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The neuropathological foundations for the restorative neurology of spinal cord injury

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ABSTRACT

An appreciation of the neuropathology of human spinal cord injury (SCI) is a basic requirement for all concerned with the medical treatment of patients with SCI as well as for the many neuroscientists devoted to finding a "cure". An understanding of the neuropathology of SCI is a necessary guide to those concerned at all levels of treatment, whether they are doctors or other health professionals. The underlying changes in the spinal cord are especially relevant to the restorative neurology (RN) of SCI. The new discipline of RN seeks to enhance the function of residual spinal cord elements which have survived the injury and so improve the patient's rehabilitative status. This is in contrast to the conventional approach in rehabilitation which works around the clinical neurological deficiencies. Following the injury a series of changes take place in the spinal cord and surrounding tissues which continue to evolve throughout the life of the patient. In flexion and extension injuries resulting from motor vehicle trauma, diving and sporting accidents the spinal cord is compressed and disrupted but usually with some continuity remaining in the white matter columns. The brunt of the injury is usually centrally placed where there is bleeding into the disrupted grey matter involving one two segments, usually cervical. The loss of central grey matter is nowhere near as important as is the tearing apart of the white matter tracts in determining the patient's clinical state. The central grey matter supplies one two overlapping segmental myotomes and sensory fields. In contrast loss of continuity in the long white matter tracts is catastrophic because all functions below the level of injury are affected, autonomic or voluntary either by paralysis or anaesthesia, usually both. It is important to determine the exact nature of the injury in every patient as a preliminary to treatment by RN. This assessment is both clinical and neurophysiological with special attention given to any part of the long white matter tracts which may have escaped the initial injury. It is these residual nerve fibres which provide the opportunity to improve the patient's neurological state by being re-activated, modulated and enhanced by stimulation or by other RN methods. The conversion of a clinically complete SCI patient to being incomplete and ambulant is a tremendous improvement in the patient's status. It is the purpose of this article to provide the reader with the essential neuropathology of SCI as a beginning point in planning treatment whether it is medical or ancillary, as well as to inform the neuroscientist about the condition being addressed in his or her research.

1. The essential neuropathology of human spinal cord injury

The neuropathology of SCI described here is derived from the post-mortem examination of more than 220 patients who died, after various periods of survival with a spinal cord injury (Table 1). Each case has been examined in detail both macroscopically and microscopically. Following the routine somatic necropsy the spinal column was removed *in toto*, the site of injury examined, photographed and fixed in formalin. After fixation the laminae are removed exposing the injured spinal cord which is then carefully removed examined and photographed. Transverse sections are taken from the level of injury and from segments above and below, for microscopic examination. The neuropathology of SCI which follows below is an amalgamation of the total experience gained from the study of each individual case condensed into narrative for the convenience of the reader [1].

Two hundred and twenty two cases were examined and classified as being acute or chronic according to their survival time which for this purpose is defined as more or less than one year. Table 1 shows how many cases were clinically and anatomically complete i.e. showing complete dehiscence of the spinal cord. The clinically incomplete cases had some continuity of white matter crossing the lesion with partial retention of voluntary motor function and/or sensation below the injury. The clinically discomplete cases are those who had no sensation or

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Table 1
SCI case data

SCI Type	Acute	Chronic	Total
Complete	16	23	39
Incomplete	20	24	44
Discomplete	23	30	53
Unknown	69	15	84
Total	128	92	220

motor control below the level of the lesion but who had retained some continuity of white matter (axons) crossing the lesion, the number of discompletes being about 25% of the total. It is the discomplete group of patients who would benefit greatly from treatment by restorative neurology.

2. The natural history of SCI

After injury the spinal cord lesion passes through the stages of acute, subacute and chronic. However it should be kept in mind that the neuropathological changes are different in each SCI so that the description which follows is generalised. It is emphasised that the spinal cord is disrupted at the moment of injury. Although further changes occur later due to the body's reaction to the injured tissues it is important to recognise that most of the damage is due to the immediate trauma. As a note of caution, in our experience unfortunately, the spinal cord injury was in several instances aggravated by poor handling of the patient at the scene of the accident or while in hospital. An unrecognised fracture of the vertebral column with associated instability may lead to severe injury to a previously intact spinal cord (Table 2).

The central grey matter of the spinal cord is more severely affected than are the white matter columns in the usual flexion and extension injuries (Figure 1). The grey matter with its rich capillary bed is disrupted and haemorrhagic. In contrast the white matter columns tend to be spared extending from the periphery to the centre. It is important to note that in the majority of SCI patients who have suffered their spinal injury resulting from a motor vehicle or diving accident there remains some white matter with myelinated axons traversing the lesion (Figure 2).

The spinal cord is disrupted to a greater or lesser degree by the trauma arising from the mechanical forces involved. It is emphasised here that the initial spinal cord injury is a physical event and is at its maximum at once. The microscopic changes in spinal cord which follow the injury are stereotyped conforming to the usual pattern found in the CNS. In roadside deaths the spinal cord and meninges are haemorrhagic and appear torn apart. However if the patient lives beyond a few hours on, the tissues being no longer viable undergo necrosis. Cellular debris is removed by macrophages followed by glial scarring. Within hours of injury, polymorphs are attracted into the necrotic area by cytokines released from the injured tissues. At this stage "secondary damage" becomes apparent but this is little more than the necrosis. However enzymes derived from the inflammatory cells promote liquefaction of the dead tissues. Attention has been given to this secondary damage by the experimentalists striving to limit its effect.

Fig. 1. A case demonstrating the brunt of the injury affecting the central grey matter which is haemorrhagic while leaving the white matter columns relatively intact.

Fig. 2. Spinal cord and vertebral column in longitudinal section showing that the spinal cord is not severely damaged despite a fracture through the disc in a hyperextension injury.

Table 2
Details of seven patients who suffered spinal cord damage sometime after the initial vertebral trauma.

Case	Neuropath	Age (years)	Sex	Accident	Reason for deterioration	Level	Cord Pathology
1	B62/4362	57	F	Fall	Fracture not recognised	C6/7	Focal Necrosis
2	B63/13	56	M	MVA	Fracture not suspected	Odontiod Cord "intact"	
3	A66/323	27	M	MVA	Transport and Lifting	T11/12	Gliosis
4	A70/487	55	F	MVA	Transport and Lifting	C6/7	Gliotic Cysts
5	X79/142	25	M	Mcycle Collapsed waking	T8	Severed	
6	X84/105	16	M	Diving Fracture not suspected	C5	Gliotic with NRR	
7	X89/274	64	M	MVA	Fracture not suspected	C5/T4	Compression