exposures—available evidence does not suggest that these factors can account for a large part of the difference.

Factors related to climate and to the incidence rates of bronchitis cannot be ruled out of consideration. There is too little evidence at present to draw any conclusions concerning this point.

It is within the realm of possibility that a part of the difference is due to under-reporting of lung cancer as a cause of death in the United States or to over-reporting of lung cancer as a cause of death in England and Wales, or both.

Clearly, the data at present available are insufficient for any definite conclusions to be drawn on why reported lung cancer death rates are so much higher in England and Wales than in the United States. However, it does not appear likely that any single one of the factors discussed above is sufficient in itself to account for all of the difference. This suggests that the difference may be due to an interaction of two or more of these factors. For example, bronchitis, which appears to be more common in England and Wales than in the United States, may increase the susceptibility of individuals to the carcinogenic effect of tobacco smoke or air pollution.

Summary

In 1955 the lung cancer death rate reported in England and Wales for males was 2.1 times as high as reported in the United States and for females 1.6 times as high. A difference of this order of magnitude has existed in male rates for several decades; while prior to 1949 there was only a small difference in the female rates.

Among both males and females in the middle- and old-age groups there are proportionately more cigarette smokers in the United Kingdom than in the United States. On the other hand, at the present time the number of cigarettes consumed per smoker per day is higher in the United States than in the United Kingdom. The proportion of male cigarette smokers who inhale appears to be approximately the same in the two countries. In the United States the average length of the remaining butt after a cigarette is discarded is about 31 mm.; about 5% are 45 mm. or longer and about 9% are under 20 mm. in length. In the United States in 1957 the main stream smoke of the average cigarette contained about 2.6 mg. of nicotine and 42.8 mg. of of tar. More recently, cigarettes with a relatively low content of nicotine and tar have become increasingly popular in the United States.

Data at present available do not support the hypothesis that a substantial proportion of the difference in lung cancer death rates between the two countries is attributable to differences in the use of cigarettes. However, more detailed information on smoking habits and on the chemical composition of cigarette smoke in the two countries is needed before this can be definitely established.

In both countries the lung cancer death rate of smokers, as well as the lung cancer death rate of non-smokers, is higher in urban areas than in rural areas. The difference in this respect is reported to be far greater between Liverpool and rural areas of Wales than between large cities and rural areas in the United States. However, Merseyside (which includes Liverpool) has a higher male lung cancer death rate than any other region in England and Wales, so it cannot be considered as typical of the country as a whole. While it is possible that some of the difference in lung cancer death rates may be due to factors associated with urbaniza-

tion—for example, air pollution or occupational exposures—available evidence does not suggest that these factors can account for a large part of the difference.

The reported death rate from cancer, exclusive of lung cancer, declined steadily from 1931–5 to 1956 in England and Wales but rose slightly in the United States during the same period of time. It is possible that differences in the diagnosis and recording of causes of death may account for a part of the difference between reported lung cancer death rates in the two countries.

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PARAPLEGIA FOLLOWING EPIDURAL ANAESTHESIA

BY

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We present a case of paraplegia that occurred after an epidural anaesthetic. So far as we can ascertain no similar case has been reported.

Case Report

A man aged 76 was admitted to hospital on March 14, 1957. He complained that for seven days before admission he had pain in his left-sided femoral hernia, with associated central abdominal intermittent pain. Two days before admission the pain in the rupture and the abdomen had become more severe and he started to vomit profusely. His bowels, usually regular, had been opened inadequately on the days before admission, and since the onset of the severe intermittent pain he had had no bowel movement nor had he passed flatus. The rupture appeared first six months before admission, but was small, and he had experienced no discomfort in it before.

In May, 1956, the patient was first seen at Ashford Hospital in congestive heart failure with pulmonary oedema. He was admitted to hospital and his cardiovascular condi-

tion was stabilized. After his discharge he once more developed gross congestive failure with bilateral bronchopneumonia. He refused to go to hospital, but shortly afterwards developed acute cor pulmonale, necessitating his admission. He was at this time grossly oedematous, and his neck veins were distended up to the angle of the jaw in the erect position. The fluid in his serous cavities was tapped, and his legs were drained with Southey's tubes. He made a surprisingly rapid recovery and was able to return home. During the ensuing months at the beginning of 1957 he was gradually deteriorating. His blood pressure, recorded at the time of his first admission to hospital, was 140/70.

