

Terminal Plate Fracture in Vertebrae of the Aged

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Summary. The terminal plate fracture of human vertebrae was studied morphologically on midsagittal specimens from 92 autopsy cases which included 82 patients over 60 years.

1. *Histologically*, the terminal plate fracture was classified into three types by its representative histology. (a) *Fracture with intervertebral disk (ID) prolapse*: a healed fracture characterized by the presence of the prolapsed ID at the fractured site. ID prolapse in the marrow cavity may or may not be present. The group includes the typical case of the Schmorl's nodule. (b) *Fracture with the granulation tissue*: The fracture site was embedded by the granulation tissue. Callus and ID prolapse, if any, were insignificant. (c) *Diffuse replacement of the hyaline cartilage layer by osteoid tissue*: The alteration is seen often at a concaved surface of the terminal plate of significantly deformed vertebrae.

2. *The fracture* may be classified by the depth of the ID prolapse. (a) *Grade 1*: ID prolapse may be seen within the fracture aperture, but, not in the marrow cavity. (b) *Grade 2*: ID prolapse is confined to a labyrinth of subcortical trabeculae. (c) *Grade 3*: ID prolapse extends to a deep marrow space. The typical Schmorl's nodule belongs to this group.

3. In the present study, the terminal plate fracture was noted in 78% of the patients over 60 years. The female group was affected more often (68% in male, 86% in female). Grade 2 was more often seen in atrophic vertebrae than Grade 1. However, in Grade 3, the fracture appeared unrelated to the atrophy of the vertebrae.

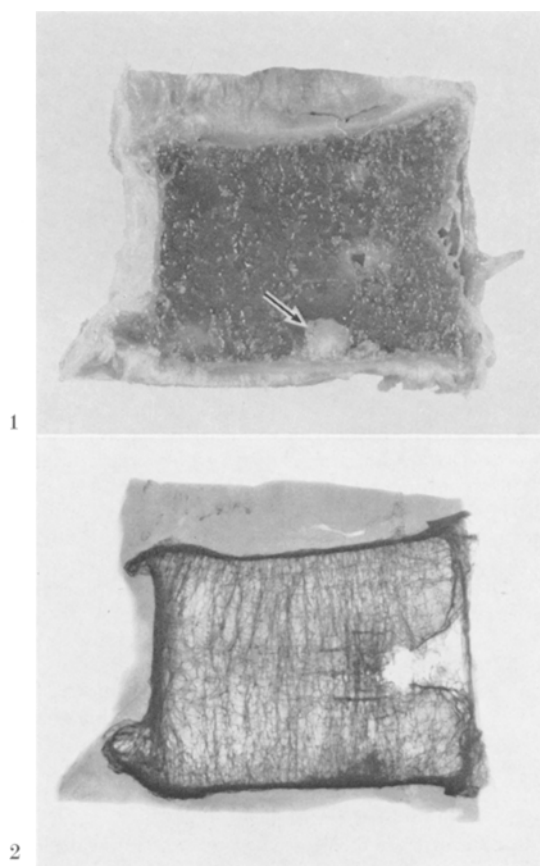
4. The terminal plate fracture was more often seen in the lower deck than the upper deck at the site of two-thirds dorsal from the ventral edge of the vertebral body. The site corresponds roughly to the nucleus pulposus.

5. Histologic variations of the terminal plate fracture and of ID prolapse may relate to senescence of both vertebrae and ID.

Key words: Terminal plate fracture — Human vertebrae — Schmorl's nodule — Ageing.

A unique prolapse of the intervertebral disk (ID) into the pars spongiosa by the terminal plate fracture of the vertebral body is known as Schmorl's nodule (Schmorl, 1927, 1928; Gardner, 1965; Doerr and Uhle, 1970; Lichenstein, 1970). According to Schmorl (1928), its incidence is high—up to 38% of nearly 3000 vertebral columns he examined. In a typical case, the involved vertebrae are distinguished macroscopically by the presence of a whitish nodule which protrudes into the pars spongiosa.

