separating patients with severe (aortic valve orifices $\leq 0.08$ sq cm) from nonsevere aortic stenosis is important because it allows sensitivity, specificity, and likelihood ratios to be determined for any cut-off value employed for a given noninvasive parameter. Bayesian analysis, taking into account not only sensitivity and specificity but also "pre-test likelihood" of having the disease, has been utilized previously in treadmill exercise testing for the prediction of coronary disease. \textsuperscript{22,23} This type of approach, evaluating a combination of noninvasive tests in the context of the history and physical examination, provides a powerful tool in assessing the severity of aortic stenosis.

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\section*{Interpretation of Arterial Blood Gases}

While arterial blood gas measurements have become increasingly easy to obtain in recent years, their interpretation has become increasingly difficult, especially in the intensive care setting. This is because of heightening awareness that factors other than intrapulmonary abnormalities can affect the arterial $P_{O_2}$ and $P_{CO_2}$. Most of us would like to use blood gas measurements as an index of the state of the lungs, at least to the extent that improved values can be equated to improved lung function and vice versa. The difficulty is that arterial $P_{O_2}$ and $P_{CO_2}$ reflect not only the state of the lung (i.e., shunt, ventilation-perfusion ($VA/Q$) inequality and incomplete diffusion equilibration), but also the conditions under which the lung is operating, that is, the composition of inspired gas and mixed venous blood.
Inspired $\text{PO}_2$ is usually easy to take into account. Thus, it is well appreciated that as $\text{FiO}_2$ is raised, hypoxemia due to $V_A/Q$ mismatching or diffusion impairment is alleviated, while that due to shunting persists, particularly when the shunt is large. This nice theory can, however, be upset in several ways, many of which may not be evident just from the blood gas measurements themselves. Shapiro and colleagues (see page 138) address one such problem: the induction of atelectasis by the very breathing of enriched oxygen mixtures. The theoretical aspects of this phenomenon have been studied\textsuperscript{1,3} and center on the cessation of expiration in lung units of very low $V_A/Q$ ratio, as first suggested by Briscoe and co-workers.\textsuperscript{1} The article by Shapiro and coauthors adds to the body of experimental evidence on this subject. Oxygen-induced atelectasis, in turn, results in two highly undesirable problems—confusion in the assessment of the state of the lungs, and potential deterioration of the patient. Another example of a problem incurred by high oxygen concentration breathing occurs in patients with chronic obstructive pulmonary disease where some poorly ventilated lung units wash out nitrogen, $(N_2)$ very slowly. In addition, $N_2$ levels in these units may transiently rise to very high values through the concentrating effect of rapid $O_2$ transfer into the blood as alveolar $\text{Po}_2$ rises. As a result, arterial $\text{Po}_2$ rises very slowly during 100 percent oxygen breathing, and even after 30 minutes it may be sufficiently low so as to falsely suggest shunt fractions of 10-20 percent\textsuperscript{3} when no shunt, in fact, exists.

Mixed venous $\text{Po}_2$ ($\text{PrV}_2$) must also be taken into account in interpretation of blood gases, as was elegantly shown by Pontoppidan and co-workers several years ago.\textsuperscript{4} Since mixed venous $\text{Po}_2$ is considerably less accessible than inspired $\text{Po}_2$, it is accordingly even more important to understand how great a role $\text{PrV}_2$ can play in determining arterial $\text{Po}_2$. Whatever the cause, a fall in $\text{PrV}_2$ results in a fall in arterial $\text{Po}_2$ in a patient with a fixed amount of intrapulmonary abnormality, and vice versa. It is therefore impossible to interpret a change in arterial $\text{Po}_2$ in a given patient without knowledge of the mixed venous $\text{Po}_2$. It should be pointed out that other factors held constant, $\text{PrV}_2$ will fall as (a) cardiac output falls, (b) $\text{O}_2$ consumption rises, (c) hemoglobin content falls, (d) alveolar ventilation falls, and (e) intrapulmonary disease worsens (because of worsening shunt, $V_A/Q$ relationships, or incomplete diffusion equilibration). It is not necessary to spell out the myriad ways in which cardiac output, $O_2$ consumption, hemoglobin content, alveolar ventilation, or lung disease can each change, sometimes simultaneously and in opposite directions, so as to complexly alter arterial $\text{Po}_2$. Thus, it is comforting to see that at least some of the data reported by Shapiro and associates are supported by direct mixed venous $\text{Po}_2$ measurements. In the remaining examples, the results must be interpreted with caution because $\text{PrV}_2$ was assumed rather than measured.

As usual, we are left in a dilemma. Inspired $\text{Po}_2$ is not difficult to monitor, but estimation of mixed venous $\text{Po}_2$ generally requires direct sampling through a pulmonary artery catheter, the placement of which carries the risk of many unlikely, but potentially dangerous, complications. We then have to balance the need for accurate and scientifically meaningful analyses of pulmonary gas exchange against the risk of placement of the pulmonary artery catheter in the individual instance.  

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