

Osteomyelitis affecting 6th and 7th cervical vertebrae.

tuberculosis. Early morning urine specimens showed no evidence of tuberculous infection and the Mantoux was positive only at 1 in 1000. He was treated with cloxacillin 1 g and benzyl penicillin 2 MU six-hourly intravenously for one week, then 1 g and 1 MU respectively intramuscularly for one week, followed by six weeks of penicillin V 500 mg and cloxacillin 500 mg six hourly by mouth. Throughout this time probenecid 500 mg six hourly was given. His neck was immobilised in a stiff collar. Over the next five months the bodies of C6/7 fused completely. He has remained well throughout the subsequent 10 months.

Samples of serum taken at the time of the two admissions were examined for streptococcal antibodies. The antistreptolysin O titre rose from 86 to 310 Todd units (normal <200) and the anti-M associated protein titre rose from 10 to 90-160 (normal <10). The antistaphylococcal titre was <2 units (normal).

Comment

The interesting features of this case are the unusual infecting organism and the presentation with chest tenderness, which in retrospect, possibly represented early cervical spine disease. Nevertheless, the chest tenderness settled very rapidly, and it was one month before the cervical osteomyelitis was recognised. Group C streptococci are found in the throats of 2-3% of healthy adults in Europe and North America. They are generally of the biotype *S. equisimilis*. *S. equisimilis* causes pharyngitis and skin infection and, formerly, puerperal sepsis.^{1,2} In the series of patients with systemic disease due to streptococci reported by Parker and Ball³ group C streptococci were the least common of the easily identifiable streptococci. *S. equisimilis* is a cause of septicaemia and arthritis in 1 to 3-week-old pigs⁴ and septic arthritis has occasionally been reported in man.⁵ Our patient had had no contact with animals. We have found no reports of osteomyelitis due to this organism.

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¹ Hutchinson, R I, *British Medical Journal*, 1946, 2, 575.

² Duma, R J, *et al*, *Medicine, Baltimore*, 1969, 48, 87.

³ Parker, M T, and Ball, L C, *Journal of Medical Microbiology*, 1976, 9, 275.

⁴ Collier, J R, *Proceedings of the American Veterinary Medical Association*, 1951, 88, 169.

⁵ Mayon-White, R T, 1978, personal communication.

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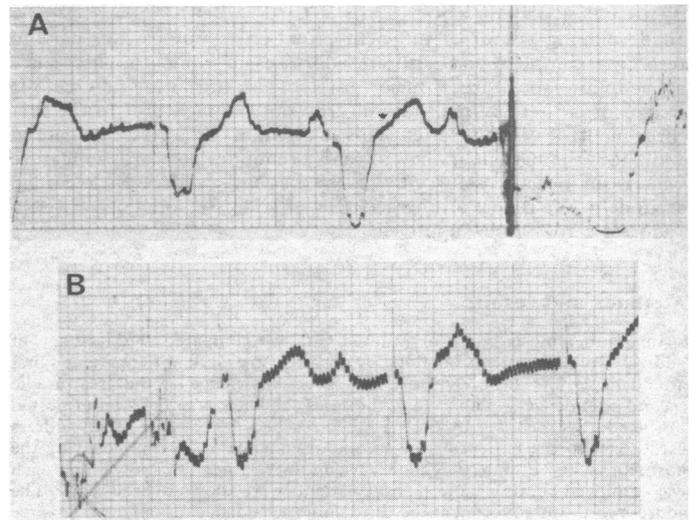
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Electroconvulsive therapy for patient with cardiac pacemaker

There have been few reports of electroconvulsive therapy (ECT) for patients with cardiac pacemakers and none in Britain. The effect of the ECT current on the pacemaker and the possibility of cardiac arrhythmias generate concern about the use of this treatment in depressed patients with pacemakers. We report a case in which treatment was successful.

