

Extradural cysts of the spinal canal

PETER GORTVAI

From the National Hospital for Nervous Diseases, Queen Square, London

Extradural cysts are rare, space-occupying lesions of the spinal canal. In 1955 Wise and Foster summarized 33 cases from the literature and added one of their own. Nugent, Odom, and Woodhall reviewed the same cases in 1959, adding several more from their own experience and from the literature.

The literature of these lesions is still limited. Fifty-six cases were found described, including those previously reviewed. There are only two cases of

cervical cyst among them. The purpose of this paper is to add five cases to the literature, two of which are cervical, and to contribute to the discussion on the clinical aspects, aetiology, treatment, and prognosis of these lesions.

CASE REPORTS

CASE 1 A woman of 53 years (hospital no. 95361) had been complaining of an intermittent dull ache in the back



FIG. 1

FIG. 1. *Myelogram of case 1, showing the cervical extradural cyst with a fluid level.*

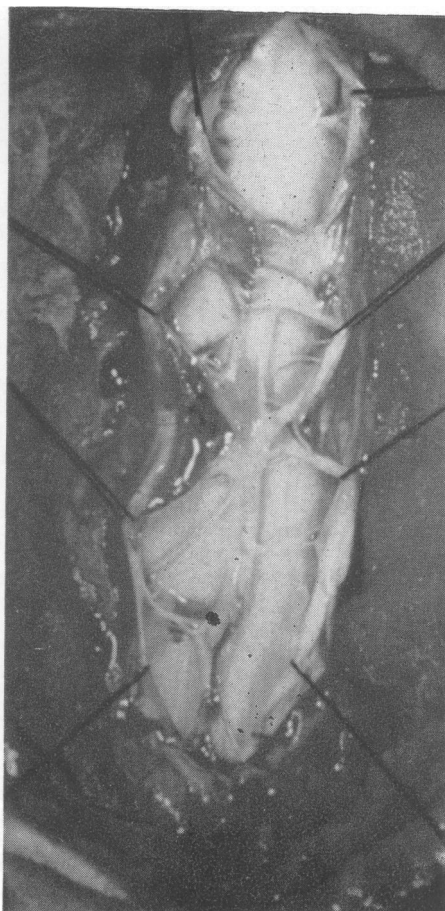


FIG. 2

FIG. 2. *The interior of the cervical cyst in case 1, showing fibrous strands.*



FIG. 3

FIG. 3. Tomogram of case 2, showing wide separation between the anterior arch of the atlas and the odontoid process. There are large areas of destroyed bone in the second and third cervical vertebrae.

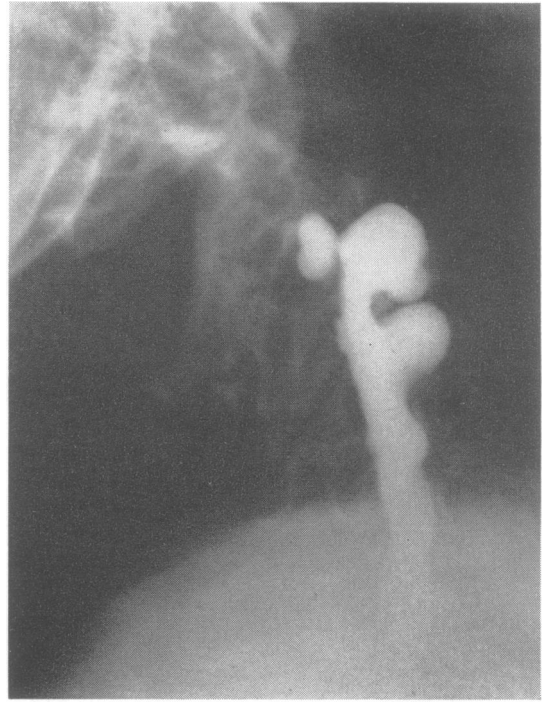


FIG. 4

FIG. 4. Myelogram of cervical cyst in case 2.

of the neck for seven months, followed by weakness and stiffness of the legs.

The pupils were small but reacted normally to light and accommodation. There was slight bilateral ptosis. The arms were weak, with muscular wasting. The biceps and triceps reflexes were absent on the right. The lower limbs were spastic and weak, with extensor plantar reflexes. Sensation to pain and temperature was impaired in a patchy manner below an ill-defined level at the cervico-dorsal junction and vibration sense was impaired in all four limbs.

Lumbar puncture yielded clear colourless fluid under 90 mm. pressure, and on bilateral jugular compression there was an incomplete manometric block. The fluid contained 120 mg. of protein in 100 ml. and no cells.

