Chapter 2
Pain in Mental Health: Myths

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Pain is a concept that has evolved over time. From antiquity to the twenty-first century, from Europe to Africa and whatever religions or doctrines, pain inspired the most varied behaviors and opinions. To varying degrees, philosophers, theologians and writers have sublimated it by giving some redeeming value or have exalted its greatness and acceptance.

Pain is an individual, subjective experience. It is a well-known and frequent clinical reality. The new scientific knowledge allows a better understanding of the main mechanisms and the necessary support for the painful symptoms that is now an essential part of any good health practice.

However, in the field of mental health and pain we are at the beginning of a complex course, which has attracted many beliefs, such as the supposed higher pain threshold of some patients suffering from mental illness. Because the expression of pain is significantly altered in specific pathophysiology such as schizophrenia, our understanding of pain in mental health becomes even more complex. Too often, painful complaint is interpreted as a clinical sign of the psychiatric symptoms in the mentally hill patients such as if a somatic painful condition was not possible.

2.1 Conceptions of Pain

Most of the studies on pain in schizophrenia are reporting atypical behavior that lead to interpret it as a form of hypoalgesia [14, 16]. This conclusion probably derived from early clinical observations in psychiatry of pain insensitivity in patients

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S. Marchand et al. (eds.), *Mental Health and Pain*,
DOI 10.1007/978-2-8178-0414-9_2, © Springer-Verlag France 2014

suffering from schizophrenia. However, these reports are often poorly structured, probably because of the lack of understanding of concepts governing this phenomenon. Thus, a number of authors have consistently reported the difficulty for schizophrenic patients to express or perceive painful stimulation. However, these observations were not followed by changes of attitude or solutions to avoid the consequences of such a problem.

2.2 The First Authors

Foucault [15] based on the writings of the classical age, described in "Histoire de la folie" the animality of a mad: *"The animality, in effect, protects the mad against everything that may be fragile, sickly in human. Animal strength of madness, and the thickness it borrows to the blind world of animal, hardened the mad against hunger, heat, cold, pain."*

It was common thinking until the eighteenth century that the mad can endure the miseries of existence. There is no need to protect them; we do not cover them, nor heat them.

The ability of the insane to support, like animals, the worst weather, will be for Pinel [34] a medical dogma. He described *"Constancy and ease with which some insane, of the two genders, support the most severe and prolonged cold. In the month of Nivôse, of the year three, during some days when the thermometer showed 10°, 11° and up to 16° below the ice, an insane from the Bicêtre hospital could not keep his blanket, and he sat on the frozen floor of the lodge. In the morning, we opened the door when we just saw him in shirt running in the courtyard, taking the ice and snow with hand, applied it to the chest and let it melt with a kind of delight. Madness, for all that it can contain of animal ferocity, preserves the human from the danger of diseases; it give him access to invulnerability, similar to that of nature, in its foresight, has spared animals."*

In 1874, Kahlbaum [22] published a book in which he raises the issue of insensitivity to pain in different types of mental illness, particularly catatonia. He studied many cases and organized the concept of insensitivity to pain as a well-defined syndrome. He does not, however, propose satisfactory hypotheses to explain this phenomenon. In cases of melancholy, he noticed that deep punctures could be made without the patients express the slightest reaction of pain. He notes, however, that this lack of response to pain is not present in all cases. He proposed that in many cases, it is more a motor incapacity to react than a real analgesia.

In 1896, Pellizzi reports in his article [32] several observations of patients with an important reduction in sensitivity to pain. This insensitivity is found both in the schizophrenic patients and in melancholic patients. Among these observations, there are also several cases of self-harm. He comes to the conclusion that self-harm is often a way for the patient to "*divert his attention from delusions or hallucinations*". In the case of melancholy, he hypothesized that the patients probably feel the pain, "*but they rarely react because they are unable to leave their self withdrawal*".

This author considered that the lack of response or expression to pain was related to hallucinatory productions and inability to react of these patients.

