



Short communication

## Therapeutic sedation for functional (psychogenic) neurological symptoms

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## ABSTRACT

**Background:** Patients with severe functional (psychogenic) neurological symptoms such as paralysis and fixed dystonia present a therapeutic challenge, particularly if no movement is possible during physiotherapy. Sedation has been discussed as a treatment for functional neurological symptoms for 100 years but technique, use of video and outcome has not been systematically described.

**Methods:** Therapeutic sedation of patients with severe functional neurological symptoms with propofol and follow up at a neuroscience centre.

**Results:** Of eleven patients (median duration 14 months), five were cured or had major improvement with sedation. At follow up (median 30 months) four were asymptomatic, two were significantly improved and one had minor improvements. We describe a standardized anesthetic and physician technique, refined over consecutive treatments.

**Conclusion:** In carefully chosen patients, therapeutic sedation with propofol can be a useful adjunctive treatment for patients with severe functional neurological symptoms. The treatment deserves randomized evaluation.

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## Introduction

Sedation with anesthetic agents has been used since at least the First World War for investigation and treatment of functional (psychogenic) neurological symptoms [1], particularly in patients who were unresponsive to other treatments such as hypnosis. The technique of narcoanalysis or abreaction was a method of facilitating psychotherapy by gaining information from the patient that they were unwilling or unable to talk about in normal consciousness.

By contrast, the idea of using suggestion under anesthesia, not to talk to the patient, but to demonstrate to the patient the possibility of recovery of a paralyzed, weak or dystonic limb and exclude contractures [12] has received scant attention. Older methods of sedation made this difficult however and only 15 cases of rehabilitation during anesthesia (moving the affected limb) were described in a systematic review of abreaction for conversion disorder since 1920 [3]. Sodium amytal, thiopentone or intravenous benzodiazepines were most commonly used in these older studies [3]. We chose propofol for its pleasant anxiolytic effects, and the rapidity with which any oversedation can be corrected.

We describe our experience of therapeutic sedation as an adjunctive treatment for eleven patients with severe and persistent functional neurological symptoms. In particular we describe a focus on movement and rehabilitation during the procedure, the use of propofol as a short acting anesthetic agent, and of video recording in aiding the patient's recovery [2,4]. We suggest a standardized method for the procedure that could be formally investigated as a treatment for functional (psychogenic) neurological symptoms.

## Methods

We performed a retrospective analysis of all patients receiving therapeutic sedation for functional neurological symptoms in our unit over the period 2002–2012. We recorded initial and long term outcomes using the Modified Rankin Scale. All patients were diagnosed by a consultant neurologist (JS). None had additional neurological diagnoses. Cases were chosen whose symptoms firstly could not be temporarily reversed during examination. For example most patients with unilateral functional leg weakness will have a positive Hoover's sign, that is weakness of hip extension which returns to normal with contralateral hip flexion against resistance. In these patients sedation is not required to demonstrate reversibility to the patient [5]. Secondly, we resorted to sedation when there was a lack of improvement despite treatment with physiotherapy, psychological therapies and/or hypnosis. All patients received a 'functional' explanation of their symptoms

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emphasizing the genuine nature of their symptoms (i.e. that there was a problem with the brain's control of the body) [5,6]. They were given written material from [www.neurosymptoms.org](http://www.neurosymptoms.org).

Table 1 describes the nature of the procedure in detail. We focused the treatment on patients who appeared to understand and believe the diagnosis, had a good relationship with the clinician (JS/AC) and were motivated. The purpose of sedation was explained as an attempt to 'kick start' movement or normalization of position in the affected limb, not to make the diagnosis or discuss new information. We endeavoured at all times to maintain transparency regarding what we were doing and why, including a discussion that the procedure may be working by a powerful form of suggestion. Written consent was obtained for the procedure from the patient or in the case of a patient with functional coma, assent from their next of kin. Consent was also obtained for video recording (so that the patient could view the procedure themselves afterwards), nail bed and sternal pressure during the procedure as well as brief anesthesia if no improvement occurred (Table 1).

Sedation was provided using a propofol infusion administered by a Consultant Anesthetist with experienced assistance and Bispectral Index (BIS) monitoring to provide an indication of anesthetic effect (target >80). The propofol infusion was increased slowly until an appropriate sedation level was attained while maintaining verbal contact and the ability to follow commands. Passive movements and active encouragement of movement through conversation and/or painful stimulation were carried out throughout the procedure which lasted 30–60 min. If function was regained the patient was encouraged to continue with the action as the sedation was lightened. Following treatment further physical and/or psychological therapy was provided as required.

## Results

Eleven patients were sedated, eight female and three male (Table 2, Supplementary Video and Supplementary Case Vignettes). Eight patients had functional limb paralysis (two with associated functional dystonia), one functional mutism, one functional dystonia alone and one functional coma (i.e. prolonged motionless unresponsiveness lasting 5 days but not truly comatose) [7]. Six patients were seen as day cases, five as inpatients. One patient was fully anesthetized to assess contractures. Three additional patients declined the offer of sedation citing concerns about being 'out of control' during sedation. All patients with limb paralysis required at least a walking aid prior to therapy and the patient with mutism communicated with an electronic communication device (Litewriter™). No patients were working prior to treatment. Mean age at time of sedation was 34 yrs (range 19–53 yrs) and median length of symptoms 14 months (range 7 days to 9 yrs.).

