Pain Insensitivity in Schizophrenia: A Neglected Phenomenon and Some Implications
by Robert H. Dworkin

Abstract

The literature on insensitivity to pain in schizophrenia is reviewed. Numerous reports indicate that, relative to normals, individuals with schizophrenia are insensitive to physical pain associated with illness and injury. In addition, insensitivity to pain of various sorts administered in experimental studies has been reported frequently in this population. This extensive and diverse literature of clinical and experimental reports suggests that many individuals with schizophrenia are less sensitive to pain than normal individuals. However, because the experimental studies—almost all of which were conducted before 1980—suffer from a variety of methodological limitations, this research provides neither a satisfactory characterization nor an adequate explanation of pain insensitivity in schizophrenia. It is argued that this widely reported but currently neglected phenomenon has important implications for physical health, self-mutilation, homelessness, premorbid development, and affective flattening in individuals with schizophrenia.


It has been recognized for some time that individuals with schizophrenia appear to be relatively insensitive to physical pain. Kraepelin (1919) observed that patients with schizophrenia are often "less sensitive to bodily discomfort; they endure uncomfortable positions, pricks of a needle, injuries ... burn themselves with their cigar, hurt themselves" (p. 34). Bleuler (1911/1950) noted that "even in well-oriented patients one may often observe the presence of a complete analgesia which includes the deeper parts of the body as well as the skin. The patients ... incur quite serious injuries, pluck out an eye, sit down on a hot stove and receive severe gluteal burns" (p. 57).

Of course, some individuals with schizophrenia do report that they suffer pain, most commonly headache (Varsamis and Adamson 1976; Watson et al. 1981; Philips and Hunter 1982; Torrey 1989), but the prevalence of pain complaints in schizophrenia appears to be lower than in other psychiatric disorders (Merskey 1965; Spear 1967; Delaplaine et al. 1978; Watson et al. 1981). It is noteworthy that pain insensitivity in schizophrenia has been described by surgeons and internists as well as by psychiatrists, and much of the attention that this phenomenon has received consists of discussions...
of its often life-threatening implications.\footnote{The terms "pain insensitivity" and "reduced sensitivity to pain" in schizophrenia will be used throughout this article. The phenomenon is not well understood, and it could be argued that other terminology would be preferable—for example, reduced nociception sensitivity, pain nonresponsivity, indifference to pain, and analgesia. The term sensitivity has been used because other terminology would be cumbersome or would make a greater number of assumptions about the nature of the phenomenon.}

With respect to abdominal disease, Marchand et al. (1959) reported that pain was absent as a presenting symptom in 21 percent of a series of schizophrenia inpatients with acute perforated peptic ulcer and 37 percent of those with acute appendicitis. Moreover, whether these patients did or did not complain of pain, most of them came to surgical attention relatively late in the course of their disease (see also West and Hecker 1952). It has been estimated that pain is a presenting complaint in 95 percent or more of nonschizophrenia patients with these disorders (Marchand et al. 1959), and case reports have also documented a marked discrepancy between the severity of acute intra-abdominal surgical disease in schizophrenia patients and the patients' clinical presentations, in which a complaint of pain was often absent (Lewis 1937; Van-derkamp 1970; Geschwind 1977; Apter 1981; Fishbain 1982; Bickerstaff et al. 1988; Katz et al. 1990; Rosenthal et al. 1990).

With respect to heart disease, pain was reported in only 18 percent of a series of schizophrenia inpatients who suffered myocardial infarction but was estimated to occur in approximately 90 percent of normals (Marchand 1955; see also Lieberman 1955, Hussar 1965, Van-derkamp 1970); pre- and postinfarction angina pectoris also appears to be rare in schizophrenia (Hussar 1965). In an analysis of causes of death in a large sample of autopsied schizophrenia patients, Hussar (1966) found that almost one-third of patients over age 40 died suddenly and he attributed this result to "painless myocardial infarction, the rarity of angina pectoris, [and] the uncomplaining patient with some abdominal catastrophe" (pp. 54-55; see also Jetter and White 1944 and Marchand 1958 on the absence of complaints of pain preceding "rupture of the heart" in psychotic patients).