On myelography there was almost complete obstruction to the flow of the contrast medium at C.3. In the lower cervical region a constant remnant of the contrast medium could be demonstrated in all positions of the patient, with a fluid level (Fig. 1).

At operation, on removing the third, fourth, and fifth cervical spines and laminae, an irregular cystic swelling was seen filling the limits of the exposure, which was opened in the midline. A loculated space was entered, with soft septa and fibrous strands running between the walls of the cyst, dividing it into incomplete compartments (Fig. 2). The spinal cord itself appeared to be quite

normal in size and position. The superficial wall of the cyst was excised and the deep layer, which had fused with the dura, was united with fine interrupted sutures.

The excised cyst wall was examined by Professor Blackwood. His report was: 'There is an outer irregularly orientated layer of fibrovascular tissue and an inner, thick, poorly nucleated layer of collagenous tissue with the fibres running parallel to the inner smooth surface.'

Three weeks after operation the patient was walking without help and there was further slow but steady improvement in the power of the arms. When seen six months after her operation there was no weakness of the lower limbs, although vibration sense was still diminished in the right leg and the right plantar response was extensor. She was walking almost normally and she had returned to her normal household duties.

CASE 2 A woman of 23 years (hospital no. 78955) was admitted on 15 May 1958, complaining of stiffness and loss of power in the left arm and leg for the last two years and pain in the right foot of six months' duration.

On examination no Horner's syndrome was noted. The limbs on the left side were spastic with generalized weakness. The right limbs were spastic and weak to a lesser extent. All tendon reflexes were exaggerated. Impaired sensation to pinprick was noted over the left side of the body up to the level of the second cervical

dermatome. Joint position sense was impaired in the feet.

Radiological examination revealed gross separation between the posterior arch of the atlas and the odontoid process. There were large areas in which bone was destroyed, involving the second and third cervical vertebrae (Fig. 3).

The cerebrospinal fluid appeared normal and contained 35 mg. of protein in 100 ml.

On myelography a hold-up of the contrast medium was demonstrated at the level of the atlas with extension of the neck but free passage with flexion. A large sac was outlined at this level in communication with the subarachnoid space, with diverticula into the second and third cervical foramina on the left side (Fig. 4).

Laminectomy was undertaken, removing the first to the fourth arches. A large cyst was found immediately below the foramen magnum, extending to the fifth cervical lamina. The cyst sent prolongations into all cervical root canals along the nerves. The posterior arch of the axis possessed unnatural mobility in the vertical plane. The cyst wall was widely incised and left open. The musculature and skin were closed in layers. At the conclusion of the operation skull traction was applied by a caliper.

Ten days later the right atlanto-occipital joint was fused by removal of the articular cartilage and packing the joint with bone chips obtained from the tip of the mastoid process. After a further 10 days the left atlanto-occipital joint was fused in the same manner. Seventeen days later the posterior cervical incision was re-opened and a longitudinally split rib inserted on either side, wiring the graft to the squama of the occipital bone. Skull traction was discontinued and a plaster cast was applied encircling the head and extending down to the angles of the scapulae. Eight weeks after the last operation the plaster cast was replaced by a small cervical support. Long before that the patient was walking well unaided.

When seen six months post-operatively no abnormality was detected in the right limbs. The left hand was a little clumsy and weak. The left leg showed no weakness but the left plantar response was still extensor. She was getting about normally and doing all her housework.

CASE 3 A man of 40 years (hospital no. 78439) was admitted in April 1958, complaining of periodic constipation and colicky abdominal pain. There was a mass in the abdomen arising from the pelvis to 3 in. above the pubis. On rectal examination a cystic tumour was felt. There were no abnormal neurological signs.

A radiograph of the lumbo-sacral spine showed a large defect of the left side of the sacrum. Examination of the clear and colourless cerebrospinal fluid yielded normal results. Myelography demonstrated a large cavity in the pelvis communicating with the subarachnoid space.

Laminectomy was performed from L.5 to S.2. Below this level the sacrum was replaced posteriorly by a tough membrane. This was incised in the midline almost to the coccyx. The sacral canal was filled with a large cyst which extended anteriorly well into the true pelvis. The dural sac itself ended normally at S.2, whence the filum terminale issued and ran over the posterior aspect of the cyst wall. The cyst contained straw-coloured cerebrospinal

fluid which on subsequent analysis was found to contain 720 mg. of protein in 100 ml. Dissection of the neck of the sac exposed its communication with the subarachnoid space at the end of the normal dural sac at the level of the second sacral vertebra. The neck was in the midline 1½ cm. long, as wide as a match-stick. It was thought that obliteration of the neck would prevent its future filling, making the almost impossible excision unnecessary. This was achieved by co-apting the walls of the neck by sutures and inserting a piece of crushed muscle.

