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Forking of the central canal in the equinal cord of children

R. G. LENDON AND J. L. EMERY

Congenital Anomalies Research Unit, University of Sheffield and Department of Pathology, Children's Hospital, Sheffield

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During a survey of the spinal cord in normal children and children with meningomyelocele (Lendon, 1968) we noticed that the central canal in the filum terminale of the normal children was occasionally forked or duplicate. The literature on equinal anatomy contains little about the incidence of canal forking in this region of the adult or infant cord. A large number of cases of canal duplication have been described in human embryos (Ikeda, 1930), but although Tarlov (1938) noted canal forking in the infant and adult filum he quoted no incidence. In view of this we were unable to assess the significance of canal forkings we had observed in the equinal cord of children with meningomyeloceles and it became necessary to investigate its incidence in normal children.

The equinal cord consists of three regions. (1) The conical region of the cord caudal to the fourth sacral nerve root is the conus medullaris. (2) The conus is followed by a region in which the central canal expands considerably to give rise to the ventriculus terminalis—itself divided into three characteristic regions (Argutinsky, 1898). (3) Caudal to the ventricular region, the canal and cord are reduced in size to become the filum terminale, which runs to an attachment with the back of the coccyx and consists of intra- and extra-dural parts (Harmeier, 1933).

MATERIALS AND METHODS

The material came from 100 necropsies carried out at the Children's Hospital, Sheffield. The majority of the children had died within a year of birth. There was a slight predominance of male children.

The equina was removed and the conus and intradural portion of the filum were divided into approximately 6 mm lengths. Using routine paraffin embedding, serial transverse sections were cut at 10 μm throughout the tissue, and the sections were stained with Harris's haematoxylin and eosin. In ten cases, only part of the intra-dural filum was available for study.

As this was a survey of canal forking in the normal infant, equinal cord material from children with central nervous system (C.N.S.) malformations or with symptoms possibly referable to the C.N.S. were not admitted to the study. Others that were found after detailed post-mortem study to have possessed a congenital deformity of any other organ in the body, such as the heart, were removed from the normal group and categorized separately. This left a normal group consisting of 77 equinae (69 of which were complete), and a second group of 23 equinae (21 of which were complete)

which we will refer to in later discussion as a 'distant congenital defect' ('D.C.D.') group. This distinction is possibly an important one and previous studies have largely ignored it.

RESULTS

The incidence of canal deformity at the three main cordal levels is shown in Table 1.

Table 1. *Incidence of canal forking at three levels of the equinal cord of 100 children*

Level		No. of cords	Unforked	Major forking	Minor forking
Conus	Normals	77	45 (58·4 %)	8 (10·3 %)	24 (31·2 %)
	'D.C.D.'	23	13 (56·5 %)	5 (21·7 %)	5 (21·7 %)
Ventricle	Normals	77	61 (79·2 %)	16 (20·8 %)	0
	'D.C.D.'	23	17 (73·9 %)	6 (26·1 %)	0
Filum	Normals	69	45 (65·4 %)	24 (34·6 %)	0
	'D.C.D.'	21	9 (42·9 %)	12 (57·1 %)	0
At all or some levels	Normals	69	19 (27·5 %)	31 (44·9 %)	19 (27·5 %)
	'D.C.D.'	21	4 (19·1 %)	12 (57·1 %)	5 (23·8 %)