During a comparative radiographic and histologic study on the vertebrae of the aged we frequently noted a minute fracture or dissociation in the cartilage layer of the terminal plate in the least deformed vertebrae. The size of the lesion varies from a minute type which can be detectable only by histologic examination to a type distinguishable grossly by the presence of ID prolapse (Fig. 1, 2). The latter type corresponds to classic Schmorl's nodule (Gardner, 1965; Lichenstein, 1970; Doerr and Ule, 1970). The original reports of Schmorl (1927, 1928) described the alteration in the aged vertebrae confining to the type which showed an extensive vertebral deformation. However, the fracture or dissociation of the terminal



Figs. 1 and 2. Classic Schmorl's nodule observed in a 86-years-old female. Photos of macroscopic specimen (Fig. 1) and its radiograph (Fig. 2). Insignificant deformation of vertebra and cartilage nodule on lower deck (arrow) seen in Figure 1. Reactive trabecular condensation apparent at corresponding site (Fig. 2). both ca. $\times 1.3$

plate we noted was present in vertebrae with insignificant deformation. An analysis on this aspect, we thought, may contribute to the understanding on vertebral changes in the aged subjects.

The purpose of this paper is to describe morphologically the alterations of the terminal plate in the aged vertebrae and discuss its relation to vertebral deformation and classic Schmorl's nodule.

Materials and Methods

The midsagittal specimens of 92 vertebrae used for the previous radiographic analysis on aged subjects (Tanaka, 1975) were the main samples for this study. The age sex classification of the materials is listed in Table 1. The terminal plate fracture was studied on the specimens which satisfied the following criteria: (1) In less atrophic vertebrae, a least deformation of its craniocaudal axis is present. (2) In atrophic vertebrae, the terminal plate alter-

Table 1. Age and sex classification of the materials and incidences of the terminal plate fracture

Age Group	Sex	Fracture		Total	Percentage	
		no	yes		sex diff. (%)	total (%)
under 59	male	2	3	5	60	40
	female	4	1	5	20	
60-69 years	male	3	5	8	63	67
	female	1	3	4	75	
70-79 years	male	4	11	15	73	82
	female	3	21	24	86	
80-89 years	male	5	10	15	67	76
	female	2	12	14	86	
90-99 years	male	0	0	0	—	100
	female	0	2	2	100	
Total		24	68	92		74%

ation should not contribute to deformation of the vertebral body. Consequently, vertebrae with significant deformation by apparent fracture were eliminated.

The midsagittal planes were decalcified by EDTA-Na and embedded in celloidin-paraffin. Thin sections of 5-10 micra were stained with hematoxylin-eosin (H & E), Masson's trichrome stain, and periodic acid Schiff's reagent.

Results

The terminal plate of the human vertebrae here defined is a bony deck which faces to the ID and consists of a hyaline cartilage and cortical bone. Histologically, the structure is similar to that of the surface of the articular joints and can be referred elsewhere (Arey, 1974).

1. The Terminal Plate Fracture

Sixty-eight vertebrae satisfied our present criteria. Histologically, the fracture (or dissociation) of the terminal plate cartilage was distinctive being associated with various changes in the neighboring tissues, such as the cartilago-osteal layer of the terminal plate and bone marrow. The alterations of the vertebrae themselves were insignificant except for a slight reactive hypertrophy and condensation of the bone trabeculae at the fracture site. Thus the definite alteration is confined to the cartilago-osteal layer. Three types may be distinguished histologically. The classification may be arbitrary and coexistence of more than two changes may be seen in one fracture. In such cases, the most representative alteration was counted.

a) Fracture with ID Prolapse. The fracture aperture of the cartilago-osteal layer may be embedded by the prolapsed ID (Fig. 3-5) and this type was most common in about 70% of all fractures. Because of its histologic characteristics, such as insignificant local reaction and distinctive bone formation, the change is interpreted as a healed fracture. The prolapsed materials were detected easily by



