REFERENCES

- ¹ Nordsted, Norgaard, and Thaysen: *Acta Med. Scand.*, April 28th, 1930, 125.
- ² Fletcher, A. A., and Campbell, W. R.: *Journ. Metabol. Research*, 1922, ii, 637.
- ³ Banting, F. G., Campbell, W. R., and Fletcher, A. A.: *British Medical Journal*, 1923, ii, 8.
- ⁴ Cammidge, P. J.: *British Medical Journal*, 1930, i, 818.
- ⁵ Sevringhaus, E. L., Kirk, E., and Heath, H. J.: *Amer. Journ. Med. Sci.*, 1923, clxvi, 677.
- ⁶ Sendrail and Planques: *Gaz. d. Hôp.*, 1927, c, 1105, 1137.
- ⁷ Harris, S.: *Journ. Amer. Med. Assoc.*, 1924, lxxxiii, 729.
- ⁸ Moore, H., O'Farrell, W. R., Malley, L. K., and Moriarty, M. A.: *British Medical Journal*, 1931, ii, 837.
- ⁹ Stowell, T. E. A.: *Ibid.*, 1930, i, 927.
- ¹⁰ Gammon, G. D., and Tenery, W. C.: *Arch. Int. Med.*, 1931, xlvii, 829.
- ¹¹ Cammidge, P. J.: *Lancet*, 1924, ii, 1277.
- ¹² Idem: *Practitioner*, 1927, cxix, 103.
- ¹³ Thalimer and Murphey: *Journ. Amer. Med. Assoc.*, 1928, xci, 89.
- ¹⁴ McClenahan, W. U., and Norris, G. W.: *Amer. Journ. Med. Sci.*, 1929, clxxvii, 93.
- ¹⁵ Wilder, Allen, Power, and Robertson: *Journ. Amer. Med. Assoc.*, 1927, lxxxix, 348.
- ¹⁶ Elliott, C. A.: *Ibid.*, 1929, xcii, 2188.
- ¹⁷ Howland, G., Campbell, W. R., and Maltby, E. J.: *Trans. Amer. Neurol. Assoc.*, 1929, 551.
- ¹⁸ Smith, M. G., and Seibel, M. G.: *Amer. Journ. Path.*, 1931, vii, 723.
- ¹⁹ Phillips: *Journ. Amer. Med. Assoc.*, 1931, xcvi, 1195.
- ²⁰ Pemberton, H. S.: *British Medical Journal*, 1925, i, 1004.
- ²¹ Finney, J. M. T., and Finney, J. M. T., jun.: *Ann. of Surg.*, 1928, lxxviii, 584.
- ²² Allen, F. N.: *Arch. Int. Med.*, 1929, xlv, 65.
- ²³ Griffith, J. P. C.: *Journ. Amer. Med. Assoc.*, 1929, xciii, 1527.
- ²⁴ Josephs: *Amer. Journ. Dis. Child.*, 1929, xxxviii, 746.

COMPRESSION FRACTURE WITH COMMUNITION OF BODY OF THIRD LUMBAR VERTEBRA AND NEURAL ARCH OF FOURTH VERTEBRA WITH PARAPLEGIA

BY

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(With Special Plate)

Watson Jones (*British Medical Journal*, February 21st, 1931) published for the first time a safe and simple method of treating crush fractures of the spine. Hitherto these cases had been looked upon apathetically, and the results of the usual simple recumbency method of treatment had inevitably been followed by serious deformity and crippling. Davis of Erie (Pa.) had devised a method where traction and forcible hyperextension were combined with suspension of the anaesthetized patient by the heels from the roof of the operating theatre, and Hersikoff described a case of reduction in which four strong men had combined to exert traction on the patient's spine.

Aetiology of Crush Fracture

The mechanism in crush fracture of the vertebral body is indirect injury—that is, a bending or hyperflexion of the spine, associated in some cases with compression in the long axis of the column. Falls on the back of the neck from a height, or a heavy weight falling on the back while it is in a stooping position, are examples of this.

When the spine is forcibly flexed the first effect is a compression of the bodies of the vertebrae and of the intervertebral disks. The upper vertebrae tend to be displaced forwards, but are held behind by the articular processes, which prevent any further movement. Should the flexion continue, the lower articular processes of the upper vertebrae rise and jump the upper processes of the vertebrae below, and a dislocation takes place. Compression fracture of the vertebral body without displacement is met with in the dorsal and lumbar regions, par-

ticularly the twelfth dorsal and the first lumbar vertebra, and also in the upper part of the dorsal vertebrae in the region of the fourth. The latter is frequently associated with a fracture of the sternum opposite the second rib. The cause is always an indirect injury compressing the vertebral column in a vertical direction, and causing hyperflexion. The soft, spongy bone is compressed between the upper and lower laminae of compact bone. Owing to the presence of the pedicles behind, the posterior part is strengthened, the crushing being more marked in front than behind. The result may be a complete comminution of the soft cancellous bone, which may be broken up into innumerable fragments and displaced in all directions—usually forwards, as this is the direction of the violence.