In 1919, Kraeplin [25] observed that patients with early dementia are "*often less sensitive to body discomfort; they endure uncomfortable positions, wound… to burn themselves with cigarettes and self-harm.*" Bleuler [3] also noted frequent analgesia without anesthesia… "*They live in a fantasy world from their sensory disturbances.*"

2.3 Clinical Studies

Numerous authors have described cases of insensitivity to pain over time, depending on their anatomical location or specificity [2, 12, 14].

Thus, Marchand et al. [30] identified the occurrence of three conditions in psychotic patients: perforation of peptic ulcer, acute appendicitis and femur fracture. There was no reported pain in 19 out of 46 patients with schizophrenia on the femur fracture, 3 cases out of 14 for perforated ulcer and 7 cases out of 19 for acute appendicitis. In total, 37 % of patients showed no painful complaint at the onset of their illness.

Rosenthal et al. [37], after reviewing various articles, attributed the absence of pain alleged by the patient to multifactorial, psychological and biological entanglement.

Observations of insensitivity to pain have also been reported in schizophrenic patients during painful medical conditions. The absence of pain in cases of myocardial infarction is a phenomenon repeatedly described in the literature, and the majority of authors agree to an average of 10 % of the cases not reporting related pain to cardial infarction. Concerning infarction, the work of Marchand [27] is interesting. He identified 83 patients being divided into 32 cases of old myocardial infarction, and 51 cases occurring during the observation period. In none of the 32 cases of old myocardial infarction, clinical or pain signs have fostered a reported clinical care. Of 51 patients, there were 26 patients with schizophrenia. In 82.5 % of cases, the infarct was painless in the initial phase and in 67.5 % of cases at 24 h. For the author, these results are associated with the loss of understanding of the meaning of pain in these patients.

Similarly, Hussar [20] conducted autopsies on recently deceased schizophrenic patients. He found that a third of patients over the age of 40 years died of sudden death. This result supports, among other causes, the absence of pain in myocardial infarction, absence of angina pain and absence of painful complaints in abdominal pathologies.

The absence of painful complaints has been widely described in cancers and arthritis. Marchand [28, 29] found that psychotic patients had virtually no postoperative pain. This decrease in tenderness has also been observed in a number of painful situations not related to pathological processes: often including severe burns caused by cigarettes or hot radiators.

2.4 The Painful Complaint in Schizophrenic Patients

Literature is less abundant in this area. Spontaneous pain can be observed in the form of hallucinations. The hallucination in this case could be secondary to a delirium of suffering from a disease causing pain. The complaint most frequently expressed concerns headache. Watson et al. [38], by analyzing various studies, concluded that "*headache and sometimes other pains are present at the initial stages of schizophrenia while analgesia, or decreased sensitivity to pain, are the hallmarks of chronic schizophrenia*." From all studies, we note that the prevalence of pain complaints in schizophrenic patients was much lower than that of patients with other psychiatric disorders.

2.5 Comparative Studies

The desire to objectify the reported insensitivity to pain in schizophrenia has encouraged teams to develop a number of experimental studies. There is a big difference in both the variability and interpretation of results. Different types of painful stimuli were studied. The responses obtained were analyzed differently depending on the type of stimulus and authors. The different stimuli were heat, cold, electricity, injection and painful pressure.

Dworkin [11] listed some important critics about these studies:

- On the methodology;
- On the diagnostic reliability;
- On small samples;
- On the associated medications and their effects;
- The lack of distinction between perception and expression of pain;
- No formal proof of the reality of insensitivity to pain in these experimental studies;
- Lack of description of the clinical form of schizophrenia;
- No discrimination of sensory, emotional or motor aspects of pain.

Numerous studies have been conducted on the expressions and reactions of the schizophrenic patient to pain. Some works came from the Signal Detection Theory [5, 12]. This approach to pain measurement distinguishes, on the one hand, the ability of sensory discrimination of the subject, and on the other hand, the quantitative and subjective evaluation by the subject of his painful experience using a categorical scale. The ability of sensory discrimination is related to the neurophysiological functioning, while quantitative painful experience depends on psychological factors.

