following intrathecal injection. The explanation may be that this is an anoxic effect due to the vasoconstrictor action. However, those workers suggest that adrenaline itself has a direct effect on nerve conduction, and Forbes *et al.* (1933) and Fog (1939) showed that there was no arteriolar constriction following direct application of adrenaline to the pia mater. Nevertheless, the clinical and pathological findings of nervous damage confined to the anterior spinal artery distribution strongly suggest that some block, albeit transient, occurred in this arterial supply, and we believe that adrenaline could have caused such a transient block.

It is appropriate at this stage to discuss briefly the significant points concerning the arterial anatomy in the region. The cord derives its supply from three vessels, one anterior and two posterior. The anterior spinal artery arises as a branch from each vertebral artery. These unite at the level of the foramen magnum to form one vessel, which runs down the whole length of the cord in the midline. This vessel, through multiple small branches, supplies the lateral column and the anterior grey and white columns. The posterior pair of arteries arise from the posterior inferior cerebellar or from the vertebral arteries, and run down the whole length of the cord in relation to the posterior nerve roots supplying the posterior third of the cord. The anterior spinal artery is virtually an end-artery receiving significant collaterals only at the level of T 1 and T 11. The posterior spinal arteries in contrast have numerous collateral vessels freely anastomosing over the posterior part of the cord. It is obvious that interference with the blood supply derived from the anterior artery will give rise to ischaemic changes in the anterior two-thirds of the cord; whereas occlusion of the posterior vessels is less likely to produce permanent changes because of their free anastomosis.

The syndrome of anterior spinal artery thrombosis was first clinically diagnosed by Spiller (1909). Since his description many cases of this syndrome have been reported. The following aetiological factors have been blamed: interference with the blood supply through aortic compression, thrombosis, aneurysm and co-arcuation, syphilitic thrombosis, arteriosclerosis, and simple vascular spasm; as well as trauma, both direct and indirect, and compression due to tumour or disk (Stegmann, 1952; Peterman *et al.*, 1958). Still, there are numerous reports of cases where no causative factors were found.

The lesion produces a definite clinical picture of destruction in the area supplied by this anterior spinal artery. Most commonly the areas affected have been the lower cervical and the thoraco-lumbar regions. Our case was typical of this syndrome, and further elaboration is unnecessary.

Summary and Conclusions

A case of the anterior artery syndrome following epidural anaesthesia is presented.

We have discussed the possible causes of the anterior spinal artery syndrome in this case, and have come to the conclusion that no single factor was responsible. We believe that there are possibly two causes: firstly, the relative hypotension imposed on the patient's already failing peripheral circulation; and, secondly, the vaso-spastic action of the adrenaline. From the histological findings that thrombosis had not occurred in this artery, we have to assume, therefore, that the occlusion was of transient nature. Unfortunately, this was of sufficient duration to produce permanent changes in the anterior part of the cord due to ischaemia. His arterial supply was probably already decreased owing to his heart failure, and therefore interference with this supply was disastrous. Finally, it should be mentioned that cases of spontaneous occlusion of the anterior spinal artery have been reported and that, although unlikely, this might have been purely coincidental.

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REFERENCES

- Bromage, P. R. (1954). *Spinal Epidural Analgesia*. Livingstone, Edinburgh.
 Ciliberti, B. J. (1948). *Anesthesiology*, 9, 439.
 Ditzler, J. W., and McIver, G. (1956). *Curr. Res. Anaesth.*, 35, 501.
 Fog, M. (1939). *Arch. Neurol. Psychiat.* (Chicago), 41, 109.
 Forbes, H. S., Finley, K. H., and Nason, G. I. (1933). *Ibid.*, 30, 957.
 Frumin, M. J., Schwartz, H., Burns, J. J., Brodie, B. B., and Papper, E. M. (1953). *J. Pharmacol. exp. Ther.*, 109, 102.
 Payne, J. P., and Bergentz, S-E. (1956). *Lancet*, 1, 666.
 Peterman, A. F., Yoss, R. E., and Corbin, K. B. (1958). *Proc. Mayo Clin.*, 33, 31.
 Priddle, H. D., and Andros, G. J. (1950). *Curr. Res. Anaesth.*, 29, 156.
 Ruben, J. E., Kamsler, P. M., and Howell, W. L. (1948). *Science*, 107, 223.
 Schultz, E. C., Johnson, A. C., Brown, C. A., and Mosberg, W. H. (1953). *J. Neurosurg.*, 10, 608.
 Spiller, W. G. (1909). *J. nerv. ment. Dis.*, 36, 601.
 Stegmann, A. T. (1952). *Neurology*, 2, 15.
 Sykes, M. K. (1958). *Anaesthesia*, 13, 78.
 Thomas, P., and Dwyer, C. S. (1950). *Anesthesiology*, 11, 635.
 Wikler, A., Marmor, J., and Hurst, A. (1937). *J. Amer. med. Ass.*, 109, 430.

