USE OF SPINAL ANALGESIA IN FORCEPS AND BREECH DELIVERIES

BY

R. T. SEARS, M.A., M.B., F.R.C.S., M.R.C.O.G.

Formerly Registrar, the City Hospital, Nottingham

For many years general anaesthesia has been the anaesthetic of choice for most operative obstetric deliveries, but recent awareness of the danger of inhalation of vomit (Jeffcoate, 1953; Parker, 1954, 1956) has caused concern. The extent of the problem is revealed by reference to the Report on Confidential Inquiries into Maternal Deaths in England and Wales, 1952-4, which shows that inhalation of vomit was responsible for no fewer than 32 of the 49 maternal deaths due to anaesthesia. Recognition of this danger has led, on the one hand, to an increase in the use of local analgesia, and, on the other, to greater precautions when general anaesthesia is administered.

Local analgesia (pudendal block) is undoubtedly safe provided care is taken to avoid overdose; mishaps have been recorded when concentrated solutions have been used (Gordon, 1955; Martin, 1955; Barnes, 1955; Rollason, 1955). Unfortunately, while local analgesia is suitable for most forceps and breech deliveries, it is inadequate for those in which there is difficulty. In order to extend the limits the use of pre-operative intravenous chlorpromazine and pethidine has been advocated. Scott and Gadd (1957) gave 100 mg. of pethidine and 12.5 mg. of chlorpromazine in this way and were able to perform 94% of their forceps deliveries under local analgesia. But this technique may introduce fresh hazards, for there is a risk of apnoea when pethidine is given intravenously in this dosage to a woman who has already had analgesics earlier in labour. The development of such apnoea may be delayed and appear at a time when the patient is unattended.

Although the safety of general anaesthesia has been increased by endeavours to reduce both the incidence of vomit and the risk of inhalation, complete success may not be achieved. The use of apomorphine (J. M. Holmes, 1956) or routine aspiration of the stomach pre-operatively is unpleasant and may be rendered ineffective by subsequent duodenal regurgitation; nor is the provision of tilting labour-ward beds and adequate suction apparatus a complete safeguard. Much depends upon obtaining the services of an anaesthetist skilled in obstetrical anaesthesia, and this is not always possible in the sudden emergencies of obstetric practice.

At a discussion at the Royal Society of Medicine (*Proc. roy. Soc. Med.*, 1957) both the dangers of general anaesthesia and the limitations of local analgesia were appreciated. Little reference was made to the place of spinal analgesia, which has neither the limitations of local analgesia nor the disadvantages of general anaesthesia. For some 25 years it has been the practice in Nottingham to employ spinal analgesia for the majority of caesarean sections, and for the past 20 years it has been used extensively for operative vaginal deliveries. There have been no deaths and no serious complications due to spinal analgesia in the three Nottingham Hospitals admitting maternity cases during this period (H. J. Malkin, 1958, personal communication). I have therefore been encouraged to present the results obtained

during the period 1951-5 in 439 cases delivered by the vaginal route at the City Hospital, Nottingham, under spinal analgesia.

Present Series

During the period 1951-5 there were 8,395 deliveries in the City Hospital. These included 391 forceps and 97 breech deliveries requiring anaesthesia. Spinal analgesia was given in preference to other methods of anaesthesia and was used for 371 (95%) forceps deliveries and 68 (70%) breech deliveries. A conservative policy has been practised in the use of forceps, which are rarely employed before one and a half to two hours in the second stage of labour unless there is foetal distress or other abnormality. Of the forceps deliveries 180 were classed as low forceps, 186 as mid-cavity (122 requiring rotation of the foetal head), and 5 as high forceps deliveries.

There were no maternal deaths and, headache excepted, few maternal complications, none of which were serious. The results to the baby were also satisfactory. A "saddle block" technique has been used, 0.8 ml. of 0.5% hyperbaric cinchocaine hydrochloride ("nupercaine") being given. This causes complete loss of sensation in the lower abdomen, perineum, and thighs. Sensation from the uterine cavity is greatly diminished; thus difficult forceps and breech deliveries requiring intrauterine manipulations can be performed without causing distress to the patient. There is complete perineal relaxation, which facilitates delivery and the performance of manipulations such as correction of the malrotated head in forceps deliveries and the bringing down of arms in breech cases.

