The Clinical Assessment of the Relative Importance of Mechanical Defects and Abnormalities of Respiratory Drive

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I am going to put before you a framework which has served me well for many years, both in practice and teaching. It represents a compromise between the rigorous and the practical. I shall introduce it in the form of two questions:

Can’t breathe or won’t breathe?

I like to know the Pco₂ of a patient not because I am worried about Pco₂ but because of what a raised Pco₂ means. It means the respiratory system (the whole thing—not just the lungs) is not breathing enough. There are two possible reasons: either he cannot breathe or he will not breathe—and in many patients, it is a bit of both and I like to try and decide how much.

Why is this patient so breathless?

A less dramatic problem, but one which nevertheless must come into one’s assessment of a breathless patient is: is there, in addition to his mechanical difficulty, some abnormal stimulus to breathe (higher nervous, pulmonary, circulatory, chemical)?

When teaching, I develop the theme using the Pco₂: FEV plot. What follows is an attempt to put in literary form something like what I say and something like what is left on the blackboard when I have finished.

The Vertical Axis

The choice of Pco₂ needs no justification. Most people think in terms of arterial Pco₂, but other sites may be preferred. The paraphrase for Pco₂ is commonsensical: substitution of more precise terms such as CO₂ production and alveolar ventilation is left to choice. The downward arrow (nonCO₂ drive) on the right is commonsensical. The choice between the two descriptions of the upward arrow (CO₂ drive and failure of CO₂ homeostasis) depends on the point of view required by a particular problem. The usual servo model of breathing taking elevation of Pco₂ as the error signal permits either.

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REFERENCES


The Horizontal Axis

The choice of FEV is justified by clinical usage. The leftward arrow below the horizontal axis indicates the obvious: a decreased FEV may be due to a decreased ability to breathe, an increased load to breathe or, as is probably common in many patients, some combination of the two.

Neither axis has been given any numbers, but the shaded band starts on the right by indicating that the normal range of Pco₂ is about one-quarter of the mean. The inflection of the curve is at about one-third on the horizontal axis and the general shape is in accord with what is usually found in a mixture of patients with bad chests.

Drive

I use “drive” in a final overall sense to indicate the muscular forces applied to the chest as a result of the summation of the chemical, reflex, higher nervous, brain stem and spinal mechanisms eventually integrated at the lower motor neurons.

The two oblique arrows (increased drive and decreased drive) are deliberately drawn half inside and half outside the shaded area of usual clinical experience to emphasize the marginal problems about which clinicians need help.

It is tempting to use the lower left boundary ever observed clinically as defining the mechanical limit and to say that any point above it signifies a defect of drive. However, I think this is unacceptable because of the uncertainty of our methods and data (see Noise below).

Noise

The usual range of Pco₂ is quite large and it is not easy to predict the “normal” for a particular person. The intraindividual variation in mixed venous Pco₂ is certainly much less than the intersubject variability. There are many factors which limit the precision with which FEV₁ estimates either the ability to breathe or the load to breathe and these limitations of the FEV₁ become more troublesome as the FEV₁ decreases.

An important contribution to noise is pulmonary gas exchange. Other things being equal, an increased dead space due to inefficient gas exchange will cause an elevation in the Pco₂:FEV curve.

The dashed rectangle outlines the main area of clinical concern. The four boundaries are arbitrarily assigned on the following grounds. First, the right side: few patients seek medical advice until they have lost more than half their breathing ability because the activities of daily living do not need more than half the breathing ability. The left side: below a certain FEV₁, the mechanical problem is inevitably dominant. The lower boundary: a Pco₂ below the lower limit of normal always implies an abnormal drive. The upper boundary is defined by the highest Pco₂ compatible with life. Breathing oxygen can extend this boundary. Note how much of the area of the rectangle is shaded—is “noisy.”

There is little practical or conceptual problem in recognizing the so-called primary hypoventilator northeast of the rectangle as having defective drive or in recognizing the asthmatic south of the rectangle as having an abnormal drive. What can one do about patients in the rectangle? Here are a few tips.

First and most important, try to define the Pco₂:FEV curve of the individual patient by following the fluctuations in his condition with simultaneous measurements of the Pco₂ and FEV₁ to see if his curve deviates from the usual. I prefer to use the mixed venous (rebreathing) Pco₂ for several reasons: first, it is less susceptible to fluctuations in the breathing and hence less likely to give spuriously low estimates of the Pco₂; secondly, it is arguably more constantly related to the longterm central CO₂ sensor; thirdly, for those who have got the method going it is less bothersome than stabbing an artery. (If asked to see a patient when this information is not available, look for past records of plasma bicarbonate in the electrolytes; these may give a hint about the likely chronic level of Pco₂.) Secondly, note the objective accompaniments of the dyspnea. In other words, in addition to recording the history, note closely the action of the muscles and the forces of movement of the thoracic cage. Thirdly, use clinical examination of the shape and movements of the chest to make allowance for these factors when interpreting the FEV₁ as an index of the ability to breathe. I cannot make this hint very precise, but I believe that a distorted inflated chest with respiratory phase lags represents, for any given FEV₁, a greater mechanical problem than if these signs are absent. To put it the other way around, for a given FEV₁ I am more suspicious of defective drive in a patient with a marginally raised Pco₂ if his chest has fairly normal shape and movements.

If I can have this sort of information, I do not often feel myself clinically wanting more of the currently available fancy tests of mechanics or chemical control, but I would like better and more widely applicable methods to add precision and understanding to this simple clinical framework.