THESIS

ON

EPIDURAL ANALGESIA

SUBMITTED FOR THE DEGREE OF

DOCTOR OF MEDICINE

BY

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I. INTRODUCTION

HISTORY AND DEVELOPMENT OF EPIDURAL ANALGESIA

The object of epidural analgesia is to produce, with a single injection, a multiple blockade of the spinal nerves as they emerge from the dura mater. Although sacral epidural analgesia was first performed in 1901 by Sicard and Cathelin, and in 1921 Pagés described his method of lumbar epidural analgesia, it is only within the last few years that the technique has been performed on any scale in this country.

Following the introduction of ether in 1842 by Morton, and of chloroform in 1847 by Simpson, the concept of anaesthesia became quickly established. Although this freed the surgeon from anxieties regarding the patient's awareness of operative procedures, other serious limiting factors, particularly infection, still remained to impede surgical progress. Another 40 years passed before Lister made the next major advance by introducing his antiseptic method. It is not surprising, therefore, that anaesthesia itself made no marked forward steps until after this time, when the anaesthetist was confronted with many new problems. Operations were taking very much longer, and surgeons were anxious to explore new fields such as the thorax and the cranium, each of which presented its own difficulties.

With the discovery of the local anaesthetic properties of cocaine, it became possible to avoid general anaesthesia altogether, and this principle gained its greatest impetus when /

when Bier introduced spinal anaesthesia in 1898. Complete relaxation in the operative field without general depression of the patient was of considerable advantage over the general anaesthetics in common use at that time.

In France in 1901, Sicard[1], who was dissatisfied with the headache and sickness that follow spinal anaesthesia, described his technique of sacral epidural analgesia, the local anaesthetic being introduced through a needle inserted into the sacral spinal canal through the sacral hiatus. Cathelin[2], working independently, also produced analgesia in this way. Although Tuffier[3] attempted to locate the epidural space by the lumbar route in 1901 (he performed a lumbar puncture then withdrew the needle slowly until the cerebro-spinal fluid ceased to flow), he did not meet with any great success and it appeared that an attempt to enter a space only two or three millimetres wide, so deeply situated in the back, was almost bound to fail. This left the sacral route almost unopposed for 20 years, although Heile[4] in 1913 had achieved lumbar epidural block by entering the space laterally through an intervertebral foramen. As a result, the method was only used for blocking the cauda equina, attempts to reach a higher level proving unreliable.

It was not until 1921 that Pagés[5] in Spain showed that the lumbar epidural space could be located with success in a high proportion of patients. He described 43 cases, 40 of which had satisfactory analgesia (although he had one case which /

which was probably an inadvertent total spinal blockade). He used a tactile method of identifying the space and this required considerable experience and skill. The needle was directed slightly laterally so that the epidural space was entered at the side of the dura mater. He was guided mainly by the sound and feel of the needle as it penetrated the ligamentum flavum, and confirmed the position by the ease with which fluid could be injected. A further development of this confirmatory sign was to use it as the so-called "loss of resistance" test. In this, pressure was maintained on the plunger of a loaded syringe attached to a needle being advanced through the spinal ligaments, until entry into the epidural space was indicated by a sudden loss of resistance to the forward movement of the plunger. Forestier[6] described this in 1922, and it was popularised by Dogliotti.[7] This method is the most commonly used at the present time.

Use of the normally occurring negative pressure within the epidural space was made by Soresi[8] and Gutierrez[9] who devised the "hanging-drop sign". In this a drop of liquid previously applied to the hub of the exploring needle is seen to be sucked into the needle as the space is entered. This technique is now usually reserved for epidural puncture in the thoracic or cervical regions, because lower down the spinal canal the negative pressure is not present in about 20 per cent. of cases.

Unfortunately, /

Unfortunately, although identification of the epidural space was no longer a problem, the lack of a satisfactory local anaesthetic put severe limitations on the method. Procaine, though safe, was not reliable for nerve blocks and its effect was too short-lived. Cinchocaine and amethocaine produced a good effect, but took up 20 to 30 minutes to act. As a result of this, epidural blockade was mainly used by individual enthusiasts, and in this respect, special mention should be made of the work in this country of Massey Dawkins[10], and later, of Bromage[11].

In the U.S.A. the series of caudal blocks in obstetrics by Hingson[12] attracted considerable attention. American anaesthetists had used the drug metycaine for this purpose with good results, but this was seldom, if ever, employed in this country.

It seemed that, with the introduction of muscle relaxants into anaesthetic practice after the second World War, spinal and epidural analgesia would suffer a complete eclipse. This eclipse, however, has only been short-lived, for, with the advent of lignocaine in 1950, we had at last the almost perfect anaesthetic for epidural analgesia. It has become apparent that, although anaesthesia has been revolutionised by muscle relaxants, they have not by any means solved all the problems. Epidural analgesia is now undergoing a phase of increasing popularity as its very real advantages become apparent.

During the years when muscle relaxants were being introduced /

introduced, the use of spinal anaesthesia was undergoing a decline. This was due to two main reasons. Firstly, it was thought that muscle relaxants could produce equally ideal operating conditions (though this is now open to question). Secondly, the fear of producing neurological damage made many anaesthetists feel that the method was quite unjustified. It was held that it was impossible to reduce the incidence of neurological damage below a certain fixed minimum, though very few anaesthetists could produce large enough personal series that would indicate just what was this minimum. More recently, doubt has been thrown on the inevitability of permanent paralysis. Vandam and Dripps[13], in a series of 10,000 cases of spinal anaesthesia, did not see one case of permanent neurological damage. As we gain knowledge of the causes of damage to nervous tissue, these unfortunate cases become more and more rare. Over the years, improvements in technique have been followed by reduction in the incidence of complications. It was common for syringes and needles to be kept in spirit and sterilised by boiling. Now autoclaving is considered essential. The keeping of ampoules of local anaesthetics in antiseptics such as phenol has been shown to allow penetration of the antiseptic into the ampoules, and this has been the cause of paraplegia (Cope[14]). Local anaesthetics that can be autoclaved are now chosen. Strict aseptic technique by "scrubbing-up" and the donning of sterile rubber gloves must /

must be observed. The use of fine lumbar puncture needles, and better training in lumbar puncture, reduce the incidence of post-spinal headache and temporary cranial nerve palsies which may follow leakage of cerebro-spinal fluid through the dural puncture hole.

When lignocaine was introduced into anaesthetic practice, it was at last possible to produce epidural blocks that were rapid in onset and reliable. Although spinal anaesthesia still remained unpopular, it was gradually realised that in many ways epidural analgesia, though almost indistinguishable from spinal blockade, was superior to general anaesthesia with relaxants. Moreover, it possessed two major advantages over spinal blockade; the dura was not punctured so that headache was conspicuous by its absence, and local anaesthetic drugs were not introduced directly into the sub-arachnoid space. The recent evidence that, if properly carried out, spinal anaesthesia is free from permanent nervous sequelae, offsets much of the latter advantage of epidural injection, but the impression remains among anaesthetists that epidural is safer than spinal analgesia, as far as neurological sequelae are concerned.

PRESENT INVESTIGATIONS

The author's experience with epidural analgesia began in 1954. At first the caudal route was chosen until it became apparent that the failure rate was rather high, and toxic reactions to the injected local anaesthetic drug were not infrequent. /

infrequent. The lumbar route was then tried and, rather surprisingly, this proved somewhat easier. Failures were fewer, and because a slow unforceful injection could be made (in contradistinction to caudal block), toxic reactions were eliminated.

Though self-taught, a reliable technique was soon adopted and epidural analgesia became the method of choice in many types of operation. To date, more than 450 epidural blocks have been performed, including 60 caudal blocks.

There are many unsolved problems relating to this type of anaesthesia, and during the five years the writer has been practising it, an attempt has been made to probe into some of them. It will be appreciated that most of these investigations were carried out while supplying a clinical anaesthetic service, often single-handed, to busy surgical units, and this imposed considerable limitations on experimental methods. All the epidural blocks were performed on patients about to undergo surgery, and their interests had to take precedence over experimental investigation. Moreover, surgeons could not be kept waiting indefinitely while observations were carried out.

In spite of these limitations, however, the following investigations were undertaken:-

(1) A small series of dissections (about 20 in number) of sacral canals was carried out in the dissecting room in order /

order to become familiar with the anatomy, and see if there were any anatomical reasons why caudal block was less reliable than lumbar epidural block.

(2) In a series of 100 cases (including 40 caudal blocks) hyaluronidase was added to the anaesthetic solution in an attempt to speed up the onset of analgesia. The results, which are discussed, had in addition a bearing on the site of action of epidurally injected local anaesthetic.

(3) Thirty cases were given the new local anaesthetic drug carbocaine, and a comparison with lignocaine was made.

(4) The incidence of hypotension during the course of epidural analgesia was noted, and views are expressed regarding the applicability of this method to controlled hypotension. A comparison is made with hypotension produced by spinal blockade and by ganglionic blocking agents.

(5) One of the major advantages of epidural analgesia is the reduction of operative blood loss. Unfortunately, almost all protagonists of various forms of anaesthesia claim this for their own particular method and almost always the claim is unsubstantiated by any evidence, other than clinical impression. In order to get more information on this point, blood loss was estimated by swab weighing on cases of pelvic floor repair (Fothergill's operation). By this method, the blood loss in a series of cases could be calculated reasonably accurately in a standard operative procedure performed under epidural anaesthesia, and this could /

could be compared with a series performed under general anaesthesia. The role of the various factors which may reduce bleeding, such as hypotension, could then be assessed.

(6) During the course of the preceding investigation on blood loss and in an effort to find the most satisfactory anaesthetic for pelvic floor repairs, local infiltration of the operative area with dilute (1/200,000) adrenaline was used. While proving satisfactory as a haemostatic agent, the adrenaline had very interesting side-effects. In the conscious subject, adrenaline given in sufficient dosage causes a rise in blood pressure by its stimulating action on the heart, tachycardia occurring at the same time. Its action on peripheral blood vessels is varied, some being constricted while others are dilated. The overall effect is one of vasodilatation (Robson and Keele[15]), so that the rise in blood pressure is solely due to increased cardiac output as a result of direct stimulation of the myocardium. Under anaesthetic conditions, however, the adrenaline would appear to act quite differently. Firstly, the patients seem more sensitive to its action, a marked rise of blood pressure occurring after a relatively small amount of adrenaline is injected. At the same time the pulse rate, instead of being raised, is unaffected. It would seem, then, that dilute adrenaline injected into anaesthetised patients causes marked vasoconstriction with little or no direct action on the myocardium.

Details /

Details of these investigations and their results are given in the course of this thesis which also includes the writer's experience of the methods of producing epidural analgesia, and the management of patients undergoing this form of anaesthesia.

References

(1) Sicard, A. (1901). C.R. Soc. Biol. (Paris), 53, 396.
(2) Cathelin, M.F. (1901). C.R. Soc. Biol. (Paris), 53, 452.
(3) Tuffier, T. (1901). C.R. Soc. Biol. (Paris), 53, 490.
(4) Heile, B. (1913). Arch. klin. Chir., 101, 845.
(5) Pagés, F. (1921). Rev. Sanid. milit. (Madr.), 11, 351.
(6) Forestier, J. (1922). "Le trou de conjugaison vertebral et l'espace epidural," p. 46. Paris: Jouve et Cie.
(7) Dogliotti, A.M. (1933). Amer. J. Surg., 20, 107.
(8) Soresi, A.L. (1932). Med. Rec. (N.Y.), 135, 165.
(9) Gutierrez, A. (1932). Rev. Cir. (B. Aires), 12, 665.
(10) Massey Dawkins, C.J. (1945). Proc. roy. Soc. Med., 38, 298.
(11) Bromage, P.R. (1954). "Spinal Epidural Analgesia." Edinburgh: E. & S. Livingstone Ltd.
(12) Hingson, R.A. (1949). Brit. med. J., 2, 777.
(13) Vandam, L.D. and Dripps, R.D. (1956). J. Amer. med. Ass., 161, 586.
(14) Cope, R.W. (1954). Anaesthesia, 9, 249.
(15) Robson, J.M. and Keele, C.A. (1956). "Recent Advances in Pharmacology," p. 17. London: J. & A. Churchill Ltd.

II. ANATOMY

BOUNDARIES OF THE EPIDURAL SPACE

The epidural space extends from the foramen magnum to the sacral hiatus. It constitutes that portion of the spinal canal outside the dura mater. At the foramen magnum the spinal dura mater is continuous with the inner layer of the cranial dura and at this point it becomes adherent to the periosteum of the foramen magnum. Within the cranium the periosteum and the inner layer of cranial dura are closely adherent except where they separate to enclose venous sinuses.

It is apparent, therefore, that fluid injected into the epidural space cannot extend higher than the foramen magnum, unlike fluid injected into the sub-arachnoid space.

The anterior surface of the vertebral canal is made up of the posterior longitudinal ligament which runs from the axis vertebra to the sacrum. It is narrow over the vertebrae but widens over the intervertebral discs and is attached to the posterior surface of the vertebral bodies and intervertebral discs.

The lateral wall of the canal is composed of the pedicles of the vertebrae and the intervertebral foraminae.

The ligamenta flava and the vertebral laminae form the posterior aspect of the epidural space. The ligamenta flava (Fig. 1) are of great importance in the identification of the space as will be seen later. They are pairs of very strong elastic sheets that connect the laminae of adjacent vertebrae, /

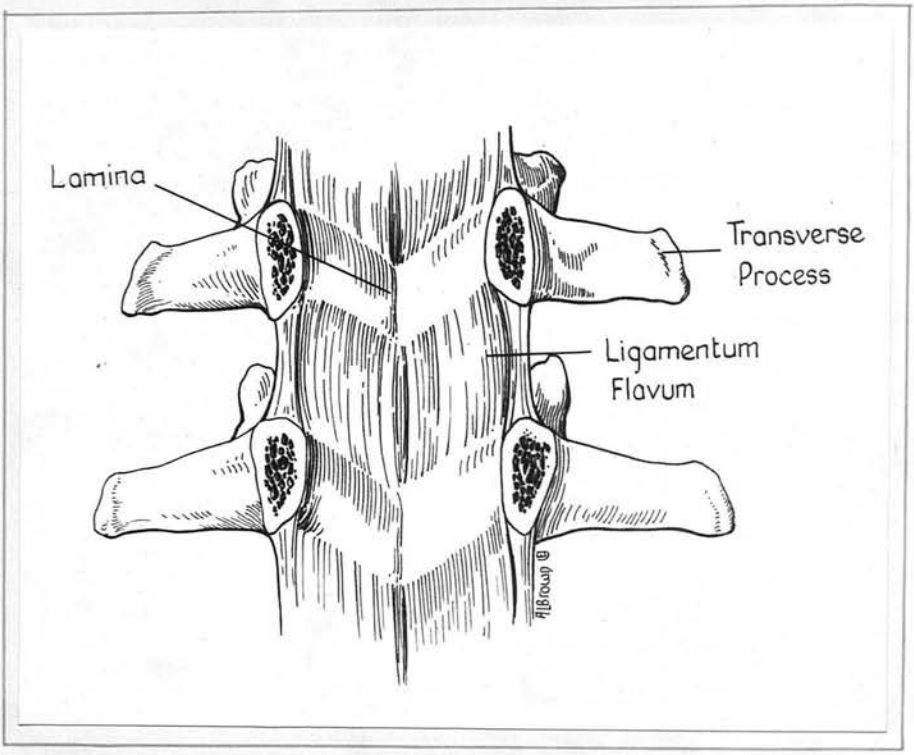

Fig. 1.- Ligamenta flava (anterior aspect).

vertebrae, being rather thin in the cervical region, but thick in the lumbar region. Laterally they fuse with the capsules of the synovial joints between the articular processes of the vertebrae, while medially they are separated by a little fatty areolar tissue. Inferiorly they are attached to the outer surface and upper borders of the laminae; and superiorly to the inner surface and lower border of the laminae (except in the thoracic region where they are only attached to the inner surfaces). They form the last structure to be traversed by a needle being inserted into the epidural space from the back.

The other structures through which a needle passes to reach the space from the back are the skin, superficial and deep fascia, supraspinous and interspinous ligaments (Fig. 2). The supraspinous ligaments form a long, continuous band attached to the tips of the spinous processes along the whole length of the column. In the neck it widens into the triangular ligamentum nuchae which separates the muscles on the two sides of the back of the neck and gives origin to the trapezius, splenius and rhomboideus minor muscles.

The interspinous ligaments connect adjacent vertebral spines.

CONTENTS OF THE EPIDURAL SPACE

The epidural space is filled with areolar tissue enclosing fatty lobules. Through it run blood vessels which supply both the structures within the vertebral canal and /

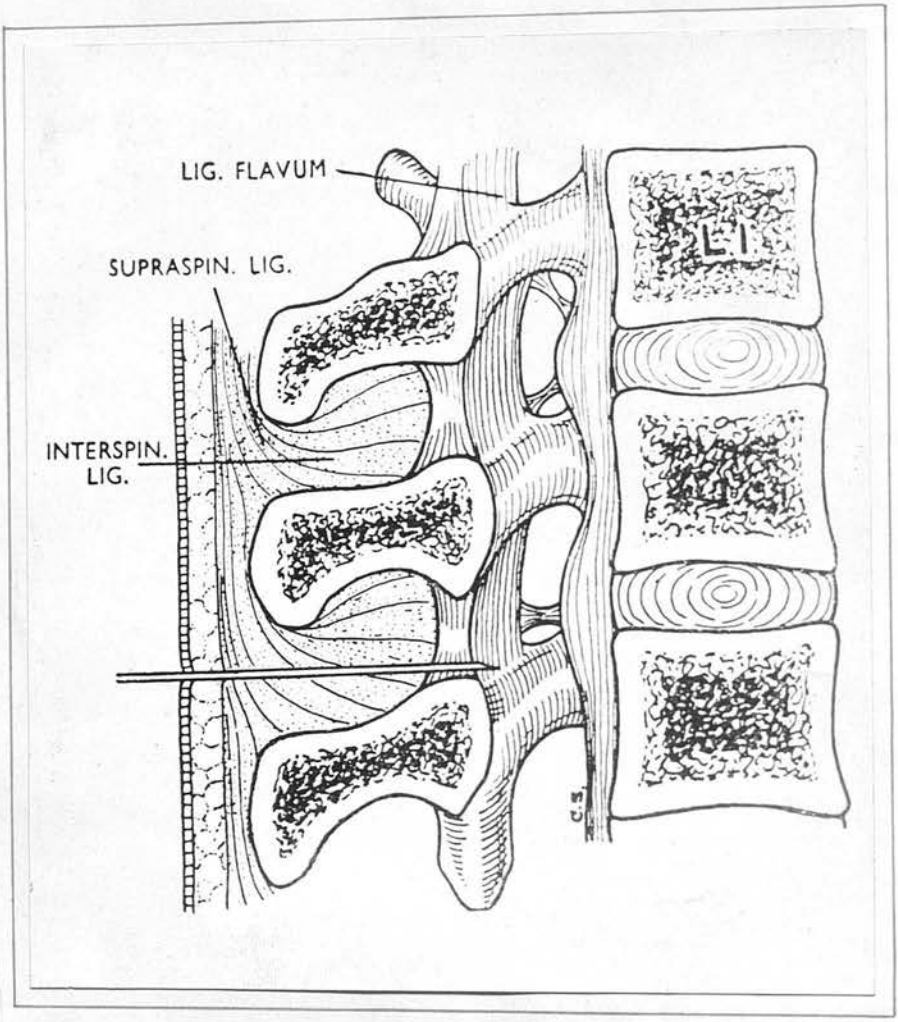

Fig. 2.- Sagittal section through lumbar spine showing structures traversed by needle inserted into epidural space.

and the walls of the canal. It is also traversed by the spinal nerve roots running from the dural sac to the appropriate intervertebral foraminae.

The blood vessels are the spinal arteries and veins and their branches. The arteries are small. In the neck, they are derived from the vertebral and the ascending and deep cervical arteries. Lower down they arise from the intercostal, lumbar and lateral sacral arteries. The veins are larger and more numerous than the arteries. In the epidural space they form networks called the internal vertebral venous plexuses. These are drained by intervertebral veins which join tributaries of the vertebral, intercostal, lumbar and lateral sacral veins. They also communicate with a network of veins on the outside of the vertebral column called the posterior external plexuses, and with the cranial venous sinuses near the foramen magnum. Two pairs of longitudinal channels, anterior and posterior, run in the intervertebral venous plexuses. The anterior channels run on the back of the vertebral bodies, at the edges of the posterior longitudinal ligaments; and the posterior channels are on the deep surface of the laminae. These channels receive lymph from lymphatics draining the vertebral canal and its contents.

It will be seen that the epidural space is a highly vascular area, being especially well supplied with veins, and that the danger of too rapid absorption of local anaesthetic is /

is real. In addition, the delicate venous networks can be readily damaged by the passage of needles or catheters.

DURAL SAC AND ITS CONTENTS

The spinal dura mater forms the outermost of the three meninges of the spinal cord, the others being the arachnoid mater and the pia mater. Each is continuous inside the cranium with the corresponding meninges of the brain.

The pia mater closely invests the spinal cord whose supplying blood vessels ramify within it. The cord and the pia mater end at the level of the space between the first and second lumbar vertebrae (though this is subject to some individual variation), except for the filum terminale which runs on to the end of the spinal canal, piercing the lowest part of the arachnoid and dura, and ending by fusing with the periosteum covering the back of the coccyx. On each side of the cord runs the ligamentum denticulatum which is a longitudinal ridge of pia mater that sends out lateral processes or "teeth" which pierce arachnoid and are attached to dura mater. There are 21 such "teeth" from the foramen magnum to the first lumbar vertebra and they serve to anchor the cord in the middle of the arachno-dural tube.

Anteriorly the pia is folded into the anterior median fissure of the cord and is thickened to form the linea splendens.

The arachnoid mater is thin and transparent and invests the /

the cord only very loosely, ending far below it, with the dura, at the level of the second sacral vertebra. Between the arachnoid and the pia mater is the subarachnoid space which contains the cerebro-spinal fluid.

The dura mater is much thicker and stronger, and is separated from the arachnoid by the sub-dural space which is a capillary interval containing only lymph.

SPINAL NERVES

The spinal cord gives off 31 pairs of nerves (eight cervical, twelve thoracic, five lumbar, five sacral and one coccygeal), before ending at the level of the second lumbar vertebra. The upper seven cervical nerves leave the vertebral canal above the corresponding vertebrae, while the eighth leaves below the seventh vertebra. All the other nerves leave below their corresponding vertebra. Each nerve derives an anterior and a posterior root from the cord. The anterior root is composed of motor fibres that arise in the anterior horn of the cord. The posterior root contains sensory fibres that enter the cord posteriorly. Each posterior root has on it a ganglion, and each nerve cell within the ganglion gives rise to a sensory fibre which divides before leaving the ganglion into a central and a peripheral branch. The former enters the cord while the latter runs distally in the mixed spinal nerve.

The anterior and posterior roots leave the arachnoid

and /

and dura mater taking with them a dural cuff and they very quickly join as they run to the intervertebral foramen in the epidural space. The posterior root ganglion is situated in or near the intervertebral foramen. Distal to the ganglion, the fibres of each root intermingle to form the mixed nerve. The dura becomes closely attached to the nerve at the distal side of the ganglion and is continuous with the perineurium. The arachnoid mater is attached to the proximal end of the ganglion. It will be seen from this that the motor and sensory roots of a spinal nerve unite proximal to the posterior root ganglion, but only distal to the ganglion do the motor and sensory nerve fibres intermingle.

Soon after leaving the intervertebral foramen the mixed spinal nerve divides into an anterior and a posterior primary ramus just after giving off a meningeal branch. A white ramus communicans leaves, and a grey ramus returns to, the mixed nerve in the paravertebral space on their way to and from the sympathetic chain. Only the nerves from the first thoracic to the third lumbar segments give off a white ramus, while all spinal nerves receive a grey ramus.

Because the spinal cord ends at the level of the second lumbar vertebra, the nerve roots, which are short and horizontal in the cervical and upper thoracic regions, become progressively longer and more oblique further down the cord. The lower lumbar, the sacral and the coccygeal roots form a loose /

loose bundle called the cauda equina, in the middle of which runs the filum terminale.

SACRAL CANAL

Any account of the anatomy of the sacral canal suffers from the marked individual variation that occurs in this region, but a description is necessary for a proper understanding of caudal analgesia.

The sacrum is formed by the fusion of the five sacral vertebrae, and the sacral canal is the lowest part of the vertebral canal. The sacral canal is wide and triangular above but tapers progressively, becoming flattened and rectangular as it goes downwards. It ends at the sacral hiatus, which is formed by the non-union of the laminae of the fifth sacral vertebra and is bounded by two small processes, the sacral cornuae, which can be felt through the skin. Often there is also non-union of the laminae of the fourth and even the third sacral vertebrae making a long, large sacral hiatus bounded by more than one pair of cornuae. The hiatus is covered posteriorly by the superficial posterior sacro-coccygeal ligament which may ossify.

