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# THE USE OF SODIUM AMYTAL IN THE ASSESSMENT AND TREATMENT OF FUNCTIONAL OR OTHER DISORDERS

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Sodium amytal (SA; sodium amobarbital), a medium action barbiturate, was first synthesized by the Lilly Company around 1927.[74] This agent quickly came to be used in many contexts, with many different patient populations, and for several different purposes. SA was approved by the Food and Drug Administration (FDA) in 1938 and is currently DEA schedule II. Perry and Jacobs[77] recommended injecting a 5% solution of SA (500 mg of the drug dissolved in 10 ml of sterile water) at a rate of IV infusion no faster than 50 mg/min or 1 ml/min. The infusion is to be continued until the "sedation threshold" is reached, i.e., sudden relaxation with slower and more regular breathing, slight slurring of speech, etc. At this point, the patient may be prompted to talk (if mute), move or feel a limb (in case of a motor or sensory deficit), remember (if amnestic), etc. While some contraindications or risks have been suggested (e.g., chronic obstructive pulmonary disease/respiratory depression), there are very few reports of any untoward or adverse effects,[20,77] even on repeated administrations,[8,20,30,37] although the potential for abuse, especially when used as a sreet drug, clearly exists.[67]

The first section of this article reviews the early history and use of SA, largely involving psychiatric cases, and its continued use in this domain. Several issues that developed out of this experience are highlighted. The next section examines the use of SA in assessment and treatment of chronic pain. It should be noted that the personal experience of the authors using this drug is

with chronic pain patients, and it is this population with whom we are primarily interested. The third section looks at possible mechanisms of effect, reviewing pharmacological or behavioral-pharmacological studies that might bear on this issue. The final section summarizes the utility of SA procedures in the assessment or treatment of functional and other disorders, reconsiders at greater length what may be the meaning of a functional disorder, and presents recommendations for future research.

Although we will return to the issue later, it should be clarified what might be meant by the terms "functional" and "organic," which will be repeatedly used in this discussion. The latter is generally attributed to diseases/disorders associated with detectable peripheral or central pathology. The confusing term "functional" has been associated with a host of concepts and symptoms (including pain) where a psychological or behavioral component contributing to presentation is typically implied.[79,87] However, we believe that, in most cases, both biomedical and psychosocial factors play a part in what is traditionally thought to be a functional disorder. An undue emphasis on, or ignorance of one or the other, of these factors risks the considerable problems and pitfalls of mind-body dualism.

## USE OF SA IN ASSESSMENT OR TREATMENT OF PSYCHIATRIC OR OTHER CONDITIONS—PAIN EXCLUDED

Bleckwenn,[8] one of the first to describe the beneficial effects of SA, administered this agent to psychotic patients with severe insomnia in order to produce sleep. On waking, many of the patients had intervals of 2–14 hours' duration when they were lucid and without catatonic symptoms. Bleckwenn and others[72] suggested that the principal value of SA was in producing prolonged periods of sedation or sleep, providing for an opportunity for "mental and physical rest." However, Lindemann[44,45] found that even smaller doses of SA that were incapable of inducing sleep were sufficient in relieving the symptoms of catatonia or depressive stupor (e.g., rigidity, muteness, inability to communicate). Several studies have subsequently confirmed that there may be beneficial effects on *catatonia* or *muteness* associated with psychiatric/schizophrenic causes[1,14,18,33,34,52,62,90,93,94,99] including a recent randomized blind comparison of the effect of SA versus placebo.[66] In contrast with "functional" cases, SA may not be helpful in cases of "organic" catatonia,[95] i.e., catatonia due to neurological lesions. Some reports as well indicate that SA may be useful in cases of suspected aphasia or muteness arising from entities other than schizophrenia and affective disorders.[47,77] Indeed, Raines and Cohn[80] had early on noted that the inhibition of verbal behaviour in reversible conditions is different from the disturbance of language accompanying focal brain lesions.

There have been numerous reports of beneficial effects of IV SA administration in several other disorders that are often considered functional. Herman[27] had early on described the use of SA interviews in resolution of cases of *psychogenic amnesia* or *fugue states* (i.e., loss of identity or memory for life events) that had been refractory to other methods. Since then, several other reports have confirmed this observation.[6,10,36,41,61,68,86,102] Furthermore, SA administration has been reported to help discriminating the organic from the functional component in cases where both co-exist.[6,17,29]