The conscious patient is able to assist delivery by use of her abdominal muscles; this is valuable in assisted breech deliveries. Nevertheless there is considerable loss of power and the perineal reflex is lost. It is therefore advisable to give the injection as late as possible in breech cases to avoid unnecessary extraction.

Since uterine tone is retained under spinal analgesia the risk of post-partum haemorrhage is reduced, though intravenous ergometrine (0.25 mg.) has been given with delivery of the anterior shoulder in forceps cases and on completion of the second stage in breech deliveries. Post-partum haemorrhage (loss of 20 oz. (570 ml.) or more) occurred in 32 forceps deliveries and one breech (incidence 7.5%), but in 11 cases bleeding was thought to have arisen from cervical or vaginal tears. Manual removal of the placenta was performed in 13 patients (incidence 3%); no undue difficulty was experienced.

Forceps delivery was difficult in five patients who had mitral stenosis, and in three who had chronic bronchitis. Saddle block analgesia was considered especially suitable in these cases.

Reactions of Patient.—Mothers interviewed after delivery under spinal analgesia seem well satisfied by their experience. Patients entering hospital know that spinal analgesia is often used, but no persuasion is attempted if they wish to have another anaesthetic. There was only one refusal in the year 1955; this woman had suffered from headache for two weeks following a diagnostic lumbar puncture some years previously. The placid manner of patients delivered under spinal analgesia is in contrast to the restlessness which is frequently present during delivery under pudendal block.

Contraindications.—Only one patient was encountered in whom spinal analgesia was contraindicated. She was a primigravida of 39 with a major degree of

kyphoscoliosis affecting the lumbar region. She was successfully delivered by forceps under general anaesthesia.

Failures.—Spinal analgesia proved unsuccessful in 13 cases: in six the subarachnoid space was not entered; in seven cerebrospinal fluid was seen to flow, but there was no loss of sensation after the injection was given.

Technique

Great care is taken in the preparation of materials used in the administration of spinal analgesia. Syringes and needles are cleansed, washed in water, and dried to ensure that they are free from extraneous chemicals likely to damage nervous tissue. Syringes, needles, and ampoules of analgesic agents are assembled in a special pack, which is autoclaved in a drum. The contents of the pack comprise: One 5-ml. syringe; one 2-ml. syringe; three lumbar puncture needles (20 S.W.G.); one file for ampoule; one 20-mm. needle; one 16-mm. needle; one ampoule containing 1-ml. of 1% procaine hydrochloride; one ampoule containing 3 ml. of 0.5% cinchocaine hydrochloride in 6% glucose.

The analgesic is given with the patient in either the left lateral or the sitting position, according to the preference of the anaesthetist or the comfort of the patient. If the left lateral is used the patient is inclined on pillows, feet downwards, to limit upward spread of the hyperbaric fluid after its injection. The skin is painted with 0.1% thiomersal, which is allowed to dry to prevent contamination of the lumen of the lumbar puncture needle. Procaine hydrochloride, 1 ml., is used to anaesthetize the skin and subcutaneous tissues of the interspinous space between the third and fourth lumbar vertebrae, and lumbar puncture is performed through this space. After a few drops of clear cerebrospinal fluid have flowed, 0.8 ml. of 0.5% hyperbaric cinchocaine is injected slowly.

Immediately after the injection has been given the patient is sat up in order to limit upward spread of the solution. Two to three minutes later the cinchocaine becomes fixed, and the lithotomy position can be adopted. Sensation is lost within five minutes. The patient is nursed flat for 12 hours and then gradually allowed to sit up.

Foetal Results

The number of cases and the cause of foetal loss in the forceps and breech deliveries are shown in the Table. There were eight foetal deaths which could reasonably be attributed to birth injury among the 371 forceps deliveries, but none was considered to have been caused by analgesia. Similarly the three deaths due to birth injury amongst the 68 breech deliveries were also

unrelated to spinal analgesia. The deaths of three babies (one forceps and two breech) were unexplained.

There was a noteworthy absence of respiratory difficulty in the surviving babies. Asphyxia livida was present at birth in 20 babies and asphyxia pallida in 7. Factors other than the analgesic—for example, foetal distress *in utero*, difficulty in delivery—were present in 18 of the 27 cases, and may have been responsible for the respiratory difficulty.

