FRACTURES OF DORSAL VERTEBRAE IN EPILEPSY AND CONVULSION THERAPY.

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[Received February 2, 1946.]

INTRODUCTION.

FRACTURES of the dorsal vertebrae remain one of the chief complications of convulsion therapy; and the problems of their mechanism and significance have not yet been completely solved. The present investigation has been developed with the intention of throwing fresh light on these matters by a comparative study of similar lesions in 27 patients suffering from the convulsions of idiopathic epilepsy, 21 of whom have also been treated by electrical convulsion therapy. A control group of 30 patients suffering from non-convulsive psychoses has also been examined, and in all cases the spine has been examined both clinically and radiologically, attention being focused, for the sake of clarity, on the dorsal region only.

REVIEW OF THE LITERATURE.

Ever since the first reports, by Wespi (1938) and Stalker (1938), of vertebral fractures produced in convulsion therapy, many workers have compared these lesions with those which occur during the convulsions of tetanus (Pearson and Ostrum, 1940; Kraus and Viersma, 1940; Worthing and Kalinowsky, 1942, etc.). The theories put forward by Lehndorff (1907), Erlacher (1920) and Roberg (1937) as to the muscle forces which produce the fractures in tetanic convulsions have been generally accepted as applying equally well to the vertebral changes of convulsion therapy.

Pearson and Ostrum (1940) have pointed out that similar lesions also follow the convulsions of insulin coma treatment—out of three patients whom they examined, one had a typical wedge-shaped fracture of the fifth dorsal vertebra.

It would therefore be reasonable to expect that similar fractures of the dorsal vertebrae should also be found in patients who suffer from the major convulsions of epilepsy, but a review of the literature on this subject reveals a strange confusion of opinion.

Kraus and Viersma (1940) X-rayed the spines of 21 epileptics, and found one case with a definite compression fracture of D11, and another case with doubtful lesions of D6 and D7 accompanied by slight osteoporosis.

Pearson and Ostrum (1940) examined three patients, who had recovered from status epilepticus, and found in one case wedge-shaped fractures of D12 and L1. They also examined the spine of a patient suffering from meningovascular

syphilis, who had had convulsions for years. X-ray revealed a compression fracture of L3.

Ziskind and Somerfeld-Ziskind (1939) have described spinal changes in three epileptics, and they too found fractures only in the lower dorsal and lumbar regions—D10, 11, 12, and L2 and 3.

Schatz and Konwaler (1941) found no fractures in the dorsal spines of epileptics whom they examined.

Worthing and Kalinowsky (1942) X-rayed 42 epileptics, and found fractured vertebrae in only two cases. One had a fracture of L1, and the other had a fracture of the spinous process of C6. They stated that " no reports of fractures of the mid-dorsal region in epilepsy are given in the literature. . . . The few instances of vertebral fractures reported in epilepsy have the usual localization of compression fractures of traumatic origin. This is easily understood, since in epileptics an occasional trauma from falls can never be excluded. This supposition and the different localization shows that fractures in epilepsy are of quite a different character, nor does it seem that they are caused by muscular contractions during the convulsions as is the case in metrazol convulsions. We feel that we have sufficient evidence for the conclusion that in epileptic seizures fractures of the same type as in convulsion therapy do not occur."

Reed and Dancey (1940) made lateral X-ray studies of the dorsal region of the spine in a group of 72 epileptics. They reported an incidence of compression deformities in the mid-dorsal region of 34·2 per cent. Unfortunately, they gave no details of the individual lesions or their actual distribution among the dorsal vertebrae, but they implied that both types of lesion and distribution were the same as in metrazol-treated patients whom they examined during their investigation; and they contrasted the localization of these fractures with those produced in the lower dorsal and upper lumbar regions by external trauma.

Cook and Sands (1941) took lateral radiograms of the spine of 134 idiopathic epileptics and 135 control cases. They found 14 cases of compression deformity in the first group and only 2 in the control series. Their epileptics included 72 males and 62 females. The incidence of fractures among the males was 14 per cent. and among the females only 4 per cent., the incidence for the whole group being 10·4 per cent. They described the type of compression fracture in these epileptics as " so like that found after induced convulsions that a similar mechanism can justifiably be assumed." They reported, however, that the distribution of the fractures was rather lower than among metrazol patients; of 18 fractured vertebrae there were 4 lesions in D7, 1 in D8, 2 in D10, 3 in D12, and the rest were in the lumbar region. They mentioned that in addition to the compression fractures, " knorpelknötchen " were found in one case, and deformity of the superior surface of a vertebral body with formation of a " cartilaginous pearl " in another. Also " non-traumatic extensive deformity of several vertebrae, apparently due to occupational or constitutional causes, appeared in four cases." They gave no details of any of these lesions, and there is thus no possibility of comparing them with those which other authorities have classified as " minor fractures " or " infractions."

It is also of interest to note that they did not describe the occurrence of such abnormalities in the control group of 135 cases.

Moore, Winkelman, and Solis-Cohen (1941) X-rayed the spines of 12 epileptics who had all suffered for years from severe *grand mal* fits. They reported an incidence of definite compression fractures of dorsal vertebrae in 50 per cent. of the cases. The fractures were located as follows:

	Dorsal vertebra.				
	8.	9.	10.	11.	12.
Number of fractures . . .	3	3	1	2	2

Three of the patients had major fractures and the others had minor fractures, of each of which they gave reasonably detailed descriptions.

Barrett, Funkhouser and Barker (1942) have also described fractures of the dorsal vertebrae in epilepsy. They took lateral X-ray films of 20 epileptics, and they found compression fractures in 45 per cent. of cases. A similar number of control cases showed no fractures. They reported that the vertebral lesions in the epileptics "are indistinguishable from those occurring with metrazol and electro-shock both as to type of fracture and localization." They found the distribution of fractures among the dorsal vertebrae to be as follows:

	Dorsal vertebra.									
	3.	4.	5.	6.	7.	8.	9.	10.	11.	12.
Number of fractures . .	2	2	2	2	4	2	2	1	1	1

And they concluded that "the deduction of earlier workers that vertebral fractures in epilepsy are due to the trauma of frequent falls during seizures is not substantiated by these findings."

Significance.