On examination the patient looked extremely ill and dehydrated, with a blood pressure of 110/82 and a pulse rate of 100. He was slightly cyanosed, particularly the tip of his nose, hands, and feet, and was dyspnoeic at rest. His neck veins were engorged to about an inch (2.5 cm.) above his sternoclavicular joints and he had rales over both lung fields. His liver was palpable two fingerbreadths below the costal margin and was tender. The abdomen was not distended, nor was there visible peristalsis, but bowel sounds were increased. In his left groin there was a tender irreducible femoral hernia. The skin over the hernia and over the inguinal ligament was reddened. Rectal examination revealed only faeces. He had oedema of his sacrum and of the legs below the knees. His haemoglobin was 70%. He was given a blood transfusion of 1 pint (570 ml.), and two hours later was considered to be fit for surgery. It was decided that he should have an epidural anaesthetic.

Premedication.—Fifty minutes before the operation the patient was given pentobarbitone 50 mg., atropine 0.6 mg., and pethidine 75 mg.

Anaesthetic Technique.—His blood pressure was 90/50. Before the epidural anaesthesia was induced a Gordh needle was inserted intravenously. He was turned on to the left side with a head-down tilt of about 5 degrees. The skin overlying the lumbar region was cleaned with Bonney's blue and 70% spirit. The epidural space was easily identified between L1 and 2 by means of a modified Dogliotti technique using a 2-ml. syringe filled with air. As there was no backflow of cerebrospinal fluid, 27 ml. of 1.75% lignocaine hydrochloride ("xylocaine") containing 1 in 300,000 adrenaline was injected slowly over five minutes into the identified space. The patient was left lying on his left side for several minutes. Immediately on the completion of the epidural anaesthesia 15 mg. of ephedrine hydrochloride was given intravenously and 15 mg. intramuscularly to prevent further hypotension. The patient was turned on to his back, and it was noted that seven minutes after the injection of the local anaesthetic he was still able to move his legs to such an extent that he was requested to lie as still as possible during the course of the operation. Inter-mittent injections of ephedrine hydrochloride, to a total dose of 100 mg., were given during the course of the operation, maintaining his systolic blood pressure between 80 and 90 mm. Hg.

Operation.—A vertical incision was made and the McEvedy approach adopted. The peritoneal cavity was opened and a loop of ileum was seen to enter the hernial sac. This was reduced with some difficulty, and the bowel was found to be gangrenous and non-viable. Eight inches (20 cm.) of ileum was resected and continuity restored by end-to-end anastomosis in two layers. The hernial sac was transfixed and ligated and the wound closed in layers.

During the whole course of the operation the patient remained conscious and was talking, but was not felt to move his legs at all after being wheeled into the theatre. On completion of the operation his blood pressure was 100/50 and he had been given 250 ml. of blood during its course. The time taken to complete the operation was 45 minutes. He was returned to the ward in the head-down position, which was maintained until his blood pressure was stabilized at over 100 mm. Hg.; this is the practice at the

hospital. Immediately on return to bed his pressure was recorded as 138/74, and during the night fluctuated between 96/48 and 140/74. Unfortunately it was not noted whether he had moved his legs at all during this time.

Post-operative Course

On the following morning his general condition was satisfactory apart from slight dehydration. He had no pain, but complained that he was unable to move his legs. On examination his general condition was good, his abdomen was soft, with audible peristalsis, and his bladder was not distended. He could not move either leg, although he had very slight movement of the sartorius and plantar flexors of the right foot and slight movement of the long flexors of his left toes. The knee- and ankle-jerks were absent and no plantar responses were elicited. He had impairment of pin-prick up to the level of the umbilicus, but fine and coarse touch were not impaired, nor were his postural and vibration senses. A diagnosis of anterior spinal artery syndrome was made.

