

THE FORCE REQUIRED TO CRUSH VERTEBRAE: ITS PROBABLE MECHANICAL RELATION TO THE POSTMETRAZOL FRACTURE*

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The initial enthusiasm for the metrazol shock treatment of psychoses has subsided since the reports of complicating vertebral fractures. The literature does not explain the high incidence of this otherwise rare fracture. The following factors may be etiologically related:

1. A constitutional factor¹
2. Age and sex variations¹
3. The chemical action of metrazol on bone
4. Disturbance of calcium-phosphorous metabolism
 - (a) Nutritional deficiency¹
 - (b) By muscular contraction²
 - (c) By endocrine stimulation producing transient hyperparathyroidism¹
5. Variation in blood supply to the vertebra
 - (a) By vascular compression
 - (b) By stimulation of the autonomic nervous system producing vasoconstriction

This paper attempts to evaluate the probable significance of the mechanical factors in the production of the postmetrazol vertebral fracture.

The peculiar anatomical nature of the thoracic spine³ may explain the frequency of fracture of the fifth, sixth and seventh thoracic vertebrae. The natural convexity of the thoracic vertebrae is somewhat increased by the thoracic intervertebral disks, which in this region are slightly narrower in front than behind.³ Mobility of this region, moreover, is limited to 90° in flexion and only 40° in extension (in the cadaver). The flexor muscles of the spine (the rectus abdominis, sternocleidomastoid, scalenus anticus, abdominal obliques, psoas major and minor, longus colli and capitis) therefore apparently have a definite mechanical advantage over the exten-

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sors (the sacrospinalis, quadratus lumborum, semispinalis, longissimus dorsi, multifidus, rotatores, interspinales and splenius).

Reed and Davis⁴ believe that, since there are few muscles extending the thoracic spine, generalized severe muscular action produces extension of the cervical and lumbar spine with flexion and angulation of the thoracic region resulting in localization of the postmetrazol fracture to the latter area. Their radiologic studies reveal a surprising similarity in the incidence between postepileptic and postmetrazol-insulin vertebral fractures. Table 1 illustrates their data.

TABLE 1

"Idiopathic" Epilepsy Group

Number examined	72
Percentage with compression deformity	34.2
Percentage of males	46.8
Percentage of females	24.5

Metrazol—Insulin Group

Number examined	86
Percentage with compression deformity	31.4
Percentage of males	50.0
Percentage of females	18.0

Midthoracic fractures also occur occasionally in tetanus.⁵

The compression force which a vertebra will withstand is probably a major factor in its resistance to fracture. The pressure necessary to crush the fifth thoracic vertebra was determined by the following method: The body of the vertebra was removed with an electric saw, fleshed and placed in formalin. The intervertebral cartilage was separated, the vertebra dried and placed under an electrically controlled press capable of measuring pressure variations within five pounds. The load was applied to the flat surfaces of the vertebra. As the pressure increased to 250-275 pounds, a cracking sound occurred. At this point, the bone appeared flattened and the periphery of the superior and inferior borders of the ventral margin was separated from the body (Fig. 1). As the load gradually increased to 750-800 pounds, the vertebra cracked apart transversely. The trabeculae also appeared crushed, although the two parts retained their shape.

