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Bodysurfing injuries of the spinal cord

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In a group of 104 patients paralysed as a result of injury while swimming or diving, 3 patients were identified in whom the injury was sustained during bodysurfing. The mechanism of the injury and the clinical and radiological findings in this group differ markedly from the findings in the 101 patients paralysed after diving into shallow water. The 3 patients were significantly older with a mean age of 46 years. No fracture or dislocation of the cervical spine was present, but evidence of osteo-arthritis was present in all cases. The pattern of spinal cord injury was that of incomplete paralysis consistent with the central cord syndrome. This combination of findings suggests that the mechanism of injury was forced hyperextension of the head and neck due to the surfers having been caught up in turbulent wave action and driven into the sandy sea bottom.

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In two previous analyses^{1,2} of patients paralysed as a result of diving injuries to the cervical spinal cord, a small, distinct, sub-group of older patients who had sustained spinal cord injuries while bodysurfing was identified. The circumstances and consequences of injury in this small group of patients differ in several significant respects from those of patients paralysed as a result of diving into shallow water. There is only one other report in the literature of cervical spinal cord injury caused by bodysurfing³ and an analysis of this group has therefore been made in order to define further the mechanism of injury, orthopaedic injury, neurological deficit and preventive measures.

Analysis of clinical and radiological findings

Out of a total of 104 patients paralysed as a result of water-related accidents,^{1,2} 3 were identified who had sustained their spinal cord injury while surfing in the waves, and not while diving into shallow water. The ages of the 3 patients were 42, 44 and 52 years, the mean age being 46 years. All 3 provided similar histories of having been caught up in turbulent wave action and driven into the sandy bottom. All 3 had sustained neurological injury with incomplete paralysis consistent with central spinal damage. None had sustained any fracture or dislocation, but all 3 showed evidence of osteo-arthrotic changes in the cervical spine. None had consumed alcohol prior to entering the sea.

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Fig. 1. Marked disc space narrowing at the C5/C6 level. A large posterior osteophytic spur (arrow), protruding into the spinal canal, is present at this level.

Discussion

This analysis has revealed marked differences in the findings between the two groups of patients analysed, i.e. the 101 patients paralysed because of diving into shallow water and the 3 patients injured as a result of surfing.

Those injured as a result of diving accidents were predominantly young, with a mean age of 22 years, while the surfing accident patients were significantly older, with a mean age of 46 years.

While the diving accident patients showed severe orthopaedic injuries, the most common being 'tear-drop' fractures of the vertebral bodies, the surfing group showed no radiological evidence of orthopaedic injury.

Neurological deficit associated with the orthopaedic injuries in the diving group was severe, with 65% of patients sustaining complete, permanent quadriplegia. In the surfing group, all 3 patients were fortunate to have sustained only incomplete paralysis consistent with the central-cord syndrome. This results from haemorrhage into the central grey matter of the spinal cord, with a varying degree of white matter involvement which severely affects the centrally located arm tracts and, to a lesser extent, the leg tracts. The central haemorrhage damages the anterior horn cells over several segments, producing a lower motor neuron paralysis of the hands and arms. The peripheral haemorrhage into the white matter results in weakness and spasticity of the legs. There is usually loss of voluntary bladder control, urinary retention, and some degree of sensory loss.⁴

While the diving patients showed no evidence of any significant osteo-arthrosis of the cervical spine, all 3 of the surfing injury patients had well-marked changes of osteo-arthrotic disease of the spine. Posterior osteophyte formation at the C4/C5 and C5/C6 levels was present.

This combination of pre-existing osteo-arthrotic changes of the cervical spine, spinal cord injury without orthopaedic injury and the onset of the central cord syndrome all suggest that the mechanism of injury was forced hyperextension.

The majority of these injuries⁵ occur in the presence of cervical spondylosis in which the cord is pinched anteriorly between the degenerate vertebral discs and osteophytes and posteriorly between protruding redundant folds of ligamentum flavum. There are usually no radiological signs of injury and only the presence of changes of cervical spondylosis are observed.⁶

The cervical spinal cord in the lower cervical spinal canal (C3 - C6) is especially liable to trauma in extension, as the subarachnoid space around the cord (in the sagittal diameter) is narrowest in this region. On extension the available space is diminished still further as the spinal cord itself increases its thickness, while the sagittal diameter of the canal narrows by approximately 2 mm in the normal adult subject.⁷ Additional decrease in diameter due to the intrusion of projecting osteophytic ridges and ligamentum flavum combines with the abovementioned physiological changes in extension to precipitate spinal cord injury.

Apart from functional narrowing of the spinal canal some individuals have congenitally narrow canals that place them at great risk of spinal cord injury. If the sagittal diameter of the cervical canal at any level is 10 mm or less, then any additional narrowing due to trauma or other processes will almost certainly result in spinal cord damage.⁸ With diameters of 10 - 13 mm, a high risk is present.

