

A STUDY OF 49 PATIENTS WITH ACUTE SPINAL EXTRADURAL ABSCESS

By D. O. HANCOCK, F.R.C.S.

National Spinal Injuries Centre, Stoke Mandeville Hospital

INTRODUCTION

ONE of the earliest references to spinal extradural abscess (S.E.D.A.) is by Morgagni in his *De Sedibus Et Causis Morborum* dated 1796. The next occurs in 1820, the references thereafter increasing steadily in number through the nineteenth and twentieth centuries. By 1958 records included 270 examples of the disease (Korbin, 1958).

The clinical pattern of this condition is by now clearly established. Superficial peripheral sepsis is the commonest source of infection, which, on occasions in association with mild trauma, may precede by only a few days or several weeks the appearance of pain in the midline of the back. Radiation due to root involvement usually follows and objective neurological involvement is added, commonly within three weeks of the onset of pain. The clinical signs are also clear; localised spinal tenderness, frequently found at the site of the abscess; neck stiffness; severe pain on attempting spinal flexion (Stammers, 1938) and fever with leucocytosis. These are the cardinal signs which should lead to laminectomy.

Unfortunately the diagnosis is made more often after the appearance of neurological signs, at which time the prognosis for recovery is worsened (Rankin & Flothow, 1946; Heusner, 1948; Hulme & Dott, 1954). Browder and Meyers (1937) commented that the most frequent error in diagnosis is failure to think of the condition. Basically it is a painful, febrile, spinal syndrome.

Paraplegia is permanent in approximately 60 per cent. of sufferers from S.E.D.A. (Gasul and Jaffe, 1935; Campbell, 1937). Hulme and Dott (1954) found that full or partial recovery occurred in 44 per cent. of their patients. However, Dandy (1926) in a review of the existing literature found that from 1833-1923 S.E.D.A. had proved fatal in 23 of 25 patients.

In the present series no deaths occurred which were directly attributable to S.E.D.A., a change undoubtedly related to the introduction of antibiotics and the appreciation of the need to operate.

One of the most important pathological observations was by Browder and Meyers (1941) who found that in seven autopsies there was no compression deformation of the spinal cord. Softening at the level of the abscess was common and histological examination pointed to impairment of the intrinsic circulation of the cord as the pathogenetic mechanism; a theory now generally accepted.

THE SERIES

This report concerns 49 patients admitted to the National Spinal Injuries Centre between July 1945 and November 1968. There were 25 females and 24 males involved, their ages ranging from 11 to 69 years. The annual rate of incidence between 1940-49 was not more than two, between 1950-59 not more than four and

between 1960-66 not more than five. The presence of an acute spinal extradural abscess was proved by operation in every patient.

Presenting Features. Spinal pain was invariable, though records of spinal tenderness were complete in only 24 patients, being present in the region of the abscess in 20 patients, five of these had neck stiffness. Tenderness was absent in the remaining four. Neck stiffness alone was found in four patients, only one of which had a cervical abscess.

The interval between the onset of spinal pain and the appearance of paralysis varied from one to 23 days.

All except one patient showed complete or partial limb paralysis before operation. Thirty-two lesions were complete and 21 of them remained so after operation. Of nine lesions neurologically incomplete before operation, all remained so afterwards. Forty-six patients had dorsal and three had cervical, neurological syndromes.

There was no record of the pre-operative neurological status in eight patients; four of these were complete post-operatively and two of the quartet of patients with incomplete lesions were able to walk without the aid of calipers.

Peripheral Sepsis. There was found to be peripheral sepsis in 26 patients, and none in 21. No information was recorded on the remaining two patients. Pus was most commonly found between the sixth and eleventh dorsal vertebrae, the most commonest site being at D9 (21 instances). The interval between the appearance of peripheral sepsis and the onset of paralysis varied from one week to three months.

The sepsis was within the area of drainage of the superior vena cava in 18 patients and of the inferior vena cava in five (one patient had a septic lesion in each area; another suffered a septic abortion two months before the diagnosis of S.E.D.A. and chickenpox two weeks before laminectomy; in a third the site was not recorded). The site of the extradural pus bore no relation to the situation of the peripheral sepsis. One patient had a septic lesion on the 'back' though none had such an infection on or near the spine.

Trauma. There was a clear history of trauma to the back shortly before the onset of the syndrome in five patients, and of trauma which may have involved the back in three more. None of these patients had any evidence of peripheral sepsis. Three further patients suffered both trauma and peripheral sepsis, one of them receiving a blow on the spine.

OPERATIVE FINDINGS

The longest interval between the onset of the paralysis and operation was three months, the shortest a few hours.

Granulation tissue as well as pus was present in 15 patients. In this group the interval between the onset of back pain and laminectomy varied between one day and four months. This interval varied from four days to four weeks in 29 patients in whom there was no granulation tissue. *Staphylococcus aureus* was recorded as the infecting organism in 31 cases.

