receptors for neurotransmitters and modulators on synaptic as well as non-synaptic membranes. We consider that, like local anesthetics, also general anesthetics stabilize nerve membranes, but via a different mechanism, *i.e.*, stabilization of the hydrogen-bonded aqueous-protein structure. Our observations both with D_2O and with neuroamines can be integrated into a single theory because replacement of H_2O by D_2O in the Ringer's solution also alters catecholamine receptors in peripheral nerve.[7] It would be interesting to study the actions of neuroamines and of D_2O on artificial membrane models. Certainly Dr. Singer's experiments with this model support our view that general and local anesthetics have some properties in common (*i.e.*, Na^+ antagonism in our case, "fluidization" of membranes in his) and also have different properties (*i.e.*, Ca^{++} and H_2O interactions in our nerve experiments, interaction with membrane negative charges in his observations).

BRUCE I. DIAMOND, PH.D
HENRI S. HAVDALA, M.D.
HECTOR C. SABELLI, M.D., PH.D.
Department of Anesthesiology
Mount Sinai Hospital Medical Center of Chicago
Chicago, Illinois 60608

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Signal Detection Theory and Pain

To the Editor:—The measurement of pain sensation has been bedeviled by problems not encountered with the other senses. Chief among them is the fact that pain is defined by the affective response to the painful stimulation. In the other senses it has been possible to approach the measurement of sensation behavioristically—does the subject discriminate a particular intensity of light from darkness or a concentration of sodium chloride from water? It does not matter greatly how the person feels about the color or salty taste. With pain, however, the essential question is the person's feeling about the stimulus that is impinging on his receptors. This would not be an insurmountable problem if we found that people agreed among themselves as to which intensities of what stimuli were painful, but it is well known that people vary greatly in their assessment of painful stimuli as a function of motivational,

cultural, and cognitive factors. Not that these problems are absent in the other senses. In fact, signal detection theory (SDT) was developed in order to make it possible to separate the subject's sensitivity to stimuli from the other factors, collectively defined as his criterion for reporting the presence of the stimulus. Several investigators have seized on this theory as an answer to the puzzle of pain measurement.[1-5] In his editorial,[7] Chapman commends this theory as one fruitful approach to the measurement of pain.

However, at the risk of appearing like the dog in the manger, I must restate my objection[8] to the use of SDT in pain research. Chapman acknowledges that there have been criticisms[8,9] of the use of SDT in the experiment of Clark and Yang,[6] but says that they involve "procedural factors that limited the precision of measurement" (reference 7, p 503). Far from it. Both Hayes *et al.*[9] and I[8] argued that *theoretical assumptions* of SDT were not met in the Clark and Yang experiment. Hayes *et al.* said further "Even explicit operational recognition of the assumptions of the model still poses serious questions about its applicability" (reference 9, p 66) and I said "Unfortunately, it does not seem possible to meet these requirements in principle" (reference 8, p 66).

Signal detection theory requires the presentation of two (or more) events that can independently be defined as noise and signal(s). The signal is some intensity of a stimulus (a blue light or a salt solution) and the noise is an event that can be considered by definition not to be blue or salty or whatever. The problem in the case of pain is that ordinarily (and perhaps always) stimuli that are too weak to elicit pain sensations do elicit sensations such as touch and the like. Therefore, there is no stimulus that can be defined as either painful or imperceptible. And, in the absence of two stimuli that can be independently defined as signal and noise, SDT cannot be employed. This problem has been recognized by those who have tried to apply SDT to pain. Their attempted fix has been to present two (or more) intensities of some stimulus, one (or more) of which can be considered to be painful to some degree. They treat the weaker one as noise and the stronger one(s) as signal(s) and proceed to draw conclusions as to changes in sensitivity and criterion as a result of nitrous oxide[4,5] or acupuncture.[6] This is a legitimate application of SDT if one is interested simply in the ability to discriminate between intensities of a stimulus. But it does not allow one to conclude anything about the presence or absence of a painful sensation. The basic reason is that SDT is a theory of how people discriminate between two states of the world. As such, it cannot be used to measure an affective state or any other attribute of a stimulus that cannot be assumed to be such by definition. Both the weaker and the stronger stimuli may have become painless without affecting the person's ability to discriminate between them. On the other hand, it is conceivable that the weaker one was affected more than the stronger, and the ability to discriminate them would increase, leading to the incorrect conclusion that sensitivity to pain has increased!