Case report

A 71-year-old man with a four-year history of recurrent agitated depression was admitted to hospital because he had ceased to respond to adequate doses of amitriptyline. He had physiological features of depression, was agitated and depressed, preoccupied with ideas of unworthiness, self-reproach, remorse, and guilt. His premorbid personality was suggestive of a conscientious, meticulous, and obsessional person. He had had a permanent demand pacemaker (model ELA staniun unit 570 NSM) implanted in April 1977, a month before his admission, for complete heart block diagnosed in 1973. In view of the severity and unresponsiveness of his depression he was treated with a course of five bilateral electroconvulsions, two a week (Ectron model 220V DC × 1.0 sec (c)), and his cardiac function was monitored throughout the treatment (Hewlett, Packard 1511 B). His pulse rate was constant at 70/min. ECG tracings before and immediately after the ECT showed no change in rate or rhythm (figure 1). In comparison a patient of the same age without a pacemaker who had ECT showed typical post-ECT bradycardia. Atropine was not given as premedication because the pacemaker itself eliminated the chances of bradyarrhythmias. He responded well to five treatments, losing all his symptoms. Six months later he was readmitted with a recurrence of his original depressive symptoms. He was once again successfully treated with a course of eight electroconvulsions and imipramine. Monitoring during treatment showed no abnormality.



Electrocardiogram of patient with pacemaker (A) before ECT, (B) immediately after ECT.

Comment

The two main hazards of ECT in patients with pacemakers, the possibility of arrhythmias and the ECT current affecting the pacemaker, have been studied by Youmans and his colleagues¹ using dogs with implanted electrodes. They found that dogs with pacemakers had one-third the arrhythmias of controls. Moreover, all the arrhythmias occurred in dogs with transvenous electrodes. In other reported cases^{2,3} there was no evidence of ECT-induced arrhythmias. On the other hand, the pacemaker provides a form of "built-in" protection against the serious bradyarrhythmias, which account for 30% of all cardiac arrhythmias seen during ECT.¹⁻³

One of the main indications for atropine premedication in ECT is to prevent bradycardia.⁴ Since the pacemaker eliminates this complication premedication with atropine is best avoided. Implanted electrodes seem not to be affected by the ECT current.¹⁻³ Nevertheless, proper grounding to ensure that no current passes over the implanted low-resistance pathway to the myocardium is essential. There should be no contact between the patient and the ground

(for example, improperly grounded monitoring device, an assistant in contact with the patient and the floor). Provided adequate precautions are maintained and means of treating cardiac emergencies are at hand, depressed patients with pacemakers, particularly demand implanted pacemakers, can be successfully treated with electroconvulsive therapy.

¹ Youmans, C R, jun, *et al*, *American Journal of Surgery*, 1969, **118**, 931.

² Blitt, C D, and Kirschvink, L J, *Anesthesiology*, 1976, **45**, 580.

³ Ballenger, J C, *Psychosomatics*, 1973, **14**, 233.

⁴ Cropper, C F J, and Hughes, M, *British Journal of Psychiatry*, 1964, **110**, 222.

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Abdominal and thoracic pressures during defaecation

Hiatus hernia is common in the Western world but said to be rare in the developing countries.¹ Its precise aetiology is unknown. Muller² suggested that raised intra-abdominal pressure when straining at stool might be a causative factor. Burkitt¹ postulated that the fibre-depleted diet of the West results in small hard stools needing greater effort to evacuate than the bulky soft stools associated with the high fibre diet of the poorer countries. Thus the greater straining at stool might create a greater pressure gradient across the diaphragm, eventually pushing up the stomach and producing a hiatus hernia. We investigated this theory by measuring the absolute intra-abdominal and intrathoracic pressures and the gradient between them during defaecation. We also compared pressures in the sitting with those in the squatting position.

Methods and results

Five healthy medical students (four men and one woman) of average build, aged 22 to 24, and not taking any drugs, were studied after an overnight fast. Each sat on a commode, screened for privacy, after pressure-recording tubes had been put into position. Intra-abdominal pressures were measured by a tube which was swallowed and positioned with its tip in the mid-point of the stomach. Intrathoracic pressures were measured by a second tube swallowed and slowly withdrawn while the pressure was constantly recorded. The position of the diaphragm was located by detecting reversal of the respiratory pressure changes and the tip of the tube positioned 10 cm above this point. Both tubes were calibrated before each study and the pressures recorded in centimetres of water. Each tube was of polyvinyl, 120 cm long with a lumen of 1.4 mm. Each had three lateral openings just proximal to the sealed distal end. They were perfused with water at 3 ml/min with a Harvard constant infusion pump. Pressures were measured with a transducer (Bell and Howell) connected to an M-19 multichannel recorder (Devices). After the tubes had been passed and the subject settled he or she was asked to prepare for passing stools, then to strain as hard as possible and attempt to empty the bowels. Three actually passed stools. Each then squatted over a bedpan on the floor and, after the tubes had been repositioned, repeated the effort.

