

Persistence of the notochordal canal in vertebrae

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INTRODUCTION

Notochordal cells normally disappear from vertebral bodies during the cartilaginous stage of development of the vertebral column. The mucoid streak or notochordal sheath usually disappears from the vertebral body as it ossifies in early fetal life. Traces of the notochordal track persist in the cartilage end plates in fetuses, infants and children in the form of small funnel-shaped depressions from the nucleus pulposus (Fig. 3), associated with a narrow clear cell-free area in the cartilage plate (Böhmgig, 1930). Notochordal cells persist in the nucleus pulposus in fetal life and they can be found in the nucleus postnatally up to the age of ten years (Peacock, 1952). Schwabe (1933) reports persistence of notochordal cells in the disc remnants of the sacrum up to mid-adult life. Persistent notochordal rests in the vertebral column (Schmorl, 1928; Ehrenhaft, 1943) are regarded as the source of chordomas. Although rare, chordomas are most commonly found in the sacrococcygeal and cranial regions.

Single cases of persistence of the notochordal canal through a vertebra are reported in L 4 by Musgrove (1891) and in T 9 by Schmorl & Junghanns (1959). Böhmgig (1930) describes and illustrates 'cartilage processes' in the centre of the vertebral body of a 16 cm fetus. In the middle of these processes 'runs the former chordacanal'. Other reports in the literature of persistence of the notochordal canal in vertebrae are associated with cleft vertebrae. Sereghy (1927), Frets (1930), and Rathke (1959) report persistence of the notochord or its sheath in cases of sagittal cleft vertebra. In his 'schemata' showing normal and abnormal vertebral development, Junghanns (1959) illustrates the development of sagittal cleft vertebra in association with persistence of the notochordal canal.

Coronal cleft is more frequently reported, though its recorded incidence varies widely from 1% to over 30% (Rowley, 1955; Cohen, Guido & Neuhauser, 1956; Reichman & Lewin, 1969; Fielden & Russel, 1970). The variation may be due to the different methods of investigation used – radiological or sectioning of post-mortem material. Coronal cleft is said to occur during fetal life and infancy, disappearing with growth. Knutsson (1940), Rowley (1955), Schmorl & Junghanns (1959), and Reichman & Lewin (1969) describe coronal cleft simply as a failure or delay of fusion of a large anterior centre of ossification in the vertebral body with a small posterior 'accessory' centre of ossification. Meyer-Burgdorff & Klose-Gerlich (1935) and Wollin & Elliott (1961) associate coronal cleft with the persistence of notochordal tissue. Lossen (1931) and Probst (1952), investigating human fetuses, discuss the relationship between persistence of the notochordal canal in vertebrae and the

Table 1. *Material examined*

	Fetuses	'Full term'	Infants	Children
Age range of group	25-38 weeks	38 weeks to 1 month	1 month to 2 years	2-10 years
Serial nos. of cases	3-18	19-36	37-47	48-59
No. in group	16	18	11	12
Cases of persistent notochord	Nil	3	1	Nil

Table 2. *Analysis of four cases suggesting persistence of notochord*

Case no.	Age	Vertebrae involved	Nature of track
24	Full term	T 8 and 9	Mainly fibrocartilage with fibrillar acellular core
31	Full term	L 4	Fibrocartilage
36	Full term	T 7 8 and 9 L 4 (trace)	Mainly fibrocartilage with fibrillar acellular core and notochordal tissue near a VB centre
44	10 months	L 4 (partly interrupted by ossification)	Fibrocartilage

T = thoracic; L = lumbar; VB = bony vertebral body.

concurrent existence of axial clefts in these vertebrae. Rathke (1959) states that sagittal and coronal clefts may coexist in the same vertebra. He also distinguishes two types of coronal clefts: a 'pseudo-cleft' with no persistent notochordal tissue but simple delay in fusion of two ossification centres; and a 'true coronal cleft' which is associated with persistence of notochordal tissue in the cleft (as in sagittal cleft) and is a permanent anomaly found in adults.