SA administration has been reported to resolve sensory or motor problems often considered to be symptoms of a *conversion disorder*. Early on, Lambert and Rees[41] reported that barbiturates were more effective than psychotherapy or hypnosis in the treatment of motor conversion disorders. During the SA interview, suggestions to move the affected limb or passive movement of the affected limb was



utilized until patients could use the limb normally in the fully awake state. Several other reports have confirmed that administration of SA resolves, at least temporarily, functional paralysis or hemiplegia[9,11,32,69,71,103] or other movement disorders with a functional component.[20] Hernandez-Peon et al.[28] reported that sensory function and evoked potentials normalized on administration of barbiturate in an "hysterical" patient with a hemianesthetic leg. Furthermore, several case reports have shown that SA procedures were helpful in assessment or treatment of psychogenic deafness,[3,41] blindness,[41] convergence eye spasm,[84] psychogenic seizures or "spells,"[20,63] visual disturbances,[75] pseudodementia,[31,63,82] and multiple personality disorder.[21] It has also been suggested that certain responses (e.g., disorientation or other neurologic symptoms) that become manifest on administration of SA are indicative of organic brain disease or lesion[35,92,100,101]; therefore, SA administration may assist in diagnosis of central nervous system lesions as well.

SA is reported to be a useful adjunct to *psychotherapy*. Frequently used terms for the administration of SA for such purposes include the SA (or amobarbital) interview, narcoanalysis, and narcosynthesis.[12,30,39,48,74,93] In brief, it has been suggested that SA infusion may be helpful in uncovering material that would otherwise not be disclosed (e.g., material that has been repressed in association with unpleasant or intolerable experiences) by providing an avenue for emotional abreaction and desensitization. Such material may then be re-integrated into a more mature or healthy mode of functioning. Particular interest in the use of such procedures exists in cases of post-traumatic stress reactions.[15,24,26,77] Nevertheless, some studies suggest that SA administration is not helpful in the interviewing or psychotherapeutic process.[18,88] Some authors, as well, believe that the benefits of SA administration are maintained only with ongoing psychotherapy, while others have suggested that the same results can be obtained with psychotherapy alone, although this may take longer.[77]

SA administration has been considered useful in *forensic* applications such as the evaluation or determination of "*truthfulness*" (giving rise to the popular misnomer of SA as the "truth serum" in various movies). Most literature reports, however, caution that this technique does not produce unequivocal results and that there may be difficulty differentiating between material that is factual or the product of fantasy.[23,40,43,49] While several reports have suggested that malingerers may have a typical reaction to SA, the indicators appear inconsistent, these behaviors under SA may appear with no other indications of malingering, and alternative explanations may apply.[7,13,41,51,71] One should therefore be quite careful in making the diagnosis of malingering solely on SA-based interpretation of behaviors observed during the procedure.

Several researchers have suggested that the effects obtained with administration of SA are associated with personality and/or situational factors[1,4,18,19,46,71,94] including interviewer characteristics and the quality of the doctor-patient relationship.[26,88]. Hart et al.[26] noted that more drug is needed for hostile, bitter, or resentful patients. The authors suggested that the amount of the drug administered is a good index of resistance to narcosis and that psychological tension seemed to raise the threshold to narcotic effects, like pain raises the threshold to morphine effects. Shagass,[85] in a series of studies with large numbers of patients and normal controls, reported that the sedation threshold on administration of SA was positively related to the degree of anxiety or tension as well as certain frontal EEG indices. In these studies, obsessional personalities were thought to have high sedation thresholds and hysterics low thresholds. However, subsequent studies were unable to replicate these findings.[2,104]

In terms of duration of the SA effects, Lang[42] had observed that subjects relapsed to their previous states on termination of SA infusion and cautioned that the response of an individual cannot be predicted. Several authors report a limited effect after SA administration, lasting approximately 2–3 hours, provided the subject has not fallen asleep.[1,67] However, long-lasting or even permanent beneficial effects have been reported in some cases.[20,32,47,86,93,102]

## USE OF SA IN ASSESSMENT OR TREATMENT OF CHRONIC PAIN AND RELATED PROBLEMS

The early literature contained a few reports regarding the effects of SA on chronic pain patients. These papers reported some increase in pain thresholds, although details were not given[44,48] and a beneficial effect was seen in cases of backache or functional limp.[26] More recently, isolated case reports[22,32,73,77] presented patients with post-traumatic or other pains and a host of systemic and emotional symptoms who responded to infusions of SA. Fackler et al.[20] conducted a retrospective chart review of serial SA infusions in 21 patients, some of whom had chronic pain. Temporary or more lasting benefit was reported in some cases.