Figs. 3—5. Terminal plate fracture with ID prolapse

Fig. 3. Terminal plate fracture of Grade 1. Fracture of terminal plate and part of prolapsed ID (arrow) seen. Female: 74 years old, Masson, $\times 22$



Fig. 4. Terminal plate fracture of Grade 2. ID prolapse into subcortical trabecular labyrinth through fracture aperture. Note presence of reactive trabeculae which surrounded material. Female: 72 years old, Masson, $\times 22$

histologic characteristics of the ID and a positive PAS reaction of mucopolysaccharides in the cartilage. Secondary ossification of various degrees may be seen around the prolapsed ID. The material spread in a cartilago-osteal junction of

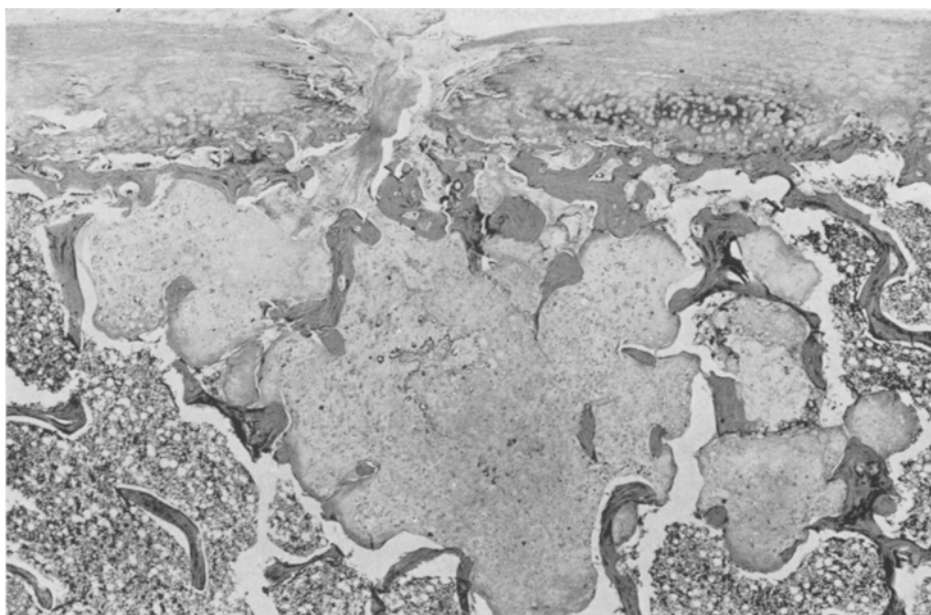


Fig. 5. Terminal plate fracture of Grade 3. Prolapsed ID, detectable by its morphologic characteristics, into deeper marrow space and reactive bone formation on surface of prolapsed ID. Female: 75 years old, Masson, $\times 22$

the terminal plate, in a subcortical labyrinth of bone trabeculae, or in a deep marrow space. Sometimes the fracture site showed almost no reaction except the presence of the ID material. This change was more common in the terminal plate with degenerative appearance. The prolapsed ID usually showed characteristics of chondrocytes, however, fibrous materials resembling the annulus fibrosus were also noted. The fracture ran in a rectangular or oblique direction in the terminal plate. The latter type lacerated the cartilage layer for a distance. Sometimes, the hyaline cartilage of the terminal plate disappeared and the defected area was reinforced by the cortical bone. The change is possibly created by absorption and remodeling of the preceding fracture.

