24.

Baker, A.H. (1947) - Brit. J. Surg. 34: 385-388.

Barrie, H.J. (1941) - Lancet 1: 242-243.

Batten, D.H. (1941) - An. & Analg. 20: 115.

Batten, D.H. (1943) - Ibid 143-150.

Beecher, H.K. (1938) - "The Physiology of Anaesthesia" New York & Oxford: P. 271 (Quoted by Sellman, P.).

Belinkoff, S. (1946) - Anaesthesiology 7: 258-275.

Bellis, C.J. (1944) - Surgery 16: 896-905.

Bier, A. (1899) - Deutsche Ztschr. F. Chir. 51: 361-369.

Billington, R.W. (1924) - J. Amer. Med. Ass. 83: 683-686.

Bittrich, N.M. (1939) - An. & Analg. 18: 29-36.

Bittrich, N.M. (1945) - The Urological & Cutaneous Review 49: 133-138.

Black, M.G. (1947) - Anaesthesiology 8: 382-388.

Booker, W.H. & Johnson, V.B. (1944) - An. & Analg. 23: 255-257.

Botterell, E.H. & Magner, D. (1945) - Lancet 1: 112.

Bourne, W. (1942) - Brit. J. An. 18: 2.

Bourne, W., Leigh, H.D., Inglis, A.N. & Howell, G.R. (1942) - Anaesthesiology 3: 272-281.

Bowman, F.B. (1946) - Can. Med. Ass. J. 54: 148-150.

Boyd, E.A. (1936) - Brit. J. An. 13: 56-65.

Brennan, H. (1934) - Brit. J. An. (July) 147-153.

Brown, G. (1946) - An. & Analg. 25: 118-131.

Burch & Harrison (1930) - Arch. Surg. 21: 330.

Burford, G.E. (1942) - Anaesthesiology 3: 159-170.

- C -

Chaikoff, J.S. (1940) - An. & Analg. 19: 121-131.

Chaikoff, J.S. (1947) - Ibid 26: 156-163 & 203-211.

Campbell, L.E., Crane, R.M. & Sankey, B.B. (1947) - An. & Analg. 26: 255-259.

de Caux, F.P. (1932) - An. & Analg. 11: 68-70.

Cave, J.N. (1938) - Brit. J. An. 15: 158-165.

Charles, R.L. (1936) - An. & Analg. 15: 206-208.

Cole, P.P. (1941) - Brit. J. Surg. 29: 168-181.

Cooper, M.P. (1939) - An. & Analg. 18: 291-294.

Corning, J.L. (1885) - New York Med. J. 42: 483-485.

Corning, J.L. (1888) - New York Med. Rec. 33: 291-293.

Cosgrove, S.A., Hall, P.O., & Gleeson, W.J. (1937) - An. & Analg. 16: 234-237.

Cotui, F.W. & Standard, S. (1932) - Surg. Gyn. Obs. 55: 290-297.

Cotui, F.W. (1934) - An. & Analg. 13: 143-151 & 183-192.

Courville, C.B. (1936) - Medicine 15: 129 (quoted by Woolmer 1948)

Cubitt, A.W. (1936) - Brit. J. Surg. 24: 215-225.

Cullen, W.G. & Griffiths, H.R. ( ) - An. & Analg. 26: 114-121.

- D -

Daley, R. (1943) - Brit. Med. J. 2: 179.

Davidson, I.M. (1947) - Lancet 2: 653-654.

Davies, L., Haven, H., Givens, J.H. & Emmett, J. (1931) - J. Amer. Med. Ass. 97: 24.

De-Lee & Greenhill (1943) - Principles & Practice of Obstretics 8th. Ed.

Dillon, J.B. (1947) - J. Amer. Med. Ass. 135: 977-981.

Dinsdale, T. (1947) - Anaesthesia 2: 17-27.

Dodd, H. & Prescott, F. (1943) - Surg. Gyn. Obs. 77: 645-656.

Doud, E.A. & Rovenstine, E.A. (1940) - Anaesthesiology 1: 82-86.

