

The Pathogenesis Of Pott's Paraplegia and The Effect Of  
Surgical Intervention in its Course and Prognosis.

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Surgical Intervention in its Course and Prognosis.

The pathogenesis or pott's paraplegia, the place of surgery in the treatment, and the best and most effective surgical approach for a rapid and reliable cure have been discussed by many investigators.

Hodgson and associates evaluated and popularised the anteriorradical debridement and fusion of the affected area in the spine.

The object of this paper is to report and demonstrate encouraging results of anterior radical debridement, decompression and fusion carried out in 74 patient by the authors in the manner described by Hodgson and associates.

**Material and Methods**

Fourteen patients 8 females and 6 males were included in this study. The ages of these patients varied between 17 and 70 years (average age 42 years).

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Symptoms were present between 6/12 and 2 years prior to operation. The most important symptom was backache usually followed by a progressive paraplegia with a fairly sudden deterioration in the month or two before admission to hospital. Usually the thoracic vertebrae were involved (see table) but in one case we had evidence of C5-C6 lesion.

On examination, one patient had apstic quadriparesis, four flaccid paraplegia, and the rest spastic paraplegia . Four had severe sphincter problems and nearly all had a sensory level to pin prick and temperature and in most cases diminution of vibration and position sense below the level of the lesion. In two cases the chestX-ray showed evidence of old pulmonary tuberculosis. Straight X-rays or tomography showed bony destruction in all cases and evidence of paravertebral abscess formation in all cases bar one. In one case(MD aged 70) the radiological changes were indistinguishable from secondary neoplastic lesion with collapse of vertebrae, intact intervertebral disc and no kyphosis. One patient was readmitted after operation with Addison's disease and in two cases there were radiological and clinical evidence and ankylosing spondylitis.

The sedimentation rate was over 50/ hour in all cases, the Montoux. Test at least ++ in 48 and +++ in 72 hours. Nearly all patients who had myelography had complete black at the appropriate level and raised CSF protein.

Specimen obtained at surgery histologically showed evidence of tuberculous osteomyelitis. The sedimentation rate was followed post operatively and found to be a good index of clinical improvement.

All patients were treated with anti T.B triple drugs (INH, Ethambutal, streptomycin) for a period of one to two

weeks prior to surgery. The cervical spine was approached through a left anterior approach as described by Southwick and Robinson as for excision of cervical disc and anterior fusion. The thoracic spine was approached through a right throacotomy and the lumbar spine through a transperitoneal approach. Abscess and caseous material were completely evacuated whenever found. All debris such as devitalized disc material, sequestered bony fragments, devitalized portion of vertebrae and granulation tissue were radically debrided down to the posterior longitudinal ligament which was also found to be involved in 2 cases with active tuberculosis and contracted and fibrotic in 2 cases with healing T.B, mechanically compressing the cord, posterior longitudinal ligament was excised to decompress the cord, whenever found necessary, after a thorough. The spinal cord was completely decompressed from any external pressure. Stabilization of the spine was carried out by a full thickness bone graft taken from the iliac crest with both inner and outer tables of the crest.

Bone grafts were shaped and inserted between 2 vertebral bodies, which were judged free of disease, above and below the location of original lesion. To insure the stability of the graft a transverse through was made into the vertebral bodies to receive the graft, after mild distraction of vertebral bodies in order to countersink the bone graft.

Post operatively, the spine was immobilized with plaster cast and patients were allowed to ambulate as soon as fusion could be demonstrated clinically by lack of abnormal motion at the operative site and confirmed radiologically.

Antituberculosis therapy was continued until sound fusion was demonstrated. This occurred between 4-6 months post

Name	Duration	Level	Neurologic Status	Paravertebral Abscess	
				Clinical Evidence	Finding of operation
I.A 35	6.M	L <sub>1</sub> L <sub>2</sub>	Weekness lower extremities	Present	Present
T.N 35 <sup>dge</sup>	8.M	T <sub>9</sub> T <sub>10</sub>	Spastic Paraplegia	Present	Present
A.B 20	10.M	T <sub>1</sub> T <sub>2</sub>	Spastic Paraplegia	Present	Present
J.H 62	6.M	T <sub>6</sub> T <sub>7</sub>	Spastic Paraplegia	Present	Present
H.A 45	12.M	T <sub>10</sub> T <sub>11</sub>	Spastic Paraplegia	Present	Present
A.S 45	12.M	T <sub>10</sub> T <sub>11</sub>	Spastic Paraplegia	Present	Absent
S.R 38	2.M	T <sub>10</sub> T <sub>11</sub>	Spastic Paraplegia	Present	Absent
Z.TCH 50	2.Y	T <sub>10</sub> T <sub>11</sub>	Flacid P no bowel and bladder Control	Present	Present
H.SH 17	12.M	T <sub>3</sub> T <sub>4</sub>	Flacid P no bowel and bladder Control	Present	Absent
SH.A 45	6.M	C <sub>5</sub> C <sub>6</sub>	S. Quadriplegia	Present	Present
M.D 70	8.M	T <sub>11</sub> T <sub>12</sub>	Incomplete S.P	Present	Present
H.J 40	12M	T <sub>8</sub> T <sub>9</sub>	Flacid P no bowel and bladder Control	Present	Present
M.B 46	6.M	T <sub>6</sub> T <sub>7</sub>	Spastic Paraplegia	Present	Present
I.A 40	2.Y	T <sub>8</sub> T <sub>9</sub>	Spastic Paraplegia	Absent	Absent