SA infusions have been used for the past 30 years consistently at the Toronto Western Hospital (TWH) in Toronto, Ontario, Canada, for assessment of chronic pain patients. Shoichet[83] presented his experience with 75 consecutive chronic pain patients and described behaviors under SA that he believed were consisted with "organic," "psychogenic," "mixed" pain or "malingering." Shoichet distinguished those behaviors as follows: (1) "non-organic" or "psychogenic" pain was markedly reduced usually in the early phases of SA administration, associated with marked improvement in symptoms and pain behavior; (2) "mixed" pain (combination of organic and psychogenic pain) is reduced to a moderate to significant degree while continuing signs and symptoms of physical pathology remain even under the deeper stages of sedation; (3) "organic" pain is only slightly reduced under SA, while objective symptoms and signs remain even under deep sedation; and (4) in "malingering," subjective symptomatology usually increases in a conscious effort to maintain complaints under weakening of subconscious defenses. Mailis et al.[53] compared a structured SA interview (based on behaviors described above by Shoichet) with the initial clinical assessment or the final decision of a multidisciplinary pain team regarding the presence and degree of "organic" and/or "psychogenic" factors in 111 chronic pain patients. They concluded that SA is a quick, safe, and cost-effective procedure that can serve as an adjunct in the diagnosis of chronic pain problems.

The Toronto Western pain group has subsequently studied systematically the responses of different types of pains to SA administration as follows:

1. The first observation of a beneficial effect of barbiturates in chronic pain arising from damage of the nervous system (neuropathic pain) was made by Tasker et al.[91] These authors reported that 82% of a group of patients with pain from brain lesions or spinal cord injury responded quite well to 50–225 mg of IV pentothal (a short action barbiturate), compared with 55% response to IV opioid administration. Mailis et al. subsequently undertook a systematic investigation of the response of *neuropathic pain* to SA.[54–56] Mailis[54] reported on two patients with CRPS type I and II, formerly known as reflex sympathetic dystrophy and causalgia, respectively,[70] who temporarily lost their touch-evoked pain (allodynia) under IV infusion of SA. We also observed a patient with CRPS II after posterior tibial nerve damage who lost his touch-evoked pain (allodynia) while his foot warmed up dramatically during SA infusion (with the effects lasting 8–10 hours). This further prompted a literature review regarding possible *thermogenic* effects of the barbiturates. Only an isolated case report was found of a patient with hand pain attributed to CRPS I who obtained pain relief under SA while the limb warmed up considerably as documented by thermography.[78] Mailis et al.[55] subsequently studied 8 normal volunteers, 15 patients with CRPS type I and II, and 13 patients suffering from other than CRPS disorders with infusions of SA. The drug was administered at a rate of 50 mg/ml/min and a maximal dose of 250 mg. The CRPS patients were characterized by severe and diffuse limb pain and widespread sensory, sudomotor, and vasomotor abnormalities, not confined to the site of injury or the distribution of a peripheral nerve. The CRPS patients proved, indeed, to have a widespread tendency to significantly warm up many of their limbs (and not just the symptomatic one) after SA infusions but not after normal saline. This effect was not seen in normal subjects or patients with other than CRPS pain. The thermogenic effect of SA was attributed to the influence of the drug on sympathetically mediated cutaneous thermoregulation, secondary to CRPS-induced alterations of the sympathetic nervous system.

A second study[56] focused attention on the response of different types of pains to IV SA, i.e., spontaneous pain and stimulus-evoked pains (to pinprick, light touch, cold and deep pressure), in 17 neuropathic pain patients (16 with CRPS and one with central pain syndrome). All patients were given SA 4–7 mg/kg of body weight intravenously over a period of 7–10 min (50 mg/ml/min, maximum 500 mg) preceded by normal saline infusions of similar duration. The patients had signed an informed consent stating the following: "I understand that I will receive IV administration of two of the following: lidocaine, normal saline, phentolamine or sodium amytal. I understand that I will not be told which drugs I will receive or in which order. I also understand that I may feel no effect at all or that I may become lightheaded, dizzy, develop a stuffed nose, may feel relaxed, happy or sad, and that my pain may increase, decrease or not change at all." Therefore, the patients (but not the primary investigator) were blinded as to the drugs they received. Since the instructions were neutral (i.e., giving general but not specific information for each drug and indicating both positive and negative effects as possible outcomes), the patients did not know what exactly to expect. Still photography and videotaping were obtained for each pair of infusions after another signed consent. After SA (but not normal saline infusion), there was a significant but partial improvement of spontaneous pain and dramatic reduction of allodynia. Pinprick and cold hyperalgesia as well as algometric pressure thresholds (as a measure of deep pain primarily arising from bone periosteum) were much less changed or not at all. In several patients, sympathetic ganglion blocks with local anesthetic, A-fiber ischemic block, and spinal stimulation (the latter in one patient) produced effects identical to those observed during SA administration. The authors concluded that IV SA administration allowed for the discrimination of two pain components in neuropathic pain patients: a cutaneous one (touch-evoked pain) mediated by large fibers as a product of "central sensitization," and a deep pain component mediated by nociceptors (pain receptors). Central sensitization, as opposed to peripheral sensitization of the nociceptors after inflammation, denotes an abnormal degree of amplification of the incoming sensory signal in the central nervous system. An example of this is touch-evoked pain or allodynia that arises from low threshold Aβ touch-sensitive fibers.[76]

The superiority of IV SA as an analgesic compared with IV lidocaine was recently shown as well in 6 spinal cord injury patients, 74% of whom reported substantial pain relief with SA as opposed to 45% relief obtained with IV lidocaine.[5] Both IV procedures were single-blinded with SA or lidocaine following normal

saline infusion as described previously.[56] Furthermore, pain relief was accompanied by modification of hyperesthesia in 4 of 6 patients administered SA and only 1 of 5 patients administered lidocaine.