There has been no final decision so far as to the significance of vertebral fractures produced by convulsion therapy as regards the future health of the patient, but the literature contains no account of any serious sequelae.

Worthing and Kalinowsky (1942) followed up eight severe cases over a period of two years and reported that Kummel's disease did not occur, the motility of the spine was not affected, and there was no involvement of the cord.

Roberg (1937), referring to the comparable lesions of tetanus, has reported that a certain amount of healing sometimes takes place, especially in a kyphosis which is treated by a plaster jacket or surgical brace. He has reported no cases of definite involvement of the cord, though one patient, a nine-year-old girl, with fractures of the second and third lumbar vertebrae, retained the gibbus three years later, as well as a "hypererflexia of one leg." Many of the cases were followed up for years and no serious complications have been recorded.

Jessner and Ryan (1942) have recorded their opinion that "few cases are reported to be seriously inconvenienced through vertebral fractures." They reported that the usual sequela is a slight gibbus.

Cook (1944) pointed out that the discovery of vertebral fractures in so many cases was at first very alarming, and it placed the future of convulsion therapy in serious jeopardy, but " fortunately they are not followed by serious sequelae and have proved to be far less important than was at first feared." He referred to the question of the comparison of these fractures with those occurring in epileptics, but was of the opinion that " no absolute conclusion can be drawn from the absence of referable symptoms in long-standing epilepsy, but it is reassuring that neither Kummel's disease nor spinal involvement occur as a result of epileptic fits." After reviewing the literature he stated: " In spite of the many thousands of convulsion courses given no serious permanent disability has been reported."

Description of Present Investigation.

Twenty-seven male patients were chosen who had all suffered at one time or another from frequent *grand mal* epileptic fits. They were carefully examined clinically to see whether they showed any neurological abnormalities which might be attributable to vertebral lesions causing pressure on the spinal cord.

The dorsal region of the spine was then carefully radiographed in each case, and examined for the presence of vertebral fractures. The latter were classified as major fractures and minor fractures. Subsequent descriptions will show how these were differentiated, but it may be briefly stated here that major fractures were those in which there was a definite compression deformity of the vertebral bodies, usually wedge-shaped, and accompanied in many cases by fragmentation and sclerosis of the articulating surfaces; minor fractures were those in which there were lesser degrees of deformity, but in which there was evidence of trabecular buckling, accompanied by a variable number of other abnormalities, e.g. flattening of the articulating surfaces, narrowing of the discs, chip fractures of the antero-superior edge of the vertebral bodies, spur formations and sclerosis of articulating surfaces, etc.

A control group of 30 patients, from the same wards, who were suffering from various chronic non-convulsive psychoses, was subjected to the same clinical and radiological investigation of the dorsal spine.

After carrying out a full radiological investigation of the dorsal spines of the epileptic patients, twenty-one of them were given electrical convulsion therapy, as described in another paper (Caplan, 1945). No attempt was made to fix the patients in hyperextension during the convulsions; they were treated in the usual way, lying on their backs on ordinary beds, and were not restrained in any way either manually or by bed-clothes. After each patient had been given an average of twelve electrical convulsions he was again X-rayed, and the films were carefully compared with the initial ones. During the course of the electrical treatment it was noticed that each patient had his own characteristic set of movements for the convulsion, and that some patients had strong flexion spasms which were absent in most cases. Also the strength of the muscular contractions appeared to vary from one to another, and even the degree of movement of the limbs and trunk during the clonic stage seemed to be a peculiarity, characteristic for each individual. It was felt that these

mechanical variables might have some bearing on the question of individual susceptibility to vertebral fractures, and an attempt was made to record them for each convulsion and investigate their possible correlation with the existence of spinal lesions. The ideal way of doing this would be to employ the method of Strauss, Landis and Hunt (1939), and use high-speed cinematograph cameras and electro-myographic apparatus, but since these were not available a rather rough-and-ready clinical method was worked out.

The mechanical variables in the convulsion were divided into three: posture, strength of muscular contractions, and clonic excursions. In each of these an arbitrary set of standards was devised, and each individual convulsion was then very carefully observed and assessed according to its behaviour in relation to these.

(*a*) POSTURE.—During a convulsion the patient does not, of course, remain in one position, but is constantly moving. There is, however, in most cases a tonically held posture on which the other movements appear to be superimposed, and this is especially so in the case of the trunk. Interest was for obvious reasons directed solely to the posture of the dorsal spine, and this was in each case investigated not only visually, but also by palpation. Three main types of posture were noted:

i. *Extension:* this varied from a slight hollowing of the back to a marked opisthotonos.

ii. *Straight:* here the back was neither flexed nor extended, although head and neck were often flexed.

iii. *Flexion:* this was divided into mild and severe flexion, and was usually accompanied by flexion of head and neck.

(*b*) STRENGTH OF MUSCULAR CONTRACTIONS.—This was ascertained by palpation during the convulsion, and was divided into strong, moderate, and weak. Standards were, of course, arbitrary, but it was found that after some practice a fairly consistent judgment was possible.

(*c*) CLONIC EXCURSIONS.—In some cases there was hardly any movement at all during the seizure, little more than a coarse tremor; such cases were recorded as *Clonus* 0.

In other cases there occurred movements of great amplitude, and these were classified as *Clonus* 2.

Intermediate degrees of movement were recorded as *Clonus* 1.

Here again, despite the subjective nature of the judgments made a satisfactory consistency was soon attained, and this was shown among other things by the fact that although the records of the case were not inspected until afterwards, there was rarely any deviation from previous markings unless there had definitely been a change from the patient's usual behaviour during his convulsion.

RESULTS.

(1) EPILEPTICS.

Radiological study of the 27 epileptics showed that nine of them were suffering from fractures of dorsal vertebrae—an incidence of 33 per cent. Four of this number had only one fracture each, and the rest had multiple fractures

varying from two to four per patient. Four patients had major fractures, and five had minor fractures only. Three of the former had minor fractures in addition.

Table I shows the distribution of the fractures among the dorsal vertebrae. It can be seen that the peak incidence is at D6, 7, 8 and 9; and that lesions of D5 and D10 were also found.

