

NEUROLOGICAL COMPLICATIONS OF SPINAL ANAESTHESIA*

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A GREAT DEAL has been written in the literature and presented in discussions at meetings concerning the neurological complications of spinal anaesthesia. Many investigations of clinical and experimental nature have been undertaken in order to discover the true perspective in the incidence of morbidity and mortality. The basis of this debate lies in the incidence and severity of the neurological complications. These may be described under the headings of Immediate and Subsequent.

The *immediate* neurological complications concern the excessive height to which the anaesthetic solution might rise, thereby producing undesired paralysis of nerves and nerve centres. This complication is mentioned as it has produced a fatal outcome in the hands of the unalert. It has been known to cause a worse result, namely irreversible anoxic changes in the brain in patients who have otherwise recovered. Amongst 77 fatal cases of cerebral anoxia studied by Courville (1) no less than 12 were due to spinal anaesthesia.

The *subsequent* possible complications are known to all; indeed some have been in the unpleasant position of being associated with one or more of the serious types.

POST-SPINAL HEADACHE

Etiology

It is now well established that the common type of post-spinal headache is caused by leakage of cerebrospinal fluid which creates low pressure within this system. The altered dynamics cause "sagging" of the architecture of the brain, the consequent tension on the sensitive parts of the brain producing this headache. McNaughton (2) has shown that the sensory nerve supply of structures above the tentorium is the fifth while the posterior fossa is supplied by the ninth, tenth and upper cervical nerves.

Kreuger (3) quotes Sicard for having put forward the suggestion of leakage in 1902. This theory has been followed by others (4, 5, 6). Thorsen (7) points out that with 84 per cent of all post-spinal headaches there is hypotension of the cerebrospinal fluid.

Kunkle (8) and others have shown that the loss of 20 ml. of cerebrospinal fluid is enough to lower the intracranial pressure; this fall is more marked when the posture is erect.

Additional Factors

Other causative factors have been well put forth by Kreuger, Stoetling and Graff (9) who describe the importance of dehydration, trauma, size of needle

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and apprehension, in the incidence of post-spinal headaches. The actual anaesthetic agent introduced does not appear to be of statistical importance; Bonica (10) states that the addition of vasoconstrictors to the solution bears no relationship to this occurrence.

As well as the factors enumerated above, McCord (11) and others feel that cervical cord pathology may be of significance and that meningeal irritation should be considered. This latter item raises a different etiological factor which will be mentioned later.

TABLE I
INCIDENCE OF POST-SPINAL HEADACHE

| Investigation | No of cases | Details | Frequency |
|--------------------------|-------------|--|---------------|
| Brady (13) | 1148 | No 22 needle Cont Spinal No 3½ catheter ¹ | 3 9% 6% |
| McCord (11) <i>et al</i> | | Vag delivery | 14-18% |
| (Cases quoted) | | Various series | 1-32 8% |
| Kreuger (3) | | "Ordinary needle" No hydration | 21 7% |
| Kruger (3) | | Average all types | 10 8% |
| Kreuger (3) | | Whitacre needle | 4% |
| Kreuger (3) | | Apprehensive patients | 14 3% |
| McCarthy (14) | | Over-all | 15% |
| Harris (15) | | No. 20 24 needle | $\frac{2}{1}$ |
| | | Catheter technique | 23 8% |
| Gilbert (15) | 912 | Over-all | 2 5% |

Many of the quoted authorities point out the higher incidence of headaches in obstetrical cases and are agreed concerning the reasons for this fact: relative dehydration, early sitting up, and the effects of pressure changes due to the abdominal tumour. To these may be added the dissimilarity to surgical patients who usually lie longer in bed and receive more anodynes than does the average obstetrical charge.

For a comprehensive chart concerning the incidence of headache, showing larger numbers of cases, reference may be made to that produced by Pitkin (12).