- E -

Ecker, A.D. (1945) - Lancet 1: 176.

Edwards, A.T. (1938) - Brit. J. Surg. 26: 166-192.

Eibert, H. (1922) - Mnschr. Psychiat. Neurol. 52: 347.

Elam, J. (1944) - Brit. J. Anaesthesia 19: 32-46.

Essex, H.F., Wegria, R.G.E., Herrick, J.F. & Mann, F.C. (1939) - Proc. Amer. Physiol. Soc.

Etherington-Wilson, W. (1933-1934) - Proc. Ry. Soc. Med. 27: 323-331.

Etherington-Wilson, W. (1943) - Brit. J. An. 2: 43-55.

Etherington-Wilson, W. (1944-1945) - Proc. Ry. Soc. Med. 38: 109-115.

Evans, C.H. (1929) - Spinal Anaesthesia, (1929) - London, William Heinman (Medical Books) Ltd.

Evans, C.H. (1929) - Spinal Anaesthesia, New York & Hoeber 103-110.

Evans, F.T. (1945) - Lancet 1: 115.

Everett, A.D. (1942) - Proc. Roy. Soc. Med. 35: 208-210.

Eversole, U.H. (1945) - J. Amer. Med. Ass. 128: 256-261.

- F -

Fairlie, H.P. (1932) - Brit. J. An. 9: 162-168.

Fairclough, W.A. (1945) - Brit. Med. J. 2: 801-803.

Ferguson, F.R. & Watkins, K.H. (1938) - Brit. J. Surg. 25: 735-752.

Ferguson, L.K. & North, J.P. (1932) - Surg. Gyn. Obs. 54: 631-634.

Fisher, S.J. & Whitacre, R.J. (1947) - Anaesthesiology 8: 584-588.

Foster, A.D., Neumann, C. & Rovenstine, E.A. (1945) - Anaesthesiology 6: 244-257.

Franken, H. (1935) - Brit. J. An. 12: 81-88 & 124-131.

Fraser, H. (1940) - Can. Med. Ass. J. 43: 251-255.

- G -

Gardner, H.B. (1930) - Brit. J. An. 7: 71-79.

Garth, W.L. (1936) - An. & Analg. (March & April) 72-76.

Garth, W.L. (1944) - An. & Analg. 23: 125-128.

Gellman, M. (1940) - J. Brit. & Jt. Surg. 22: 980-985.

Gillespie, N.A. (1944) - Brit. J. An. 19: 1-16.

Girth, W.L. (1936) - An. & Analg. 73: 72-75.

Gorham, A.P. (1933) - Brit. J. An. 11: 12-15.

Gowar, F.J.S. (1941) - Brit. J. Surg. 29: 3-18.

Grant-White, H. (1937) - Brit. J. An. 14: 55-79.

Gregory, R. & Levin, W.C. (1945) - J. Lab. & Clin. Med. 30: 1037-1043.

Gregory, R., Levin, W.C., Ross, G.Y. & Bennett, A. (1946) - Arch. Int. Med. 77: 385-392.

Griffiths, H.F. (1934) - Brit. J. An. 11: 89-109.

- H -

Hand, L.V. (1940) - Surg. Cl. N. Amer. 20: 647-652.

Harbord, R.P. (1943) - Brit. J. Surg. 30: 236-237.

Harris, T.A.B. (1939) - Brit. J. An. 16: 4 (July).

Harris, T.A.B. (1942) - Brit. J. An. 18: 11-26.

Harrison, P. (1940) - Surgery 7: 910-917.

Haugen, F.P., Ruth, H.S. & Taylor, I.B. (1942) - Anaesthesiology 3: 52-60.

Haugen, F.P. (1943) - An. & Analg. 22: 152-157.

Hawksley, M. (1944) - Brit. J. Surg. 31: 242-245.

Haynes, W.G. & Smith, F.A. (1942) - Anaesthesiology 3: 444-447.

Heard, K. (1938) - An. & Analg. 17:

Heard, K. ( ) - Surg. Gyn. Obs. 70: 657-661.

Heard, K. (1946) - An. & Analg. 25 (Sept.-Oct.): 191-199.