The effect of SA infusion in neuropathic patients with allodynia and pinprick hyperalgesia is illustrated in the case report[56] presented in Figure 1. The patient had undergone transaxillary sympathectomy for pain associated with CRPS I of the right hand. Subsequently, he developed severe chest allodynia and chest wall spontaneous pain. Prior to SA infusion, a very large area of allodynia surrounded by pinprick hyperalgesia was noted and marked (Fig. 1*A*). Subsequent to 250 mg of SA infusion (but not after blinded normal saline infusion), allodynia disappeared, allowing for a small area of numbness in the territory of the surgical scar to be disclosed. Furthermore, the pinprick hyperalgesia "shrunk" to the territory of the intercostobrachial nerve of the arm (Fig. 1*B*). Up to the end of the infusion, the patient remained alert and euphoric with no signs of sedation. Based on this (and repeated other blinded SA interviews with similar results), the diagnosis of intercostobrachial nerve damage with superimposed "central sensitization" was made.

2. Regarding *nociceptive pain* (i.e., pain arising from activation of nociceptors in muscles, bones, ligaments, etc.), its rather poor response to IV SA as opposed to the response of neuropathic pain described above has been demonstrated in several studies from our group.[53–56]

3. The first systematic study on the effect of placebo-controlled SA infusions in *unexplainable non-dermatomal somatosensory deficits* (NDSDs) was reported in 1997 by Mailis and Nicholson.[57] The authors described 10 chronic pain patients with pronounced hypoalgesia (as opposed to the hyperesthesia described above in certain neuropathic pain patients) to pinprick (as well as touch and cold) covering large body areas outside the distribution of peripheral nerves or nerve roots. Either radiological and other investigations had failed to disclose peripheral pathology, or the physical abnormalities were insufficient to explain the patient's pain and sensory abnormalities. One patient experienced resolution of her 1.5-year-old hemisensory deficit and pain after infusion of normal saline, which she perceived as the active drug, despite neutral suggestions as described above. The remarkable effects were persisting at 2 years follow-up while the patient had returned to full time work. In 6 other patients, both pain and NDSD responded dramatically to SA (but not normal

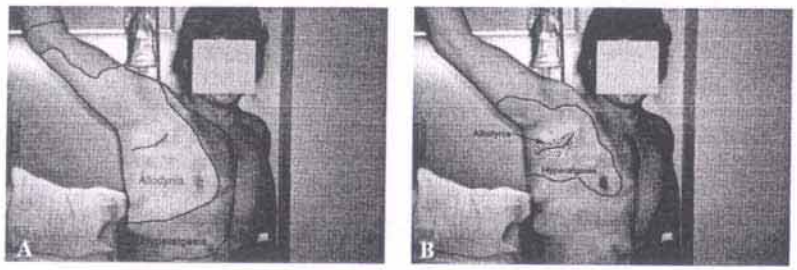


*FIGURE 1.* *A*, Case report. Patient prior to SA infusion presents with a large area of allodynia surrounded by a larger area of pinprick hyperalgesia after a transthoracic sympathectomy. *B*, After 250 mg of IV SA (in the absence of sedation), allodynia has been replaced by a small area of hypoesthesia surrounding the sympathectomy scar, while pinprick hyperalgesia has "shrunk" to the territory of the intercostobrachial nerve.



saline), and in 3 patients pain improved but sensory deficits persisted. In 8 of 10 of these patients, personality profiles indicated that somatoform processes or psychological factors may be involved in their presentation. In contrast, the 11th patient, in whom SA infusion had failed to alter both pain and sensory deficit, was found on quantitative sensory testing (QST) to have suffered spinothalamic tract injury after spinal cord tumor surgery. It is worth mentioning that the effect of SA on sensory deficits of this sort has been reported before[32] in patients with conversion reaction (unexplainable sensorimotor abnormalities in the absence of underlying physical disorder), most of whom had significant psychiatric disorders identified or confirmed during SA infusion. The majority of our patients reported above would indeed fit the concept of "conversion" reaction or "hysteria." However, we believed that dissociating "the mind from the body" was rather inappropriate and we proposed at that time that a psychobiological substrate at cortical and/or subcortical levels was responsible for NDSDs, possibly associated with certain personality organization.