Histologically, an incomplete fracture or a recessus was seen in the cortical bone and cartilage at the marrow side. The site was repaired completely by the callus. Neither ID prolapse nor local alteration in the ID at the corresponding site was observed. This fracture was frequently seen at a distant position from a larger fracture. We interpret the lesion as an incomplete fracture which may be formed by a hinge-effect when the terminal plate fracture occurred at a distant position.

b) Fracture with the Granulation Tissue. In place of ID prolapse, the granulation tissue embedded the fracture aperture. The change may be a healing fracture. An example is shown in Figures 6 and 7. A fracture of this type was noted in 20% of all fractures. ID prolapse or fibrocartilaginous callus, if any, was insignificant. In the case of a large fracture, a nodular granulation tissue

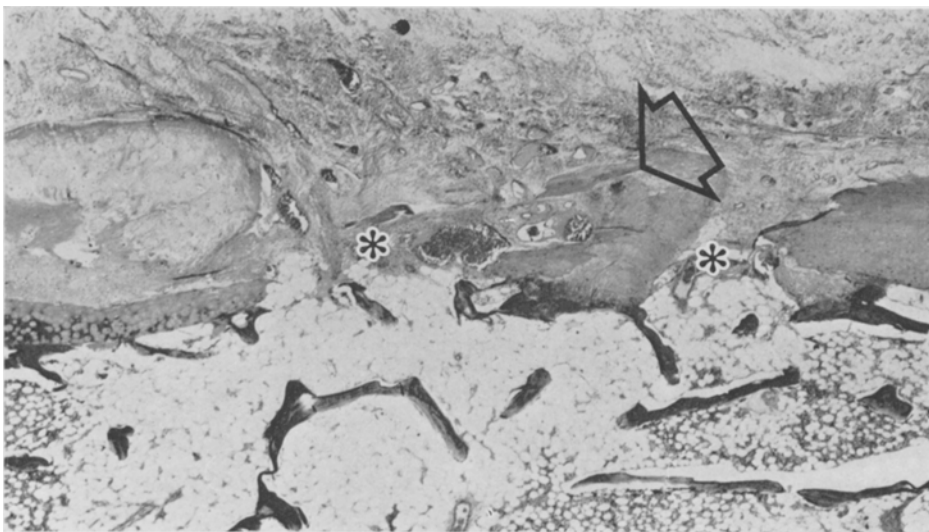


Fig. 6

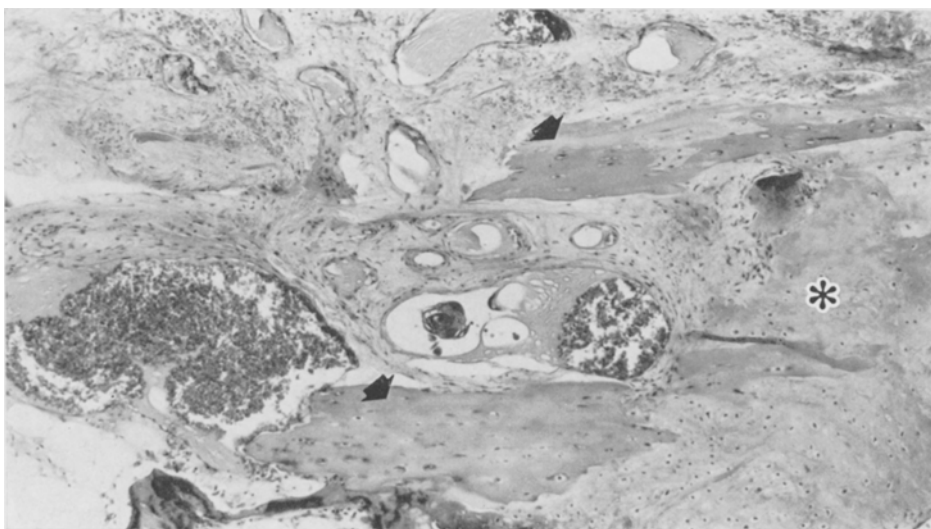


Fig. 7

Figs. 6 and 7. Fracture with granulation tissue. Terminal plate defected at two locations where granulation tissue embedded fracture apertures (asteriks). Many venules seen in ID (Fig. 6). Higher magnification of area indicated by open arrow in Figure 6 is illustrated in Figure 7. Note presence of fragmented hyaline cartilages (closed arrows) of the terminal plate and fibrocartilaginous callus (asterisk). Female: 87 years old, Masson, $\times 22$ and $\times 80$

extended to a deep marrow cavity inducing local fibrosis and reactive hypertrophy of adjacent bone trabeculae. Vascular extension may be seen in the ID at the fracture site. Vasculatures in the granulation tissue must originate from the marrow side since no vascular supply is available from the ID side. The terminal

plate of the affected vertebrae was usually thin and degenerative in comparison to the previous group.