One of the first studies was that of Bender and Schilder [2] on 60 catatonic patients receiving an electrical stimulus. These authors studied the possibility of eliciting a nociceptive reflex in these patients. The experimental protocol consisted

of an electrode placed on the palm of the hand, the other was in contact with the fingertips. Before the electric shock, the patient was warned by a bright flash, or in non-responsive patients, with a touch of the skin on the forehead. The amplitude of the electric shock was not reported in the study.

The authors concluded that:

- The response to painful stimulation was often delayed and incomplete;
- The response is based on the quality and strength of the stimulus;
- The response is local, in the form of stiffness;
- The overall body response did not exist in these patients;
- Emotional responses or increased respiratory rate can be observed;
- Avoidance response is not predictable;
- There was frequent spontaneous repositioning of the hand, independently of the responses caused by stimulations.

The authors found that the defense response observed is most often a partial response implicating only a localized response. The pain is no longer seen as a global phenomenon, but only as an unpleasant sensation confined to the stimulated area and most of these patients adopt a passive attitude. The authors proposed that the non-responsiveness to pain was the result of a primary organic modification, and that malfunctions result in a decrease in integration capabilities. They emphasize that such a change in the response to pain may be a reflection of a significant impairment of mental functioning.

Another interesting study is from Collins and Stone [7]. They found that pain responses of schizophrenic patients were related to certain parameters. One of the parameters was the general activity of a patient measured by an activity scale. They pointed out that the responses to pain were amplified when the patients activity were greater or below average. Another parameter influencing the response to pain, according to these authors, was the age of the patients. Younger and older patients were more responsive. This variability according to age was attributed to uneven neuroleptic dosages of these populations.

The aim of the study was to reassess the relationship between pain sensitivity and general activity in chronic schizophrenic patients. Eighteen male (20–54 years old) chronic schizophrenic patients were included in this study. They received no treatment at the time of experiment. The experimental protocol was to deliver electrical stimuli of increasing intensity. After each stimulation, the investigator asked the patient to classify his perception among the following three answers:

- Not perceived at all;
- Perceived as a painful stimulation;
- Perceived as very intense pain, almost unbearable.

This experiment was repeated for each patient weekly for 5 weeks. During those 5 weeks, the activity of these patients was observed and quantified by the health care team using an activity scale. This scale consisted of 20 items measuring the movement from one place to another, the movements performed without walking and the intensity of these responses. The authors measured the perception threshold, the

pain threshold and the tolerance threshold. The responses were stable over time for each patient. There was no link found between the pain thresholds and the level of activity, and no relationship between age and the different thresholds.

The same study was conducted in 50 healthy U.S. Army subjects aged between 18 and 53 [7]. Pain sensitivity was not correlated with age. However, the pain threshold was related to age, in a linear and curvilinear manner. The older the subjects, the lowest were pain and tolerance thresholds. In this article, the populations tested (schizophrenics/soldiers) were aged matched. Comparison tests between the two samples on the thresholds of pain and tolerance showed a non-significant difference. However, there was a significant difference when the comparison tests were applied only between pain thresholds of the two samples. Thus, the average threshold of pain of schizophrenics was 0.600 mA against a threshold of pain in healthy subjects of 0.300 mA. The authors' proposed that the control group of soldiers, even if matched to the sample of schizophrenic patients for age, included 50 % of subjects belonging to racial minorities and that this could have had an effect on the results observed. However, the finding of a significant difference between the pain thresholds of two samples suggests an overall decrease in pain sensitivity in schizophrenic patients. But because of the small number of cases, further studies seem necessary to refine these conclusions.