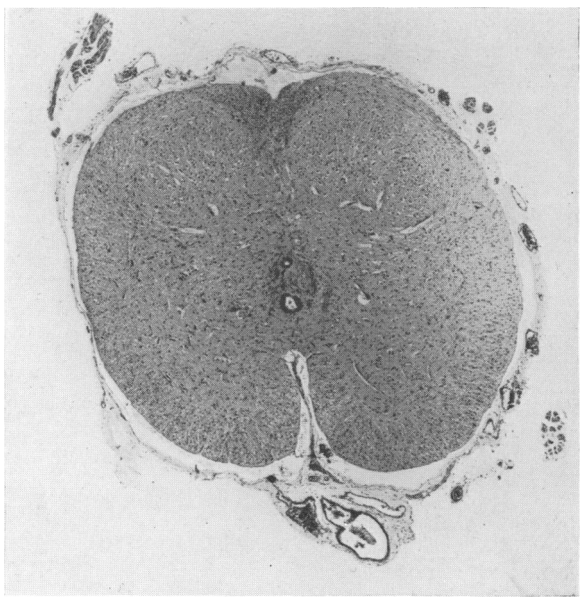


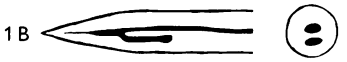
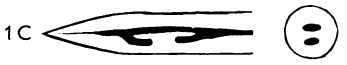
Fig. 1. Transverse section of the conus medullaris from a normal child showing minor forking of the central canal. × 26.

Almost a third of the equinae showed localized minor forking of the central canal in the conus region (Fig. 1). These minor forkings were short dorsal loops extending a distance usually of only a few consecutive serial sections. They often reappeared in

the same cord lower down the conus. Twenty-nine of the 100 cords exhibited minor canal forking of this type. We have seen similar loops at more cephalic levels of the normal cord. A further minor type of deformity which frequently occurred was localized splitting of the most distal part of the canal in the intra-dural filum prior to its disappearance, but these have not been included in Table 1.

Major bifurcation of the central canal occurred in 31 of the 69 apparently normal children at some or all levels of the equinal cord (45 %); and the incidence was higher in the smaller 'D.C.D.' group (57 %). The major canal deformities encountered and their incidences are shown in Table 2. Some cords exhibited more than one type of defect.

Table 2. *Types and incidences of major canal forking in the human equinal cord*

Type of forking	Incidence of forking: Present series infants: Normal	Incidence of forking: Present series infants: 'D.C.D.'	Incidence of forking: Ikeda (1930) embryos
1A 	6	3	5
1B 	0	0	14
1C 	1	0	4
1D 	5	3	7
2A 	23	9	12
2B 	4	3	6
3 	2	1	15
4 	0	1	1
5 	0	2	0
Total number of forkings	41	22	64
Total number of cases	69	21	181

The deformities are shown as they would appear in sagittal and transverse sections, except types 2A and 2B, which are in frontal and transverse planes.

A common major anomaly was dorso-ventral duplication of the central canal or ventricle (Type 1, Figs. 2, 3). This usually started in the conus region and often continued into the ventricle region but rarely further. A small ventral or dorsal diverticulum was given off from the main canal and increased in size in the ventricular region. Occasionally the diverticulum would disappear in the conus region leaving the original canal as the main one. In other cases (Table 2, Type 1 D) the two canals would re-fuse in the conus or ventricular region. In one cord (Table 2, Type 1 C) a ventral canal was given off in the conus region and travelled caudally before terminating, while a second ventral canal rose from the ventricle region to travel in a cephalad direction before disappearing without meeting the first canal.

The most commonly occurring type of defect was simple lateral duplication of the central canal to give right and left canals (Table 2, Type 2 A). This occurred in the ventricle region (Fig. 4) or in the filum terminale (Fig. 5), or in both. Occasionally the two canals would re-fuse (Type 2 B). We have not seen the type 2 A or 2 B defect in the conus region.

Two cords had a small accessory canal ventral to the main canal and ventricle totally unconnected with the main system (Type 3).