Pus was anteriorly placed in only three patients and two of these who had pus in the cervical extradural space had evidence of *vertebral osteomyelitis*. Osteo-

myelitis was probably present in no more than four others who unlike the two already mentioned did not have any peripheral sepsis.

Post-Operative Improvement. Post-operative improvement of three segments or more in the neurological level, or a change from a complete to an incomplete lesion, occurred in 20 patients. Of the five patients able to walk without the use of calipers, two had a complete motor paralysis pre-operatively; two were neurologically incomplete and in one there was no pre-operative record.

The least paralysed patient presented with *paralytic ileus* and urinary retention only; immediately after her operation she had a complete paraplegia below T12, walked unsupported a year later but four years after that developed an incapacitating spasticity.

CONCLUSIONS

Clearly this series is specially selected in being referred to the National Spinal Injuries Centre and useful limb recovery would not be expected to be frequent. In addition, all but one patient had signs of limb paralysis before operation, which is of bad prognostic import.

S.E.D.A. may occur at any age and is uncommon but not rare. This condition should be considered whenever a painful, febrile, spinal syndrome is seen in a patient. As soon as the diagnosis is entertained it must be immediately pursued by myelography and laminectomy. The appearance of neurological abnormalities worsens the prognosis and delay in operation may be disastrous.

Only four patients in this group had this operation on the same day as the appearance of neurological signs, while operation was on the second day or later in 33—a delay probably accounting for the preponderance of persisting paralysis in the whole series. Even the four patients operated on quickly remained paralysed!

There is no correlation between the age of the lesion and its appearance at operation. Heusner (1948) pointed out that it was not possible to predict whether abscess or granuloma would be found.

The infrequency of cervical syndromes reflects the anatomy of the extradural space which is only potential in the neck and increases in depth from the seventh cervical vertebra downwards to reach its maximum between the fourth and eighth dorsal vertebrae (Dandy, 1926). It is important to note the delay between the appearance of peripheral sepsis and paralysis. In the group described it extended for as long as three months. It is probable that in several instances the patient forgot about the infection and specific enquiry should always be made about this.

In any situation, operation is essential—the mortality without it is close to 100 per cent. (Korbin, 1958). Completely paralysed patients may recover fully and at the very least this series has shown that nine patients suffered incomplete syndromes throughout their illness.

Spinal tenderness was absent in only four patients, though all of these complained of spinal pain. Neck stiffness is a common accompaniment and may be the only local sign present on spinal examination.

Trauma was recognised as a predisposing cause in 1925 by Schmalz—perhaps local bleeding occurs in the extradural space to produce a locus minoris. Eleven of this group suffered such trauma and paralysis occurred between one and six weeks after it.

SUMMARY

Forty-nine patients (25 female, 24 male) whose abscess was diagnosed at laminectomy were studied. Their ages ranged from 11 to 69 years. The interval between the onset of the paralysis and operation varied between a few hours and three months.

Forty-six patients had dorsal and three had cervical neurological syndromes. All patients suffered back pain. Where the records showed it, 31 of the abscesses were infected with *Staphylococcus aureus*.

There was a history of peripheral sepsis in 26 patients and no evidence of sepsis in 21.

There was a history of trauma only, probably affecting the back, in eight patients; and of trauma and peripheral sepsis in three. Only five patients walked without the use of calipers post-operatively; one of these subsequently deteriorated.

REFERENCES

- BERGAMASHI, G. (1820). Sulla mielitide stenica e sul tetano, loro identia. *Thesis: Parvia*, G. Torri.
- BROWDER, J. & MEYERS, R. (1937). *Amer. J. Surg.* **37**, 4.
- BROWDER, J. & MEYERS, R. (1941). *Surgery*, **10**, 296.
- CAMPBELL, M. M. (1937). *Bull. neurol. Inst. N.Y.* **6**, 574.
- DANDY, W. E. (1926). *Arch. Surg. Chicago*, **13**, 477.
- GASUL, B. M. & JAFFE, R. H. (1935). *Arch. Paediat.* **52**, 361.
- HEUSNER, A. P. (1948). *New Engl. J. Med.* **239**, 845.
- HULME, A. & DOTT, N. M. (1954). *Brit. Med. J.* **i**, 64.
- KORBIN, W. M. (1958). *Bull. Los. Ang. neurol. Soc.* **23**, 21.
- MORGAGNI, G. B. (1796). De Sedibus et Causis morborum per anatomem indagatis. *Epist. X. art. 13*. (Translated by B. Alexander.)
- RANKIN, R. M. & FLOTHOW, P. G. (1946). *West. J. Surg.* **54**, 320.
- SCHMALZ, A. (1925). *Virchow's Arch. f. Path. Anat. u. Physiol.* **257**, 520.
- STAMMERS, F. A. R. (1938). *Brit. J. Surg.* **26**, 366.