Hershey, S.G. (1945) - New York State J. Med. 45: 1761-1764.

Hewer, C.L. (1941) - Brit. J. Surg. 28: 615-627.

Hewer, C.L. (1944) - "Recent Advances in Anaesthesia & Analgesia" (J. & H. Churchill Ltd.) Chap. XIV.

Hill, E. Falkner, (1931) - Brit. J. An. 8: 132-150.

Hill, E. Falkner (1933) - Brit. J. An. 11: 3-11.

Hill, E. Falkner (1934) - Ibid 11 (July): 142-146.

Hill, E. Falkner (1935) - Ibid 12: 2 (Jan) 70-77.

† Hirschfelder, A.D. & Bieter, R.N. (1932) - Physiol Rev. 12: 190-282.

Holles, K.E. (1942) - Can. Med. Ass. J. 46: 351-354.

Holmes, F. (1948) - Anaesthesia 3: 67-72.

Hopkins, (1902) - J. Amer. Med. Ass. 38: 355.

Hughes, C. (1927-1928) - Proc. Roy. Soc. Med. 21: 189-196.

Hurxthall, L. (1932) - J. Amer. Med. Ass. 100: 1489.

Hutton, J.H. (1943) - An. & Analg. 22.

Hyman, I.R. & Wood, P.M. (1942) - An. & Surg. 115: 864-868.

- I -

Iason, A.H., Lederer, M. & Steiner, M. (1930) - Surg. Gyn. Obs. 51: 76-80.

Iglarer & Altschule (1940) - J. Clin. Invest. 19: 503.

- J -

Jackson, D.E. (1947) - Cur. Res. in An. & Analg. 26: 1-11 & 64-73.

Johnson, B.R.M. (1931) - Brit. J. An. 8: 104-111.

Jones, W.H. (1930) - Brit. J. Surg. 7: 99-113.

Jones, W.H. - Ibid 7: 146-156.

Jones, W.H. (1931) - Brit. J. An. 9: 3-12.

Jones, H. & Trempé, F. (1937) - An. & Analg. 16: 26-30.

- K -

Kaye, G. (1936) - Brit. J. An. 13: 110-127.

Kelly, F.A. (1939) - Brit. J. An. 7: 17-22.

Kennedy, F.G., Somberg, H.M. & Goldberg, B.R. (1945) - J. Amer. Med. Ass. 129: 664-667.

Kershner, D. & Spapiro, A.L. (1946) - Amer. J. Surg. 72: 43-46.

Keys, T.E. (1945) - An. & Analg. 24: 104-108.

Keys, T.E. (1945) - "The History of Surgical Anaesthesia" New York, Schuman's.

Kirtlin & Sudarf (1939) - Proc. Mayo. Clin. 14: 502.

Knight, R.T. (1945) - Anaesthesiology 6: 225-230.

Koster, H. & Kasman (1949) - Surg. Gyn. Obs. 49: 617-630.

Koster, H., Shapiro, A., & Leikensohn, A. (1938) - Arc. Surg. 37: 603.

Kremer, M. (1945) - Brit. Med. J. 2: 309-313.

- L -

Labat, G. (1930) - Surg. Clin. N. Amer. 12: 643.

Lahey, F.H. (1940) - Surg. Clin. N. Amer. 20: 611-614.

Latham, R.M. (1942) - An. & Analg. 21: 34-40.

Lee, J.A. (1944-1945) - Proc. Roy. Soc. Med. 38: 115-118.

Lee, J.A. & Essex (1944) - An. & Analg. 23: 171-173.

Leech, B.C. (1947) - Can. Med. Ass. J. 56: 28-34.

Lemmon, W.T. (1939)

Lemmon, W.T. (1940) - Ann. Surg. 111: 140-144.

Lemmon, W.T. & Paschal Jr., G.W. (1941) - Penn. Med. Jr. 44: 975-981.

Lemmon, W.T. & Heger, H.G. (1944) - Ann. Surg. 120: 129-142.

Lemmon, W.T. (1946) - Can. Med. Ass. J. 54: 363-368.

