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Diaphragmatic paralysis: a difficult diagnosis

Sir,

The aetiology of orthopnoea can occasionally represent a formidable diagnostic challenge to the clinician. We report a case of diaphragmatic paralysis which had been misinterpreted as left ventricular insufficiency. In December 1988, a 72 year old man was hospitalized because of dyspnoea resistant to inotropic, diuretic and vasodilating therapy. The dyspnoea typically occurred during the night and after major effort. The patient gave a history of pulmonary tuberculosis treated by left phrenic nerve crush and lower lobe resection in 1949. The physical examination revealed right basal rales, whereas the left base was at the level of the 9th dorsal vertebra. The electrocardiogram showed left anterior hemiblock and non-specific ST-T changes in the precordial leads. On the chest X-ray, some Kerley B lines were evident in the right lower zone. The echocardiogram revealed mild left ventricular (LV) hypertrophy with a LV ejection fraction of 50%. The arterial tension of oxygen and of carbon dioxide were 72 mmHg and 42 mmHg respectively, the pH was 7.39.

The nocturnal dyspnoea was thought to be an angina equivalent, but neither isosorbide dinitrate and verapamil could prevent it nor were perfusion abnormalities shown by a thallium-201 myocardial scintigraphy. As a moderate rise of the blood pressure up to 170/100 mmHg usually paralleled the occurrence of nocturnal dyspnoea, a pathogenetic role of hypertension was supposed. Despite

an effective antihypertensive treatment, dyspnoea still occurred during the night and occasionally by day if the patient lay down. After 9 days of hospital stay, a nurse noticed that during sleep the patient's abdomen moved outward and inward during expiration and inspiration respectively. Bilateral diaphragmatic paralysis was considered and was confirmed by measurement of the transdiaphragmatic pressure (Pdi). The patient could generate a maximum Pdi of only 2 cm H₂O versus normal values of 10–20 cm H₂O.¹

Unfortunately, extensive investigation did not provide an explanation for the right diaphragmatic paralysis. A neuritis seemed the most likely cause. A chest cuirass was used at night which completely prevented dyspnoea. After about 15 months of such therapy, the patient remains asymptomatic.

Owing to a peculiar compensatory breathing pattern, the physical examination of a patient with a diaphragmatic paralysis may be misleading as may either the fluoroscopic assessment of diaphragmatic motion or the finding of normal or near normal arterial gas values.² The ineffectiveness of cardiological therapy, the dissociation between severe nocturnal and mild effort dyspnoea, the absence of upper lung blood diversion and cardiomegaly on the chest X-ray all contribute to suggest a noncardiac origin of the dyspnoea. Eventually, a consistent fall of the vital capacity and of the arterial tension of oxygen with passage from the seated to the supine posture is highly suggestive of diaphragmatic paralysis.³ Awareness of this condition will enable physicians to care for patients who would otherwise undergo useless investigations and ineffective or dangerous therapy.

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'Spread-eagle' position for the rectal examination of the prostate

Sir,

Rectal prostatic examination is an integral part of a urological physical examination in male patients. Generally, for this purpose, the left-lateral (Sims') or knee-elbow position is advised in British texts,^{1,2} and the standing position (with the patient bent over the end of an examination table and his toes pointed inwards) is preferred in American books.^{3,4}

I routinely use the supine position with the knees brought up and gently abducted ('spread-eagle' position)