Prophylaxis

Kreuger (3) has quoted Antoni as having described the importance of a small needle in 1923. Associated with this has been the assertion that a needle should separate the fibres of the arachnoid rather than cut them; hence other modifications have been described in the form of the needle. By these means the finest of holes is made which may readily be sealed off.

Green (17) and others have stressed the importance of preventing a headache by efficient hydration and by making use of the antidiuretic property of posterior pituitary extract as well as of a small gauge needle.

Kaplan (18) has suggested and carried out the procedure whereby 10–20 ml. normal saline is introduced (epidurally) as the spinal needle is withdrawn.

Treatment

Nursing in the supine position and treatment by aspirin combinations have, in the past, cured the majority of headaches. Are there other measures which should be undertaken in the early stages? What should be done in the case of severe persistent headaches? These are questions which should be considered if spinal anaesthesia is still to be used.

Weed and McKibben (19) described the changes which occurred in the pressure of cerebrospinal fluid following intravenous injections of varying concentration. This concept has led to the intravenous administration of 5 per cent dextrose or 2.5 per cent dextrose in .45 per cent saline. To this solution has been added 100 mgm. nicotinic acid (3).

Brady (13) recommends Empirin and nicotinic acid. McCarthy (14) advises caffeine sodium benzoate. McCord (11), Ahearn (6), Wolff (4) and Rice (20) all advocate, as do many others, the intrathecal or epidural injection of saline 70–100 ml, given to treat a severe headache, will cure all but 20 per cent. This group will respond to a second treatment. A modification of this has been advocated at the Montreal Neurological Institute when an artificial C.S.F. produced by Dr. K. A. C. Elliott is used intrathecally. This has a physiological rationale to recommend it (21). Glesne (22) recommends the use of intrathecal glucose, with which 50 per cent success is claimed.

Deutsch (23) reports the treatment of severe post-spinal headache by intravenous ethanol. One litre of 5 per cent alcohol in 5 per cent glucose is given. This appears a rational form of treatment. The alcohol is an analgesic and provides euphoria, it gives rise to dilatation of pial vessels (24), it aids sodium retention (25) and causes a shift of water from the intracellular to the extracellular compartment (26)

Other types of headache

There remain a small percentage of headaches which are not of the previously described type. They are usually associated with a high cerebrospinal fluid pressure and a high C.S.F. cell count. The symptom usually suggests meningismus rather than the frontal or occipital headache of the low pressure type of syndrome. This type of headache is not relieved by posture. The occurrence of this pain calls for close observation and investigation, following which specific treatment may be indicated. Apart from this, treatment is symptomatic.

CRANIAL NERVE LESIONS

These may occur following spinal anaesthesia owing to different etiological factors. First, and most commonly, they may be produced by alterations in the architecture of structures within the skull brought about by altered hydrodynamics whereby the nerves become stretched. Secondly, they may be brought on by a meningitic phenomenon either septic or aseptic. Thirdly, they may be the outcome of widespread adhesive changes such as will be described later. Kennedy and Lockhart (27) have enlarged upon these etiological factors.

The first and common type, that of the sixth nerve, occurs with varying frequency. Some report large series with scarcely a case while others report its occurrence with apparent frequency. The fortunate fact concerning this diplopia-producing complication is that it nearly always rectifies itself in time. For this reason there should be no undue haste in seeking ophthalmic surgeons to perform muscle operations.

Though of much rarer occurrence, paralysis of the other cranial nerves has been reported (7).

Other Nerve Palsies

Isolated nerve palsies, usually reversible, may occur in relation to the cauda equina—a typical example is that which gives rise to foot drop. Other sensory or motor nerves may be temporarily or more rarely, permanently, involved.

The other types are of much graver consequence and are symptoms from other clinical pictures.