TABLE I.—*Distribution of Vertebral Fractures in Epileptics.*

Number of Cases.	Fractures. Type.	Number.	Distribution in dorsal vertebrae. 1.	2.	3.	4.	5.	6.	7.	8.	9.	10.	11.	12.
4	Major	6	0	0	0	0	0	2	1	1	2	0	0	0
8	Minor	10	0	0	0	0	1	2	2	3	1	1	0	0
9	Total	16	0	0	0	0	1	4	3	4	3	1	0	0

Table II shows the details of the lesions in the different patients, together with their present ages and also the age of onset of epilepsy in each case. This table shows quite clearly that neither length of history nor age of onset has

TABLE II.—*Vertebral Fractures in 27 Epileptics.*

	Case No. 99.	100.	101.	102.	103.	104	105.	106.	107.
Present age	42	53	38	45	37	28	35	30	26
Age of onset of epilepsy	14	15	22	20	12	16	15	17	20
Major fractures	D6, 8, 9	0	D7	0	0	0	0	0	0
Minor fractures	D7	D8	0	0	0	0	D8, 9	0	0

	Case No. 108.	109.	110.	111.	112.	113.	114.	115.	116.
Present age	22	27	50	45	48	32	44	33	37
Age of onset of epilepsy	20	14	23	11	14	21	40	18	23
Major fractures	0	0	0	D9	0	0	0	D6	0
Minor fractures	0	0	0	D10	D6	D7, 8	0	D5	0

	Case No. 117.	118.	119.	120.	121.	122.	123.	124.	125.
Present age	41	45	24	45	49	24	22	35	37
Age of onset of epilepsy	19	28	9	25	35	9	13	14	12
Major fractures	0	0	0	0	0	0	0	0	0
Minor fractures	D6	0	0	0	0	0	0	0	0

any influence on the incidence of fractures. In no case did clinical examination show any neurological complication pointing to cord involvement, but in one patient (Case No. 99) there was some degree of kyphosis at the site of the fractures.

The X-ray pictures showed no trace of osteoporosis in any case, nor was there any caries or other bone disease, though a number of patients were found to be suffering from various mild postural defects which were not related in any way to the incidence of fractures. One patient (No. 114) was suffering from an old adolescent kyphosis affecting D7, 9, 10 and 11. The following are the details of the individual lesions in each case:-

https://doi.org/10.1192/bjp.92.389.766 Published online by Cambridge University Press

CASE NO. 99.—*Major Fractures—D6, 8 and 9.*

The sixth dorsal vertebra showed flattening and shortening of the body with elongation transversely. There was irregularity and sclerosis of both articulating surfaces. There was indentation of the anterior surface of the body, which was wedge-shaped in appearance.

The body of the eighth dorsal vertebra was more acutely wedge-shaped. Its upper articulating surface was very irregular and had the appearance of healed and sclerosed fragmentation. There was narrowing of the disc between this vertebra and the seventh dorsal.

The ninth dorsal vertebra had a body which was a little less wedge-shaped. Its upper articulating surface was the one chiefly involved, and it showed irregularity and sclerosis.

Minor Fracture—D7.

The body of the seventh dorsal vertebra showed a mild degree of both anterior and lateral wedging. The upper articulating surface had lost its normal concavity—it was flat and sclerosed. There was a little shortening of the body vertically, and some degree of notching of its anterior surface.

The other vertebrae appeared normal. There was some kyphoscoliosis with its apex at the level of D9.

CASE NO. 100.—*Minor Fracture—D8.*

The eighth dorsal vertebra showed a small amount of flattening of the body and shortening in its vertical axis. This was more marked anteriorly, giving a mild degree of wedging. The superior articulating surface was less concave than normal, and there was a zone of increased density just at the margin. The other vertebrae were normal.

CASE NO. 101.—*Major Fracture*—D7.

The seventh dorsal vertebra showed irregularity and sclerosis of its upper articulating surface. There was an irregularly shaped shadow superimposed on the upper part of the body, which looked like a piece of excess callus from an old healed fragmentation of the upper articulating surface. The height of the body was materially diminished and there was marked wedging. There was an indentation of the anterior surface. There was some increase in density in the region of the lower articulating surface, but its contour was quite normal.

The other vertebrae showed no abnormality.

CASE NO. 105.—*Minor Fractures—D8 and 9.*

The lower anterior end of the body of the eighth dorsal vertebra was compressed and sclerosed. There was a spur formation, and the edge was roughened and thickened by old arthritis. The upper anterior articulating tip of the ninth dorsal vertebra was changed in a similar manner. The anterior part of the upper articulating surface was flattened and sclerosed, and there was some hypertrophic arthritis surrounding it.

The other vertebrae were quite normal, there was no kyphosis or scoliosis, and there was no trace of arthritis elsewhere in the spine.

CASE NO. 111.—*Major Fracture—D9.*

The body of the ninth dorsal vertebra was flattened and its height was shortened. The anterior surface showed a semi-circular indentation. The body as a whole showed a mild degree of wedging. The anterior part of the upper articulating surface was flattened and sclerosed. There was some arthritis between this portion and the vertebra above. The lower articulating surface was irregular in shape as though the result of healed fragmentation, and there was a thick zone of increased density at this lower margin.

Minor Fracture—D10.

The anterior portion of the superior articulating surface of the tenth dorsal vertebra was markedly flattened and depressed. It showed dense sclerosis, and

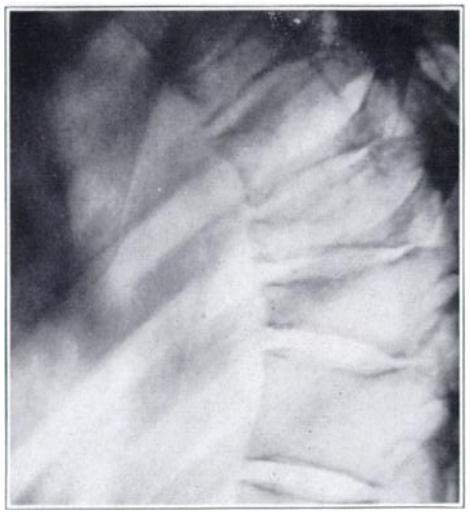

FIG. 1.—X-ray of epileptic, Case No. 99. Major fractures—D6, 8, 9. Minor fracture—D7.

