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## Editorial Views

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# *On the Possible Painful Consequences of Misapplying Signal-detection Theory*

SIGNAL-DETECTION THEORY was originally developed by communications engineers to distinguish statistically situations where a weak signal plus noise is likely to be present from situations ("blanks") where noise alone is likely to be present. When signal-detection theory is used to study analgesics, a higher-intensity stimulus takes the place of the "signal," and a lower-intensity stimulus takes the place of the "blank." The methodology under these circumstances is often referred to as sensory decision theory, rather than signal-detection theory. In sensory decision theory, a human subject is asked to make a decision about whether pain is present without knowing whether or not the high-intensity stimulus (the "signal") or the low-intensity stimulus (the "noise") is being applied. The situation is adjusted so that subjects identify as "painful" mainly (but not exclusively) those intervals where the higher-intensity stimulus is presented.

For a reader to interpret the paper of Yang *et al.*[1] intelligently, two other articles[2,3] are particularly helpful. The use of signal-detection theory does provide an approach to analgesic studies that is more sophisticated than are the older "threshold" techniques. Signal-detection theory provides an answer to two questions: 1) Can the difference between two stimuli of different magnitudes be detected?; 2) Can the intensity at which a subject reports a stimulus as painful be modified? It thus seems to provide more information about "analgesia" than do threshold studies, since threshold studies can be affected by, among other things, either a change in the ability to discriminate painful from nonpainful stimuli or a change in the subject's verbal or behavioral response independent of what is actually "experienced."

Unfortunately, there may be as little relationship between discrimination and conscious experience as there is between conscious experience and verbal or behavioral response. Despite the disclaimers of Chapman,[3] signal-detection theory proponents, including the present authors, do use language which at least tempts both clinicians and researchers to equate decreased discriminability with analgesia. For example, Clark states that studies that do not use sensory decision theory provide "no clear evidence" that an intervention has actually altered the pain "experience."[4] At least to me, this clearly implies that studies that do employ sensory decision theory do tell whether the pain "experience" is altered. In the present article,[1] the authors suggest that the results (*i.e.*, decreased discriminability and decreased response criteria) show the "predicted results of an effective analgesic." Unfortunately, it is not at all clear that an "effective" analgesic would necessarily decrease the ability to discriminate between two stimuli, or that a drug that fails to decrease discriminability would be ineffective as an analgesic.[3]

Shifts in signal discriminability tell one only about the ability to discriminate between stimuli. Decreased ability to discriminate between stimuli is neither necessary nor sufficient to demonstrate "analgesia." It is this linkage (intended or not) between the concepts "analgesia" and "decreased signal discriminability" that can confuse or mislead the nonexpert reader. Worse, because the statistical methods involved are unusually complex, it is hard to keep the non-

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statistical issues clearly in mind. Hence, I would like to emphasize that the possible confusion does not lie in failure to understand the complex statistics of signal-detection theory.

If its limitations are clearly understood, signal-detection theory can provide more overall information about analgesic drugs and techniques than that provided by less complex threshold studies. However, it has not yet been shown that this added information increases our ability to predict the clinical usefulness of analgesic drugs or techniques, or to predict the circumstances when a given drug will be effective. Indeed, the additional information, while interesting, may be misleading if it is directly employed in deciding which drugs or techniques have clinical merit, or even which drugs or techniques warrent further clinical evaluation.

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# *The Changing Arterial Oxygen Tension—Disease or Physician?*

SINCE THE OXYGEN ELECTRODE became readily available to clinicians and research investigators two decades ago, there has been a tremendous increase in our knowledge of the variables influencing arterial oxygen tension ($Pa_{O_2}$). Nowhere is this truer than with pulmonary failure, including its management by techniques designed to maintain or restore lung volume.

It is probably not surprising, however, that we have focused disproportionately on factors influencing distribution of ventilation, rather than on the determinants of regional pulmonary blood flow. Decreases in lung volume are discernible clinically, roentgenographically, and by laboratory methods, and an associated increase in alveolar–arterial oxygen tension differences ($AaD_{O_2}$) is commonly observed. It is easy to conclude that an increase in lung volume, by one of the common clinical techniques such as positive end-expiratory pressure (PEEP) and the associated decrease in $AaD_{O_2}$ are directly related. Similarly, in following the pulmonary course of a patient with pneumonia or congestive heart failure, we relate a decrease in $AaD_{O_2}$ to an improvement in pulmonary status, primarily from a resolution of low-ventilation areas resulting from the disease process. In so doing, it is important to recognize that considerable changes in $Pa_{O_2}$ values and $AaD_{O_2}$ may occur independent of changes in lung volume or in pathologic status.

Changes in $AaD_{O_2}$ secondary to techniques for positive-pressure distention of the lung may reflect the effect of airway pressure on pulmonary vascular resistance in ventilated regions. Kanarek demonstrated adverse redistribution of whole-lung pulmonary blood flow with PEEP and, in that instance, the magnitude of this adverse redistribution effect on $Pa_{O_2}$ values exceeded any beneficial effect of positive pressure.[1] It is reasonable to assume that this trade-off occurs in all patients treated with PEEP and that the effect on $AaD_{O_2}$ is the resultant of increasing ventilation to some low-ventilation/blood flow ($\dot{V}/\dot{Q}$) regions and increasing perfusion to others. In this issue, Benumof *et al.*[2] address this important problem using a dog model, and find considerable changes in distribution of pulmonary blood flow. Thus, one set of opposing factors acting during mechanical ventilation is defined.