The patient made an uneventful recovery. A fortnight after operation the only residual deficiency was a small area of anaesthesia to the right of the anus, probably representing the fourth sacral dermatome.

The patient was seen at regular intervals for two years. There were no physical signs apart from the small patch of anaesthesia. He has remained free from symptoms.

CASE 4 A man of 64 years (hospital no. 94236) was admitted to hospital on 8 August 1960. Ten years previously he developed pain in the small of the back and in the left leg. For the last 18 months both legs had become progressively weak.

On clinical examination of the spine no abnormality was detected. The lower limbs were spastic, more so on the left than on the right. Muscular power was impaired to the extent of just being able to lift the feet off the bed. The plantar responses were extensor. Sensation to pinprick was impaired from the groin downwards and it was

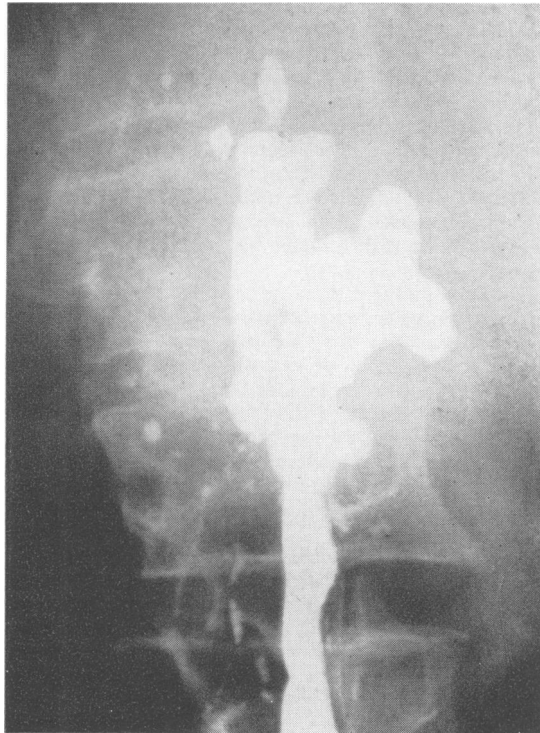


FIG. 5. Myelographic appearance in case 4.

densest over the fifth lumbar and first sacral dermatomes. Position sense and vibration sense were grossly impaired in both legs.

A radiograph of the thoraco-lumbar spine demonstrated erosion of the posterior aspects of the vertebral bodies from D.10 to L.1. On lumbar puncture no obstruction to the flow of clear cerebrospinal fluid could be demonstrated. The protein and cellular contents of the fluid were normal.

Myelography showed cystic dilatations communicating with the subarachnoid space in the dorso-lumbar area. The cysts were irregular, some were loculated, and they were considerably more extensive on the left side than on the right (Fig. 5).

At laminectomy the spines and laminae of D.11 to L.2 were removed. The ligamenta flava were greatly thickened. The extradural space was filled with sacs of thin membrane. All the sacs were removed without difficulty, leaving the dura looking quite normal. Sections of the wall of the cyst were examined by Dr. Mair, who reported that the material consisted of dense collagen. It was not infiltrated by any cellular elements.

Two months after operation sensation to pinprick was normal over the left lower limb but remained slightly impaired on the right. The muscular power remained unchanged.

CASE 5 A police constable of 33 years (hospital no. 88148) was admitted on 17 November 1959. For three years he had been suffering from a constant ache in the lower part of the back, worse on standing and walking. Gradually the pain increased in severity and spread diffusely along the back of both thighs. The patient had a mild mid-dorsal kyphosis. There were no abnormal neurological signs.

Lumbar puncture yielded clear and colourless fluid containing no cells. The protein was 30 mg. in 100 ml.

In view of the patient's long-standing, persistent symptoms myelography was performed. With the patient in the erect position, cystic spaces, apparently multilocular, were filled with the contrast medium in the sacral region.

At operation the fifth lumbar lamina and the adjacent three sacral posterior arches were removed. The second and third posterior bony arches were markedly thin. A large multilocular, bluish cyst was seen filling the sacral canal (Fig. 6). The cyst extended into the sacral foramina on both sides. The theca and the cyst were opened in the midline to go over a narrow communication between the two structures. The filum terminale was found and around it ran the narrow canal from the theca into the cyst. This communication was obliterated by interrupted fine sutures.

The patient was discharged from hospital on the eighteenth post-operative day free of pain and walking normally. In a few weeks he developed a multiplicity of symptoms, including backache, and he remains incapacitated.