NEUROLOGICAL COMPLICATIONS OF EPIDURAL ANAESTHESIA

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Epidural anaesthesia is generally regarded as being safe, easy to carry out, and free from complications. Many authors, in reviews of its use in thousands of cases, record very low mortality and morbidity. The technique of the injection involves the introduction of a lumbar-puncture needle between two spinous processes into the epidural space. In this situation a negative pressure exists, as demonstrated by the suction effect upon a drop of saline placed on the hilt of the needle. 2 ml. of 2% procaine solution is injected, and if during the next 10 minutes no reaction occurs a further 25-30 ml. is introduced.

Two cases are described in which neurological complications of a very serious nature appeared after epidural anaesthesia with the technique described above.

Case 1

A 26-year-old housewife had a caesarean section at another hospital four months previously, and because of a history of bronchial asthma epidural anaesthesia was decided upon. No untoward incidents were noted at the time of operation, but the same day she complained of headache and double vision, the latter passing off in a few hours. Headache and a feeling of intracranial pressure persisted for a few weeks, and at this time the patient noticed gradually increasing difficulty in walking. Weakness in the legs, first right and then left, became progressively more pronounced until a few weeks later she was quite unable to walk. Impairment of sphincter control followed some weeks later and became complete. She was readmitted to hospital, where lumbar puncture revealed partial manometric block. On myelographic examination the oil column was held up at D 11.

The patient was transferred to this hospital and showed paralysis of the lower limbs, complete apart from feeble flexion of the right knee. The muscles were hypotonic, the right knee-jerk and abdominal reflexes were absent, and there was left bilateral ankle clonus; sensation, both

superficial and deep, was much impaired up to D 11. The bladder was paralysed, with retention of urine. In the hope that the cause of the paraplegia might conceivably be a local constrictive arachnoiditis at the level of the myelographic block, the patient was transferred to the neurosurgical department. Laminectomy at D 11-L 1, performed by Dr. Herzberger, revealed gross arachnoiditis, the cord being embedded in an opalescent jelly-like mass which extended as far as could be seen cranially and caudally. The condition of the patient was not influenced by the decompression, and she remained permanently crippled.

Case 2

A 66-year-old man was admitted for surgical treatment of vesical calculi. Five years previously he had undergone a spinal operation for a tumour at D 12, which proved to be a chordoma. His symptoms at the time were pains in the back and legs, and these were relieved by the operation. Up to the time of this second admission he had been entirely free of any complaints referable to the lower limbs and there were no objective neurological residua. Transurethral lithotomy under epidural anaesthesia passed off without incident.

A month later a second-stage operation was performed with transurethral resection of the prostate, again under epidural anaesthesia (L 2-3). The patient was seen in consultation on the following day because power in the legs had failed to return. He was found in good general condition, with paralysis of ilio-psoas and quadriceps muscles bilaterally; muscles below the knees were active. Deep reflexes were absent and sensation was reduced below the level of L 3. With this distribution of paralysis it was thought less likely that there had been inadvertent intrathecal injection than that for some reason the roots at the level of the injection had failed to recover from the anaesthesia. During the succeeding weeks there was only minor improvement until the patient died from cardio-pulmonary complications.

At post-mortem examination the remains of a nodular growth were seen at D 12 with an intrathecal extension the size of a pea. Considering the time relationship as outlined above, it was not thought that this tumour played a part in the production of the post-operative paralysis.