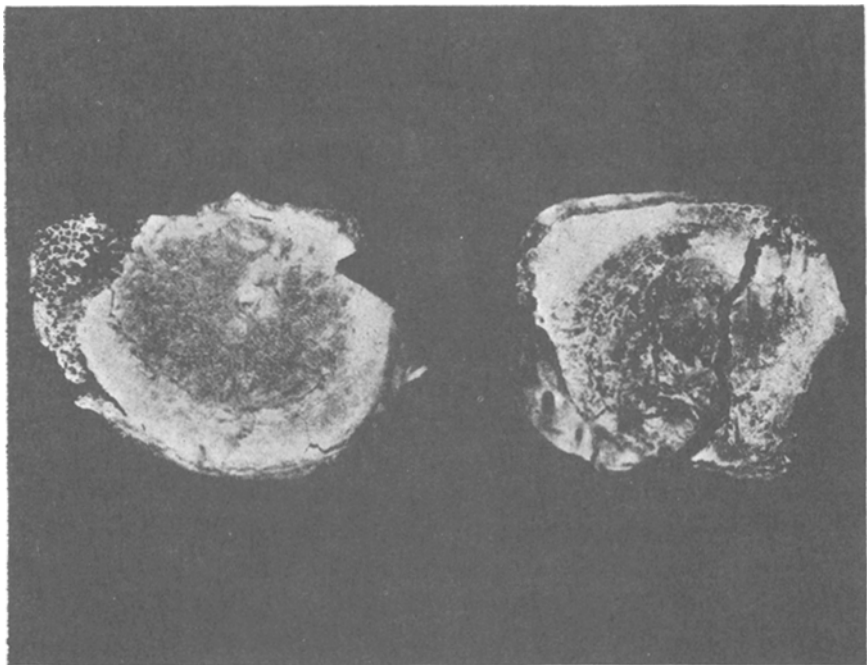
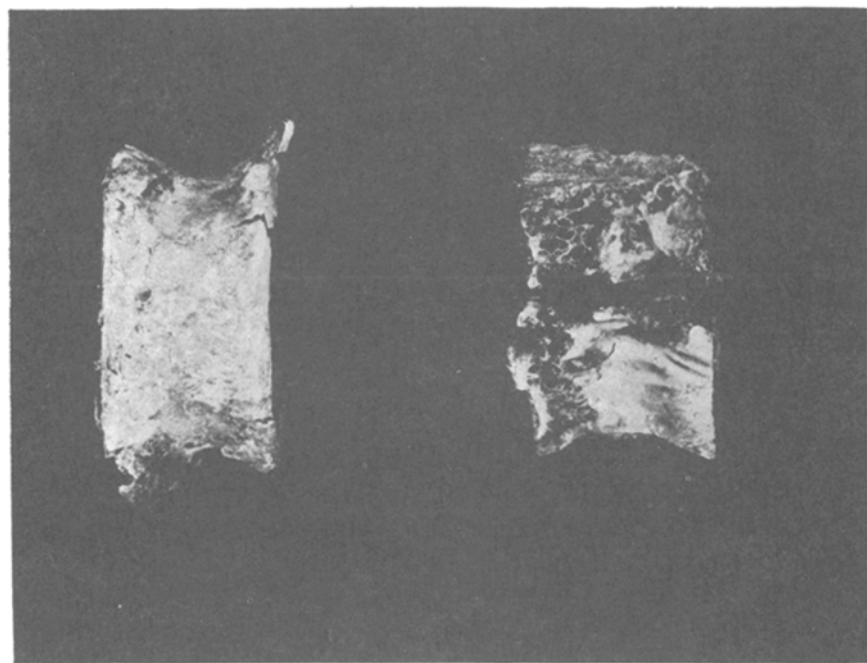


Fig. 1. The appearance of vertebrae following experimental mechanical compression.
 A—Ventral view. The upper vertebrae was subjected to 250 pounds,
 the lower to 780 pounds pressure
 B—Superior aspect of the same vertebrae

The results of two experiments are charted in Table 2.

TABLE 2

Patient	Sex	Age	Flattening load	Crushing load
1. A. R.	M.	28	250 pounds	750 pounds
2. J. W.	M.	56	275 pounds	780 pounds

Although the method of preparation of the vertebrae and the circumstances of this experiment are subject to criticism, one fact appears significant, namely: The fifth thoracic vertebra when longitudinally compressed will be crushed at the peripheral portion of its superior and inferior ventral margins by a force approximately one-third that required to crush the body.

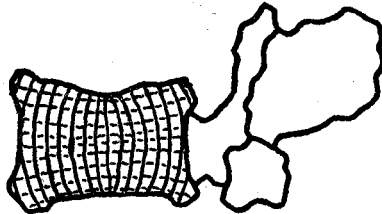


Fig. 2. Vertebral body in cross-section, showing the bony structure.
(Transcribed from Morris' *Human Anatomy*, p. 105)

The unique construction of the vertebral body³ may be partly responsible for the wedge-shaped nature of the postmetrazol fracture. It is evident from Fig. 2 that the trajectories described by the trabeculae of the vertebrae are prone to disruption at their centers when the vertebra is subjected to a longitudinally compressing flexion force. The midpoint of the ventral border of the vertebral body in its transverse axis is therefore its weakest portion.

The study of the metrazol convulsion by Strauss, et al.,⁴ utilizing high speed motion pictures, reveals the fact that the seizure is tripartite: a 10-second clonic stage, a 10-second tonic stage and a second clonic stage of 30 seconds duration. This study probably indicates an important factor in the postmetrazol fracture, which may best be illustrated by analogy with the engineering aspect of the problem of bridge construction. In computing the strain tolerated by a bridge, allowance must be made not only for the total load of 100 soldiers but also for the increased force incident to the

recurrent impacts of their marching. The compression load placed upon a vertebra likewise seems of less importance than the recurrent impacts of the clonic-tonic-clonic convulsion.

The fate of the intervertebral disk in the pathogenesis of the postmetrazol fracture has received scant attention. Tureen and Key⁷ suggested that, due to direct longitudinal pressure on the vertebral bodies, there is relatively slight tendency to wedging but the intervertebral disk may be forced into the bodies of the vertebrae. Of 15 cases of fracture of the thoracic vertebral bodies (T ix to T xii) from external trauma, Olin⁸ observed compression of the intervertebral disk in 86 per cent. The commonest result of this complication was the invasion of the prolapsed nucleus pulposus of the disk into the spongiosa of the vertebral body forming a Schmorl's node. Rathmell⁹ confirmed this finding histologically in a case of postmetrazol vertebral fracture. Posterior protrusion of the intervertebral disk into the spinal canal following this type of fracture, although not yet reported, should be anticipated.

