Oxygen Uptake and Mechanical Ventilation

To the Editor:

I would like to address the findings reported by Husbayar et al in their article, "Oxygen Uptake During Weaning From Mechanical Ventilation" (Chest 1988; 94:1148). This study hypothesizes that assessing ventilatory muscle loads and \( V_O_2 \) consumption should predict ventilatory muscle overload, fatigue and consequent ventilator dependence. The data presented, however, do not appear adequate for completely testing this hypothesis. I say this for two general reasons, one technical and one related to patient selection.

The reported work and \( V_O_2 \) measurements may not be a reliable reflection of actual patient demands during prolonged spontaneous ventilation. My reasons for saying this are as follows: first, it would seem to me that the estimate of work during spontaneous ventilation should be based on the minute ventilation needed for adequate gas exchange and not just the minute ventilation the patient chooses to generate during a short period off the ventilator. That the study patients all needed to be returned to the ventilator with rising end tidal values for \( CO_2 \) suggest that these spontaneous minute ventilations, in fact, may not have been adequate. Work calculated for the patient during the measurement period may thus have been an underestimate of the work needed for prolonged ventilation without the ventilator.

Also, significant errors can be introduced into the estimation of ventilatory muscle loads when the control tidal volume-frequency pattern used for the trapezoid pressure measurement technique does not mimic the patient's actual ventilatory pattern. Specifically, it can be shown from a computer simulation that the work/min associated with an alveolar ventilation of 6 L/min, respiratory system compliance 50 ml/cm \( H_2O \), and airway resistance 12 cm \( H_2O \) L/s and \( VD/VT \) 0.25 (values seemingly comparable to this patient population) can range 9.0 joules/min to 17.9 joules/min as the respiratory rate varies from 6 to 40 breaths/min.\(^1\) Data on what settings were used in this study would be helpful. Finally, I agree that the mean inspiratory pressure is probably a better reflection of muscle \( V_O_2 \) under high impedance loads. This has both theoretic as well as experimental basis.\(^4\) Inspiratory pressure data would thus be helpful.

If it is true that, despite the concerns noted above, the calculated load and \( V_O_2 \) values are reasonable, a different problem then arises. Specifically, these measurements indicate that ventilator dependency in these patients is not a consequence of overload of the ventilatory muscles. Calculated work of breathing in the five ventilator-dependent patients was only 11.99 joules/min. While this is almost double the normal work of breathing (around 5 to 6 joules/min), it is well below estimates of work that are usually associated with muscle fatigue and a consequent need for mechanical ventilation.\(^3\) Second, although not listed, I believe I can calculate the average inspiratory pressure in these patients to be on the order of only 12 cm \( H_2O \). This is a very low pressure demand on the ventilatory muscles and, even if the negative inspiratory force is severely reduced (values not given), this level of pressure should still be consistent with weaning if ventilator dependence is a consequence of ventilatory muscle overload (ie, fatigue and ventilator dependence appears to be associated with a mean inspiratory pressure to maximum diaphragmatic pressure ratio of greater than 40 percent). Data on muscle pressure generation capabilites would be helpful. Third, the diagnoses given for the ventilator-dependent patients are not the kind of diagnoses we associate with muscle overload. Specifically, there is a sleep apnea patient and four patients with a primary diagnosis of heart disease. Lung mechanical function and gas exchange information would be helpful in clarifying this. Finally, the authors point out that work as an index of muscle load in group 1 and group 2 are not significantly different.

It thus seems that these patients have ventilator dependence as a result of something other than muscle overload (eg, cardiovascular instability, gas exchange abnormalities, neurologic dysfunction, severe muscle weakness).\(^4\) Consequently, this patient population does not seem to be the one that should be used to test a hypothesis involving muscle overload.

To summarize, I think this study addresses some very important ways of assessing some characteristics of ventilator dependence. These measurements of muscle load and muscle oxygen consumption, however, would seem to me to be most useful in patients in whom muscle overload was the reason the patients were mechanically ventilated. Because of this, I am not surprised that they are not particularly useful in patients such as these with other reasons for ventilatory dependence. Complicating this is the concern I have regarding the accuracy of these indirect measurements. At the present time, then, I must conclude that the stated hypothesis remains a reasonable one and I would speculate that proper monitoring of load/muscle relationships should be a useful parameter to follow in ventilator dependence resulting from relative muscle overload.

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To the Editor:

In contrast to the conclusions we reached\(^6\), Dr. MacIntyre suspects that proper monitoring of load/respiratory muscle relationships is useful in following patients with ventilator dependence from relative muscle overload. He suggests that our patients were not in a steady state, had causes of respiratory failure other than respiratory muscle overload, and that the inspiratory work, as we have calculated it, is an inadequate predictor of the load on the inspiratory muscles during spontaneous breathing. In order to measure oxygen uptake (\( V_O_2 \)) in the absence of phasic inspiratory muscle activity, awake subjects must be mechanically hyperventilated, which results in an arterial \( CO_2 \) tension below that during spontaneous breathing.\(^7\) Based on the stability of minute ventilation, \( V_O_2 \) and breathing pattern (which were continuously monitored during weaning), we believe that our patients were indeed in steady state. We agree with Dr. MacIntyre that the reasons for ventilator dependency in our patients were multifactorial and recognize the limitations of inspiratory work as a measure of inspiratory load. In fact, as we have stated in the paper, it is the recognition of these limitations that lead us to conclude that routine monitoring of either \( V_O_2 \) or inspiratory work in the hope of predicting weaning outcome or severity of disease state has little or no role in clinical practice. In a mechanistic sense, Dr. MacIntyre's hypothesis that muscle
overload may lead to respiratory failure is probably correct. However, for this hypothesis to find clinical applications, the critical care practitioner must be in a position to diagnose muscle overload with confidence, rule out confounding variables (ie, other causes for respiratory failure), and possess the means to accurately quantitate inspiratory load. It is our firm belief that with the current state of technology, these expectations are unrealistic. Even with the resources of a research study, the measurement of VO2 in critically ill patients is exceedingly tedious, carries a high chance for experimental error, and has no predictive value. It has not been our intent to discourage clinical research on the mechanisms of respiratory failure, but merely to caution against the indiscriminate use of monitoring technology in clinical practice.