Discussion

The mechanism of production of the cord changes described above must remain to some extent speculative. Although sepsis cannot be ruled out, it is probably an unlikely explanation. In Case 1 there was nothing to suggest an acute post-operative meningitis; in Case 2 the complications were immediate, but limited to the anatomical segments innervated from the level of the injections. Taking into consideration the headaches and double-vision experienced by the first patient on the day of operation, it seems difficult to exclude the likelihood that the arachnoiditis and cord changes were a direct expression of a chemical meningo-myelopathy resulting from inadvertent injection of the anaesthetic into the subarachnoid space. Since complications of this nature are known (Payne and Bergentz, 1956) to follow deliberate intraspinal anaesthesia where total quantities of only 40 mg. of procaine are employed, it is not unreasonable to suppose that they may result from the introduction of some portion of the total of 500 mg. commonly employed in epidural anaesthesia. The clinical course of the case described by Payne and Bergentz resembles very closely that of our Case 1. Progressive paraplegia began seven days after the anaesthesia and became complete in two weeks. These authors quote experimental work on dogs which demonstrated the appearance of local arachnoiditis following spinal anaesthesia in clinical doses. Haxton (1956) mentions an experience of his own in which an intrathecal injection of phenol was unwittingly given when the intention was to place the needle paravertebrally, let alone epidurally.

Local hypersensitivity of nerve roots to the anaesthetic reagent or chemical changes or impurities may perhaps be invoked to account for the persistent paralysis in our second case.

General hypersensitivity to the anaesthetic agent would seem to be an unlikely explanation, as a reaction of a more widespread nature might be expected. In this connexion it is of no small interest that in the course of our investigation into the complications of epidural anaesthesia prompted by the above experiences it was discovered that the only two cases of cardiac arrest during anaesthesia which had occurred in this hospital happened while this method was being employed and before the operation itself had started.

Both patients were in good general health and were admitted for elective operations. In one, 15 ml. of anaesthetic was injected, when the heart suddenly stopped. In the second it was thought that a portion of the anaesthetic may have been introduced intrathecally. The needle was partially withdrawn and the anaesthesia proceeded with, as there were apparently no reactions; however, a few minutes later, as the skin was being incised, the pulse stopped. Cardiac massage succeeded in restoring the pulse in both cases, and one patient made a reasonable recovery in the course of some weeks; the other died in coma. In these two cases one may postulate the existence of hypersensitivity to procaine, though a reaction to the flooding of the cerebrospinal fluid with large quantities of anaesthetic cannot be ruled out.

Though the numerical incidence of complications following epidural anaesthesia as seen in this hospital is low (4 out of 200 lumbar and several hundred sacral injections) their seriousness and their tragic consequences suggest that this method should be employed only after very careful consideration. Although this method is claimed to be safe, we cannot be sure that all complications are indeed brought to light or even thought of when they are of delayed onset. It seems that Walshe's (1956) criticisms of intrathecal anaesthesia may be equally pertinent when applied to epidural anaesthesia.

Summary

Neurological complications are described in two patients following epidural anaesthesia: they are believed to have been due to a local chemical meningo-myelopathy or neuropathy.

Reference is made to two cases of cardiac arrest attributable to this technique.

REFERENCES

- Haxton, H. (1956). *Lancet*, 1, 1015.
Payne, J. P., and Bergentz, S.-E. (1956). *Ibid.*, 1, 666.
Walshe, F. M. R. (1956). *Ibid.*, 1, 859.

The Third International Congress of Photofluorography was held in Stockholm last month under the presidency of Professor CARL WEGELIUS. It was attended by 600 delegates from all over the world. The papers read at the Congress gave clear evidence that here is a field in a ferment of activity. Important advances such as alkali halide intensifying screens and new methods of film processing suggest that up to a tenfold reduction in received radiation may be expected from technical innovations. With modern coning and filtering of the x-ray beam the dose to the skin and gonads in taking a single chest film can be reduced to less than the weekly permitted safe dose for radiographers who spend their whole working lives exposed to x-radiation, while image intensification can increase the brightness of the screen picture a thousand times. This allows screening at very low radiation levels, a degree of brightness that makes dark adaptation unnecessary, and the making of permanent multiple or cine photographic records of physiological or pathological functions for analysis at leisure in "slow motion." A special edition of *Excerpta Medica* summarizes most of the papers presented at the Congress.