Lewis, I. (1936) - Brit. J. Surg. 24: 326-367.

Lewis, N.H. (1944) - An. & Analg. 23: 29-33.

Light, G., Sweet, W.H., Livingstone, H. & Engel, R. (1940) - Surg. 7: 138-156.

Lindy, J.S., Essex, H.E. & Kernohan, J.W. (1933) - J. Amer. Med. Ass. 101: 1564-1567.

Livingstone, H., Wellman, V., Clarke, D. & Lambross, V. (1943) - Surg. Gyn. Obs. 77: 217.

Lorhan, P.H. & Mosser, D. (1947) - Ann. Surg. 125: 171-176.

Lotz, A.K. (1937) - An. & Analg. 16: 70-74.

Lovell, R.M. & Dunn, H.L. (1932) - An. & Analg. 11: 37-41.

Lund, P.C. & Rumball, A.C. (1947) - Anaesthesiology 8: 181-199

Lyford, 3rd. J., Berger, O.L. & Shumacker Jr. H.B. (1942) - Bulletin of J. Hopkins' Hosp. 70: 488-503.

- M -

Madan (1944) - Brit. J. A. An. 19: 87-94.

Macpherson, G. (1939) - Brit. J. Surg. 27: 34-37.

Magill, I.W. (1936) - Brit. J. An. 13: 92-109.

Mallinson, F.B. (1938) - Brit. J. An. 16: 22-27.

Margottini, M. (1937) - Polyclinico 44: 1385.

Martin, R.C., Livingstone, H. & Wellman, V. (1945) - Arc. Surg. 50: 130-136.

Martin, S. & Schwab, J.M. (1945) - Anaesthesiology 6: 145.

Maxson (1938) - Spinal Anaesthesia, J.B. Lippincott Co: Phildalphia.

Mc Cann, J.C. (1947) - An. & Analg. 26: 89-104.

Mc Cann, J.C. (1947) - Ibid 26: 140-154.

Mc Carthy, K.C. (1947) - An. & Analg. 26: 30-33.

McDonald, A.D. & Watkins, K.H. (1938) - Brit. J. Surg. 25: 879-883.

Mc Elligott, D.C. & Watkins (1936) - An. & Anlg. 15: 288-291.

Melville & Stehle (1931) - J. Ph. & Exper. Ther. 42: 455.

Melville, K.I. (1946) - Anaesthesiology 7: 176-189.

Meredith, R.H. (1941) - Can: Ass. J. 44: 394-396.

Milowsky, J.L. & Betancourt, R.R. (1945) - Anaesthesiology 5: 600-602.

Milward, F.J. & Grout, J.L.A. (1936) - Lancet 2: 183-184.

Morgan, A.D. (1943) - An. & Anlg. 22: 81-92.

Morrison, D.R. (1948) - Surg. 23: 561-570.

- N -

Nathanson, M.H., Engleberg, H. & Hersh, J. (1942) - Amer. Heart J. 34: 153-166.

Nicholson, M.J. (1940) - Surg. Cl. N. Amer. 20: 639-646.

Nicholson, M.J. & Eversole, U.H. (1946) - J. Amr. Ass. 132: 679-685.

Nitikman, G.

Noble, A.B. (1946) - Can. Med. Ass. J. 54: 378-379.

- O -

O'Shaughnessy, L. & Slome, D. (1935) - Brit. J. Surg. 22: 589-618.

- P -

Peace, C.N. (1935) - Amer. J. Dis. Childr. 49: 849-860.

Peterson, M.C., Robertazzi, R. & Goebel, J. (1943) - Anaesthesiology 4: 625-629.

Pisetsky, J.E. (1945) - Anaesthesiology 6: 522-527.

Pitkin, G.P. (1928) - Amer. J. Surg. 5: 537-553.

Pitkin, G.P. (1929) - Brit. J. An. 7: 2-16.

Pitkin, G.P. (1940) - An. & Analg. 19: 241-260 & 315-329.

Pitkin, G.P. (1942) - Ibid 21: 1-12 & 83-95.

