SPINAL INJURIES IN CONVULSION THERAPY.

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Therapeutic convulsions were introduced by Meduna in 1934, but it was not until the latter part of 1938, after many thousands of fits had been induced all over the world, that spinal injuries were first recorded (Wespi, 1938; Stalker, 1938). The reason for this is not far to seek; it lies in the fact that these injuries are for the most part symptomless, and consequently to be found only by means of routine radiographic examinations. In 1939 alarming reports of the high incidence of compression fractures following induced convulsions were published, and the possibility of subsequent permanent disability led in many hospitals to the abolition of a very useful form of treatment.

Certain facts, however, suggested that these injuries might be less important than was at first feared. First, no evidence of subsequent disability had come to light, although the treatment was introduced six years ago. Delayed vertebral collapse (Kümmel's disease) usually manifests itself within a year of the original injury, and if its occurrence were a real danger, it seemed strange, considering the high percentage of compression fractures, that in not even one of the earliest cases had any secondary symptoms been reported. Secondly, the fact that these injuries were brought to light only by radiography rendered it possible that similar injuries might sometimes occur after spontaneous fits. If symptomless compression fractures were to be found in cases of long-standing epilepsy, obviously the danger of subsequent disability after induced convulsions would assume far less terrifying proportions.

With these ideas in mind we have taken lateral radiograms of the spine of 134 idiopathic epileptics and 135 control cases, as well as of 143 patients who have undergone a course of induced convulsions.

RESULTS.

TABLE I.—*Incidence of Vertebral Fractures with Induced Convulsions, Idiopathic Epileptics and Control Cases.*

	Induced convulsions.			Idiopathic epilepsy.			Control cases.		
	M.	F.	Total.	M.	F.	Total.	M.	F.	Total.
Cases	73	70	143	72	62	134	97	38	135
Traumatic fractures	13	8	21 (14·7%)	10	4	14 (10·4%)	2	0	2 (1·5%)

TABLE II.—*Distribution of Vertebral Fractures.*

Vertebra.	Th. IV.	V.	VI.	VII.	VIII.	IX.	X.	XI	XII.	L. I.	II.	III.	IV.	Total (vertebræ).
Induced fits	1	7	6	4	3	4	1	..	..	1	..	..	..	27
Epiletics	..	..	..	4	1	..	2	..	3	3	3	1	1	18
Controls	1	..	..	1	..	..	..	..	..	..	..	..	..	2

(a) *Induced Convulsions (Cardiazol and Triazol).*

Twenty-one out of 143 patients showed recent fractures of the spine, almost certainly due to the convulsions (Table I). Of these, 18 (12 male and 6 female) were compression injuries, varying from severe flattening and wedging of two bodies to slight compression of a single body. The anterior aspect of the vertebral body usually suffered most. The three remaining fractures involved the anterior or superior borders of thoracic vertebral bodies without producing any signs of compression.

The mid-dorsal region was predominantly affected, although fractures of all vertebrae were seen from Th. IV to L. I, excepting Th. XI and XII (Table II).

Other traumatic lesions were "knörpelknötchen" (3 cases), "cartilaginous pearls" (1 case), and herniation of the nucleus pulposus (1 case). Arthritic changes were seen in a few cases.

The number of fits induced appeared to have little bearing on the incidence of fractures. On two occasions fractures were caused by the first fit. In most cases fractures were discovered only through routine radiography of the spine some time after treatment had ended. The view of Reed and Dancey (1940) that the involvement of more than one vertebra is due to multiple fits is not always true, as one of our patients sustained severe compression of the 5th and 6th thoracic vertebrae after a single fit. The severity of the convulsion appeared to be little guide as to the probability of fracture. Of five males who had conspicuously severe fits, three showed no signs of injury.