DISCUSSION

Wise and Foster (1955) summarized 34 cases in the form of a table. A summary of their table and its

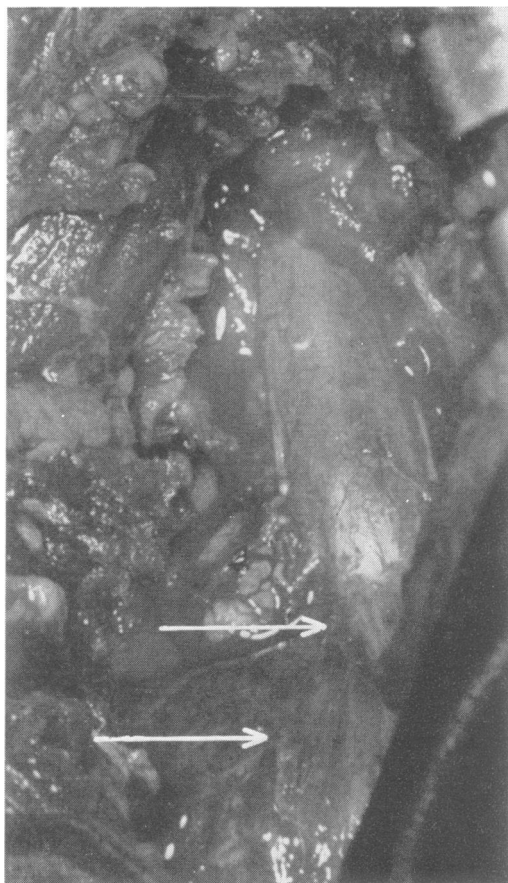


FIG. 6. Sacral cyst of case 5, displayed at operation. The marker above points to the communication between the theca and the cyst and the one below to the cyst itself.

continuation by further cases is given in Table I. (In one case of Teachanor no details were available.) These 60 cases will be analysed.

AGE INCIDENCE The age of the patients at the time of their presentation is shown in Figure 7.

One-third of the cases occurred in the first half of the 'teens, and apart from the extremes of the life span the rest of the cases are fairly evenly distributed.

SITE OF THE LESION Most lesions extended over more than one vertebra. The incidence of involvement of each vertebral level is given in Figure 8.

The lower half of the thoracic column is involved most frequently, the upper half of the thoracic region and the lumbar region fairly commonly, while the disease is rare in the cervical and sacral regions.

TABLE I

SUMMARY OF CASES IN THE LITERATURE

Age	Sex	Duration	Site	Remissions	Communication	Block	Kyphosis	
<i>Wise and Foster (1955)</i> 34 Cases	Average 21.9 yr. Range 6 to 48 yr.	M 23 F 10	Range 3 mth. to 16 yr.	Yes 13 No 18	Yes 11 No 9	Yes 13 No 6 Partial 5	Yes 19 No 11	
Schmidt (1904)	16	M	18 mth.	D4-8	No	?	Yes	?
Roger <i>et al.</i> (1933)	25	F	2 yr.	D6-8	No	?	Yes	No
Guillaume <i>et al.</i> (1947)	14	M	17 yr.	D6-9	No	No	Yes	Yes
Olsson (1948)	13	F	1 yr.	D9-11	No	No	No	Yes
Rouquès <i>et al.</i> (1948)	14	M	11 mth.	D5-8	Yes	No	Yes	Yes
O'Connell (1953)	46	F	6 mth.	L5-S1	No	Yes	—	—
Jacobs <i>et al.</i> (1954)	22	M	9 yr.	T6-8	Yes	Yes	Yes	Yes
Strully <i>et al.</i> (1954)	36	M	2 yr.	S2	No	Yes	—	—
Strully <i>et al.</i> (1954)	43	F	Many years	S3	No	Yes	—	—
Strully <i>et al.</i> (1954)	37	F	2 yr.	?	No	?	?	?
Strully <i>et al.</i> (1954)	57	F	Short	S2-3	No	Yes	—	—
Strully <i>et al.</i> (1954)	32	F	2 yr.	L5-S1	No	Yes	—	—
Cuneo (1955)	48	M	—	D5-8	No	Yes	—	—
Balestrieri (1958)	19	F	1 yr.	D1-10	No	Yes	?	Yes
Garcin <i>et al.</i> (1958)	6	M	1 yr.	D5-10	No	No	Yes	No
Nugent <i>et al.</i> (1959)	29	F	4 yr.	L1-4	No	Yes	No	No
Nugent <i>et al.</i> (1959)	43	F	7 yr.	D6-10	Yes	No	Yes	Yes
Nugent <i>et al.</i> (1959)	50	M	28 yrs.	C7-T1	Yes	No	No	No
Nugent <i>et al.</i> (1959)	13	M	3 yr.	T8-9	No	No	Yes	Yes
Nugent <i>et al.</i> (1959)	28	M	7 yr.	T8-10	Yes	No	Yes	No
Nugent <i>et al.</i> (1959)	46	M	5 yr.	T8-10	Yes	No	Yes	No
Nugent <i>et al.</i> (1959)	14	M	8 wk.	T6-8	Yes	Yes	Yes	Yes
<i>Present series</i>								
Case 1	53	M	7 mth.	C3-C5	No	Yes	—	—
Case 2	23	F	23 yr.	C1-C4	No	Yes	—	—
Case 3	40	M	2 yr.	S2-S4	Yes	Yes	—	—
Case 4	64	M	10 yr.	T10-L1	No	Yes	—	—
Case 5	33	M	3 yr.	S2-S3	No	Yes	—	Yes