The circumstances of injury, neurological deficit, age of the patients and absence of orthopaedic injury are similar to the findings reported by Cheng *et al.*³ in their larger series of 14 patients who sustained bodysurfing accidents.

Conclusion

This study has shown that spinal cord injuries due to bodysurfing accidents differ markedly from those sustained as a result of diving into shallow water. The mechanism of these injuries is forced hyperextension of the head and neck due to the surfers being caught up in turbulent wave action and driven into the sandy sea bottom. The presence of pre-existing osteo-arthrosis predisposes these patients to injury of the central portion of the spinal cord, without associated fracture or dislocation of the cervical spine. Middle-aged people are prone to these injuries.

The absence, therefore, of fracture or dislocation of the cervical spine in middle-aged patients presenting with spinal cord injury and paralysis sustained during bodysurfing, should not cause surprise. An exhaustive search for subtle fractures or dislocations, utilising sophisticated imaging techniques such as computed tomography and magnetic resonance imaging, is not indicated. Careful inspection of the plain radiographs for changes of osteo-arthrosis and a narrow spinal canal is required. If either or both of these conditions are present on the plain radiographs, it is

unnecessary to continue with the imaging investigation of these patients, as these examinations will not contribute towards the initial clinical management of the patient.

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100 years ago . . .

We had to cool down before we could write this article. After listening to Dr Beck's address [Presidential address to the Cape Town Branch of the BMA] we became intoxicated with ecstatic visions of a firm, solid medical organisation, embracing our profession in all South Africa, gathering in our wanderers in its remotest parts as civilization advances, and firmly holding within to itself, and without to the public, those ideal relationships which have been so often pictured but never realized. Waking, we imagined all the good fellowship it might bring about; sleeping, we dreamt of how it might be discharged like a load of bricks on the head of offenders against its laws, worked out, as we confidently trust they will be, in the spirit of unselfishness and of justice. We believe that as we listened we heard the first step of a great progress, tentative and uncertain yet, it is true, but nevertheless coming audibly nearer each day. It is difficult to keep sober in prophecy (*sic*) on such a theme; but since then we have put our heads under the cold douche of practical facts, and, though we have lost thus none of the warm enthusiasm stirred in us by the vision called up, it has somewhat contracted and concentrated into a problem of ways and means. We have remembered that the skeleton is not alive till it is clothed with the flesh of things accomplished, and has a heart beating and blood circulating steadily. That it can be so vitalized, if we set about it cautiously but untiringly, we feel morally certain.

(Editorial, *S A Medical Journal*, October 1895, p. 171.)

PHYSIOLOGY FOR PHYSICIANS

The activated neutrophil — formidable forces unleashed

R. Anderson

The human neutrophil is the small, aggressive, front-line, circulating phagocyte. The enormous intrinsic destructive potential of neutrophil is captured in the following quotation, which dramatically emphasises the critical requirement for latency and self-limiting activation of these cells:

'Unlike cytotoxic lymphocytes and the complement system, which destroy their targets with a drop of poison, the professional phagocytes kill like Attila the Hun, deploying a battery of weapons that lay waste to both the target and the nearby landscape with the subtlety of an artillery barrage.'

In this review recent advances in neutrophil physiology and function, as well as mechanisms of inflammation-related tissue injury and carcinogenesis, are highlighted.

Neutrophil production

About 55 - 60% of bone marrow is dedicated to the production of one cell type — the neutrophil, the most abundant professional phagocyte.² During maturation in the marrow the neutrophils undergo progressive differentiation and loss of biosynthetic activity. After the myelocyte stage these cells become end-cells and enter a large storage pool from which they are released into the circulation after about 5 days, where they have a half-life of about 6 hours.² Although they are end-cells, mature neutrophils retain some residual biosynthetic capacity which is activated on exposure to leuco-attractants or the cytokines GM-CSF and TNF,³ enabling limited adaptation to a changing micro-environment. The steady state production of mature neutrophils is about 1×10^9 /kg body weight per day, but this is dramatically increased by co-operative interactions between leucocytosis-promoting cytokines (TNF- α , IL-1, IL-3, IL-6, IL-8, G-CSF and GM-CSF) and leuco-attractants (C5a, PAF and LTB₄).⁴

Neutrophil cytoplasmic granules

The most notable structural features of the neutrophils are the abundant, heterogeneous cytoplasmic granules and the highly dynamic plasma membrane; these make this cell ideally suited to the performance of its primary function, viz. adherence to locally activated vascular endothelium, extravasation, rapid migration to sites of infection and engulfment and intracellular destruction of invasive microbial pathogens.

With regard to function, the plasma membrane is equipped with adhesion molecules, receptors for leuco-attractants, opsonins and cytokines, as well as the unique

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