Reduced pain sensitivity in individuals with schizophrenia has been reported for a variety of other medical conditions. Painless fractures (Marchand et al. 1959; Fishbain 1982), third-degree burns (Shattock 1950), cancer (Talbott and Linn 1978), "megacolon" (i.e., an atonic and tremendously dilated colon; Ehrethiel and Wells 1955), peptic ulcer (West and Hecker 1952; Ehrethiel 1957; Hussar 1968), and arthritis (Gowdy 1979) have been described in schizophrenia. With respect to surgery, Arieti (1945) reported that schizophrenia patients rarely complain when wounds are sutured, and Marchand (1959) observed that psychotic patients seldom complain of postoperative pain. Two studies have reported that schizophrenia patients have a lower incidence, duration, and severity of postlumbar-puncture headache than normals (Ballenger et al. 1979; Torrey 1979). In a sample of 31 children with schizophrenia, observed for 1 to 3 years in an inpatient setting, Goldfarb (1958) reported clearly diminished or absent reactions to physical trauma that typically cause pain in normal children, including cuts, bruises, burns, inflammation, and dental work. Although patients with chronic pain (i.e., pain lasting more than 3 months; Merskey 1986) suffer from a variety of psychiatric disorders (Dworkin and Caligor 1988), schizophrenia appears to be very rare in this population. A number of studies have conducted DSM-III (American Psychiatric Association 1980) diagnostic assessments of chronic pain patients (reviewed in Dworkin and Gitlin 1991), but only two cases of schizophrenia have been reported, both of which were in a study of patients with atypical facial pain (Remick et al. 1983).

Reduced sensitivity to pain in schizophrenia has also been studied in the laboratory. Different types of painful stimuli have been investigated and patients' responses to pain have been assessed and analyzed using a variety of methods. Studies have examined the responses of patients with schizophrenia to thermal pain (Malmo et al. 1951; Hemphill et al. 1952; Hall and Stride 1954; Kane et al. 1971; Clark and Mehl 1976; Dworkin et al. 1993a), electrical pain (Bender and Schilder 1930; Parsons et al. 1949; Collins and Stone 1966; Ax et al. 1970; Seppington 1973; Watson and Jacobs 1977; Davis et al. 1979b; Ingvar 1980; Buchsbaum et al. 1984, 1986), cold-pressor pain (Earle and Earle 1955; Maricq and Edelberg 1975; Albus et al. 1982), pin-prick and pressure pain (Stengel et al. 1955; Merskey et al. 1962), painful pinch (May 1948), and imagined painful situations (Petrovich 1960). On the
basis of verbal, avoidance, and/or psychophysiological measures, it has typically been reported that patients with schizophrenia have reduced sensitivity to pain.

The majority of these studies of insensitivity to experimental pain in schizophrenia have a variety of problematic aspects and often lack important methodological information. The methodological problems characteristic of these studies, most of which were conducted before 1980, include small sample sizes, questionable diagnostic reliability, problematic control groups (e.g., college students), lack of careful examinations of medication effects, lack of attention to the distinction between perceptual deficits and pain expression, and limitations of the psychophysical methods used in assessing pain sensitivity.

Moreover, these studies do not provide a satisfactory description of insensitivity to experimental pain in schizophrenia. Specifically, several important questions have not been adequately addressed, including whether experimental pain insensitivity in schizophrenia varies as a function of the type of painful stimulus, whether certain patients with schizophrenia are more likely to have pain insensitivity than others, the degree to which pain insensitivity in schizophrenia reflects sensory and/or affective processes (see below), the nature of the relationships between pain insensitivity in schizophrenia and other psychological and neuropsychological abnormalities that are characteristic of the disorder, and, because few of these studies examined individuals with other psychiatric disorders, whether pain insensitivity is specific to schizophrenia. Methodologically rigorous research on these questions could be conducted, and the results would provide a better understanding of insensitivity to the physical pain of illness and injury in individuals with schizophrenia.

Neither the reports of clinical pain insensitivity nor the studies of experimental pain insensitivity provide a satisfactory explanation of reduced sensitivity to pain in schizophrenia. Although it has been suggested that pain insensitivity in schizophrenia may be a result of neuroleptic medication (e.g., Fishbain 1982), many of the reports describing this phenomenon predate the introduction of these medications. It has been proposed that pain insensitivity in schizophrenia reflects a motor deficit in responding to painful stimuli (Malmo et al. 1951; compare with Hall and Stride 1954), but many of these clinical and experimental reports did not depend on motor responses to document this phenomenon. It has also been suggested that an excess of endorphins in schizophrenia may explain clinical and experimental pain insensitivity and other symptoms of the disorder (Buchsbaum et al. 1980), but the results of research addressing this hypothesis have been inconsistent (Mueser and Dysken 1983; Meltzer 1987).