CLINICAL FEATURES The history of symptoms may be as short as a few weeks or as long as a few years. It more commonly extends over several years.

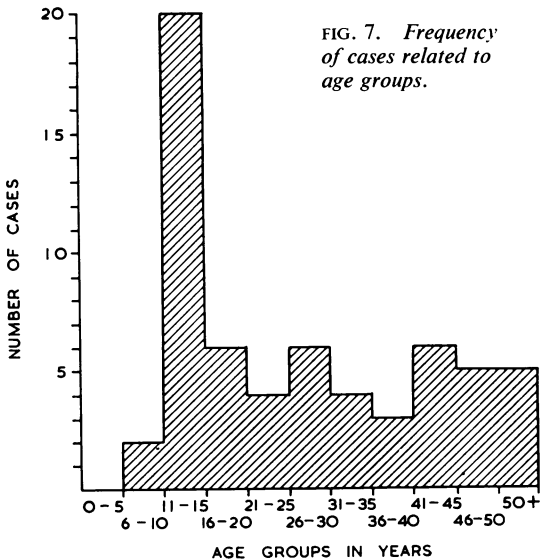
Remissions occurred in 21 of the cases but there were no remissions in thirty-five. Elsberg, Dyke, and

Brewer (1934) place much importance on the history of spontaneous remissions in their description of the clinical syndrome, but this is commonly absent. When such a history is obtained, it does help in the clinical differentiation of the disease from a neoplasm, although remissions may occur rarely in the history of spinal neoplasms (Connolly, 1962).

Pain is quite common and it may be a dull backache or sharp root pains radiating in a segmental distribution.

The neurological picture is commonly that of spinal cord compression of variable severity. The motor signs are usually more marked than the sensory signs but a sensory level may be found. A segmental distribution of sensory loss may also be observed when there is involvement of nerve roots, as in case 4 described here. One of our cases of cervical cyst had Horner's syndrome, just as both previously described cases.

In the majority of patients plain radiographs of the spinal column showed erosion of the pedicles or scalloping of the vertebral bodies. The cyst was in communication with the subarachnoid space in 26 cases. Not all of these had myelographic studies. It is possible that with careful technique the majority of cysts would have filled with contrast medium. In all our cases the cyst filled and a correct pre-operative diagnosis was possible.



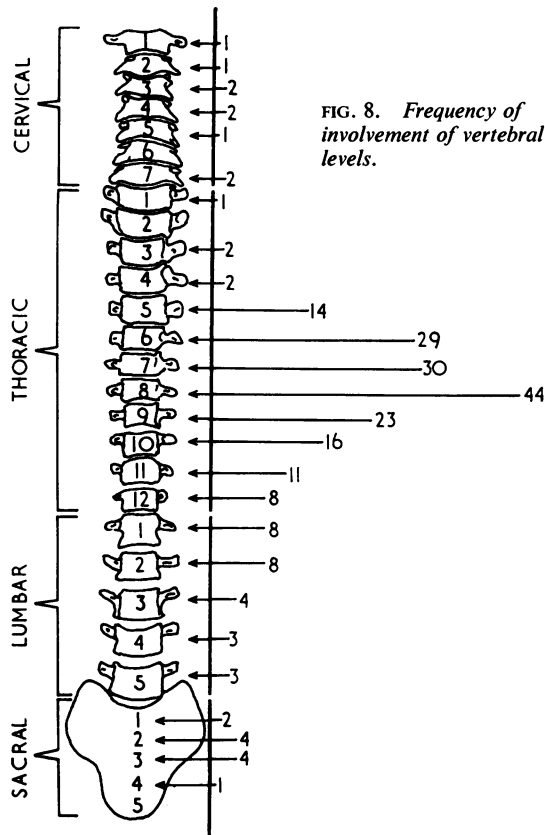


FIG. 8. Frequency of involvement of vertebral levels.

