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CORONAL CLEFT VERTEBRAE IN GROWING INDIVIDUALS

A Preliminary Report

By

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Sagittal and coronal bands of uncalcified tissue may occur in vertebral bodies.

The morphology of coronal cleft vertebrae in the foetus and newborn has been described by *Meyer-Burgdorff & Klose-Gerlich* (1935), *Schinz & Töndury* (1942), *Cohen et al.* (1956), *Rettig* (1958) and *Töndury* (1958). *Cohen et al.* have also reported on the appearance of such clefts in lateral projection roentgenograms. These papers have demonstrated a considerable variation in the appearance of the cleft, which is generally shaped like a horseshoe with the convexity facing forward. The anterior part of the vertebral body is larger than the posterior. The cleft does not always go right through the vertebral body; instead it may be confined to one side or to an isolated region in the middle. An otherwise complete septum is frequently bridged by a central osseous process joining the two halves of the vertebral body. The tissue comprising the cleft consists of the same type of hyaline cartilage that encloses the centres of ossification of the vertebrae.

Coronal cleft vertebrae occur almost exclusively in the newborn (*Töndury* 1958) and ossification of the cleft is considered to occur during the first year or two of life (*Meyer-Burgdorff & Klose-Gerlich* 1935, *Knutsson* 1940, *Cohen et al.* 1956, *Fagerberg* 1963).

Opinions differ concerning the mechanism responsible for coronal cleft vertebra. Abnormal persistence of the notochord has been held to be an important factor (*Meyer-Burgdorff & Klose-Gerlich* 1935, *Rathke* 1959, *Blauth & Hopf* 1960, *Zukschwerdt et al.* 1960). This opinion is based upon the histological demonstration, at relatively late stages of development, of tissue having an apparently notochordal origin. It has

moreover been stated that coronal cleft vertebrae usually occur in several segments in the same spine. *Schinz & Töndury* (1942) reported that the bone nuclei of the vertebral body always develop in a uniform manner but that their shape may vary, resulting in extreme cases in one anterior and one posterior part united by only a minimal bridge of bone. *Rettig* (1958) ascribed the occurrence of coronal clefts to the occasional division of the bone nucleus, which is normally single.

The majority of coronal clefts occur in the lumbar spine (*Töndury* 1958), though they have also been demonstrated in thoracic and cervical segments (*Knutsson* 1940, *Cohen et al.* 1958, *Fagerberg* 1963). *Cohen et al.* (1956) considered that coronal cleft vertebrae are more common in children with anomalies in other organs. *Fagerberg* (1963), on the other hand, found no increased incidence of skeletal malformations in children with coronal cleft vertebrae. Instead he asserted that children with such clefts display a higher mortality, *e.g.* in complications during pregnancy, and that about 90 per cent of all children with clefts are male.

The extent to which coronal clefts produce secondary states has not been investigated. *Zukschwerdt et al.* (1960) consider that they can cause instability in the motion segment.

The present paper deals with the following questions:

1. How common are coronal clefts at different ages in a postmortal series? What is their distribution in segments $L_1 - S_1$?
2. Can other structures give rise to similar roentgenograms?
3. Does ossification occur at the border between the cleft and the bone nucleus?
4. Does growth occur within a coronal cleft? Do asymmetric clefts result in asymmetric vertebrae?
5. What light does the present postnatal study throw on the genesis of coronal cleft vertebrae?

MATERIAL

The study was made on autopsy specimens comprising the segments L_1 to S_1. The specimens came from individuals autopsied in Gothenburg having died in a hospital during the collection period. Only subjects below 25 years were accepted. The drop-out was less than 4 per cent for the age groups up to 16 years but could not be established for older groups. No primary consideration was paid to the cause of death.

The age distribution of the 132 cases is shown in Figure 4. The autopsy reports of these cases were also studied.

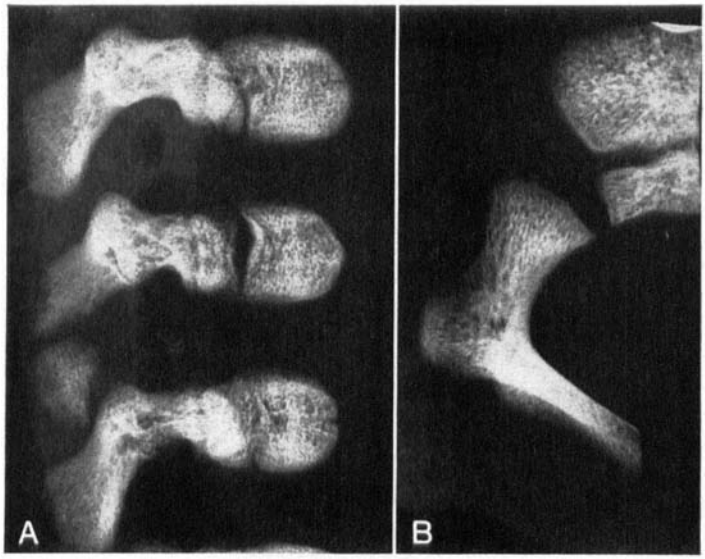


Figure 1. Coronal cleft vertebrae at birth seen (A) in lateral projection and (B) in axial projection. A cleft in an inferior articular process is visible in (A).

