REFERENCE


The Diaphragm in SLE

To the Editor:

We were interested to see the recent report entitled "Phrenic nerve function in patients with diaphragmatic weakness and systemic lupus erythematosus" from Wilcox et al (Ches 1988; 93:352-8). Like previous reports, it suggests that diaphragmatic weakness is a common finding in patients with SLE, probably due to a specific myopathy affecting the diaphragm, even in the absence of any generalised muscle disorder. This conclusion was based on the finding of an apparent reduction in transdiaphragmatic pressure during a maximal static occluded inspiration in 11 of 30 women with SLE. It is inherently surprising that the diaphragm should be the only muscle affected by this systemic disorder.

Results reported were compared to a quoted normal range obtained from ten normal male subjects. The normal ranges obtained from larger groups of normal subjects (and more particularly from patients without muscle disorders) are much lower; many normal subjects are unable to generate pressures higher than those reported for SLE patients. In addition, the normal range for women is considerably lower than that for men in all reports. Thus the conclusion that the diaphragm was weak based on the data given in this paper must be questioned.

We also have investigated diaphragmatic strength in 12 patients with unexplained loss of lung volume (vital capacity $54 \pm 11$ percent predicted) associated with SLE, using additional maneuvers such as the maximal "sniff", which most patients find easier to perform to generate maximum pressures (Am Rev Respir Dis 1988; 137:66). We found low mouth pressures values in some patients, but nine of 12 patients had entirely normal sniff maximal transdiaphragmatic pressures. In addition, when measured during bilateral twitch stimulation of the phrenic nerves—a technique independent of patient technique and co-operation—transdiaphragmatic pressure was normal in all patients.

Thus, we did find evidence of diaphragmatic dysfunction in three of 12 patients when assessed using voluntary maneuvers, but normal twitch pressures excluded the possibility of primary muscle abnormality. These results could be explained by a lack of maximal effort or the presence of an upper motor neurone lesion affecting diaphragmatic activation, which seems not unlikely in view of the high reported incidence of cerebral involvement in SLE.

We do not agree that diaphragmatic myopathy is a frequent feature of SLE in the absence of a generalised myopathy or polymyositis.

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To the Editor:

After carefully reading Miller and Staats' article "Impaired Exercise Tolerance after Inferior Vena Cava Interruption" (Ches 1988; 92:776-78), I find their conclusions seriously flawed. Pulmonary exercise testing showed no limitation due to obstructive lung disease, but in all four cases the alveolar-arterial oxygen gradient was widened after exercise, signifying subtle but definite gas exchange abnormalities. No physical findings were presented to support venous stasis with pooling of blood in the lower extremities (which would have to be present to support the hypothesis of inadequate venous return to the heart). It is known that venous collaterals develop after cava ligation; otherwise, venous stasis would develop in all these patients.
Studies from Swan-Ganz catheterization with determination of pulmonary wedge pressure and calculation of left ventricular end diastolic pressure would have given the data needed to confirm or disprove their theory. In addition, thermolodiation studies with the catheter in place to determine cardiac output would have been valuable to substantiate their hypothesis.

If Swan-Ganz catheterization showed elevated pulmonary arterial pressures signifying pulmonary hypertension, a more likely explanation of dyspnea would be recurrent pulmonary embolism. These could arise via collateral venous channels or recanalization of the ligated inferior vena cava.

By their own admission, in the three of four cases where lung scans were done an intermediate probability for pulmonary embolus was present. The present state-of-the-art mandates the performance of pulmonary arteriography—the gold standard—to confirm or deny the presence of pulmonary emboli. This would be particularly true in patients with a history of emboli where there is high clinical suspicion of recurrent emboli. Only an absolutely negative ventilation/perfusion lung scan rules out pulmonary thromboembolism; in all other cases pulmonary angiography should be performed.