c) *Diffuse Replacement of the Cartilage Layer by the Osteoid Tissue.* The type classified here is a complicate alteration and the term, "fracture", may not be appropriate to describe the lesion. In a typical case as shown in Figures 8 and 9, the terminal plate cartilage was replaced diffusely by the osteoid tissue or the callus. The cortical bone became extremely thin, but, detectable beneath the osteoid tissue or the callus. Sometimes, the osteoid tissue involved the cartilage layer for a distance interrupting it at multiple locations. The alteration was noted more often in atrophic vertebrae of advanced deformation, the type which was eliminated from the present investigation.

2. *Extent of ID Prolapse and its Relation to Schmorl's Nodule*

The terminal plate fracture may be classified into three types by the depth of the ID prolapse in the marrow cavity. The classification may be arbitrary since several microscopic sections cut from the same block showed a difference in grade classification. *Grade 1:* ID prolapse confined to the fracture site of the terminal plate (Fig. 3). *Grade 2:* The ID material spread in a subcortical labyrinth of bone trabeculae (Fig. 4). In atrophic vertebrae, this zone became thin, but, reactive hypertrophy of the bone trabeculae was usually seen. *Grade 3:* The ID material reached a deep marrow space and formed a nodular lesion (Fig. 1, 2, 5) compatible to classic Schmorl's nodule.

Grossly, Schmorl's nodule was noted in 9 vertebrae. In addition, the ID material was detected histologically in a deep marrow cavity of 6 vertebrae which failed to show a nodule in macroscopic samples. Thus 15 vertebrae had Schmorl's nodule. In a typical case, as of Figures 1, 2, and 5, the prolapsed ID showed a typical histology of the cartilage and embedded local trabeculae and marrow space. Usually ossification was noted on the surface of the prolapsed ID (Fig. 5). In macroscopic specimens which showed typical Schmorl's nodule, sometimes, no corresponding alterations were seen in histologic slides. The phenomenon may be due to change in an observation level by trimming of the paraffin blocks during the preparation of histologic sections. In macroscopic specimens classified in both Grades 1 and 2, indistinctive thickening of the trabeculae was seen at the fracture site. However, such alteration may be easily overlooked due to its insignificance.

3. *Incidence of the Terminal Plate Fracture*

An incidence of the terminal plate fracture in bone over 60 years reached 78% (68/82) of the total specimens of the corresponding age group. The value is significantly higher than those reported on Schmorl's nodule by others (Schmorl, 1928; Wissing, 1930). The terminal plate fracture was noted in 40% (60% in male, 20% in female) of the specimens of under 59 years. However, the data are contributory only as a reference because of a limited number of cases examined. It should be kept in mind that our selection of material was not made primarily for the study of vertebral fracture. We selected rather grossly, from the individual lumbar column which consisted of more than 3 consecutive vertebral bodies, the vertebrae with least deformation in order to study age-related alterations of