MATERIAL

Material was obtained from 57 apparently normal vertebral columns. This included in 24 cases a minimum of two vertebrae and two discs from each of the cervical thoracic and lumbar regions, and, in the remainder, all the lumbar vertebral bodies and discs. After fixation in formalin and embedding in celloidin, 150 μ m sagittal, coronal or transverse sections were cut. In those specimens obtained earliest in the series all sections were mounted. In later specimens only one in three sections was mounted. Cases were grouped and numbered according to age (Table 1).

RESULTS

In four individuals (an incidence of 7.0%) a persistent cylinder of unossified material was found passing from the superior and inferior surfaces of the vertebra towards its centre, but interrupted there by a bony nodule (Fig. 1). The position of this cylinder or canal corresponded to that of the notochord in fetal life. The upper and lower halves of the canal within each vertebra formed an angle, the apex of which lay at the bony nodule in the centre of the vertebral body. In the thoracic vertebrae

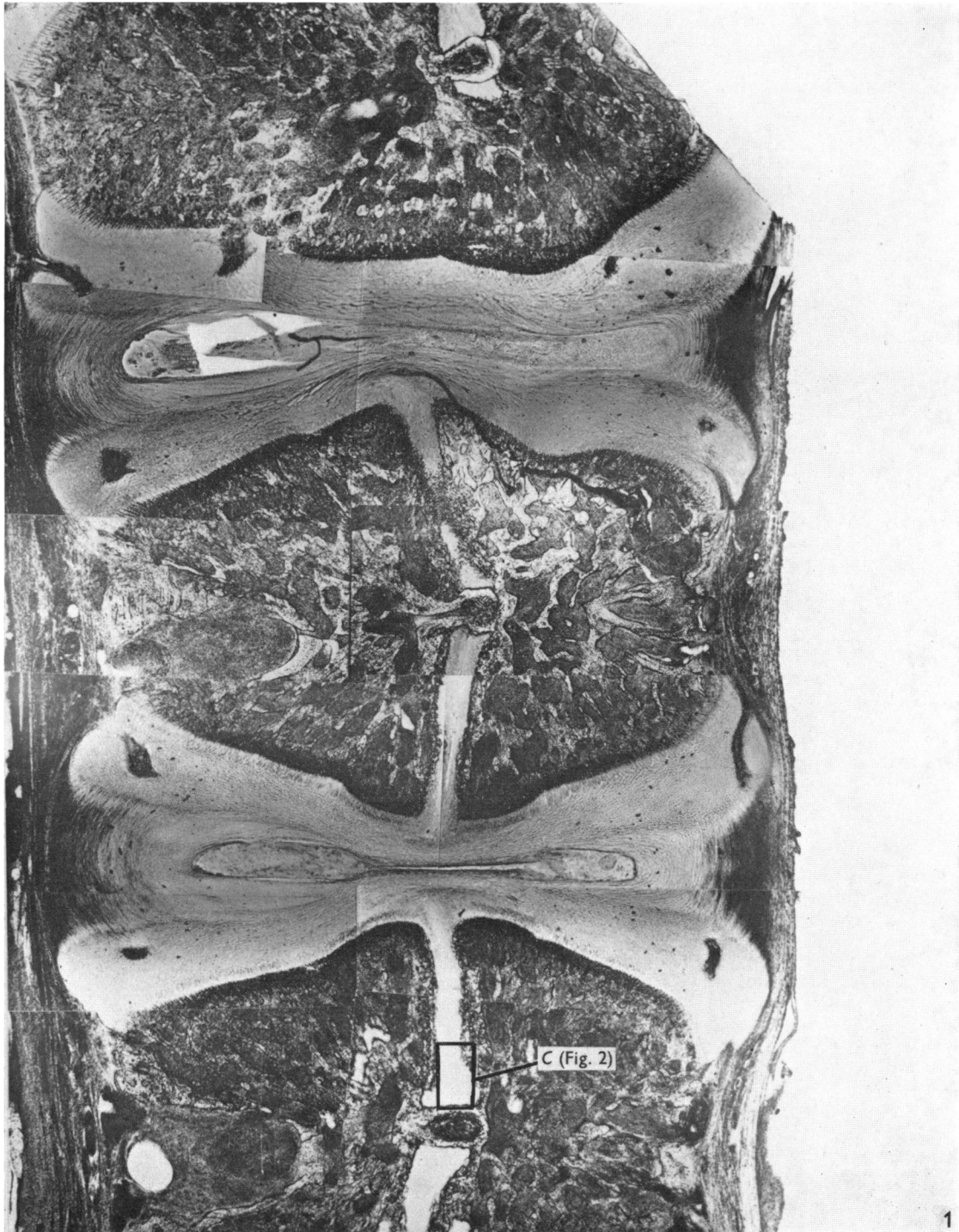


Fig. 1. Case 36, a full-term fetus showing anomalies in T7, 8 and 9 and the intervening discs.
× 12. (Cf. Fig. 4.)

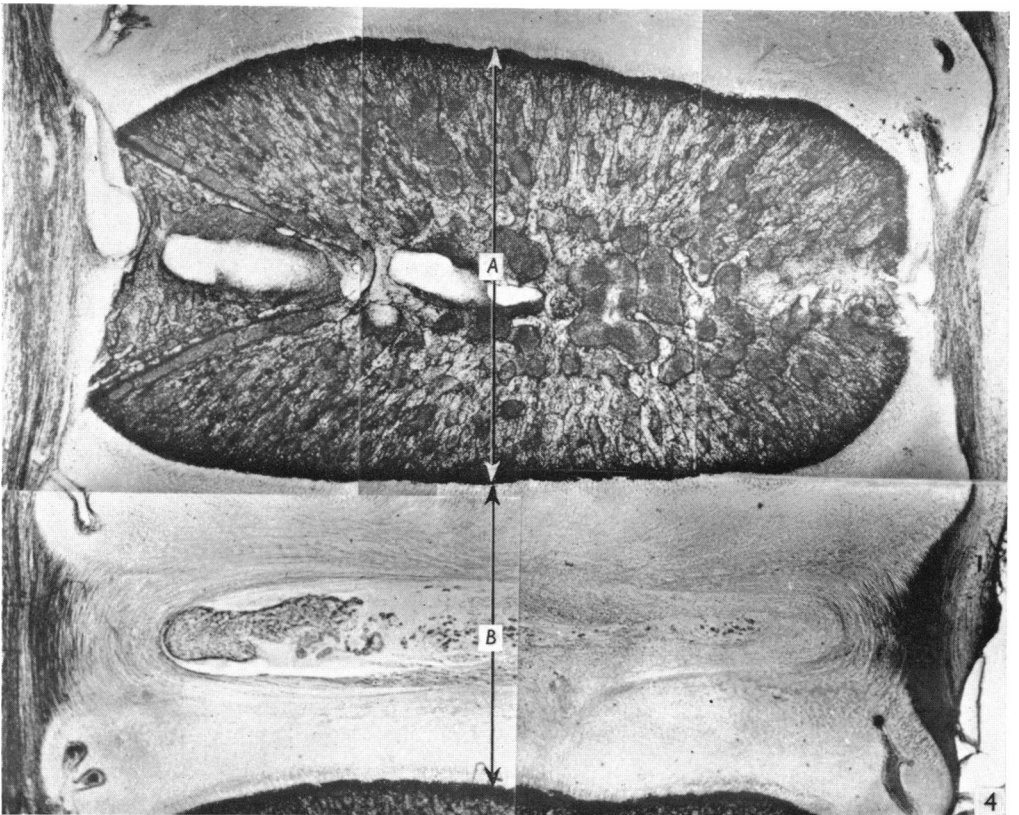
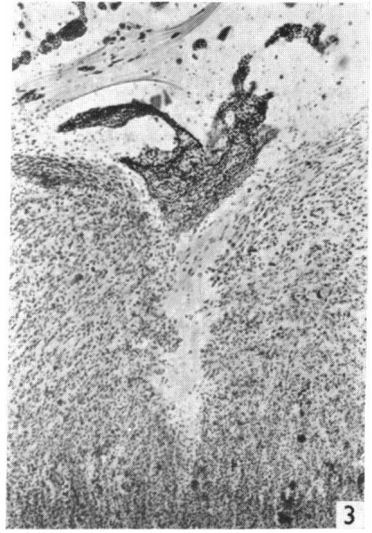
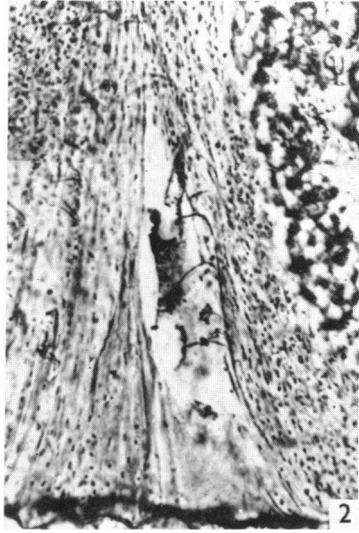