An important feature of the sacral canal is the angulation it undergoes in its course. Until it reaches the sacrum, the vertebral canal follows the gentle curvature of the spinal column. In the lower lumbar region the canal is inclined downwards and slightly backwards. As it enters the /

the sacrum it bends sharply backwards and runs an arched course to the sacral hiatus where it is again inclined downwards. The posterior surface of the sacrum when viewed externally gives a somewhat false idea of the degree of this angulation because, although the canal ends at the posterior surface of the sacrum, it starts considerably deeper than the posterior surface.

In dissecting out a number of sacral canals, the writer was struck by the individual variation of this angulation which was so marked in some cases as to form almost two right angles, one at the lumbo-sacral junction and the other at the level of the third or fourth sacral vertebrae. Where angulation of this degree is present, it obviously imposes a considerable impediment to the upward flow of local anaesthetic introduced through the sacral hiatus. This is especially so when the patient is lying face downwards (the usual position for caudal block) when the lumbo-sacral joint is extended, for gravity plays a part in resisting the upwards spread of solution. On the other hand lumbar epidural blocks are usually performed with the patient in the lateral position, when gravity is relatively unimportant. Moreover, the patient is asked to bend into the fully flexed position when the lumbo-sacral angle tends to be straightened out, thus allowing an easy flow of local anaesthetic solution caudally to reach the sacral nerve roots. This change in the angulation of the spinal canal with posture is shown in Fig. 3.

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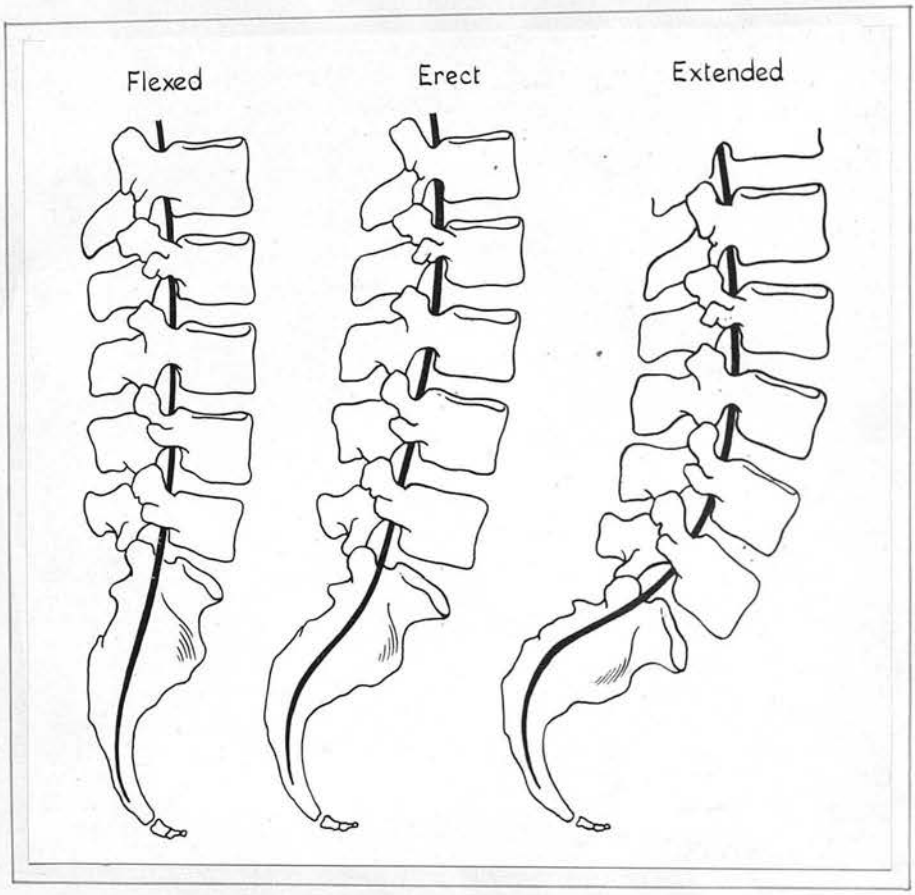


Fig. 3.- Variation in angulation of lower spinal canal with posture.
Fluid can spread with greater facility in the flexed position than in the extended.

Inside the sacral canal the dura and arachnoid end in most cases at the level of the first or second sacral vertebra. The sacral and coccygeal nerve roots run from the dura to their appropriate intervertebral foraminae, where they divide into anterior primary rami, which leave through the anterior sacral foraminae; and posterior primary rami, leaving through the posterior sacral foraminae.

The filum terminale pierces the tip of the dural sac and runs down to fuse with the periosteum covering the back of the coccyx.

The two roots that make up each sacral nerve leave the dura close together and quickly unite, but do not intermingle proximal to the posterior root ganglion. The ganglia are situated at a variable distance from the dura. In some individuals they are quite close to the dura, while in others they are close to the intervertebral foraminae (Fig. 4). In any individual it may be said that the second sacral ganglion is lower than the first, and third lower than the second. The fourth, however, is at about the same level as the third and the fifth is a little higher than the fourth. The coccygeal nerve ganglion is said to be intradural, though it is seldom visible macroscopically. As in the case of nerve roots higher up, the sacral and coccygeal nerve roots have a prolongation of dura and arachnoid as far as the posterior root ganglion. If, as there is some reason to believe, local anaesthetic introduced into the epidural space exerts much of its action on the posterior root /

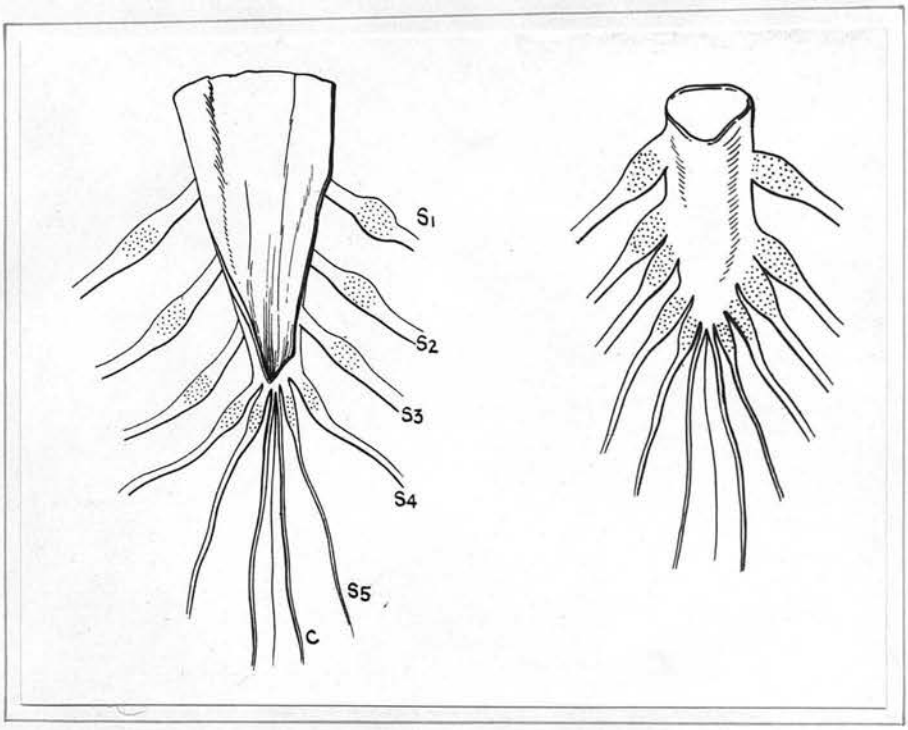


Fig. 4.- Sacral nerve roots and lower ends of dural sacs (from specimens removed at dissection). These specimens show the variability in the position of the posterior root ganglia. The left specimen shows the ganglia at some distance from the dural sac while in the right specimen they are close to the sac.

root ganglia, then the variability in the position of these structures in different individuals may be of importance.

As in the vertebral canal higher up, the sacral canal contains a quantity of fat and numerous blood vessels. Branches of the lateral sacral arteries enter the canal via the anterior sacral foraminae, while the posterior foraminae transmit the terminal branches of the arteries with their accompanying veins. Inside the canal the veins form a plexus communicating with the venous plexus in the lumbar vertebral canal.

The fat in the sacral canal shows one difference from that found in the rest of the vertebral canal, in that it appears to be very much more closely packed. Even in the very thin bodies seen in the dissecting room, fat bulges out of the canal as the posterior wall of the sacrum is removed. It is quite possible that this tight packing of fat constitutes an impediment to the spread and penetration of local anaesthetic.

From the limited number of dissections carried out (20 cases), it may be said that the variable degree of angulation, the variable position of the sacral posterior nerve root ganglia, and the tight packing of fat all contribute to unpredictability in the action and spread of local anaesthetic injected into the sacral canal. It is well known that much of the fluid escapes through the anterior sacral foramina and it is easy to see how, on simple /

simple hydrostatic grounds, an impediment to the upward flow of solution may cause an increased loss of local anaesthetic through these foramina.

Two common anatomical anomalies are also of importance in caudal block analgesia. Firstly, non-fusion of the laminae of the fourth sacral vertebra and occasionally of the third also, creates an abnormally large sacral hiatus, account of which must be taken when inserting the exploratory needle. Secondly, the dura mater may extend below the second sacral vertebra, and sub-arachnoid penetration may occur though the needle has not been advanced far into the sacral canal.

SPREAD OF SOLUTION IN THE EPIDURAL SPACE

Except in the sacral canal, which is packed tight with fatty tissue, the contents of the epidural space offer little or no resistance to the passage of injected fluid. When fluid is injected into the lumbar epidural space its spread will depend upon several factors:-

(a) Volume of fluid injected.- The amount of fluid injected obviously affects the distance that it will travel from the needle. This is the most important single factor.

(b) Gravity.- This is relatively unimportant if the patient is in the lateral position, although its effect can often be seen when the patient exhibits a higher segmental block, by up to three dermatomes, on the dependent side.

Some /

Some anaesthetists use the sitting position when gravity would obviously play a much bigger part. Others attempt to augment the spread of their injection by tilting the patient, either head up to obtain a low block, or head down for a high block. The author has given up tilting the patient, as the evidence that it produced a substantial effect was unconvincing. The angle to which a patient can be tilted is quite limited, and with the exception of the use of the sitting position, gravity is only of small practical value.

(d) Speed of injection.- Rapid injection causes the solution to run much farther than slow injection although its spread will be "thinner." In practice forceful injection is not necessary in lumbar epidural analgesia (though it may be in sacral epidural analgesia). Because the epidural space is richly supplied with blood vessels, rapid absorption of local anaesthetic with toxic reactions is a constant hazard. The higher the pressure used during injection, the more likely is such a reaction to occur. Moore, Bridenbaugh, Van Ackeren, Belda and Cole[1] have shown, by the injection of iodopyracet into cadavers, that 20 ml. injected epidurally through a suitable vertebral interspace is sufficient for any operation below the diaphragm. They used a rapid injection of not less than 1 ml. per second. In their clinical practice, however, this fast injection of a relatively small amount of local anaesthetic produced inadequate anaesthesia in 4 per cent. of cases, presumably because the spread was too "thin."

(d) /

(d) Site of injection.- For success, regional anaesthesia must block a minimum number of dermatomes, depending upon the surgery to be carried out. The nearer the needle is introduced to the centre of the prospective region of analgesia, the less local anaesthetic need be injected. For example, a laparotomy requires a band of analgesia from the seventh thoracic segment to the first lumbar segment, a total of seven segments. If a needle is introduced between the ninth and tenth, or the tenth and eleventh thoracic vertebrae, only enough anaesthetic to cover four dermatomes on either side will be required. On the other hand, if the needle is placed between the first and second lumbar vertebrae, the fluid must spread upwards to the seventh thoracic segment, a distance of seven dermatomes (Fig. 5).

Some anaesthetists use this principle to reduce the volume of local anaesthetic. Its application, however, involves a high percentage of thoracic epidural punctures which are technically more difficult, and less safe, than lumbar epidural punctures. The use of a larger volume of local anaesthetic by the safer lumbar route has not proved to be any great disadvantage, provided it is given slowly.

A caudal block starts from an immediate disadvantage because as the spread is unidirectional, the local anaesthetic must travel a considerable distance to reach the thoracic nerves.

(e) /

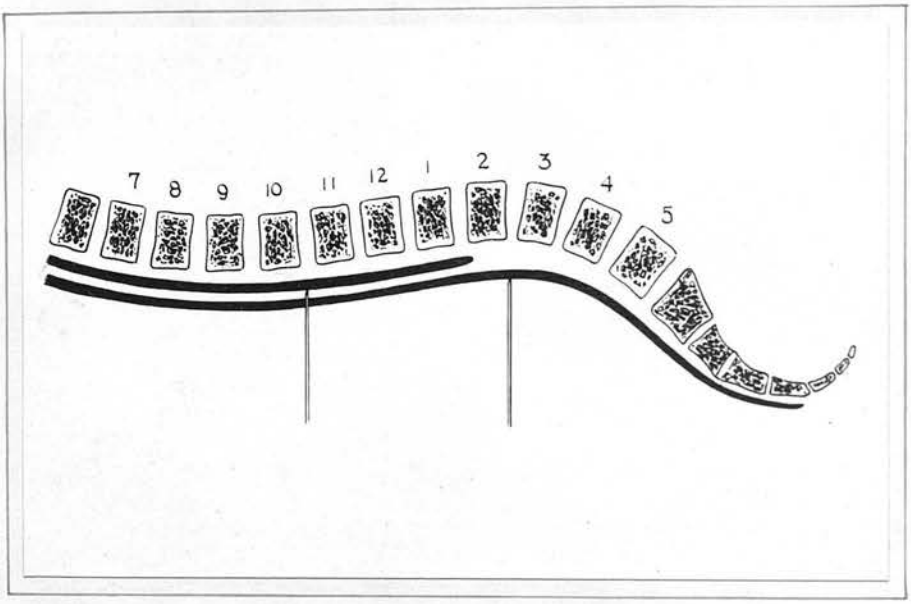


Fig. 5.- Diagram indicating the spread of local anaesthetic injected into the epidural space. Injection at the centre of the intended band of analgesia requires considerably less local anaesthetic than when made at a lumbar interspace.

(e) Loss of fluid through intervertebral foramina.- The epidural space is not a closed space. The intervertebral foramina form openings on each side, through which injected fluid can escape into the paravertebral space. The level to which local anaesthetic will spread largely depends on how much leakage takes place in this way. It should be noted that with increasing age, the foramina tend to become stenosed, and it is an observable fact that the more elderly patient requires considerably less local anaesthetic than a younger one, to produce the same level of anaesthesia.

Bromage[2], as a result of radiographic studies, states that while the loss through the intervertebral foramina is considerable in the lumbar and upper thoracic regions, it is minimal or absent in the mid-thoracic region. Moore et al.[1], however, found leakage from every foramina. This discrepancy is probably due to a difference in technique. In Bromage's cases, 5 ml. of non-viscous opaque oil ("Neohydriol") was injected after a slow injection of local anaesthetic. It did not necessarily follow that the oil reached to the limits of the aqueous injection. Moore and his co-workers used rapid injections of 20-40 ml. of an aqueous solution of iodopyracet in cadavers. In interpreting the results of such experiments, it should be remembered that conditions in the epidural space are quite different after death, when there is cessation of the normal pulsations of the dural sac and of the blood vessels within the

epidural /

epidural space.

SITE OF ACTION OF EPIDURALLY PLACED LOCAL ANAESTHETICS

For many years it seemed obvious that in epidural analgesia the spinal nerves were being blocked as they left the dural sac and ran through the intervertebral foramina to reach the paravertebral space. Indeed, this was stated to be one of the great advantages over spinal analgesia, as no local anaesthetic was introduced into the cerebro-spinal fluid.

In 1951, Rudin, Fremont-Smith and Beecher[3] published a paper describing the results of experiments on dogs in which they showed clearly that procaine injected into the epidural space did penetrate the dura mater, and up to 10 per cent. could be recovered in the cerebro-spinal fluid. These experiments, however, though carefully designed to avoid error, differed radically from conditions prevailing during clinical epidural analgesia. The entire epidural space was filled with 2-3 per cent. procaine, the pressure of which caused considerable emptying of cerebro-spinal fluid from the spinal sub-arachnoid space. In addition, the sub-arachnoid space was continuously perfused throughout the experiment. Under these conditions they found that the concentration of procaine in the sub-arachnoid space rose to 0.3-0.8 mg. per ml., and that this was sufficient to affect the spinal nerve roots and the spinal cord itself.

Frumin, /

Frumin, Schwartz, Burns, Brodie and Papper[4] in 1953 also noted this phenomenon in humans under ordinary anaesthetic conditions. They inserted two plastic catheters, one into the epidural space, and one into the sub-arachnoid space, both being advanced cephalad to the region of the twelfth thoracic vertebra. On injecting 20 ml. of 2 per cent. procaine through the epidural catheter, procaine could be detected in the cerebro-spinal fluid collected from the other catheter and its level seemed to follow the intensity of the clinically determined analgesia. These results have been criticised on the grounds that the leakage of procaine into the cerebro-spinal fluid could occur round the point at which the intrathecal catheter pierced the dura. In fact, this was not a serious cause of error, because when the tip of the intrathecal catheter was placed at the level of the second sacral vertebra, i.e. at the same distance from, but distal to, the dural puncture, no procaine could be detected in the cerebro-spinal fluid. If any criticism is to be made of these experiments, it must be of the conclusion that the average concentration of procaine in the cerebro-spinal fluid was about 0.2 mg. per ml. which is the threshhold concentration for effective spinal block. In fact, of eight cases studied, only two reached this level, while three were below 0.15 mg. per ml. (one as low as 0.06 mg. per ml.), although analgesia was satisfactory. In one case there was no analgesia though the procaine level in the cerebro-spinal fluid reached 0.17 mg. per ml. However, the authors only claim /

claim that part of the analgesia was due to the dural penetration of procaine.

As a result of these two publications, many anaesthetists now believe that epidural analgesia produces its main effect within the sub-arachnoid space. On the other hand, there is much evidence against such a conclusion.

Even allowing that 10 per cent. of the local anaesthetic is absorbed into the cerebro-spinal fluid this would not be enough to produce full surgical analgesia. Four to six times more anaesthetic must be injected in an epidural block than would be required for a spinal block; but not 10 times. From an injection of 20 ml. of 2 per cent. procaine (400 mg. of procaine), only 40 mg. would reach the sub-arachnoid space. For a spinal anaesthetic this is a very modest amount, and unlikely to produce a widespread blockade.

Foldes and Davis[5], using substantially the same technique as Frumin and his co-workers[4] (the intrathecal catheter being replaced by a 22 gauge needle), showed that, with 3 per cent. 2-chloro-procaine, the level of the drug in the cerebro-spinal fluid lagged behind the sensory and motor anaesthesia determined clinically. The average cerebro-spinal fluid concentration at seven to twelve minutes (the time of fully developed sensory and motor blockade) was only 0.06 mg. per ml. The highest concentration was reached after 30 minutes when it was, on average, 0.23 mg. per ml. Moreover, it was still 0.2 mg. per ml. when the motor block ceased and 0.17 mg. per ml. when /

when the sensory block ceased.

On anatomical grounds it would seem more likely for a local anaesthetic in the epidural space to produce its main effect where the nerves are least protected by their coverings. As we have seen, proximal to the posterior root ganglion, there are coverings of dura and arachnoid mater. The arachnoid ends at the proximal end of the ganglion while the dura becomes attached to the nerve at the distal end of the ganglion (where it continues as the perineurium). Thus the nerve possesses greater barriers to penetration by local anaesthetic proximal to the posterior root ganglion than it does distal to the ganglion. There are some grounds for believing that the injected drug affects the ganglion itself, for sensory loss is the most obvious feature of the epidural blocks. This can be produced with concentrations of 1 per cent. lignocaine and even 2 per cent. still spares much of the motor function of the nerve.

One other region of the body, where nerve blocks are frequently carried out and have a bearing on this question, is the orbit. Retro-bulbar injections of 2 per cent. lignocaine produce complete sensory blockade of the eyeball, but leave sight unimpaired. The optic nerve, like spinal nerves in their intrathecal course, has a covering of dura and arachnoid with the cerebro-spinal fluid circulating right up to the eyeball itself, and is apparently protected from local anaesthetic drugs.

Hyaluronidase has been added to the local anaesthetic solutions /

solutions used in epidural anaesthesia in an attempt to hasten the onset of analgesia (Scott[6]). This desired effect was produced in some degree, more noticeable in caudal than in lumbar epidural blocks. It is known that hyaluronidase does not exert its spreading effect through fascial planes (Moore[7]), and therefore it is unlikely to increase the rate of absorption through the dura and arachnoid mater. If the onset of analgesia is more rapid when hyaluronidase is used, its effect must, therefore, be peripheral to the dura. That hyaluronidase does not increase absorption through the dura is again shown by retro-bulbar injection, which does not affect vision even when hyaluronidase is added to the anaesthetic solution.

Rudin et al.[3] showed in their experiments that the procaine absorbed into the cerebro-spinal fluid affected conduction in the spinal cord itself. If this were applicable under normal conditions of epidural analgesia, segmental block would be virtually impossible to produce, for spinal cord conduction would be blocked, and all segments below the site of the blockade would be affected. However, segmental analgesia with normal sensation both above and below the area of analgesia can be easily produced by epidural block.

While the whole question of the site of action of epidurally placed local anaesthetics is open to further research, it is reasonable, at the present time, to say that, though penetration does occur into the cerebro-spinal fluid, the /

the amount absorbed is insufficient to produce the full effect of the blockade. The main action would, therefore, appear to be distal to the proximal end of the posterior root ganglion.

References

(1) Moore, D.C., Bridenbaugh, L.D., Van Ackeren, E.G., Belda, F.B. and Cole, F.V. (1958). Anesthesiology, 19, 377.

(2) Bromage, P.R. (1954). "Spinal Epidural Analgesia," p. 17. Edinburgh: E. & S. Livingstone, Ltd.

(3) Rudin, D.O., Fremont-Smith, K. and Beecher, H.K. (1951). J. appl. Physiol., 3, 388.

(4) Frumin, M.J., Schwartz, H., Burns, J.J., Brodie, B.B. and Papper, E.M. (1953). J. Pharmacol. exp. Ther., 109, 102.

(5) Foldes, F.F. and Davis, D.L. (1954). J. Pharmacol. exp. Ther., 110, 18.

(6) Scott, D.B. (1956). Brit. J. Anaesth., 28, 187.

(7) Moore, D.C. (1951). Anesthesiology, 12, 611.

III. PHYSIOLOGY

The way in which local anaesthetic solutions spread in the epidural space, and their probable site of action, have been discussed.

The physiological response to epidural nerve block can be divided into: (a) the local effects on each affected nerve and (b) the generalised effects of the multiple nerve block that ensues. Motor and sensory nerve paralysis produces changes that are confined mainly to the segmental distribution of the affected nerves. Autonomic blockade, however, and, in particular, sympathetic blockade, causes widespread physiological changes.

EFFECTS OF EPIDURAL BLOCKADE UPON SPINAL NERVES

When the soluble salts of local anaesthetics are injected into tissues the buffering mechanism of these tissues causes precipitation of the insoluble anaesthetic base, e.g. procaine hydrochloride reacts with sodium bicarbonate to form procaine base, sodium chloride and carbonic acid:

Procaine HCl + $NaHCO_3$ ⟶ Procaine base + NaCl + H_2CO_3.

The local anaesthetic base, though insoluble in water, is relatively soluble in lipoids, and is, therefore, selective in its action, being absorbed by nervous tissue.

A mixed spinal nerve contains motor, sensory and autonomic fibres. These differ from each other in size, motor being the largest and autonomic the smallest. Somatic motor /

motor fibres to skeletal muscle vary in diameter from 12-30 μ. Afferent somatic fibres vary from 1-20 μ, although those fibres in the range 14-20 μ are derived from muscle nerves (these fibres are proprioceptive and mediate stretch reflexes). Cutaneous nerve fibres are almost all in the range of 5-13 μ. Sympathetic nerve fibres are either myelinated (pre-ganglionic), and have a size of 1-3 μ, or non-myelinated (post-ganglionic) of less than 1 μ in diameter (Fulton[1]). It is a common observation that the thicker the nerve fibre, the more difficult it is to block with a local anaesthetic, and this differential action is thought to be due to the differences in the thickness of the myelin sheath (Sollman[2]). With normal microscopic techniques, the myelin sheath appears to form a considerable proportion of the diameter of a nerve fibre, and the differences between small and large fibres have been thought to be mainly due to the thickness of the myelin. With the electron microscope however, very much thinner sections can be studied, and by this method the myelin sheath is seen to be considerably less bulky than when viewed by an optical microscope. Although the myelin/axon ratios of the various types of nerve fibre have not yet been finally worked out, it may be said that the main bulk of a fibre is made up by its axon (Muir[3]). In view of this, it is probably the axon which constitutes the main barrier to the action of local anaesthetics, and this concurs with the view (Evans and Gray[4]) that these drugs exert their action at the nodes of Ranvier, where the myelin sheath is /

is absent.

As a result of the difference in sensitivity of nerve fibres to the blocking action of local anaesthetic drugs, the extent to which a mixed nerve is anaesthetised depends upon the concentration of drug injected. By selecting the concentration, different degrees of nerve block can be obtained and the so-called "differential nerve block" produced. In epidural block 0.5 per cent. lignocaine produces autonomic blockade without sensory or motor loss. One to two per cent. produces sensory blockade in addition, while motor loss will not be complete unless a solution in excess of 2 per cent. is used.

