Postoperative Sleep Disturbance: Influences of Opioids and Pain in Humans

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Study Objectives: To test the hypothesis that opioids and pain contribute independently to postoperative sleep disturbance, 10 women undergoing surgery requiring a low abdominal incision for treatment of benign gynecologic conditions were randomized to receive either epidural opioid (fentanyl) (n=6) or epidural local anesthetic (bupivacaine) (n=4) for intraoperative and postoperative analgesia.
Design: N/A
Setting: N/A
Patients or Participants: N/A
Interventions: N/A
Measurements: Polysomnography was performed in a standard patient room on the preoperative and first three postoperative nights. Pain at rest and with coughing was evaluated using a visual-analogue pain scale each evening and morning.
Results: On the first postoperative night, rapid eye movement (REM) sleep was abolished in all patients. On the third postoperative night, the mean±SE REM sleep time increased significantly (p=.003) to 9.8%±3.1% in the fentanyl group, and 12.9%±3.8% in the bupivacaine group. Conversely, light non-REM (NREM) sleep (%stage 1 + %stage 2) was higher on the first postoperative night and significantly lower on the third postoperative night (p=0.011). Between group comparison revealed only that the mean % slow-wave sleep (SWS) in the fentanyl group (6.0%, 2.0%, and 14.7%) was different from the bupivacaine group (7.8%, 9.1%, and 10.6%) in the postoperative period after adjusting for the preoperative night % SWS (p=0.021). Pain was well controlled in all patients, but was slightly better controlled in the fentanyl group than in the bupivacaine group on postoperative night 2
(p=0.024). There was no statistically significant association between pain score and any polysomnographically defined stage.
Conclusion: Postoperative patients suffer a profound sleep disturbance even when opioids are avoided and pain is well controlled.
Key words: Postoperative sleep disturbance; circadian rhythm; sleep; opioid; pain

INTRODUCTION

SURGICAL PATIENTS SUFFER A PROFOUND SLEEP DISTURBANCE EARLY IN THE POSTOPERATIVE PERIOD.[1] For example, following open cholecystectomy, rapid eye movement (REM) sleep falls from 18% of the nighttime sleep period to 0% on the night of surgery, and slow-wave sleep (SWS) falls from 23% to 3%.[2] Although postoperative sleep disturbance has been repeatedly documented, its cause remains unclear.[1]

Opioids have been proposed as a cause of postoperative sleep disturbance.[2] Morphine, despite its sedating effect, increases wakefulness and inhibits REM sleep and SWS in a dose-dependent fashion in normal volunteers.[3] In cats, opioid REM sleep inhibition is dose-dependent, naloxone-reversible, and receptor subtype specific.[4,5] In one study of postoperative patients, morphine use was highest and REM sleep time was lowest on the first postoperative night, and as morphine use declined, REM sleep time increased over the subsequent five nights.[2] These lines of evidence suggest that opioids contribute to postoperative sleep disturbance. However, an additional factor, pain, complicates the opioid-sleep relationship in surgical patients.

This opioid-pain linkage confounds the study of postoperative sleep disturbance since pain alone disturbs sleep.[6] A questionnaire study specifically addressed the contribution of pain to sleep disturbance in postoperative patients.[7] In that study, pain was the reason most often provided by patients as the cause for their subjective impression of poor sleep and nighttime awakenings, and the provision of opioid pain medications was reported by patients as the most effective means of enabling them to return to sleep.

With the exception of this questionnaire report and a study which recorded sleep only on the first postoperative night,[8] we are unaware of any prospective study of the cause of postoperative sleep disturbance. Fifty-three patients having noncardiac surgery have been studied using polysomnography for more than one postoperative night.[2,9-12] A unifying observation from these five studies is that the greater the magnitude of the surgical procedure, the greater the magnitude of the sleep disturbance. What constitutes the "magnitude" of a surgical procedure is undeter-

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mined. It is clear, however, that following open cholecystectomy[2] the sleep disturbance is more profound than following laparoscopic cholecystectomy,[1] and the sleep disturbance following inguinal herniorraphy is even less severe.[12] Since "larger" procedures are likely to involve more tissue damage, pain, and higher opioid requirement, these factors need to be dissected to determine their relative contribution to postoperative sleep disturbance.