The individual intra-abdominal and intrathoracic pressures are shown in the table. Straining on the commode produced a mean (\pm SEM) intra-abdominal pressure of 195 ± 28.2 cm H₂O and a mean intrathoracic pressure of 67 ± 8.1 cm H₂O. This difference is statistically significant ($P < 0.01$).

Pressures (cm H₂O) within chest and abdomen during defaecation when sitting and squatting. Figures for each individual subject are means of five recordings

Subject	Sitting			Squatting		
	Abdomen	Chest	Difference	Abdomen	Chest	Difference
1	292	94	198	100	60	40
2	213	67	146	138	83	55
3	173	59	114	173	43	130
4	178	45	133	175	45	130
5	120	72	48	75	40	35
Mean (\pm SEM)	195.2 ± 28.2	67.4 ± 8.1	127.8 ± 21.8	132.2 ± 19.7	54.2 ± 7.9	78.0 ± 19.2

Mean pressures when squatting were intra-abdominal 132 ± 19.7 cm H₂O and intrathoracic 54 ± 7.9 . This difference also is statistically significant ($P < 0.05$). Although the pressures and the pressure gradient across the diaphragm were lower when squatting than when sitting on the commode the differences were not significant. The woman had lower pressures than the men.

Comment

This study shows that the intra-abdominal pressure always considerably exceeds the intrathoracic when straining maximally to defaecate. If this pressure gradient across the diaphragm occurs often and for prolonged periods the stomach might gradually be pushed up into the chest. This is consistent with Burkitt's hypothesis that the higher incidence of hiatus hernia in Western countries may be related in part to prolonged periods of straining at hard stools. Squatting for defaecation seems to offer a little protection, since the pressure gradient was less, but the difference was not statistically significant. Martin and Odling-Smee³ found that squatting was no better than sitting in preventing the transmission of intra-abdominal pressure to the leg veins. Hiatus hernia is usually asymptomatic. Symptoms of oesophagitis due to reflux of gastric juice often occur without any detectable upward shift of the cardia. Nevertheless, oesophagitis associated with hiatus hernia causes much ill health: its prevention would be worthwhile. Our findings are consistent with Muller's and Burkitt's hypotheses.

¹ Burkitt, D P, and James, P A, *Lancet*, 1973, **2**, 128.

² Muller, C J B, *South African Medical Journal*, 1948, **22**, 376.

³ Martin, A, and Odling-Smee, W, *Lancet*, 1976, **1**, 768.

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Unusual complication after suprapubic bladder puncture

Suprapubic bladder puncture is a useful method of obtaining uncontaminated urine specimens for bacteriological investigations. Complications are infrequent and usually unimportant. A case of osteomyelitis after suprapubic bladder puncture is reported.

Case report

During a suprapubic bladder puncture in a 53-year-old woman with recurrent urinary tract infections the pubic bone was scratched with the puncture needle. An adequate urine specimen was obtained and *Escherichia coli* were isolated from it. Intravenous pyelography and cystoscopy two and three days respectively after the bladder puncture were normal. On the day of cystoscopy the patient developed septic fever, and *E coli* were isolated from three blood cultures. Gentamicin was given, and the patient became afebrile after six days. Fourteen days later clinical signs of osteomyelitis in the left pubic bone developed. X-ray examination was normal, but two weeks later radiological signs of osteomyelitis were evident. *E coli* were isolated from a bone biopsy. The patient was treated with gentamicin for another six weeks. She made an uneventful recovery. The *E coli* isolated from urine, blood, and bone had similar biochemical identification reactions