(b) *Idiopathic Epileptics.*

Traumatic compression fractures were found in 14 (10 male and 4 female) out of 134 epileptics, all of whom were subject to major fits (Table I). In 9 of these 14 cases the fits started after the age of 25 years, when ossification

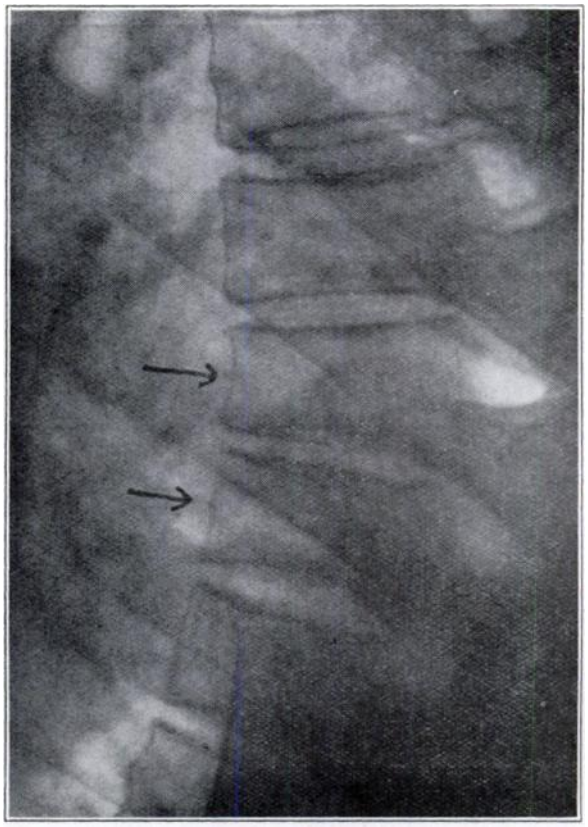

FIG. 1.—Compression fracture of 5th and 6th thoracic bodies after one cardiazol convulsion.

is usually complete. The type of injury (Fig. 3) was so like that found after induced convulsions that a similar mechanism can justifiably be assumed. The distribution of the fractures was rather lower, the last thoracic and first two lumbar vertebrae being more often affected (Table II). Besides the compression fractures "knörpelknötchen" were found in one case and deformity of the superior surface of a vertebral body with formation of a "cartilaginous pearl" in another. Of non-traumatic lesions extensive deformity of several

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vertebrae, apparently due to occupational or constitutional causes, appeared in four cases, while six others showed arthritic changes. As with induced convulsions, no correlation between the number or severity of fits and the occurrence of fractures could be ascertained.

FIG. 2.—Same case as in Fig. 1 before treatment.

(c) *Controls.*

Control cases consisted of patients radiographed before treatment and other adult mental patients under 50 years of age. 135 cases (97 male and 38 female) produced two compression fractures (both male). In one case there was a definite compression fracture of the 4th thoracic body, typical of the injury associated with induced fits; in the other slight wedging of the anterior border of the 7th thoracic body was seen. Both were cases of schizophrenia of recent admission. In addition one woman showed herniation of the nucleus pulposus and another was the subject of osteo-arthritis.

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Published figures show an incidence of vertebral fractures varying between 15·7 and 47·1 per cent., with an average of 30 per cent. in 243 cases (Table III). American figures tend to be higher than European. The lower incidence of fractures in our cases may be due in part to careful avoidance of restraint during fits and to excluding elderly patients from treatment, but we feel that

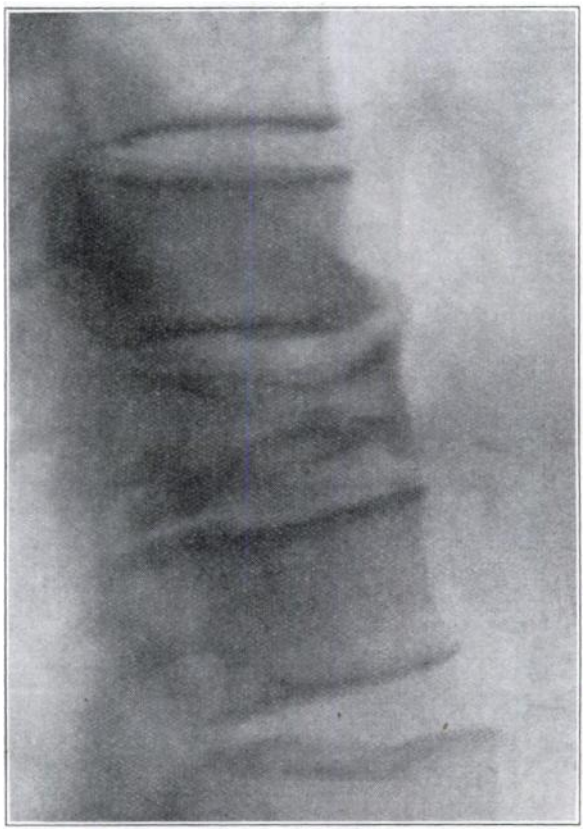

FIG. 3.—Severe crushing of 1st lumbar vertebra in a case of idiopathic epilepsy.