Epidural block differs in this respect from spinal anaesthesia. In the latter a concentrated solution of local anaesthetic is injected into the sub-arachnoid space where it spreads, being diluted as it travels by cerebro-spinal fluid. Thus, at the upper extent of its spread it is only concentrated enough to affect autonomic nerve fibres; lower down sensory block is also present, and lower still all three types of nerve fibre, autonomic, sensory, and motor, are affected. Only above the level of the main nerve block is there an area of differential block.

When local anaesthetic solutions are injected into the epidural space, however, no dilution occurs and the same degree of differential nerve block will occur at all levels (Fig. 6).

When discussing the problem of the site of action of epidurally /

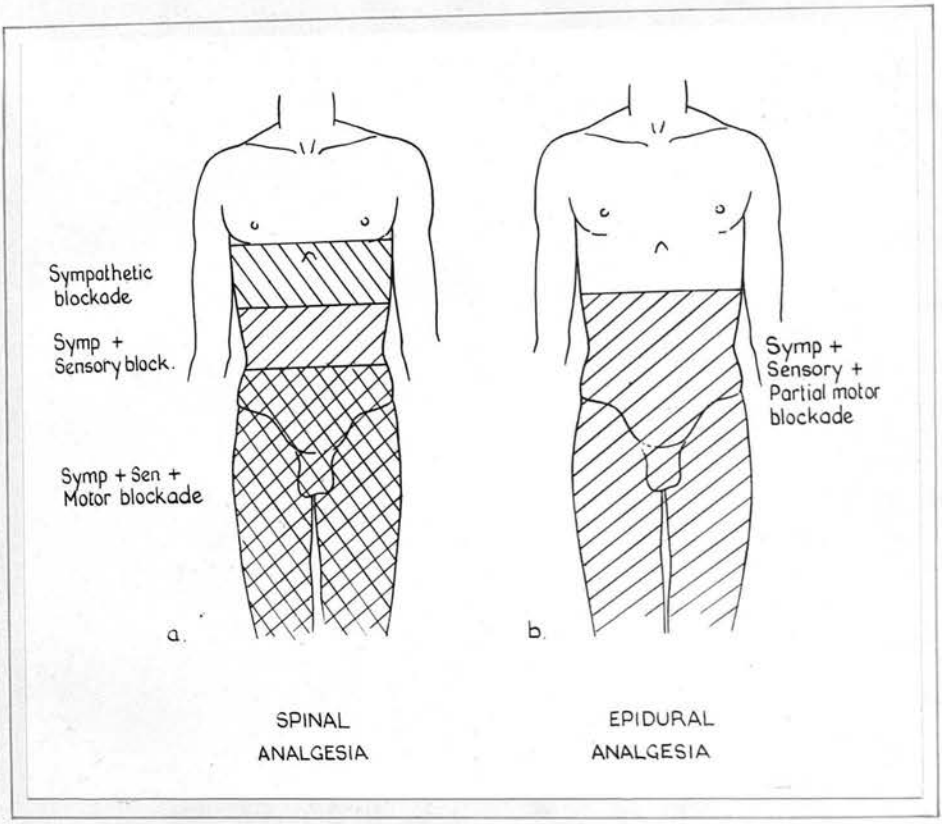


Fig. 6.- Pattern of nervous blockade during (a) spinal and (b) epidural analgesia. Owing to the dilution of local anaesthetic in its upward spread in the cerebrospinal fluid, spinal analgesia produces three bands of nerve block. As local anaesthetic in the epidural space is not diluted, only one type of nerve block is produced throughout, depending upon the strength of solution chosen. In clinical practice, a concentration is used that produces only partial motor blockade.

epidurally placed local anaesthetics (p. 27), it was noted that a certain quantity of drug is absorbed into the sub-arachnoid space. It is theoretically possible that the drug so absorbed could diffuse in the cerebro-spinal fluid and produce autonomic blockade at a higher level than was reached by the epidural solution (Fig. 7). The author has noticed on occasion the typical signs of high spinal block-ade, i.e. hypotension and bradycardia, although sensory block was only demonstrable up to the seventh thoracic segment. This aspect of epidural analgesia requires further investigation. Unfortunately, there are very few objective tests of autonomic blockade which are applicable under anaesthetic conditions. The author has measured skin temperature and skin resistance to test the onset of sympathetic paralysis, but premedication itself produces a considerable increase in the blood flow to the limbs. The limbs are therefore warm and dry (the typical picture of sympathetic blockade) before epidural block is attempted.

With the concentrations of local anaesthetic commonly used for epidural analgesia, motor loss is far from complete. Nevertheless, muscle relaxation is very marked. This is due to the interruption of the afferent component of the spinal reflex arc. Though the muscle will respond to an efferent nerve impulse, its resting tone is abolished. No stimuli are received from the operation area, and the muscles remain slack. This explains why intercostal movement remains unimpaired during respiration, although other muscles

supplied /

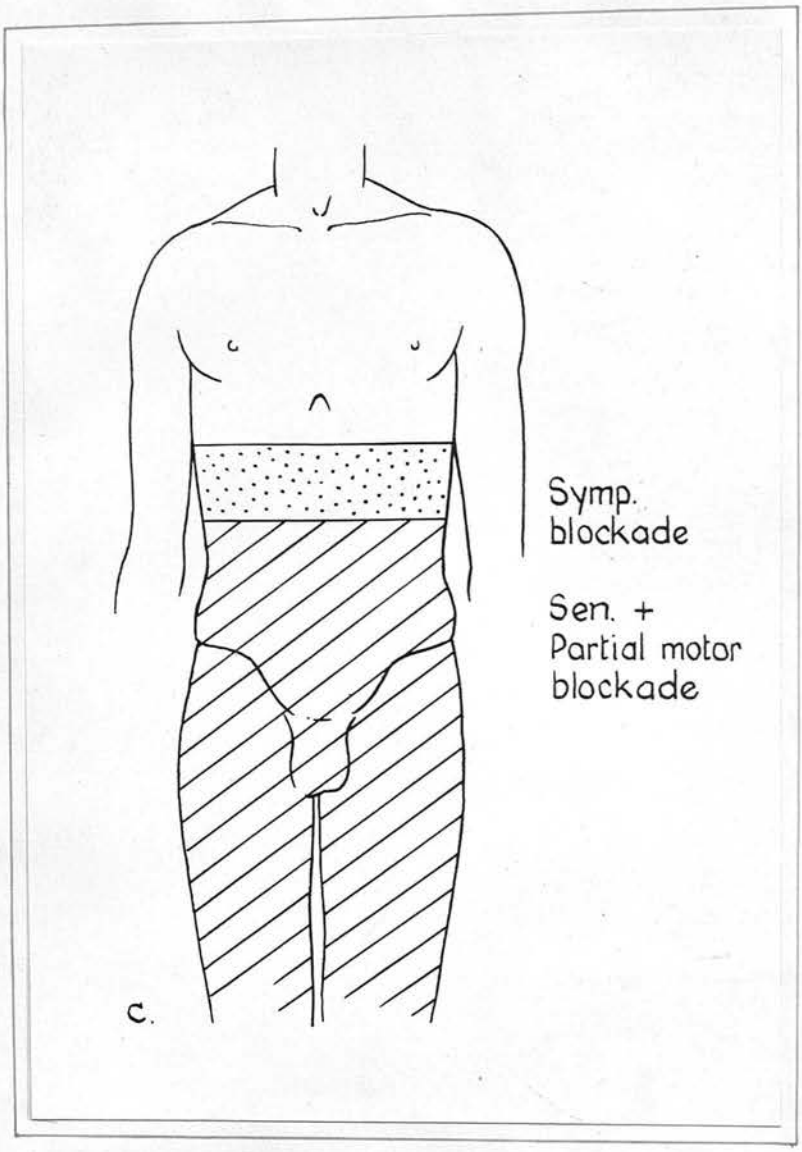


Fig. 7.- Suggested pattern of nervous blockade in epidural analgesia. The sympathetic blockade occurring above the band of analgesia is due to absorption of local anaesthetic into the cerebro-spinal fluid, where a further upward spread can occur.

supplied by the lower thoracic nerves, in particular the abdominal muscles, are relaxed. This action on skeletal muscles can, therefore, be said to be a true relaxation, rather than a paralysis as produced by spinal anaesthesia or specific muscle "relaxants".

While the advantages of completely normal respiration are obvious, it must be remembered constantly in epidural analgesia that the abdominal muscles do retain, to a certain extent, the ability to contract. Should coughing or straining occur for any reason, e.g. due to interference with the pharynx or larynx, abdominal relaxation can quickly disappear.

The effects, then, of motor and sensory blockade are mainly localised to the area supplied by the affected nerves. Unless the block is a very high one and motor loss is considerable, there will be few generalised physiological changes produced in the patient. Cases of total epidural blockade, in which apnoea occurs as a result of paralysis of both the intercostal muscles and the diaphragm, have been reported (Lee[5]), but it is very difficult in these cases to decide whether or not a total spinal anaesthetic has not been produced inadvertently. In the author's experience, respiration has been uniformly normal and unimpaired.

MODIFICATIONS OF PHYSIOLOGY PRODUCED BY EPIDURAL BLOCKADE

Generalised physiological changes are almost entirely the result of autonomic blockade. The sympathetic outflow arises /

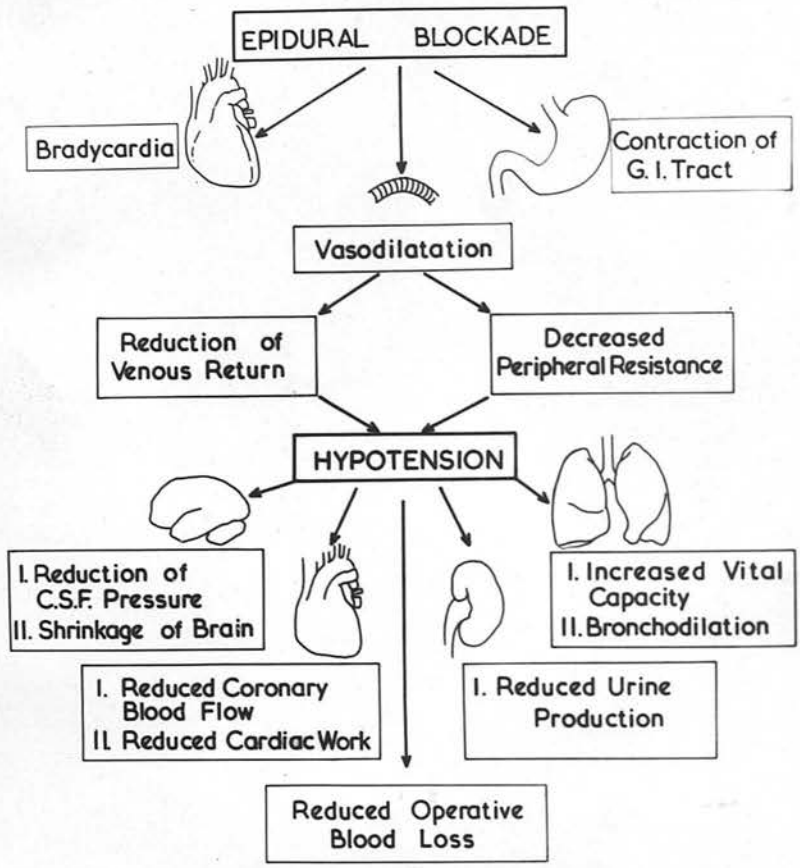


FIG.8 PHYSIOLOGICAL EFFECTS OF EPIDURAL ANALGESIA

Fig. 8.- Schematic diagram of the physiological effects of epidural blockade.

arises from the first thoracic to the second lumbar spinal segments. As most abdominal operations require analgesia up to at least the seventh or eighth thoracic segments, it is obvious that epidural analgesia will often block a considerable number of sympathetic nerve fibres. Post-ganglionic fibres from the sympathetic chain (grey rami communicantes) reach each spinal nerve in the paravertebral space and epidural block can, therefore, cause sympathetic blockade in spinal nerves below the second lumbar segment. Parasympathetic fibres arise in the sacral segments of the spinal cord and will be likewise affected by low blocks.

The main effects of epidural analgesia are on:

(A) Cardiovascular system (leading to hypotension which itself affects other organs in the body).

(B) The gastro-intestinal tract.

(A) Cardiovascular System

Autonomic blockade produces its main effect on blood vessels, but in addition the heart itself is affected.

Heart.- The only direct effect of nervous blockade upon the heart is bradycardia. Secondary effects due to hypotension, which may result from paralysis of sympathetic vasoconstrictor nerves, can occur, and these will be considered later.

Both spinal and epidural block produce bradycardia if there is an associated fall in blood pressure. Pugh and Wyndham[6] found that a fall of 25 per cent. in arterial pressure /

pressure is necessary before bradycardia develops. Slowing of the heart rate has been attributed to paralysis of the cardiac accelerator fibres. These fibres arise from the upper five thoracic segments of the spinal cord (White and Smithwick)[7], and therefore a very high blockade would be required to produce bradycardia if this were the only mechanism. Moreover, blockade of the cardiac accelerators can only explain why tachycardia does not occur, not why bradycardia does occur.

There are two main nervous reflexes which could affect the pulse rate under conditions of reduced venous return and hypotension. The stretch receptors situated in the great veins and the right atrium can cause tachycardia, if venous pressure rises, or bradycardia if venous pressure falls. This reflex is mediated in both the afferent and efferent pathways through the vagus nerve, which is unaffected by epidural or spinal blockade. Though the existence of this reflex has been questioned (Wiggers)[8], one clinical observation supports its role during spinal or epidural block. If hypotension and bradycardia are established, raising the legs and tilting the patient into the Trendelenburg position leads to an increase in the pulse rate as the venous return increases (Griffiths and Gillies[9]; Greene)[10].

The second reflex affecting cardiac rate is that arising in the baroreceptors of the aorta and carotid sinus which causes tachycardia as the arterial blood pressure falls. /

falls. Its effect, therefore, is exactly opposite to the former reflex. It can only be assumed that, as tachycardia does not occur under epidural or spinal anaesthesia, the reflex from the great veins and right atrium is more important in these conditions.

Ganglionic blocking drugs such as hexamethonium and trimetaphan cause hypotension with tachycardia. This difference is most probably due to the fact that these drugs affect both sympathetic and parasympathetic ganglia, and some degree of vagal block occurs.

Blood Vessels.- The autonomic nervous system exerts a large measure of control over the state of tone of blood vessels, and this tone is one of the main factors maintaining the level of the blood pressure. Although parasympathetic nerves can produce dilation in certain vessels, such as those to the brain, salivary glands and erectile tissue, it plays little or no part in the general haemodynamics. Though some authorities believe in the existence of parasympathetic vasodilators to the parietes and limbs (Mitchell[11]), their presence is difficult to prove, and there is little evidence that they play any part in the control of blood pressure.

Interference with vasomotor tone by sympathetic blockade leads to vasodilation. This causes pooling of blood and a reduction in the circulating blood volume. In a low epidural block, only a small amount of pooling will occur (in the legs), and this will be compensated for by vaso-

constriction /

vasoconstriction at a higher level. The higher the blockade reaches, fewer vessels are available to constrict and at some point compensation will become inadequate. The blood pressure will fall as a result of a reduced cardiac output (due to inadequate venous return) and reduced peripheral resistance.

Pugh and Wyndham[6] studied cardiac output and peripheral resistance in a series of patients, some normotensive and some hypertensive, who received high spinal analgesia. They found a reduction in cardiac output in all cases, varying from 5 per cent. to 46 per cent. (average 23 per cent.) in the supine position, and from 16 per cent. to 54 per cent. (average 36 per cent.) in the reverse Trendelenburg position.

There was no appreciable fall in total peripheral resistance unless the systolic pressure fell below 80 mm.Hg., and above this level the fall of blood pressure was due to reduction of the cardiac output. There was evidence that, in some hypertensive cases, an excessive degree of sympathetic vasoconstrictor tone existed, and in them the spinal anaesthesia produced a very considerable fall in blood pressure. The dangers of the reverse Trendelenburg position are well illustrated by these studies.

The height of blockade required for the production of hypotension varies according to the patient's ability to compensate for vasodilation. This depends upon the state of the vessels. Patients with normal vessels need a

blockade /

blockade well up into the thoracic region before any substantial drop in pressure occurs. Hypertensive patients on the other hand are very susceptible to the hypotensive action of both spinal and epidural blockade. This is presumably due to inability to compensate adequately. This is easily explained in the more elderly cases who have widespread arteriosclerotic changes in their blood vessels, for such vessels are only capable of a small degree of constriction. Many hypertensive patients, however, have reasonably elastic vessels with little or no arteriosclerosis. Wiggers[12] has pointed out that, though the main peripheral resistance is provided by the arterioles, the aorta and its larger branches form what he terms the "compression chamber", and even quite mild vasospasm in them can cause a considerable increase in systolic pressure. It follows therefore that vasodilation of the larger arteries, which would have little effect in normal patients, can cause considerable drops in the systolic pressure of hypertensives. Moreover, reduction of cardiac output leads to a decrease in the amount ejected by the heart into the compression chamber. If the aorta is relatively inelastic, the systolic pressure will fall disproportionately. This is also of importance if atherosclerosis of the larger vessels is present.

Susceptibility to hypotension is also seen in patients suffering from a diminished circulating blood volume and who are only maintaining their blood pressure by vasoconstriction. Vasodilation produced by even a low epidural block could, in these /

these circumstances, cause profound hypotension, which, in the continued presence of low blood volume, might well be very dangerous. Shocked patients therefore, are usually unsuitable for this form of anaesthesia.

At one time, it was believed that hypotension was slight or absent in epidural analgesia as compared with spinal anaesthesia (Odom[13]; Massey Dawkins[14]). This is not now held to be true, and hypotension is comparable in every way with that produced by a sub-arachnoid injection, when the height of analgesia is similar. An important factor in lowering blood pressure is the concomitant use of general anaesthesia. This greatly facilitates the onset of hypotension for two reasons. Firstly, general anaesthesia produces some degree of vasodilatation itself and depresses the compensatory mechanisms. Secondly, consciousness itself can have a marked pressor effect (Taylor and Page[15]). It is significant that all the writers who believed blood pressure was relatively unaffected in epidural block, did not use supplementary general anaesthesia, and frequently a dose of ephedrine was given to prevent a fall in blood pressure.

If hypotension ensues as a result of epidural blockade, immediate reversal by vasoconstrictors is not now carried out if it is thought that the patient can tolerate it well, and if the operation warrants its use. The physiological effects of hypotension must, therefore be considered.

Effects /

Effects of Hypotension on:

Brain.- In spite of a considerable reduction in cardiac output, the cerebral blood supply may be relatively unaffected, provided the patient is horizontal or in the head-down position, and the cerebral venous oxygen content is not then reduced (James, Coulter and Saunders[16]). Other workers who demonstrated a reduction of cerebral blood flow found a normal oxygen uptake and no cerebral hypoxia (Morris, Moyer, Snyder and Haynes[17]). With a reduction of systolic blood pressure, there is a decrease in the cerebro-spinal fluid pressure and the brain tends to shrink. These changes are useful when neurosurgery is performed under hypotension.

Posture is an important factor in modifying the cerebral circulation of the hypotensive patient. The head-up position is particularly liable to cause a marked decrease in cerebral blood flow and great care is required if this position is adopted. The state of the patient's cerebral vessels also affects the safety of hypotension. It is obvious that advanced cerebral arteriosclerosis makes hypotension more liable to produce serious hypoxia of the brain and, if hypotension is used in the elderly, it is prudent to maintain the systolic blood pressure at a somewhat higher level than in younger subjects.

It is not possible to predict in any given patient what level of systolic blood pressure is safe or unsafe. As a marked reduction in bleeding occurs with the systolic pressure /

pressure between 60 and 80 mm.Hg., it is generally considered that falls below 50 mm.Hg. should be avoided.

It has been suggested by Nilsson[18] that transient and otherwise unsuspected cerebral damage may be caused by hypotension. Applying the "flicker-fusion" technique of Berg[19], most of his patients in whom an impairment of cerebral function was detected by this method, had been operated upon in the reverse Trendelenburg position.

Heart.- The supply of blood to the heart depends largely upon the level of systolic blood pressure and, as this is lowered, so the coronary circulation decreases. At the same time the peripheral resistance also decreases, and the heart has less work to do than normally. Cardiac output is reduced as the venous return falls, and the heart has to contract against a decreasing resistance. Myocardial insufficiency occurring after coronary infarction or in angina of effort, is usually considered a contra-indication to hypotension. It may be, however, that a reduction in cardiac work as occurs in hypotension can benefit some of these cases.

Lungs.- Respiration is not depressed during epidural analgesia, and it remains quiet and regular. The autonomic blockade, however, produces changes in the lungs. The bronchi have both a sympathetic and a parasympathetic innervation, the former causing broncho-dilation and the latter broncho-constriction. Under high epidural blockade the sympathetic nerves are paralysed, leaving the parasympathetic pathway (through the vagus nerve) intact. It

would /

would be expected, in this case, that broncho-constriction would occur and the method be unsuitable for asthmatic or emphysematous subjects. The reverse, however, is true, for these types of patients do very well and, in fact, their vital capacity is increased. Using three methods of induced hypotension in conscious patients (spinal, epidural and ganglionic block) Bromage[20] found an increase of up to 30 per cent. in vital capacity, the effect being most marked in cases of cardio-pulmonary insufficiency. The explanation of this probably lies in the vascular hypotension that ensues whenever the blockade is high enough to leave the vagus nerve unopposed. This hypotension has two main effects. Firstly, reduction of the pulmonary arterial pressure relieves pulmonary congestion, if present. Secondly, it has been shown that hypotension excites a baroreceptor reflex whereby normal vagal broncho-constrictor tone is inhibited (Daly and Schweitzer[21]).

Kidneys.- During hypotension the renal blood flow and the filtration pressure are reduced. Urine secretion diminishes and finally ceases as the systolic pressure approaches the filtration pressure (Mendelsohn and Szutu[22]). Hypoxia of the renal tissues does not occur, however, and when the blood pressure is restored to normal, urine production is resumed.

Liver.- It has been observed that the liver becomes darker in colour and firmer in consistence under hypotension, and doubts were at one time expressed as to its ability to withstand /

withstand severe hypotension (Bromage)[23]. These fears are now considered to be unfounded.

In summary, it may be said that almost all cases of serious complications occurring under hypotensive anaesthesia are due to cerebral or myocardial hypoxia.

Factors affecting the safety of hypotension:

The safety of hypotension depends upon many variables. Some, such as the state of the patient's vessels, are outwith the control of the anaesthetist. The factors outlined below directly concern the conduct of the anaesthetic procedure.

(a) Degree of hypotension.- In general it may be said that the lower the systolic blood pressure the greater is the risk of serious complication. As the object of hypotension is to reduce bleeding, it would appear logical to produce, in any given patient, that level of hypotension which produces satisfactory operating conditions in so far as preventing blood loss is concerned. If a dry field is produced at a systolic blood pressure of 80 mm.Hg., there would appear to be little purpose in reducing the pressure to 60 mm.Hg. The effective hypotensive level for any particular patient cannot be predicted, and may vary from 55 to 85 mm.Hg.

It is not always easy to achieve a preselected systolic pressure. Often a low pressure is produced and the anaesthetist may be in doubt as to whether it should be raised by means of a vasopressor. Epidural analgesia offers advantages /

advantages in this respect over ganglionic blockade. With the former the pressure falls slowly, taking 20-30 minutes to reach its lowest level, whereas hypotensive drugs act rapidly. It is easier, therefore, to stop the fall at a level that gives satisfactory hypotensive operating conditions. Under sympathetic blockade also a patient is much more susceptible to the action of a vasopressor agent, and by using small amounts intravenously, it is possible, with experience, to raise a low pressure without eliminating the hypotension altogether.

If, in spite of hypotension, bleeding occurs during operation, a quite modest loss of blood can produce a further fall in blood pressure. Adequate blood replacement is therefore necessary in operations performed under hypotensive anaesthesia.

(b) Posture.- Posture modifies hypotension in two ways. Firstly, the blood pressure at any given part of the body depends on the level of the part relative to the heart (Enderby[24]). If possible, it is advantageous to have the site of operation at the highest possible level. If, however, the head is raised above the level of the heart, the pressure in the cerebral arteries may fall below the critical pressure and cerebral hypoxia will ensue.

Secondly, pooling of the blood may be augmented by posture, especially if the legs are lowered, and in consequence venous return is further reduced.

In patients tilted into the reverse Trendelenburg position, /

position, not only is the cerebral blood pressure lowered, but the cardiac output is further reduced by pooling in the dependent legs. This combination of factors may be very dangerous.

(c) Respiration.- During anaesthesia, respiration may be spontaneous, assisted or controlled. In spontaneous breathing, intrathoracic pressure is entirely negative, in assisted respiration it is partly negative and partly positive, and in controlled respiration it is positive. The normal negative intrathoracic pressure has an important part to play in cardiovascular function, for it is one of the main factors responsible for venous return, especially in anaesthetised patients when muscular activity is absent.