Maternal Complications

There have been few complications in this series and none have been serious. But since complications, though rare, constitute such a strong deterrent to the use of spinal analgesia, mishaps which have not occurred in this series are discussed at some length. Evidence is presented that they should be almost eliminated in carefully administered "saddle block" analgesia.

Spinal Shock

There were 10 cases in which the post-partum blood pressure fell to below 100 systolic, but post-partum haemorrhage and trauma to soft parts were thought to be responsible. Each case responded readily to intravenous therapy. There were no cases in which shock could be attributed to the analgesia itself. Nevertheless spinal shock has been responsible for maternal deaths; Klein *et al.* (1953) and Lock and Greiss (1955) report 21 maternal deaths from this cause, but in 17 of these cases caesarean section had been performed and a relatively large dose given.

Spinal shock is a condition of circulatory and respiratory failure probably caused by peripheral vasodilatation and paralysis of respiratory muscles. Respiratory failure is especially apt to occur in the parturient woman because the diaphragm is splinted by the full-term pregnant uterus, and paralysis of the thoracic region of the spinal cord is therefore sufficient to cause respiratory embarrassment. F. Holmes (1957) thinks there is evidence of vena caval obstruction by the uterus in late pregnancy, causing a reduction in venous return to the heart. When spinal analgesia is given, venous return is still further reduced by peripheral vasodilatation. The combination of these two factors he regards as responsible for the special susceptibility to spinal analgesia which some have supposed to exist in the pregnant woman. The risk of spinal shock should therefore be least where peripheral vasodilatation and muscular paralysis are limited to the lumbo-sacral region, as in the saddle block technique using low dosage and a hyperbaric solution.

In order to test the effect on blood pressure of the spinal analgesic administered in cases of forceps delivery a study was made of the records of 68 uncomplicated cases where systolic blood pressure was noted before and shortly after delivery. The blood pressure was unchanged in 22 cases, rose in 20, and fell in 26 cases. In six the fall was as much as 30–40 mm. Hg, but none fell below 100 mm. systolic. These results do not suggest any consistent effect upon blood pressure.

It might also be argued that, although saddle block analgesia does not cause a consistent fall in blood pressure, there may be sufficient effect upon the cardiovascular system to lower the threshold level at which shock develops after blood loss or trauma. As stated above, there were 10 cases in which the blood pressure fell to below 100 systolic following haemorrhage or trauma, but, on the other hand, post-partum

Number of Cases and Cause of Foetal Loss in Forceps and Breech Deliveries, City Hospital, Nottingham, 1951–5

Stillbirths		Neonatal Deaths	
<i>Forceps Delivery (371)</i>			
Intrauterine foetal death ..	6	Hydrocephalus	1
Congenital abnormalities ..	2	Intracranial haemorrhage ..	1
Hydrops foetalis	1	Cause unknown	1
Intracranial haemorrhage ..	3		
Asphyxia (difficulty in delivery)	4		
Total	16	Total	3
<i>Breech Delivery (68)</i>			
Hydrocephalus ..	2	Hydrocephalus	1
Asphyxia (difficulty in delivery)	1	Intracranial haemorrhage ..	2
Cause unknown	1	Cause unknown	1
Total	4	Total	4

haemorrhages of 30 oz. (850 ml.) or more were sustained in nine patients without signs of shock appearing. There seems no reason to conclude that there is serious lowering of the threshold level at which signs of shock appear.

Meningitis; Headache; Ocular Nerve Palsy

Meningitis.—No case of meningitis has occurred in this series and there has been none in Nottingham since 1939. Meningitis should be avoided by careful technique. It is mentioned in few modern series, though one maternal death is recorded both by Klein *et al.* and by Lock and Greiss; both series cover years when antibiotics were limited.

Headache.—This was a common complication, and 62 (14.1%) patients complained of it. It was mild and did not persist for more than three days in 41 patients, but lasted five days in 16, and persisted for two to three weeks in 5 patients. The headache was throbbing in character and was felt both behind the eyes and over the occiput. It was aggravated by sitting up and was usually relieved by lying prone. Headache was treated by a variety of analgesics, nicotinic acid, and 30% glucose given intravenously. None of these measures was uniformly successful, and the strict maintenance of the prone position appears to be the most effective remedy. A 5–10% incidence of headache following spinal analgesia seems common to most series.