This study attempts to break the linkage between opioid use and pain in order to test the hypothesis that opioids independently contribute to postoperative sleep disturbance. Patients undergoing a uniform surgical procedure were prospectively randomized to receive either opioid or nonopioid analgesia. Sleep was recorded using polysomnography. Pain was measured to test the secondary hypothesis that pain independently contributes to postoperative sleep disturbance.

METHODS

The Institutional Review Board of the Milton S. Hershey Medical Center approved this study. Written informed consent for participation in this study was obtained from 10 adult patients with benign gynecologic disease requiring surgery through a low abdominal incision. Patients over age 65 were excluded from the study because of potential age-related differences in sleep. Patients reporting frequent nighttime awakenings, daytime somnolence, snoring, or chronic use of benzodiazepines or opioids were excluded because of potential confounding influences of these factors on postoperative sleep, pain, or opioid requirement.

Preoperative preparation: At 20:00 on the preoperative evening, patients were admitted to a standard private hospital room on the gynecologic surgery ward. No caffeine was permitted after 16:00. The lights, television, and radio were turned off and the patients were not disturbed while polysomnography was performed between 22:00 and 06:00.

Intraoperative management: On the following morning, patients had an epidural catheter placed at the T10-11 or T11-T12 spinal level. Location of the catheter in the epidural space was confirmed with a test dose consisting of 3 ml of 1.5% lidocaine with 5 ug/ml epinephrine. After testing of the epidural catheter, patients were randomized to receive either fentanyl (opioid) or bupivacaine (local anesthetic) for intraoperative and postoperative analgesia. The fentanyl group received a 1.5-2.0 ug/kg bolus dose followed by a 1 ug/kg/hr infusion of fentanyl via the epidural catheter. The bupivacaine group received a 10 ml bolus dose followed by a 10 ml/hr infusion of epidurally administered 0.25% bupivacaine. General anesthesia with endotracheal intubation and mechanical ventilation was then induced with 2 mg/kg of propofol and 0.2 mg/kg cis-atracurium intravenously. Anesthesia was maintained for the duration of the surgical procedure with 70% nitrous oxide in 30% oxygen and 2—4% desflurane.

Postoperative management: Following a one-hour stay in the recovery room, patients returned to their room on the surgical ward. For analgesia, the fentanyl group received a 0.75—1.0 ug/kg/hr infusion of fentanyl via the epidural catheter. The bupivacaine group received a 10 ml/hr infusion of 0.125% bupivacaine via the epidural catheter. In addition to the epidural analgesia, patients were free to request 15 mg of intravenous ketorolac every six hours for inadequate analgesia. Patients were blinded to their treatment. Because of obvious clinical differences between fentanyl and bupivacaine treatment, no attempt was made to blind the medical personnel. On the second postoperative morning, the epidural infusion was terminated and patients received oxycodone/acetaminophen (percocet) tablets when requested for pain relief.

Between 22:00 and 06:00 on the postoperative nights, patients were undisturbed except to have their vital signs measured once. Patients had an indwelling urinary catheter throughout the postoperative period to minimize the number of sleep interruptions. Three ml of blood was drawn hourly from an indwelling arterial or venous catheter, with the sampling port separated from the patient by six feet of tubing. No patients received any benzodiazepines postoperatively.

Sleep recording: Using silver/silver chloride adhesive patch electrodes (BioTac, Graphic Controls, Buffalo, NY), polysomnography was performed (Oxford MedilogSAC, Clearwater, FL) comprising two channels of the electroencephalogram (EEG), two channels of the electrooculogram (EOG), and one channel of the submental electromyogram (EMG). Bilaterally symmetric EEG was recorded from electrodes placed on the lateral forehead anterior to the hairline and referenced to the contralateral mastoid. The EOG was recorded from an electrode adjacent to the lateral canthus of each eye and referenced to the contralateral mastoid. The EMG was recorded from two electrodes overlying the submandibular musculature. A 60 Hz filter was used during the recording. The sleep period was 22:00 to 06:00 on all of the study nights. With blinding for subject, treatment, and date, each record was manually scored in 30-second epochs using Rechtschaffen and Kales criteria.[13] Epochs with too much motion artifact to permit reliable scoring were excluded from the analysis. The results were reported as the percentage of the recording time (22:00 to 06:00) spent in wakefulness (% wake), light non-rapid eye movement sleep (% light NREM), slow-wave sleep (% SWS), or REM sleep(% REM).