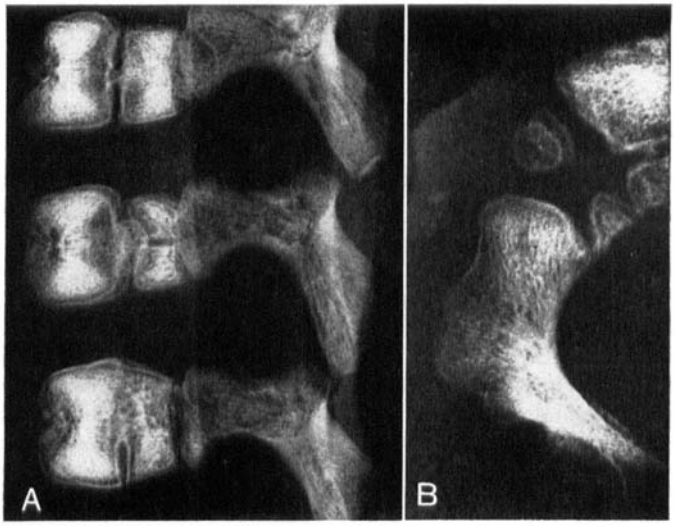


Figure 2. Coronal cleft vertebrae and supernumerary bone nuclei 19 days after birth. One vertebral body in (A) has a horizontal cleft in its posterior part. Two separate bone nuclei are visible laterally and one medially (histologically verified) in (B). The superior contour of the lowermost vertebral body is raised in the region of the cleft.

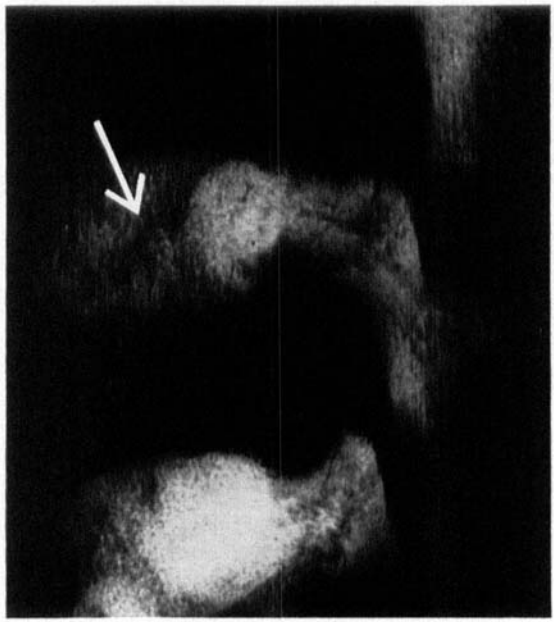


Figure 3. Radiolucent streak dividing caudally in vertebral body at birth.

METHODS

The specimens were prepared as follows:

1. Fixation in 10 per cent, neutral formalin.
2. Frontal roentgenograms, using a very fine-grain non-screen film ("industrial film").
3. The specimen was divided down the mid-line.
4. Lateral and oblique roentgenograms were taken of the two halves of the specimen. The median surface was photographed with panchromatic or infrared emulsion (the latter gives a clearer picture of cartilaginous tissue in the newborn, cf. Figure 9).
5. The specimen was sawed into segments and vertical roentgenograms were taken of these. One half of each specimen from newborn individuals was, however, sawed into large pieces because these can be quite conveniently embedded and sectioned as they are.
6. Decalcification followed by embedding in paraplast and sectioning with a standard microtome. Staining chiefly with Ehrlich's haematoxylin-eosin, mounting in Canada balsam. Unstained specimens were mounted in pre-polymerised metachrylate.

The autopsy records provided data concerning the individual's sex, age, cause of death and any malformations.

The statistical analysis was performed with the χ_2 method even though the χ_2 hypothesis gave expected values between 0 and 5. Recent studies have shown that this method of analysis is reliable even with these expected frequencies (*Carlström* 1967).

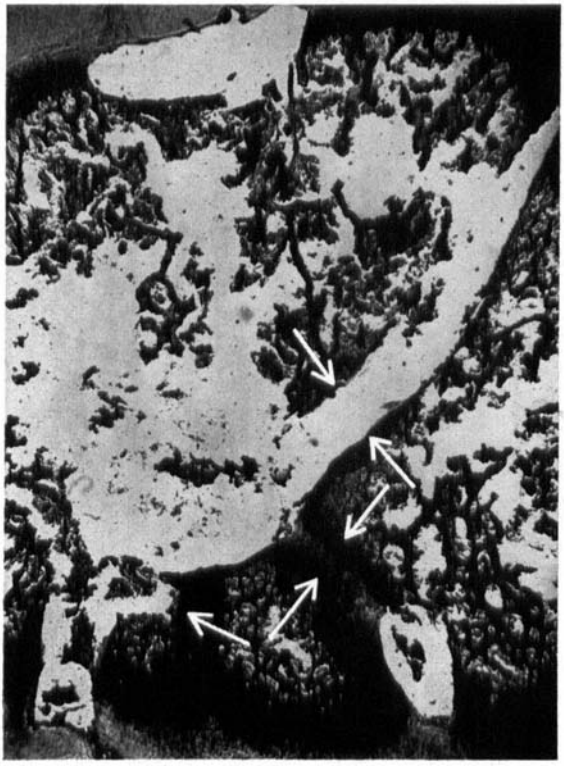


Figure 4. Histological section in the sagittal plane from the specimen in Figure 3. The radiolucent formation corresponds to blood vessels, which are surrounded by a thin zone of cartilage.