Other studies have used different stimulation and measurement modalities [18, 19]. The experimental protocol consisted of placing the patient in a comfortable position so that his blood pressure was stable. Under these conditions, the patient's right hand was immersed in a water bath at 37 °C, while the blood pressure was measured every minute for 5 min. The hand was then transferred into a water bath at 4 °C, and blood pressure was collected every minute for 5 min. This manipulation causes an increase in systolic blood pressure of 20 and 15 mmHg for diastolic blood pressure. This protocol has been studied in populations in psychiatric populations with contradictory results. Through these studies, the change in blood pressure requires integrity of pain pathways to be observed. Any alteration of these pathways at any level whatsoever, produces abolition of the effect on blood pressure, thus inhibiting the response linked to the 'cold pressor test' (test of immersion in cold water).

This test has been used in patients with schizophrenia. Examples include the study of Earle and Earle [13] in which 36 schizophrenic patients and 10 psychotic patients with 15 control subjects were included. These authors showed that over a third of schizophrenic patients (36 %) had no response to the cold pressor test. Schizophrenic patients showed no neurological abnormality. Their autonomic system allowed a positive response to emotional stimulation tests, which proves the emotional integrity of this system. The authors conclude that it is at the higher level of the interpretation of the meaning and the integration of sensory information that dysfunction seemed to fall.

Based on works concerning the threshold of pain, some authors proposed to study the withdrawal reflex of the lower limb (RIII) during percutaneous electrical stimulation of the sural nerve. Willer's work [39] showed a good correlation of the withdrawal reflex with subjective perception of pain. He argues that there is a

correlation between the magnitude of the muscular response and the intensity of perceived pain.

Guieu et al. [17] decided to evaluate this experimental method. Ten patients participated in this study, aged 20–54 years old. Of these patients, three were suffering from paranoid schizophrenia, three from hebephrenic schizophrenia and four cases of schizophrenia. The originality of this study is that all patients had given their informed consent and were 'naive' of any antipsychotic, anxiolytic and analgesic treatment for 30 days. None of the patients showed signs of peripheral neuropathy. Each patient received three sets of five stimuli each at increasing and decreasing intensity. Each intensity level was tested six times. The results supported that there was no significant difference between patients and control group. In addition, this study shows a good correlation between the RIII and pain perception in patients. Based on theses results, the authors attribute the apparent patients hypoalgesia to a kind of indifference to pain more than insensitivity. Malmo et al. [31] demonstrated that pain reactivity in schizophrenia was correlated to autonomic responses such as heart rate and blood pressure increase.

Clinical observations report the absence of a painful complaint in a number of usually painful situations. The uses of experimental pain in experimental contexts studies aimed at verifying if these patients are really hyperalgesic, without too much success. The assessment of pain in schizophrenic patients depends on a number of parameters, not only from one subject to another, but also in the same patient over time. There are still needs for studies on the phenomenon of apparent analgesia or hypoalgesia.

2.6 Hypothesis for the Reported Hypoalgesia

2.6.1 Biochemical Hypothesis

A biological model was proposed assuming that there is an increase in brain activity of opioids, particularly endorphins, a factor that may be involved in this apparent insensitivity to pain in schizophrenic patients.

The level of endorphins of schizophrenic patients was measured in blood and cerebrospinal fluid by different authors and was compared with healthy controls [9, 10]. The results of these studies were contradictory. One of the studies focused on three schizophrenic patients in whom naloxone, an opioid receptor antagonist, was given to block the activity of endorphins while electrical stimulation was applied [33]. Pain perception was normalized with the administration of naloxone.

Studies on the level of endorphins in schizophrenic patients were published by Brambilla et al. [4]. The results were contradictory; the endorphin level in the cerebrospinal fluid was found high, normal or low. In fact, we are confronted with a plethora of interpretations based on uncertain measurements. Brambilla et al. [4] then tried to conduct a study with more refined measurement methods. They tested

the stimulation and inhibition of secretion of beta-endorphin, beta-lipotropin and ACTH, finding a significant difference between schizophrenic patients and the control group. The level of opioids was significantly higher in the cerebrospinal fluid of chronic schizophrenic patients and blood level of beta-endorphin was correlated with pituitary secretion of beta-lipotropin.