There is considerable evidence that positive pressure respiration reduces cardiac output by impeding venous return, especially in the presence of circulatory inadequacy such as low circulating blood volume due to shock (Mahoney, Elam, Handford, Balla, Eastwood and Brown[25]; Beecher, Bennett and Bassett[26]; Carr and Essex[27]). Hubay, Waltz, Brecher, Praglin and Hingson[28], using a bristle flow-meter inserted into the superior vena cava of dogs, have measured the venous flow under varying conditions. They found that during the application of a positive intrapulmonary pressure of up to 20 cm. of water, normovolaemic dogs suffered a 10 per cent. reduction in venous blood flow, compared with the flow at atmospheric pressure. By adding a negative phase to the respiratory cycle during expiration (and thus making artificial respiration as close as possible to normal respiration /

respiration) an increase of 33 per cent. in venous blood flow was obtained. The inference of this experiment is that positive pressure respiration causes at least 33 per cent. reduction in venous return (and therefore in cardiac output) compared with spontaneous respiration. The effect of this in normo-volaemic cases is of little consequence, if not prolonged beyond a few hours. The same experiment performed on dogs made hypotensive and hypo-volaemic by bleeding, showed that the negative phase increased venous blood flow by 100 per cent. All these experiments were performed with the chest closed. Opening the thorax reduces to negligible proportions the increase in flow from the application of a negative phase.

Though hypotension from bleeding cannot be strictly compared to purposely induced hypotension, it would be logical to assume that positive pressure respiration will cause a considerable reduction in cardiac output under both conditions. It follows, that, under controlled hypotension when the cardiac output is already reduced, positive pressure respiration would add a further burden to the circulatory dynamics. It is well known that controlled respiration, when instituted in the hypotensive patient, causes a further fall in blood pressure (Evans and Gray[29]), and this is even used by some anaesthetists to augment hypotension. The fallacy of the procedure is that it is by no means certain that a given level of blood pressure represents the same cardiac output under different conditions. On theoretical grounds /

grounds it is likely that a patient, breathing spontaneously, with a systolic pressure of 60 mm.Hg., has a considerably better cardiac output than one whose respirations are controlled and who has the same systolic pressure. The advantage of epidural, or spinal analgesia, in this respect is obvious, for respiration is in no way decreased.

It must be remembered that, under anaesthetic conditions, the only measurements of cardiovascular function that can be carried out conveniently, are the blood pressure and pulse rate. Clinically the cardiac output, which is probably much more important than the blood pressure, can only be inferred from the pressure. In doing this, the pulse pressure is of value as it is obvious that a pressure of 60/50 mm.Hg. represents a smaller cardiac output than 60/30 mm.Hg. In support of the foregoing contention, that spinal and epidural analgesia with spontaneous respiration produce hypotension with a better cardiac output than ganglionic blockade with controlled respiration, it is a frequent observation that the pulse pressure is much greater in the former than in the latter, for any given level of systolic blood pressure. This, incidentally, offers two advantages to the anaesthetist in the management of epidural or spinal anaesthesia; the pulse can readily be felt, and the blood pressure is easily determined by sphygmomanometry.

(d) Concomitant general anaesthesia.- General anaesthesia facilitates the production of hypotension. Moreover, deepening the anaesthesia causes a further drop in blood /

blood pressure. This is due to the fact that general anaesthesia causes a degree of vasodilatation in proportion to the depth of the anaesthesia which correspondingly restricts the patient's compensatory mechanisms.

Anaesthesia must not be so deep as to cause respiratory depression, since this would lead to carbon dioxide retention, and a further fall in blood pressure. Hypercarbia has a dual action on blood vessels. It has a stimulant effect on the vasomotor centres leading to vasoconstriction, and a direct dilating action on the vessels themselves. Normally, the former predominates and a rise in blood pressure occurs. Under sympathetic blockade, however, this central action is blocked, and vasodilation being unopposed, the pressure falls.

In the course of anaesthesia, inadvertent coughing and straining can cause an excessive rise in intrathoracic pressure, and consequently the cardiac output may fall almost to zero. Hypoxia even though transient, will not be tolerated by the hypotensive patient, and anaesthetic skill is a necessity before hypotension can be used safely.

(B) Gastro-Intestinal Tract

When the sympathetic supply to the gastro-intestinal tract is blocked, the vagus can act unopposed. This causes active peristalsis and a contracted gut, which considerably facilitates surgical exposure within the abdomen.

As the vagus nerve is unaffected by epidural blockade, reflexes /

reflexes may be activated by traction on the mesentery or viscera, especially in the upper abdomen. Nausea and vomiting may then ensue, especially if no concomitant general anaesthesia is administered.

References

(1) Fulton, J.F. (1955). "Textbook of Physiology," p. 53. Philadelphia: W.B. Saunders & Co.

(2) Sollman, T. (1957). "A Manual of Pharmacology," p. 324. Philadelphia: W.B. Saunders & Co.

(3) Muir, A.R. (1958). Personal communication.

(4) Evans, F.T. and Gray, T.C. (1958). "Modern Trends in Anaesthesia," p. 65. London: Butterworth & Co.

(5) Lee, J.A. (1958). Personal communication.

(6) Pugh, L.G.C. and Wyndham, C.L. (1950). Clin. Sci., 9, 189.

(7) White, J.C. and Smithwick, R.H. (1944). "The Autonomic Nervous System," p. 47. London: Henry Kempton.

(8) Wiggers, C.J. (1949). "Physiology in Health and Disease," p. 625. Philadelphia: Lea & Febiger.

(9) Griffiths, H.W.C. and Gillies, J. (1955). Personal communication.

(10) Greene, N.M. (1958). "Physiology of Spinal Anaesthesia," p. 66. Baltimore: Williams & Wilkins Co.

(11) Mitchell, G.A.G. (1948). J. roy. Coll. Surg. Edinb., 4, 1.

(12) Wiggers, C.J. (1952). "Circulatory Dynamics," p. 29. New York: Grune & Stratton.

(13) /

(13) Odom, C.M. (1936). Amer. J. Surg., 34, 547.

(14) Massey Dawkins, G.J. (1945). Proc. roy. Soc. Med., 38, 302.

(15) Taylor, R.D. and Page, I.H. (1952). Amer. J. Physiol., 170, 321.

(16) James, A., Coulter, R.L. and Saunders, J.W. (1953). Lancet, 1, 412.

(17) Morris, G.C., Moyer, J.H., Snyder, H.B. and Haynes, B.W. (1953). Ann. Surg., 138, 706.

(18) Nilsson, E. (1953). Brit. J. Anaesth., 25, 24.

(19) Berg, O. (1949). Acta psychiat. (Kbh.), Suppl. No. 58.

(20) Bromage, P.R. (1956). Anaesthesia, 11, 139.

(21) Daly, M. de B. and Schweitzer, A. (1951). J. Physiol., 113, 442.

(22) Mendelsohn, M.L. and Szutu, C. (1953). Amer. J. Physiol., 173, 355.

(23) Bromage, P.R. (1952). Lancet, 2, 10.

(24) Enderby, G.E.H. (1954). Lancet, 1, 185.

(25) Mahoney, J.V., Elam, J.O., Handford, S.W., Balla, G.A., Eastwood, D.W., Brown, E.S. and Ten Pas, R.H. (1953). J. Amer. med. Ass., 152, 212.

(26) Beecher, H.K., Bennett, H.S. and Bassett, D.L. (1943). Anesthesiology, 4, 612.

(27) Carr, D.T. and Essex, H.E. (1946). Amer. Heart J., 31, 53.

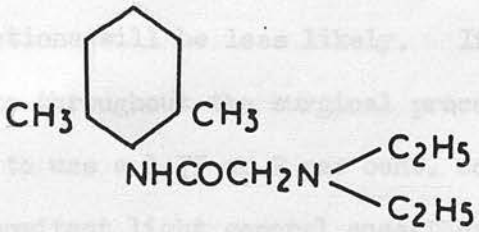
(28) Hubay, C.A., Waltz, R.C., Brecher, G.A., Praglin, J. and Hingson, R.A. (1954). Anesthesiology, 15, 445.

(29) Evans, F.T., and Gray, T.C. (1958). "Modern Trends in Anaesthesia," p. 238. Butterworth & Co., London.

IV. PHARMACOLOGY

A great many drugs have been employed to produce epidural analgesia; only those with which the writer has personal experience will be discussed.

LIGNOCAINE (w - diethylamino - 2 - 6 - dimethylacetanilide)



The introduction of lignocaine in 1948 (Gordh)[1] provided a considerable impetus to the use of epidural analgesia, for it proved to be a much more efficient agent for nerve block than any drug previously in common use.

It is a remarkably stable drug, being unaffected by autoclaving (even in solution), and resistant to the action of acids and alkalis. It is rapid in action, producing nerve block in 5-10 minutes. The effect lasts for 2-2½ hours. Compared with procaine, it acts more quickly, and is more certain in its effect.

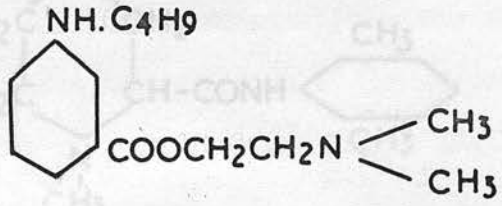
It is somewhat more potent than procaine and can be used as a surface anaesthetic. While toxic reactions similar to those seen with procaine can occur, it has a wide margin of safety and has been used intravenously as an adjuvant to general anaesthesia (de Clive-Lowe, Gray and North)[2].

For epidural analgesia it can be used in strengths varying from 1-2 per cent. The writer began by using a 1.25 per cent. solution, but has increased this to 1.5 per cent., /

cent., following a few cases in which the analgesia was not complete. The 2 per cent. solution will naturally produce a greater degree of motor block than the weaker solutions, but even so, it does not paralyse all the motor fibres. The advantage of using the weaker solutions is that toxic reactions will be less likely. If the patient is to be awake throughout the surgical procedure, it is probably better to use a 1.75 or 2 per cent. concentration. With concomitant light general anaesthesia, however, 1.5 per cent. has always proved adequate.

Lignocaine is supplied in three strengths for injection, 0.5 per cent., 1 per cent. and 2 per cent. A 1.25 per cent. solution is obtained from equal parts of 2 per cent. and 0.5 per cent. and 1.5 per cent. from equal parts of 2 per cent. and 1 per cent. As re-autoclaving does not cause deterioration of lignocaine, any unused solution can be re-sterilised for a future occasion.

AMETHOCAINE (p - butylaminobenzoyl dimethylaminoethanol)



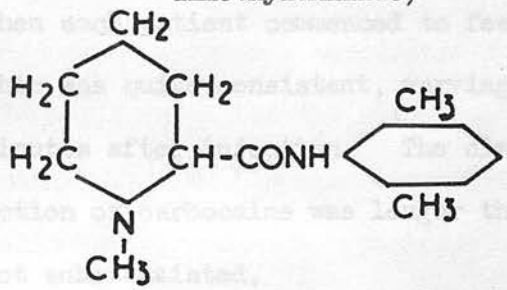
This drug has been used for many years and is satisfactory for blocking nerves. Unfortunately, it is only effective after 20-25 minutes, which is too long for the smooth running of an operating list. Its effect, however, lasts /

lasts for 3-3½ hours, which is rather longer than with lignocaine. It is quite useful, therefore, to use a mixture of lignocaine and amethocaine. The former gives a rapid onset allowing surgery to be commenced without delay, while the prolongation of analgesia by amethocaine allows lengthier operations to be performed, and gives a longer period of post-operative analgesia.

Amethocaine can be obtained in ampoules containing 100 mg. of the crystalline powder which can be autoclaved. This makes 50 ml. of 0.2 per cent. concentration, which is satisfactory for nerve block. It is dissolved in the required strength of lignocaine.

When using a combination of drugs in this manner, it must be remembered that their toxic effects may summate. Adrenaline, therefore, should be added to reduce the rate of absorption, and the injection should be made slowly.

CARBOCAINE (d - l - N - methyl - pipecolic acid 2 - 6 - dimethylanilide)



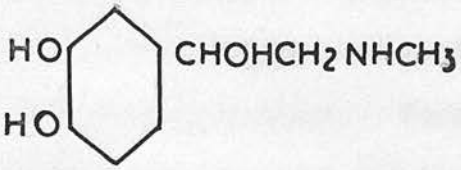
This local anaesthetic drug has recently been introduced from Sweden (Ekenstam, Egner, Ulfendahl, Dhuner and Oljelund).[3] It has all the advantages of lignocaine and its effect is said to last up to half an hour longer. It is also somewhat less acid in solution than lignocaine, a theoretical /

theoretical advantage for its use as a spinal anaesthetic. Though it has been used extensively in Sweden for the production of spinal anaesthesia, there are few reports on its efficacy in epidural analgesia. The writer has used carbocaine to produce epidural nerve block on 30 occasions, and in 1.25 per cent. solution to permit comparison with lignocaine. Though the speed of onset was not measured accurately, carbocaine did not seem to be any slower or quicker in action than lignocaine. Following the epidural injection, each patient was immediately anaesthetised with thiopentone followed by nitrous oxide. On average, 10 minutes elapsed between the completion of the epidural injection and the initial incision, and in no case was the onset of analgesia delayed beyond this time. In two of the 30 cases it was considered that analgesia was not so complete as expected, but this could have been prevented by using a stronger solution such as 1.5 per cent. or 2 per cent.

To examine the duration of analgesia, the time was noted when each patient commenced to feel pain post-operatively. This was quite consistent, varying from 115 minutes to 135 minutes after injection. The claim that the duration of action of carbocaine was longer than that of lignocaine was not substantiated.

In summary it may be said that carbocaine was indistinguishable from lignocaine in producing epidural analgesia. A 1.5 - 2 per cent. solution is recommended.

ADRENALINE



The addition of adrenaline to local anaesthetic solutions is a well established practice. By causing vaso-constriction in the tissues into which it is injected, it delays absorption of the analgesic drug into the blood stream. Toxic reactions are avoided and analgesia lasts longer than with solutions not containing adrenaline. This is of particular importance in epidural analgesia, for the epidural space is richly supplied with blood vessels and toxic reactions from absorption are a real hazard.

For local anaesthesia, adrenaline in a dilution of 1 in 80,000 to 1 in 250,000 is usually recommended. The total amount of adrenaline should not exceed 0.5 mg. (i.e. 0.5 ml. of 1 in 1,000). Unfortunately, adrenaline deteriorates on autoclaving though this effect has been overstressed in the past. After being autoclaved in ampoules, 1 in 1,000 adrenaline retains a considerable proportion of its potency (Thomas[4]). Neither the decomposition products nor the stabiliser commonly used (potassium metabisulphite) are neuro-toxic, and have been injected into the sub-arachnoid space without deleterious effect.

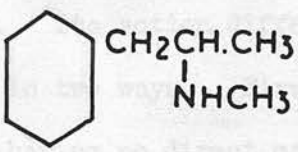
It has been said that the administration of adrenaline prior to induction of general anaesthesia is a dangerous procedure. This may be true if induction is carried out with /

with chloroform or cyclopropane. With thiopentone followed by nitrous oxide, however, serious side-effects from the use of adrenaline are unlikely to occur. Tachycardia and an increase in blood pressure may occasionally be seen but they are short-lived and no case of cardiac arrhythmia or other significant complication has been observed in the writer's series.

VASOPRESSORS

Vasopressors may have to be administered during epidural analgesia to counteract, or reverse, hypotension. It should be remembered that, in common with other forms of sympathetic blockade, epidural analgesia renders the patient more sensitive to the action of vasoconstrictors. Indications for the use of such drugs will be given when the management of cases undergoing epidural block is discussed.

(a) Methylamphetamine



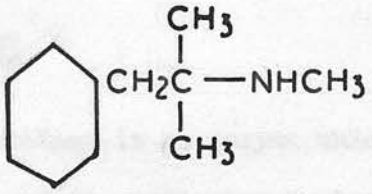
This drug has three main actions. Firstly, it causes peripheral vasoconstriction with a consequent increase in venous return. Secondly, it is a cardiac stimulant causing the heart to beat faster and more strongly. Thirdly, it is a cerebral stimulant, like its related analogue d-amphetamine. This last property can be a distinct disadvantage in a patient under light general anaesthesia, since it tends to rouse /

rouse the patient who may then cough and strain.

The actions of methylamphetamine are said to be the result of its interference with the action of the enzyme amine-oxidase. The latter substance is responsible for the break-down of adrenaline in the body, so that methylamphetamine, by inhibiting the activity of the adrenolytic enzyme, potentiates the action of endogenous adrenaline.

Methylamphetamine is administered intravenously or intramuscularly in doses varying from 1 to 30 mg. Given intravenously it acts within three minutes and its effect lasts 20 minutes. Intramuscularly its action persists for one hour.

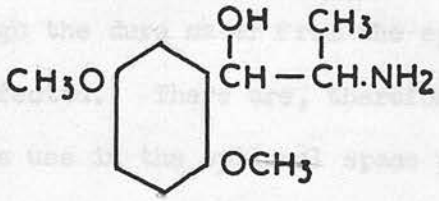
(b) Mephentermine



This substance is closely related chemically to methylamphetamine. Its action differs from that of methylamphetamine in two ways. Firstly, it is a pure vasoconstrictor having no direct cardiac effect and thus it resembles noradrenaline, except that its action is much more prolonged. Secondly, it is not a cerebral stimulant. It is given in similar doses and by similar routes to methylamphetamine.

(c) Methoxamine /

(c) Methoxamine



This drug also is solely a vasoconstrictor and is, therefore, of value in the management of cases of epidural block combined with light general anaesthesia. Restoration of blood pressure is prompt and, as it has no analeptic effect, smooth anaesthesia is uninterrupted. From clinical experience, methoxamine appears to be more potent, and quicker acting, than methylamphetamine and in the writer's view it is the drug of choice for reversing hypotension.

HYALURONIDASE

Hyaluronidase is an enzyme which hydrolyses mucopolysaccharides and in particular hyaluronic acid, which is an essential component of the "ground substance" of tissues. Softening of this "ground substance" facilitates the extracellular spread and absorption of fluids.

The use of hyaluronidase with local anaesthetics has been advocated for two reasons. Firstly, it acts as a spreading agent and permits a reduction in the amount of local anaesthetic drug used. Secondly, it accelerates penetration of the nerve coverings and nerve block is established more readily. It should be remembered that hyaluronidase does not aid the penetration of drugs through fascial /

fascial barriers (Moore[5]) and passage of the analgesic through the dura mater from the epidural space will not then be affected. There are, therefore, no contra-indications to its use in the epidural space provided adrenaline is added to the solutions to counteract rapid absorption into the blood stream.

One disadvantage of epidural analgesia is the length of time taken to produce an effective blockade. If hyaluronidase could expedite the onset of analgesia, this limiting factor would be mitigated. To test the possibility of doing so the author (Scott[6]) conducted a series of 40 sacral epidural and 60 lumbar epidural blocks, in half of which lignocaine was given and in the remainder lignocaine and hyaluronidase. Adrenaline was added in each case. The expectation that the effect of the injection would be more widespread if hyaluronidase were added, was not fulfilled. On considering the anatomy of the epidural space this is understandable. The space contains only fat and blood vessels bound in loose areolar tissue. Hyaluronidase acts as a spreading agent by making cell membranes more permeable and its effect is best seen in structures such as muscle, where the cells are closely packed. Where tissues are less dense as in the epidural space (excluding the sacral canal) little resistance to injected solutions is offered, and the action of hyaluronidase would be insignificant. The sacral canal, as we have seen, is much more tightly packed with fat, and in the sacrum the hyaluronidase would have more opportunity /

opportunity to exert its action and be effective.

The rate of onset of analgesia, however, was shown to be more rapid in those cases in which hyaluronidase was added, this being more apparent in caudal blocks than in lumbar epidural blocks. The effect was not sufficient, however, to warrant the routine use of hyaluronidase in lumbar epidural analgesia, but in caudal blocks, it may have a place. Due to its almost complete non-toxicity, the dosage of hyaluronidase is relatively unimportant. It is supplied as a freeze-dried powder in ampoules, and 1,000 units (100 mg.) are added to each 20 ml. of anaesthetic solution.

The significance of this increase in the rapidity of onset of analgesia, in relation to the site of action of epidurally placed local anaesthetics, has been discussed (see p. 30).

References

(1) Gordh, T. (1948). Svenska Läk.-Tidn., 45, 117.

(2) de Clive-Lowe, S.E., Gray, P.W.S. and North, J. (1954). Anaesthesia, 9, 96.

(3) Ekenstam, B.A., Egner, B., Ulfendahl, L.R., Dhuner, K.G. and Oljelund, O. (1956). Brit. J. Anaesth., 28, 503.

(4) Thomas, D.V. (1956). Anesthesiology, 17, 752.

(5) Moore, D.C. (1951). Anesthesiology, 12, 611.

(6) Scott, D.B. (1956). Brit. J. Anaesth., 28, 187.

V. METHODS AND MANAGEMENT OF EPIDURAL ANALGESIA

LUMBAR EPIDURAL ANALGESIA

Identification of Epidural Space

Basically there are two ways in which the epidural space can be located. The first depends upon the presence of negative pressure within the space, and the second upon loss of resistance to injection that occurs as the exploring needle passes the ligamentum flavum and enters the epidural space.

The negative pressure in the epidural space does not appear to serve any physiological purpose. Janzen[1], its discoverer, believed from his observations that it was due to depression, or dimpling, of the dura by the advancing needle. He maintained it was an artefact and produced only in the locality of the needle. Later workers, however, have produced evidence that the negative pressure was present throughout the epidural space, and was due to transmission through the intervertebral foramina of intrathoracic negative pressure (Macintosh and Mushin[2]; Bryce-Smith[3]). Modern opinion inclines to the view that, although this can produce a slight negative pressure, especially in the thoracic region, the main cause is the local dimpling of the dura mater.

Whatever the cause, it must be noted that pressure in the lumbar epidural space is negative in not more than 80 per cent. of cases, and only in the thoracic region does it approach 100 per cent. Methods of localising the space using /

using the negative pressure are therefore not suitable for routine use in the lumbar region. They are to be preferred in high thoracic epidural blocks, because at that level the ligamentum flavum is thin, making the loss of resistance test less effective. The methods using this principle are:-

(a) Hanging drop sign.- In this technique, a drop of water is applied to the hub of the exploring needle after it is engaged in the interspinous ligament. When the needle has passed through the ligamentum flavum the drop is sucked into the epidural space.

(b) Odom's[4] indicator.- This is a small piece of glass capillary tubing, ground so that it fits on to the hub of the spinal needle. A drop of liquid is introduced into the capillary, and any negative pressure encountered at the needle tip causes the drop to move towards, and into, the needle hub. It is said that this indicator is very sensitive provided it fits accurately.

(c) Macintosh[5] balloon indicator.- A small balloon is attached to an adaptor which fits the spinal needle. When the needle is engaged in the ligaments, 2 ml. of air are injected into the balloon through its thick walled neck. As the needle is advanced into the epidural space the bag deflates.

The loss of resistance sign has been used in a variety of ways.

(a) Without any mechanical aid.- This is the simplest and /

and probably the best method. It depends upon the fact that if a charged syringe is attached to the spinal needle, and a constant pressure exerted upon the plunger, the resistance to injection encountered in the interspinous ligaments disappears as soon as the ligamentum flavum is traversed. Fluid leaving the needle tip pushes the dura away and reduces the likelihood of dural puncture. The use of a wide-bore short bevelled needle greatly facilitates this method as changes in resistance are much more easily appreciated.

(b) Spring-loaded syringe (Brunner and Ilke[6]).- By attaching a spring to the plunger of a 2 ml. syringe, pressure can be constantly applied without the aid of the operator. The syringe discharges itself automatically as the epidural space is entered. The disadvantage of this syringe is that it cannot differentiate between the epidural space and the laxity of the interspinous ligaments that can occur in some elderly patients. In such cases it is very easy to inject 2 ml. of fluid without much resistance developing. Similarly, if the needle is directed laterally and enters the sacro-spinalis muscle, easy injection is possible.

(c) Macintosh extradural space indicator[7].- In this device, a blunt stilette, attached to a spring, is within the spinal needle, and protrudes through the needle hub. As the epidural space is entered, the loss of resistance at the needle tip allows the spring to advance the stilette.

Again /

Again this only works well in patients with well-marked ligaments.

All mechanical aids have the inherent disadvantage that they replace the operator's intelligent touch by a piece of unintelligent machinery.

To achieve success consistently, it is very important for the anaesthetist to find a technique that suits him and to persevere with it. In the writer's experience, the best method is the simple loss of resistance technique. One finds, with continuing practice, that the loss of resistance becomes merely a confirmatory sign that the epidural space has been reached. Appreciation of the exact position of the needle point at any given time is more important, and once the ligamentum flavum has been reached the procedure becomes simple. Such appreciation of position is not possible with mechanical aids.

Equipment

As all the apparatus and drugs used must be autoclaved, it is preferable to have epidural packets or drums, each containing enough equipment for one case. These can be rapidly re-packed and sterilised. Each drum should contain the following:-

(a) One 20 ml. Luer sized syringe (all-glass).