RESULTS

In keeping with previous investigations, large variations were found in the shape of the coronal cleft (around the general form) a ventrally concave band filled with hyaline cartilage (Figures 1 and 2). No asymmetry of the vertebral body was found.

Roentgenological Morphology

Most coronal clefts were visible in a lateral roentgenogram (cf. Figure 13). In those cases in which the line of the cleft coincided for a considerable distance with the direction of the X-rays, the cleft appeared to be bordered by a thin zone of sclerotic bone. Roentgenograms of specimens in the upper age groups showed that clefts in these were not as wide as in specimens from newborn individuals. Raising of the chondro-osseous interface is described under "micromorphology".

Two structures of differential diagnostic importance are vascular

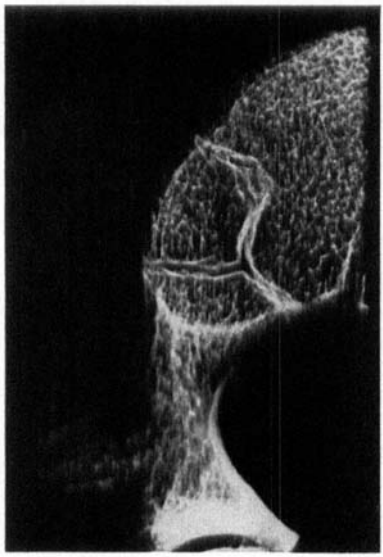


Figure 5. Supernumerary bone nucleus in a 3-year-old. In a lateral projection the posterior chondrous shank may be mistaken for a coronal cleft in the vertebral body.

clefts and supernumerary bone nuclei in the cartilaginous zone between the vertebral body and the pedicle.

A roentgenogram of a vertebral body with a vascular cleft is reproduced in Figure 3. The cleft appears as a somewhat diffuse radiolucent area with an atypical course. Serial sectioning of this specimen (Figure 4) revealed the vessel surrounded by a thin zone of hyaline cartilage that continued laterally to the cartilaginous part of the vertebral body. In sections that did not include the vessel the cleft differed from usual coronal clefts only in its course. A similar casing of cartilage around relatively large vessels occurred in many places where blood vessels entered the vertebral body. An axial view gives the best roentgenographic picture, the generally short course of the vessels producing a small indentation in the periphery of the bone nucleus.

The band of cartilage between the vertebral body and pedicle may contain supernumerary, unattached bone nuclei (Figure 2). These develop within the cartilage, which surrounds them only as a thin layer at the age of two to three years. At this stage the posterior shank of cartilage may lie in the frontal plane (Figure 5); in a lateral projection it then appears very similar to a coronal cleft of the type described above.

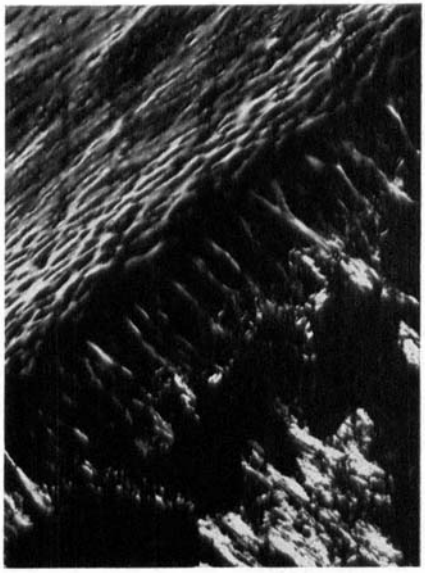


Figure 6. Polarisation microphotograph of normal, endochondral zone of ossification. The collagen fibres change direction at the zone of proliferation (× 110).

Micromorphology

The sclerotic border zone between the coronal cleft and bone nuclei corresponds micromorphologically to a thin layer of bone. The cytological changes from the centre of the cleft to its periphery are partly the same as those in a zone of endochondral ossification. A regular feature is the degenerative picture with swollen cells.

Two regular morphological features in a normal zone of endochondral growth are cell proliferation and reorganisation of the collagen. Cell proliferation is visible in the form of mitoses that result in the typical, radial arrangement of the chondrons. The direction of the collagen can be studied with a polarisation microscope, a regular arrangement resulting in bi-refringency with the axis along the line of the collagen (*Pfeiffer* 1949).