Ocular Nerve Palsy.—One patient, who had severe headache, also had blurring of vision and palsy of the external rectus muscle which persisted for three days. Paralysis of the abducent nerve, diplopia, and transient disturbance of the cerebral cortex have been reported by Macdonald Critchley (1937), Kennedy *et al.* (1950), and Vandam and Dripps (1956). It is probable that these disorders are caused by changes in intracranial tension due to loss of cerebrospinal fluid at the site of lumbar puncture, for they are known to follow diagnostic lumbar puncture (Dattner and Thomas, 1941; Robinson, 1945) and are reversible.

Disturbance of Spinal Cord

Disturbance of the spinal cord is the complication of spinal analgesia which is most feared, since it can reduce a healthy young adult to a helpless cripple, with paralysis of the legs and loss of control of bladder and rectum.

Only one patient developed any disturbance of locomotion; she underwent a difficult rotation of the foetal head and Kielland forceps delivery for foetal distress. A left peroneal palsy developed, and was still present four months after delivery. There was no interference with function of either the bladder or the rectum. Peroneal palsy is a well-recognized complication of forceps delivery, usually attributed to pressure of the foetal head upon the lumbo-sacral nerve, and was probably unrelated to the spinal analgesic given in this case.

Retention of urine occurred in 39 cases (8.9%). Thirty-six were mild and responded to treatment within 48 hours, but in three it persisted for three to seven days. Temporary and partial incontinence of urine, persisting for one week, was observed in two patients; in neither was there retention or infection. Retention of urine is a common complication of forceps delivery conducted under any anaesthesia, and the incidence in this series is not unreasonable. It is possible that incontinence was due to temporary damage to the cauda equina, though such damage usually causes retention of urine.

There were no cases of more serious neurological disorders such as those described by Kennedy *et al.* (1950). Degeneration of the cauda equina and lower levels of the spinal cord (cauda equina syndrome), and chronic adhesive arachnoiditis, are the commonest of these. It is difficult to assess the incidence of these complications since reports are so varied. The clinical picture is described by Macdonald Critchley (1937) and Ferguson and Watkins (1938). In cauda equina syndrome there is loss of sensation over the sacrum and coccyx, anal incontinence, and retention of urine. In severe cases, where damage is more widespread, the legs are involved; there is muscular weakness and serious interference with walking. Damage is usually maximal at the onset and may improve a little in time, but a case of progressive damage leading to death is recorded by Brain and Russell (1937).

Chronic adhesive arachnoiditis produces similar symptoms but arising weeks or months after administration of the analgesic. In this condition damage is produced by ischaemia, and may progress until all the lower segments of the spinal cord are affected. Rosenbaum *et al.* (1952) have emphasized that symptoms may not arise until 6 to 12 months after administration of the analgesic, and may therefore be overlooked in a short follow-up.

The aetiology of both cauda equina syndrome and adhesive arachnoiditis is uncertain. Trauma, infection, chemical impurities, and sensitivity to the analgesic agent have all been suggested. From considerations of the time interval between administration of the analgesic and development of paresis, it is clear that cauda equina syndrome is due to damage sustained at the time of injection, whereas adhesive arachnoiditis is a chronic inflammatory reaction in the meninges. Since cauda equina syndrome does not follow diagnostic lumbar puncture, it is unlikely to be due to damage to the cauda equina by the lumbar puncture needle.

Weston Hurst (1955) has shown that a variety of chemical substances, including antiseptics and detergents, can cause both immediate degeneration of nervous tissue and chronic arachnoiditis when injected into the subarachnoid space of rhesus monkeys. Damage may be done with high dilutions of the substances. In a recent judgment given in the High Court (*The Times*, 1953), it was considered that paralysis following spinal analgesia was caused by contamination of the anaesthetic drug by passage of phenol through minute cracks in the ampoule. Such events may have passed unrecognized in the past. The risk should be avoided by care in the manufacture of anaesthetic solutions, sterilization by autoclave of all materials used, and precautions to ensure that syringes and needles used do not contain impurities.