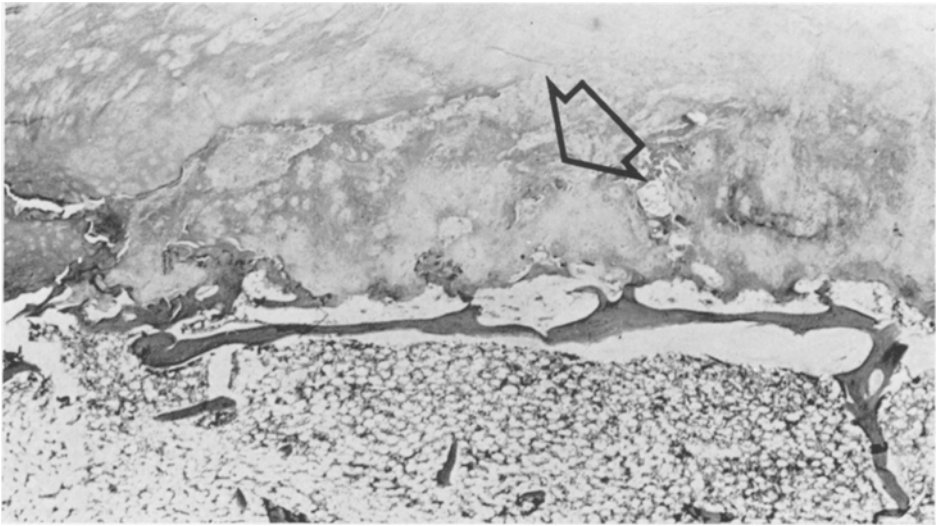


Fig. 8

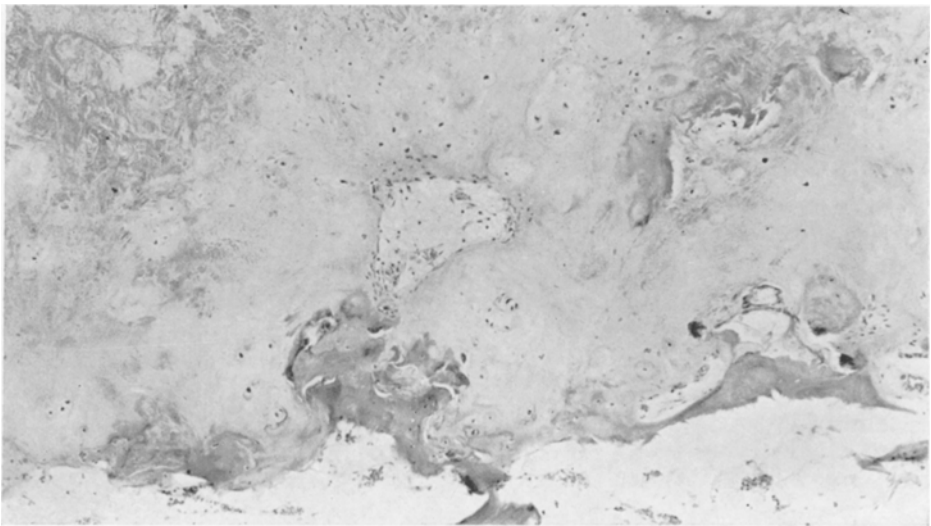


Fig. 9

Figs. 8 and 9. Terminal plate replaced diffusely by osteoid tissue or callus which covered by thin layer of bone. Higher magnification of part of osteoid tissue indicated by open arrow in Fig. 8 illustrated in Fig. 9. Change is unique and often seen in terminal plate of highly deformed vertebrae. Female: 83 years old, H. & E., $\times 22$ and $\times 80$

vertebral bodies (Tanaka, 1975). Thus the incidence of the terminal plate fracture may differ when the level of the target vertebra is defined. As shown in Table 1, the present analysis indicates that the highest incidence was in the materials of the 7th decade group (82%). In female the terminal plate fracture was more often seen in the group over 60 years. In Grade 2, the terminal plate

Table 2. Relation between grades of the terminal plate fracture and the vertebral astrophy classified by radiography

Grades of ID prolapse	Grades of atrophy in radiography					Cases examined
	I (%)	II (%)	III (%)	IV (%)	uc (%)	
No fracture	25	21	33	17	4	24
Grade 1	12	42	22	24	0	33
Grade 2	0	9	62	24	5	21
Grade 3	7	21	36	29	7	14
Total						92