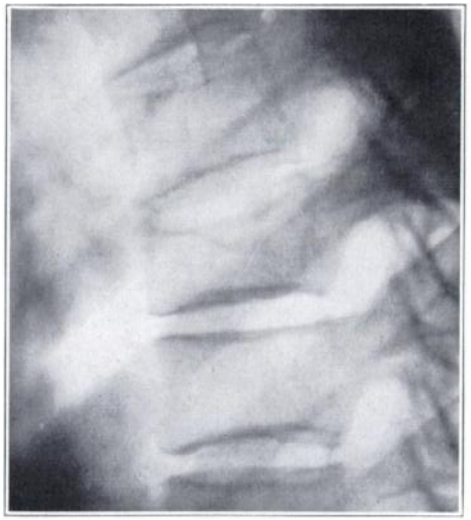

FIG. 2.—X-ray of epileptic, Case No. 101. Major fracture—D7.

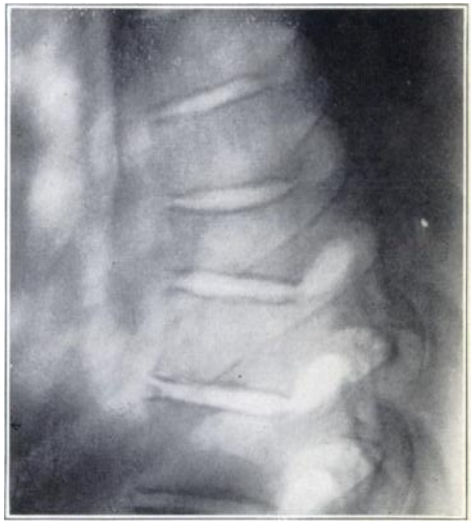

FIG. 3.—X-ray of epileptic, Case No. 115. Major fracture—D6. Minor fracture—D5.

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there was a zone of hypertrophic arthritis between it and the adjoining portion of the vertebra above. There was narrowing of the intervertebral disc between the ninth and tenth dorsal vertebrae. The anterior surface of the body of the tenth dorsal vertebra showed a marked indentation, but there was no wedging of the body itself.

The other vertebrae appeared normal.

CASE NO. 112.—*Minor Fracture—D6.*

The body of the sixth dorsal vertebra showed some flattening and shortening in its height. There was a mild degree of wedge-shaped deformity present. The upper articulating surface showed some loss of the normal concavity, and there was a slight reduction in the thickness of the intervertebral disc.

The other vertebrae showed no abnormality, though there was a slight degree of scoliosis with its peak at D3.

CASE NO. 113.—*Minor Fractures—D7 and* 8.

The body of the seventh dorsal vertebra showed a mild degree of wedging, with some shortening and flattening. There was loss of the normal concavity of both the upper and the lower articulating surfaces, and a narrow zone of increased density in both margins.

The body of the eighth dorsal vertebra showed similar slight wedging and flattening. Its upper articulating surface was irregular in outline and sclerosed.

The other vertebrae in the dorsal spine showed no abnormality.

CASE NO. 115.—*Major Fracture—D6.*

The body of the sixth dorsal vertebra showed flattening and vertical shortening. There was a gross degree of wedging, and the anterior surface showed an indentation due to crushing. The lower articulating surface was flattened and somewhat irregular in outline, and showed some sclerosis.

Minor Fracture—D5.

There was some flattening of the body of the fifth dorsal vertebra, and it showed a mild degree of anterior wedging. The upper articulating surface showed loss of its normal concavity, and there was a reduction in thickness of the intervertebral disc, between it and the vertebra above.

There was no kyphosis in the dorsal spine, and the other vertebrae appeared normal.

CASE NO. 117.—*Minor Fracture—D6.*

The body of the sixth dorsal vertebra showed some flattening and vertical shortening. There was a mild degree of anterior wedging. The upper articulating surface showed loss of the normal concavity, and there was narrowing of the intervertebral disc.

There was no abnormality in the other dorsal vertebrae.

From the above details it can be seen that the main changes in the X-ray appearance of these vertebral lesions in epileptics are as follows:

(1) Loss of the normal concavity of the upper articulating surfaces of the bodies of the vertebrae.

(2) Irregularities of both upper and lower articulating surfaces, giving the appearance of old healed fragmentations; and in many cases showing sclerosis and sometimes roughening.

(3) Zones of increased density at the articulating margins of the vertebrae.

(4) Narrowing of the intervertebral discs.

(5) Indentations of the anterior surfaces of the vertebral bodies.

(6) In occasional cases, spur formations at the upper or lower anterior tips of the vertebral bodies, usually accompanied by arthritic changes.

(7) Flattening of the vertebral bodies, shortening vertically, and sometimes elongation transversely.

(8) Greater or lesser degrees of anterior wedging of the bodies of the vertebrae, giving the characteristic picture of compression fractures.

(9) The fractures seemed to be old ones, and there was no evidence of recency in any of the lesions.

(10) There were no gross associated spinal changes, no evidence of Kummel's disease, and no sign of displacement of the vertebral bodies to produce danger of cord involvement.

(11) There was no evidence of decalcification or rarefaction in any of the vertebrae to suggest interference with calcium or phosphorus metabolism.

(2) CONTROL GROUP.

No such lesions as the above were to be found in any of the control cases. Three of the patients had a mild degree of kyphosis, probably of postural origin, but the vertebral bodies were normal in contour, and there were no changes in any way suggestive of fractures.

(3) EFFECT OF ELECTRICAL CONVULSION THERAPY ON EPILEPTICS.

Twenty-one of the above epileptic patients were given courses of electrical convulsion treatment (Caplan, 1945). They included among their number all the cases in which vertebral fractures had been discovered.

The electrical current used to induce the convulsions was kept down to about the threshold value in order to reduce the initial flexion effect of stimulation of the cortical cells when the current was flowing. As has previously been described (Caplan, 1945), the induced convulsion resembled closely the spontaneous fit which was characteristic for each individual. After an average of 12 convulsions the spines were again examined radiologically.

In no case was there any change to be seen from the condition of the vertebrae which had existed before treatment was commenced.

No additional fractures were found, and the old lesions were not affected in any way by the electrically-induced convulsions.