One intriguing possibility is suggested by recent research indicating that a dysregulation of N-methyl-D-aspartate (NMDA) receptor-mediated neurotransmission might occur in schizophrenia (Javitt and Zukin 1991; Ulas and Cotman 1993). Because NMDA antagonists have analgesic properties and administration of NMDA can cause hyperalgesia (e.g., Woolf and Thompson 1991), it can be hypothesized that a decrease in NMDA receptor transmission accounts for pain insensitivity in schizophrenia. This hypothesis is not inconsistent with the suggestion that arachidonic acid (a second messenger at the NMDA receptor) and its prostaglandin derivatives may play a role in the pathogenesis of negative symptoms in schizophrenia, including pain insensitivity (Horrabin et al., in press).

A variety of other biological, psychodynamic, psychological, and sociological hypotheses have been proposed to explain pain insensitivity in schizophrenia (Talbott and Linn 1978; Jakubaschek and Boker 1991). However, these hypothesized mechanisms cannot be adequately evaluated because of the limitations of both the clinical literature, which with few exceptions consists of case reports, and the experimental studies, few of which have used modern diagnostic criteria. Nevertheless, any consideration of these hypotheses must take into account the finding by Link et al. (1986) that the occupations of schizophrenia patients before onset of the disorder were more frequently “noisome,” that is, characterized by hazards, noise, heat, humidity, fumes, and cold. Link et al. interpret their results as evidence of the importance of stress associated with socioeconomic status in the etiology of schizophrenia (but see Dohrenwend et al. 1992). However, in view of the literature discussed above, it could be argued that reduced sensitivity to painful and aversive stimuli also distinguishes individuals with schizophrenia from normals even before the development of their disorder.

In attempting to account for pain insensitivity in schizophrenia, it will also be important to consider the evidence that patients with mood disorders have reduced pain sensitivity (e.g., Hall and Stride 1954; Davis et al. 1979a;
Ben-Tovim and Schwartz 1981; Dworkin et al. 1993a, submitted for publication). It has been suggested that pain insensitivity in schizophrenia and mood disorders may be mediated by different mechanisms (Davis et al. 1979a, 1980). Because depressed patients often report that they suffer from pain, their relative insensitivity to experimental pain is strikingly similar to the relative insensitivity to experimental pain often found in patients with chronic pain syndromes (e.g., Clark and Yang 1983; Nalibof and Cohen 1989), recalling Hippocrates' aphorism that "Of two pains occurring together, not in the same part of the body, the stronger weakens the other" (Meskev and Evans 1975, p. 77). Indeed, in a preliminary analysis of a small sample of depressed patients it was reported that those patients who reported greater somatic distress during the 3 days before an experimental pain assessment had less sensitivity to the experimental pain than patients with lower levels of somatic distress (Davis et al. 1979a). These results suggest that pain insensitivity in mood disorder (and chronic pain syndromes) may reflect adaptation or other compensatory mechanisms and may therefore provide an important contrast to pain insensitivity in schizophrenia (Davis et al. 1980).

Some Implications

The extensive and diverse literature of clinical and experimental reports of pain insensitivity in schizophrenia suggests that many individuals with this disorder are less sensitive to pain than normals. As discussed above, this literature provides neither a satisfactory characterization nor an adequate explanation of this phenomenon. Our current lack of knowledge about pain insensitivity in schizophrenia, however, is not the only justification for conducting additional research on this topic. An increased understanding of this topic has important implications, not only for the health and well-being of individuals with schizophrenia, but also for other aspects of schizophrenia, such as premorbid development and affective flattening. In considering the following implications of pain insensitivity in schizophrenia, however, it is important to remember that these implications are largely speculative because they are based on a widely reported but not well-understood phenomenon.

Physical Health. In their discussions of clinical findings, the authors cited above have noted that pain insensitivity can be detrimental to the health of schizophrenic patients and can even have life-threatening consequences. Because pain is a symptom of many illnesses, insensitivity to pain can delay recognition of a condition requiring diagnosis and treatment. In addition, pain serves "as a warning signal for impending injuries, and causes most of us, most of the time, to avoid sprains, fractures, burns, etc., that would otherwise leave us crippled and open to severe infection. As a result of early experiences with pain, we now behave in such a way as not to damage ourselves. ... In fact, pain is what keeps most of us from injury" (Sternbach 1968, p. 106).