Anatomical Considerations

The anterior common ligament is a strong band of fibres which extends along the anterior surfaces of the bodies of the vertebrae. It is broader below than above, thicker and narrower in the thoracic than in the cervical and lumbar regions, being somewhat thicker and narrower opposite the bodies of the vertebrae than opposite the intervertebral fibro-cartilages. It consists of dense longitudinal fibres, which are intimately adherent to the intervertebral fibro-cartilages and the prominent margins of the vertebrae, but not to the middle parts of the vertebral bodies. In the latter situation the ligament is thick, and fills up the concavities on the anterior surfaces, and makes the front of the vertebral column more even. It is composed of several layers of fibres, of which the most superficial are the longest, and extend between four or five vertebrae. The intermediate fibres extend between two or three vertebrae, and the deepest reach from one vertebra to the next. At the sides of the bodies the ligament consists of a few short fibres, which connect adjacent vertebrae (Gray). Reduction depends on the integrity of this anterior common ligament. The process of hyperextension puts this ligament into tension, which in its turn presses the fragments back into position, and so moulds the vertebrae back into form. Thus pressure on the cord by the laminae of the intact vertebrae above is immediately released, and unless irrevocable damage, such as crushing or severance, is effected, recovery takes place.

CASE RECORD

This is well illustrated by the following case.

J. D., aged 50, was admitted to Queen Mary's Hospital on August 11th, 1931, having been knocked down by a lorry. He did not lose consciousness, and remembers being crushed between the front axle of the lorry and the ground—that is, his shoulder being under the front axle, his trunk in a position of hyperflexion.

On admission he was suffering from shock, and complained of pain in the small of his back, inability to move his legs, and also that "he could not feel them." He was immediately turned over on his face, and nursed in that position until two days later, when reduction was effected.

On examination the spines of the dorsal and upper lumbar region were normal until the mid-lumbar region, when a depression was felt, the spine above forming an elevation with a depression for two spines level below this. X-rays taken in lateral and antero-posterior positions showed marked comminution of the body of the third lumbar vertebra, with forward displacement of the greater part of the body into the abdominal cavity, and fracture of the spine and neural arch of the fourth (Fig. 1 on Plate).

Examination of lower limbs showed: in the right leg, flaccid tone with complete motor paralysis; all reflexes and sensation absent. Left leg: flaccid; complete paralysis, barring an ability to contract the quadriceps extensor muscle; sensation, touch, and sense of position were absent, and the patient was unable to void urine and evacuate the bowels. Catheterization was performed.

Progress

August 12th, 1931.—Patient was given morphine 1/4 grain, and laid face downwards, his arms resting on a high window-ledge, the lower part of the thighs on an operating table, which was lowered to its extreme limit, his body being curved, and with the shoulders about 2 feet higher than the knees. He had to be supported by two porters, one each side, holding a shoulder. Plaster-of-Paris was applied from the shoulder to the greater trochanter. An x-ray taken next day showed the result—complete reduction of the fracture (Fig. 2). Immediately the plaster jacket was applied he was very comfortable and in no pain. Next day there was some return of power in the left leg, and he could contract some groups of muscles, and he complained of tingling in his legs. The bowels were costive, and an enema, aided by pituitrin, was necessary to secure an evacuation.

During the next few days motor power and sensation gradually returned, particularly on the right side, reflexes on both sides being, however, still absent. Urine was drawn off twice daily by catheters under strict aseptic precautions.

August 26th, 1931 (fifteen days after the accident).—The patient passed 12 ounces of urine normally, but he had to be catheterized until September 13th, since which time he has been able to void in the usual manner. Motor power and sensation and reflexes returned gradually. By August 24th there was complete recovery.

October 10th.—He was allowed up and out of bed.

October 16th.—Able to walk unaided up and down the ward.

December 21st.—Plaster removed.

February 1st, 1932.—Electric reaction of muscles of lower limb showed normal responses and complete recovery. He could now walk unaided. X-rays taken later confirmed the reduction. The spine was in excellent position, and there was no atrophy of body of third lumbar vertebra. He would now have been able to resume his work but for polyarticular rheumatism, an intercurrent infection.

SUMMARY

This case illustrates a simple method of reducing severe comminuted fracture of the spine, accompanied by severe cord injury. It would have been impossible by any other method to approach, replace the fragments, and maintain them in position. He has recovered completely, both as regards his fracture and the cord injury. This method provides an easy way of nursing patients, changing their position, and avoiding the usual complications of bed-sores and respiratory troubles.

I wish to express my thanks to Mr. Percival P. Cole, F.R.C.S., under whom this case was admitted, and by whose kindness I had the opportunity of treating the case and publishing these notes.

CHRONIC GASTRIC ULCER IN A BOY AGED 13

BY

MICHAEL OLDFIELD, B.M., B.CH.