CONVULSION DETAILS.

Table III gives the details of seven successive convulsions in each case, and also of the incidence of vertebral fractures.

It can be seen that there is a definite correlation between the posture during the convulsion and the presence or absence of fractured vertebrae.

In 7 out of 9 cases which had vertebral lesions, the posture during some or all of the convulsions was one of flexion.

This posture did not occur in any patient who had no fractures.

Of the other two fracture cases, one showed postures which were occasionally straight and sometimes extension.

Of the eleven patients whose spines were normal only three showed occasional straight postures during convulsions, and the rest were entirely confined to extension.

Only one patient with vertebral fractures had his convulsions in extension during the period of observation.

These observations afford strong evidence that susceptibility to fracture in these patients is associated with individual peculiarities of convulsion pattern, and that the majority of fractures occur among those patients whose fits usually take place in an attitude of flexion, or in certain cases in a straight posture. They also show that the majority of patients usually have fits in an attitude of tonic extension of the spine, and that this is associated with their freedom from vertebral fractures.

When attention was directed to the details of the individual convulsions, it was discovered that although the general pattern was characteristic for each person, there were variations from day to day in the patient's convulsion picture. This is shown very well in Table III ; Case No. 117 is a good example. Successive fits showed the following postures : Ext. ; Ext. ; Strt. ; Ext. ; Strt. ; Flex. ; Strt. This was also found to hold for the spontaneous fits. As has been described elsewhere (Caplan, 1945*a*), some patients developed the habit of having a spontaneous fit whilst in bed waiting for their E.C.T. It was thus possible to record in a few cases the details of these fits and compare them directly with the electrically induced convulsions. Sufficient material has not so far been forthcoming to enable any sort of statistical table to be drawn up, but the general clinical impression has been that spontaneous fits resemble induced convulsions in showing individual variations superimposed on a general pattern characteristic for each patient.

The Table also shows that day-to-day variations were more marked among the patients who had the fractures. Out of 7 cases where flexion was found, only 3 were consistent in this posture. This can be compared to the 16 cases of extension, among whom 9 were consistent. There were 8 cases in which straight postures were found, and all of these showed variations from one convulsion to the next.

As for the other variable factors in the convulsions, Table III shows quite clearly that neither the degree of clonus nor the strength of muscular contractions has any significance as regards the incidence of fractures. Patients who had consistently strong muscular contractions, and extensive clonus, were fracture free ; while other patients who did have vertebral fractures had weak or moderate muscular contractions, and mild or intermediate degrees of clonus. Apart from this lack of correlation with susceptibility to fractures, it is of interest to note that these factors, too, showed a tendency to variation from one fit to the next, although this variation was less noticeable than it had been in the case of posture.

Discussion.

The results of the present investigation allow some light to be thrown on the various unsolved problems of vertebral fractures.

(1) *Incidence of Vertebral Fractures in Epileptics.*

Our results regarding lesions of the dorsal spine in idiopathic epilepsy confirm the findings of Reed and Dancey (1940), who reported an incidence of

TABLE III.—*Correlation of Convulsion Details and Incidence of Fractures in Epileptics.*

		Case No. 99.	Case No. 100.	Case No. 101.	Case No. 102.
Successive convulsions	1	Flex.; Mod.; C.1	Flex.; Mod.; C.0	Flex.; Stng.; C.1	Ext.; Mod.; C.2
	2	Flex.; Mod.; C.1	Flex.; Mod.; C.0	Flex.; Stng.; C.1	Ext.; Mod.; C.0
	3	Flex.; Mod.; C.1	Flex.; Stng.; C.0	Strt.; Mod.; C.1	Ext.; Mod.; C.0
	4	Flex.; Mod.; C.0	Flex.; Stng.; C.0	Flex.; Stng.; C.1	Ext.; Mod.; C.1
	5	Flex.; Mod.; C.1	Flex.; Mod.; C.0	Flex.; Stng.; C.1	Ext.; Mod.; C.0
	6	Flex.; Mod.; C.0	Flex.; Stng.; C.0	Strt.; Mod.; C.1	Ext.; Mod.; C.1
	7	Flex.; Mod.; C.1	Flex. Mod.; C.0	Flex.; Mod.; C.1	Ext.; Mod.; C.1
Vertebral fractures	Major	D6, 8, 9	0	D7	0
	Minor	D7	D8	0	0

		Case No. 103.	Case No. 104.	Case No. 105.	Case No. 106.
Successive convulsions	1	Ext.; Mod.; C.1	Ext.; Stng.; C.1	Ext.; Mod.; C.0	Ext.; Mod.; C.2
	2	Ext.; Mod.; C.0	Ext.; Stng.; C.1	Strt.; Stng.; C.1	Ext.; Stng.; C.2
	3	Ext.; Stng.; C.0	Ext.; Stng.; C.1	Strt.; Stng.; C.0	Ext.; Stng.; C.2
	4	Ext.; Stng.; C.0	Ext.; Stng.; C.1	Flex.; Mod.; C.2	Ext.; Stng.; C.1
	5	Ext.; Stng.; C.1	Ext.; Mod.; C.1	Flex.; Mod.; C.1	Ext.; Mod.; C.2
	6	Ext.; Stng.; C.0	Ext.; Mod.; C.1	Flex.; Stng.; C.1	Ext.; Stng.; C.2
	7	Ext.; Mod.; C.0	Ext.; Stng.; C.2	Flex.; Stng.; C.2	Ext.; Mod.; C.2
Vertebral fractures	Major	0	0	0	0
	Minor	0	0	D8, 9	0

		Case No. 107.	Case No. 109.	Case No. 110.	Case No. 111.
Successive convulsions	1	Ext.; Stng.; C.2	Ext.; Mod.; C.2	Ext.; Stng.; C.2	Ext.; Stng.; C.2
	2	Ext.; Stng.; C.2	Ext.; Mod.; C.2	Ext.; Stng.; C.2	Ext.; Stng.; C.2
	3	Ext.; Stng.; C.2	Ext.; Mod.; C.2	Strt.; Stng.; C.2	Flex.; Stng.; C.1
	4	Ext.; Stng.; C.2	Ext.; Mod.; C.2	Ext.; Stng.; C.2	Flex.; Stng.; C.2
	5	Ext.; Stng.; C.2	Ext.; Mod.; C.2	Strt.; Stng.; C.2	Flex.; Stng.; C.2
	6	Ext.; Stng.; C.1	Ext.; Mod.; C.2	Strt.; Stng.; C.2	Strt.; Stng.; C.2
	7	Ext.; Stng.; C.2	Ext.; Mod.; C.2	Ext.; Stng.; C.2	Strt.; Stng.; C.1
Vertebral fractures	Major	0	0	0	D9
	Minor	0	0	0	D10

