SPINAL ANAESTHESIA IN ELECTRICAL CONVULSIVE THERAPY.

H. J. SHORVON, M.R.C.S., D.A., D.P.M.Lond.,
Psychiatrist, E.M.S., Sutton Emergency Hospital, and

L. M. SHORVON, M.R.C.S., D.A., D.C.H.,
Anaesthetist, E.M.S., Sutton Emergency Hospital.

[Received October 30, 1942.]

THE value of electrical shock therapy is becoming increasingly recognized and used by psychiatrists in the treatment of depressive states, particularly of the involutional period, states of depersonalization, and to a lesser extent in schizophrenia. One of the major objections to its use, however, has been the complication of fractures, generally crush fractures of the bodies of the mid-dorsal vertebrae (thoracic 4–8), and uncommonly that of limb bones, such as the femur. Another aspect of this problem arises with the occasional case in which the use of shock therapy is desirable, but a previous fracture of the spine or limb bones, or marked bone disease with an increased liability of fracture resulting during treatment, acts as a deterrent to the physician. Even more so is the question of continuing treatment without further damage to the patient, should a fracture have unfortunately resulted before the full course of convulsions is completed. Various methods have been introduced to minimize or abolish the muscular pull producing fractures, including intravenous injections of curare, of concentrated magnesium sulphate solution, or of beta-erythoidin hydrochloride; spinal anaesthesia has been similarly employed.

The intravenous injections have a generalized muscular effect, while the protective action of the spinal anaesthetic is, of course, only on the thoracic and lumbar spine and the lower limbs.

None of these methods has proved entirely satisfactory, but this paper illustrates the decided value of spinal anaesthesia in cases where the problems mentioned above have arisen.

Numerous papers have been published analysing the incidence of vertebral fractures in different series of cases. With improvements in technique, such as keeping the patient's spine in a position of hyperextension and applying counter pressure to the hips and shoulders while allowing freedom of movement of the extremities, there has been a marked drop in the incidence of these fractures.

The first vertebral fracture following induced convulsions was recorded by Stalker in November, 1938, and in 1939 Polatin, Friedman, Harris and Horwitz recorded 43 per cent. vertebral fractures. This figure has been rapidly reduced by successive workers, such as the 23 per cent. of Easton and Sommers, 14 per cent. of Cook and Sands in 1940 without using hyperextension, down to the very low incidence of rate of 2 fractures in 112 cases recorded by Cheney, Hamilton and Heaver in 1941, using hyperextension. All workers agree that the vertebral fractures are usually found to occur between the 4th and 8th dorsal vertebrae, and that they are compression fractures with anterior wedging of the vertebrae. As stated by Worthing and Kalinowsky the general opinion is that the rigid mid-dorsal curvature becomes subjected to strong converging muscular forces which act above and below the dorsal region. It is also agreed that the incidence of vertebral fractures using electrical means is about a quarter that seen when metrazol (cardiazol) is used. One group by L. B. Kalinowsky had 10 per cent. vertebral fractures with electrically induced convulsions, compared with 43 per cent. when

metrazol was used, all cases having sandbags placed under the mid-dorsal spine and the same radiologist and X-ray technician employed. Roberg, in studying the spinal changes, concluded that the forces exerted by tetanic muscular contractions produced compression fractures of the mid-dorsal vertebral bodies, usually those of the 5th and 6th dorsal vertebrae, in definite contrast to ordinary traumatic compression fractures which centre about the body of the first lumbar vertebra. There may be a predisposing factor, i.e. the possible presence of bone atrophy. In addition to vertebral fractures bilateral fractures of the femoral necks caused by metrazol-induced convulsions are recorded by Androps, Sommers and Richardson, and by Hemphill; and A. E. Bennett in 1941 recorded serious fractures of humerus and femur in 1·5 to 2 per cent. and dislocations in 17·2 per cent. The incidence of vertebral fractures is greater over 50 and under 20 years than between these ages. Thus it can be concluded that where X-ray of the dorsal spine shows a normal state of affairs and the patient's age is between 20 and 50, with recent improvements in technique, the routine use of spinal anaesthesia is unnecessary; but in cases where the above conditions do not hold, it may be a useful aid.