THE ORIGIN OF EXTRADURAL CYSTS Elsberg *et al.* (1934) considered the extradural cysts they described to be congenital, either arising as a congenital diverticulum of the dura, or as a protrusion of the arachnoid through a congenitally weak place in the dura. Nugent *et al.* (1959), in their review of spinal extradural cysts, also thought that the cysts were congenital. Their main argument in favour is the work of Rexed (1947) who found areas of intense proliferation of the arachnoid around human spinal nerve roots which in some cases were connected to small cysts. Nugent *et al.* (1959) point out that the cysts may arise at the midline of the dural sac, at the attachment of the dural sleeve covering a root at its attachment to the dural sac, or in the dural sleeve of the root itself. Rexed's observation may support the congenital origin of the second and third groups. In my case 2 the cyst included dilated dural sleeves, in cases 1 and 3 the cyst communicated with the subarachnoid space in the region of spinal nerve roots, and in case 5 the communication was around the filum terminale.

It is reasonable to suppose that those cysts which

communicate with the subarachnoid space near the midline are also congenital in origin. Embryonic structures fuse in the midline to cover the neural tube and a minor disturbance of fusion, sometimes referred to as 'status dysraphicus', may weaken the dura congenitally and predispose to future cyst formation. In one of our patients (case 4) the cysts had such thin walls that they appeared to be protruding sacs of arachnoid through weak spots in the dura. In the case of Balestrieri (1958) the extradural cyst co-existed with other congenital abnormalities, such as atresia of the foramina of Luschka and Magendie (the Dandy-Walker syndrome) and bony malformations. The atresia of the outlet foramina of the fourth ventricle may also be caused by an abnormality of mesodermal fusion over the neural tube. In one of our own patients (case 2) cyst formation was accompanied by subluxation of the odontoid process, probably congenital in origin.

Although it seems that most, if not all, extradural cysts are congenital in origin, it is possible that occasionally they arise as a result of trauma. A history of past injury is not uncommonly given by patients harbouring these cysts, but in most of these cases it is more likely that injury aggravated a pre-existing disorder, or merely called attention to it. Meredith (1940) found haemosiderin in the cyst wall in two cases with a previous history of injury. This observation is often quoted in defence of the theory of a traumatic origin of the cysts, but it is equally feasible that trauma to a congenital cyst caused a slight haemorrhage.

In spite of these considerations one must agree that trauma can cause cyst formation in the extradural space. An operative defect of the dura can lead to the formation of cysts (Hyndman and Gerber, 1946; Schreiber and Nielsen, 1950; Strully and Heiser, 1954). These cysts were caused by herniation of the arachnoid through a substantial dural defect, a state of affairs unlikely to be caused by injuries of a minor nature such as occur in the past histories of patients suffering from spinal extradural cysts.

THE MECHANISM OF SPINAL COMPRESSION Originally the cysts must be merely small diverticula of the subarachnoid space, and while they produce no symptoms they presumably do not compress the spinal cord and do not erode bone. The factors which may cause enlargement of the cyst are (1) the hydrostatic pressure of the cerebrospinal fluid, (2) osmosis of water into the cyst, and (3) secretion by the cyst wall.

Hydrostatic pressure of cerebrospinal fluid Considering enlargement of the cysts by hydrostatic pressure, Schmidt (1904) pointed out long ago that so long as there is free communication the pressure

within the cyst cannot rise above the pressure of cerebrospinal fluid in the subarachnoid space.

The pressure head behind the circulation of cerebrospinal fluid would not alter this consideration so long as there was no compression of the spinal subarachnoid space. Should there be compression of the theca by the cyst, as the cross-sectional area of the main channel decreases, the velocity of flow must increase proportionately (Lewitt, 1945). According to Bernoulli's theorem, and the Venturi principle which can be deduced from it, with the increased velocity of flow at the constriction the hydrostatic pressure falls. This is the familiar principle underlying the construction of the common suction pump connected to a running water tap. It can be seen, therefore, that should a constriction exist, the dynamic pressure of circulating fluid would tend to empty the cyst rather than inflate it.

O'Connell (1953) ascribed the distension of the cysts to the pulsatile nature of cerebrospinal fluid pressure. At the inlet of the communication of the cysts eddies must arise in the flow. Eddies cause a loss of kinetic energy, consequently a drop in pressure. The pulsatile nature of cerebrospinal fluid pressure should again tend to empty the cyst rather than inflate it. If the opening is simple, neither the static pressure of cerebrospinal fluid, nor the dynamic pressure of its circulation, nor the pulsatile nature of the pressure could account for enlargement of the cysts and spinal compression. Instead of a simple opening the communication of the cyst with the subarachnoid space may be valvular. It is conceivable that in the erect posture fluid enters the cyst and when the patient is recumbent the inflated cyst may cause some degree of spinal compression. On straining or coughing, when the cerebrospinal fluid pressure is temporarily raised, fluid may enter the cyst and become trapped by the valvular opening. Should the flap of the valve across the opening of the cyst deflect the flow of cerebrospinal fluid, the pressure in the cyst could rise to the sum of the static pressure and the dynamic pressure which causes circulation of the cerebrospinal fluid. Assuming that the velocity of the circulation is slow, the additional pressure head must be small. Nevertheless, a deflecting valve flap can add to enlargement of the cyst.