The dynamic nature of NDSDs under SA administration was demonstrated not only in pain associated with minimal or no peripheral pathology as described above, but also in painful disorders associated with major structural damage.[16] Dramatic but short-lived reversal or elimination of dense and diffuse sensory deficits was shown during SA (but not saline) infusion in two patients with extensive cavitation of the spinal cord (syringomyelia). The authors concluded that the deficits that are temporarily modified by SA are "dynamically" maintained as a result of central nervous system plasticity, *superimposed* on phenomena arising from structural neural tissue damage. On their own, "structural" sensory deficits like those observed after neurectomy, at the level of spinal cord lesion, after brachial plexus avulsion, etc., have well-defined sensory borders that are not altered by SA infusions.[56]

Most recently, we reported on the prevalence and speculated on the nature of NDSDs[59,60] in chronic pain patients. These widespread non-dermatomal hypoesthetic or anesthetic abnormalities were seen in 25% of 194 consecutive patients with *little or no detectable physical pathology* in the context of litigation/compensation. Hypoalgesia to pinprick ranged from slight alteration of the prick (in whole limb, quadrotomal, or hemisensory distribution) as compared with the control site, to complete anesthesia. Most of these patients reported widespread pains, were often diagnosed as suffering from fibromyalgia or diffuse myofascial pain syndromes, and displayed intense pain behavior and disability. Those born in countries other than Canada or injured at work were more likely to present with NDSDs. NDSD subjects were more likely to have pain lateralized or worse in one side of the body (at the site of NDSD) and to complain of more pain and increased number of pain sites as compared with the original complaints at onset of pain. They were also more likely to present with abnormalities of deep pain perception in the form of either excessive sensitivity or, to the contrary, insensitivity within the NDSD area as demonstrated by algometry or digital pressure. They were virtually all unemployed, versus 31% of non NDSD subjects who were still employed. They demonstrated unusual gestures or posturing, pain behaviors, and discrepancies in straight leg raising between distraction and confrontation. Most of these patients again presented with symptoms and signs easily classifiable as non organic or "hysterical."

Preliminary functional MRI studies on our pain population with NDSDs have so far supported our concept of functional brain alterations in individuals with non-anatomical deficits and intense/bizarre pain behavior. These data demonstrate striking abnormalities with lack of activation (or actual deactivation) of somatosensory

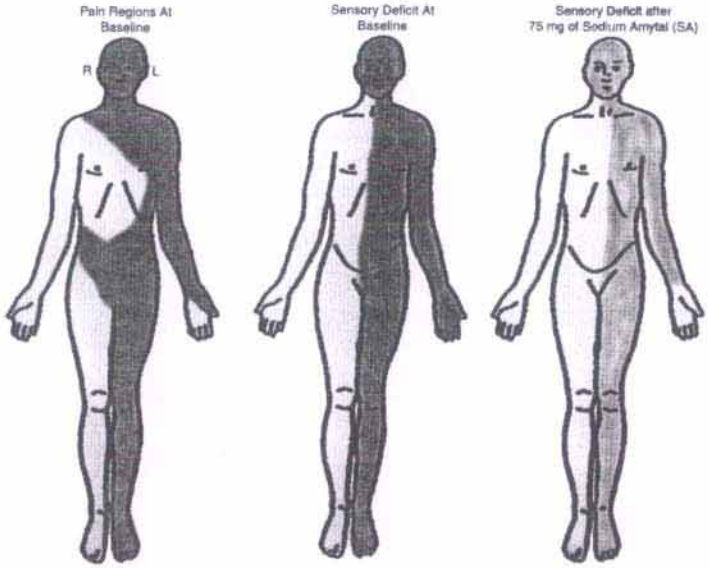


*FIGURE 2.* Patient with axial and left-sided pain (*left*) and profound hemisensory anesthesia (*middle*) to pinprick in the absence of detectable pathology. After 75 mg of SA infusion, pain is dramatically reduced while sensory deficit normalizes almost completely (*right*).

cortex and several subcortical structures after both noxious and non-noxious stimulation of the symptomatic limb.[58] Based on our studies, we speculated that, given the dynamic nature of NDSDs as shown by their dramatic but temporary response to SA infusions, these phenomena seem to constitute an "unsuccessful attempt" of the central nervous system to "shut down all peripheral inputs in an effort to control pain."[60] In this context, NDSDs seemed to be examples of "functional deafferentation" (as opposed to structural deafferentation exemplified by brachial plexus avulsions) resulting from "*maladaptive neuroplasticity*."[60] Given the clinical picture and exuberant behavior of patients with NDSD, we also stressed that further studies should explore the role of personality organization and psychosocial factors in the generation of NDSDs and pain.