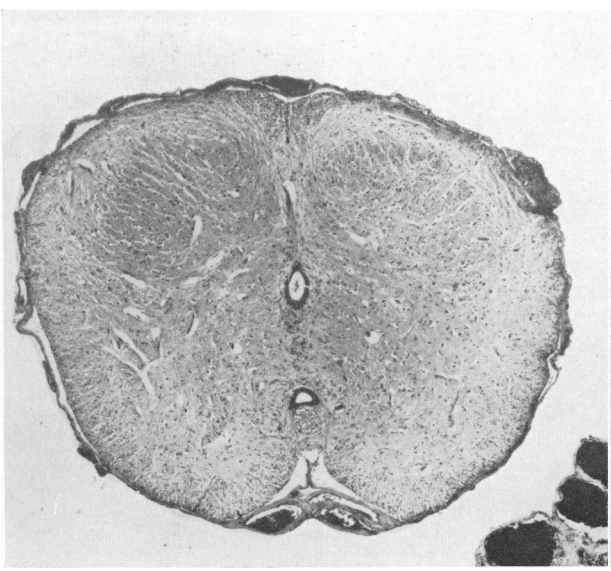


Fig. 2

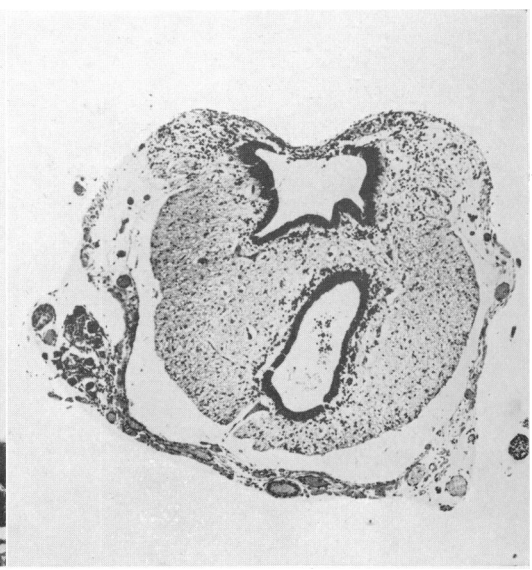


Fig. 3

Fig. 2. Transverse section of the conus medullaris from a normal child with Type 1 duplication of the central canal. × 39.

Fig. 3. Transverse section through the cephalic end of the ventriculus terminalis of a normal child with Type 1 duplication of the ventricle. × 52.

In one cord (Type 4) the normal central canal disappeared in the ventricle region gradually being replaced by a ventral canal that was completely isolated from the original central canal and which became the main canal of the filum terminale.

Two cords in the 'D.C.D.' group exhibited an increase in canal number to between

3 and 5 in a region at the caudal end of the conus and cephalic end of the ventricle (Type 5). This was accompanied by a great increase in the number of glial cells similar to those seen normally beneath the ependyma of the cerebral ventricles in immature brains, and they showed a tendency for the type of pseudo-rosette formation seen in neuroblastomas. These children were the only two in this series who had cystic fibrosis (muco-viscoidosis).

The eight types of forking we have described were the basic deformities. However, occasionally in the filum terminale the pattern was complicated locally by a reduplication or out-pouching of an already duplicated canal. To avoid making the data too unmanageable we have listed only the major forking patterns encountered.

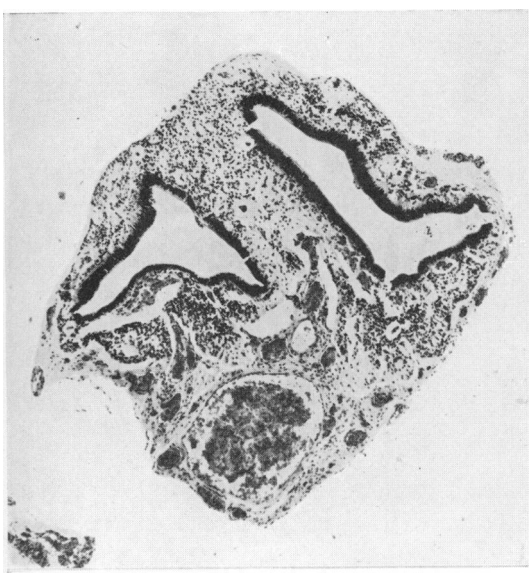


Fig. 4

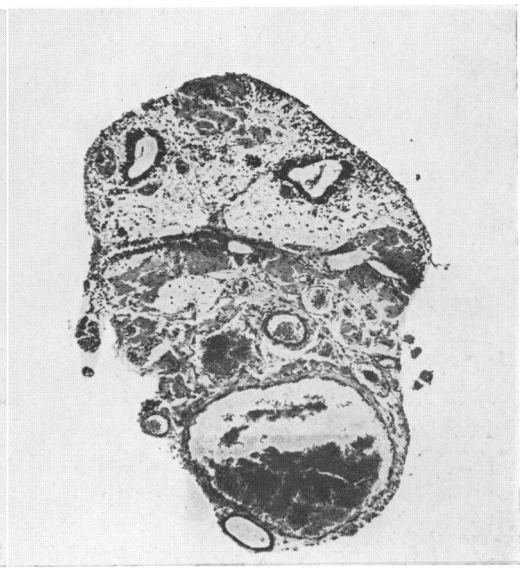


Fig. 5

Fig. 4. Transverse section through the ventriculus terminalis of a normal child with Type 2 duplication of the ventricle. ×43.