the rigorous elimination of old injuries, constitutional and other deformities, arthritic changes and artefacts due to radiographic distortion, all of which may at times simulate convulsion fractures, has played no insignificant part. In this connection we tend our grateful thanks to Dr. J. Duncan White, who has carefully examined our radiograms, and without whose kind co-operation and great experience of spinal radiography our figures would have been far less accurate and considerably higher.

Discussion.

1. *Incidence of Fractures with Induced Fits.*

Table III.—*Vertebral Fractures after Induced Convulsions.*

Author.	Cases.			Vertebral fractures.			Percentage.
	M.	F.	Total.	M.	F.	Total.	
Polatin *et al.* (1939)	20	31	51	6	16	22	43·1
Bennett and Fitzpatrick (1939)	..	..	17	..	..	8	47·1
Palmer (1939)	..	..	20	..	..	5	25·0
Kraus and Viersma (1940)	13	38	51	6	2	8	15·7
Wyllie (1940)	..	..	18	..	3	3	16·7
Reed and Dancey (1940)	..	..	86	50%	18%	27	31·4
Total	..	..	243	..	..	73	30·0
Cook and Sands	73	70	143	13	8	21	14·7

Sex incidence appears to vary considerably (Table III). Reed and Dancey (1940) give no exact figures, but find 50 per cent. fractures in their male and 18 per cent. in their female cases; Kraus and Viersma (1940) also find a male preponderance, which our figures tend to support. On the other hand, Polatin *et al.* (1939) find a marked female preponderance, while Wyllie's three fractures out of 18 unspecified cases were all in female patients. If force of muscular contraction is the main criterion of fracture, one would naturally expect male cases to suffer most, but it must be admitted that neither powerful musculature nor severe fits have in our experience been any guide to probability of fracture.

2. *Incidence of Fractures in Epileptics.*

We have been able to find only two references to vertebral fractures in epilepsy. Kraus and Viersma (1940) found one compression fracture and one doubtful lesion of a vertebral body in 21 epileptics. Reed and Dancey (1940), supplying only percentage figures, state that they found 34·2 per cent. compression deformities in 72 idiopathic epileptics—46·8 per cent. of 32 males and 24·5 per cent. of 40 females. Elementary arithmetic proves these figures to be inaccurate, possibly due to misprints. The incidence of fractures in our epileptic cases (14 out of 134—10·4 per cent.) is considerably lower than that published by Reed and Dancey, but the ratio of fractures found after induced fits to those occurring in idiopathic epileptics is comparable. Actually Reed and Dancey find vertebral fractures slightly more common in epileptics (34·2 per cent.) than after induced fits (31·4 per cent.), while our figures are 10·4 per cent. for epileptics and 14·7 per cent. after induced convulsions.

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3. *Localization and Multiplicity of Injured Vertebrae.*

There is no dispute that fractures following induced convulsions mainly affect the mid-dorsal region (Polatin *et al.*, 1939; Palmer, 1939; Kraus and Viersma, 1940; Reed and Dancey, 1940; Graves and Pignataro, 1940), and that the anterior part of the vertebral body bears the brunt of the injuries. The lumbar region is rarely injured—one fracture of the 1st lumbar body reported by Kraus and Viersma (1940) and our own single case of fracture of the same vertebra appear to be the only cases noted. In idiopathic epilepsy we have found a somewhat lower distribution, 11 out of the 18 fractured vertebrae lying between Th. XII and L. IV (inclusive) (Table II).

In none of our cases were more than two vertebrae affected, a single body being injured in 15 out of 21 post-therapeutic cases and in 10 out of 14 epileptics. Injury to more than two vertebrae in the same patient, however, has been published.

4. *Symptoms of Fracture.*

Most workers agree that these fractures are usually symptomless, but Bennett and Fitzpatrick (1939) state that pain in the back was present in all of their eight cases of fracture. Our experience is in accord with the majority, but we have noted that a number of patients, not limited to those in whom bony injuries were found, complained of an aching type of pain in the back after fits, which passed off within 48 hours. This symptom appears to be due to the severe and unaccustomed muscular action of the convulsion and is no criterion of vertebral fracture. We noticed that some patients who complained of pain most emphatically showed perfectly normal spinal radiograms, and we are able to support the view of Polatin *et al.* (1939) that the degree of injury bears no relation to the severity of backache.