Osmosis of water Osmosis of water may act as an adjuvant to the enlargement of the cyst and eventually to the mechanism of spinal compression. The opening of the cyst has to be valvular and not simple because otherwise the osmotic pressure within the cyst could not rise above that of the circulating cerebrospinal fluid. There are no data in the literature to reveal the composition of fluid in the cysts and our own observations are incomplete. It is often mentioned, however, that at operation the cyst

fluid was xanthochromic. Xanthochromia implies concentration and it is possible that the osmotic pressure of xanthochromic fluid is higher than the osmotic pressure of tissue fluid. In one of our patients (case 3) the straw-coloured cyst fluid contained 720 mg. of protein, whereas the protein content of cerebrospinal fluid withdrawn on lumbar puncture was only 35 mg. In this case the existence of a valvular opening of the cyst was demonstrated at operation. The electrolytes in the fluids were not estimated, but it is possible that the osmotic pressure of the xanthochromic cyst fluid exceeded the osmotic pressure of tissue fluid.

Secretion by the cyst wall This as a mechanism for enlargement of the cysts cannot be disproved by existing data. Histological examination of the walls of cysts nearly always proved them in the past to be composed of simple connective tissue, unlikely to possess secretory powers. In two of our patients (cases 1 and 4) the previously reported histological picture was repeated.

In conclusion, there seems to be sufficient evidence to regard the extradural cysts of the spinal canal as congenital in origin. A valvular opening may cause enlargement in later life resulting in gradual spinal compression. A cyst with a simple opening cannot enlarge and cause spinal compression. Osmosis of water may act as an adjuvant to the increase in size of the cysts when the opening is valvular and for a time even after the opening has been shut off.

KYPHOSIS ASSOCIATED WITH LESIONS OF THE SPINAL CORD Cloward and Bucy (1937) first mentioned the association between spinal extradural cysts and juvenile kyphosis. Twenty-eight cases of the 61 under discussion suffered from kyphosis. The kyphosis became worse post-operatively in five (Elsberg *et al.*, 1934, case 1; Lehman, 1935, case 1; Kelly, 1937; Rouquès, Guillaume, Ribadeau-Dumas, and Rogé, 1948; Olsson, 1948). Cloward and Bucy believe that the bony vertebral changes which lead to the development of kyphosis are due to venous stasis caused by pressure of the cysts on the epidural veins. They account for the post-operative progression of kyphosis by a lessened resistance to movement in the spine which has been subjected to laminectomy. In the experience of Shenkin, Horn, and Grant (1945) the development of post-operative kyphosis is not rare in young persons after dorsal laminectomy. In Olsson's view (1948) the kyphosis is due to the laminae and ligamenta flava.

It seems that although cysts of the spinal cord, especially when situated in the thoracic region, are not uncommonly associated with kyphosis, the development of kyphosis is not restricted to cystic lesions. It is more likely that the development of

kyphosis is due to damage to the nervous system. Partial segmental denervation of the erector spinae musculature causes imbalance of movement and diminished protection against minor trauma. These factors will eventually lead to deformation of the vertebrae and kyphosis. In support of this theory is the development of kyphosis in non-space-occupying diseases of the thoracic cord and the occasional progression of the kyphosis after the removal of a spinal tumour.

TREATMENT The only definitive treatment possible is operation and relief from compression. Operation should be undertaken when the patient is disabled by the disease.

The cysts are sometimes easily separable from the dura although in some cases they were found to be closely adherent. The lesions were always posterior and thus laminectomy gave good exposure.

When feasible the cyst is removed. Of our own patients, case 4 was treated in this way. When the cyst is extensive and removal could not be easily accomplished, excision of the accessible portion of the cyst wall may be sufficient. This was done in case 1 and was followed by gradual improvement in the patient's neurological state. The communication with the normal subarachnoid space was ignored but there was no leak of cerebrospinal fluid post-operatively. Presumably the communication was small and eventually it became obliterated. It is possible that the communication is displayed with relative ease at operation but removal of the entire lesion is not practicable as in our case 3. Emptying of the cyst followed by obliteration of the communication by suture and a free muscle graft resulted in a permanent cure.

