INTERCOSTAL NERVE DAMAGE FOLLOWING CLOSED CHEST INJURY

Case Report

BY

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SUMMARY

A case of traumatic intercostal paresis following chest injury is described. It is suggested that this lesion should be suspected when an otherwise unexplained respiratory dysfunction occurs after a severe impact to the chest wall has been sustained. Ventilatory support may be necessary in the early management of the condition, especially when hypovolaemic shock is also present.

Closed chest injuries may cause respiratory inadequacy in a number of ways. These include damage to the bony thoracic cage, ruptured diaphragm, haemothorax, lung contusion, pneumothorax and surgical emphysema.

Two recent patients showed an unexplained reduction in normal inspiratory expansion on the side of the chest which sustained a direct impact. In the absence of any more obvious cause this was originally ascribed to a lesion in the rib cage or lung not radiologically demonstrable. It was later suspected that weakness or possibly paralysis of the intercostal muscles might be responsible.

Unfortunately, owing to other severe injuries the first of these patients did not survive for long and a more precise diagnosis was not established. An account of the second patient whom it was possible to study electromyographically is presented here.

CASE REPORT

A male patient aged 19 was admitted to hospital following multiple injuries sustained in a rail accident. He had been trapped in the wreckage for 24 hours, lying on his left side with his arms extended above his head. He was shocked but fully conscious and complained of severe pain on his left side with his arms extended above his head. A repeat chest film showed clear lung fields and no fractured ribs. On both sides the diaphragm was held in the normal inspiratory position as the film was taken.

Following initial resuscitation he was taken to theatre for immediate exploration and toilet of his axillary wound, suture of superficial lacerations and immobilization of his fractured humerus under general anaesthesia. Because of cyanosis when breathing room air and diminished movement of the left side of his chest the anaesthetist used controlled ventilation with an oxygen-rich mixture during the operation. Blood-gas estimations were not available at this stage.

After operation a decision was made to maintain the patient on IPPV. This was justified on the grounds of his pre-operative cyanosis, the diminished expansion of his left lung and the need to provide an adequate degree of relief from the pain and distress of spontaneous ventilation. Tracheostomy was performed and auffed tracheostomy tube was inserted.

The patient was transferred to the intensive care unit where he was ventilated at a rate of 12 l/min with 40 per cent oxygen in air. Intermittent doses of morphine 15 mg were employed for analgesia and sedation. IPPV was maintained throughout the next 24 hours. If it was discontinued he became cyanosed and distressed by the effort of breathing. Because of technical problems blood-gas estimations were not yet available.

In the succeeding 24 hours he was able to sustain satisfactory spontaneous ventilation for increasing periods. At 52 hours after injury, breathing room air unassisted, his blood estimations showed: Po, 89 mm Hg; Pco, 37 mm Hg; pH 7.42. It was noted, however, that the left side of the chest was still not moving as much as the right and there was a slight indrawing of the intercostal spaces. By now there was relatively little pain on inspiration. A repeat chest film showed clear lung fields and no fractured ribs. On both sides the diaphragm was held in the normal inspiratory position as the film was taken.

An electromyographic examination was requested and this was carried out on the 3rd post-accident day. The report stated:

"A concentric needle electrode was used to explore the 5th, 7th and 9th intercostal muscles on the right and left side. On the left side there was no spontaneous activity and no potentials were seen to recruit even when the patient was hyperventilating. On the right side normal appearing units were present waxing and waning with respiration.

"There is no evidence of any denervation potentials but the time lapse since the accident is too short for such activity to appear. What could be said, therefore, is that there is no electrical activity in the left-sided intercostal muscles. It seems unlikely that this total suppression of activity could be on an entirely voluntary basis and it therefore appears probable that some nerve lesion is responsible."

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The intercostal weakness, although diminishing especially in the upper part of the chest. A repeat e.m.g. was performed on the 22nd day after the original incident. The report read:

"A concentric needle electrode was used to explore the 5th, 7th, and 9th intercostal muscles on the left side. There was some spontaneous fibrillation and positive sharp wave activity. Volitional activity was, however, evident during respiratory movements and on attempted hyperventilation there was an increase in the amount of activity though a full interference pattern was not produced. There is certainly evidence of damage to the intercostal nerves though the return of some volitional activity suggests that the damage has been partial."

Owing to the widespread area of skin abrasion over the left side of the chest it was not possible to test sensation satisfactorily in the early stages. As the abrasion healed, no gross sensory deficit could be elicited. Initially there was severe pain on this side, presumably due partly to the skin damage, and it would therefore appear that motor conduction was affected more than was sensation.

On the 23rd day he was discharged to be followed up in a centre nearer to his home town. There was still some slightly diminished movement in his upper left intercostal muscles, but in spite of this he achieved a vital capacity of 4.3 l. without difficulty. Six months later he was reported to have made satisfactory progress following further orthopaedic and plastic procedures. His brachial plexus damage had almost fully recovered spontaneously and there was no evidence of any residual chest lesion.

DISCUSSION
The actual mechanism of the nerve injury is a matter of speculation. The intercostal nerves are well protected against direct external injury. It is known, however, that impact to the chest can cause considerable temporary deformation in the bony cage without producing a fracture, especially in young people, and thus may bring about a traction injury. In the case described the fact of being trapped in an abnormal position may well have been a contributory agent. Haemorrhage into and around the nerve could also be a factor.

Some of the initial apparent respiratory inadequacy may have been associated with the reduced pulmonary blood flow and increased physiological deadspace known to occur in hypovolaemic shock (Brooks, 1967). These factors result in arterial desaturation when breathing air and lead to increased ventilation. Temporary partial denervation of some of the intercostal muscles would reduce the ability to respond to this hyper-ventilatory drive and thus give rise to a relative respiratory insufficiency.

Campbell (1966) has pointed out that too much attention should not be paid to the presence or otherwise of gross damage to the thoracic cage when in fact the damage to the underlying lung may be more serious from a functional standpoint. Our patient was fortunate in that there appeared to be minimal damage to the lung parenchyma as observed from radiological and blood oxygen studies. Had more severe contusion occurred it is unlikely to have resolved so rapidly. The presence of such damage or pre-existing lung disease would undoubtedly have affected the outcome.

It is interesting to note that in neither of the cases seen were there any fractured ribs. In both the condition occurred on the left side: presumably it can occur on either side or possibly bilaterally.

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REFERENCES

ENDOMMAGEMENT DU NERF INTERCOSTAL APRES TRAUMATISME A THORAX FERME: DESCRIPTION D'UN CAS

SOMMAIRE
L'auteur décrit un cas de paralysie intercostale après traumatisme thoracique. Il suggère qu'une telle lésion doit être soupçonnée, lorsqu'une dysfonction respiratoire non-expliquée se manifeste après coup grave contre la paroi du thorax. Le soutien de la ventilation peut être indispensable dans le traitement précoce du patient, surtout en présence d'un choc hypovolémique.

SCHADEN AN INTERCOSTALNERVEN NACH GESCHLOSSENER THORAXVERLETZUNG: BERICHT ÜBER EINEN FALL

ZUSAMMENFASSUNG
Es wird ein Fall von traumatischer intercostaler Lähmung nach einer Thoraxverletzung beschrieben. Es wird nahe gelegt, daß man an eine derartige Verletzung denken soll, wenn nach einer ernsten Verletzung der Brustwand Atmungsstörungen auftreten. In diesem Fall kann eine künstliche Beatmung notwendig werden zur Frühbehandlung, besonders bei gleichzeitig bestehendem hypovolämischen Schock.