TABLE III (*cont.*).—*Correlation of Convulsion Details and Incidence of Fractures in Epileptics.*

		Case No. 112.	Case No. 113.	Case No. 114.	Case No. 115.
Successive convulsions	1	Flex.; Stng.; C.1	Ext.; Stng.; C.1	Strt.; Wk.; C.1	Ext.; Mod.; C.1
	2	Flex.; Stng.; C.1	Strt.; Mod.; C.2	Strt.; Wk.; C.1	Ext.; Mod.; C.2
	3	Flex.; Stng.; C.1	Ext.; Stng.; C.2	Ext.; Stng.; C.1	Ext.; Mod.; C.1
	4	Flex.; Stng.; C.0	Strt.; Stng.; C.2	Strt.; Stng.; C.1	Ext.; Mod.; C.2
	5	Flex.; Stng.; C.0	Strt.; Stng.; C.2	Strt.; Stng.; C.1	Ext.; Mod.; C.1
	6	Flex.; Stng.; C.0	Strt.; Stng.; C.2	Ext.; Stng.; C.2	Ext.; Mod.; C.2
	7	Flex.; Stng.; C.1	Strt.; Stng.; C.2	Strt.; Mod.; C.1	Ext.; Mod.; C.0
Vertebral fractures	Major	0	0	0	D6
	Minor	D6	D7, 8	0	D5

		Case No. 116.	Case No. 117.	Case No. 118.	Case No. 120.
Successive convulsions	1	Ext.; Stng.; C.2	Ext.; Mod.; C.1	Ext.; Stng.; C.2	Ext.; Stng.; C.2
	2	Ext.; Stng.; C.2	Ext.; Stng.; C.1	Ext.; Stng.; C.2	Ext.; Stng.; C.2
	3	Ext.; Stng.; C.2	Strt.; Stng.; C.1	Ext.; Stng.; C.2	Strt.; Stng.; C.2
	4	Ext.; Stng.; C.2	Ext.; Stng.; C.1	Ext.; Stng.; C.2	Ext.; Stng.; C.1
	5	Ext.; Mod.; C.1	Strt.; Stng.; C.1	Ext.; Stng.; C.2	Strt.; Stng.; C.1
	6	Ext.; Mod.; C.2	Flex.; Stng.; C.2	Ext.; Stng.; C.2	Ext.; Stng.; C.1
	7	Ext.; Stng.; C.2	Strt.; Stng.; C.0	Ext.; Mod.; C.2	Ext.; Stng.; C.2
Vertebral fractures	Major	0	0	0	0
	Minor	0	D6	0	0

KEY.

Posture:
Ext.: Extension.
Strt.: Straight.
Flex.: Flexion.

Muscular power:
Stng.: Strong.
Mod.: Moderate.
Wk.: Weak.

Degree of clonus:
C.0: Mild clonus.
C.1: Intermediate clonus.
C.2: Extensive clonus.

34·2 per cent. in 72 cases. Barrett *et al.* (1942) and Moore *et al.* (1941) gave a higher incidence of 45 per cent. and 50 per cent. respectively.

Our figure is about the same as the average incidence of fractures in metrazol-treated patients, where no precautions are taken; and is just what one would expect from the fact that the seizures in epilepsy and pharmacological convulsive therapy are so similar.

Cook and Sands' (1941) figure of 10·4 per cent. of fractures in epileptics is very much lower than our own, but it has already been pointed out that these workers neglected a number of lesions which we would probably have included in our list of fractures—lesions which did not occur in their control group of non-epileptic patients. That these investigators are very conservative in their X-ray diagnosis of fractures can also be seen from the fact that they have given the incidence of compression fractures in a series of 143 patients who received metrazol treatment without restraint or other precautions as 14·7 per cent. Thus they too have given the fracture rates in metrazol treatment and epilepsy as about equal, though by neglecting what we would have called "minor fractures" they have reduced both figures by approximately 50 per cent. Another point to note is that their figures were derived from mixed material while all our patients were males. It will be remembered that in their 72 male cases the incidence was higher (14 per cent.).

(2) *Location of Fractures in Epileptics.*

The results of the present investigation are again in line with the findings of Reed and Dancey (1940) and Barrett *et al.* (1942) in localizing the vertebral fractures throughout the middle and lower dorsal spine. In this we disagree once more with Cook and Sands (1941), who found most of their fractures in the lower dorsal and in the lumbar regions of the spine. They did, however, find that D7 had the highest number of lesions of any single vertebra, and this can be compared to our peak incidence, which was localized in D6, 7, 8 and 9. It must be admitted that our own figures show a somewhat lower distribution for epileptics than for post-convulsive fractures, which have a peak incidence at D4, 5 and 6. Cook and Sands gave no explanation for this difference in location, but a reasonable theory appears to be that convulsions in epileptics often take place with the patients in an erect posture, and this tends to displace the muscle strain further down the back than in the case of those patients who are given therapeutic convulsions invariably in a horizontal position on a bed or couch. The description of Ziskind and Somerfeld-Ziskind (1939) of two cases of epileptics who had fits whilst sitting upright, and who immediately afterwards complained of pain in the back and were found to have sustained fractures of D12, and D10 and 11 respectively, makes the theory sound more plausible. The effect of the upright posture on the localization of strains on the dorsal vertebrae can also be seen in regard to the not uncommon condition of adolescent kyphosis, where the peak of vertebral changes is in the region of D8, 9 and 10 (Roberg, 1937). Also the location of vertebral fractures in tetanus fits in satisfactorily with our theory—in this disease the convulsive movements occur while the patient is lying in bed, and as would be expected, the majority of lesions are found in D5 and 6 (Roberg, 1937).