ARACHNOIDITIS, RADICULITIS, NEURITIS, MYELITIS, CAUDA EQUINA SYNDROME
AND THE NEUROGENIC BLADDER

These form a group of complications of the utmost severity. They are far more grave than those complications which might be expected to follow general anaesthesia. It is complications as these especially which have given rise to such controversy (28, 7, 29). Two such cases have been admitted to the Montreal Neurological Institute during the last four years.

Pathology

These cases may present a variety of lesions, some or all of which are found. By the time some cases reach autopsy, fibrosis and degeneration are the most prominent features.

Nerve roots present early degeneration of the medullated fibres followed by complete degeneration.

The dura, arachnoid and pia become thickened and adherent. Eventually they fuse one with the other.

Myelitis of static or ascending nature may be found.

In some cases there has been reported involvement of meninges and cranial nerves, also evidence of encephalitis.

A case was seen recently (30) in which the patient never recovered from the effects of a spinal anaesthetic with 1/1500 Nupercaine given for gastrectomy. A standard technique was used. Following this procedure myelography determined

a spinal block from T₅-L₂. Laminectomy was performed within a few days. At this operation, the portion of the cord involved appeared grossly congested and swollen. The appearance suggested infarction. The decompression has not yet resulted in recovery from this paraplegia.

Investigations

McCarthy (14) quotes but gives no reference to Davies as having produced experimentally changes in the meninges, ganglion cells and nerve fibres which are similar to changes found in cases which have had fatal outcome, owing to neurological damage.

It has been considered that in humans a number of cases may develop early but reversible changes. This is an unknown factor.

Sterilization solutions and green soap have been injected in larger than clinical doses into the subarachnoid space of dogs (29); all have recovered.

Clinical Picture

The onset of symptoms may be immediate or of gradual development during the course of days or weeks. The progress may become arrested, resulting in partial or complete paraplegia. The condition more commonly, however, progresses, either rapidly or slowly, to a fatal termination.

Causation

There are a number of possible causes:

1. The *drug* itself may have specific affinity for a specific spinal cord or it may be in a too high concentration in a particular case. In either of these cases an irritant process is initiated leading to reversible or irreversible changes.
2. There may be direct injury to nerve, cord or blood supply. The conus medullaris may be injured by too high a puncture. A recent case may be cited (31) in which a cauda equina syndrome followed rapidly a spinal anaesthetic. The site of lumbar puncture could still be seen, a silver clip was fixed thereon and a film was taken. The X-ray showed the puncture was made between T₁₂ and L₁.
3. There may be chemical contamination of the anaesthetic solution injected.
4. There may be a low-grade infection, either exogenous or endogenous
5. There may be coexisting disease of the central nervous system.

Concerning the latter possibility, a personal case can be mentioned in which a patient, who had previously suffered from infantile paralysis, was anaesthetized for an operation on the knee joint. Subsequently, that patient appeared to have paraplegia. The patient was removed to the neurological department of another hospital with the diagnosis, spinal complication. In actual fact, the patient had received a general anaesthetic, as her chart bore witness. The diagnosis was then altered to one of hysteria.

LESS COMMON COMPLICATIONS

Meningitis

Little need be said concerning this dreadful and entirely avoidable complication.

Paravertebral Block

A paravertebral block with pontocaine has been reported (32) as causing radiculitis. Elocaine injected paravertebrally (33, 34) has caused paraplegia.

Herniated Intervertebral Disc

This is not a common sequela but there is convincing evidence that undue trauma at lumbar puncture may, by piercing the annulus fibrosus, precipitate the herniation of a Nucleus Pulposus. It may be that that disc is already the site of degenerative changes.

PROS AND CONS OF SPINAL ANAESTHESIA

There are ardent protagonists of spinal anaesthesia such as Cole (35). There are other men who cannot condemn the method too strongly.

The arena is also entered by our surgical and obstetrical colleagues, some of whom maintain that they will only operate under spinal anaesthesia while others refuse to allow their patients to be subjected to such a form of anaesthesia.