(b) Two Tuohy needles. This needle was designed for the insertion of plastic catheters into the sub-arachnoid space. Its curved tip allows the catheter to emerge at a right /

right angle to the needle shank. With an ordinary needle the catheter would merely impinge on the dura, and be unable to turn the corner and advance up or down the sub-arachnoid space. The Tuohy needle is also especially suitable for epidural analgesia. It has a wide-bore and is of strong construction, allowing easy re-direction while still within the interspinous ligament. The curved tip ensures that only very little bevel has to enter the space before the loss of resistance test becomes positive, thus minimising the danger of dural puncture.

If these needles are unobtainable, a wide-bore, short bevelled needle can be used.

Tuohy needles are all Luer fitting.

(c) Two wide-bore aspirating needles for drawing up solution.

(d) Two fine intradermal needles for skin infiltration.

(e) Ampoules or bottles of lignocaine. Though lignocaine can be obtained in 25 ml. ampoules of 1.5 per cent., the writer prefers to use rubber capped bottles of 0.5 per cent. and 2 per cent. In this way varying strengths of lignocaine can be made up, and as unused amounts of the drug can be re-autoclaved, it is more economical. To avoid mistaking the strength of solution in the bottles (labels being very likely to come off in the sterilisation), 20 ml. bottles of 0.5 per cent. and 50 ml. bottles of 2 per cent. are used.

(f) One ampoule of 100 mg. of amethocaine.

(g) /

(g) One ampoule of $\frac{1}{2}$ ml. 1/1000 adrenaline.

(h) Ampoule files.

(i) Towels.

(j) Swabs and swab-holder.

(k) Galley pot.

The writer prefers to have all these items in a roll which can be easily lifted from the sterilising drum. The operator can then easily "set up" his own trolley.

Preparation of Patient

In addition to the normal pre-operative examination the anaesthetist should take careful note of the lumbar spine. Skin infection near the proposed site of epidural puncture contra-indicates the method. The ease with which the lumbar vertebral spines can be palpated and the range of movements of the vertebrae are determined. Gross osseous deformity rules out epidural puncture, but mere obesity and senile spinal rigidity, though they may make the procedure more difficult, are not contra-indications. No patient in this series has been refused epidural analgesia on such grounds.

The patient is told briefly that the anaesthetic will consist of an injection in the back, followed a few minutes later by another in the arm which will cause sleep to ensue. The great majority of people require no further explanation, but this is given if directly requested.

For premedication the writer uses a combination of papaveretum and atropine, in doses appropriate to the patient, /

patient, given subcutaneously one hour before operation.

Epidural Puncture

The anaesthetist must observe full aseptic precautions during epidural puncture. "Scrubbing-up" and the donning of sterile rubber gloves are essential.

As epidural analgesia is time-consuming, it is necessary for the anaesthetist to work swiftly and to avoid unnecessary delays. Minutes can be saved by an analysis of the manoeuvres required, and avoidance of all unnecessary movements and actions.

The patient is turned into the lateral position and asked to flex the back as much as possible. The skin is cleaned with 2.5 per cent. iodine, and sterile towels are draped round the patient. A suitable interspinous space is chosen (usually the one between the second and third lumbar vertebrae) and the skin over it is infiltrated using the 20 ml. syringe fully charged with 1.5 per cent. lignocaine. Infiltration of the ligaments is quite unnecessary, as they are insensitive to painful stimuli. Moreover, the insertion of a short fine needle into the ligaments incurs the risk of breakage.

A Tuohy needle is now inserted into the interspinous space at right angles to the spine, in the sagittal plane, until it has passed the supraspinous ligament and is well engaged in the interspinous ligament. The opening at the tip of the needle can either be pointed caudad or cephalad, depending on whether a low or a high block is required. The stilette /

stilette is removed and the fully loaded 20 ml. syringe is attached to the needle hub. On attempting to depress the plunger, resistance to injection, offered by the ligament, will be easily discernible. The left hand now steadies the needle and the right hand holds the syringe, the thumb keeping a constant pressure upon the plunger. Syringe and needle are advanced together until the increased density of the ligamentum flavum is felt. As this ligament is pierced, the plunger moves forward and injection becomes quite easy. Epidural tap has now been performed.

If the line of entry is wrong, bone will obstruct the forward movement of the needle. Invariably it is the vertebral lamina which is thus impinged upon, and note is taken of the depth of the needle, for, when the alignment is corrected, the ligamentum flavum will be found at that depth. The thickness and strength of the Tuohy needle allows for easy re-direction within the interspinous ligament. The needle point can then be slipped above or below the lamina and into the ligamentum flavum.

Test Dose

Once the ligamentum flavum has been passed and injection is rendered easy, the needle point is in one of two places: the epidural space or the sub-arachnoid space. These must, of course, be differentiated. The syringe is at once disconnected and any efflux of fluid from the needle is noted. The continuous dripping of cerebro-spinal fluid should be quite obvious if the sub-arachnoid space /

space has been pierced. Quite often some of the lignocaine will drip back from the epidural space and is identified by its temperature (felt on the back of the wrist) which will be cold. Cerebro-spinal fluid on the other hand would be warm.

If there is no efflux of cerebro-spinal fluid, the possibility of sub-arachnoid puncture is not completely ruled out, for the needle point may be only partially within the sub-arachnoid space or a nerve root may obstruct it. Consequently, a test dose 5-8 ml. of 1.5 per cent. lignocaine is now given. Injected into the sub-arachnoid space this would produce anaesthesia up to the seventh to tenth thoracic segment within five minutes. If the needle is correctly placed in the epidural space such a dose would cause little or no anaesthesia in five minutes.

The patient is reassured that the needling is now finished and is asked to remain quite still on the side for five minutes. These five minutes can be used by the anaesthetist for re-charging the syringe with lignocaine, adding adrenaline (0.25 ml. to 20 ml. of local anaesthetic), and amethocaine if desired (40 mg. to 20 ml. of lignocaine). The writer prefers to add amethocaine in those cases undergoing abdominal surgery (when the post-operative analgesia is most useful), or for operations expected to last more than two hours. Patients over 60 years old are usually not given amethocaine (unless the surgery is to be prolonged) as it is desirable that they regain vasomotor control as soon as /

as possible post-operatively.

As soon as five minutes have elapsed, the patient is tested to ascertain whether spinal analgesia is present. The writer asks two questions: (a) "Can you move your toes and feet?" and (b) "Your legs don't feel numb, do they?" These questions are designed to obtain information as to motor and sensory paralysis. The second question is purposely phrased to avoid suggesting to the patient that the legs should feel numb. If a sub-arachnoid injection has been made, the patient will have no doubt at all that numbness is present. In one case in the writer's series where the test dose was injected intrathecally, the patient was quite numb and analgesic up to the umbilicus, but could still move the toes, so reliance should not be made only on motor paralysis. If the answers to the questions are at all equivocal, the patient can be further tested by pricking the legs with a needle, a procedure that has only been necessary in 3 out of over 400 cases.

If no signs of spinal analgesia have appeared in five minutes, the anaesthetist can be reasonably sure that his needle is in the epidural space and can proceed with the main injection.

The use of a test dose has been challenged by Bromage[8] on the following grounds:-

(a) It is time consuming. Five minutes can appear to be considerable time to wait, but if it is used by the anaesthetist to add adrenaline and amethocaine it is not all lost. /

lost. Minutes can be saved by the elimination of all unpurposeful actions, and the time spent on a test dose will not then be such a burden.

(b) Five minutes is inadequate for the development of sub-arachnoid block and the test dose may therefore be misleading. This opinion was made when cinchocaine was the drug commonly used for epidural analgesia. Lignocaine is much more rapid in its action and analgesia will certainly be apparent in five minutes if injected intrathecally. Moreover, after considerable experience with cinchocaine in spinal anaesthesia, the writer seriously doubts whether five minutes is insufficient even when that drug is used.

(c) The patient must be awake during the procedure, if a test dose is used. This is little or no disadvantage. Patients must, of course, be properly handled to get maximum co-operation. Extremely nervous subjects or actual psychoneurotic or psychotic patients are best excluded from the procedure, but these cases amount to only a minute fraction of all patients. Methods have been devised for using a test dose in the unconscious patient. A drop in blood pressure and absent knee jerks are said to be positive signs of spinal anaesthesia, but such information may be equivocal under general anaesthesia. A test dose under these circumstances is best avoided.

The most convincing evidence on the efficiency of the test dose comes from Bonica, Backup, Anderson, Hadfield, Crepps and Monk.[9] In a series of 3,637 cases of epidural analgesia, spinal puncture was inadvertently made in 35 patients. /

patients. In 16 of these, efflux of cerebro-spinal fluid made the diagnosis obvious at once. In 17 of the remaining 19 cases, however, the true position of the needle only became apparent after the test dose produced spinal analgesia. Seventeen cases (approximately 1 in 200) of inadvertent total spinal analgesia were, therefore, prevented.

In the writer's own cases, the test dose has been of use in one case, mentioned above. No case of total spinal block has occurred when a test dose was used. One case in which a total spinal may have been produced was on one of the rare occasions when the test dose was omitted. This case will be discussed more fully later.

While for the great majority of cases the test dose is considered essential, there are occasions when it may be dispensed with, provided the anaesthetist has acquired some skill at the procedure, e.g. therapeutic block in a case of eclampsia when tranquil conditions may not be obtainable for very long.

Every anaesthetist must be prepared to deal appropriately with an accidental intrathecal injection should it occur (see p. 99), but it is also prudent to avoid such an occurrence if possible.

Injection of Local Anaesthetic

The main dose of local anaesthetic, having been prepared during the test dose period, is now injected.

There /

There is a temptation to inject quickly and get the procedure over. This should be resisted, otherwise pressure builds up quickly in the space and increased absorption of the local anaesthetic into the blood stream may lead to toxic reaction to the drug. In addition, rapid injection may cause the solution to be spread too far and too thinly, leading to inadequate analgesia.

The writer injects at the rate of 1 ml. every 3 seconds and a rate of more than 1 ml. every 2 seconds should not be used. No case of toxic reaction has been seen. Bromage[10] reported two cases of severe toxic reaction after injecting at a rate of only 0.5 ml. per second, but the total dosage in these cases was relatively high.

During the injection, the operator should watch carefully the patient's respiration. The first sign that a massive sub-arachnoid injection has been made will be apnoea, and the injection must be stopped immediately.

Some workers have described patients who experience sensations as of cold water running down their legs, parasthesiae and dizziness during the injection. This may be so, but the writer has never had a patient who voluntarily offered any such information. The whole procedure of epidural analgesia is uniformly painless provided the operator manages his patients properly, and is not clumsy in executing the needling and injection.

Dosage.- One of the advantages of spinal anaesthesia over epidural analgesia, is the greater accuracy with which the /

the height a given dose will spread can be predicted. This is because the sub-arachnoid space is enclosed, whereas the epidural space is not. Local anaesthetic can escape through every intervertebral foramen and the amount that escapes varies from patient to patient.

To overcome this problem some authorities (Bonica et al.[9]; Foldes, Colavincenzo and Birch[11]) prefer to make their epidural puncture as near the centre of the required zone of analgesia as possible. In this way the dosage can be kept to a minimum, 10-20 ml. sufficing for almost any type of surgery. While this may be the best way of ensuring adequate analgesia with the minimum of local anaesthetic, it suffers from the considerable disadvantage that a large proportion of cases will need epidural puncture in the thoracic spine. This is considerably more difficult than lumbar epidural puncture, due to the angulation of the thoracic vertebral spinous processes and the decreasing thickness of the ligamenta flava higher up the spinal column. As a consequence, failure will be more common, and, should the dura be punctured, there is a risk of direct trauma to the spinal cord. In their review of 3,537 cases of epidural block, Bonica et al.[9], who always tried to perform the epidural puncture at the middle of the required zone of analgesia, had a failure rate of 6 per cent. (varying from 3 to 17.5 per cent. among anaesthetists, according to experience). This undoubtedly could have been much less if the lumbar route had been used exclusively. The

advantage /

advantage of keeping the dose to a minimum is that toxic reactions should be reduced. However, Bonica et al. noted generalised toxic manifestations in 116 cases (3.2 per cent.) 8 of whom (0.2 per cent.) had convulsions, so that it would appear that the speed of injection is more important than the total amount injected, for, using much larger doses, the writer has not seen toxic reactions in over 400 cases of lumbar epidural analgesia. No doubt the use of concomitant general anaesthesia, which was not used routinely by Bonica and his co-workers, has also helped to keep reactions to a minimum.

Another method of making the injection at the centre of the required zone of analgesia, but without using thoracic epidural puncture, is by using a plastic catheter. Through a Tuohy needle inserted through a lumbar intervertebral space, a nylon catheter can easily be inserted into the epidural space and directed in a cephalic direction. The problem now is to push it upwards to the required level which may be several inches above the tip of the needle. This procedure can cause considerable damage to the numerous delicate blood vessels within the epidural space and in the presence of such damage, toxic reactions can readily occur.

Moreover, it is not easy to be sure that the catheter will proceed in the right direction, for it can be deflected and curl up, and has even been known to leave the epidural space through an intervertebral foramen. Unless radiography is used, the position of the catheter tip cannot be known accurately, /

accurately, and this would not be practical for routine anaesthesia.

By trial and error, the writer quickly decided that the only rational approach to the problem is to inject a quantity which is likely to produce analgesia several segments higher than the operation strictly requires. Toxic reactions are avoided by slow injection and though hypotension is more commonly produced, this is considered to be an advantage. If the patient's condition contra-indicates hypotension (and this is an uncommon occurrence) it can be prevented or quickly reversed with vasopressors.

Using this very rough method of dose calculation it is seldom necessary to use more than 30 ml. or less than 15 ml. of lignocaine. Thirty millilitres is adequate to anaesthetise to the seventh thoracic segment and most abdominal operations can be performed with such a dose. It is well known that elderly patients require less local anaesthetic than younger patients because the intervertebral foramina tend to close with age and thus prevent escape of the injected solution into the paravertebral space. Account is taken of this when judging the dose. If only unilateral anaesthesia is required, e.g. for nephrectomy or herniorrhaphy, a smaller dose can be used if the patient is kept in the lateral position for some minutes after the injection, with the operative side down. These dosages refer to the total amount injected and thus include the test dose.

Catheterisation /

Catheterisation Technique

Single dose injection enables the anaesthetist to obtain analgesia for 1-3 hours depending upon the agent used. Occasionally, however, a longer period of analgesia will be required for certain operations, or where the epidural block is induced for therapeutic purposes. To achieve this the anaesthetist can introduce a nylon catheter, which can be autoclaved without deterioration, into the epidural space. The catheter is introduced easily through a Tuohy needle, and advanced not more than 2 inches past the needle tip to avoid damaging blood vessels. The needle is then withdrawn. Injection of the test dose and main dose proceeds as before. The catheter is led round the patient so that further injections can be made from the head of the operating table. At the first sign of the analgesia wearing off, further local anaesthetic is injected through the catheter. Epidural blocks have been continued for several days by this method.

CAUDAL EPIDURAL ANALGESIA

Preparation of Patient

The patient is prepared in the same way as for lumbar epidural analgesia. The anaesthetist should look carefully for the external landmarks of the sacrum, especially the sacral cornuae which mark the position of the sacral hiatus. The presence of any bony abnormality such as non-fusion of the /

the laminae forming a very large sacral hiatus should be noted. Obesity is a much greater impediment to successful epidural puncture by the caudal route than with the lumbar route, as the hiatus must be clearly felt for success. It is not uncommon to find a pad of fat over the sacrum even though the patient is not particularly obese.

On reaching the anaesthetic room, the patient is turned into the prone position and a small pillow placed under the pubis. This makes identification of the sacral hiatus easier.

The skin over the buttocks and sacrum is painted with iodine (being careful not to let it run forward onto the perineum where it may cause smarting and discomfort). It is customary to use a spinal needle for the procedure, although a shorter 18 gauge needle about 2 inches long is easier to manage. Through a skin weal raised over the sacral hiatus, this needle is advanced at an angle of 45° until it impinges upon the sacro-coccygeal ligament which covers the sacral hiatus. This is traversed and the needle point quickly reaches the anterior wall of the sacral canal. After withdrawing the needle 1-2 mm., it is re-aligned so that it is pointing up the axis of the sacral canal. The hub of the needle at this point will be close to the skin between the buttocks. The needle is now pushed forward and advanced gently up the sacral canal for 1-1½ inches. This part of the procedure requires the most skill, due to individual variations in the angulation of sacral canals. It is a

common /

common error to withdraw the needle completely from the sacral hiatus and advance it superficially along the posterior surface of the sacrum.

With the needle in situ, note is taken of any reflux of fluid. Clear fluid would indicate a sub-arachnoid tap, while frank blood shows that either a blood vessel has been damaged or the needle point is actually in the lumen of a vessel. When these possibilities have been ruled out the injection of local anaesthetic can begin.

A test dose is again employed. Even though dural puncture is less likely than by the lumbar epidural approach, it is by no means impossible. The writer has had one such puncture in 50 cases. The injection should be quite easy to make and if resistance is met, it casts considerable doubt on whether the needle is correctly placed. On disconnecting the syringe, to allow time for the test dose to take effect, it is a common finding to see some of the local anaesthetic escape from the sacral canal, and quite often it is blood-stained. In contradistinction to lumbar epidural analgesia, where the needle only penetrates the epidural space for 1-2 mm., caudal analgesia necessitates penetration for up to $1\frac{1}{2}$ inches, often with considerable scraping of the needle on the bony walls of the canal. Some damage to blood vessels is, therefore, inevitable. The anaesthetist must be prepared to meet some manifestations of toxic reaction, and if blood-stained fluid escapes, the main injection must be given slowly.

Should /

Should the test dose produce no widespread analgesia in five minutes, the main dose can be injected. If a block extending up into the lumbar or thoracic regions is required, the injection has to be made with some force and this necessitates a rapid injection. The risks in doing this are well borne out by the writer's experience, for there were no fewer than five generalised toxic reactions in 50 cases, fortunately none very severe. As a result of these reactions, forceful injection is not to be recommended and caudal analgesia is unsuitable if a block higher than the fifth lumbar spinal segment is required. For blocks lower than this, slow, unforceful injection can be made. While the injection is being made, the back of the sacrum should be scrutinised for signs of a misplaced injection which will raise a visible swelling in the superficial tissues. As in lumbar epidural analgesia, the respiration should also be watched, apnoea being the first sign of massive sub-arachnoid injection.

Dosage

Prediction of the height to which a given volume of solution will reach, is even more difficult than with lumbar epidural analgesia. In the writer's cases, using forceful injection, 30 ml. of 1.5 per cent. lignocaine (including 5 ml. test dose) produced analgesia varying in height from the ninth thoracic segment to the second sacral segment. Even 45 ml. did not guarantee that the solution reached the thoracic /

thoracic segments. This, then, is another good reason for reserving caudal analgesia for low blocks, preferably of the sacral nerves only. For this purpose 20 ml. of 1.5 per cent. lignocaine is adequate.

Adrenaline should always be added to the solution to keep toxic reactions to a minimum. If the time factor is important, hyaluronidase can be added to speed up the onset of analgesia, 1,000 units being added to 20 ml. of solution.

Catheterisation of the sacral canal is easily performed. A needle of sufficient bore to allow the passage of the catheter is used. A Tuohy needle is of no value in this case as the catheter is to proceed in the line of the needle and not at right angles to it. This technique is useful in the management of pain in labour.

MANAGEMENT OF PATIENTS RECEIVING EPIDURAL ANALGESIA

Concomitant General Anaesthesia

Advantages.- In many medical centres, general anaesthesia is considered unnecessary as an accompaniment to spinal or epidural analgesia. In Great Britain, however, the great majority of patients prefer to be asleep during the surgical procedure. Moreover, most anaesthetists and surgeons would much rather have their patients unconscious.

From the anaesthetic point of view, the addition of general anaesthesia makes the procedure somewhat more complicated, and calls for special skills in management. Nevertheless, it does offer certain definite advantages.

(a) /

(a) The patient is asleep which relieves his or her anxiety.

(b) Any minor defect in the analgesia will not interfere with smooth anaesthesia. It should be remembered that the differential nerve block produced by epidural injection does leave certain sensations intact, such as deep pressure, and the patient can have an awareness of what is being done without actually feeling pain. As some motor function is retained, a relaxed abdomen can be made suddenly tight by the patient's voluntary contraction of the abdominal muscles. As the general anaesthesia is kept as light as possible, major defects in the analgesia will be quickly discernible.

(c) The production of hypotension is facilitated by general anaesthesia (see p. 52). This is of value when hypotension is considered desirable. If the systolic blood pressure is not low enough under light general narcosis, deepening of the anaesthesia frequently causes a further fall of the pressure. General anaesthesia does not interfere with the actions of vasopressors so that hypotension can be abolished or prevented if it is contra-indicated.

(d) General anaesthesia will prevent the onset of toxic reactions to the injected local anaesthetic, provided it is induced soon after completing the epidural injection. Thiopentone is the antidote to local anaesthetics producing toxic manifestations. It follows that thiopentone, given before such manifestations appear, raises the threshold for the production of a toxic reaction.

(e) /

(e) The presence of a conscious patient in the operating theatre imposes considerable restrictions upon the surgical team. Conversation concerning the operation or the patient's condition must be strictly limited. In the event of an emergency arising during the surgery, the commands issued, and the actions taken, may occasion much anxiety in an apprehensive patient.

(f) The patient is protected from any unpleasant effects of hypotension, such as dizziness or faintness. Similarly, vagal stimulation arising from traction on viscera (the vagus nerve being unaffected by epidural analgesia), can cause retching and vomiting in the conscious subject.

Requirements and Management of the General Anaesthesia

(1) Induction.- Thiopentone is the ideal drug for induction, which is rapid and pleasant for the patient. As the epidural injection is itself time-consuming, slow induction by inhalational anaesthetic is best avoided if possible. The use of thiopentone has two difficulties. Firstly, its hypotensive effects can be very marked if the blood pressure is lowered before administration. As the epidural block will produce hypotension in a certain number of patients, it is advisable to inject the thiopentone as soon as possible following completion of the epidural injection, before the systolic pressure has fallen appreciably. Secondly, apnoea can occur if thiopentone is injected too rapidly. As apnoea is one of the main signs

of /

of inadvertent total spinal blockade, it behoves the anaesthetist to avoid apnoea from any other cause so that a proper evaluation of the situation can be made.

Thiopentone, therefore, should be given shortly after the epidural injection is completed, and it should be given slowly. It is a common mistake to use too little thiopentone. In the average case, the author uses 400-500 mg. in 5 per cent. solution. Anything less than this can lead to interruption of smooth anaesthesia when inserting an airway or transferring the patient from the anaesthetic room to the operating theatre.

(2) Maintenance.- As the epidural blockade produces analgesia and relaxation in the operation area, the main purpose of the general anaesthesia is merely to keep the patient asleep. For this purpose an inhalational anaesthetic should be chosen. Nitrous oxide is suitable for most cases, but some patients cannot maintain a fixed plane of anaesthesia with this agent and may start to breathe irregularly, swallow, move the head or cough, especially in the presence of an oral airway or endotracheal tube. Should this occur, anaesthesia must be deepened, and for this cyclopropane is ideal, though any inhalational anaesthetic can be used. Intravenous agents, such as thiopentone, relaxants or pethidine, should be avoided for they all interfere with respiration. In addition, thiopentone, even in small doses, can cause profound hypotension and should be avoided if the systolic pressure is below 90 mm.Hg. Relaxants are /

are only indicated in cases where the epidural analgesia has not reached a high enough level and the tightness of the patient's muscles are interfering with the surgical exposure. Pethidine is primarily an analgesic, and patients who become too light under the general anaesthetic are not suffering from painful stimuli. The use of pethidine is, therefore, illogical.

Maintenance of this very light plane of general anaesthesia requires considerable skill and at the first indication of the patient emerging from unconsciousness (usually irregularity of respiration), the anaesthesia must be deepened. The importance of avoiding coughing and straining cannot be over-emphasised. Not only are they dangerous in the presence of hypotension (see p. 53), but they can cause the abdominal muscles to contract and temporarily ruin previously perfect surgical conditions.

Oxygen must, of course, be supplied to the patient in adequate amounts. Cyclopropane allows the use of a high percentage of oxygen and is of especial value in this respect. When using nitrous oxide, at least 25 per cent. oxygen should be added.

(3) Care of the Airway.- It is prudent for the anaesthetist to insert a mechanical airway of some kind in every case. The choice lies between a simple oral airway of the Guedal type or an endotracheal tube. After considerable experience with both methods, the author now prefers the former provided the patient's airway can be so maintained. /

maintained. Endotracheal intubation has the big advantage of ensuring complete control over the airway and there need be no hesitation in using it if an oral airway proves unsatisfactory. Its routine use, however, is beset with difficulties. To intubate a patient who is to maintain spontaneous respiration, the use of succinyl-choline, a short-acting muscle relaxant, is indicated. Following the epidural injection, the patient is given thiopentone and succinyl-choline, the lungs are inflated and the endotracheal tube inserted. This procedure, however, renders the patient apnoeic for a period of 3-8 minutes, at a time when observation of the respiration yields valuable evidence on whether or not total spinal anaesthesia has ~~not~~ been accidentally produced. Moreover, when the effect of the succinyl-choline wears off, the patient may cough and strain under the stimulus of a tube in the trachea, and deeper anaesthesia is required than with a simple oral airway.