Potter, J.K. & Whitacre, R.J. (1946) - Anaesthesiology 7: 499-504.

Prescott (1944) - Brit. Heart J. 6: 214.

Prickett, M.D., Gross, E.C. & Cullen, S.C. (1945) - Anaesthesiology 5: 469-475.

- Q -

+ Quincke (1891) - Berlin klin. Wochenschrift 28 (929-933).

- R -

Ramson, D.B. (1946) - J. Lab. Clin. Med. 17: 115.

Rapoport, B. (1941) - An. & Analg. 20: 19-24.

Rea, C.E. (1944) - Surgery 16: 731-738.

Reynolds, K.E. & Wilson, G. (1934) - J. Amer. Med. Ass. 102: 1460-1462.

Reid, L.C. (1941) - Anaesthesiology 2: 161-169.

Richards, R.K. & Keuter, K. (1947) - An. & Analg. 26: 22-29.

Rochford, J.W. (1944) - J. R.A.M.C. 83: 250-251.

Rochberg, S. & Apgar, V. (1942) - Anaesthesiology 3: 48-51.

Roman, D.A., Vega & Adriani, J. (1946) - An. & Analg. 25: 79-83.

Roman, D.A., Vega & Adriani, J. (1946) - Anaesthesiology 3: 421-428.

Rombergger, F.T. (1943) - An. & Analg. 22: 252-263.

Rovenstine, E.A. & Apgar, V. (1944) - Anaesthesiology 5: 40-44.

Rovenstine, E.A., Papper, E.M. & Bradley, S.E. (1942) - Anaesthesiology 7: 62-68.

Rowbotham, S. (1947) - Anaesthesia 2: 3 (July).

Ruben, J.E. (1947) - Surgery 22: 826-833.

Ruben, J.E., Kamsler, P.M. & Howell Jr., W.L. (1948) - Science 107: 223.

Ruth, H.S., Haugen, F.P. & Grove, D.D. (1947) - J. Amer. Med. Ass. 135: 881-884.

Ruzicka, E.R. (1945) - Anaesthesiology 6: 385-401.

- S -

Sachs, L. (1941) - An. & Analg. 20: 200-206.

Salb, R.L. & Mueller, L. (1946) - An. & Analg. 25: 84-87.

Sappenfield, R.S. & Rovenstine, E.A. (1940) - An. & Analg. 19:

Sarnoff, S.J. & Farr, H.W. (1944) - Anaesthesiology 5: 69-76.

Sarnoff, S.J. & Arrowwood, J.G. (1946) - Surgery 20: 150-159.

Sarnoff, S.J. & Arrowwood, J.G. (1947) - J. Clin. Invest. 26: 203-216.

Sarnoff, S.J. & Arrowwood, J.G. & Chapman, W.P. (1948) - Surg. Gyn. Obs. 86: 571-581.

Schmidt, C.F. (1945) - Anaesthesiology 6: 113-123.

Schumacher, L.F. & Eversole, U.H. (1942) - Anaesthesiology 3: 630-643.

† Sebrechts, J. (1930) - Bull. Acad. Roy. de Med. de Belg. 10: 543-683.

Sebrechts, J. (1934) - Brit. J. An. 12: 4-27.

Sebrechts, J. (1935) - Ibid. 13: 176-182.

Seevers, M.H. & Waters, R.M. (1932) - An. & Analg. 11: 2-85.

Sellman, P. (1940) - Surg. Cl. N. Amer. 20: 621-629.

Sellman, P. (1941) - Anaesthesiology 2: 333-338.

Shields, H.J. (1933) - Can. Med. Ass. J. 29: 528-530.

Sise, L.F. (1932) - An. & Analg. 11: 23-28.

Sise, L.F. (1940) - Surg. Cl. N. Amer. 20: 631-638.

Slome, D. & O'Shaughnessy, L. (1938) - Brit. J. Surg. 35: 900-909.

Smith, F.A. (1946) - Anaesthesiology 7: 419-421.

Smith, W. & Smith, M.M. (1941) - Lancet 2: 783-785.