If spinal anaesthesia is to be used, the greatest care must be exercised in the preparation of the equipment, infinite dexterity and sterility must govern the procedure, prophylactic measures should be taken to ensure a minimum of even the more minor complications.

My opinion remains the same as when written in 1952 (36): "In spite of the crippling conditions inevitably seen at the big Neurological Centres, the general impression remains that these conditions are in reality rare and there is no need to abandon a sound form of anaesthesia WHEN INDICATIONS ARISE FOR ITS USE."

RÉSUMÉ

On a beaucoup écrit et discuté dans les conférences médicales sur les complications neurologiques de la rachi-anesthésie. La base de ces discussions réside dans l'incidence et la gravité de ces complications.

Les complications neurologiques immédiates sont en relation avec une élévation trop marquée de la solution anesthésique, qui produit une paralysie imprévue des nerfs et centres nerveux. Cette complication peut entraîner la mort ou des altérations irréversibles du cerveau dues à l'anoxie.

Parmi les complications ultérieures de la rachi-anesthésie, les céphalées sont les plus courantes. On a bien établi aujourd'hui que le type courant de céphalée post-rachi-anesthésique est provoqué par une perte du liquide céphalo-rachidien, qui déclenche une baisse de pression à l'intérieur du système. D'autres facteurs importants sont la déshydratation, les traumatismes, la dimension de l'aiguille et l'appréhension. L'agent anesthésique lui-même ne paraît pas revêtir d'importance statistique. Pour prévenir l'apparition de céphalées, on recommande les mesures suivantes: emploi d'une petite aiguille, hydratation efficace, emploi des propriétés anti-diurétiques de l'extrait post-hypophysaire, et injection de soluté physiologique normal dans l'espace épidual quand on retire l'aiguille.

Dans le passé, on a pallié à la plupart des céphalées consécutives à la rachi-anesthésie en couchant le malade en position dorsale, et par l'administration

de composés d'aspirine. Les céphalées plus sérieuses et persistantes ont été traitées par des injections intraveineuses d'une solution à 5 pour cent de dextrose, pouvant contenir de l'acide nicotinique, par l'"Empirine" et l'acide nicotinique; par la caféine et le benzoate de sodium; par des injections intra-rachidiennes ou épidurales de soluté physiologique normal (70-100 ml) ou de liquide céphalo-rachidien artificiel ou de solution de glucose, et par des injections intraveineuses d'éthanol.

D'autres types de céphalées post-rachi-anesthésiques peuvent être associées à des signes méningés, à une hypotension et à une présence élevée de cellules dans le liquide céphalo-rachidien.

Les lésions des nerfs craniens qui suivent une rachi-anesthésie peuvent être dues à une altération des réactions intra-cervicales ou à un phénomène méningé septique ou aseptique. Le nerf le plus couramment affecté est le sixième nerf crânien. On a signalé la paralysie d'autres nerfs craniens. Heureusement cette complication se résout toujours d'elle-même avec le temps.

L'Arachnoidite, la névrite radiculaire, la Névrite, la Myélite, le syndrome Cauda Equina et la Vessie neurogène composent un groupe de complications extrêmement sérieuses. L'apparition des symptômes peut être immédiate ou se développer peu à peu en quelques jours ou en quelques semaines. Parmi les causes possibles, citons:

1. L'irritation due à l'agent anesthésique rachidien lui-même
2. La lésion directe d'un nerf, de la moelle épinière, ou des vaisseaux sanguins
3. La contamination chimique de la solution anesthésique
4. L'infection mineure, exogène ou endogène
5. La présence simultanée d'une maladie du système nerveux central

L'auteur est d'avis "qu'en dépit des infirmités que l'on rencontre inévitablement dans les grands centres neurologiques, l'impression générale demeure que ces cas sont en réalité rares et qu'ils ne doivent pas entraîner l'abandon d'une forme d'anesthésie qui reste valable *quand son emploi est indiqué.*"

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