Hamsa and Bennett state: "The following is the prophylactic treatment to remove the potential forces of the lower extremity and spinal flexor muscular contractions: 30 minutes to one hour before induction of convulsion, 10 mgm. of pantocaine hydrochloride or 100 mgm. of procaine hydrochloride dissolved in 4 c.c. of spinal fluid obtained by puncture between L1 and L2 are injected.

"Transient anaesthesia with complete sensory and motor paralysis of all thoracic and lumbar segments wears off in about an hour after the convulsion. The convulsive shock ensues without contraction of the anaesthetized areas." We do not consider it necessary to produce complete motor paralysis of all the thoracic segments to avoid fracture; if a sufficient damping down of the force of muscular contraction is obtained, vertebral fractures will not occur. More so is this the case if hyperextension is used in addition; thus if, as we make a practice of doing, the spinal anaesthetic introduced effects a marked degree of motor paralysis no higher than D7, the undesirable results of the lowered blood pressure which follows the paralysis of the vasomotor fibres are avoided to a great extent, as also is the danger of respiratory embarrassment and failure which would be produced by paralysis of the intercostal muscles. The muscles involved, such as the abdominal muscles, the multifidus and the sacro-spinalis are largely put out of action, and there is little pull on the mid-dorsal vertebrae.

Technique.

The results obtained by the following technique were viewed from two angles: (*a*) The prevention of new fractures, and the prevention of further damage to old fractures or bone disease, such as Paget's disease, old tuberculous disease, osteoarthritis, etc.; (*b*) the effect of repeated spinal anaesthetics at short intervals on the spinal cord and cerebro-spinal fluid. Stovaine (amylocaine hydrochloride) was used in all instances, made up according to Barker's formula: amylocaine hydrochloride, 0·10 gm.; glucose, 0·10 gm.; with distilled water up to 2 c.c. Specific gravity 1025. Thus a hypertonic solution of strong concentration was used. Ordinary precautions such as are used before any spinal anaesthetic were observed, namely a full preliminary physical examination, no food for some hours before, aseptic surgical technique. Novocaine 1 per cent. was used to anaesthetize the skin, subcutaneous tissues, and ligaments over the intervertebral space selected. Lumbar puncture was performed between L1 and L2, using a Howard Jones needle, the patient in a sitting-up position on the table. The C.S.F. pressure was measured and the fluid collected for examination. With each patient the first specimen was examined for Wassermann reaction, Lange's curve, cells and protein percentage, and the subsequent specimens were examined for cells and protein only. After each lumbar puncture 1·8–2 c.c. of stovaine, depending on the height and configuration of the patient, was introduced into the spinal canal and the patient laid flat on the table, which was then tilted to an 8° Trendelenburg position. An interval of roughly half an hour was allowed to elapse before the current was passed, and in this interval the patient was tested for motor and sensory paralysis, and the electrical apparatus (in this series the Strauss-Macphail apparatus was used)

fitted up. Fifteen minutes after the injection of the stovaine the table was lowered to a horizontal position, the spinal anaesthetic being regarded as " fixed " by that time. As a rule the paralysis rapidly reached a level of D7. Immediately before the shock therapy was applied a rolled blanket or small pillow was placed under the mid-dorsal region so as to put the spine in a position of hyperextension. After each convulsion the foot of the bed was raised on blocks for a few hours and the usual treatment following a spinal anaesthetic was carried out. The patient is confused for a short but variable time, and it is found that as soon as he appreciates his surroundings the anaesthetized limbs have recovered their functions. In Case 2 below, where arteriosclerotic features were prominent, the blood pressure was recorded before, immediately after, and ten minutes after each convulsion. In addition to this technique described, use was made of a free oxygen supply after the convulsion by applying a B.L.B. mask in Cases 2 and 4. We consider that in cases of suspected myocardial insufficiency the use of the B.L.B. mask immediately after the convulsion is of definite value. Also 20 c.c. of 33 per cent. glucose were injected intravenously after the spinal anaesthetic, and before the current was passed in Case 2, so as to counteract possible ill-effects on the myocardium as a result of the convulsion. In Case 4, 2 c.c. of coramine were injected intramuscularly immediately after the convulsion for a similar reason. In all cases 70 volts on the Strauss-Macphail machine were sufficient to produce a convulsion. Where the patient was on heavy doses of sedative, such as sodium amytal, this was omitted the night before each treatment.