Airways or endotracheal tubes should be smeared with an analgesic ointment to minimise the stimuli they produce from prolonged insertion. Pre-operative spraying of the mouth with 4 per cent. lignocaine facilitates the maintenance of an oral airway, and if endotracheal intubation is decided upon, it is advantageous to spray the pharynx, larynx and trachea prior to the passage of the endotracheal tube.

(4) Management of hypotension.- Regular and frequent observations of the pulse and blood pressure are made on all cases of epidural blockade. If the patient enters the hypotensive /

hypotensive range, i.e. the systolic pressure falls below 80 mm.Hg., special care is necessary.

Posture is of importance and fortunately no operation that can be performed under epidural analgesia requires a reverse Trendelenburg position; indeed most are facilitated by the head-down position which is the ideal for hypotension.

It is a common observation that, on becoming hypotensive, the average patient's requirement of general anaesthetic is reduced. Nevertheless, for the previously stated reasons, the patient must not be allowed to become too light.

Epidural analgesia takes 20-30 minutes to produce its maximum hypotensive effect. If the systolic pressure falls below 50-55 mm.Hg. (or higher in an elderly patient) the anaesthetist should be prepared to raise it with a vaso-pressor drug. Simple reversal of hypotension is easy. To elevate the systolic pressure without exceeding 80 mm.Hg., thus maintaining hypotension, is more difficult. Methoxamine, for the reasons stated on p. 64, is the most satisfactory drug for this purpose. Two milligrams should be injected intravenously and the effect noted. Methoxamine exerts its action usually within one minute and its effect has worn off in 15-20 minutes. Subsequent injections can be made, the dose being determined by the response to the first injection.

It is a uniform finding that, in spite of hypotension, these patients have a good pulse and readily measured blood pressure. As long as regular, quiet breathing is maintained and oxygenation is good, no anxiety need be felt.

If /

If the fall in blood pressure is thought to be inadequate, deepening of the general anaesthesia often lowers the pressure by a further 10-20 mm.Hg.

Should operative blood loss, in spite of hypotension, exceed 250 ml., replacement should be undertaken. Compared with normotensive patients, haemorrhage in hypotensive patients produces a disproportionate effect.

Vigilance is the key to success in the management of hypotension.

Post-operative Care

At the completion of operation, the inhalational anaesthetic is discontinued, and the patient will invariably recover consciousness within a few minutes, thus relieving the anaesthetist of any worry concerning the airway. This recovery is usually accompanied by a rise in blood pressure. Should the systolic pressure remain below 80 mm.Hg., it is wiser to raise it by giving a vasopressor drug before returning the patient to the ward. For this purpose an intravenous dose of vasopressor should be combined with an intramuscular dose, thus giving both an immediate and a sustained rise of blood pressure. In most cases 5 mg. of methoxamine intravenously and 10 mg. intramuscularly are sufficient. If hypotension should ensue after the patient has returned to the ward, vasopressors can be given similarly, provided there is no evidence that haemorrhage or shock is causing the hypotension. It may be thought that /

that this sudden reversal of hypotension might lead to reactionary haemorrhage, but in fact this does not occur, provided all major vessels have been secured during operation. This is of importance, for the surgeon can be misled by the reduction in operative blood loss under hypotension, and may not ligate quite sizeable vessels from which only a small amount of blood is being lost. It is wisest to tie off all bleeding points, however small, during operations under controlled hypotension.

On return to the ward, the patient is placed in bed, the foot of which is raised so that a slight head-down tilt is obtained. This position is maintained for about one hour after the epidural block has worn off. Its purpose is to have the patient in a favourable position should hypotension ensue. Once vasomotor control has been re-established, the patient can be moved into any position that is comfortable or is required for surgical reasons. This ability to move the patient in this way is in marked contrast to spinal anaesthesia where, if headaches are to be prevented, the head-down position must be maintained for 24 hours (see p. 148).

During the immediate post-operative period, epidural analgesia offers considerable advantages over general anaesthesia. Pain in the wound does not appear for some time (depending on the length of the operation and the drugs used for the epidural blockade). When it does appear, the patient has regained full consciousness and is able to cope with /

with it rationally. After general anaesthesia, however, reactions to pain often appear before the patient is fully awake, and this leads to post-operative restlessness, which can be a burden on the nursing staff, and is only effectively treated by the injection of an analgesic drug such as morphine. The result is that a respiratory depressant drug has to be given to the semi-conscious patient who may then relapse into unconsciousness. After epidural blockade, analgesics are only required when the patient is fully conscious and, in these circumstances, they produce much less respiratory depression and their weak hypnotic action does not cause unconsciousness.

Quick recovery of consciousness with continued analgesia of the wound can be exploited for the purpose of breathing and coughing exercises. These exercises are of great value in reducing the incidence of post-operative respiratory complications, especially in patients with concomitant respiratory disease such as bronchiectasis. In such cases the bronchial tree can be cleared by the patient's own efforts. When pain from the wound occurs, respiration is voluntarily reduced, and coughing may become too painful to be effective.

Some patients may suffer anxiety when they discover soon after awakening, that they are unable to move their legs properly. To avoid this, the writer usually informs them, after the epidural injection, but before the thiopentone is given, that the legs will feel numb and heavy for an hour or so /

so after they wake up.

In summary, it may be said that, provided the simplest precautions are taken against circulatory collapse, epidural analgesia offers post-operative conditions superior to any other form of anaesthesia.

Complications

A. Immediate complications

(1) Dural puncture.- This is the commonest cause of failure in attempting to locate the epidural space. If too much force is used to pierce the ligamentum flavum, the needle travels too far forward in the epidural space and penetrates the dura mater. It is sometimes recommended that the needle should be rather blunt, making it more difficult to pierce the dura. The writer has found, however, that a sharp Tuohy needle allows the ligamentum flavum to be traversed with greater ease and control, and the adoption of such needles has led to a considerable reduction in the number of sub-arachnoid taps. Very occasionally, in spite of careful insertion of the needle, cerebro-spinal fluid escapes and it would appear that, in these cases, the dura mater is adherent to the wall of the spinal canal, thus obliterating the epidural space. Increasing experience of the technique is the most effective way in which dural puncture can be prevented.

It is usually quite obvious from the escape of cerebro-spinal fluid that dural puncture has occurred. The possibility /

possibility that the escape is prevented, e.g. by a nerve root blocking the needle tip, is confirmed or ruled out by the use of the test dose.

Once the sub-arachnoid space has been entered the anaesthetist has the choice of three courses of action: (a) abandon the whole procedure and give a general anaesthetic instead, (b) give a spinal anaesthetic, or (c) attempt epidural puncture at a different level. The only choice that the writer would question would be the third. Once a hole is made in the dura mater it takes some considerable time to close, because of the lack of elastic tissue in the dura. If, following dural puncture, local anaesthetic is injected into the epidural space through an adjacent intervertebral space, it is very easy to build up enough pressure to cause a considerable amount of anaesthetic to leak through the hole into the sub-arachnoid space. At least one case has been recorded when this caused a total spinal blockade (Sykes[12]). As there is a good chance that some of the solution may pass through the dural puncture hole (even though it does not produce total spinal blockade), it would appear more rational to inject a known amount direct into the sub-arachnoid space.

(2) Massive sub-arachnoid injection.- As four to five times more local anaesthetic is used to produce epidural analgesia than is used for a spinal blockade of the same extent, it is obvious that should injection into the sub-arachnoid space be made accidentally, a profound

and /

and widespread nerve block will ensue. A dose of 20-30 ml. would block all the spinal nerves and spread through the foramen magnum to affect the cerebrum. There is some discussion among anaesthetists as to the fate of any solution reaching the intra-cranial sub-arachnoid space in this manner. Some believe that it may pass through the foramen of Magendie and the foramina of Lushka to reach the fourth ventricle, where it could affect vital centres. This, however, is most unlikely, since it is much easier for it to spread round the outside of the brain and block the cranial nerves.

The responses to a large sub-arachnoid injection appear quickly, often before the patient is turned onto the back at completion of the injection. The patient becomes unconscious, apnoeic and hypotensive. The pupil will dilate as the occulomotor nerve becomes affected. When these changes occur rapidly, diagnosis is not difficult. Occasionally, however, they may take a few minutes to attain their full effect, by which time the patient may have received thiopentone. One case has been described (Sykes[12]) where the correct diagnosis was not obvious for some 20 minutes. In this case, epidural injection had been made after the dura mater had been pierced at a lower vertebral interspace. There was thus a more gradual entry of local anaesthetic into the sub-arachnoid space and only part of the solution would have found its way through the puncture hole. The significance of the apnoea was masked in this case /

case by pethidine which had been given to the patient to stop a bout of coughing.

A combination of profound hypotension and apnoea places the patient in a very precarious condition, and, if prompt treatment is not instituted, death will follow within a few minutes. Fortunately, treatment is quite simple and the patient can be quickly restored to a safe state.

Treatment.- To combat apnoea, artificial respiration with oxygen must be commenced. Keeping in mind that positive pressure respiration in the presence of hypotension causes a further reduction of an already depleted cardiac output, immediate measures must be taken to raise the blood pressure. Vasopressors will do this rapidly if given intravenously in adequate dosage. If a vein has been kept open by the use of a Mitchell needle inserted before the attempted epidural block, rapid administration of intravenous drugs is facilitated. If, for any reason, it is difficult to inflate the patient's lungs while using an anaesthetic mask, endotracheal intubation should be performed. No difficulty in doing this will be encountered, for the pharynx and larynx will be anaesthetised by the paralysis of the cranial nerves.

When oxygenation has been effected and hypotension reversed, artificial respiration must be continued until the spinal blockade has worn off. This will take from 1-2 hours if lignocaine has been used, but up to 3 hours if amethocaine was added. Further doses of vasopressor should be given

when /

when the blood pressure shows any sign of falling. Once the patient has been brought into the safe state, there is no contra-indication to carrying out the operation as planned.

If, unfortunately, effective treatment is delayed too long, cardiac arrest may occur, in which event cardiac massage must be carried out immediately.

Avoidance of massive sub-arachnoid block is obviously preferable to treatment however efficient. Prophylaxis depends upon a good technique to ensure accurate placement of the main injection in the epidural space. The value of the test dose has been discussed on p. 75 . In the writer's opinion, five minutes spent on each case to use the test dose, is not time wasted if this very worrying complication can be prevented. It is significant that almost all the reported cases of massive sub-arachnoid injection have occurred when the test dose was omitted (de Saram[13]; Lund, Cwik and Magaziner[14]; Gordon Jones[15]).

Case Report

In the author's series, there has been one case where total spinal block occurred and this was on one of the rare occasions when the test dose was not used. The patient was a lady of 76 years suffering from a strangulated femoral hernia of 3 days duration, who had been vomiting for 24 hours. The general condition was not good and in view of the dehydration and reduced circulating blood volume, epidural block would not now be considered. At the time it was thought that regional anaesthesia would allow the patient to retain consciousness and thus prevent aspiration of stomach contents. It was obvious when lumbar epidural puncture was attempted that the patient would be unable to co-operate intelligently with the use of a test dose, and it was dispensed with. Twenty millilitres of 1.25 per cent. lignocaine were injected. When the patient was turned onto her /

her back her colour was noticed to be poor. She rapidly became apnoeic and there was no palpable pulse. Artificial respiration was commenced and endotracheal intubation performed easily although some pharyngeal and laryngeal movements had been retained. Methamphetamine 30 mg. was given intravenously and the pulse returned. Using very large doses of methamphetamine (110 mg. over 1 hour), the systolic blood pressure was maintained at around 100 mm.Hg. Nitrous oxide and oxygen were administered and the operation (which required excision of 6 inches of gangrenous small intestine) was performed in 45 minutes. Fifteen minutes later spontaneous respiration re-started and when the nitrous oxide was discontinued the patient attempted to remove the endotracheal tube. When this was done for her, she soon became fully conscious and made an uneventful recovery. It is just possible that this case was one not of total spinal blockade, but of hypotension following epidural block in a hypovolaemic patient. The fact that the pharynx and larynx were not completely anaesthetised is against total spinal blockade. The pupil, which would have given valuable information, was unfortunately not observed in the anxiety of the moment. In any case the treatment was identical whichever of the two possibilities had occurred. That a very large dose of methamphetamine was necessary to maintain the blood pressure is explained by the pre-existing hypovolaemic shock.

If epidural analgesia is to be performed, the possibility of this complication must always be kept in mind and facilities for immediate treatment must be at hand. The great danger is not that total spinal blockade may occur, but that its treatment may be delayed. If the anaesthetist is prepared to treat it promptly and energetically, massive sub-arachnoid injection can be regarded as a nuisance with potential danger, rather than a calamity. That it can occur should not be accepted as a contra-indication to epidural analgesia.

(3) Toxic reaction.- It is now recognised that toxic reactions to local anaesthetics are due to high blood levels of the drugs and not to any hyper-sensitivity of the patient /

patient to the drugs. When injecting into a highly vascular area like the epidural space, rapid absorption into the blood can occur. Such absorption is aided by:

(a) The use of undue force during injection. In this way the local anaesthetic is given too rapidly and the pressure of fluid within the epidural space is raised. Moreover, forceful injection causes a greater spread of the drug thus making more vessels available for absorption.

(b) Damage to, or even cannulation of, blood vessels during insertion of the needle. This is of particular importance in caudal epidural analgesia.

(c) The use of large quantities of local anaesthetic.

Absorption can be slowed by the addition of adrenaline to the local anaesthetic, which causes a local vasoconstriction.

The toxic effects of a local anaesthetic are confined to the central nervous and cardiovascular systems. As the blood level of the drug increases, the central nervous system is first stimulated, leading to tremors followed by convulsions, and then depressed, with coma and death supervening. Depression will be due not only to increasing blood levels of the drug, but also to hypoxia, which occurs during the convulsions, and is accentuated by cardiovascular collapse. Both the heart and the peripheral vascular tone are depressed by toxic levels of local anaesthetics leading to decreasing cardiac output and hypotension.

The /

The avoidance of convulsions is of first importance, for in their presence, adequate oxygenation cannot occur. Fortunately, there is an appreciable difference in the blood level of local anaesthetic causing minor tremors and the level causing convulsions. When lignocaine is used intravenously to augment general anaesthesia, tremors are used as an indication that enough drug has been given, and if administration is stopped at that point, cardiovascular depression does not occur (de Clive-Lowe, Gray and North[16]). In the series of 3,637 cases of epidural block reported by Bonica[9] et al., 212 cases of toxic reaction were noted, only 8 of which had convulsions. Minor toxic reactions, therefore, are not uncommon, though major reactions are fortunately rare.

Prevention.- (a) Slow unforceful injection of local anaesthetic should be made. For lumbar epidural block the author injects at the rate of 1 ml. every 3 seconds, and has yet to see a toxic reaction. Unfortunately, if epidural block by the caudal route is to be used for an operation requiring anaesthesia above the sacral segments, considerable force may have to be applied to push the solution high enough. Such force led to the appearance of 5 instances of minor reaction in a series of 50 caudal injections. The patients complained of dizziness, faintness and nausea and were seen to have muscle tremors, sometimes starting in an isolated muscle group and spreading. In one case hypotension was severe enough to warrant the use of a vasopressor /

vasopressor, but no convulsions were seen perhaps because thiopentone was given quickly when the tremors occurred. Because of these reactions, caudal block is only recommended for cases requiring anaesthesia of the sacral nerves.

(b) Adrenaline in a concentration of 1 in 80,000 should be added to the solution, especially if a combination of local anaesthetics is used, or if hyaluronidase is employed.

(c) The total dose of lignocaine should be limited to 40 ml. of 1.5 per cent. solution. This is well within the maximum safe dose for infiltration and will be quite sufficient for any operation below the diaphragm.

(d) By inducing general anaesthesia with thiopentone as soon as the epidural block is completed, a higher blood level of local anaesthetic can be tolerated before a toxic reaction will occur. This is because thiopentone is a central nervous system depressant, and thus antagonises the stimulation produced by local anaesthetics.

Treatment.- If a toxic reaction appears before general anaesthesia has been induced, thiopentone should be administered forthwith. In the presence of severe convulsions, this may be a difficult manoeuvre, unless a vein has been made available by the prior insertion of a Mitchell needle. Any cardiovascular depression is corrected by intravenous vasopressor. Pulmonary ventilation will quickly reverse any hypoxia once thiopentone has /

has controlled the convulsions.

(B) Remote complications

(1) Infection.- The possibility of introducing infection into the epidural space must be seriously considered. This can occur only if the sterilisation of drugs and equipment is at fault, or if the anaesthetist does not use a proper aseptic technique. These deficiencies are obviously preventable. The same precautions used in the administration of a spinal anaesthetic must be taken for epidural blockade. However, if for any reason pathogenic bacteria were introduced into the epidural space, the outlook would not be quite as serious as a similar amount of contamination of the sub-arachnoid space. The epidural space is richly supplied with blood vessels and would be able to resist infection as well as any other region of the body. The sub-arachnoid space, on the other hand, has a poor resistance to infection. This, of course, does not allow any laxity in the precautions to be taken against infection.

(2) Neurological sequelae.- It has always been maintained that epidural analgesia is much less liable to produce neurological damage than spinal blockade. In the epidural space, the spinal nerves have a protective coating of dura mater and perineurium. In the sub-arachnoid space, not only are the nerves relatively unprotected, but the spinal cord itself is within easy reach of any neurotoxic process. Reported cases of neurological complications

following /

following epidural blockade are uncommon and this has tended to confirm the view that the procedure was relatively free from these unfortunate occurrences. Three cases of nervous damage occurred in the series of Bonica et al.[9] Two of these cases followed accidental sub-arachnoid injection and direct trauma to the spinal cord, which would have been averted if the lumbar and not the thoracic route had been chosen. The third case had some weakness and numbness of one lower limb which cleared completely in six weeks. In the series of over 2,000 cases by Lund et al.[14], there was only one case of nerve damage and this also occurred after massive sub-arachnoid injection.

In two recent reports, 3 cases of neurological damage have been described. A patient reported by Davies, Solomon and Levene[17] developed thrombosis of an anterior spinal artery with consequent cord damage and paraplegia. The patient, however, was an old man of 76 suffering from congestive cardiac failure of some years duration to which was added the dehydration and shock from an inguinal hernia which had been strangulated for 3 days. He was given 27 ml. of 1.75 per cent. lignocaine and 1 in 300,000 adrenaline. In the writer's opinion, such a case would be most unsuitable for epidural analgesia, and in any case 27 ml. was a gross overdose for the surgical procedure that was carried out.

Two cases described by Braham and Saia[18] developed paraplegia following epidural analgesia using procaine. One patient, who suffered from a typical post-operative spinal headache, /

headache, had obviously received all or part of the injection into the sub-arachnoid space. The other patient was an old man who had had a spinal tumour incompletely removed some years previously. How much effect this had on the post-operative muscle weakness is difficult to judge. As in similar cases following spinal anaesthesia, it is often impossible to assess all the contributory factors that may produce nervous damage, unless one has been present during the administration. Possible contamination, chemical or bacterial, of the solution injected, faulty technique and subsequent management have to be ruled out before the accident can be accepted as inevitable.

It would be unwarranted to say that epidural analgesia is quite free from unavoidable neurological complications, but from the evidence so far produced in the literature, the danger cannot be said to be common enough to be considered as a contra-indication.

In the presence of pre-existing neurological disease, it is always prudent for the anaesthetist to avoid spinal and epidural blockade. It cannot be said with any conviction that such blockade could affect the disease, but any deterioration in the patient's neurological state will immediately be attributed to the procedure.

Without wishing to underestimate the possibility of neurological damage following either spinal or epidural analgesia, it is interesting to note reports of cases where such damage followed conventional general anaesthesia.

Sinclair /

Sinclair[19] described an ascending spinal paralysis occurring 6 days after total hysterectomy performed under thiopentone, gallamine, nitrous oxide and ether anaesthesia. Paresis of the ocular muscles persisting for some months has also been noted after tubocurarine administration (Hewer;[20] Norman).[21] Ditzler and McIver[22] reviewed, from the literature, several cases of spinal cord damage following general anaesthesia and describe a case of anterior spinal artery thrombosis which occurred in their practice, immediately following thoracotomy under general anaesthesia. The possibility that nervous damage can occur unrelated to the anaesthetic procedure must be borne in mind. If spinal or epidural analgesia had been employed, they would certainly have been incriminated in all the above cases.

References

(1) Janzen, E. (1926). Dtsch. Z. Nervenheilk, 94, 280.

(2) Macintosh, R.R. and Mushin, W.W. (1947). Anaesthesia, 2, 100.

(3) Bryce-Smith, R. (1950). Anaesthesia, 5, 213.

(4) Odom, C.B. (1936). Amer. J. Surg., 34, 547.

(5) Macintosh, R.R. (1950). Anaesthesia, 5, 98.

(6) Brunner, C. and Ilke, A. (1949). Schweiz. med. Wschr., 79, 799.

(7) Macintosh, R.R. (1953). Brit. med. J., 1, 398.

(8) Bromage, P.R. (1954). "Spinal Epidural Analgesia," p. 57. Edinburgh: E. & S. Livingstone Ltd.

(9) /

(9) Bonica, J.J., Backup, P.H., Anderson, C.E., Hadfield, D., Crepps, W.F. and Monk, B.F. (1957). Anesthesiology, 18, 723.

(10) Bromage, P.R. (1954). "Spinal Epidural Analgesia," p. 116. Edinburgh: E. & S. Livingstone Ltd.

(11) Foldes, F.F., Colavincenzo, J.W. and Birch, J.H. (1956). Anesth. et Analg., 35, 33.

(12) Sykes, M.K. (1958). Anaesthesia, 13, 78.

(13) de-Saram, M. (1956). Anaesthesia, 11, 77.

(14) Lund, P.C., Cwik, J.C. and Magaziner, R. (1956). Anesthesiology, 17, 605.

(15) Gordon Jones, R.G. (1953). Anaesthesia, 8, 242.

(16) de Clive-Lowe, S.G., Gray, P.W.S. and North, J. (1954). Anaesthesia, 9, 96.

(17) Davies, A., Solomon, B. and Levene, A. (1958). Brit. med. J., 2, 654.

(18) Braham, J. and Saia, A. (1958). Brit. med. J., 2, 657.

(19) Sinclair, R.N. (1954). Anaesthesia, 9, 286.

(20) Hewer, C.L. (1946). Proc. roy. Soc. Med., 39, 409.

(21) Norman, J.E. (1955). Anaesthesia, 10, 87.

(22) Ditzler, J.W. and McIver, G. (1956). Anesth. et Analg., 35, 501.

VI. CLINICAL RESULTS AND ASSESSMENT OF THE PLACE OF EPIDURAL ANALGESIA IN ANAESTHETIC PRACTICE

GENERAL OPERATING CONDITIONS

Operating conditions with epidural analgesia are very similar to those prevailing during spinal anaesthesia. Relaxation is maximal and can only be simulated by such large doses of relaxant drugs that apnoea would certainly ensue. Within the abdomen, the gut is contracted by the unopposed action of the vagi, and access is thus facilitated. Breathing is quiet and regular, and the movement of the diaphragm does not interfere with intra-abdominal exposure. Intercostal movement is unimpaired (in contrast to spinal anaesthesia which often produces lower intercostal paralysis). This is good evidence of the fact that epidural analgesia produces relaxation without total paralysis (see p. 37).

Some authorities believe that respiration during epidural blockade is shallow and somewhat depressed, and, although they agree that it has little or no deleterious effect upon the patient, they feel that carbon dioxide retention probably occurs (Lee[1]). The writer disagrees with this view. The breathing is certainly quiet, but not more so than in natural sleep. It is easily forgotten that anaesthetists seldom have patients asleep without stimulation. As a result they tend to take as a norm the respiration expected from a lightly anaesthetised patient undergoing surgery. This is quite different from a patient lightly anaesthetised, but receiving no stimulation from the surgery, as with epidural analgesia.

A /

A measure of the excellence of operative conditions, is the fact that, once surgeons have had experience with epidural analgesia, they will not only request its use, but are also prepared to wait the extra time for it to be performed.

Good conditions depend upon the skill of the anaesthetist, not only in producing the epidural block, but also in keeping the patient asleep without coughing and straining. With experience premonitory signs, such as irregularity of breathing or swallowing, can be properly assessed and the necessary action taken before straining occurs. In the writer's opinion, as much, if not more, judgment and care are required in the management of the patient than is needed during the performance of the epidural blockade. This is especially so if hypotension is to be used.

HYPOTENSION

Some earlier workers thought that, unlike spinal anaesthesia, epidural blockade only rarely affects the blood pressure (Odom[2]; Massey Dawkins[3]). While it is true that it is more difficult to produce hypotension with epidural than by spinal analgesia, this is due to the greater ease with which a high block is effected with the latter. There is no reason why blockade of the same number and height of spinal nerves by either method should not produce the same fall in blood /

blood pressure.