An ordinary zone of proliferation photographed with a polarisation microscope is shown in Figure 6. In this zone the collagen lies in the same direction as the radial chondrons (*Tonna* 1964). Outside the proliferation zone itself, the collagen may lie mainly at right-angles to this (Figure 6) or its direction may vary.

Owing to postmortal autolysis, mitoses cannot be demonstrated with certainty in this material. The grouping of chondrocytes may vary in the periphery of coronal cleft vertebra (Figures 7a and 8a). In a few cases (cf. the right border of the cleft in Figure 7a) the cell organisation

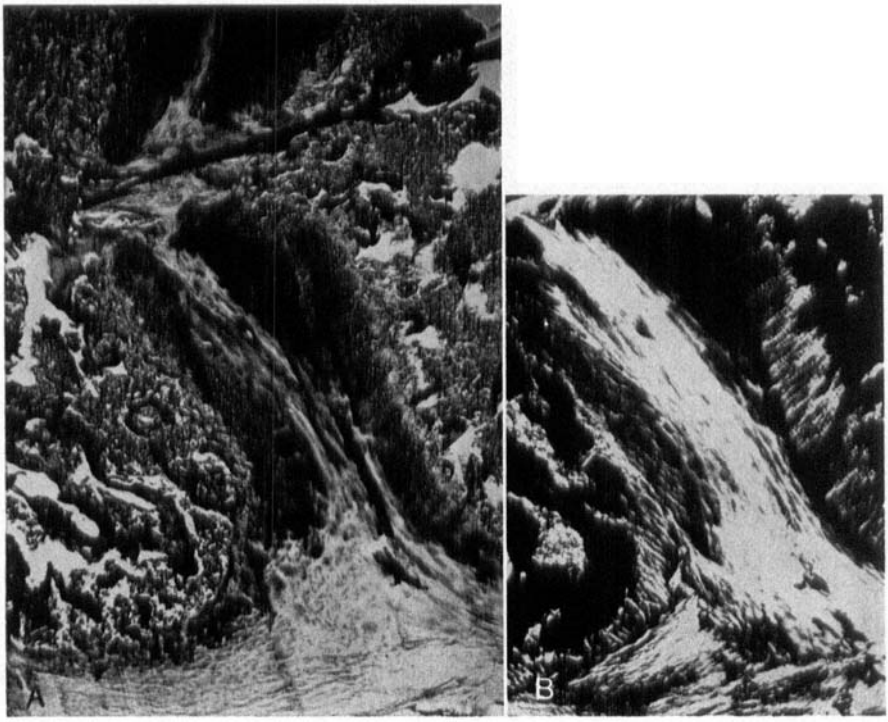
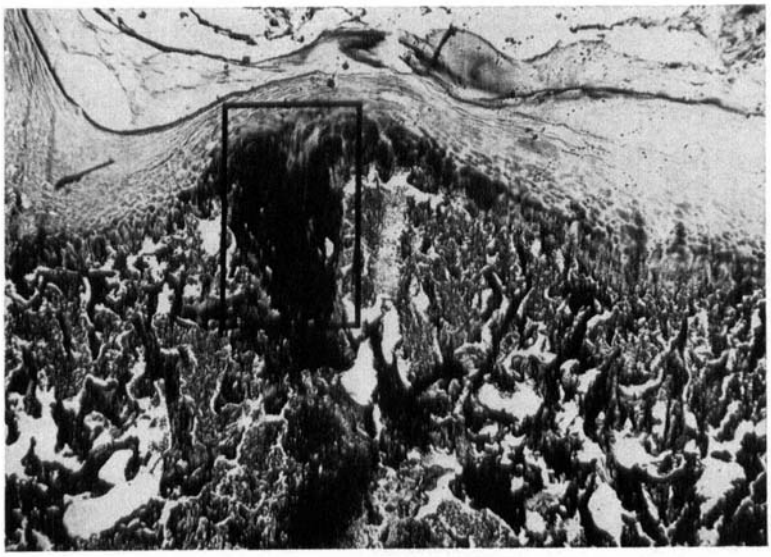


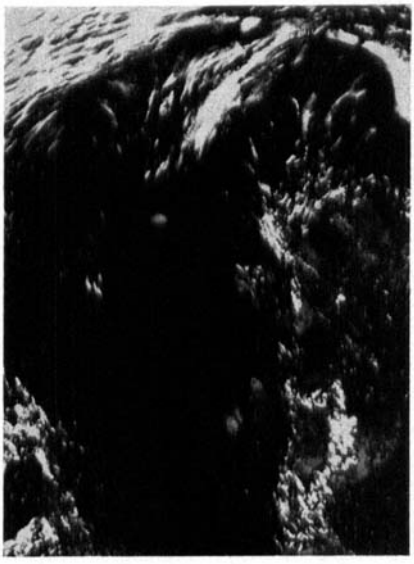
Figure 7 A. Sagittal section through a coronal cleft vertebra, showing normal cell organisation to the right of the cleft and a zone of weak staining in the centre (× 45). Figure 7 B. Polarisation microphotograph of the same vertebra as in Figure 7 A, showing normal orientation of the collagen at the right margin of the cleft and no demarcation of the central zone collagen at the basis of the cleft (× 45).

is much the same as that found in normal zones of growth and the picture shown by the polarisation microscope is then normal too (Figure 7b). In most cases, however, the cells are less organised and the polarisation microscope then usually shows isolated streaks of bi-refringency running in the direction of the cleft or, alternatively, there may be no bi-refringency (cf. Figure 8 and the left border of the cleft in Figure 7).

