

LETTERS TO THE EDITOR

Sensory anal examination in spinal cord injury

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I enjoyed reading the paper by Krogh *et al.*¹ I would recommend the addition of sensory anal examination to their physical examination. This would involve superficial anal examination with cotton and pinprick and asking about the amount of sense perceived on both the right and left sides. Comparison between both sides and with areas of normality is recommended. Observation of anal wink during sensory examination is a helpful test to see the tone of the anus. For example, if the examiner asks the patient to contract the anal sphincter and the patient answers that he/she cannot do so, but an anal wink is seen during the pinprick stimulation, it shows that tone is present. The bulbocavernous reflex and observation of the

anus during a gentle pinch of glans penis should also be performed.

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Re: Case report: A fatal metastasis of Klebsiella pneumonia to the lungs

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Dr Frisbie presents a case of a 61-year-old tetraplegic who developed a respiratory tract infection with Klebsiella pneumonia, which he suggests migrated to the lungs and caused an overwhelming infection from which the patient died. He suggests that the origin of pulmonary infections can be extra-pulmonary.

There is a very rich literature on the subject, which I feel should be acknowledged in such a presentation.

Charcot in 1877 showed that pressure sores became infected and caused septic emboli in the lungs.

We shall also notice gangrenous emboli. In this variety, thrombi impregnated with gangrenous ichor are transported to a distance and give rise to gangrenous metastases, which are principally observed in the lungs. This is a point upon which Dr Ball and myself have insisted in a work published in 1857. But long before us, and even long before the theory of

embolism had been Germanised, M Foville had expressed his opinion that a considerable number of cases of pulmonary gangrene, observed in the insane, and in different diseases of the nervous centres, are caused by ' the transport into the lungs of a part of the fluid which bathes the eschars of the breech'.²

Wagner who set up a dedicated spinal unit also described the systemic effect of pressure sores.³

'Prior to the opening of centres for the treatment of paraplegic patients, the majority died soon after injury from the effects of renal sepsis. In 1917 Thompson Walker found that 47.2% of the patients admitted with spinal injuries to the King George V military Hospital died from urinary infection 8 to 10 weeks after admission. With the advent of specialised centres during the 1939–45 war, this early mortality was reduced largely due to the better understanding of the management of the paralysed bladder, regular turning and the availability of antibiotic therapy and blood transfusions. If the patient survived the dangerous six weeks immediately after injury they still were liable to die from the long-term complications of low grade infection of



the renal tract, which are chronic pyelonephritis, renal failure, hypertension and amyloidosis. However, despite the advances in treatment there was still an appreciable number of patients dying of acute effects of bacterial infection. The most recent and comprehensive figures are those of Tribe 1969 who found that out of a series of 220 necropsies on paraplegic patients over a 20 year period, 3 died of overwhelming toxaemia and 4 of acute septic endocarditis.'

For this reason, to investigate this problem further, when I was appointed consultant in charge of the Liverpool regional paraplegic centre in 1965, I carried out repeated blood cultures on the following:

- All patients who at the time of admission had pressure sores, during the early dressing and operative inference with these sores and on any patient whose sores had deteriorated;
- (2) patients with presumed severe urinary flare-ups;
- (3) any patient with haematuria, which was suspected to be caused by a traumatized urethra.

Many patients were apyrexial or with sub-normal temperatures. When a positive culture was obtained in these ill patients, it was considered that they were suffering from the systemic effects of these bacteria in their blood stream, this constituting a septicaemia and not a transient bacteraemia. I described my experience in five cases, one of whom had a proteus and a Klebsiella aerogenes infection.⁵

The message in this study is that it should be emphasized that if the diagnosis of a septicaemia is to be established and effective treatment administered, blood cultures should be collected before any antibiotics are instituted as the indiscriminate use of antibiotics in the case of septicaemia may render the isolation of an organism from the blood impossible, while failing to treat the initial focus adequately.

The large number of aspiration pneumonias as a cause of fever in the early stages after spinal injury in patients with paralysed expiratory muscles has been emphasized by Silver (1968), where 18 of 50 patients admitted within a few days of injury had pneumonia diagnosed by chest X-ray. In all, 17 of these 18 patients had paralysed expiratory muscles.⁶

The point at issue is that bacteraemia/septicaemia is not a new phenomena, but had been known ever since spinal cases have been treated with a very high mortality. The clinician should always be on his guard, the blood culture should be carried out before the administration of antibiotics and a chest X-ray is an essential part of the investigation of the patient.

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