Fig. 5. Transverse section through the film terminale of a normal child showing Type 2 duplication of the central canal. ×54.

DISCUSSION

Ikeda (1930) investigated the caudal end of the nerve cord in a group of 181 human embryos and found 62 cases with forking of the cord lumen (34 %). He noted seven main types (Table 2), but also described other individual anomalies. He found that more than one type could occur in a single embryo. Table 2 shows the close similarities between Ikeda's findings and those of the present series. However, we have not seen the Ikeda type 1 B defect and the relative frequency of the types of forking differ from his findings. In particular, we found a much greater incidence of type 2 A forkings whereas Ikeda found a larger number of type 3 defects. The Ikeda type 1 A category differs slightly from ours. He noted that the forking was most frequently a ventral one

but in our series it was either dorsal or ventral. However, Ikeda described a 32·5 mm embryo and Politzer (1951) a 22·5 mm embryo with dorsal forking of the canal, similar to our type 1 A with dorsal fork. Politzer also noted a 'concertina' effect in the caudal neural tube of several embryos. This he believed was due to over-production of neural tissue in what is to be a rudimentary organ. Similar bucklings of the neural tube can be seen in some of Ikeda's illustrations and perhaps his type 1 B is a transitory example of a 'concertina' effect. This might account for it not being present in our series.

Ikeda described a 17 mm embryo showing a defect almost identical to the one we have seen in one of our 'D.C.D.' group (Table 2, type 4). In his embryo, however, the more caudal canal extended up as far as the 3rd sacral segment, whereas in our case it only reached the upper coccygeal region.

There was a greater incidence of major canal forking in the equinae of our 'D.C.D.' infants than in the normal ones (Tables 1, 2). It appeared probable that this difference was due to the 'D.C.D.' cords showing a higher incidence of multiple forkings. To test this hypothesis we divided the normal and 'D.C.D.' material into cases with one major forking per equina and those with more than one major fork per equina. Seven normal and seven 'D.C.D.' cords had more than one major forking, while 24 normal and five 'D.C.D.' equinae showed only a single major fork. These results were subjected to the Fisher exact probability test. The null hypothesis that there are equal proportions of single and multiple forkings in the two classes is rejected at the 4 % level, and the alternative hypothesis that there are more multiple forkings in the 'D.C.D.' group can be accepted.

Much has been written on the subject of whether duplication of the central canal in the embryonic equinal cord is of primary or secondary origin, (Holmdahl, 1925; Schumacher, 1927; Ikeda, 1930; Politzer, 1951). Canal forking has been seen in embryos both before and after the time at which tail regression and filum formation begins. It is beyond the scope of the present paper to discuss the embryogenesis of canal forking in the equinal cord but our observation that major forking is most prevalent at the more caudal end of the region, particularly in the filum terminale (Table 1), suggests that either this area is more prone to primary embryonic misconstruction, or redifferentiation associated with the development of the filum must contribute to the high incidence of canal forking.

Our observation that multiple canal forking may be of more frequent occurrence in infants with distant congenital defects suggests that development of the equinal cord may perhaps be affected by, or share a common origin with, anomalies of distant organ systems.

SUMMARY

The incidence of major canal forking in the equinal cord of 100 human infants was investigated. A major anomaly of the canal was found at some level in 45 % of a group of normal children, and 57 % of a group with non-nervous congenital defects.

Several distinct types of central canal deformity were found common to both groups and these correspond closely with those seen in embryos by previous workers.

There are indications that a higher incidence of canal forking found in the congenital defect group may be due to a greater number of multiple forkings per equina in this group.

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