A subjective impression of sleep quality was obtained each morning by asking the patients if they had slept "poorly, normally, well, or exceptionally well" and if they felt "fatigued, average, or unusually well rested." Before each postoperative recording period, patients were asked to estimate how many hours they had slept that day.

Pain assessment: A visual-analog pain scale, an unmarked 100 mm line with "no pain" at 0 and "worst imaginable pain" at 100, was completed by the subjects on the preoperative night and immediately before and after the sleep recording periods. At each evaluation point, pain at rest and pain with coughing were determined. The average of the night and morning score was used to estimate the overnight pain, both at rest and with coughing.

Statistical analysis: The exact Wilcoxon rank sum test was used to compare the fentanyl and bupivacaine groups with respect to background characteristics such as age, height, weight, duration of surgical procedure, estimated blood loss, and intraoperative fluid replacement. Descriptive statistics were produced for %wake, %light NREM, %SWS, and %REM for each treatment group and study night separately. To evaluate the difference in these sleep measures between the treatment groups over the postoperative period, linear repeated measures analysis of covariance models were fit to each outcome with treatment group and time as the explanatory factors. The primary analysis included the preoperative night measurements as a baseline covariate to

increase the power and precision of the treatment comparison. However, because of the concern that the sleep pattern on the night before surgery would not represent a meaningful baseline condition, supplemental analysis excluding the preoperative night data was performed. For the categorical sleep measures (sleep quality and feeling of well being), logistic regression models within the repeated measures framework were used to detect changes over the postoperative period and differences between treatment groups, again with treatment group and time as the explanatory factors.

Using linear repeated measures analysis of covariance models, differences in pain severity between treatment groups and changes in pain severity over time were described. To evaluate the association between pain and sleep, repeated measures linear regression models were fit to each of the sleep outcomes, with pain at rest and pain with coughing included separately as the explanatory factor. The exact Wilcoxon rank sum test was also used to confirm comparisons between the fentanyl and bupivacaine groups at each postoperative night separately.

Table 1—Mean and range of demographic characteristics of the fentanyl and the bupivacaine treatment groups. There were no statistically significant differences between the groups.

	Fentanyl	Bupivacaine
Age (years)	36 (29-42)	41 (32-49)
Height (cm)	158 (155-166)	154 (137-164)
Weight (kg)	80 (54-141)	86 (61-117)
Surgery Duration (mins)	141 (104-253)	140 (80-182)
Blood Loss (ml)	467 (200-1000)	450 (100-1100)
Intravenous Fluid (ml)	2650 (1400-5500)	3100 (1200-5300)

RESULTS

Ten patients were enrolled, with six randomizing to the fentanyl group and four to the bupivacaine group. One patient in the fentanyl group withdrew from the study after the first postoperative night. There were no significant differences found using the exact Wilcoxon rank sum test between the groups in age, height, weight, duration of surgical procedure, estimated blood loss, or intraoperative fluid replacement (Table 1).

Polysomnography: Technically adequate sleep studies were obtained on 36 of a possible 38 nights. On the preoperative night, the group mean±SE % REM was 7.5%+1.7% and % SWS was 13.9% + 2.6%. All subjects demonstrated the ability to have polygraphically documentable REM sleep and SWS in these conditions. There were differences in the % REM (6.5% vs. 8.9%) and % SWS (10.6% vs. 18.8%) on the preoperative night between the fentanyl and bupivacaine groups respectively (Table 2). Although these preoperative differences were not statistically significant according to the exact Wilcoxon rank sum test, the preoperative night measurements were included in the model as a covariate.

Over the first three postoperative nights, both treatment groups demonstrated significant changes in % REM (time main effect p = 0.003) and % light NREM (time main effect p = 0.011), but not in % wake or % SWS (Table 2). In the fentanyl group, REM sleep was abolished on the night of surgery and returned to 10.9% on the second postoperative night. In the bupivacaine group, REM sleep was also abolished on the first postoperative night and returned to 5.5% and 12.9% on the subsequent two nights. The postoperative changes in % REM were balanced by opposite changes in % light NREM in both treatment groups.