Fig. 2. Area 'C' from Fig. 1 showing notochordal tissue. $\times 70$.

Fig. 3. Funnel-shaped depression and 'cell-free area' in cartilage plate of a 36 week fetus. $\times 60$.

Fig. 4. Case 21, mid-line section of a normal thoracic vertebra (T8) and intervertebral disc. $\times 12$.

Table 3. Comparison between heights of normal and affected centra

	Five normal full-term specimens		Specimen 24	Specimen 36
	Mean	Range		
T 8 height (‘A’ of Fig. 4) (mm)	4.95	4.6–5.4	5.7	6.7
T 8–9 ‘Total disc’ (‘B’ of Fig. 4) (mm)	3.05	2.5–3.3	1.3	1.6

(cases 24 and 36, Table 2) this apex was directed forwards (Fig. 1). In sections from cases 31 and 44 the lumbar vertebral bodies involved (L 4) were incomplete, but the orientation of the canals in each case suggested that the apex in the lumbar vertebra was directed backwards. The notochordal segment forming the nucleus pulposus was predominantly posterior in both thoracic and lumbar intervertebral discs in all four cases. Canals appeared to consist mainly of fibrocartilage with cells oriented vertically parallel to the canal. In two of the four cases, a core of acellular fibrillar material passed through the centre of the canal. Notochordal cells in a mucoid matrix, similar to the fetal nucleus pulposus, were found in a dilated portion of one canal near the centre of a vertebral body (Fig. 2).

The peripheral height of the affected centrum was normal, but its central height was increased compared with that of normal centra (Table 3; Figs. 1 and 4). The convexity of the upper and lower bony surfaces was associated with a corresponding deformity of the cartilage plates, in which the cartilage columns were perpendicular to the underlying bony surface. In two cases the nucleus pulposus had a bilocular appearance in mid-line section (Fig. 1).

In a large proportion of normal vertebrae in this age group small isolated islands of cartilage were found near the centre of the bony vertebral body. No cleft vertebra was found in this series.