It is possible that damage may be caused by hypersensitivity to the analgesic drugs. Strong concentrations of analgesic drugs have been shown to cause degeneration of nervous tissue after intrathecal injection in animals (Davis *et al.*, 1931; Macdonald and Watkins, 1937). The concentrations of drugs used were higher than those usually given in humans. Permanent neurological damage is also known to be a rare complication of regional analgesia such as brachial plexus block. In the case of spinal analgesia it is also possible that local factors may sometimes be present within the subarachnoid space and may prevent normal diffusion of the analgesic solution. This would result in an unusually high concentration of analgesic drug. Similar reasoning

has been used by Payne and Bergentz (1956) to explain cases of arachnoiditis which have been found to occur with light cinchocaine analgesia (Hewer, 1933; Thorsén 1947). It therefore seems desirable to use minimal doses in spinal analgesia. Hingson and Hellman (1951), Lock and Greiss (1955), and Mitchell (1955) consider that overdosage may have been responsible for some cases of neurological damage following spinal analgesia. Lock and Greiss recommend that not more than 1.8 mg. of cinchocaine hydrochloride should be used in saddle block analgesia.

Adhesive arachnoiditis has been shown to follow both acute benign lymphocytic meningitis (Barker and Ford, 1937) and cerebrospinal meningitis (Turner, 1948), but there is no convincing evidence to suggest that infection is an aetiological factor in adhesive arachnoiditis following spinal analgesia.

In some instances spinal analgesia appears to activate pre-existing neurological conditions such as disseminated sclerosis or subacute combined degeneration (Macdonald Critchley, 1937; Nicholson and Eversole, 1946). Spinal analgesia should not be used in patients known to suffer from neurological disease.

Discussion

In the American literature, in recent years, large series of cases have been recorded where spinal analgesia has been given without causing either death or serious complications. Gibbs and Cooley (1956) report 12,202 uncomplicated cases in which saddle block analgesia was used for vaginal delivery. Similar series without complications are recorded by Torkelson and Cooley (1951), 4,000 cases; Crowder and McNulty (1951), 1,000 cases; Lewis and Austen (1950), 1,032 cases; and Hundley (1955), 27,925 cases. Dripps and Vandam (1951, 1954) and Vandam and Dripps (1956) quote their experience of 10,098 spinal analgesics given for a variety of operations, including some in the upper abdomen. There were no deaths, and only one case developed serious neurological disorder (subsequently shown to be due to a meningioma) within a follow-up period of between six months and five years. Minor neurological disorders were, however, more frequent: 66 patients showed transient sensory and motor changes in the legs and 34 had temporary visual disturbances.

Low spinal analgesia has several advantages. Good operating conditions are produced, and there is no depression of the foetal respiratory centre. The technique is easy to learn and there are few failures. In an emergency the analgesic can be administered by the obstetrician. The absence of serious complications in this series and in the much larger ones reported in the U.S.A. shows that the fear of unpleasant sequelae of spinal analgesia can be exaggerated. Since only a saddle block is required for forceps and breech delivery the danger of spinal shock is minimal. The use of careful technique and low dosage reduces the risk of serious neurological damage.

Few would dispute the present-day preference for the use of general anaesthesia in most surgical operations, but there is room for second thoughts in the limited field of anaesthesia for forceps and breech deliveries which cannot reasonably be conducted under local analgesia. Here some complications inherent in general anaesthesia occur more often than usual, whereas those of spinal analgesia are reduced, since only a saddle block is used. The incidence of serious complications is

probably less with spinal analgesia than with general anaesthesia, particularly if the latter has to be administered in an emergency by an unskilled anaesthetist. Low spinal analgesia is therefore worthy of serious consideration for difficult forceps and breech deliveries.

Summary

371 forceps and 68 breech deliveries were conducted under spinal analgesia in the City Hospital, Nottingham, during the years 1951 to 1955.

There were no maternal deaths and no serious post-analgesic complications.

Headache occurred in 14.1% of patients.

There were 20 stillbirths and 7 neonatal deaths, but these included 15 babies who had congenital abnormalities or died *in utero*, and none were attributed to spinal analgesia.

The advantages of spinal analgesia for forceps and breech deliveries that cannot be performed under local analgesia are discussed.