Occasionally severe pain was encountered, but Wyllie's experience that "two (out of three cases of vertebral fracture) complained of persistent severe pain in the back" must be exceptional. One only of our patients suffered severe pain, but this was so intense as to necessitate morphine injections over a period of several days. Radiograms showed severe compression of the 5th and 6th thoracic vertebrae, but no displacement could be seen. The pain, which was accompanied by tenderness over the 5th, 6th and 7th thoracic spines, was sharp and well-defined, and was also referred to the upper abdomen and to the right side of the chest in the areas subserved by the cutaneous branches of the 5th and 6th thoracic nerves. In this case alone was there definite local tenderness. A spinal jacket was applied with excellent result and the patient was discharged six months later without any disability.

5. *Mechanism of Fracture.*

It is well established that convulsion fractures are dependent upon two variables, namely, the force of the muscular contractions and the strength and resistance of the bones. Beyond this we know little of their mechanism. Hypotheses have been advanced to explain why the mid-thoracic region is chiefly affected in induced fits, but none of them accounts for the frequent occurrence of fractures of the lower thoracic and upper lumbar vertebrae in epileptic convulsions. The special vulnerability of the anterior parts of the vertebral bodies has also been much discussed, and in this connection we would suggest that this may be due, not to any particular lines of muscular force, but to the support given to the posterior part of the bodies by the pedicles, whose structure is of compact bone.

(6) *Significance of Vertebral Fractures.*

In discussing the significance of vertebral fractures it is necessary to consider what relevant information can be gained from two other conditions in which vertebral deformities occur, namely, Kümmel's disease and tetanus.

Kümmel's disease has been defined as the delayed collapse of vertebral bodies after spinal injury. It is usually referred to collapse following external injuries, such as falls from a horse or a ladder, heavy blows on the back, etc. (Cardis, Walker and Oliver, 1928), but can be applied equally well to delayed collapse following injury of muscular origin. Kraus and Viersma (1940) and other writers have pointed to the possibility of Kümmel's disease following the fractures of induced fits, but we believe this to be very unlikely. In the first place Kümmel's disease is apparently unknown as a complication of idiopathic epilepsy, although we now know that compression fractures of vertebral bodies are not uncommon in this condition. Secondly, Kümmel's disease usually shows itself within the first year after the original injury, and no cases, as far as we can discover, have been reported six years after the introduction of therapeutic convulsions. From our own experience we can say that no symptoms suggestive of delayed vertebral collapse have come to light in 225 cases treated over 12 months ago, and either now in hospital or followed up a year or more after treatment. On the other hand, the possibility of delayed collapse after induced fits must not be overlooked altogether, if only because one instance of Kümmel's disease has been reported in connection with tetanus. In this case collapse of three vertebral bodies occurred six months after an attack of tetanus, although the spine was apparently normal immediately after the illness (Roberg, 1937).

Compression fractures due to tetanus have been described in children and adults (Roberg, 1937; Wheeler, 1939; Dietrich *et al.*, 1940). The spasms of epilepsy and tetanus have much in common—trismus, severe tonic spasm and

tendency to opisthotonos occurring in both conditions. The chief differences are that in tetanus clonus is less regular and relaxation between clonic spasms incomplete, tonic contraction is more prolonged and repeated, and consciousness is retained. In both conditions the mid-dorsal vertebrae are predominantly affected and doubtless the mechanism of fracture is the same. Fractures caused by tetanus have been identified eleven years after the attack. Persistence of symptoms such as radiating back pains or of functional incapacity is unknown.

Now that so much negative evidence has accumulated regarding permanent disability and secondary vertebral collapse after spinal fractures caused by spontaneous and induced convulsions, we are convinced that the risk of these fractures occurring may be viewed without apprehension, and certainly should not be allowed to preclude convulsion treatment in suitable cases.