The only difference in postoperative sleep between the fentanyl and bupivacaine groups which approached statistical significance was the % SWS. Over the three-night period, the fentanyl group had 6.0%, 2.0%, and 14.7% SWS, and the bupivacaine group had 7.8%, 9.1%, and 10.6% SWS respectively. The postoperative treatment effect was marginally significant (treatment main effect p=0.112), but became statistically significant (treatment main effect p=0.021) after adjusting for the preoperative night. In each model the treatment by time interaction term was tested; however, the small sample size limited the power of this study to detect significant interactions and provide stable estimates of the sleep measures at each time point.

The average of the subjects' estimate of the number of hours napping on postoperative days one, two, and three was 2.9, 2.7, and 1.3 hours respectively (Table 3). The change over the three-day period was marginally significant (time main effect p=0.055). There was no statistically significant difference in the

Table 2—Mean±SE percentage of the time between 22:00 and 06:00 spent awake (% wake), in stage 1 or 2 NREM sleep (% light NREM), in stage 3 or 4 NREM sleep (% SWS), or in REM sleep (% REM) on the preoperative (Pre) and first 3 postoperative (Post 1, Post 2, and Post 3) nights for the fentanyl and bupivacaine groups. During the postoperative period, there were significant changes over time in % light NREM and % REM for both treatment groups. The only significant difference between the treatment groups was in % SWS.

		% Wake	% Light NREM	% SWS	% REM
Pre	Fentanyl	30.0 ± 6.4	51.6 ± 6.7	10.6 ± 1.0	6.5 ± 2.4
	Bupivacaine	28.7 ± 4.9	43.3 ± 3.0	18.8 ± 6.0	8.9 ± 2.9
Post 1	Fentanyl	23.9 ± 8.8	58.8 ± 6.3	6.0 ± 4.50.0	0.0
	Bupivacaine	36.4 ± 13.1	55.7 ± 11.8	7.8 ± 4.8	0.1 ± 0.1
Post 2	Fentanyl	27.0 ± 8.8	53.6 ± 12.6	2.0 ± 0.7	10.9 ± 5.4
	Bupivacaine	31.9 ± 9.7	53.5 ± 11.3	9.1 ± 3.0	5.5 ± 2.1
Post 3	Fentanyl	23.9 ± 8.7	40.7 ± 6.0	14.7 ± 7.0	9.8 ± 3.1
	Bupivacaine	21.9 ± 4.7	39.8 ± 11.8	10.6 ± 5.9	12.9 ± 3.8

Table 3—Mean and range of the estimated number of hours spent sleeping on the first three postoperative days (Post 1, Post 2, and Post 3) for the fentanyl and bupivacaine treatment groups. There was no significant difference between the treatment groups. Although the trend over time was for less daytime sleep, there was no statistically significant difference over the three-day period.

	Fentanyl	Bupivacaine
Post 1	3.3 (1.5-5.0)	2.3 (0-3.0)
Post 2	2.4 (1.0-4.0)	3.0 (1.0-6.0)
Post 3	1.2 (0.5-2.0)	1.5 (0-3.5)

Table 4—Mean+SE visual-analog pain scale scores at rest and with coughing for the fentanyl and bupivacaine treatment groups on the preoperative (Pre) and first three postoperative nights (Post 1, Post 2, and Post 3). Compared with the fentanyl group, the bupivacaine group reported significantly more pain on Post 2. There was no statistically significant association between pain score and any polysomnographic evaluated in this study, but the fentanyl group was more likely to report feeling unusually well rested.

	Fentanyl		Bupivacaine	
	Rest	Cough	Rest	Cough
Pre	1.5 ± 0.8	1.6 ± 1.3	0.5 ± 0.3	0.5 ± 0.5
Post 1	1.0 ± 0.5	3.9 ± 0.9	3.7 ± 1.4	5.0 ± 1.2
Post 2	0.6 ± 0.4	3.7 ± 1.1	2.7 ± 0.5	4.7 ± 0.4
Post 3	0.6 ± 0.4	3.9 ± 1.3	2.2 ± 0.8	3.7 ± 1.0

estimated number of daytime hours of sleep between the fentanyl and bupivacaine groups.

Pain: Pain scores were highest on the first postoperative night and fell slightly over the next two nights (Table 4). Pain at rest was lower than pain with coughing, but even with coughing, pain was judged to be at least moderately controlled throughout the study period. No significant association was found between the pain score, either at rest or with coughing, and the amount of any of the sleep/wake variables.