During the ensuing days there was very slight recovery of his left foot in that he developed some eversion, and he also had scattered areas of recovery to pin-prick in both legs. His bladder was treated with an indwelling catheter and became automatic after about five weeks. His bowels were emptied with the aid of enemata. His legs became oedematous and he developed pressure sores over both heels and his sacrum. These healed with ultra-violet light and infra-red irradiation and massage of the surrounding areas, but recurred in the terminal stages.

His congestive heart failure was treated with a salt-free diet, digoxin, and mersalyl, and the anaemia, which on occasion reached a level of 50% haemoglobin, was treated with intravenous iron and blood transfusions. He also had recurrent attacks of subacute intestinal obstruction relieved by enemata. Two months after the onset of the paraplegia he developed a urinary infection with *Escherichia coli* and, later, a proteus infection which resisted all forms of treatment. His blood urea gradually rose and his general condition deteriorated; finally he became uraemic, with a blood-urea level of 100 mg. per 100 ml., and he died on December 13, 1957.

Post-mortem Findings

The body was that of a small, thin old man with oedema of the legs and sacral and gluteal pressure sores. The heart was slightly enlarged, weighing 300 g. The myocardium was free from scarring, the valves were competent, and the coronary arteries showed only a mild degree of atheroma. In the left lower lobe pulmonary artery there was a recent pulmonary embolus occluding the lumen. The lungs were small and collapsed, with gross bullous emphysema, most marked on the left, the largest bulla being the size of a tangerine. The liver, spleen, and pancreas were normal. The kidneys were slightly enlarged, the capsule of the left stripping easily, but that on the right stripped to reveal multiple abscesses. The left renal pelvis was capacious, leading to a dilated ureter. On the right side, section of the kidney revealed a pyonephrosis with considerable excavation of the parenchyma. The ureter, too, was dilated, with peri-ureteric fibrosis and mucosal ulceration, the dilatation stopping abruptly at the bladder wall. No stone was found in the urinary tract. The bladder, distended with turbid infected urine, had a thin trabeculated wall with multiple diverticula. The prostate was small and adenomatous. The alimentary tract showed no abnormality, the site of the anastomosis being identified with difficulty by the presence of few silk sutures. The brain was normal; no vascular disease of the vessels was seen. No abnormality was found in the spinal canal. There was no evidence of arachnoiditis; the only comment was that the lumbar swelling was smaller than normal.

Sections were taken at each segmental level in the cord and stained with Ehrlich's haematoxylin and eosin and methasol. The pia-arachnoid was normal. Apart from

mild hyaline change in some of the veins and a short segment of medial thickening in the anterior spinal artery, no other vascular abnormality was found. The spinal cord from T 10 downwards showed a bilaterally symmetrical degeneration of uniform severity (see Fig.) The anterior horns were atrophied, with loss of motor neurones, and showed a round-celled infiltration. The lateral and anterior columns showed incomplete demyelination, the pyramidal tracts being spared. The posterior columns were unaffected. The sections from the segments below T 10 showed that these changes were clearly confined to the anterior part of the cord in the distribution of the anterior spinal artery.

Transverse section of spinal cord at the level of S 2, showing the symmetrical degeneration of the cord in the distribution of the anterior spinal artery. (Stained with methasol. $\times 6$.)

Discussion

In this patient the choice of anaesthetic was based on the following considerations. The state of his cardiovascular system, together with the bronchopneumonia and severe emphysematous changes in his lungs, made it appear wiser to use some form of local anaesthetic. Also, there was the added hazard of inhalation of vomit which might well have occurred under general anaesthesia. The choice of local anaesthetic in our minds lay between subcutaneous infiltration and local nerve block, spinal or epidural. It was thought that the inflammatory changes present over the hernial sac and inguinal ligament were sufficient to make it inadvisable to use either the subcutaneous method or the local nerve block. Spinal anaesthesia is less often used in this country, and was rejected because of its risk of nervous sequelae. Epidural anaesthesia was used because of the advantage of obtaining a pure sensory block, thus obviating any respiratory embarrassment due to motor paralysis. In addition, the unexplained bronchodilator effect of the epidural anaesthetic would be beneficial to this patient's emphysematous condition (Bromage, 1954). These advantages were considered to outweigh the risk of hypotension that might result. It was possible to prevent a further fall in blood pressure by using a moderate dose of ephedrine, without having to resort to the stronger vasopressor drugs, such as noradrenaline.