Perhaps an example outside of pain would be helpful. When one presents weak intensities of a blue light to a dark-adapted eye, some will be so weak as to be invisible. Somewhat greater intensities will be visible but colorless. Still stronger ones will appear blue. This well-known effect is owing to the existence of two receptor mechanisms in the eye; the more sensitive rod system, which is color-blind, and the less sensitive cones, which mediate color experience. One could do a SDT analysis of sensitivity to weak lights, but there can be no independent assessment by SDT of whether the lights appeared to the person as blue or white (so long as only one wavelength were used). In fact, by differential dark adaptation and recovery, it is possible to manipulate the sensitivities of the two systems independently so that the blue "threshold" can be varied without affecting the light "threshold."

Unfortunately, there is no solution to this problem within SDT. What looks superficially like the ideal solution to the problem of pain measurement turns out to be totally inappropriate. And this is so because pain is, in the

last analysis, a subjective response to a stimulus that does not permit independent definition of the stimulus as either necessarily painful or imperceptible.

DONALD H. MCBURNEY, PH. D.
Professor of Psychology
Department of Psychology
University of Pittsburgh
Pittsburgh, Pennsylvania 15260

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To the Editor:—McBurney has charged that sensory decision theory (SDT) methodology is inappropriate for the problem of pain measurement. His argument is based on three assumptions, and these warrant consideration.

First, McBurney argues that pain must be defined by the perceiver's affective response, rather than by sensory responses. Unfortunately, he has assumed a very limited view of the human pain experience. Recent models of human pain have emphasized that it is a complex perceptual experience involving the entire personality of the individual. Melzack and Casey[1] list three dimensions of the human pain experience: a sensory–discriminative dimension concerned with detection and intensity discrimination of pain signals; a motivational–emotional dimension that involves the aversive qualities of pain and the fear responses that it generates; and the central control dimension, which consists of cognitive and judgmental processes occasioned by the sensory input. The affective aspects of pain are indeed important, but this does not negate the scientific relevance of the sensory–discriminative dimension of the pain experience. Information about the effects of an analgesic on an individual's ability to detect and discriminate painful events is clearly pertinent to many research and clinical issues.

McBurney's second assumption is that there is no stimulus that can be defined as either painful or imperceptible. Recent reports do not support this perspective. There is now strong evidence that certain sensory end organs serve as nociceptors, and that small, unmyelinated C and larger A-delta fibers transmit nociceptive information.[2,3] Studies of tooth pulp show that it is innervated, perhaps exclusively, by C and A-delta afferents, and it has been argued on this basis that electrical stimulation of the pulp provides an excellent laboratory model for pain.[4] Electrical stimulation of tooth pulp produces pain[5] just as electrical stimulation of the retina generates an experience of light.[6]

Finally, McBurney has equated the formal theory of signal detection with the methodology used by signal-detection theorists. He assumes that the procedure requires the use of a stimulus defined as noise and another defined as a signal plus noise, and that SDT cannot be employed without this definition. Historically, the real origins of the methodology antecede the signal-detection theorists. In 1927, Thurstone introduced his discriminal process model for category judgments, a probability theory conceptualization intended to be a means of measuring subjective states.[7,8] The familiar overlapping normal curves of SDT are actually an extension of Thurstone's discriminal distributions.[9] The concepts of signal plus noise and noise alone were not employed in the Thurstonian model, and, indeed, were not