Occasionally the upper or lower centre of ossification in the vertebral body is deformed where it connects with a coronal cleft (Figures 8 and 9). The deformation consists in the cartilaginous part of the vertebral body being raised together with the chondro-osseous interface. Consequently the deformation can be detected in roentgenograms (Figure 2a). Histologically, these cases show an atypical transition between the



A



B

Figure 8. Sagittal section of coronal cleft vertebra as seen in the light microscope (A) and the polarisation microscope (B). No bi-refringency can be detected in the margins of the cleft. The upper zone of the vertebral body is raised and some irregular bi-refringent streaks are visible at the junction (× 25; × 90).

cartilaginous cleft and the cartilage of the vertebral body. Large clumps of cartilage cells alternate with areas of intercellular substance, in which the distribution of bi-refringent substance is irregular (Figure 8b).

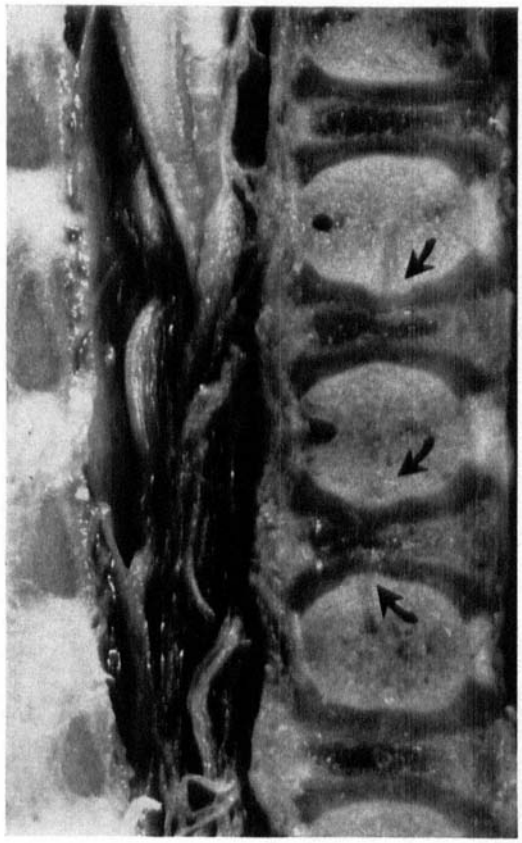


Figure 9. Infra-red photograph of median section. Hyaline cartilage appears dark. Three vertebrae display coronal clefts and deformation of adjacent parts of the chondrous tissue.

A varying amount of tissue that differs from the surrounding cartilage may occur in the centre of coronal clefts (Figure 7). Both the cells and the intercellular substance stained poorly in the specimens investigated. The cells, clearly distinguishable in the phase contrast microscope, are closely packed with no apparent grouping. They are oval in form with the major axis parallel to the line of the cleft. The polarisation microscope shows pronounced bi-refringency, with the axis in line with the cleft. The bi-refringent substance does not appear to penetrate to surrounding parts of the cleft but does continue into the chondrous part of the vertebra, which suggests that it consists of collagen. One such central zone was studied in the fluorescence microscope. Its autofluorescence was bluish-white, whereas the rest of the cleft emitted only slight amounts of blue light.

Figure 10. Unexplained radiolucent areas in vertebral bodies at birth.

Incidence of Coronal Cleft Vertebra

The incidence has been determined on the basis of the roentgenograms, only clear-cut cases being included. In three cases (Figure 10) small, well-defined radiolucent areas were seen in the centre of vertebral bodies from newborn individuals. Histological specimens have not yet been prepared in these cases and consequently the nature of these radiolucent areas has not been investigated. They have therefore not been included among the cases with coronal clefts.

The number of individuals with coronal cleft vertebra demonstrated roentgenologically is reported in Figure 11. The division into subgroups in age groups II and III is shown in Figure 12. It will be seen that approximately one-third of all individuals below the age of 4 years in this autopsy series displayed one or more coronal cleft vertebrae in the segments $L_1 - S_1$. The distribution by segments is shown in Figure 13 and the sex distribution in Table 1.

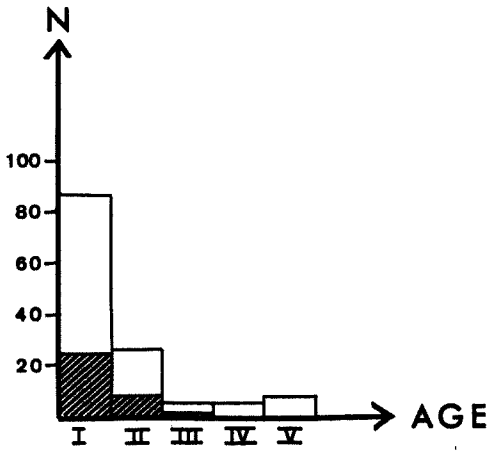


Figure 11. Distribution of the series by age; the shaded areas represent cases of coronal cleft vertebra. I, 0–14 days; II, 2 weeks–23 months; III, 2–7 years; IV, 8–15 years; V, 16–25 years.