The necessity for maintaining spinal hyperextension is therefore emphasized by these observations. Counterpressure against the hyperextended spine probably offsets a compression force between 250 and 750 pounds. This may prevent crush fracture of the vertebral body, fracture of the periphery of the superior and inferior ventral margins, or injury to the intervertebral disk. It is probable that pressures of about 250 pounds are related to the group II type, and pressures of about 750 pounds to the group IV type of postmetrazol fractures as classified by Rathmell.⁹

At the Norristown State Hospital, the author has utilized a simple but efficient means of maintaining spinal hyperextension. The patient is placed upon a flat table with hyperextension increased by a small pillow under the midthoracic spine, and counterpressure exerted by assistants against the chin, shoulders, hips and knees. By this method, it has been possible to reduce the incidence of vertebral fractures to 8 per cent in 37 consecutive cases. Graves and Pignataro,¹⁰ using a similar means of restraint, have reported the same incidence in 187 cases. This is considerably less than the reported percentages of: 20,¹¹ 43,¹² 47¹³ and 50.⁹

SUMMARY AND CONCLUSIONS

1. Several related factors probably explain the high incidence of postmetrazol vertebral fractures.
2. The relation of mechanical factors to the production of the fracture is confirmed.
3. The peculiar anatomical nature of the thoracic spine with its limitation in extension may explain in part the mechanical advantage of the spinal flexor muscles.
4. The postmetrazol fracture of the thoracic vertebral body is probably as frequent as that following epileptic convulsions.
5. It is found by direct measurement that the fifth thoracic vertebra, when longitudinally compressed, will be crushed at the periphery of its superior and inferior ventral margins by a force approximately one-third of that required to crush the body.
6. The unique construction of its trabeculae may predispose the vertebral body to a wedge-shaped deformity.
7. The compression load placed upon a vertebra seems of less importance than the recurrent impacts of the clonic-tonic-clonic convulsion.
8. Compression of the intervertebral disks following fracture of the thoracic vertebral body may occur in about 86 per cent of cases. Invasion of the prolapsed nucleus pulposus of the disk into the spongiosa of the vertebral body, resulting in the formation of a Schmorl's node, has been demonstrated.
9. The necessity for maintaining spinal hyperextension in preventing postmetrazol vertebral fractures and intervertebral disk complications is emphasized. Its use may result in a reduction of fractures from 50 to 8 per cent.

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BIBLIOGRAPHY

1. Bellinger, C. H.: Comments on metrazol treatment (discussion). *PSYCHIAT. QUART.*, 13:569, July, 1939.
2. Coombs, H. C., Searle, D. S., and Pike, F. H.: The changes of the concentration of inorganic calcium and phosphorous during convulsions of experimental origin in cats before and after thyroparathyroidectomy with and without bromide therapy. *Am. Jour. Psychiat.*, 13:761, 1934.
3. Morris: *Human Anatomy*. Ninth edition, 1933. Blakiston and Co., Philadelphia, Pa., pp. 294, 300, 301, 305.
4. Reed, G. E., and Davis, T. E.: Compression fractures of the vertebral bodies following induced and "idiopathic" convulsions. *Can. Med. Ass. Jour.*, 42:38, January, 1940.
5. Roberg, D. T., Jr.: Spinal deformity following tetanus and its relation to juvenile psychosis. *Jour. Bone and Joint Surg.*, 19:603, 1937.
6. Strauss, H., Landis, C., and Hunt, W.: The metrazol seizure and its significance. *Jour. Nerv. and Ment. Dis.*, 90:439, 1939.
7. Tureen, L. J., and Key, J. A.: Fracture of the spine during metrazol treatment. *Weekly Bull., St. Louis Med. Soc.*, 34:61, 1939.
8. Olin, H. A.: The intervertebral disc involvement in vertebral fractures and in spinal pathology. *Am. Jour. Roentgen and Rad. Therapy*, 42:435, 1939.
9. Rathmell, T. K.: The mechanism of postmetrazol gibbus. *Penn. Med. Jour.* (In press.)
10. Graves, C. C., and Pignataro, F. B.: Injuries sustained during the course of metrazol shock therapy. *PSYCHIAT. QUART.*, 14:128, January, 1940.
11. Palmer, H. A.: Vertebral fractures complicating convulsion therapy. *Lancet*, 2:181, July 22, 1939.
12. Polatin, P., Friedman, M. M., Harris, M. M., and Horwitz, W. A.: Vertebral fractures produced by metrazol induced convulsions. *J. A. M. A.*, 112:1684, 1939.
13. Bennet, B. T., and Fitzpatrick, C. P.: Fractures of the spine complicating metrazol therapy. *J. A. M. A.*, 112:2240, 1939.