Neurologic Status At last Visit	Clinical Fusion At last visit	Radiological Fusion At last visit
Complete Recovery	Solid	Solid
Complete Recovery	Solid	Solid
Complete Recovery	Solid	Solid
Complete Recovery	Solid	Solid
Walking With Crutches	Solid	Solid
Walking With Crutches	Solid	Solid
Complete Recovery	Solid	Solid
Recovery of bowel and blader control S.P Wheel Chair	Solid	Solid
Complete Recovery Upper extrimity walking with crutches	Solid	Solid
Complete Recovery	Solid	Solid
Recovery of B+B Control, spastic.P Wheel Chair	Solid	Too early (under 6/12)
Recovery of voluntary Motion, not ambulated yet	-	-
Recovery of voluntary Motion, not ambulated yet	-	-

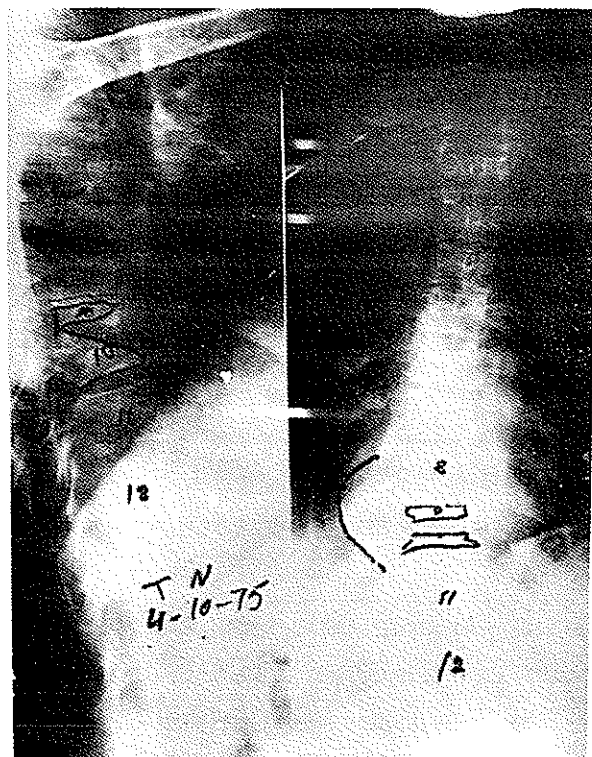


Fig. 1

T.N. 45.F. Spastic paraplegia of 8 months duration there is collups of  $T_9T_{11}$ , shadow of abcess all AP is evident.

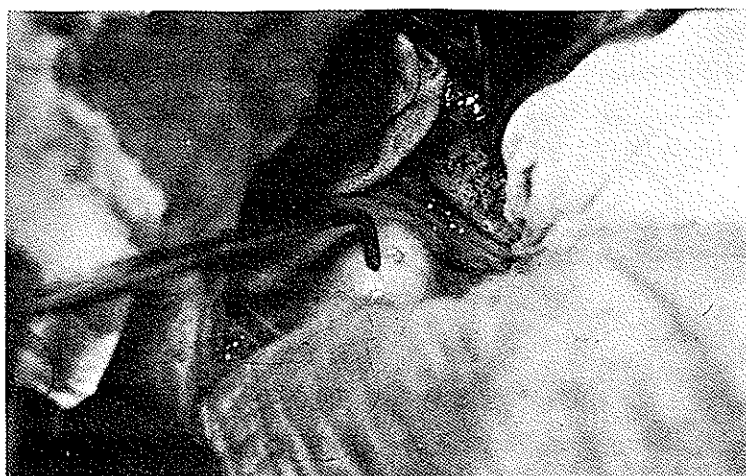


Fig. 2

Same patient as figure 1 on surgery a bulging abcess was found.



Fig. 3

After evacuation of abscess and all devitalized material.