Therefore, as the clinical literature referred to above (e.g., Marchand 1958; Bickerstaff et al. 1988; Katz et al. 1990; Rosenthal et al. 1990) notes repeatedly, clinicians who are responsible for the medical evaluation and treatment of individuals with schizophrenia must be especially vigilant for illnesses and injuries in which a complaint of pain is typically expected. Otherwise, a "dangerous delay may occur in diagnosing medical illnesses whose presenting symptoms typically include pain" (Rosenthal et al. 1990, p. 322). When clinicians suspect such an illness or injury, they must rely on signs that do not involve a report of pain. Bickerstaff et al. (1988) suggest that the "best line of defense is a high index of suspicion and an understanding of the special diagnostic problems presented by the schizophrenic patient" (p. 51).

Another important implication of pain insensitivity in individuals with schizophrenia is provided by congenital insensitivity to pain, an intriguing but poorly understood phenomenon (McMurray 1975). Life expectancy appears to be considerably shortened in individuals with congenital pain insensitivity with death occurring as a result of the effects of unrecognized trauma and associated infections (Sternbach 1989). However, individuals with congenital insensitivity to pain often develop strategies for avoiding injury, including learning to attend to other cues, such as sensations of tingling, itching, pressure, warmth, and cold, to detect an illness or impending injury (Sternbach 1963, 1968; Thrush 1973). Indeed, Sternbach (1963, 1968) has noted that injuries and burns appear to be less common in adults than in children with congenital pain insensitivity, and he attributes this to an increased awareness of the importance of
such strategies for detecting potential tissue damage. The implication of this for individuals with schizophrenia who have insensitivity to pain is that they could be taught such strategies to avoid illness and injury. Training of this sort would have the potential to ameliorate some of the adverse consequences that pain insensitivity has with respect to physical health in individuals with this disorder.

Self-Mutilation. Self-injurious behavior of various sorts occurs in an appreciable number of individuals with schizophrenia, as well as in those with other disorders (Burgess 1991). Reports of self-mutilation, the most dramatic example of self-injurious behavior, in patients with schizophrenia include descriptions of unilateral and bilateral eye enucleation (Feldman 1988), self-laceration (Shore et al. 1978; Sweeny and Zamecnik 1981), and self-amputation of various parts of the body, including the hand (Schweitzer 1990), breast (Coons et al. 1986), ear (Silva et al. 1989), penis and testicles (Schweitzer 1990), and, in what is arguably the most extreme case reported to date, virtually the entire face (Scheftel et al. 1986). It has been noted that during their acts of self-mutilation, these individuals seem to have been in what has been termed a state of "psychotic analgesia" (Scheftel et al. 1986). Shore (1979) suggests that this absence of pain may be related to the blunted affect that is characteristic of schizophrenia and notes that it would be unexpected for "the emotional aspects of pain to be spared when the affective expressions of fear, anger, love, etc., are so grossly distorted or blunted" (p. 386).

The results of recent research suggest that patients with borderline personality disorder and a history of self-injurious behavior are less sensitive to experimental pain than borderline patients with no history of such behavior (Russ et al. 1992; see also Russ 1992). Although the relevance of this finding to self-mutilation and other forms of self-injurious behavior in schizophrenia is speculative, this finding does suggest that self-injurious behavior may be facilitated by insensitivity to pain in patients with borderline personality disorder and in patients with schizophrenia. Of course, it is also possible that a history of self-injurious behavior decreases sensitivity to experimental pain. Such hypotheses could be addressed not only by comparing pain sensitivity in individuals with schizophrenia who do and do not have a history of self-injurious behavior, but also by examining pain insensitivity in the biological relatives of patients with schizophrenia. If pain insensitivity is found in individuals with schizophrenia who do not have a history of self-injurious behavior and also in their relatives, it would suggest that this insensitivity may be a predisposing factor for, rather than a consequence of, self-injurious behavior.