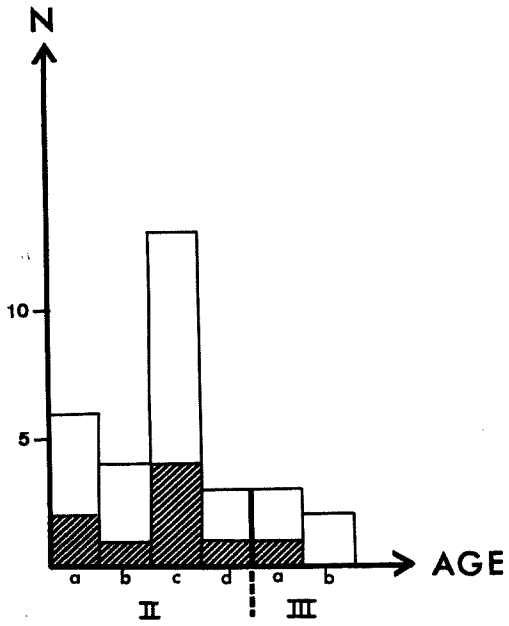


Figure 12. Age groups II and III subdivided. IIa, 2–7 weeks; IIb, 2–6 months; IIc, 7–14 months; IId, 15–23 months; IIIa, 2–3 years; IIIb, 4–7 years.

Correlation between Coronal Clefts and other States

Table 2 shows the number of malformations discovered at routine autopsy in those age groups in which coronal cleft vertebra occurred. Cases with coronal cleft vertebra were not concentrated to any of the individual malformations in each group and it will be seen that no correlation could be found between such vertebrae and the malformations detected.

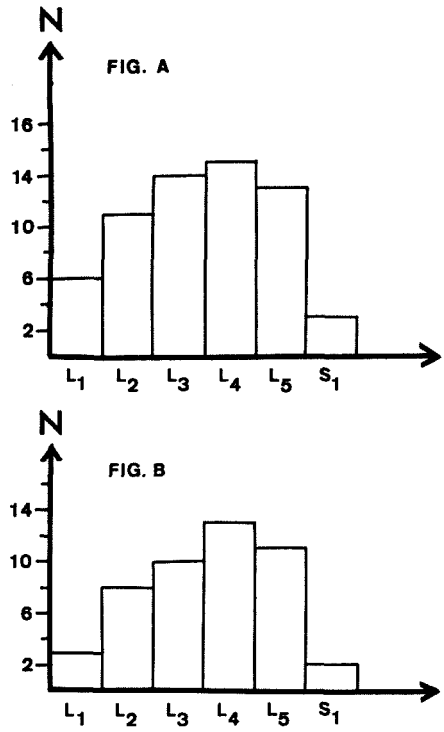


Figure 13. Distribution by segments studied in all projections (A) and in lateral projection only (B).

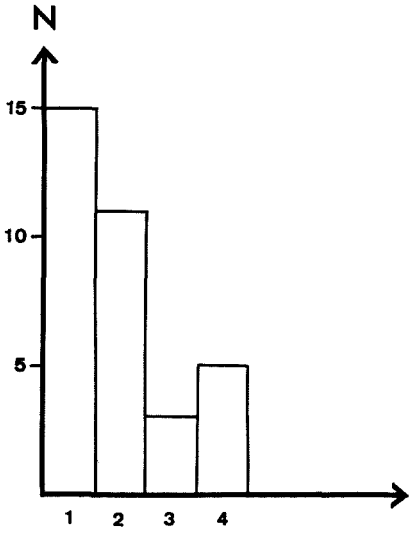


Figure 14. Number of individuals (ordinate) with different numbers of coronal cleft vertebrae in the segments L_1–S_1.

The number of cases with coronal cleft vertebra are related to the cause of death in Table 3. Many individuals died of multiple causes, each of which has been tabulated separately. The category "Respiratory disease" includes neonatal asphyxia. The causes of death listed as "Fetus mortus ante partem" have certainly been various and are largely ascribable to complications of pregnancy, which are to be investigated. The cases of coronal cleft vertebra are evenly distributed among the various categories with the exception of "Tumor cerebri" and "Rh immunisation", for which the incidence of such vertebrae is significantly higher than one would expect to find with an even distribution between the causes of death. The same result is obtained if the cases with coronal cleft vertebra are related only to the primary cause of death (cf. Table 4).

Table 1. Distribution of cases with coronal cleft vertebra by sex and age.

Sex	Age				
	0–3 years		> 3 years		
	No cleft	Cleft	No cleft	Cleft	
♂	46	23(33%)*	7	0	76
♀	34	11(24%)*	9	0	54
Unknown	2	0	0	0	2
	82	34	16	0	132

* Incidence for age group 0–3 years.