(3) *Type of Lesion in Epileptics.*

The radiological changes found in the dorsal vertebrae of the epileptics in this investigation have been very similar to those described in the literature as occurring in patients who have had convulsion therapy (e.g. Pearson and Ostrum, 1940), and they are almost identical with those which we have found in our own patients after E.C.T. The few differences between them are probably due to the fact that the lesions in the epileptics are much older, and have in many cases become involved in a greater or lesser degree of secondary change in the form of sclerosis of the injured bone and hypertrophic arthritis, etc. In both epileptics and convulsion therapy patients the lesions are confined to the vertebral bodies and the intervertebral discs, and are obviously the result of compression and crushing which is more marked anteriorly. The fact that the types of lesion are so similar is another confirmation of their similar origin.

(4) *Mechanism.*

The views expressed above regarding the incidence and distribution of fractures in the dorsal spines of epileptics are diametrically opposed to the opinions of Kraus and Viersma (1940), Pearson and Ostrum (1940), Schatz and Konwaler (1941), and Worthing and Kalinowsky (1942).

The explanation for the strange discrepancy in the different reports is obscure. It may be true, as Cook (1944) says, that this shows " all too convincingly that radiological diagnosis of vertebral fractures is by no means an exact science." But it is rather hard to believe that authorities of the standing of Worthing and Kalinowsky, who have had a lot of experience with vertebral fractures in patients after convulsion therapy, should suddenly change their diagnostic criteria when faced with the same lesions in epileptics. It seems more reasonable to assume that findings so different depend chiefly on variations in choice of material. It may be not without significance that the fracture cases in the present study occurred entirely in that group of patients whose epilepsy was so bad that they were all put down for treatment by E.C.T. If the material were available it would be interesting to compare the radiographic appearances of the spines of different types of epileptics, e.g. mental hospital in-patients, epileptic colony patients, out-patients, etc.

Whatever the explanation of the discrepancy of the results of different workers, there can be no doubt as to the findings in our case, and they allow us to take up a definite attitude to the theory put forward by Worthing and Kalinowsky (1942) as to the mechanism of production of fractures in convulsion therapy. They believed that vertebral fractures do not occur during the tonic or clonic stages of a convulsion because they are not found in epilepsy. Also that the only real difference between the epileptic fit and the metrazol and E.C.T. convulsion was in the mode of onset, and therefore the fractures after therapy were due to flexion spasms during the " first clonic phase " of metrazol and the " initial flexion phase " of E.C.T.

From our results, and in substantial agreement with Reed and Dancey (1940), Cook and Sands (1941), Moore *et al.* (1941), and Barrett *et al.* (1942),

we can say that fractures in epilepsy are *mutatis mutandis* of the same type and distribution as those resulting from convulsion therapy, and that their incidence is comparable to that resulting from metrazol therapy. The main plank on which the theory of Worthing and Kalinowsky rests thus falls to the ground, and we can now express our agreement with the views of those workers like Wespi (1938), and Friedman, Brett and Vogt (1940), who have maintained that fractures may occur at any phase of the convulsion. There can, of course, be little doubt that the acute flexion spasms of onset of the therapeutic seizure may be a potent factor in producing fractures, but the absence of such phenomena in epilepsy proves that vertebral compression can be produced also during the tonic and clonic stages of the fit.

It is recognized that the electrically-induced convulsion is less strenuous than the metrazol or epileptic fit (Cook, 1944), and this is probably the main reason why fractures are less common after E.C.T. than after metrazol or epilepsy.

Further light is thrown on this subject and further evidence obtained against the theory of Worthing and Kalinowsky by our interesting findings regarding the effect of E.C.T. on the spines of epileptics. It will be remembered that 21 epileptics were given courses of E.C.T. without taking any precautions to prevent vertebral fractures, and in no case did any fresh fractures occur. If the mechanism of fracture production in E.C.T. had been different from that of epilepsy, we would have expected to find at least two or three patients with fresh fractures of dorsal vertebrae at the end of the treatment. On the other hand, the results do not surprise us if we believe that the mechanism is in fact similar in both cases, though weaker in E.C.T.

(5) *Individual Liability to Fractures.*

We are now in a position to attempt to solve the important question: Why do some patients get fractures and not others?

The findings of the present investigation agree with those of Easton and Sommers (1942), who reported that it is not previous spinal damage which determines the incidence of fractures. In no case among our patients was there evidence of E.C.T. producing fresh damage to old compression fractures due to epileptic fits. Some of our patients had kyphosis, scoliosis and spinal arthritis before treatment, but these changes did not appear to render them more liable to sustain vertebral fractures during electrical convulsion therapy.

A positive contribution towards the solution of the problem is afforded by the results of our clinical observations on the type and individual variations of the convulsions electrically induced in our epileptics. We have seen that certain patients have their fits in a predominantly flexed posture, and that others have convulsions in extension; while some patients are intermediate in pattern, sometimes straight, and at other times flexed or extended. We have also seen that each individual varies his posture to a certain degree from fit to fit, but that on the whole he remains in his own class. And the most important finding was that there was a definite correlation between these classes and the incidence of vertebral fractures. Fractures were present in

the "flexion class" and absent in the "extension class," while the intermediate posture class had a certain number of fractures. This means that, other things being equal, a patient's liability to fractures depends on his individual convulsion pattern, and this in turn probably depends on individual variations as regards the central neurological mechanism governing the muscular movements. From the fact of the day-to-day variation it would appear probable that this pattern is rather of a functional than a structural nature, and it may be that further investigation may bring to light factors which are capable of influencing it. One possible line of approach would be to investigate whether different electrode positions or varying electrode sizes have any effect on the convulsion posture, and it is planned to study this question in the future. The possibility of different electro-encephalographic pictures in the different classes of patients is also of interest.

In connection with the views here put forward it is appropriate to quote from the paper of Strauss, Landis and Hunt (1939), who investigated the metrazol seizure with the aid of accurate apparatus. They said: "Certainly all human epileptic seizures are not the same . . . some of our patients developed a flexion (of the arms) in one attack and an extension in another attack. This proved that in spite of the simultaneous innervation of all muscles the final position of the body is certainly not the result of an *equal* activity of these muscles. There must be central factors determining differences in the degree of innervation of the various muscle groups. What the reason may be for these differences in innervation in repeated experiments on the same subject we do not know."