Homelessness. Pain insensitivity may also play a role in other self-injurious behaviors found in individuals with schizophrenia, including some aspects of homelessness. Individuals with schizophrenia make up a substantial portion of many samples of homeless persons (e.g., Lamb and Lamb 1990; Marshall and Reed 1992). Disabling functional deficits associated with major mental disorders may be important contributing factors to homelessness in individuals with these disorders (Lamb and Lamb 1990), and negative symptoms and housing instability are associated in individuals with schizophrenia (e.g., Drake et al. 1991). Homeless individuals with a history of psychiatric hospitalization were more likely to have been sleeping outdoors, in an accident, injured, and victimized in the previous year than homeless individuals who had never used mental health services (Gelberg and Linn 1988; Gelberg et al. 1988). It is possible that insensitivity to pain in individuals with schizophrenia may make it somewhat less aversive for these individuals to tolerate homelessness and its consequences. Indeed, many aspects of homelessness recall the list of noisome occupational characteristics found by Link et al. (1986) in the premorbid histories of individuals with schizophrenia—hazards, noise, heat, humidity, fumes, and cold. However, many factors contribute to becoming homeless in individuals with schizophrenia (Lamb and Lamb 1990), and any contribution made by pain insensitivity probably plays no more than a very small role in the extremely complex causation of homelessness in this population.

Premorbid Development. The excess of noisome premorbid occupations found in individuals with schizophrenia (Link et al. 1986) may reflect premorbid insensitivity to pain, which could make it more likely that individuals with the predisposition to develop this disorder choose hazardous and aversive occupations (such occupations are typically better paid). Premorbid pain insensitivity in schizo-
Affective Flattening. Affective flattening, which has long been considered a prominent symptom of schizophrenia (Bleuler 1911/1950; Kraepelin 1919; Strauss et al. 1974), plays a central role in recent theories and research on negative symptoms (Andreasen 1982; Crow 1985; McGlashan and Fenton 1992; Knight and Valner 1993) and is the only negative symptom included in major rating scales for the assessment of these symptoms (Fenton and McGlashan 1992). In recent research, affective flattening and anhedonia were the only negative symptoms that predicted long-term outcome in schizophrenia independently of premorbid functioning and the severity of positive symptoms (Fenton and McGlashan 1991), and negative symptoms, including affective flattening, were associated with frontal lobe dysfunction (i.e., “hypofrontality”) in studies using single-photon emission tomography (Andreasen et al. 1992b) and positron emission tomography (Wolkin et al. 1992).

The importance of affective deficits in schizophrenia has been established, but it has been argued recently that current approaches to the conceptualization and assessment of affective flattening have not taken into account social skills deficits and neuromotor abnormalities (Dworkin 1992; Dworkin et al. 1993a, 1993b). Existing approaches to the assessment of affective flattening are based on motor behaviors—for example, eye contact, facial expressiveness, and vocal inflection—during an interview or other interpersonal situation. It is certainly possible that a patient’s behavior lacks these qualities because he or she suffers from an affective deficit. But it is just as plausible that a patient has, for example, diminished vocal inflection or facial expressiveness because he or she suffers from neuromotor or social deficits. Indeed, affective flattening has been found to be associated with both neuromotor (Manschreck et al. 1982, 1985; Butler et al. 1992) and social skills (Jackson et al. 1989; Bellack et al. 1990; Mueser et al. 1990) deficits in schizophrenia.

The possibility that current approaches to the assessment of affective flattening may reflect social skills and neuromotor deficits has important implications for research on the role of affective flattening in the diagnosis, etiology, and treatment of schizophrenia (Dworkin 1992). Given the potential confounding of these variables, it is not possible to make a compelling argument that affective flattening characterizes individuals with schizophrenia. It would be just as valid to conclude that characteristics such as diminished vocal inflection and facial expressiveness are evidence of social or neuromotor deficits. Because the conceptual and measurement overlap among these variables has not been recognized, approaches to affective flattening in schizophrenia that do not reflect social and neuromotor processes have not been developed.