An illustrative example of a patient with profound hemianesthesia of the NDSD type as described above is shown in Figure 2. This female patient (who had been reported elsewhere) presented with intractable pain, excessive pain behavior, and dense hemianesthesia 2.5 years after a motor vehicle accident where only soft tissue injuries were sustained.[60] At original consultation, 2nd degree burns were noted in the forearm sustained 2 weeks prior to consultation, which the patient stated were painless. SA infusion at low doses (75 mg), which failed to generate at this level any significant sedation, produced temporarily near-normalization of the dense anesthesia, significant reduction of spontaneous pain, and improvement of the excessive pain behavior. A deep "conversion V" profile was obtained on the MMPI-II.

In summary, our group has demonstrated multiple and separate effects of SA infusion on different types of pain and associated somatosensory abnormalities.



## MECHANISMS OF EFFECT

In terms of behavior alterations, Thorner[93] early on considered that effects associated with administration of SA were largely attributed to "psychic release" and suggested that the mechanism of effect involves *disinhibition* or, more specifically, an *anti-inhibitory action*. He noted that catatonic and negativistic states are sometimes regarded as representing a state of widespread inhibition. Kubie[38] thought that SA produces a loosening of the "tight grip" with which the patient clings to reality. The subject's psychological state was thought to be associated with and/or influence the sensorimotor organization of the nervous system. Lipton[48] suggested that SA acts as a "psychic analgesic." Ludwig[51] later proposed a neuropsychobiological theory of conversion symptoms involving increased inhibition of sensory and motor function from corticofugal tracts. The role of barbiturates or other sedative drugs in relieving symptoms of conversion disorder was stressed, suggesting that these serve to produce disinhibition with subsequent return of normal attentional function. In our opinion, a consistent theme seems to appear through much of the literature, in the sense that SA may exert an effect on inhibition of processes associated in some way with "repression or dissociation of an intolerable experience." We believe that the "intolerable" nature of such experiences may be determined more so by the *premorbid personality organization* than by any objective indicator of the current stimulus/trauma. Perry and Jacobs[77] suggested that SA procedures were useful specifically in disorders that involve repression and dissociation, and cautioned that they should not be used in "fishing expeditions."

McKim,[66] reviewing behavioral-pharmacological or other literature, noted that at lighter levels barbiturates cause a speeding up of brain waves. This begins in the frontal cortex and spreads over the entire cortex. With high doses, the EEG slows and larger amplitude waves characteristic of sleep occur. McKim noted that behavioral studies indicate that barbiturates may act either as a stimulant or a depressant depending upon environmental contingencies or experiential history of the subject. It was suggested that barbiturates have a spectacular effect on behavior suppressed by punishment, i.e., they cause a dramatic increase in punished behavior at doses that have little effect on positively motivated behaviour. For example, animals will continue to make responses that are punished by electric shock at normal, unpunished rates. This occurs despite the fact that the animals feel the shock as they jump and flinch when it happens.[25] McKim also noted that humans, rats, and monkeys will all readily work to give themselves infusions of all types of barbiturates. The pattern of self-administration in non-humans appears more similar to the pattern for opiates than for alcohol.

Rall[80] reviewed the effects of barbiturates in detail. In summary, barbiturates produce reversible depression of CNS (from mild sedation and sleep to general anesthesia). Some possess anticonvulsant properties and may exert euphoric effects comparable with those of morphine. In smaller doses, they may lower the pain threshold (algesic effect). Chronic use can produce both pharmacodynamic and pharmacokinetic tolerance, and cross-tolerance to all general CNS suppressants including ethanol. Non-anesthetic doses suppress polysynaptic responses and depress facilitation while they enhance inhibition, the latter primarily at GABA sites. Inhibition is both presynaptic (spinal cord) and postsynaptic (cortical and cerebellar pyramidal cells, cuneate nucleus, substantia nigra, thalamic relay nuclei, etc). In the peripheral nervous system, barbiturates selectively depress transmission in autonomic ganglia and reduce nicotinic excitation by choline esters resulting in fall in blood pressure. Some also produce facial flushing. Apart from GABAmimetic effects, barbiturates

possess ionotropic AMPA, kainate, and NMDA receptor non-competitive antagonistic effects. In neuropathic pain patients, it is unclear which one of these (or other) effects are responsible for the selective modulation of allodynia. However, NMDA receptors have been implicated in centrally mediated allodynia in multiple reports. For review of the above, see Rall[51] and Mailis et al.[55,56]

In essence, however, it remains essentially unknown what are the mechanisms of effects of SA on mood, behavior, pain, and sensory abnormalities.