(7) *Radiographic Examination.*

It is now our routine practice to take lateral spinal radiograms before starting convulsions and at the end of the course. The original radiogram is not only a preliminary safeguard, but serves as a basis of comparison in reading later films. Naturally radiograms are taken at any time during the course, if there are any clinical indications of spinal injury. Graves and Pignataro (1940) have shown that the great majority of injuries occurs during the first five fits; consequently it must be considered whether radiograms should be taken after each of the first five fits. We do not believe this to be necessary in the absence of clinical symptoms, because we have found that symptomless fractures are not aggravated by further fits, and therefore should not be allowed to hold up treatment. In most of our cases of fracture treatment had been completed before the risk of this complication had become recognized, and consequently many fits must have been induced after the fractures had occurred. In no case has there been any subsequent disability. If, however, there is definite pain or tenderness and fracture has been confirmed by radiography, treatment should be discontinued and a plaster jacket applied.

(8) *Preventive Measures.*

No satisfactory method of preventing spinal injury has so far been found. Among those suggested have been (1) spinal anaesthesia (Hamsa and Bennett, 1939), (2) injection of curare (Palmer, 1939) and of beta-erythroidin hydrochloride (Rosen, Cameron and Ziegler, 1940), (3) mechanical methods, e.g. acute flexion of the spine in the lateral position (Polatin *et al.*, 1939), extension of the spine (Furst, 1940; Rankin, 1940; Graves and Pignataro, 1940).

In the recently advocated method of Furst a small pillow is placed under the mid-thoracic vertebrae and counter-pressure exerted by assistants against

chin, shoulders, hips and knees. By this procedure Furst claims to have reduced the incidence of vertebral fractures to 8 per cent. in 37 consecutive cases. Using a similar means of restraint Graves and Pignataro confirm these findings in 187 cases. Since the anterior borders of the vertebral bodies sustain the greatest damage as a rule, this might appear to be a logical procedure, but we cannot envisage any form of mechanical restraint capable of reducing the chances of bony injury without incurring a greater risk of tearing muscle fibres or insertions. Neverthless, we are trying out Furst's method in electrically induced fits, without as yet experiencing very encouraging results. Probably the only certain way to prevent vertebral fractures is to employ a spinal anaesthetic. This obviously complicates an otherwise relatively simple procedure, and is unlikely to find general acceptance so long as these fractures remain largely symptomless.

Apart from the present investigation into Furst's method, we have been careful to avoid all forms of restraint, and we feel that this policy accounts to some extent for the relatively low incidence of fractures in our cases.

Summary and Conclusions.

1. Lateral radiograms of the spine showed vertebral fractures in 21 out of 143 patients who had received convulsion treatment (14·7 per cent.), 14 out of 134 idiopathic epileptics (10·4 per cent.), and 2 out of 135 controls (1·5 per cent.).

2. The incidence of fracture after induced fits in our series is lower than the average published (30 per cent.).

3. The predilection for post-therapeutic fractures to occur in the mid-thoracic region is confirmed. In epileptics the distribution is somewhat lower.

4. With one exception, marked by severe radiating and referred pain and local tenderness, none of the fractures was accompanied by referable symptoms. Generalized pain in the back was found to be an unreliable symptom, and was often felt by patients who were not the subject of vertebral injury.

5. It is suggested that the relative immunity of the posterior parts of the vertebral bodies may be due to the support afforded by the pedicles, which are of compact bone.

6. The significance of these fractures appears to have been greatly overestimated. We take this view because (*a*) we have found similar fractures in over 10 per cent. idiopathic epileptics, and yet no disability due to vertebral fracture has, to our knowledge, ever been reported as a complication of epilepsy, and (*b*) delayed vertebral collapse (Kümmel's disease), which is the main cause for apprehensiveness, has not been observed in epilepsy or after six years of convulsion therapy. This is particularly important, as Kümmel's disease usually shows itself within the first year after the original injury. We feel that there is no justification for allowing the risk of vertebral fractures to preclude convulsion treatment in suitable cases.

7. Lateral spinal radiograms should be taken before and after treatment. Intermediate films are unnecessary unless indicated by symptoms suggesting fracture.

8. Attempts to prevent vertebral compression fractures by any form of restraint during fits are unlikely to meet with much success, and may involve a serious risk of tearing muscle fibres or insertions. Spinal anaesthesia is scarcely a practical proposition for general use. Insistence on reducing restraint during fits to the barest minimum may well be a relevant factor in the low incidence of fractures in our cases.

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