Given the diverse conditions in which pain insensitivity has been reported in schizophrenia, this phenomenon may provide a method of investigating affective flattening that is unconfounded by social skills and neuromotor dysfunction. However, as was recognized by both Kraepelin (1919) and Bleuler (1924), pain insensitivity in schizophrenia could reflect sensory abnormalities as well as affective deficits. Arieti (1945) appreciated the importance of this and noted that it was possible “that pain and temperature sensations are perceived [in patients with schizophrenia], but that only the affective components of such perceptions are lost” (p. 380). In attempting to account for diminished responsiveness to pain in schizophrenia, Arieti “was inclined to believe that the real sensation of pain and temperature is not lost. … However, these patients seem to be unaware of the painful and thermic stimuli and do not show any emotional reaction to them” (p. 382; see also Shore 1979).}

It is interesting that distinctions have been made between congenital insensitivity to pain and congenital indifference to pain [e.g., Sternbach 1963, 1968; Jewesbury 1970; Landrieu et al. 1990]. These distinctions often differentiate the “absence of sensation or failure to receive a stimulus” [Jewesbury 1970, p. 187] from a “lack of concern to a stimulus well received” [Landrieu et al. 1990, p. 574]. Similarly, McMurray [1975] notes that in cases of congenital universal insensitivity to pain the sensory aspects of painful stimuli appear to be preserved, but the motivational-affective processing of these stimuli is abnormal and the aversiveness of the sensation is absent.
The distinction between sensory and affective processes is a fundamental feature of recent theory and research on the measurement of clinical and experimental pain (Melzack and Casey 1968; Gracely and Dubner 1987; Price 1988; Clark et al. 1989; Fernandez and Turk 1992). With few exceptions (Clark and Mehl 1976; Davis et al. 1979b; Dworkin et al. 1993a), investigations of pain sensitivity in schizophrenia have not distinguished these aspects of pain and have not attempted to examine the extent to which sensory and affective processes account for pain insensitivity in this disorder. However, there are well-researched methods of measuring pain sensitivity that distinguish its sensory and affective aspects; these methods could be used in experimental research on pain insensitivity in schizophrenia. A brief overview of these methods follows.

### Measuring Sensory and Affective Aspects of Pain Insensitivity in Schizophrenia

A wide variety of approaches have been used to study individuals' responses to pain in experimental and clinical settings (Chapman et al. 1985), but only two types of methods are available for investigating experimental pain that distinguish its sensory and affective aspects. One approach is based on psychophysical methods, including magnitude estimation and cross-modality matching, in which subjects scale both the sensory intensity and the unpleasantness of painful stimuli using words, numbers, line length, handgrip force, or other methods (e.g., Gracely 1984; Gracely and Dubner 1987; Price 1988). Although the reliability of some of these methods has been questioned (Clark et al. 1989; Fernandez and Turk 1992), they have provided important information about both experimental and clinical pain. However, these methods can be complex and often require well-trained subjects with adequate verbal ability who have had similar prior experiences with pain and are culturally homogeneous (Chapman et al. 1985; Turk 1989). 

At the present time, the use of these methods to measure pain sensitivity in patients with schizophrenia appears to be limited by the logistical difficulties that would be encountered.

The second approach to distinguishing sensory and affective aspects of pain is based on signal detection theory (SDT; also referred to as sensory decision theory or statistical decision theory). This approach to the measurement of pain distinguishes the sensory-discriminative aspects of subjects' responses from the extent to which subjects report their sensory experience as painful. SDT approaches yield two measures, an index of sensory discrimination ($d'$ or $P(A)$), which is related to the functioning of the neurosensory system, and a measure of response criterion ($L_2$ or $B$), which is related to the subject's affective response to the sensory experience, that is, how readily he or she reports pain (Clark 1974). “At a descriptive, or qualitative level, the sensory and emotional components of pain have long been recognized. SDT now permits the quantification of these two components into indices of discriminability and pain report criterion” (Clark and Yang 1983, p. 23). This methodological approach to the measurement of subjects' responses to stimuli was developed in research on sensation and perception (Green and Swets 1966) and has been extensively investigated. These investigations include studies of personality and psychopathology (Grossberg and Grant 1978), medical decision making (McNeil et al. 1975), and experimental and clinical pain (Chapman 1980; Clark and Yang 1983; Fernandez and Turk 1992).