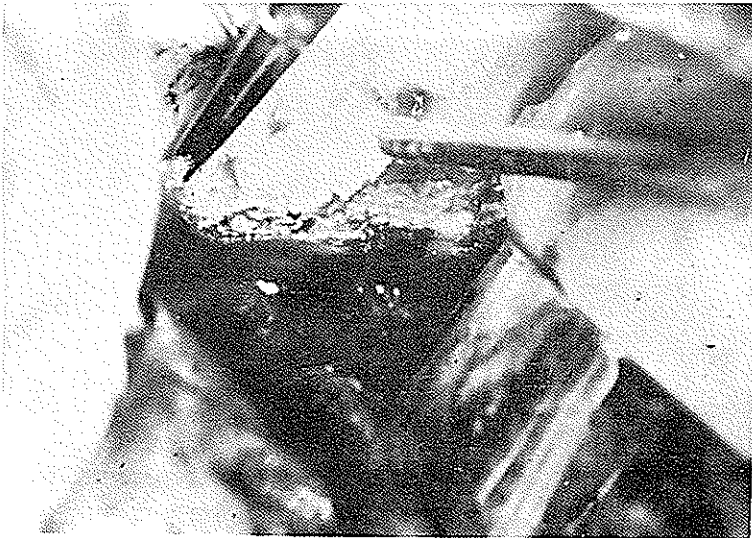


Fig. 4

Bone graft bridging T<sub>8</sub> to T<sub>11</sub> thinp. thad full recovery.

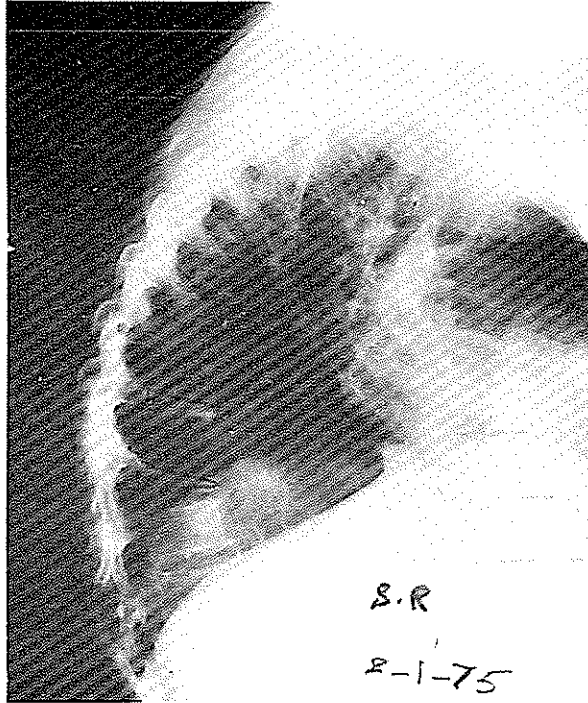


Fig. 5

Spastic paraplegia of 2 months duration with a history of 10 months back pain, patient had been known to have ankylosing spondylitis, destruction of body of  $T_9T_{10}$ .

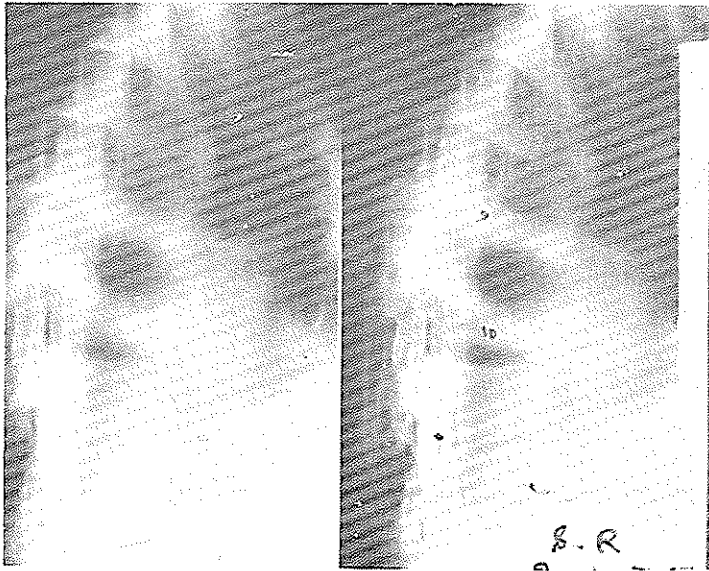


Fig. 6

Myelography of the same patient acomplete block of  $T_{10}$  level is seen.