Table 2. Incidence of coronal cleft vertebra in cases with malformations in the age group 0–3 years.

Type of malformation	No. of cases with malformation	Of which with coronal cleft vertebra	
		No. found	No. expected*
All types	28	8	8
CNS anomalies	12	3	4
Skeletal anomalies	8	3	2
V.O.C. cong.	11	3	3.2
Other viscera	9	2	2.6
Mongolism	1	0	0
Multiple anomalies	4	0	1

* No. expected, assuming an equal distribution between the groups with and without malformations.

The number of specimens with one, two, three or four clefts in the segments $L_1 - S_1$ is shown in Figure 14.

A comparison between the occurrence of coronal cleft vertebrae and supernumerary bone nuclei in the zone of cartilage between the vertebral body and the pedicle (Figure 2) or in the inferior articular process (Figure 1a) is given in Table 5, concerning the ages in which such clefts were found. As shown by the χ_2 analysis, there is a positive correlation between the occurrence of coronal cleft vertebrae and clefts in the inferior articular process. Likewise if the clefts of the articular process are regarded as the result of supernumerary bone nuclei as the histology suggests and their frequency is added to the one of the pedicle zone nuclei a significant correlation exists with coronal cleft vertebrae. No significant correlation could however be demonstrated between coronal cleft vertebrae and bone nuclei in the pedicle's region of growth.

Table 3. Incidence of coronal cleft vertebra in relation to cause of death in the age group 0–3 years.

Cause of death	No. of cases*	Of which with coronal cleft vertebra		χ_2
		No. found	No. expected†	
Intracranial haemorrhage	16	3	4.7	–
Prematurity	40	9	12	–
Tumor cerebri	2	2	0.6	4.91
Respiratory disease	43	14	13	–
Injury of birth	12	3	3.8	–
V.O.C.	11	3	3.2	–
Accident	1	0	0	–
Cerebral anomaly	9	3	3	–
Fetus mort. ante partum	27	9	8	–
Rh-immunisation	2	2	0.6	4.91
Urinary tract disease	3	1	1	–
Infectious disease	9	5	3	–
Other causes	9	2	3	–
Unknown cause	3	0	1	–

* Individuals with multiple causes are included in all the relevant groups.

† No. expected, assuming an equal distribution between the cause of death in question and other causes.

The χ_2 value is given for the groups in which the χ_2 analysis refuted the hypothesis.

Table 4. Incidence of coronal cleft vertebra in relation to primary causes of death in the age group 0–3 years.

Primary cause of death	No. of individuals	Of which with coronal cleft vertebra		χ_2
		No. found	No. expected*	
Prematurity	15	3	4.5	–
Tumor cerebri	2	2	0.6	4.91
Respiratory disease	23	9	7	–
Injury of birth	12	3	3.8	–
V.O.C.	11	3	3.2	–
Accident	1	0	0	–
Intracranial haemorrhage	16	3	4.7	–
Cerebral anomaly	9	3	3	–
Fetus mort. ante partum	13	3	4	–
Rh-immunisation	2	2	0.6	4.91
Infectious disease	5	1	1.5	–
Other causes	4	2	1.2	–
Unknown cause	3	0	1	–

* No. expected, assuming an equal distribution between the cause of death in question and other causes.
The χ_2 value is given for the groups in which the χ_2 analysis refuted the hypothesis.

DISCUSSION

According to *Töndury* (1958), the variation in the shape of the original nucleus, which may lead to the development of a coronal cleft vertebra, is entirely dependent upon the course of the vessels that grow in towards the bone nucleus. We have nevertheless made a distinction between coronal cleft vertebra and clefts occasioned by large blood vessels because, in the coronal clefts studied here, such vessels were never sufficiently large or displayed such a course as to suggest that they were the cause of the cleft as such.

Coronal cleft vertebrae were found in one or more segments studied in approximately one-third of all autopsy cases below the age of 4 years. This incidence corresponds to that reported by *Rettig* (1958) and is considerably higher than those published by other investigators (*Meyer-Burgdorff & Klose-Gerlich* 1935, *Cohen et al.* 1958, *Fagerberg* 1963) owing to differences in the composition of the material and the methods used for the investigation.

The incidence declines from the age of 4 years onwards. Since coronal cleft vertebrae are not found in the adult, it seems that they generally disappear during the fourth year of life. This is supported by the ob-

Table 5. Comparison between the incidences of supernumerary bone nuclei and coronal cleft vertebrae in the age group 0–3 years (n = 16).

Individuals with bone nucleus in the pedicle's zone of growth 7	of which, with coronal cleft vertebra 4 ($\chi_2 = 2.79$)
Individuals with cleft in inferior articular process 5	of which, with coronal cleft vertebra 4 ($\chi_2 = 6.49$)
Individuals with bone nucleus in the pedicle's zone of growth and cleft in inferior articular process 2	of which, with coronal cleft vertebra 2 ($\chi_2 = 4.91$)
Individuals with bone nucleus in the pedicle's zone of growth and/or cleft in inferior articular process 10	of which, with coronal cleft vertebra 8 ($\chi_2 = 13.57$)

servation that the cleft becomes narrower with increasing age. The histological demonstration of a thin border of bone tissue and the occurrence of cell degeneration in the periphery should therefore be taken to indicate a process of ossification.