Three patients became asymptomatic, three had major improvement and two had minor improvement within 24 h of sedation. A further two cases improved only during the sedation itself but relapsed once it had worn off. In one patient who was bedbound with contractures there was no change. There were no adverse events associated with the procedure. Oversedation was not an issue with propofol because it is such a short acting anesthetic. Therefore if the patient did become too sedated to co-operate then within a few minutes of adjusting the propofol infusion they were able to continue with the procedure. Video recordings of the procedure were shared with the patient but we found that patients who had improved could generally remember what happened and did not need to see the video. In those patients who only improved temporarily, viewing the video didn't help. We do strongly advise video however, as a record of the procedure and at the very least to avoid dispute about what happened.

At median follow up of 30 months (range 1–120 months) all improvements were maintained. Two patients who were "cured" and two patients with major improvement during sedation sustained this improvement over lengthy follow up (removal of knee dystonia (duration 35 months) and complete cure of right arm monoplegia (41 months)). Of the two patients with minimal initial improvement, one subsequently went on to have a full recovery attributing some of this to small improvements he had made during sedation. The other had deteriorated back to her baseline (Table 2).

We did not collect detailed data to assess prognostic factors and the numbers were too small and not normally distributed to allow meaningful statistical analysis. Our impression however was that the procedure worked best in patients where there was a good understanding of their disorder, an understanding of the rationale for treatment and unambiguous evidence of motivation to improve. In those patients where it didn't help, the presence of an unresolved work retirement issue, persistent doubts from the patient about the diagnosis, and a lack of active engagement in attempts at physiotherapy were identified as potentially most relevant. Although shorter duration appeared to be a positive prognostic factor in some cases (Case 1 and 3), there were cases who benefited after having had symptoms for several years (Cases 4 and 5) so we do not think duration should prevent clinicians considering this treatment.

**Table 1**

Practical guidance for therapeutic sedation for functional movement disorder

Suitable patients
<ul style="list-style-type: none"> <li>• Functional (psychogenic) movement disorder, e.g. paralysis, dystonia, mutism</li> <li>• Severe deficit in which reversibility cannot be demonstrated to the patient using the examination – e.g. fixed dystonia, paraplegia</li> <li>• Good doctor patient relationship.</li> <li>• Good patient confidence in the diagnosis of functional movement disorder</li> <li>• Good patient motivation. The clinician judges that the patient is genuinely 'stuck' and desperate to have a 'kick start' in treatment. This is in distinction to the patient who is fed up and miserable at situation but has ambiguous motivation for change of symptoms and pattern of disability/family support.</li> </ul>
Pre-procedure explanation to patient – recommended ingredients
<ul style="list-style-type: none"> <li>• The procedure is for treatment not diagnosis – sedation allows 'automatic movements to re-emerge'</li> <li>• Recovery may or may not occur</li> <li>• Video being used to record any improvement and can be viewed afterwards</li> <li>• May need to use painful stimuli to induce movement</li> <li>• Movement may occur more after procedure as much as during it</li> <li>• The procedure is not about finding out verbal information. If the patient speaks whilst heavily sedated then attention will be gently directed back to limb.</li> <li>• An understanding that if nothing happens during procedure it will probably be unhelpful to have repeated sedation procedures, or if they only provide temporary benefit.</li> <li>• Discussion of how the patient would handle sudden recovery with family and friends (i.e. would they worry that others thought they had been feigning if they suddenly improved? If so anticipate strategies to compensate. e.g. "family and doctors know you are not feigning", "if others happen to think this then that is a small price to pay for regained function")</li> </ul>
Procedure
<p><i>Anesthetic guidance</i></p> <ul style="list-style-type: none"> <li>• Senior Anesthetist with appropriate assistance (ODP or anesthetic nurse)</li> <li>• Anesthetic room and equipment – suitable for managing a general anesthetic with machine and monitoring according to AAGBI guidelines (<a href="http://www.aagbi.org">www.aagbi.org</a>)</li> <li>• Target controlled infusion of propofol and suitable infusion pump.) A target controlled infusion (TCI), Marsh model for propofol was commenced with a starting level of 0.2 to 0.5 µg/ml. This was increased slowly until an appropriate sedation level was attained while maintaining verbal contact and the ability to follow commands. Typically propofol TCI levels under 2.5 to 3 µg/ml.</li> <li>• Processed EEG signal analysis (optional) e.g. bispectral index monitoring system (Covidien), as an indication of sedation level with a lower limit of 80. Continual verbal contact with the patient should be maintained.</li> </ul> <p><i>Physician guidance</i></p> <ul style="list-style-type: none"> <li>• Plenty of encouragement throughout. Use of humour, personal information from patient as appropriate</li> <li>• Expect improvements most commonly in deeper states of sedation or during emergence from them <ul style="list-style-type: none"> <li>• Example movements include <ul style="list-style-type: none"> <li>○ Elbow flexion against resistance</li> <li>○ Knee flexion against resistance</li> <li>○ Ankle plantar flexion against resistance</li> </ul> </li> </ul> </li> <li>• Examples of sensory stimuli (which all should be consented prior to the procedure) include <ul style="list-style-type: none"> <li>○ Nail bed or sternal pressure</li> <li>○ Induction of plantar or deep tendon reflexes</li> </ul> </li> <li>• Examples of comments to make during procedure <ul style="list-style-type: none"> <li>○ "Your brain is in an altered state. Let's see if we can get those automatic normal movement going"</li> <li>○ "That was a good movement let's do that again"</li> <li>○ "Don't fight the strange feeling of your arm/leg moving again"</li> </ul> </li> <li>• Continue to reinforce movements after returning to ward, 0–2 h after. This period is also a good time to attempt more bold movements, preferably with the aid of physiotherapy – e.g. attempting walking in someone previously in a wheelchair.</li> </ul>

The patients' experience of the procedure was positive, especially when there was a rapid improvement in a longstanding symptom. Case 1 in the table went on to full time work and was pleased for her case to be shown with this report to give hope to other patients in the same situation. Case 2 went in to and remained in full time employment.

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