DISCUSSION

It is believed that all four cases described above are instances of persistence of the notochordal track through the vertebral bodies. The canal of fibrocartilage in each case had a similar position and histological appearance, and was associated with a similar deformity of ossification of the vertebral body. In two cases an acellular fibrillar core extended through the centre of the cylindrical canal, resembling the embryonic mucoid streak. In one case notochordal tissue was found in the canal near the vertebral body centre (Fig. 2). The finding of the notochordal cells near the centre of a vertebral body recalls the description by Dursy (1869) of a fusiform swelling of the notochord in a corresponding position in human and other mammalian embryos. The angulation of the canals within the vertebral bodies (Fig. 1) resembles the notochordal flexures seen in the fetus. The different orientation of the canals in thoracic as opposed to lumbar specimens supports Schaffer's (1910) claim that the notochordal flexures differ according to the region of the vertebral column where they are found, and that these flexures are related in some way to the regional curvature of the vertebral column.

The rare anomaly described as persistent notochordal canal by Musgrove (1891) and by Schmorl (1959) is present in the adult. This is a wide central channel of more than 1 cm diameter in a vertebra with concave upper and lower surfaces resembling a fish vertebra. The anomaly found in this series has a high incidence, is present in infants, and the vertebral bodies have convex upper and lower surfaces.

In their textbook, Schmorl & Junghanns (1959) note the connection of a smaller 'persistent chordacanal' with sagittal cleft vertebrae. The cases observed here are not associated with cleft vertebra and no coronal clefts have been observed in the 36 cases in this series from full term to four years, despite the claim of Reichman & Lewin (1969) that approximately one in three individuals of less than four years of age show coronal clefts. However, Reichman & Lewin (1969), in defining coronal cleft, state 'the cleft does not always go right through the vertebral body. It may be confined . . . to an isolated region in the middle'. Reichman & Lewin's illustrations of ossification irregularities at the cartilage plate junction bear a striking resemblance to the deformity of ossification observed in this series. Wollin & Elliott (1961) include in a series of 'coronal cleft' vertebrae a case (diagnosed from lateral X-ray) which on section proved to contain 'a cylindrical core of gelatinous white cartilage . . . a loose matrix of mucin . . . indicating notochordal derivation'. It is possible, as some authors (e.g. Rathke, 1959), maintain that 'persistent chordacanal' may predispose to coronal cleft, but a cylindrical core of cartilage containing notochordal tissue should not be confused with a coronal cleft. Coronal cleft is usually described as situated at the junction of the anterior two thirds and posterior one third of the vertebral body. The 'persistent notochordal canals' in this series were nearer the centre of the vertebral body.

The persistence of the canal indicates its resistance to ossification. The mucoid streak in the fetus is surrounded by primitive hyaline cartilage. The presence of elongated spindle-shaped cells around the acellular fibrillar core, as observed in this series, may indicate a change in the character of these cells under the influence of persistent notochordal tissue. The canal in case 36 shows hyaline cartilage cells outside the fibrocartilage layer. The frequent finding of small islands of cartilage in the centre of vertebral bodies in normal infants may be associated with the former presence of the notochord at this situation. Theiler (1953) cited by Rathke (1959) states that the criterion for true cleft vertebra lies in the structure of the tissue in the cleft ' . . . if it is intervertebral disc-like tissue, then it cannot be replaced by bone, as it would be in the case of cartilage'. However, since case 44 shows incipient ossification across the canal, and no persistent notochordal canal is observed in the fifteen cases older than case 44, it may well be that resistance to ossification is only of a temporary or delaying nature in canals of this size.

There is an apparent stimulation of ossification in the cartilage plates around the ends of the canal. The presence of the canal may be associated with a change in the orientation of the metaphyseal cartilage columns in its immediate vicinity and the persistence of the canal may induce the observed bony deformity in this way (cf. Figs. 1, 4). The corresponding alteration in the shape of the cartilage plates and nucleus pulposus in the midline (Fig. 1), if it were to persist in a surviving infant, might reduce the functional efficiency of the intervertebral disc.

SUMMARY

Four cases of persistent notochordal canal in vertebral bodies are described. This represents an incidence of 7.0 % of the apparently normal 57 vertebral columns from fetuses, infants, and children examined. The relationship of persistent notochordal canal to cleft vertebra is reviewed, and the effect of persistent notochordal canal on ossification is discussed.

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