Case Reports.

Case 1.—Female patient, aged 60, suffering from a fairly severe depressive illness following an air raid which caused injuries necessitating amputation of both legs. On X-ray it was found that she also had a traumatic crush fracture of the 2nd and 3rd lumbar vertebrae and some arthritic changes in the dorsal spine. A course of convulsive shock therapy with the aid of spinal anaesthesia was decided on. She had nine spinal anaesthetics followed by convulsions, at intervals of three days to a week, using 1·8 c.c. stovaine on each occasion. The C.S.F. pressure was normal throughout; the first specimen showed no cells and 20 mgm. per cent. protein, whilst the final specimen (i.e. after nine spinal injections) showed 17 lymphocytes per cmm. and 50 mgm. per cent. protein. Throughout the course of treatment the patient complained of no symptoms and showed no signs referable to the C.N.S.; and the final X-ray showed no change on the first one. She was discharged four months after admission, much improved.

Case 2.—Male patient, aged 54, was admitted suffering from a depressive illness with hysterical features. He had a forcible apex beat, blood pressure 190/110 mm. Hg; the peripheral vessels were hardened; the cranial nerves and reflexes normal; some rigidity of the limbs, and a persistent tremor. A diagnosis of arteriosclerotic Parkinsonism was made. His blood pressure varied between 170–200 mm. Hg systolic and 105–115 mm. Hg diastolic. In this case it was thought that preliminary spinal anaesthesia would, in addition to its protective action, lower the blood pressure, and it was found that after stovaine was injected and before the current was passed there was an average drop of 20–30 mm. Hg in systolic readings. The blood pressure rose during the convulsion to an average reading of 170–180 mm. Hg systolic, and fell again to the earlier reading ten minutes after the convulsion. His ordinary blood-pressure reading after completion of treatment was 145/95 mm. Hg. The cerebro-spinal fluid reports were of much interest in this case. Seven spinal administrations each of 2 c.c. stovaine using technique as described were given at intervals varying from three days to a week. The Wassermann reaction was negative, and C.S.F. pressure within normal limits. The specimen after the first anaesthetic had 81 lymphocytes per c.mm. with 40 mgm. per cent. protein. The reports after the next two anaesthetics gave lymphocytes plus with a positive Pandy reaction. After the fifth stovaine injection there were only nine cells per c.mm., but after the seventh spinal anaesthetic the cell count rose to 229 per c.mm. with small lymphocytes predominating and 80 mgm. per cent. protein. This patient made slow progress, but a contributory factor was domestic difficulties. There were no symptoms attributable to the spinal anaesthetic and no abnormal physical signs. Patient showed no ill-effects some months later.

Case 3.—Female patient, aged 42, admitted suffering from depression. She had an old tuberculous lesion of the left hip, which had been in plaster for months at a time. The X-ray report on admission stated: " Left hip shows destruction of head of femur and acetabulum with bony ankylosis. Spine normal apart from arthritic lipping. Abdomen shows calcified mesenteric glands and there is a calcified focus in the chest." Three spinal anaesthetics each of 1·9 c.c. stovaine were administered preliminary to passage of current at weekly intervals. The C.S.F. was not under pressure, did not show any cells, and had 20 mgm. per cent. protein before treatments began. There was no change in the C.S.F. after each spinal anaesthetic, and final X-ray showed no change. Patient was discharged recovered with no abnormal physical signs or symptoms.