(6) *Significance of the Fractures.*

The fact that we have established so firmly the similarity of vertebral lesions in epilepsy and convulsive therapy is of the utmost importance when we come to discuss the significance of these fractures in practice. The main weakness of previous assessments of the seriousness of this complication of E.C.T. was the comparatively short period during which the treatment has been carried out. It may well be that such fractures of the dorsal spine produce no immediate serious ill-effects, but this is no guarantee of what will happen in fifteen or twenty years' time, especially in view of the gross structural changes which take place in the vertebral bodies in many cases.

The present study has shown that a large proportion of epileptics have fractures which are identical in type, similar in location, and probably produced by the same forces as those found after convulsion therapy. The literature contains no accounts of any disabilities suffered by epileptics which could be ascribed to these lesions, and in our own cases a careful clinical investigation has failed to uncover any significant sequelae. We cannot be sure of the age of the fractures in our own epileptic patients, but they may well be of fifteen or twenty years' duration in some instances, and they can be used as demonstrations of what the E.C.T. fractures will be like in the distant future.

Why gross compression fractures of dorsal vertebrae should be harmless when similar lesions in the lumbar and cervical regions are so dangerous is a

difficult matter to explain, and the literature on the subject contains no satisfactory theory. My own suggestion is that structural changes in vertebral bodies are only of importance in regions where movement is taking place; the dorsal part of the spine, especially the middle region of it, is normally not required to move in the antero-posterior plane, and in fact acts as a steady fulcrum for the respiratory movements; because of this, changes in contour of these vertebral bodies are of little significance.

Conclusions.

(1) Fractures of dorsal vertebrae occur in 33 per cent. of male epileptics who suffer from frequent *grand mal* fits.

(2) The incidence of vertebral fractures in epilepsy is the same as in patients who have had pharmacological convulsion therapy.

(3) The type of vertebral lesion in epilepsy is the same as after convulsion therapy.

(4) The fractures in epilepsy are located a little lower down in the dorsal spine than those due to convulsion therapy. This may be associated with the fact that epileptic fits often occur with the patient in a vertical position, whilst convulsive therapy is invariably carried out with the patient lying horizontally.

(5) The mechanism of production of fractures in epilepsy and convulsion therapy is probably identical, the main factor being the force of muscular contraction during a flexion spasm.

(6) Vertebral fractures may be produced at any stage of the convulsive seizure.

(7) The smaller incidence of fractures after electrical convulsion therapy is probably associated with the fact that the seizures are less strenuous than those of epilepsy or cardiazol.

(8) The details of the convulsion in any individual vary on different occasions, but conform with some consistency to a characteristic pattern.

(9) A patient's liability to sustain fractures of the dorsal spine depends partly on his type of convulsion pattern.

(10) Patients may be divided into three main classes as regards posture during the convulsions: (1) Those who have their fit in a position of spinal flexion; (2) those who have their fit in a position of spinal extension; (3) an intermediate group whose spines are either straight, flexed or extended on different occasions.

(11) Patients in class (1) are most liable to sustain fractures, and patients in class (2) are least susceptible.

(12) Previous vertebral damage does not increase the patient's susceptibility to fractures.

(13) Fractures of the dorsal spine following convulsion therapy are not likely to lead to trouble in future years, and this risk should not prevent the treatment being carried out.

My best thanks are due to Dr. N. Moulson, Medical Superintendent, Dr. I. Davies, Consulting Radiologist, and Mr. A. Dignam, Radiographer of Cefn Coed Hospital, for their advice and assistance.

REFERENCES.

BARRETT, J. E., FUNKHOUSER, J. B., and BARKER, W. A. (1942), *Am. J. Psychiat.*, **90**, 387.
CAPLAN, G. (1945), *Brit. Med. J.*, **1**, 511.
Idem (1945*a*), "Studies on Electrical Shock Therapy in Neuropsychiatry," Thesis, University of Manchester.
COOK, L. C. (1944), *J. Ment. Sci.*, **90**, 435.
Idem and SANDS, D. E. (1941), *ibid.*, **87**, 230.
EASTON, N. L., and SOMMERS, J. (1942), *Am. J. Psychiat.*, **98**, 538.
ERLACHER, P. J. (1920), *Ztschr. f. Orthop. Chir.*, **40**, 385.
FITZPATRICK, C. P. (1939), *J. Nerv. Ment. Dis.*, **90**, 515.
FRIEDMAN, E., BRETT, A. L., and VOGT, E. C. (1940), *New England J. Med.*, **222**, 704.
JESSNER, L., and RYAN, V. G. (1942), *Shock Treatment in Psychiatry*. Heinemann, London.
KRAUS, G. R., and VIERSMA, H. J. (1940), *J. Ment. Sci.*, **86**, 76.
LEHNDORFF, H. (1907), *Wien. med. Wchnschr.*, **67**, 2477.
MOORE, M. T., WINKLEMAN, N. W., and SOLIS-COHEN, L. (1941), *J. Nerv. Ment. Dis.*, **94**, 309.
PEARSON, M. M., and OSTRUM, H. W. (1940), *Am. J. Roent. and Rad. Ther.*, **44**, 726.
RAND, C. W. (1938), *Bull. Los Angeles Neur. Soc.*, **3**, 141.
REED, G. E., and DANCEY, T. E. (1940), *Canad. Med. Assoc. J.*, **42**, 38.
ROBERG, O. T. (1937), *J. Bone and Joint Surgery*, **19**, 603.
SCHATZ, I., and KONWALER, B. E. (1941), *J. Nerv. Ment. Dis.*, **93**, 169.
STALKER, H. (1938), *Lancet*, **2**, 1172.
STRAUSS, H., LANDIS, C., and HUNT, W. A. (1939), *J. Nerv. Ment. Dis.*, **90**, 439.
WESPI, H. (1938), *Schweiz. Arch. f. Neurol. u. Psychiat.*, **42**, 404.
ZISKIND, E., and SOMERFELD-ZISKIND, E. (1939), *Bull. Los Angeles Neur. Soc.*, **4**, 45.