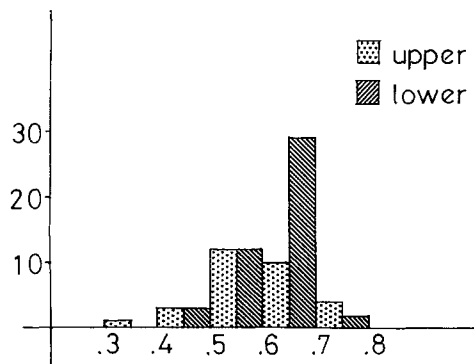


Fig. 10. Histogram of incidence of terminal plate fracture in upper and lower decks. Numbers of fracture on ordinate and ratios of distance between ventral edge of vertebrae and fracture point to ventrodorsal length of deck on abscissa. The Fracture is most often seen at center of upper deck and at two-thirds point of lower deck from ventral edge

fracture was more often seen in atrophic vertebrae than in Grade 1. Grade 3, which includes classic Schmorl's nodule, was seen often in vertebrae with less atrophy (Table 2). The finding suggests that the larger fracture may occur unrelating to vertebral atrophy.

The fracture occurred on both upper and lower terminal plates. Sometimes in one vertebra, both plates were simultaneously affected without significant vertebral deformation. Analysis on the fracture location showed that the lower plate was more often affected (ratio of 2 to 1 dominant in the lower plate). The result appeared to be in good agreement with a classic observation of Wissing (1930) on Schmorl's nodule. The fracture occurred frequently at a position two-thirds dorsal from the ventral edge of the vertebrae (Fig. 10). The position corresponds roughly to the site of the nucleus pulposus.

4. Relations to Others

a) *The Terminal Plate of the Nonfractured Group.* Histologically nonfractured specimens from all age groups showed degenerative change including irregularity in thickness, disarrangement of chondrocytes, vacuolization or rarefaction of the matrix, and invasion of small vasculatures within the terminal plate cartilage.



Fig. 11. An apparent split (arrow) in terminal plate otherwise nonsignificant alteration. Change counted as disposition rather than fracture. Female: 91 years old, Masson, $\times 22$

Changes occurred either singularly or multiply, although to a lesser extent, these changes were already noted in materials of nonfractured vertebrae of the 3rd decade group. Occasionally, the terminal plate became so thin that a fracture could be expected at any moment. This alteration may be a disposition for future fracture. One example with an apparent split in the terminal plate cartilage is shown in Figure 11.

b) Effects of the Local Fracture to the Entire Vertebral Body. Large fractures frequently induced local condensation and disarrangement of the trabeculae detectable by both radiography (Fig. 2) and histology (Fig. 4). Radiographically local condensation and disarrangement of the trabeculae were also seen in vertebrae with a minute fracture which was hardly detectable by macroscopic examination. The fractured vertebrae associated frequently with an extensive hypoplasia or fatty changes of the bone marrow without fibrosis. We interpret presently that the lesion is induced by direct vascular damage which creates an environmental alteration unfavorable to generate hematopoiesis.

Discussion

Schmorl (1927, 1928) reported that the nodule was present in 38% of nearly 3000 vertebral columns and noted more often in adult males and aged females. The latter observation is in good agreement with our present result. Possible contribution of the trauma for the adult male and osteoporosis for the aged female to the formation of the Schmorl's nodule was suggested. However, a direct relation to the trauma was denied in his later publication (Schmorl, 1928). The presence of the unique vertebral lesion was confirmed by others (Putschar, 1927; Wissing, 1930). Wissing reported as follows; (1) a lower incidence of the lesion,

13.5% in 421 vertebral columns by radiography, (2) both upper cervical and lower lumbar vertebrae were favorable sites, (3) incidence was equal in both sexes, (4) the lower terminal plate was involved more often, (5) no clear relation to the trauma. In our present study, Grade 3 lesion was noted in 16% (15/92) of the materials. Apparently the incidence of Schmorl's nodule differs among investigators, however, these data cannot be compared easily because of differences in the methods used and the definition of the lesion. In our present study, the incidence of the terminal plate fracture, which may or may not relate to classic Schmorl's nodule, is exceedingly high probably reflecting advancing senescence of the vertebrae in the aged. In his original reports, Schmorl (1927, 1928) showed some histologic characteristics of the terminal plate fracture here described in relation to the mechanism of ID prolapse and its healing. However, the description was not confined to the alteration in the aged vertebrae and the terminal plate fracture in advanced osteoporosis was of highly deformed vertebrae. Therefore, our present study may be contributory to further understanding of physiopathology of the human vertebrae in the aged.