Although SDT has been widely used, this approach to distinguishing sensory and affective components of pain has been controversial. Much of the controversy involves the interpretation of the measures of sensory discrimination and response criterion. It has been suggested that the SDT measure of sensory discrimination may reflect nonsensory processes, including context effects (Craig and Prkachin 1978; Rollman 1979; Chapman 1980) and various other nonsensory factors (Coppola and Gracely 1983; Malow et al. 1989). The SDT measure of response criterion can also be interpreted in different ways (Jones 1979) because it can reflect conceptual, judgmental, emotional, motivational, and sociocultural factors (Chapman 1977, 1980). However, even though SDT may not provide “pure” measures of discrimination and criterion, the extent to which these measures reflect sensory and nonsensory affective processes can be determined by examining hypothesized relationships between these SDT parameters and other types of data (Jones 1979; Yang et al. 1991). Another criticism of the use of SDT in pain research has been that the model has stringent methodological requirements and that data often do not fulfill its strict assumptions (Rollman 1977), but nonparametric data analysis methods satisfactorily address these concerns (McNicol...
Because the experience of pain reflects a complex interplay of sensory and affective processes, it is not surprising that any single approach to assessing both of these components of pain can be criticized. Although “SDT procedures provide useful information about discrimination and response behavior … neither these measures nor those from other direct procedures provide pure measures of sensation or response” (Coppola and Gracely 1983, p. 265); nevertheless, “SDT remains a powerful measure of scaling performance … that can provide significant information” (Gracely 1989, p. 262). At the present time, SDT appears to be the only approach to distinguishing sensory and affective aspects of pain that has been studied in diverse experimental and clinical contexts by different groups of investigators (Chapman et al. 1985; Fernandez and Turk 1992) and that is suitable for research on pain sensitivity in major psychiatric disorders (Clark and Mehl 1976; Davis et al. 1979a, 1979b, 1980).

In a recent report in which SDT measures were used in two studies of pain insensitivity in schizophrenia (Dworkin et al. 1993a), patients with schizophrenia had significantly poorer sensory discrimination of painful thermal stimuli than control subjects, but—unexpectedly, given the literature reviewed above—did not differ from controls with respect to their response criterion for reports of pain. As predicted, however, higher (i.e., more “stoical”) response criteria were significantly associated with greater affective flattening and less intense affective experience (as well as with fewer positive symptoms and poorer premorbid adjustment) in patients with schizophrenia, but not in controls or patients with mood disorders.

The results of this study suggest that pain insensitivity in schizophrenia may reflect affective as well as sensory abnormalities. In addition, the results suggest that pain insensitivity may be a manifestation of an affective deficit in schizophrenia, one that is relatively independent of the social and neuromotor deficits that confound existing measures of affective flattening. The continued development of such approaches to affective flattening would allow this symptom to be studied in a manner consistent with arguments that emphasize the importance of the neurobiological and psychological processes underlying the symptoms of schizophrenia (Strauss et al. 1974; Neale et al. 1985; Persons 1986; Carpenter and Buchanan 1989; Andreasen et al. 1992a; Carpenter 1992).

Conclusions

Continued investigation of pain insensitivity in schizophrenia and its relationship to other biobehavioral aspects of the disorder will lead to an increased understanding of the neurobiological and psychological processes underlying this phenomenon. Research on pain insensitivity also has important implications for physical health and self-injurious behavior in individuals with schizophrenia. Such research may eventually make it possible to ameliorate or even prevent the often extremely negative consequences of pain insensitivity for the well-being of individuals with this disorder. In addition, research on pain insensitivity has the potential to contribute to a better understanding of related aspects of the psychopathology of schizophrenia, including affective flattening.

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Errata

The article by Anne L. Hoff et al. entitled "Anomalous Lateral Sulcus Asymmetry and Cognitive Dysfunction in First-Episode Schizophrenia" (Schizophrenia Bulletin, 18(2):257-272, 1992) contained an error on page 260 (first column, third paragraph, third sentence). The corrected sentence should read as follows: The length was measured beginning at the anterior most portion of the lateral sulcus where it was visualized through to the bifurcation of its ascending ramus (measurement 1). Measurement 2 was the same measurement including the ascending ramus. The authors state that this change does not alter the results of the article in which it was found that female schizophreniform patients had a reduction in the normal asymmetry pattern of left greater than right sulcal length on measurement 2 ($F = 4.07$, $df = 1, 75, p < 0.05$).

In the article by Cornblatt and Keilp entitled "Impaired Attention, Genetics, and the Pathophysiology of Schizophrenia" (Schizophrenia Bulletin, 20(1):31-46, 1994), figure 2 that appears on page 41 should have included the following credit line: Reprinted with permission from Cornblatt, B.A.; Lenzenweger, M.R.; Dworkin, R.H.; and Erlenmeyer-Kimling, L. Childhood attentional dysfunctions predict social deficits in unaffected adults at risk for schizophrenia. British Journal of Psychiatry, 161(Suppl. 18): 59-64, 1992.

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