## CONCLUSION

Numerous reports over the past 70 years indicate that SA procedures may have a beneficial effect, at least temporarily resolving the symptoms of many different disorders as outlined above. With the exception of its use in chronic pain, most of what is known today about SA in the assessment and treatment of functional or other disorders has been known already for 50 years or more. The lack of more recent research on barbiturates is likely due to the fact that less attention was paid to the barbiturates since the introduction of neuroleptics, anti-depressants, benzodiazepines, and other pharmacological agents that were thought to target more specific problems. Furthermore, with the exception of few studies on pain patients, the existing literature is not controlled, as most of the studies reviewed here are case series (some involving very large numbers) or single case studies. Several researchers have commented upon the need for further, more rigorously controlled studies.[20,35] While we believe that there is considerable evidence that SA procedures may be useful for a variety of purposes, many questions need to be resolved.

Based on our own research and experience, SA seems to exert the following effects in patients with different types of pain:

1. Behaviors and signs (tenderness, range of movement, gait, etc.) arising from pure *nociceptive pain* (i.e., spontaneous pain arising from fractures, inflammation, muscle tears, etc.) do not seem to be affected or are minimally affected by SA infusions. In such cases, SA may modulate the "affective" component of pain, or other central effects associated with nociception, thus reducing slightly spontaneous pain. Of course, in mixed pictures where there is a minor nociceptive component with a superimposed significant "functional" overlay, SA administration or different other interventions (including administration of inert normal saline as we reported above by Mailis and Nicholson[56]) may produce dramatic reduction in spontaneous pain.
2. In *neuropathic pain patients with hyperesthesia*, SA dramatically reduces touch-evoked pain (allodynia secondary to central sensitization) and much less pinprick hyperalgesia, while it does not alter deep pain as evidenced by failure to reduce deep seated tenderness. Spontaneous pain may be partially reduced.
3. In *neuropathic pain patients with hypoesthesia* due to damaged neural tissue, SA may partially reduce spontaneous pain but will not alter the borders of these anatomical deficits. Again, one should be aware of the potential co-existence of both structural and dynamic deficits in patients with somatic or neural tissue damage as reported above.
4. When structural damage is minimal or absent, *unexplainable non-anatomical hypoesthesias*, most often attributable to "non-organic pain" or "conversion/hysterical" phenomena, may respond dramatically to SA administration (preliminary data from our unit indicate a 70–80% response). Patients with such abnormalities experience significant or total reduction of sensory deficits to all sensory testing modalities, i.e., touch, pinprick, cold, vibration sense, and deep tenderness, as well as pain. Again, this particular subgroup of patients would easily fit the description of



"hysteria" or "conversion" reaction, as it does present quite often with intense and bizarre pain behavior; certain personality and psychosocial factors may be prominent while conventional investigations show minimal or non-detectable peripheral or central physical pathology.

In our experience (over 700 SA infusions over the past 7 years alone), it seems that the following observations hold true in patients with chronic pain: (1) Even in patients with unequivocal physical disorders and insignificant psychological factors, one should expect a slight amelioration of spontaneous pain (maybe 10–20%) as a result of the relaxing and euphoric or other effects of SA that may influence the affective component of pain. (2) Tolerance to SA effects may be seen physiologically in some (but not all) patients on high-dose opioids or with history and/or current ethanol abuse. Similarly, young children and adolescents may need doses up to 15 mg/kg of body weight to produce sedation (probably owing to the pharmacokinetic and pharmacodynamic effects of the drug in the young). (3) Symptom exacerbation (with the exception of occasional algesic effects of SA in 50–100 mg dose) may be consistent with "disease simulation" or "malingering," but one should be very cautious in labeling the patient as a "malingerer" based exclusively on the SA interview findings. (4) In the absence of cross-tolerance to other CNS suppressants or adolescence, complete failure to experience any pain modulation (often associated with denial of obvious sedation) may be indicative of unconscious resistance or need to protect fragile defenses, i.e., to maintain a sense of control. (5) It seems that true analgesic effect in neuropathic pain patients with concommitant reduction of allodynia occurs in low doses of SA (see case report in Figure 1), often well before sedation and other CNS effects appear. Remarkably, in our experience, oral administration of SA in serum concentrations equivalent to those obtained via SA infusions almost always fail to produce the analgesic effect seen during infusion of the drug. We and others speculate that this may be due to the difference in the speed with which the drug crosses the blood-brain barrier.