2.6.2 Glutamatergic Hypothesis

Studies have also discussed the possibility that a deregulation of NMDA glutamatergic system (receptors activated by N-Methyl-D-Aspartate) may explain in part some symptoms in schizophrenia [21]. Because of the analgesic effect of NMDA antagonists and the hyperalgesic effect of NMDA agonists, a decrease in the number of NMDA receptors or in the transmission capacity of these receptors may play a role in schizophrenia symptoms. Future research on the role of NMDA receptors in pain perception and mental health are of interest.

2.6.3 Influence of Neuroleptics

Various authors have studied the influence of neuroleptics in reducing the sensitivity to pain. Kocher [23] states that neuroleptics could act as analgesics and potentiate their effects. The author interprets the analgesic effect of these drugs by dissociation of the mental representation of pain. It evokes a kind of asymbolia, a loss or reduction of the mental amplification of the pain phenomenon. One of the levers of action of these molecules is on the affective components of pain. Haloperidol is able to bind to opioid receptors, and this could explain the analgesia of schizophrenic patients taking these drugs. It implies that this molecule has an activity similar to morphine [6]. However, a recent meta-analysis from Potvin et al. [35] permitted to conclude that the reported hypoalgesia in schizophrenia was independent of the use of neuroleptics.

2.6.4 Psychopathological Hypotheses

Data from the literature found a decrease in behavioral reactivity to pain in schizophrenic patients, but provide no evidence of a real analgesia. The assumption that one can formulate for this decrease in behavioral reactivity to the pain seems to be a different mode of expression of the pain associated with schizophrenic pathology. Namely, communication disorders and social adaptation [8], disorders of body image [24], and some cognitive disorders as thought disorders and disorders related to the management, expression and recognition of emotions [1]. Painful stimuli result in a physiological and psychological stress that cannot be discharged by the usual modes of regulation and behavioral expression of pain.

Stress can also be a factor distorting the perception of pain, resulting in impaired behavioral reactivity to pain in schizophrenia. The existence of this decrease in behavioral reactivity to pain in schizophrenia, even if not related to a real endogenous analgesia, can be a serious risk factor of not reporting adequately or rapidly enough life-threatening organic diseases. Premature death observed in schizophrenic patients may be related to this difficulty in interpreting and reporting important pain signals.

2.7 Current Clinical Research

Based on these conflicting results between the reported clinical cases of hypoalgesia in schizophrenia and the apparent lack of differences in experimental research, Marchand and colleagues [26, 36], refined the methodological approaches to understand the painful experience of the schizophrenic patient. To determine whether hypoalgesia is explained by hypoactivity of excitatory mechanisms or inhibitory mechanism hyperactivation, these authors measured pain perception and spinal (RIII) excitatory mechanisms through a protocol of temporal summation of pain and inhibitory mechanisms by the efficiency of diffuse noxious inhibitory control (DNIC) in schizophrenic patients and controls. They found that the patients suffering from schizophrenia presented an inhibitory response comparable to the control group, suggesting a normal inhibitory control. However, they had a lower pain threshold, but an absence of perceived temporal perception. The spinal temporal summation activity was comparable to healthy subjects, suggesting a supraspinal effect.

It can be concluded that schizophrenic patients experience pain but do not have this pain awareness signal, which is an adaptive phenomenon to avoid injury due to persistent stimulation [26, 36].

2.8 Conclusion

The literature review showed a probable decrease in pain perception in patients with schizophrenia but no real analgesia. The relationship between pain and mental health seems to be emerging now, thanks to recent works on excitatory and inhibitory mechanisms that interact to modulate pain information and produce the perception of pain. The perception and communication of pain in patients with schizophrenia have important clinical implications and can be related to physical risk or even be life-threatening.

Often, it is not allowed for the patient to speak about his pain, in his own language, about his suffering, his disease, if his speech does not fit in our organic and anatomical references.

The approaches we have with patients suffering from mental health disorders make sterile the too categorical organic-psychic opposition. How can we help our patients to talk about pain when they suffer and with which words?

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