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Echocardiography vs CT in Lung Cancer

To the Editor:

I would like to comment about the report, "Echocardiography in Determining Nonresectability of Carcinoma of the Lung" by Howard et al (Chest 1988; 94:1306-07).

The paper deals with a failure of CT in determining invasion of the heart by a bronchogenic carcinoma. Echocardiography is presented as an important and underutilized tool for staging bronchogenic carcinomas. I would like to take issue with the chest radiograph and particularly with the CT scan displayed in the article.

The chest radiograph (Fig 1 in their article) likely shows a peripheral right upper lobe mass with hilar and lower paratracheal lymph node enlargement indicative of stage IIIa. The legend to this film reads: "Hilar mass with postobstructive pneumonia. No radiographic evidence of unresectability." I cannot concur with this interpretation.

The legend to Figure 2 reads: "No evidence of vascular pericardial or cardiac invasion or hilar adenopathy is present." The CT scan shown is at the level of the aortic arch and does not include the pericardium, the cardiac silhouette or the right hilus. This picture could not demonstrate invasion of these structures even if it were present. What is more disturbing is the fact that the CT scan reproduced in the article definitely shows an enlarged right pretracheal, retrocravical lymph node which is highly suspicious for metastatic disease and would probably preclude resection of this bronchogenic carcinoma.

In conclusion, I have to disagree with the authors of this paper: the chest radiograph and the CT scan in this case show evidence for stage III disease. Echocardiography cannot compete on a routine basis with CT in staging bronchogenic carcinoma.

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To the Editor:

We feel that the questions Dr. Stark has raised regarding our report are good and we will try to answer them as best we can.

The official reading of the radiograph on our case report was hilar mass with postobstructive pneumonia. It may very well be that Dr. Stark's interpretation that the x-ray film showed a peripheral upper lobe mass with hilar and lower paratracheal lymph node involvement may be true. In either case, at the University of South Carolina radiographic evidence of unresectability means that the disease is not confined to the side of the chest involved. Hilar adenopathy or lower paratracheal node enlargement does not mean that the patient is unresectable. On biopsy, some enlarged nodes in the hilum and mediastinum often show no evidence of tumor. For this reason, our protocols mandate mediastinoscopy (or Chamberlain procedure for left upper lobe lesions) to assess mediastinal nodes. For staging purposes and prognosis it should be pointed out that N₂ disease is not equivalent to metastatic disease. It has been shown that a stage III-A lesion can be resected with improved survival. We do not feel it would be in the patients' best interest to deny them a chance at a cure based upon a chest x-ray film showing hilar or paratracheal mass alone.

Your comments on the CT scan are absolutely correct. Space limitations precluded us from showing the whole CT scan. We chose a cut that showed both the peripheral and the hilar nature of the lesion. At our institution, we use the CT scan to look for M₁ disease to guide us to biopsy an area outside the chest. CT scan findings of enlarged lymph nodes do not deem our patients as unresectable for the reasons cited above. Again, we like to emphasize that stage III disease does not mean that the patient has an unresectable tumor.

We do not advocate that echocardiography compete with CT scanning in preoperative staging, but we feel it can be used to help evaluate what appears to be local extension into the pericardium or great vessels. Obviously, since some of the atra can be included in the resection (if no tumor is left behind), the extent of invasion will need to be quantified in some way. At this point in time we are looking at this question, but we do not have an answer as yet as to how to fully utilize the echocardiogram. If the echocardiogram were to show invasion of most of the atria or a ventricle, then that patient should not undergo an exploratory thoracotomy.

Since the echocardiogram is not invasive and may allow us to make a judgment as to whether or not a thoracotomy will lead to a curative resection, we feel that it may have a role in the preoperative staging of the patient with a bronchogenic carcinoma.

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Diagnosing Sarcoïdosis

To the Editor:

The article, "Diffuse Interstitial Pneumonitis and Fibrosis in Sarcoïdosis" by Aisner and Albin (Chest 1988; 94:193-95) is of considerable interest. The authors report a case of sarcoïdosis in which extensive interstitial pneumonitis and fibrosis were demonstrated in areas without active granuloma formation. They stress that: 1) interstitial pneumonitis has rarely been reported in sarcoïdosis, and in their case was diffuse; 2) interstitial pneumonitis and fibrosis were found in regions distinct from granuloma formation; and 3) the finding of interstitial pneumonitis on transbronchial biopsy does not exclude a diagnosis of sarcoïdosis. We believe these