If the ossification is combined with endochondral growth, a coronal cleft should be able to function as a zone of growth. Such growth in an asymmetric coronal cleft might be liable to result in deformation of the vertebral body. In the majority of cases, however, there are histological differences in the chondro-osseous interface between normal zones of growth and coronal clefts. In such cases the ossification appears to be preceded by true degeneration of the cleft's periphery. It may be noted, however, that certain coronal clefts display areas that cannot be distinguished from normal zones of endochondral growth with the methods used. The deformations described in the chondrous part of the vertebral body in connection with a coronal cleft may possibly express an increased proliferation in conjunction with the cleft. As mentioned above, no asymmetry of vertebra could be demonstrated as a result of growth in connection with a coronal cleft but, in view of the infrequency of the histological phenomena reported, further study of this question may be called for.

Malformations in other internal organs are largely due to impairment of the normal process of development. The occurrence of coronal cleft

vertebra has also been included in this category and, as mentioned above, has been regarded as a result of abnormal persistence of the notochord. The only possibly support for this hypothesis in the present material is the finding of central parts which stained poorly. In the polarisation microscope, however, their collagen is connected so naturally with that in the chondrous part of the vertebra that it appears unlikely that this tissue represents a notochordal remnant. Moreover, persistence of the notochord should have resulted in clefts in several segments in the same spine to a greater extent than was actually the case. Since there was also no statistical correlation to anomalies in other organs, which may largely be ascribed to impaired development, it seems improbable that coronal cleft vertebrae in general are to be regarded as malformation. Children who died from cerebral tumours or Rh immunisation displayed a significantly higher incidence than expected of coronal cleft vertebra. From the investigations made to date, however, no explanation is possible.

Individuals with coronal cleft vertebrae also display statistically inward frequency of supernumerary bone nuclei in the inferior articular process as well as such nuclei in combination with bone nuclei in the chondrous zone between the vertebral body and pedicle. It therefore seems reasonable to suppose that at least the majority of coronal cleft vertebrae constitute a normal variation in the development of the vertebral body's bone nucleus.

SUMMARY

The study was made on 132 autopsy cases. In the age group 0–3 years, about one-third of the cases displayed one or more coronal cleft vertebrae in the segments $L_1 - S_1$. Such clefts should be distinguished roentgenologically from radiolucency as resulting from large blood vessels as well as from supernumerary bone nuclei in the chondrous zone between the vertebral body and pedicle. Ossification appears to be a regular micromorphological finding, while proliferation appears to be impaired in most places. In some cases the picture in the light microscope corresponded to the zone of normal growth and consequently coronal clefts may, perhaps, play a significant role in the development of asymmetrical vertebral bodies. The majority of coronal cleft vertebrae probably represent an atypical pattern in the development of the vertebral body's bone nucleus.

RESUME

Cette étude a été pratiquée sur 132 cas d'autopsies. Dans le groupe d'âges 0–3 ans, il y avait dans un tiers des cas une ou plusieurs fissures coronaires des vertèbres des segments $L_1 - S_1$. Ces fissures doivent être distinguées radiologiquement des taches lumineuses resultant des larges vaisseaux sanguins aussi bien que des noyaux osseux surnuméraires dans la zone chondrale entre le corps de la vertèbre et le pédoncule. L'ossification se révèle comme une véritable trouvaille micromorphologique, la proliferation apparaissant endommagée en bien des endroits. Dans certains cas, le tableau au micriscope lumineux correspondant à la zone de la croissance normale et il est par consequent possible que les fissures coronaires aient une influence sur le développement de corps vertebraux asymétriques. La majorité des vertèbres ayant une fissure coronaire représentent probablement un modèle atypique du développement des noyaux osseux du corps vertébral.

ZUSAMMENFASSSUNG

Eine Studie wurde an 132 Autopsiefällen ausgeführt. In der Gruppe 0–3 Jahre zeigte ungefähr ein Drittel der Fälle einen oder mehrere koronale Spaltwirbel der Segmente L_1–S_1. Solche Spalten sollten röntgenologisch von Röntgendurchsichtigkeit, wie sie sowohl durch grosse Blutgefässe als auch durch überzählige Knochenkerne in der Knorpelzone zwischen Wirbelkörper und -stiel entsteht, unterschieden werden. Verknöcherung scheint ein regulärer mikromorphologischer Befund zu sein, während Proliferation in den meisten Fällen herabgesetzt zu sein scheint. In einigen Fällen entspricht das Bild im leichten Mikroskop der Zone des normalen Wachstums und es kann daher möglich sein, dass koronale Spalten für die Entwicklung von asymetrischen Wirbelkörpern bezeichnend sind. Die Mehrzahl von koronalen Spaltwirbeln stellen wahrscheinlich eine atypische Form in der Entwicklung des Knochenkernes des Wirbelkörpers dar.

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