Smilair (1902) - Surg. Obs. Gyn. Brit. Emp. 22: 1902.

Snyder, W.H. & Snyder, M.H. (1939) - An. & Analg. 18: 221-226.

Speller, W.C. & Payne, F.L. (1924) - J. Amer. Med. Ass. 82: 106.

Stanley, G.D. (1942) - Can. Med. Ass. J. 46: 164-167.

Stanley, G.D. (1943) - An. & Analg. 22: 290-296.

Stanley, L.L. (1940) - An. & Analg. 19: 112-114.

Starr, Gamble, Mangolies, Donal, Joseph & Engel (1937) - J. Clin. Invest. 16: 799.

Steinberg, B. & Bishop, H.F. (1946) - Anaesthesiology 7: 296-298.

Symonds, C.P. (1925) - Lancet 1: 434-435.

- T -

Tainter, M.L. (1946) - Anaesthesiology 7: 132-145.

Taylor, T.B., Bennett, J.H. & Waters, R.M. (1937) - An. & Analg. 16: 187-192.

Teffier, M. (1899) - Comptes Rendus Hebdomadaivus des Seances et memoirs de la Societe de Biologia 51: 882-884.

Telford, E.D. (1939) - Roy. Soc. Med. 32: 1145-1148.

Thomas, S.J. & Sica, P.B. (1946) - Penns. Med. J. 50: 137-143.

Thompson, K.W. (1934) - Surg. Gyn. Obs. 58: 852-866.

Torrie, A.M. (1945) - Anaesthesiology 6: 154-158.

Tovell, R.M. & Dunn, H.L. (1932) - An. & Analg. 11: 37-41.

Trempe, F. (1937) - An. & Analg. 16: 26-30.

Tuffier (1899) - Compt. Rend. de la Soc. de Biol. 51: 882-884.

Tuohy, E.B. (1945) - S. C. N. A. 834-840.

Tuohy, E.B. (1947) - J. Amer. Ass. 135: 751-753.

Turner, F.P. & Wilkinson, F.A.H. (1945) - Abst. in An. & Analg. 24: 12-24.

- V -

Vander Post, C.W.H. (1938) - Brit. J. Anaes. 15: 117-124.

Vehrs, G.R. (1936) - An. & Analg. 15: 33-43.

Vuylesteke, C.A. (1947) - B.M.J. 1: 179-180.

- W -

Walker, J.B. (1936) - Brit. J. Surg. 24: 105-121.

Wallace, G.B. & Brodie, B.B. (1936) - J. Pharm. & Exper. Ther. 65: 220-226.

Wardle, E.N. (1945) - Lancet 1: 369-402.

Waters, R.M. & Gillespie, N.A. (1944) - Anaesthesiology 5: 113-128.

Watson, T.P. (1943) - Can. Med. Ass. J. 49: 121.

Watter, L. (1941) - An. & Analg. 20: 132-144.

Weed, L.H. (1914) - J. Med. Res. 31: 51-59.

Weed, L.H., Wedgeworth, P., Ayer, J.P. & Felton, L.D. (1920) - Monograph 12. Rockfeller Inst. Med. Res.

Weed, L.H. (1938) - J. Anat. 72: 151-215.

Whitacre, F.M. (1944) - An. & Analg. 23:

Whitacre, R.J. & Sankey, B.B. (1943) - An. & Analg. 22: 99-104.

Whitacre, R.J. & Potter, J.K. (1948) - An. Surg. 127: 338-341.

Wilkinson, F.A.H. (1942) - Anaesthesiology 3: 437-443.

† Woodbridge, P.D. (1940) - Surg. Clin. N. Amer. 20: 615-620.

Woolmer, R. (1948) - Anaesthesia 3: 73-76.

Woltman, H.N. (1936) - Wisconsin Med. J. 35: 427-436.

† Wossildo, E. (1932) - J. Amer. Med. Ass. 99: 993.

- Y -

Yanick, J.J. (1939) - An. & Analg. 18: 174-180.

* * * * * * * * *

NOTE :- THE references marked † have been quoted by others and not personally consulted by me.