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To the Editor:

We thank Dr. Singer for his careful critique of our article. However, we disagree with several points. 1) We agree that D (A - a)O2 increased in all four cases with exercise, but in only one patient did the value exceed the upper limit exercise value of 30 mm Hg. 2) These patients did have varying degrees of edema, but the emphasis of our article is that flow through the venous collaterals was relatively normal at rest but inadequate during exercise, a well-recognized phenomenon occurring in other vascular beds, including the arterial circulation to the lower extremities and coronary arteries. 3) Swan-Ganz catheterization would have allowed determination of elevated pulmonary artery pressures, but knowing pulmonary wedge and LVED pressures would provide no additional information to distinguish recurrent pulmonary emboli from impaired venous return to the heart as the limiting feature of these patients’ exercise intolerance. There was no evidence for pulmonary hypertension on either physical examination or electrocardiogram in any of the patients. In the only patient who underwent an echo-Doppler study, a technique that has been shown to reliably measure RV pressure, RV pressure was only 30 to 35 mm Hg. 4) Determination of cardiac output by thermolodiation during exercise can be quite inaccurate relative to the Fick technique. Varat et al. measured exercise cardiac outputs by the Fick technique and found them to be reduced in their patients. We measured cardiac output in one patient using acetylene rebreathing, a well-accepted technique, and found it to be decreased below normal values for the given level of exercise, confirming the findings of Varat et al. 5) Pulmonary angiography was not performed to further investigate the intermediate probability lung scans. We therefore cannot exclude the possibility of recurrent pulmonary emboli. However, none of the patients presented clinically with historic evidence for acute pulmonary emboli. Absence of findings on physical examination and electrocardiogram to suggest right ventricular hypertrophy, along with normal diffusing capacities, would make chronic recurrent pulmonary emboli very unlikely. Additionally, if recurrent emboli were the responsible factor for these patients’ clinical dyspnea, abnormal gas exchange should have been a prominent finding. Again, we emphasize that these findings were not observed.

Despite lung scan results, the clinical impression was that none of these patients had pulmonary emboli and, therefore, pulmonary angiography was not deemed warranted. Two of the patients had positive smoking histories, one of whom also had evidence of obstructive lung disease by pulmonary function testing which may have accounted for abnormalities on the lung scans.

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Resuscitation in the Elderly

To the Editor:

I can think of no better illustration of the critical issue of high-tech healthcare so elegantly discussed by Dr. Cohn1 than a case report found in the same issue of CHEST.1 In this report, a 95-year-old woman collapsed at a nursing home and it appeared that extremely vigorous measures were taken to resuscitate her. Resuscitation of all individuals, regardless of age or quality of life, will no doubt result in enormous expenditures both in terms of services offered by paramedics and emergency departments who perform the resuscitation, and also from the cost of caring for survivors of resuscitation in intensive care units. As Dr. Cohn suggests, it is crucial to include the elderly in studies on the effects of high-tech healthcare. Both prehospital and in-hospital resuscitation should be a part of this effort.

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Digoxin-Like Immunoactivity

To the Editor

Using radioimmunoassays, digoxin-like immunoactivity has been found in the blood,1,2 urine,3 and amniotic fluid4 of human subjects not receiving cardiac glycosides. In some studies, digoxin-like immunoreactive substances (DLIS) have also been shown to have digitals-like bioactivity.5 High levels of DLIS have been found in the blood of patients with chronic renal failure,4 in cord blood1 and in the blood of pregnant women.6 However, the clinical relations of urinary DLIS have not been determined. Accordingly, our purpose is to report on the findings in a heterogeneous population of human subjects whose urine was examined for DLIS.

Urine specimens submitted to a hospital laboratory for chemical analysis were selected at random for digoxin assay. Urine samples of healthy, ambulatory subjects were also assayed. Whenever DLIS was detected, the case was reviewed to ensure that cardiac glycosides had never been administered. Urine samples were derived from a heterogeneous population, of both sexes, ranging from 1 to 93 years of age. In 40 patients, urinary creatinine concentration was also measured and in 30, a 24-hr urine collection was obtained and creatinine clearance was calculated.

Digoxin levels were determined by radioimmunoassays. Assays

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