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# Bilateral diaphragm weakness

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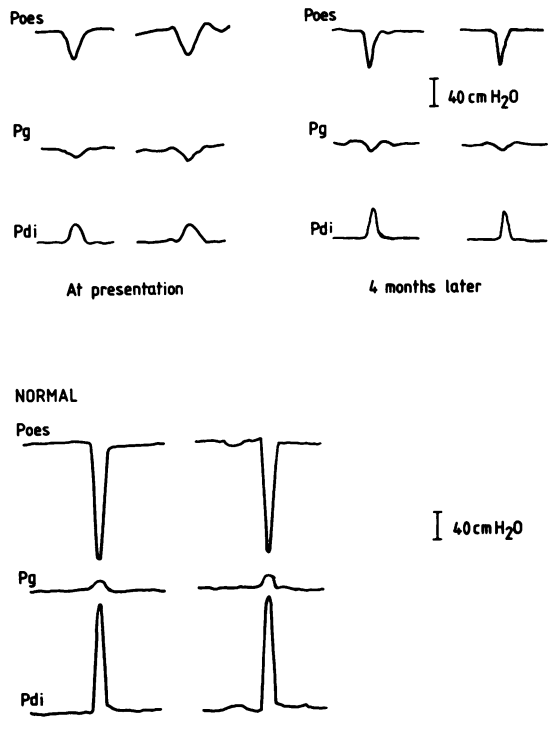
Paralysis of the diaphragm occurring as a result of damage to the phrenic nerves during thoracic and neck surgery is well recognised.[1 2] It is likely that weakness, as opposed to complete paralysis, of the diaphragm could occur after less severe trauma to the phrenic nerves. We describe a patient who developed bilateral diaphragm weakness after complicated surgery on a cervical disc, with subsequent improvement.

## Case report

A 62 year old concert pianist was referred with a one week history of dyspnoea after cervical disc surgery. He had been well and active until September 1983, when he developed a staggering gait, paraesthesiae, and weakness of both arms, with urgency of micturition. A myelogram showed a cervical disc lesion at C3/4 level. His neurological condition deteriorated so that he could no longer play the piano and increasing spasticity of both legs eventually made him immobile. At no time during this period did he complain of breathlessness. Surgery was undertaken to relieve pressure on the cervical cord by an anterior approach. During the procedure the vertebral artery was accidentally punctured, so that extensive exploration of the neck was needed. The following day the patient developed acute respiratory failure and required ventilation for 24 hours. A week later he was still dyspnoeic and was referred for further assessment.

He was found to have residual pyramidal weakness of the lower limbs, but was able to walk with ease and could now play the piano without difficulty. He was orthopnoeic, and on careful examination there was slight paradoxical inward inspiratory motion of the anterior abdominal wall when he was supine and breathing quietly. A chest radiograph was normal, and diaphragm screening in the supine position was assessed as showing "slightly diminished movement of both domes with some paradoxical movement of the lateral part of the left dome on sniffing." The vital capacity was low at 2.0 l (predicted 3.2–4.2 l), and it fell by 30% to 1.4 l when he was supine. Maximum static respiratory pressures were low, with inspiratory ($PI_{max}$) and expiratory ($PE_{max}$) values of 10 and 40 cm $H_2O$ (normal greater than 44 and 80 respectively).[3] Transdiaphragmatic pressure (Pdi) was measured with balloon catheters.[4] The Pdi developed during a maximal static inspiratory effort (Pdi $PI_{max}$) was 22.5 cm $H_2O$, at the lower limit of the normal range (NR 18–137 cm $H_2O$.)[5] The Pdi developed during a maximal sniff, however, was considerably reduced, with a mean of 21.5 cm $H_2O$ (n = 20) compared with the predicted normal for men of 112–204 cm $H_2O$[6] (fig), confirming bilateral diaphragm weakness.

Four months later the patient's symptoms of dyspnoea and orthopnoea had improved and his vital capacity had increased to 2.8 l while he was standing, falling by 25% to 2.1 when he was supine. $PI_{max}$ was 32 cm $H_2O$, while $PE_{max}$ was now normal at 92 cm $H_2O$. Pdi $PI_{max}$ had risen to 40 mm $H_2O$, well within the normal range. Although maximal sniff Pdi had increased to 34.5 cm $H_2O$ (n = 20), this was still considerably below the normal lower limit of 112 cm $H_2O$.




*Changes in oesophageal (Poes), gastric (Pg), and transdiaphragmatic (Pdi) pressures during two maximal sniffs, in the seated position, at presentation (top left) and after four months (top right). Records of two sniffs in a normal subject (bottom) are also shown for comparison.*

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## Discussion

Paralysis of the diaphragm may be suspected in a patient who develops dyspnoea and orthopnoea, with paradoxical inward inspiratory motion of the anterior abdominal wall,[7 8] and in whom the vital capacity falls appreciably in the supine position. In our patient, however, the physical signs were more subtle. Radiological assessment was unhelpful. He showed reduction in vital capacity when supine of 30%, which was outside the range for normal subjects (less than 25%),[9] but not as great as the range previously reported for patients with complete diaphragm paralysis (35–97%).[7] His maximum static pressures were impaired, suggesting some respiratory muscle dysfunction; but these tests depend on the patient's cooperation as well as on cheek and mouth muscle strength, and are not specific for the diaphragm. It has been suggested that diaphragm strength may be assessed by measuring Pdi developed during a maximal static inspiratory effort. The range of normal values, however, is wide (18–137 cm $H_2O$),[5] and by these criteria our patient's results were normal. By contrast, Pdi developed during a maximal sniff in normal subjects has better defined lower limits (112 cm $H_2O$ for men)[6] and should therefore be more sensitive, as seen in this case, in detecting diaphragm weakness in the range between complete paralysis with zero Pdi and normality.[10] There was a 60% increase in our patient's sniff Pdi over the period of four months after his initial assessment.

A high index of suspicion is required to make a diagnosis of respiratory muscle weakness, since it may easily be overlooked clinically. Recording sniff Pdi allows the severity of diaphragm weakness to be measured specifically.

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