Future studies might address virtually any of the issues that have been raised in the past, plus various other questions. A partial list of such questions might include: (1) For what disorders is administration of SA actually helpful in comparison with placebo or other pharmacological agents? (2) Who is a responder and who is not, and what premorbid experiences, personality characteristics, or situational factors, including quality of the doctor-patient relationship, have an effect on response? (3) Is there any interaction or relationship with placebo effects? (4) What exactly does lack of response indicate, e.g., psychoemotional factors, drug tolerance, etc? (5) What, if any, role do SA procedures have in forensic issues such as the determination of truthfulness or malingering? (6) Are long-term beneficial effects likely to be seen with any particular disorders or persons? (7) Why are alcohol, benzodiazepines, or other agents not as useful as SA? (8) What is the biological mechanism of effect? (9) Could related pharmacological agents, including new or to be developed agents, delivery systems, etc., be more helpful, possibly allowing for routine administration? (10) Is the mechanism of effect primarily or only biomedical, or do psychosocial issues need to be addressed? (11) Are SA procedures useful as an adjunct to the psychotherapeutic process or in specific psychotherapeutic contexts or with certain disorders such as post-traumatic stress disorder? (12) What is the relative effectiveness of repeated procedures, with or without psychotherapy? (13) What is the relationship between the mechanism of effect in perceived pain versus phenomena seen in conversion or other disorders?

SA procedures in our opinion have utility in the differential diagnosis and possible treatment of many disorders that are often considered "functional." The term

"functional" has been associated with a host of concepts, e.g., somatoform, hysterical, psychogenic, abnormal illness behavior, malingering, etc.[79,87] However, we believe that the terms "functional" and "organic" can be conceptualized across a continuum. At one extreme, the symptoms/signs are quite reproducible, are based on distinct peripheral or central pathological processes, psychological factors are not contributory, and symptoms fail to respond appreciably to SA administration. At the other end of the extreme, one might consider symptom complexes that are generated in the absence of detectable physical pathology by conventional means, psychosocial factors are pivotal in the presentation, and SA administration produces marked resolution of all symptoms. In between these extremes, there may be a wide variety of possible presentations that might best be characterized as involving a *psychophysiological interface* with diverse mechanisms of effect, both psychosocial and biomedical. We believe that it remains largely unknown whether *all* disorders described above as "functional" involve a psychosocial component, whether there may be some "maladaptive neuroplasticity" or other pathological process independent from psychosocial factors, or whether psychosocial factors are involved in the generation or maintenance of "maladaptive neuroplasticity" and observed symptoms/signs. A remarkable example relates to unexplainable widespread somatosensory deficits (NDSDs) as described above, classically attributed to "conversion" or "hysteria," seen in pain patients in the absence of pathology and associated with high disability and significant pain behaviors.[60] It is unclear if the functional brain alterations seen in these individuals[58] are the cause or effect of perceived pain, whether they contribute to the maintenance of pain, and how or if psychosocial factors may be associated with onset or maintenance of the problems. Currently, several functional neuroimaging studies have begun to explicate what might be the underlying neurobiology of some functional disorders.[58,64,89,96] Imaging techniques in the future may attempt to discriminate between "conversion" disorders with anesthesia or paralysis and *outright malingering* with fabrication of symptoms solely for the purposes of external incentives/monetary gain in which symptoms come and go primarily as a matter of whether the person is being examined.

We caution that one should not fall into the many possible pitfalls and problems associated with *mind-body dualism*. Many functional disorders respond well to purely behavioral or psychological interventions[65,98] without need for biomedical interventions, but in other cases pharmacological or other medical treatments may be necessary. In several cases, conjoint psychosocial and biomedical treatments are indeed more efficacious (e.g., depression, chronic pain). It may be that SA administration is most useful in disorders involving *repression/dissociation or inhibition associated with intolerable experiences*, be it *physical* or *emotional* or both.

In conclusion, SA has shown and continues to show considerable promise in the assessment and treatment of a wide variety of functional or other disorders. However, there is clearly a need for further research to clarify a host of outstanding issues and unanswered questions.

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# USEFUL PSYCHOLOGICAL INSTRUMENTS FOR ASSESSING PERSONS WITH FUNCTIONAL MEDICAL PROBLEMS

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No text on the subject of functional medical disorders, or physical disorders and presentations in which psychological disturbances constitute primary influence over symptomatology, would be complete without a chapter that discusses psychological assessment. In this chapter, a brief biopsychosocial model is presented that recognizes the importance of evaluating the collective influence and interaction of psychological, social, and cultural factors with biological factors, in explaining disease and its variable expression in terms of health outcomes.

A list of many of the most relevant instruments for identifying important vulnerability and stress and coping factors is presented. Specific emphasis is given to evaluating psychological variables that impact on and mediate the variability in behavioral expression of health and disability. This is done with the intention of increasing understanding about the influence of these variables, their measurement, and their relevance to enhancing clinical rehabilitation interventions.

## A BIOPSYCHOSOCIAL CONCEPTUALIZATION OF FUNCTIONAL DISORDERS

Medical presentations in which psychological disturbances constitute primary influence over symptomatology are a common and costly health care problem (e.g., Chaudhuri, this volume).