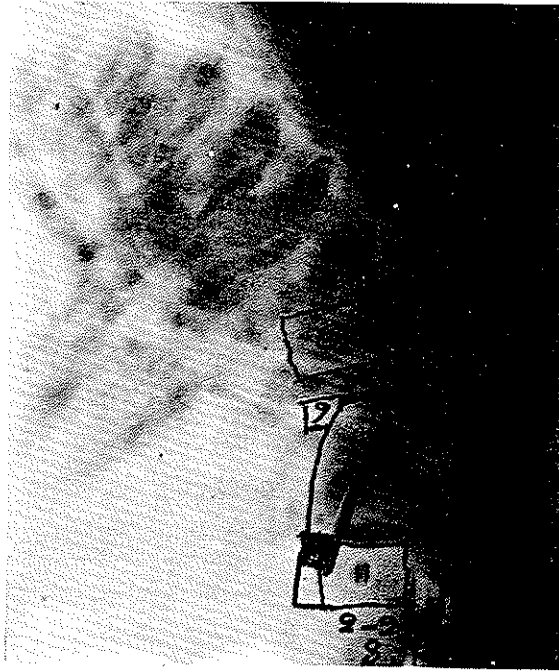


Fig. 7

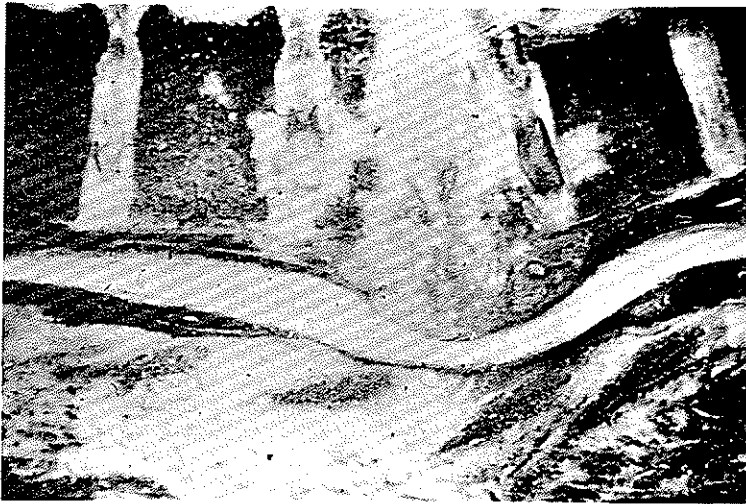


Fig. 8

After radical debridement bone graft is bridging  $T_9$  to  $T_{11}$ , no abscess was found at surgery but the spinal cord was compressed anterior by dense fibrous tissue and bony sequestrated the patient had full recovery.

operatively. Plaster casts were only discarded after radiological evidence of fusion between bodies of the vertebrae as recommended by Pott.

### Discussion

Hodgson et al following their observation after surgical treatment of Pott's paraplegia, discussed the etiological reasons for the clinical symptoms and divided the causes of cord compression into intrinsic and extrinsic causes related to different factors occurring active disease or healing stage.

Extrinsic causes include, during active disease, abscess formation (fluid or caseous material) granulation tissue, sequestered bone, and disc material, and pathological subluxation and dislocation of vertebrae with mechanical compression of the spinal cord. During or after healing, fibrosis of the dura and posterior longitudinal ligament, transverse ridges of bone, or dense fibrous tissue anterior to the spinal cord with compression of the cord were responsible.

Amongst intrinsic causes mention should be made of the passage of tuberculous inflammation across the dura to involve the meninges and eventually, the spinal cord with associated tissue destruction. Rare causes include infections, thrombosis and spinal tumour syndrome described by Seddon.

Most of the above etiological factors were met with in our cases. Operations by the above mentioned approach gave good results as regards reduction of pain and duration of patient's stay in hospital. Prognosis as regards neurological status was improved after operation and most patients became ambulant and independent. It is argued that such an operation in appropriate cases should be performed as early

as possible and the cause of the spinal cord compression found and corrected as thoroughly as possible and mechanical stability of the spine achieved as described.

#### Conclusion and Summary

14 cases of pott's disease of the spinal column treated by surgery is described. The operation was performed by an anterior approach and evacuation of abscess, removal of granulation tissue, sequestered bone and disc material achieved. Any mechanical compression by bony ridges, dense fibrotic tissue or fibrotic posterior longitudinal ligament was removed making it possible for anti TB treatment to have full effect.

Stability of the spine was achieved by means of anterior bone grafts which accelerated bony fusion and eradication of the disease.

It is stressed that this type of radical surgery in pott's allows for histological diagnosis, corrects the deformity thus preventing future cardio-respiratory complications, prevents paraplegia of late onset, and shortens the duration of hospital stay allowing the patient to return to his previous occupation.

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