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Editorial Views

Pain, Analgesia and Anti-Analgesia

IT IS UNFORTUNATE that *pain* continues to constitute not only a scientific problem but a semantic one as well. Pain has been considered a philosophic problem in terms of its meaning and purpose, a theological problem in terms of a moral force, a psychological problem in terms of modifying behavior and a medical problem in terms of disease. It has been considered good as a homeostatic protective device and bad because of its deleterious physical and mental effects. Pain has been described in subjective terms (burning, aching), in terms of transmission (slow, fast), in terms of receptors (specific, free endings), in terms of site of origin (cutaneous, somatic, visceral, deep, superficial) and in terms of disease (anginal pain, colic pain). In contrast to the difficulties in defining pain, the definition of analgesia is quite clear, *viz:* insensibility to pain and by extension, relief of pain, amelioration of pain, prevention of pain, or loss of pain sensation. Clearly, analgesia is defined only in terms of whatever constitutes pain.

As early as 1895, Strong proposed a concept of pain, now widely accepted, which states that pain consists of two components: (1) the perception of the stimulus at the site of stimulation and (2) an affective reaction to the stimulus as it reaches consciousness. Hardy, Wolff, and Goodell, while accepting this hypothesis, carried out extensive experiments during the early 1940's on alterations in thermal pain thresholds of man under many circumstances including the administration of drugs. They quantified pain in terms of pain threshold and analgesia in terms of increases in threshold, ignoring the reaction component of pain. During the succeeding years, Beecher pointed out in great detail the limitations of such pain threshold measurements and his own observations further documented the validity of Strong's original hypothesis. Beecher concluded that pain was undefinable except as every man defined it introspectively for himself. Pain existed only as the total experience ("suffering") of an individual responding to a stimulus which had been modified at all levels of central nervous system integration of such factors as memory, conditioning, anxiety, distraction, etc. He even doubted that pain threshold techniques measured perception of a pure stimulus free from any affective component.

As an operational definition the pain experience served as an important unifying concept. It was supported by anatomical studies which demonstrated the many pathways by which pain stimuli could be modulated at all levels of the central nervous system. It provided an explanation for the placebo response, the effect of emotion and suggestion on pain, the lack of correlation between the degree of stimulation and the degree of "suffering" and the lack of quantitative correlation between analgesic induced elevations in pain thresholds and the clinical effectiveness of the analgesic. The strongest support for this concept came from studies of those rare and unique individuals who have congenital indifference to pain. These individuals have no neuroanatomic abnormality, either macro- or microscopically.

They perceive pain normally. Their tactile and thermal sensibilities are normal. Their biologic response to injury is normal. They are not mentally retarded, socially irresponsible, nor immoral. They simply have a defect in the nervous mechanism responsible for this reaction component of pain.

Against this background, Clutton-Brock revived measurements of pain thresholds in man utilizing graded tibial pressure as the stimulus to demonstrate the threshold elevating effect of hyperventilation. He extended his observations to show that barbiturates decreased the pain threshold and interpreted this as an increase in pain or to use his phrase, "anti-analgesia." Dundee then applied the same technique to a study of a large number of compounds used for preanesthetic medication and anesthesia. He developed a classification according to which drugs were analgesic, slightly analgesic, or anti-analgesic depending on changes in threshold.

In the current issue of the Journal, Robson and his associates report on their extension of the work of Clutton-Brock and Dundee to include measurement of changes in both the tibial pressure pain threshold and the thenar thermal pain threshold in healthy subjects. They too found that thiopental decreased the tibial pressure pain threshold. However, it increased the thermal pain threshold. They were unable to confirm Dundee's observations on the anti-analgesic effect of halothane, since halothane, like nitrous oxide, increased the threshold of both types of stimulation. To explain this curious difference in the effect of thiopental on the two thresholds they noted that barbiturates and inhalation anesthetics act differently on presynaptic inhibition within the spinal cord. They then concluded that the information traveling in the central nervous system as a result of these two pain stimuli was different. Their experiments again illustrated the difficulties in defining pain in terms of pain thresholds and analgesia in terms of alterations in thresholds.

In this light, the introduction of the term "anti-analgesia" is inappropriate, since the term only describes a laboratory observation limited to tibial pain thresholds. "Anti-analgesia" is uninterpretable in terms of the pain experience or of clinical medicine. Hyperalgesia would describe the increased appreciation of a painful stimulus better than the double negative of "anti-analgesia" or the pretentious "antanalgesia."

To criticize the terminology is not to criticize the merit of experimentation utilizing pain threshold measurements. Robson and associates demonstrate the type of valuable information which can be derived from such experiments. Any definition of these changes in anatomic, physiologic and pharmacologic terms is worth exploring. Some studies of Dundee suggest that these changes represent increased reflex activity. He has also noted that those drugs which produce decreases in tibial pressure threshold also increase the incidence of "excitatory phenomena" during thiopental anesthesia. These leads merit investigation. However, until an interpretation of these observations in terms of the pain experience can be established, this laboratory observation should not be labeled "anti-analgesia" lest it be confused by the less discriminating as having pertinence for the patient with pain.

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