PROGNOSIS The prognosis seems fair on reading the case reports in the literature. This is borne out by our own experience. Three of our five patients were significantly improved by operation. The fourth patient was improved two months after operation but was not helped much as his lower limbs remained weak. The fifth patient, who suffered only from pain in the back, was not relieved by operation.

SUMMARY

Five cases of extradural spinal cysts of the spinal canal are added to the 56 previously described in the literature. In two of the five cases the lesion was in the cervical region, which has only been reported twice previously.

One-third of the 61 cases occurred in adolescents in the first half of the teens but no age is exempt.

Most lesions occur in the mid-thoracic region. Cervical and sacral lesions are rare.

The majority of cases presented as compression of the cord. Pain was not a rare feature. An intermittent history is helpful but not diagnostic. Myelography establishes the diagnosis if the cysts can be filled with contrast medium.

The cysts are considered to be congenital in origin. When the opening of the cyst is simple, hydrostatic pressure alone is inadequate for their production or enlargement. A valvular opening may aid enlargement of the cyst and the production of spinal compression. Osmosis may play a part in the enlargement of the cysts.

The association of kyphosis with the lesions is thought to be due to damage to nervous tissue.

Operation for the neurologically disabled patient carries a fair prognosis: the cyst is removed or drained and its re-forming prevented.

The patients reported in this paper were treated under the care of Mr. Harvey Jackson and he performed the surgical operations. I wish to thank him for permission to publish the case reports and for helpful criticism and advice. Cases 1 and 2 were admitted under Dr. Denis Brinton, case 4 under Dr. S. P. Meadows, and case 5 under Dr. Michael Kremer. My thanks are due to them for their kind permission to discuss their patients. I should also like to thank Professor W. Blackwood and Dr. W. G. P. Mair for permission to include their histological reports in this paper. I am indebted to Mr. A. H. Prickett of the Department of Medical Photography for taking the photographs and preparing the drawing.

REFERENCES

- Balestrieri, A. (1958). *Riv. Neurol.*, 28, 17.
 Connolly, Campbell R. (1962). Paper read to the 65th meeting of the British Society of Neurological Surgeons at St. Bartholomew's Hospital, London.
 Cloward, R. B., and Bucy, P. G. (1937). *Amer. J. Roentgenol.*, 38, 681.
 Cuneo, H. M. (1955). *J. Neurosurg.*, 12, 176.
 Elsberg, C. A., Dyke, C. G., and Brewer, E. D. (1934). *Bull. neurol. Inst. N. Y.*, 3, 395.
 Garcin, R., Launay, C., Guillaume, J., and Hadengue, A. (1958). *Rev. neurol.*, 98, 54.
 Guillaume, J., Ribadeau-Dumas, C., and Rogé, R. (1947). *Ibid.*, 79, 507.
 Hyndman, O. R., and Gerber, W. F. (1946). *J. Neurosurg.*, 3, 474.
 Jacobs, L. G., Smith, J. K., and Van Horn, P. S. (1954). *Radiology*, 62, 215.
 Kelly, T. S.-B. (1937). *Lancet*, 2, 13.
 Lehman, E. P. (1935). *Amer. J. Surg.*, 28, 307.
 Lewitt, E. H. (1945). *Hydraulics and the Mechanics of Fluids*, 7th ed. Pitman, London.
 Meredith, J. M. (1940). *Virginia med. Mth.*, 67, 675.
 Nugent, G. R., Odum, G. L., and Woodhall, B. (1959). *Neurology (Minneapolis)*, 9, 397.
 O'Connell, J. E. A. (1953). *Brain*, 76, 279.
 Olsson, O. (1948). *Acta radiol. (Stockh.)*, 29, 279.
 Raxed, B. (1947). *J. Neurosurg.*, 4, 414.
 Roger, H., Arnaud, M., Poursines, Y., and Alliez, J. (1933). *Rev. neurol.*, 40 (1), 1155.
 Rouquès, L., Guillaume, J., Ribadeau-Dumas, C., and Rogé, R. (1948). *Ann Med.*, 49, 369.
 Schmidt, A. (1904). *Dtsch. Z. Nervenheilk.*, 26, 318.
 Schreiber, F., and Nielsen, A. (1950). *Amer. J. Surg.*, 80, 124.
 Shenkin, H. A., Horn, R. C. Jr., and Grant, F. C. (1945). *Arch. Surg. (Chicago)*, 51, 125.
 Strully, K. J., and Heiser, S. (1954). *Radiology*, 62, 544.
 Wise, B. L., and Foster, J. J. (1955). *J. Neurosurg.*, 12, 421.