I am grateful to Mr. H. J. Malkin, Mr. P. C. Barkla, and Mr. J. B. Cochrane, under whose care these cases were admitted, for their encouragement and permission to publish. I thank Professor C. S. Russell and Mr. C. J. Dewhurst for their advice in the preparation of this paper.

REFERENCES

- Barker, L. F., and Ford, F. R. (1937). *J. Amer. med. Ass.*, **109**, 785.
 Barnes, J. E. M. (1955). *Brit. med. J.*, **2**, 677.
 Brain, R., and Russell, Dorothy (1937). *Proc. roy. Soc. Med.*, **30**, 1024.
 Critchley, M. (1937). *Ibid.*, **30**, 1007.
 Crowder, R. C., and McNulty, J. V. (1951). *Ann. west. Med. Surg.*, **5**, 531.
 Dattner, B., and Thomas, E. W. (1941). *N.Y. J. Med.*, **41**, 1660.
 Davis, L., Haven, H., Givens, J. H., and Emmett, J. (1931). *J. Amer. med. Ass.*, **97**, 1781.
 Dripps, R. D., and Vandam, L. D. (1951). *Ibid.*, **147**, 1118.
 — (1954). *Ibid.*, **156**, 1486.
 Ferguson, F. R., and Watkins, K. H. (1938). *Brit. J. Surg.*, **25**, 735.
 Gibbs, C. E., and Cooley, C. L. (1956). *West. J. Surg.*, **64**, 76.
 Gordon, J. (1955). *Brit. med. J.*, **2**, 489.
 Hewer, C. L. (1933). *Proc. roy. Soc. Med.*, **26**, 506.
 Hingson, R. A., and Hellman, L. M. (1951). *Anesthesiology*, **12**, 745.
 Holmes, F. (1957). *J. Obstet. Gynaec. Brit. Emp.*, **64**, 229.
 Holmes, J. M. (1956). *Ibid.*, **63**, 239.
 Hundley, J. M., jun. (1955). *Amer. J. Obstet. Gynec.*, **70**, 872.
 Hurst, E. W. (1955). *J. Path. Bact.*, **70**, 167.
 Jeffcoate, T. N. A. (1953). *Brit. med. J.*, **2**, 951.
 Kennedy, F., Effron, A. S., and Perry, G. (1950). *Surg. Gynec. Obstet.*, **91**, 385.
 Klein, M. D., Clahr, J., Tamis, A. B., and Solkow, M. L. (1953). *N.Y. J. Med.*, **53**, 2861.
 Lewis, M. S., and Austin, R. B. (1950). *Amer. J. Obstet. Gynec.*, **59**, 1146.
 Lock, F. R., and Greiss, F. C., jun. (1955). *Ibid.*, **70**, 861.
 Macdonald, A. C., and Watkins, K. H. (1938). *Brit. J. Surg.*, **25**, 879.
 Martin, W. I. (1955). *Brit. med. J.*, **2**, 676.
 Mitchell, R. McN. (1955). *Obstet. and Gynec.*, **5**, 123.
 Nicholson, M. J., and Eversole, U. H. (1946). *J. Amer. med. Ass.*, **132**, 679.
 Parker, R. B. (1954). *Brit. med. J.*, **2**, 65.
 — (1956). *Ibid.*, **2**, 16.
 Payne, J. P., and Bergentz, S. E. (1956). *Lancet*, **1**, 666.
Proc. roy. Soc. Med., 1957, **50**, 547.
 Robinson, H. M., jun. (1945). *Amer. J. Syph.*, **29**, 422.
 Rollason, W. N. (1955). *Brit. med. J.*, **2**, 677.
 Rosenbaum, H. E., Long, F. B., Hinchey, T. R., and Trufant, S. A. (1952). *A.M.A. Arch. Neurol. Psychiat.*, **68**, 783.
 Scott, J. S., and Gadd, R. L. (1957). *Brit. med. J.*, **1**, 971.
The Times. Law Report, November 13, 1953.
 Thorsén, G. (1947). *Acta chir. scand.*, **95**, Suppl. 121.
 Torkelson, H. P., and Cooley, C. L. (1951). *West. J. Surg.*, **59**, 533.
 Turner, J. W. A. (1948). *Lancet*, **1**, 398.
 Vandam, L. D., and Dripps, R. D. (1956). *J. Amer. med. Ass.*, **161**, 586.