From the writer's series, 130 unselected cases of lumbar epidural analgesia had their systolic blood pressures carefully recorded and analysed. Falls in blood pressure from the pre-operative level have only been considered significant if they have persisted for 20-30 minutes. This eliminates any transient reduction due to thiopentone. Blood loss was not thought to have exerted an effect in any of the cases.

Table I shows that 92 per cent. of cases had a drop in systolic pressure exceeding 20 mm.Hg. and in 60 per cent. the drop was over 50 mm.Hg.

TABLE I

Fall in Systolic Pressure	No. of Cases
0 - 20 mm.Hg	12 (9%)
20 - 50 mm.Hg	41 (32%)
Over 50 mm.Hg	77 (60%)

Under anaesthetic conditions, hypotension is considered to exist whenever the systolic blood pressure has fallen to 80 mm.Hg. or lower. This may mean a fall of only 30 mm. in a case whose normal systolic pressure is 110 mm.Hg., but four times such a fall in a patient with a pressure of 200 mm.Hg. The patients have been divided into three groups according to the lowest pressure reached and sustained for 20-30 minutes during the course of the anaesthesia. In 26 per cent. of cases the systolic pressure did not fall below 100 mm.Hg.;

in /

in 32 per cent. it fell to between 80 and 100 mm.Hg.; and in 42 per cent. it reached the hypotensive level, i.e. below 80 mm.Hg. (see Table II).

TABLE II

Lowest systolic pressure reached	No. of Cases
Above 100 mm.Hg	34 (26%)
80 - 100 mm.Hg	42 (32%)
Below 80 mm.Hg	54 (42%)

To assess the effect of age upon the hypotensive action of epidural analgesia, the number of cases becoming hypotensive in different age groups is shown in Table III.

TABLE III

Age of Patient	No. of Cases	Systolic Pressure above 80 mm.Hg	Systolic Pressure below 80 mm.Hg
20 - 40 years	41	31 (76%)	10 (24%)
41 - 60 years	64	35 (54%)	29 (46%)
Over 60 years	25	10 (40%)	15 (60%)

It will be noted that more of the elderly patients became hypotensive than did the younger ones. It should be remembered that the dosage of local anaesthetic (and therefore the height of the analgesia) was reduced in the elderly otherwise the relationship between age and the fall in pressure would have been even more marked.

As /

As the dosage varied from 15 ml. to 35 ml. of lignocaine, the number of cases becoming hypotensive were calculated according to the dose administered (Table IV).

TABLE IV

Dose of Lignocaine (1.5 per cent.)	No. of cases	Non-hypotensive	Hypotensive
15 - 20 ml.	18	11 (61%)	7 (39%)
21 - 25 ml.	17	10 (59%)	7 (41%)
26 - 30 ml.	57	34 (60%)	23 (40%)
31 - 35 ml.	38	21 (55%)	17 (45%)

There is only a slight correlation between the drop in systolic pressure and the dose of lignocaine. This is, at first sight, rather surprising, until it is remembered that the elderly in whom a blood pressure reduction is most easily produced, received smaller doses than the younger patients.

In the analysis of these figures, it must be remembered that hypotension was not a primary aim of the method. A higher proportion than 42 per cent. of cases could undoubtedly have been made hypotensive, if that were desired, by using higher doses of local anaesthetic.

Because caudal analgesia seldom reaches a high level, the incidence of hypotension is rather less than with lumbar epidural block. An analysis of 38 cases of caudal analgesia receiving 35-40 ml. of lignocaine (Table V) shows that in 68 per cent. the systolic pressure remained above 100 /

100 mm.Hg. (compared with 26 per cent. in lumbar epidural analgesia) and in only 8 per cent. did it fall below 80 mm. Hg. (42 per cent. in lumbar epidural analgesia).

TABLE V

Lowest systolic pressure in caudal analgesia	No. of Cases
Above 100 mm.Hg	26 (68%)
80 - 100 mm.Hg	9 (24%)
Below 80 mm.Hg	3 (8%)

Comparative figures in 40 cases under general anaesthesia are shown in Table VI. The anaesthesia consisted of a thiopentone, relaxant and nitrous oxide sequence, with controlled respiration and small divided doses of pethidine. In 34 of these patients the systolic pressure remained above 100 mm.Hg and in none did hypotension occur.

TABLE VI

Lowest systolic pressure in general anaesthesia	No. of Cases
Above 100 mm.Hg	34 (85%)
80 - 100 mm.Hg	6 (15%)
Below 80 mm.Hg	0 (0%)

It will be seen that, whereas general anaesthesia seldom lowers the systolic pressure below 100 mm.Hg., lumbar epidural /

epidural analgesia, in the doses used by the writer, causes such a lowering in 74 per cent. of cases. The results with caudal analgesia are of interest because they show that even a low nerve block has a small, but definite, hypotensive effect. The sympathetic outflow ends at the second lumbar spinal segment and spinal anaesthesia must reach above this level to effect any vasodilatation. Epidural analgesia, however, can theoretically effect vasodilatation by blocking the grey rami communicantes to the nerves below the second lumbar spinal segment. That such a blockade occurs is borne out by the hypotension induced by caudal analgesia in spite of a low level of analgesia.

The fact that almost all cases under epidural block suffer a fall in pressure, while 42 per cent. become hypotensive, may not be regarded by some as a particularly good feature of an anaesthetic technique. However, in judging hypotension occurring in the course of anaesthesia, the important considerations are (a) the cause of the hypotension, (b) the circumstances in which it is produced, and (c) its response to treatment. If hypotension is due to direct depression of the myocardium, as occurs with chloroform, it should certainly be viewed with caution, but epidural analgesia exerts its hypotensive effect through sympathetic blockade and the myocardium is not directly affected. If an anaesthetic agent or technique causes hypotension in circumstances requiring controlled respiration, then /

then apprehension is justified. Epidural analgesia does not affect respiratory function, and hypotension is occurring, therefore, under favourable conditions. The reaction of patients to vasopressors is enhanced during epidural analgesia (in common with other forms of sympathetic blockade) and hypotension is easily and rapidly reversed.

Paradoxically, if criticism is to be made of epidural analgesia, it is because it does not produce hypotension with any certainty in young patients. If hypotension is considered essential in such patients, spinal anaesthesia is to be preferred (Scott[4]).

OPERATIVE BLOOD LOSS

In describing any anaesthetic technique, the enthusiasts can seldom forbear to say that bleeding during operation is reduced. It is seldom stated from what standard it is reduced, but it is usually inferred to be any other technique available to the anaesthetist. Actual figures are rarely given, and even more rarely is any rationale advanced as to why bleeding should be reduced.

The writer has made an attempt to collect accurate measurements of operative blood loss during both epidural analgesia and conventional general anaesthesia. For the results to be of value, the following procedures were adopted:-

(a) /

(a) Measurement of operative blood loss.- This was done by swab weighing. The weight of clean swabs being known, the amount of blood being absorbed by them during operation can easily be measured. It is not difficult to collect almost all the blood on the swabs, unless the loss is very heavy. The little that finds its way onto towels and operating gowns can be allowed for; this rarely exceeds 100 ml., and is usually less than 50 ml. With practice, the method can be reasonably accurate, and the error should not exceed 10 per cent. Apart from investigations into blood loss, swab weighing is of considerable use in clinical practice for it allows accurate blood replacement.

(b) Choice of a standard operative procedure.- Fothergill's operation of pelvic floor repair is well suited for comparisons. Local pathology is unlikely to lead to much difference in the amount of blood lost from case to case. It is an operation in which bleeding can be a considerable annoyance, if only very occasionally so profuse as to require replacement transfusion. The operative technique is standardised in the gynaecological unit in which the investigation was carried out, and the operations performed by the four surgeons are, therefore suitable for comparison.

(c) Controls.- The control cases were anaesthetised with thiopentone, a relaxant, pethidine and nitrous oxide, respiration being controlled. This method was chosen

because /

because its protagonists maintain that bleeding is minimal, especially if manual hyperventilation is used. It certainly prevents hypercapnoea, which is a well known cause of increased blood loss, and it eliminates the need for an agent such as cyclopropane, which has a poor reputation in respect to operative bleeding. It would have been ideal to allot alternate cases to the control series. Unfortunately, for the smooth and easy management of long operating lists this could not be done. However, there was no medical selection of cases. If time was available for the performance of epidural analgesia it was usually carried out, otherwise general anaesthesia was employed.

(d) To eliminate observer bias, all the weighing of swabs was carried out by nurses who had no knowledge of the investigation.

(e) Adrenaline infiltration.- To obtain the best possible conditions while performing the operation, the surgeons preferred to inject adrenaline at the site of operation. This not only causes vasoconstriction but also allows easier separation of fascial planes. A 1 in 200,000 solution was infiltrated round the cervix and along the line of the incision made in the anterior vaginal wall. When the cervix had been amputated and the anterior part of the repair completed, more adrenaline was injected into the posterior vaginal wall and perineum prior to the posterior colpotomy and repair which completes the operation. The amount /

amount of adrenaline used varied from case to case but on average 15-20 ml. was used anteriorly, and 10-15 ml. posteriorly. Injection of adrenaline into patients while under general anaesthesia is often regarded as dangerous. The generalised effects of localised adrenaline infiltration have proved somewhat surprising and will be discussed in more detail later (see p. 125).

The blood loss during pelvic floor repair is indicated in Table VII and Fig. 9. The former shows that there is markedly less bleeding in patients under epidural analgesia than in those under general anaesthesia.

TABLE VII

Blood Loss	Epidural Analgesia	General Anaesthesia
No. of Cases	36	29
Average	3.6 fl. oz. (102 ml.)	8.9 fl. oz. (253 ml.)
Range	1-8.5 fl. oz. (28-240 ml.)	2-22.0 fl. oz. (56-616 ml.)

In assessing the blood loss during operation, the variation from case to case is important, for consistency is highly desirable. Fig. 9 shows the comparative ranges in blood loss in the two groups of cases.

It will be seen that in every epidural case less than 10 fl. oz. (284 ml.) of blood was lost, the maximum being 8.5 fl. oz. (240 ml.). The results were much more consistent than in the cases done under general anaesthesia, which

sometimes /

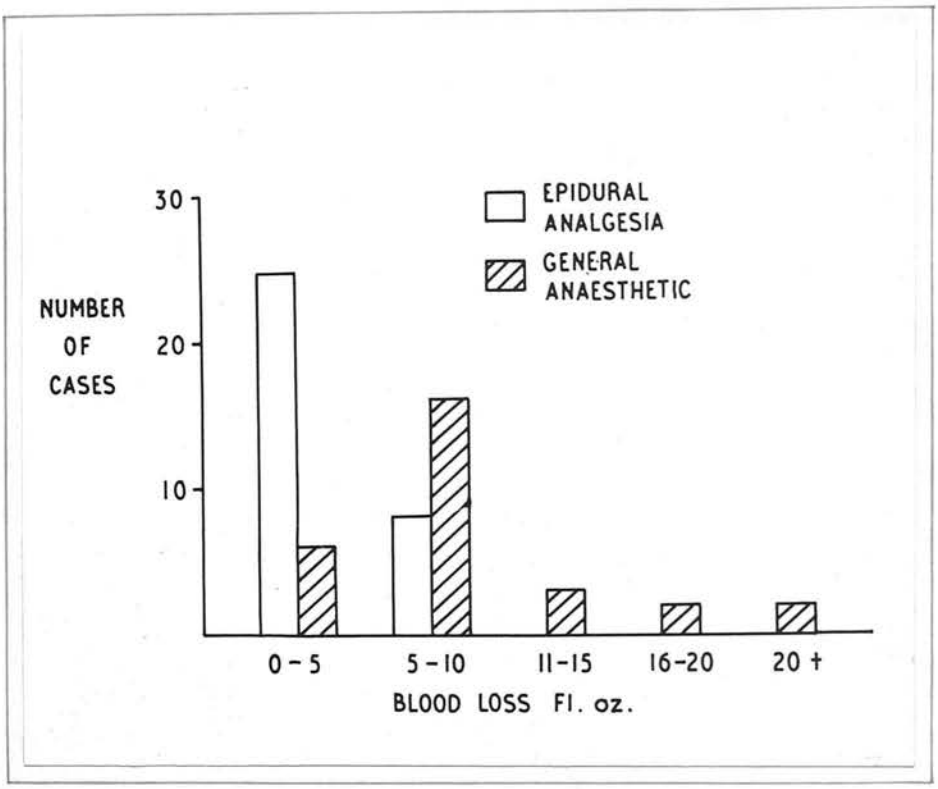


Fig. 9.- Blood loss during pelvic floor repair operations. The chart shows the distribution of cases performed under epidural analgesia and general anaesthesia. It shows that the former is far more consistent in producing good operating conditions.

sometimes produces a relatively bloodless field but often allows considerable bleeding.

To assess the effect of hypotension in epidural cases, they were divided into those who became hypotensive and those who did not.

TABLE VIII

	Epidural analgesia with hypotension (Systolic pressure below 80 mm.Hg.)	Epidural analgesia without hypotension (Systolic pressure above 80 mm.Hg.)
No. of Cases	12	24
Average blood loss	2.3 fl. oz. (65 ml.)	4.2 fl. oz. (120 ml.)

It will be seen that, even without hypotension, the results with epidural analgesia are superior to those with general anaesthesia.

Bleeding in pelvic floor repair operations is seldom heavy enough to cause concern, but it may impede the work of the surgeon considerably, and prevent the best surgical result. In general it may be said that, with losses of less than 5 fl. oz. (135 ml.) operating conditions are excellent, while with 5-10 fl. oz. (135-270 ml.) they are satisfactory. Losses in excess of 10 fl. oz. (270 ml.) interfere with the operation, and if the loss approaches 20 fl. oz. (540 ml.), it can seriously impair the procedure.

A great many factors can modify operative bleeding. Those /

Those that have a beneficial part to play in epidural analgesia are:-

(a) Hypotension. This may be thought to be the main factor but Table VIII shows that it is by no means the only one.

(b) Spontaneous respiration. This keeps venous pressure low, unlike controlled respiration, which, by eliminating the normal intrathoracic negative pressure, raises venous pressure.

(c) Complete relaxation of the abdominal muscles. Intra-abdominal tension cannot then rise sufficiently high to exert pressure upon the inferior vena cava. It is well recognised that pressure on the vena cava can cause increased venous bleeding.

(d) The local haemostatic effect of adrenaline. Its action is more marked in epidural analgesia than in general anaesthesia. This is probably due to the fact that the arterioles supplying the uterine cervix and vagina are temporarily sympathectomised by the epidural block and are, therefore, more sensitive to the vasoconstrictor effect of adrenaline.

Adrenaline.- Although the concentration of adrenaline used for local infiltration is kept very low (1 in 200,000) it still produces well marked systemic effects. Twenty millilitres of such a solution contains 0.1 mg. of adrenaline, which is well within the limit of safety in a conscious patient. With general anaesthetics, especially agents /

agents such as chloroform or cyclopropane, adrenaline is considered to be dangerous because of the risk of producing cardiac irregularity and ventricular fibrillation. This is a result of the well known cardiac stimulating action of adrenaline, and fatalities have certainly occurred when the dosage of adrenaline has been high, or when it has been given, inadvertently, into a vein.

When injected into the conscious subject, adrenaline has two main actions on the cardiovascular system. It produces changes in the tone of blood vessels, and it stimulates the myocardium. Contrary to previous opinion which attributed the marked rise in systolic blood pressure mainly to vasoconstriction, it is now known that adrenaline causes an overall vasodilatation (Robson and Keele[5]). Some vessels, such as those in the skin, are constricted, but this is countered by vasodilatation in vessels supplying voluntary muscle. The marked rise in pressure is caused by increased cardiac output resulting from the myocardial stimulation, and tachycardia. These actions are in contrast to those of nor-adrenaline which is a pure vasoconstrictor and has no action upon the myocardium.

Under general anaesthesia, the cardiovascular system undergoes changes that may alter the effects of adrenaline. Anaesthetics cause vasodilatation in varying degree. Further dilatation produced by adrenaline in the vessels which dilate under its action, may not, therefore, be very marked. Those which constrict, however, may respond even more readily, /

readily, in the same way as sympathectomised vessels respond to other vasoconstrictors. This will be seen especially under conditions of sympathetic paralysis as occurs with epidural blockade. It is possible, therefore, that adrenaline may produce an overall vasoconstriction under general anaesthesia and this would elevate the blood pressure independently of any direct action upon the myocardium. Stimulation and excitation of the myocardium would be evidenced by a tachycardia and cardiac irregularity, and these are the signs that should be viewed apprehensively during general anaesthesia.

With large doses of adrenaline elevation of the blood pressure, tachycardia and irregularity undoubtedly occur. With small doses, however, as used in infiltration with a 1 in 200,000 solution, no change can be detected in either the rate or regularity of the pulse, in spite of the fact that, in almost every case, some elevation of the blood pressure occurs. This elevation can often be as much as 40-50 mm.Hg. in the systolic pressure. It occurs within 1-2 minutes after infiltration, and lasts 5-10 minutes. A typical chart is shown in Fig. 10.

This raising of the blood pressure without any change in the cardiac rate has occurred in both those receiving epidural analgesia and the control series with general anaesthesia, though the effect is somewhat more pronounced in the former. Tachycardia has only been seen once, and

in /

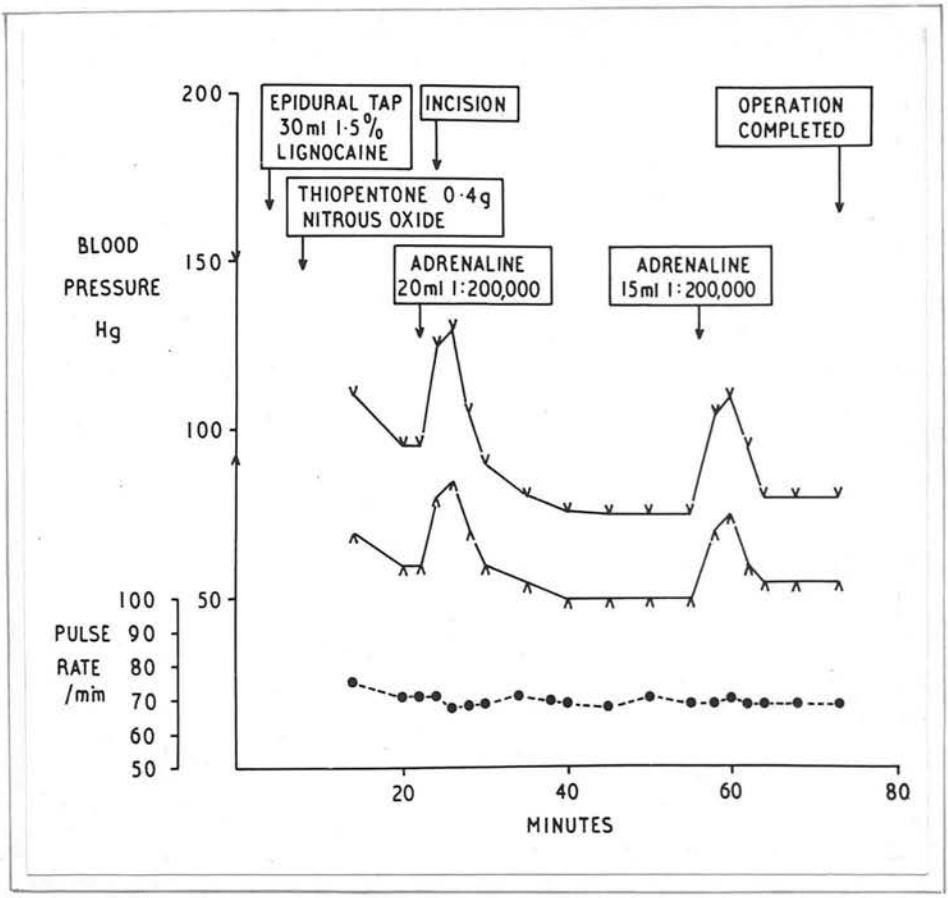


Fig. 10.- Chart showing the action of locally injected adrenaline during anaesthesia. Note the marked rise in blood pressure without any variation in pulse rate.

in this the pulse was only raised from 72/min. to 100/min. and no arrhythmia occurred. The hypertensive action was so constant that doubts as to the potency of the adrenaline were entertained when it did not occur.

It is significant that doses of less than 0.1 mg. of adrenaline, which produce obvious changes in blood pressure during anaesthesia, would have little or no effect on conscious subjects. This is further evidence that, in the former case, the adrenaline is acting upon blood vessels made more sensitive to its vasoconstrictor effects.

It is reasonable to suppose that, in small doses under anaesthetic conditions, adrenaline acts as a pure vaso-constrictor, without any excitation of the myocardium. It should be noted that elevation of the diastolic pressure also occurs, unlike the response in conscious patients when the diastolic pressure falls (this being evidence of the overall vasodilatation in conscious subjects). These results have encouraged the writer in the belief that infiltration of adrenaline in dilute solution is unlikely to predispose to ventricular fibrillation. Intravenous injection can be avoided by constant movement of the infiltrating needle.

These observations are of practical application not only in pelvic floor repairs, but in other operations, such as thyroidectomy or craniotomy, where adrenaline infiltration may be considered advantageous.

POST-OPERATIVE STATE /

POST-OPERATIVE STATE

Provided the blood pressure is adequate before leaving the operating theatre and the blood lost (if it exceeds 250-350 ml.) has been replaced, the condition of patients after epidural analgesia is uniformly good. It is desirable that the systolic blood pressure should be over 80 mm.Hg. at the completion of surgery, and if it is below this level, a vasopressor can be given. Any subsequent fall in pressure is similarly treated, provided it is not due to haemorrhage or shock.

The striking feature in the post-operative period is the absence of restlessness. This is due to the continuing analgesia combined with rapid recovery of consciousness, and is in marked contrast to the state of affairs which often follows general anaesthesia (see p. 96).

With proper encouragement from the nursing staff and physiotherapists, early breathing and coughing exercises can be started during the period of analgesia, when the maximum respiratory effort can be made painlessly. The following case illustrates this point:-

A woman aged thirty years, suffering from bronchiectasis, was referred by the thoracic unit for sterilisation. She had had bilateral segmental resections of the lower lobes of her lungs, but bronchiectasis was still present with considerable sputum. The thoracic unit expressed the opinion that she should be bronchoscoped post-operatively to remove secretions. The operation was performed under epidural analgesia and light general anaesthesia with thiopentone and nitrous oxide. The patient awoke a few minutes after the nitrous oxide was discontinued, and was encouraged to cough. Several ounces of sputum were expectorated, and bronchoscopy was unnecessary.

Pre- /

Pre-operatively the importance of breathing exercises is explained to the patient and instruction is given on how to perform them.

Once the epidural blockade has worn off and vasomotor control re-established, no limitations need be placed upon movement or positioning of the patient that may be required in the surgical treatment. Early ambulation can be carried out. This is in contrast to the post-operative period following spinal anaesthesia when, to prevent post-spinal headache, the patient is kept flat, or in a slight Trendelenburg position, for 24 hours. Moreover, should this prophylaxis fail, the patient will be unable to assume the erect position without severe headache occurring for some days post-operatively.

FAILURES

There are two main reasons why attempted epidural blockade fails to produce adequate analgesia for the operative procedure.

A. Failure to place local anaesthetic within the epidural space.- In attempting to approach the epidural space through the sacral hiatus it is possible for the needle to pass superficially along the posterior surface of the sacrum. In so doing it may scrape along the bone, giving the impression that it is within the sacral canal. In thin subjects this error can be diagnosed when injection is made, for a lump will appear in the superficial tissues.

In /

In obese patients this can easily be missed. Passage of the needle through the sacral hiatus and the sacro-coccygeal joint to reach the anterior surface of the sacrum has been described, but must be extremely rare. Occasionally it is found to be impossible to enter the sacral hiatus and this may be due either to calcification of the sacro-coccygeal membrane or to a failure to identify the hiatus. There is considerable individual variation in the amount of fat covering the sacral hiatus and this does not always depend upon the obesity or otherwise of the patient. If the hiatus cannot be felt, the difficulty in entering the epidural space is obvious. While it cannot be denied that increasing experience of the technique leads to a decrease in the failure rate, there still remains a proportion of cases (probably 5-10 per cent.) where the sacral canal cannot be entered.

In the lumbar region a much higher success rate is to be expected. The commonest error is to push the needle too far and thus enter the sub-arachnoid space. Some authorities would not accept this as a failure and would attempt epidural tap at a different level (Lund, Cwik and Magaziner[6]). Current opinion is against this (see p. 99). If the sub-arachnoid space is tapped it is better to give a spinal anaesthetic or abandon the whole procedure in favour of a general anaesthetic. In the writer's series, dural puncture occurred six times in the first hundred cases but with /

with increasing experience and adoption of the use of the Tuohy needle, this error is now uncommon, certainly no more than 1-2 per cent.

Inability to pass the needle into the spinal canal at all is the only other cause of failure in the lumbar region. This is also the main cause for failure to perform spinal anaesthesia. Arthritis of the spine, calcification of the ligamenta flava or inadequate spinal flexion all militate against entry of the needle into the spinal canal. Epidural puncture is somewhat more difficult than sub-arachnoid puncture because the movement of the needle must be slower and under more control. As epidural blockade is performed on the conscious patient, the anaesthetist should not persist too long in attempting a successful puncture. If two interspaces have been tried without entering the epidural space it is advisable to abandon the procedure.