To date, various mechanisms have been proposed for the formation of loci minoris resistentia in the terminal plate (Doerr and Ule, 1970). Our present study showed that the terminal plate fracture occurred most frequently at the anatomical position of the nucleus pulposus. An impaired function of the ID in absorbing both static and dynamic stresses on the vertebral column due to decrease in elasticity of the ID may be important for the terminal plate fracture. Anatomically, development of the subcortical trabeculae of the terminal plate is extremely inferior to that of the articular joints of the extremities. These features, together with exceedingly high incidence of the terminal plate fracture in the aged vertebrae, suggest that the design of the human vertebral column is insufficient to support a heavy stress. The architectural insufficiency may be exaggerated by ageing and may result in terminal plate fracture in aged vertebrae.

In our present investigation, the time when the terminal plate fracture occurred was estimated based on histologic characteristics of the fractures. In the fracture with granulation tissue, callus formation and ID prolapse were noted but to a lesser extent, in contrast to the presence of a large fracture aperture. Diffuse replacement of the cartilage layer by the osteoid tissue suggests that the lesion is formed more recently and there must be certain factors which prevent ID prolapse, such as a decrease in liquidity of the ID. In addition diffuse replacement of the cartilage layer by the osteoid tissue suggests that there may be multiple fractures on the terminal plate. These changes may be more characteristics of the aged vertebrae than the fracture with ID prolapse. Though an apparent fracture is detectable in the terminal plate, these two types of fracture cannot be included in classic Schmorl's nodule because of a failure in the formation of nodular ID prolapse.

Clinical significance of the terminal plate fracture is not known at present. The terminal plate fracture here described may or may not be a consequence of vertebral ageing. In a healed fracture, the fracture site may not be the locus minoris resistentia for the vertebral deformation since a trabecular reinforcement by reactive bone formation is available at the fracture site. In aged vertebrae, when the vertebral deck becomes insufficient to hold the stress, the terminal plate

may be ruptured or bent concavely toward the marrow cavity. Two types of fractures, fractures with the granulation tissue, and one with diffuse replacement of the cartilage layer by the osteoid tissue may be formed during this process.

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References

- Arey, L. B.: Human histology. Philadelphia-London-Toronto: Saunders 1974
Doerr, W., Ule, G.: Spezielle pathologische Anatomie, III, Heidelberger Taschenbücher B 70b: S. 260-263. Heidelberg-Berlin-New York: Springer 1970
Gardner, D. L.: Pathology of the connective disease, pp. 324-326. London: Arnold 1965
Lichenstein, L.: Disease of bone and joints, pp. 155-156. St. Louis: Mosby 1970
Putschar, W.: Über Knorpelinseln in den Wirbelkörpern. Verh. dtsch. path. Ges. **22**, 262-265 (1927)
Schmorl, G.: Über die an den Wirbelbandscheiben vorkommenden Ausdehnungs- und Zerreißungsvorgänge und die dadurch an ihnen und der Wirbelspongiosa hervorgerufenen Veränderungen. Verh. dtsch. path. Ges. **22**, 250-262 (1927)
Schmorl, G.: Über Knorpelknötchen an den Wirbelbandscheiben. Fortschr. Röntgenstr. **38**, 265-279 (1928)
Tanaka, Y.: A radiographical analysis on human lumbar vertebrae in the aged. Virchows Arch. Abt. A **366**, 187-201 (1975)
Wissing, O.: Über Knorpelknötchen in der Columna. Acta path. microbiol. scand., Suppl. **III**, 499-506 (1930)

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