From the clinical picture and the pathological and histological findings our patient was quite clearly suffering from the syndrome of anterior spinal artery occlusion. Other causes for the paraplegia that might be related to the anaesthetic technique, such as disk protrusion, extradural haemorrhage, and haematomyelia, were considered. The disk protrusion could have been caused by flexion of the spine during the induction of anaesthesia. Extradural haemorrhage either spontaneously occurring through twisting or straining (Schultz *et al.*, 1953) or traumatic in origin by damage to the spinal veins, may have resulted. Haematomyelia may have been caused by damaging the cord. All these possible events were excluded on the

pathological findings. Further consideration must be given to the possibility that an accidental spinal anaesthesia was induced, causing the paraplegia. On clinical grounds this was excluded because the patient was able to move his legs some while after the epidural anaesthetic was given. But Sykes (1958) points out that this sign is not infallible, and a spinal anaesthesia may have resulted through seepage via a small dural puncture. A total spinal anaesthesia can be excluded on the grounds that there was no respiratory paralysis, no profound fall in blood pressure, or, as would be expected with a quantity of anaesthetic of this magnitude, no loss of consciousness and no pupil signs developed.

Two further causes of the neurological damage—chemical contamination of the spinal cord and infection—have to be excluded. The former can be ruled out because no chemical agent was used to sterilize the anaesthetic solution or the instruments. The possibility of the introduction of infection does not seem likely, because of the aseptic technique adopted. This consisted of autoclaving drums (30 minutes at 20 lb.), the use of sterile gowns and gloves, and the preparation of the skin over the lumbar region with Bonney's blue and 70% surgical spirit. Clinically, the onset of the paraplegia was rapid enough to make it unlikely that there was an infective cause. Furthermore, the absence of adhesive arachnoiditis at post-mortem examination ruled out both these possibilities.

It should also be mentioned that cases of paraplegia have been reported as occurring after general anaesthesia. These were due to either a thrombosis following hypotension (Ciliberti, 1948; Ditzler and McIver, 1956) or to air embolism during the induction of an artificial pneumothorax (Wikler *et al.*, 1937) or injury to the pulmonary vein during thoracotomy (Thomas and Dwyer, 1950).

Finally, the epidural anaesthetic as the prime cause of the paraplegia must be discussed. Because of the known hypotensive action of an epidural anaesthetic, precautions were taken at the onset to prevent a severe fall in blood pressure. However, a systolic pressure between 80 and 100 mm. Hg might well have been too low to maintain an adequate blood supply to the cord with his already failing circulation. This might possibly have led to stasis and subsequent thrombosis in the vessels. We are aware of a similar case where a temporary paraplegia resulted from the use of an epidural anaesthetic to provide controlled hypotension for a total parotidectomy (Massey Dawkins, 1958, personal communication).

The stasis might have been produced by compression of the spinal vessels by the volume of local anaesthetic solution introduced into the epidural space. This almost certainly can be excluded, for there is easy dispersion of the solution through this space and the paravertebral foramina. Furthermore, it would be more likely for the posterior spinal vessels to be affected rather than the anterior.

Many authorities have suggested that the local anaesthetic itself may cause permanent neurological damage (Payne and Bergentz, 1956). Frumin *et al.* (1953) have produced strong experimental evidence that the anaesthetic solution will diffuse from the epidural space into the spinal canal. Most of the local anaesthetic drugs have been incriminated in producing cord damage, but, so far as we can ascertain, no report following the use of lignocaine hydrochloride has been made. However, it would seem likely that this too might have a similar action on nervous tissue. From the clinical signs and the pathological findings of damage solely in the distribution of the anterior spinal artery, and lack of arachnoiditis, it is unlikely that this was a direct toxic action of the drugs on the cord.

A further possible cause is the vasoconstrictor action of adrenaline. The solution used during the anaesthetic procedure contained 1 in 300,000 adrenaline. Ruben *et al.* (1948) reported the spinal anaesthetic effects in the human following the use of vasoconstrictor substances alone. Priddle and Andros (1950), using adrenaline solution of 1 in 2,500 dilution, produced hypoaesthesia in the saddle area

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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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