If cases are selected to exclude the obese and the elderly it would not be difficult to reduce the rate of failure to less than 1 per cent. Such selection, however, would be quite unwarranted. The only cases that should be excluded on the grounds that passage of the needle may be difficult, are those suffering from active disease, or gross deformity, of the spine.

B. Failure of local anaesthetic within the epidural space to produce adequate analgesia.- Inadequate analgesia can be due to:-

(i) /

(i) Insufficiently widespread analgesia. With the method of calculating the dose of local anaesthetic outlined on p. 80 such insufficiency is most unlikely in lumbar epidural blockade. Caudal analgesia, however, is often deficient in this respect if a blockade extending up into the lumbar and lower thoracic regions is required.

(ii) Inadequate analgesia from partially blocked nerves. This is due to the concentration of the local anaesthetic being too low to block all the sensory fibres. Using 1.25 per cent. lignocaine, insufficient analgesia was noticed on three occasions in the writer's series, but since adopting a 1.5 per cent. solution no further cases have occurred. Two other causes for this inadequacy have been described, both the result of rapid injection. This may not only spread the solution too "thinly", but it can also lead to rapid absorption into the blood stream before the nerves can be adequately blocked. Cases where toxic reactions occur often have little or no analgesia. By using a slow injection technique these difficulties are obviated.

In summary it may be said that, with caudal analgesia, failures are common but with lumbar epidural analgesia they are rare. Unfortunately more anaesthetists have had experience with the former than with the latter, and the inevitable failures thus encountered have prejudiced some against any form of epidural analgesia. Compared with spinal anaesthesia, lumbar epidural blockade is slightly more /

more liable to fail, though in the majority of failures (i.e. those due to dural puncture) it is a simple matter to give a spinal anaesthetic instead. In cases where entry into the spinal canal cannot be effected, similar difficulty (though not perhaps to the same degree) will be experienced in a planned spinal anaesthetic.

In the writer's opinion, too much emphasis has been placed upon the fact that occasionally the technique is unsuccessful. All cases where epidural analgesia cannot be produced are easily anaesthetised by other means. Epidural blockade offers both the patient and the surgeon advantages over conventional general anaesthesia, and if a few failures occur, nothing has been lost except time. Successful analgesia, which is achieved in the great majority of cases, more than compensates for the failures.

As in all techniques requiring manual dexterity, experience leads inevitably to an increasing success rate, and in the best hands this will be in excess of 98 per cent.

INDICATIONS

There are three main indications for the use of epidural analgesia: surgical, obstetrical and therapeutic.

A. Surgical

Epidural analgesia can be employed for any surgical procedure below the diaphragm. Some authorities recommend it /

it for thoracic operations, but as the patient must also receive controlled respiration, which entails deep anaesthesia or the use of relaxants, there would seem to be little value in adding regional analgesia. In the United States, cervical epidural blocks have been employed for such operations as thyroidectomy, but the risks involved in performing epidural puncture in this region outweigh the advantages. Upper abdominal operations which involve traction reflexes mediated through the vagus nerve, e.g. gastric operations, can lead to difficulties under epidural analgesia as vagal function is left intact. Traction upon the stomach or oesophagus is a well known cause of vomiting and retching during operations on conscious patients under spinal anaesthesia, and the same is true for epidural analgesia. Light concomitant general anaesthesia partially depresses these reflexes, but if they do occur, good operating conditions may quickly disappear. Deep general anaesthesia added to the epidural blockade will reduce these complications to a minimum, but this detracts from the advantage in using epidural analgesia.

Vagal nerve block by paraoesophageal infiltration can be carried out by the surgeon once the abdomen has been opened. Unfortunately, this can cause relaxation and dilatation of the gastro-intestinal tract, reversing the contracted state of the gut that accompanies epidural analgesia.

Epidural /

Epidural blockade is seen to best advantage in operations in the lower abdomen, for vagal stimulation is rarely illicited in this region. All abdominal gynaecological operations can be carried out under ideal conditions with epidural analgesia. Rectal operations, for example, abdomino-perineal resection, are indications for this type of anaesthesia, the hypotension produced being of particular benefit in radical surgery, where, under conventional general anaesthesia, excessive blood loss can occur. The author has given epidural analgesia for 10 prostatectomies and a striking difference was seen in the operative conditions compared with general anaesthesia. No case required blood transfusion either during or after operation. While this is a small number of cases, the impression of the superiority of epidural blockade for prostatectomy has been confirmed by larger series (Morris and Candy[7]; Gardner[8]). Hemicolectomy, partial or total cystectomy, and transplantation of the ureter are all indications for the method.

Outside the abdomen, the following operations can be performed with advantage, under epidural analgesia.

(i) Renal and adrenal operations such as nephrectomy, nephrolithotomy or adrenalectomy. Special care must be taken if the pleura is inadvertently opened (as sometimes occurs if the 12th rib is removed) and, in such circumstances, the respiration should be either assisted

or /

or controlled temporarily to prevent the lung from collapsing. The reduction in bleeding in these cases is again very noticeable.

(ii) Perineal operations including those on the vagina or vulva. The comparative bloodlessness obtained in Fothergill operations has been described (see p. 123). Radical vulvectomy can also be performed with the minimum of blood loss.

(iii) Removal of prolapsed intervertebral discs. The spontaneous respiration and complete relaxation of the abdominal muscles eliminate bleeding from the epidural veins provided the patient is properly positioned without pressure upon the abdomen and chest.

(iv) Major operations upon the hip joints and lower limbs.

The routine use of epidural blockade for less major operations such as appendicectomy, herniorrhaphy or haemorrhoidectomy is probably unwarranted. If it is desirable, however, for the patient to remain awake while such operations are carried out, this is the most suitable form of regional analgesia.

B. Obstetrical

Regional anaesthesia has the great virtue in operative obstetrics, that perfect operating conditions can be obtained without giving the mother, and therefore the foetus, any depressant drug. This can be of importance if the condition /

condition of the foetus is in any way precarious, e.g. in maternal diabetes, severe pre-eclampsia or foetal distress from any cause.

For Caesarean section, regional anaesthesia can be obtained by either spinal or epidural block on the one hand or local infiltration on the other. The writer has had some experience of local infiltration, by abdominal field block, for this operation and while it was possible to extract the foetus without much discomfort to the mother, subsequent manipulations invariably required a supplementary general anaesthetic. Spinal and epidural blockade produce essentially the same operating conditions. Spinal anaesthesia is more rapid in execution (and this may be of importance in foetal distress) while epidural analgesia is free from post-operative headache. With either method the anaesthetist must remember that a fall of systolic blood pressure below 80 mm.Hg. will not be tolerated by the foetus, as gaseous exchange across the placenta is greatly reduced below this level. Vasopressors are best administered routinely to prevent any fall.

For forceps delivery, pudendal nerve block is commonly used and is satisfactory if the foetal head is sufficiently low. If a more widespread analgesia is called for, epidural injection can be employed.

The control of pain in childbirth by means of regional analgesia has not found great support in this country, where /

where reliance is put upon analgesic drugs such as morphine and pethidine. While these drugs are very easy to administer, they seldom produce complete analgesia, and are prone to cause respiratory depression of the foetus. In the United States considerable use is made of epidural analgesia which is maintained by means of a plastic catheter inserted into the epidural space (Lull and Hingson[9]). The most popular route for insertion of the catheter is through the sacral hiatus, though the lumbar route has also been used, and, to the anaesthetist practiced in the method, it presents no great technical difficulties. The blockade can be re-established when it wears off by injecting further doses of local anaesthetic through the catheter. Parturition can be made quite painless in this way, and if operative delivery becomes necessary, no general anaesthetic need be given.

The last indication for epidural blockade in obstetrical practice is in the treatment of eclampsia. This condition is now fortunately a comparative rarity, because of improved ante-natal care. When it does occur, however, it can be extremely dangerous to both mother and foetus, especially the latter. The convulsions are due to cerebral oedema, and this can be combated either by lowering the blood pressure or administering a cerebral depressant such as bromethol which, incidentally, has a secondary effect of lowering the blood pressure. By means of its hypotensive action, /

action, epidural blockade causes a reduction in the cerebral oedema and improvement in the patient's condition. As it is unlikely that the disease will be cured until the foetus has been delivered, the earlier this can be effected the better for both foetus and mother. This will, of course, be dependent upon the maturity of the foetus, but if it is thought to be viable, induction of labour by pitocin drip can be started or Caesarean section undertaken. Under epidural analgesia operations can be carried out without the addition of any drug that could depress either the baby or the mother.

The writer has had occasion to employ epidural blockade in eclampsia on two occasions, and the results warrant a further trial.

Case 1

A primigravida, aged 32, had 12 days' inpatient treatment for pre-eclampsia at 35 weeks, and was re-admitted one week before the expected date of delivery with a blood pressure of 150/105 mm.Hg. After three days' rest a surgical induction was performed, but in spite of this, regular contractions were not established for 48 hours. After becoming four fingers dilated, the cervix did not dilate further, and the blood pressure rose steadily over the next 15 hours reaching 200/130, when two fits occurred. She was given morphine gr. $\frac{1}{4}$ and 100 ml. of 50 per cent. dextrose solution intravenously after the first fit, and the pressure fell to 170/110. Two hours later a lumbar epidural block was performed with 25 ml. of 1 per cent. lignocaine. Anaesthesia developed up to T10 and the pressure dropped to 100/60 when methedrine (10 mg. intramuscularly) was given to prevent any further fall. The uterine contractions had now almost ceased but the patient was greatly improved and quite co-operative. After one hour the blood pressure was 115/80 and an oxytocin drip was commenced with immediate re-starting of uterine contractions. The blood pressure finally settled at 150/110. In spite of good contractions the cervix still remained at four fingers. The anaesthesia wore off after two hours, but as the blood pressure did not rise, no further lignocaine was given. Four and a half hours /

hours after the start of the oxytocin drip the foetal heart ceased, and, as the labour had not progressed, the drip was stopped. A further eight hours passed before forceps delivery could be attempted. Under general anaesthesia a contraction ring was found to be present, and the dead foetus was extracted with difficulty. Six hours later the blood pressure was still 150/110. Recovery was uneventful. Caesarean section at the time when the epidural block was established might have saved the foetus.

Case 2

A primigravida, aged 25, was being delivered at home. Two and a half hours after rupture of the membranes a fit occurred and the blood pressure was found to be 160/110. Two hours later there was a further fit just following admission to hospital. Two hundred millilitres of 2.5 per cent. bromethol was given per rectum, but the pressure remained high at 180/140. A lumbar epidural block was performed with 30 ml. of 1.25 per cent. lignocaine, and the pressure fell steadily to 150/115. From being almost comatose the patient became reactive and answered questions. As the cervix was fully dilated forceps delivery was performed forthwith, no further anaesthesia being required. The baby cried immediately it was delivered, and was in good condition. Within one hour the mother's blood pressure was 130/90 where it remained.

C. Therapeutic

The use of regional anaesthesia in the treatment of certain conditions is well established. Epidural block can be used either for its analgesic effects or for the sympathetic paralysis it produces. The local pressure exerted by injecting saline into the epidural space can also be of benefit in post-spinal headache.

Epidural blockade is of value in the following conditions:-

(1) Acute pancreatitis.- While analgesic drugs can usually be given with good effect in this disease, they

may /

may not suffice even in large doses in some patients who continue to have considerable pain. By repeated injections of local anaesthetic through a plastic catheter inserted into the epidural space, complete analgesia without respiratory depression can be obtained. As the pain relief is due mainly to the sympathetic blockade the strength of the lignocaine need not exceed 1 per cent. The spinal segments concerned are the 7th, 8th and 9th thoracic (Orr and Warren[10]). A series of 15 cases so treated has been reported by Walker and Pemberton[11].

(2) Dissecting aortic aneurysm.- Pain associated with this condition can be of extreme severity and quite intractable, even with large doses of morphine. Epidural blockade, in addition to relieving the pain, will reduce the blood pressure and limit further spread of the aortic dissection (Bromage[12]).

(3) Painful disorders of extremities.- Causalgia and amputation stump neuralgias often respond to repeated paravertebral injections (White and Smithwick[13]). Continuous epidural block lasting three to five days would be technically easier, have the same effect, allow for more continuous analgesia and save the patient from repeated needling.

(4) Hypertensive cardiac failure.- In these cases, and reduction in the peripheral resistance, blood pressure and venous return, leads to a considerable lessening of the load /

load upon the heart. In this way pulmonary oedema is improved and breathlessness disappears. As hypoxia, resulting from pulmonary oedema, has adverse effects upon the myocardium, reduction of the oedema by epidural blockade can cut across the vicious circle and the benefit will outlast the blockade.

(5) Eclampsia.- The use of epidural block in the treatment of this condition has already been described (see p. 139).

(6) Headache following lumbar puncture and spinal anaesthesia.- Because of the lack of elastic fibres in the dura mater, holes made in it during lumbar puncture do not close readily. Thus the cerebrospinal fluid may pass into the epidural space. If this loss of fluid is great enough, the cushioning effect of the cerebrospinal fluid is lost and the brain tends to descend, causing headache and, in severe cases, ocular palsies due to stretching of the abducent nerves over the petrous temporal bone. The headache is quite characteristic, being severe if the patient assumes an upright posture and relieved by lying flat or head down. While it is common after spinal anaesthesia, it can occur after any lumbar puncture.

Injection of 30 to 50 ml. of normal saline into the epidural space raises the pressure in the space to above that in the sub-arachnoid space and further escape of cerebrospinal fluid is prevented. For reasons that are not quite /

quite clear, a single epidural injection can produce permanent cure of the headache, though in some cases repeated injections, through a catheter, are required for some 24 hours. Rice and Dabbs[14] described 22 cases treated in this way with good results in all but one patient. As the headache is primarily due to dural puncture, it behoves the anaesthetist to be sure that he does not re-puncture the dura when attempting the epidural injection.

CONTRA-INDICATIONS

(a) Spinal deformity or active disease of the spine.- Slight deformities do not prevent successful epidural puncture but in severe cases, the attempt to locate the space may be too prolonged to be justified.

Active disease such as tuberculosis of the spine and ankylosing spondylitis are best avoided as attempts to flex the spine may be very painful.

(b) Skin sepsis.- Skin sepsis near the proposed site of injection rules out epidural puncture.

(c) Hypovolaemia due to shock.- This is a contra-indication to any form of induced hypotension. In hypovolaemic shock, the blood pressure is being maintained by compensatory vaso-constriction, and interference with such compensation is fraught with danger. In these circumstances some anaesthetists have used epidural blockade, maintaining the blood pressure with a vasopressor such as nor-adrenaline (Bromage[15]). /

(Bromage[15]). The use of vasopressors, however, in the presence of hypovolaemia is not considered rational therapy, and shocked patients are probably better served by general anaesthesia.

(d) Neurological disease.- There is no evidence that epidural analgesia can or cannot influence the course of neurological conditions. The method is best avoided in such cases, as deterioration of the neurological condition will be attributed to the nerve block.

COMPARISON OF EPIDURAL ANALGESIA WITH GENERAL ANAESTHESIA

The whole concept of general anaesthesia was changed with the advent of the muscle relaxant drugs. These enable the anaesthetist to maintain a light plane of anaesthesia instead of the deep plane required when inhalational anaesthetics are used alone. Relaxation, only obtained previously by deepening the level of anaesthesia, often to dangerous levels, can now be obtained independent of the general anaesthetic agent. The change has brought new problems. Relaxant drugs, though providing satisfactory operating conditions, also paralyse the respiratory muscles, making assisted or controlled positive pressure respiration necessary. With increasing knowledge of respiratory physiology, it is now realised that, while the vast majority of cases suffer no adverse effects, this type of respiration can be a serious disadvantage if the patient's condition /

condition is poor, or if controlled hypotension is to be used, because positive intra-pulmonary pressure interferes with the venous return to the heart.

Compared with general anaesthesia, epidural analgesia offers the following advantages:-

(a) Relaxation of abdominal muscles is maximal and is obtained without any paralysis of respiratory muscles. Because the strength of the local anaesthetic solution used produces analgesia without complete motor paralysis, all the muscles of respiration, including the lower intercostals, work normally.

(b) The general anaesthesia used to supplement the epidural blockade need only be very light. Respiratory depression does not occur and post-operative recovery is very rapid. Unless very large doses of relaxant are used, general anaesthesia often requires the addition of an analgesic drug, such as pethidine, which, apart from depressing respiration, retards the recovery of consciousness. Moreover, it is a common sequel to general anaesthesia to have to reverse apnoea at the conclusion of surgery. This is usually quite simple, but can be time-consuming and often requires the administration of prostigmine, a drug not devoid of danger.

(c) Bleeding is less with epidural analgesia, whether or not hypotension occurs. The writer has shown this in the measurements of blood loss in pelvic floor repair operations /

operations previously described. In addition, there is a strong clinical impression of reduced blood loss in such operations as hysterectomy, prostatectomy, nephrectomy and abdomino-perineal resection of the rectum.

(d) If controlled hypotension is planned, it can be obtained under general anaesthesia with ganglionic blockers such as hexamethonium or trimetaphan. When this entails controlled respiration, the patient is in a potentially more precarious situation than when breathing spontaneously during epidural analgesia.

(e) Spontaneous respiration allows the anaesthetist freedom to move from the patient in order to deal with such things as intravenous drips.

(f) During epidural analgesia the gastro-intestinal tract is contracted as a result of the unopposed action of the vagus nerve. This facilitates exposure within the abdomen.

(g) For the reasons given on p. 96 , the post-operative state of patients after epidural analgesia is very superior to that seen after general anaesthesia.

COMPARISON OF EPIDURAL ANALGESIA WITH SPINAL ANAESTHESIA

The operating conditions produced by spinal and epidural blocks are almost identical. Each has its advantages and disadvantages and in the writer's considerable experience of both methods, they are not mutually exclusive, and for the best /

best results, both should be employed.

The advantages of epidural over spinal analgesia are:-

(1) Absence of post-spinal headache. This is probably the most important advantage. Although the use of fine needles and good technique can reduce the incidence of headache following spinal blockade, cases do still occur and even if not severe, they often prevent adequate post-operative mobility of the patient. Moreover, to prevent headache, spinal anaesthetic cases are kept flat or head down for 24 hours. Such restriction is unnecessary following epidural blockade.

(2) Neurological sequelae are rarer. While the causes of nerve damage following spinal anaesthesia are still not fully understood, with increasing knowledge they are much less common than formerly. An impressive series of over 10,000 cases of spinal blockade without any permanent neurological damage (all cases being followed up for five years) has been recorded by Vandam and Dripps.[16] However, it would be optimistic to say that the danger has been completely eliminated. As was mentioned on p.107 nervous lesions have followed epidural analgesia, but to judge by reports in the literature, these would appear to be much rarer than after spinal anaesthesia. Because of the extra coverings acquired by spinal nerves as they leave the sub-arachnoid space, this deduction is not surprising.

(3) Epidural analgesia need not paralyse muscles to the /

the same extent as spinal blockade, and, whereas lower intercostal paralysis is quite common with the latter method, it is not seen with the former.

The main advantage of spinal over epidural analgesia is that the height of the blockade is more predictable. This is of importance when inducing hypotension, especially in younger patients. The writer considers spinal block the technique of choice in cases of malignant neoplasm where radical surgery, such as Wertheim hysterectomy, is performed. In such cases, hypotension can be produced by spinal blockade more consistently than with epidural analgesia, and this gives such advantageous operating conditions that the risk of neurological incapacity is far outweighed by the more satisfactory and complete removal of the tumour. A large proportion of patients undergoing surgery for cancer succumb to the disease within five years, and their survival depends upon removal of all malignant tissue. If the excision is attempted in a welter of blood, the chances of it being complete are less than with a bloodless field. Dissection close to important structures, such as the ureter, is also easier and safer. In these circumstances, it is wrong to consider that the patient's best interests are served by the administration of a "safe" anaesthetic.

In less radical surgery, however, the neurological risk must be more seriously considered and because of the associated /

associated freedom from headache, epidural analgesia is more suitable than spinal anaesthesia in most cases.

References

(1) Lee, J.A. (1958). Personal communication.

(2) Odom, C.M. (1936). Amer. J. Surg., 34, 547.

(3) Massey Dawkins, C.J. (1945). Proc. roy. Soc. Med., 38, 298.

(4) Scott, D.B. (1958). Anaesthesia, 13, 19.

(5) Robson, J.M. and Keele, C.A. (1956). "Recent Advances in Pharmacology," p. 17. London: J. & A. Churchill Ltd.

(6) Lund, P.C., Cwik, J.C. and Magaziner, R. (1956). Anesthesiology, 17, 605.

(7) Morris, D.D.B. and Candy, J. (1957). Brit. J. Anaesth., 29, 376.

(8) Gardner, E.K. (1958). Anaesthesia, 13, 406.

(9) Lull, C.B. and Hingson, R.A. (1948). "Control of Pain in Childbirth," p. 211. Philadelphia: J.B. Lippincott Co.

(10) Orr, R.B. and Warren, K.W. (1950). Lahey Clin. Bull., 6, 204.

(11) Walker, T. and Pemberton, W.E. (1953). Anesthesiology, 14, 33.

(12) Bromage, P.R. (1954). "Lumbar Epidural Analgesia," p. 109. Edinburgh: E. & S. Livingstone Ltd.

(13) White, J.C. and Smithwick, R.H. (1944). "The Autonomic System," p. 229. London: Henry Kimpton.

(14) Rice, G.G. and Dabbs, C.H. (1950). Anesthesiology, 11, 17.

(15) Bromage, P.R. (1954). "Lumbar Epidural Analgesia," p. 106. Edinburgh: E. & S. Livingstone Ltd.

(16) Vandam, L.D. and Dripps, R.D. (1956). J. Amer. med. Ass., 161, 586.

VII. CONCLUSIONS

The initial prime object of this series of epidural blocks was to discover if the method had any value over more conventional anaesthetic procedures, and, if so, in what way it could be adopted into routine practice. It soon became obvious that lumbar epidural analgesia was the most effective type of epidural blockade and that it possessed several advantages over general or spinal anaesthesia. For its adoption into routine anaesthetic work, two difficulties had to be overcome. Firstly, it had to be reasonably dependable with a success rate in excessof 95 per cent. Secondly, the time taken for its performance had to be reduced to the minimum if the anaesthetist was to work single-handed. With practice, and perseverence with a single reliable method of identifying the epidural space, a high success rate is not difficult to obtain. Previous wide experience of lumbar puncture and spinal anaesthesia is of considerable assistance. Once confidence has been established, the anaesthetist can reduce the time the procedure takes by speeding up his movements and eliminating those which are not purposive.

Our knowledge of epidural blockade, though it has increased considerably in the 50 years it has been practised, is not yet complete. During the writer's series, several investigations have been undertaken and some problems have been /

been elucidated.

(a) By dissecting out a number of sacral canals, the anatomical reasons why caudal blockade is far less dependable than lumbar epidural analgesia have been made obvious. Inadequacy of caudal analgesia can be explained by the variations in curvature of the sacral canal, by the variable position of the posterior root ganglion and the difficulties met in inserting a needle into the canal in certain cases.

(b) The effect of hyaluronidase in the epidural space has been tested. Its main effect is to speed up the onset of analgesia, this being most marked in caudal blockade. Reasons are given for believing that this shortening of the time of onset of analgesia supports the theory that the main effect of epidurally placed local anaesthetics is upon the spinal nerves extradurally and not intradurally.

(c) The efficiency of the new local anaesthetic drug carbocaine has been tested and found to be satisfactory.

(d) The hypotensive effects of lumbar epidural analgesia have been measured and compared with caudal blockade and with general anaesthesia. Reasons have been advanced why hypotension in epidural and spinal blockade is considerably safer than by any other method.

(e) An objective inquiry into operative blood loss has been carried out and the clinical impression that epidural analgesia reduces bleeding has been confirmed.

(f) The generalised effects of the local infiltration of adrenaline have been studied and reasons given for the belief /

belief that in proper amounts, such infiltration is a safe procedure under general anaesthesia.

Other problems remain, the chief amongst which concerns the absorption of the local anaesthetic into the sub-arachnoid space and into the blood stream. To inquire into these questions requires the detection and measurement of minute amounts of local anaesthetic. To do this chemically is not only tedious but liable to error and it is probable that the use of radioactive isotopes would not only be simpler but very much more accurate. It is a simple problem to label a local anaesthetic with a radioactive tracer, very small amounts of which can be detected with great accuracy. With regard to the absorption into the blood stream, the effects of adding adrenaline to the injected solution could be much better assessed than hitherto.

In the past, the major benefit of a new anaesthetic agent or technique was measured by a lowered mortality following its use. Nowadays, anaesthetic mortality is not accepted as inevitable, and we must look elsewhere to judge the good effects of anaesthetic methods. It may be felt that the extra time and skill that must be put into epidural analgesia are not worth the benefits gained. In the writer's opinion, however, every anaesthetist should be familiar with the method for it has a wide range of usefulness both surgical, obstetrical and therapeutic. An anaesthetic procedure is none the worse because it requires some extra skill both in performance and management.