CASE 4.—Male patient, aged 50, was admitted suffering from recurrent depressive psychosis. Intravenous cardiazol was used without spinal anaesthesia, and with the first convulsion a chip fracture of the left acetabulum occurred. The hip was put up in plaster for a fortnight, and with further treatment no disability resulted. He was given seven spinal anaesthetics followed by convulsions induced electrically, and he was discharged from hospital recovered. Stovaine was used in each instance and the final C.S.F. reading was—Wassermann reaction negative, cells 5 per c.mm., and protein 30 mgm. per cent. After some months at work patient's symptoms returned and he was readmitted. An X-ray taken soon after was reported on as " Paget's disease of left ilium involving left acetabulum with diminution of joint space. Pieces of bone detached from upper rim are still ununited. Kyphosis of thoracic spine with arthritic changes. No Paget's disease of spine." We gave this patient nine spinal anaesthetics each of 2 c.c. stovaine, using technique described, at intervals of three days. Apart from a mild headache no complaints were made. The C.S.F. pressure was within normal limits throughout and the first specimen of fluid (before the first injection of stovaine in this second series) showed no cells and 20 mgm. per cent. protein. After the first injection there were 2·3 cells per c.mm. with no excess protein, after the second injection 1·6 cells per c.mm. and no excess protein. The cells rose to 6 per c.mm. (small lymphocytes) after the third anaesthetic with a slight excess of protein, and after the fifth injection the cell count was 36 cells per c.mm. (majority small lymphocytes) and moderate excess protein. After the seventh spinal injection the cells fell to 13·6 per c.mm. with slight excess protein, and after the final anaesthetic examination gave 38 cells per c.mm., and slight excess protein. A specimen taken nine days later without using anaesthesia showed the spinal fluid practically back to normal, namely 6 cells per c.mm. and 25 mgm. per cent. protein. On physical examination there were no abnormal signs as a result of the anaesthetics. Patient made a good recovery. A specimen of C.S.F. taken two months later was normal.

DISCUSSION AND SUMMARY.

1. It is not uncommon in hospital practice to find patients recommended for convulsive shock therapy who have a history of previous traumatic injuries to the spine or limb bones, or previous bone or joint disease, such as tuberculosis, arthritis or Paget's disease, hence rendering the use of spinal anaesthesia a distinct aid. The cases described were seen in the same hospital within the last year, one with traumatic fracture of lumbar vertebrae, another with an old tuberculous disease of hip, and a third with Paget's disease of the ilium with an ununited fracture of the acetabulum following convulsive therapy without spinal anaesthesia.

2. The effect of repeated injections of stovaine is not necessarily deleterious; it may give a mild meningitic reaction which is temporary, the reaction tending to increase with the number of anaesthetics administered. This does not result in abnormal symptoms and signs in the C.N.S.

3. Stovaine is a hypertonic solution of high concentration generally regarded as an irritant to the tissues. It is therefore of interest to find it has no apparent harmful effect if often repeated.

4. A possible use of spinal anaesthesia in cases of hyperpietics requiring convulsion therapy to help avert possible vascular catastrophes is indicated by successful result in the second case.

5. The administration of oxygen by the B.L.B. mask immediately after the convulsion, and the intravenous injection of glucose before the convulsion, are suggested as useful aids in myocardial insufficiency.

We wish to thank Dr. Louis Minski, the Medical Superintendent, and Dr. Eliot Slater, the Clinical Director, for their advice.

REFERENCES.

ANDROP, S. (1941), *J. Nerv. and Ment. Dis.*, **93**, 701.
BENNETT, A. E. (1940), *J. Amer. Med. Ass.*, **114**, 322.
CHENEY, C. C., HAMILTON, D., and HEAVER, W. (1941), *Psychiat. Quart.*, **15**, 214.
COOK, L. C., and SANDS, D. E. (1941), *J. Ment. Sci.*, **87**, 208.
EASTON, N. L., and SOMMERS, J. (1942), *Amer. J. Psychiat.*, **98**, 538.
HAMSA, W. R., and BENNETT, A. E. (1939), *J. Amer. Med. Ass.*, **112**, 2240.
HEMPHILL, R. E. (1942), *Lancet*, ii, No. 6, 152.
POLATIN, P., FRIEDMAN, M. M., HARRIS, M. M., and HORWITZ, W. A. (1939), *J. Amer. Med. Ass.*, **112**, 1684.
ROBERG, O. T. (1939), *J. Bone Jt. Surg.*, **19**, 603.
SOMMERS, J., and RICHARDSON (1939), *Amer. J. Psychiat.*, **95**, 1193.
STALKER, H. (1938), *Lancet*, **2**, 1172.
WORTHING, H. J., and KALINOWSKY, L. B. (1942), *Amer. J. Psychiat.*, **98**, 533.