(With Special Plate)

Chronic gastric ulcer is rare in children, although acute peptic ulceration, both of the stomach and of the duodenum, is not uncommon in infants under a year old. Acute and chronic ulcers are more common in adult life, and it would be interesting to know what percentage of patients who come for treatment at a later date have had preceding ulcer dyspepsia in childhood. Proctor¹ describes sixteen such cases at the Mayo Clinic.

Children are rarely brought up for treatment for dyspepsia of the ulcer type, since this is only associated with the chronic disease. They are seen more commonly following the dramatic complications of acute ulceration,

such as melaena, haematemesis, or perforation. Paus,² Theile,³ Robinson,⁴ and Holm⁵ record cases of perforated chronic gastric ulcer in children, which were operated upon successfully; and Proctor,¹ Weber,⁶ and Miller⁷ report cases of chronic gastric ulcer in childhood not submitted to operation. Among 1,596 cases of gastric ulcer observed in the Mayo Clinic, only one occurred in a child. Professor Stewart,⁸ in a series of 200 cases of chronic gastric ulcer coming to post-mortem at the General Infirmary at Leeds, found none under 10 years, and three between 10 and 20 years of age. Deaths from chronic ulceration are obviously extremely rare in childhood. It is possible, however, that if a careful history were taken, an account of a preceding typical dyspepsia in childhood might be obtained in many adult cases of chronic peptic ulcer. This would emphasize the importance of giving rigorous and prolonged treatment to children suffering from chronic dyspepsia.

CASE REPORT

H. W., a pale, thin boy, aged 13, was admitted to the General Infirmary at Leeds, under the care of Dr. Veale, on December 4th, 1931, for haematemesis.

History.—When the patient was 11 years old he had a series of sore throats, followed by "indigestion." This consisted of an aching pain in the epigastrium when he was hungry. The pain was so severe that it wakened him during the night. It was relieved by food, and was occasionally accompanied by vomiting. Symptoms were intermittent; bouts of pain, lasting two to three weeks, alternating with periods of freedom. The pain gradually grew worse, but it was not for over a year after the onset of this symptom that the patient was taken by his mother to the doctor. Frequent small milk feeds and alkalis were given, and later tonsillectomy was performed. The symptoms disappeared within a fortnight.

Before breakfast, on the morning of December 4th, 1931, the boy had a sudden stabbing pain in the epigastrium and mid-sternum. He became thirsty, and then vomited about half a pint of dark red clotted blood. He was admitted to hospital the same day.

Physical Examination.—His face was pale, his lips and conjunctivae were blanched. Pulse 120. Temperature 98.1°. Respirations 24. There was slight epigastric tenderness. There was no evidence of bleeding from the nose or mouth. The spleen was not palpable. The heart, lungs, nervous system, optic disks, and urine showed nothing of note. No septic foci were found in the nose, ears, or throat.

Special Examinations.—(1) Occult blood was present in the faeces. (2) A blood count showed mild secondary anaemia. (3) A Rehfuess test meal showed a "high normal" acidity—that is, 1½ hours after the meal the total acids were equivalent to 80 c.cm. N/10 NaOH per 100 c.cm. of gastric contents. Free hydrochloric acid reached 0.2005 per cent., equivalent to 55 c.cm. N/10 NaOH per 100 c.cm. of gastric contents. There was slight delay in emptying of the stomach, complete after 2½ hours. (4) X-ray examination showed a small ulcer crater on the posterior surface of the stomach, close to the lesser curvature, three inches from the pylorus.

Progress.—The patient was treated medically by prolonged rest in bed, starvation, followed by Hurst's dieting. Olive oil and belladonna were given alternately before meals; alkalis after meals. He remained in bed in hospital for seven weeks, and was free from symptoms when discharged. He stayed in bed at home for another five weeks.

On March 1st, 1932, he came up to hospital. A radiograph was taken; no ulcer crater was to be seen. A Rehfuess test meal showed the gastric ulcer diathesis to be present still. There was a "high normal" acidity—that is, in the specimen taken 1½ hours after the meal the total acids were equivalent to 95 c.cm. N/10 NaOH per 100 c.cm. of gastric contents. Free hydrochloric acid reached 0.219 per cent.—that is, equivalent to 60 c.cm. N/10 NaOH per 100 c.cm. of gastric contents. There was still slight delay in emptying of the stomach, complete after 2½ hours. Seven weeks have elapsed

H. DAVID ISAACS: COMPRESSION FRACTURE WITH COMMINATION OF LUMBAR VERTEBRAE

FIG. 1.—Radiograph taken on August 11th, 1931, before treatment.

FIG. 2.—Radiograph taken on August 13th, 1931.

MICHAEL OLDFIELD: CHRONIC GASTRIC ULCER
IN A BOY

FIG. 1.—Showing small ulcer crater on posterior surface near lesser curvature of stomach. There is a translucent zone around crater corresponding to oedematous ulcer margins.

SIDNEY B. RADLEY: SUBPERITONEAL UTERINE
FIBROID

FIG. 1.—Naked-eye appearance of tumour.