A comparison of the pain control in the fentanyl versus the bupivacaine groups was performed because of the concern that a pain difference would confound interpretation of the treatment effect on sleep. Pain at rest was the only outcome where the treatment by time interaction was significant with p=0.026, so the treatment comparisons were performed for each night separately. The bupivacaine group had marginally more pain at rest on the second postoperative night (p=0.033, with p<0.02 required for statistical significance because of multiple comparisons). Using the exact Wilcoxon rank sum test, the statistical significance of this difference was confirmed (p=0.024). On that night, despite the higher pain score, the bupivacaine group actually had more SWS (9.1% vs. 2.0%) but less REM sleep (5.5% vs. 10.9%) than the fentanyl group. Confirming the increased pain in the bupivacaine group, intravenous ketorolac was requested more frequently by them than by the fentanyl group during the first two postoperative nights (exact Wilcoxon rank sum test, p=0.014). After the epidural infusion was discontinued, there was no difference between groups in pain score or oxycodone/acetaminophen (percocet) use on the third postoperative night.

Subjective sleep quality: Subjective sleep quality did not change significantly over time or differ between groups. Feelings of well-being did not change significantly over time, but was significantly better (treatment main effect p=0.034) for the fentanyl group (odds ratio of feeling better in the opioid group was 23.3) (Table 5). No significant association was found between the subjective evaluation of sleep quality or well-being and any polysomnographically defined sleep variable. However, a weak association was found (p=0.068) between lower subjective sleep quality and increased pain with coughing.

DISCUSSION

This study confirms previous observations of a profound postoperative sleep disturbance, and extends our understanding of its causes by demonstrating that it occurs regardless of opioid use or good pain control.

The finding of such a small inhibitory effect of opioids on postoperative SWS was unexpected. Human volunteers receiving 0.43 mg/kg of intramuscular morphine at 22:00 experience a 75% loss of SWS.3 In analgesic potency, this single morphine dose was equivalent to one half of the nighttime fentanyl dose administered in the current study. The results from the current study support the hypothesis that clinically appropriate doses of opioids may be contributing factor, but not the major cause of postoperative SWS inhibition.

Although this study did not detect any opioid effect on the amount of postoperative nocturnal REM sleep, incontrovertible evidence from studies in normal human volunteers and animal models documents the REM sleep inhibiting effect of opioids.[3-5] In normal volunteers, 0.22 mg/kg of intramuscular morphine decreased REM sleep time by 50%, and 0.43 mg/kg of morphine abolished REM sleep.[3] In a cat model where cholinergic mechanisms in the medial pontine reticular formation (mPRF) have been demonstrated to be critical to the brain stem generation of REM sleep, microinjection of morphine into the mPRF inhibited REM sleep.[4,14,15] This inhibition was dose dependent, naloxone reversible, and receptor subtype selective.[5] Intravenous administration of morphine to the cat decreased stimulation-evoked acetylcholine release in the mPRF.[16] The strength of this evidence requires that our findings not be interpreted as contradictory; rather, they should be viewed as evidence for additional unidentified and more powerful REM sleep inhibiting influences

Table 5—Percentage of responses over the three postoperative mornings of having slept "normally, well, or exceptionally well" and feeling "average or unusually well rested" for the fentanyl and bupivacaine treatment groups. Although there were no differences between groups in subjective sleep quality, the fentanyl group had a significantly higher rate of feeling "average or unusually well rested."

	Fentanyl	Bupivacaine
Sleep quality "normal" or better	61.5%	58.3%
Feeling average or unusually well rested	78.6%	25.0%

in postoperative patients.

Since an unidentified influence abolished REM sleep on the first postoperative night in both treatment groups, the potential for detecting the influence of opioids on REM sleep was lost. On the second and third postoperative nights, REM sleep returned, permitting detection of a difference between groups. While no difference was observed, a much larger study would be required to detect the relatively small suppressive effect of opioids on REM sleep in the presence of the overwhelming, but undefined, cause of postoperative REM sleep inhibition. This study allows us to conclude only that profound postoperative REM sleep inhibition occurs even in the absence of opioids.

This study found no correlation between pain scores and any of the polygraphically defined sleep variables. The data supporting pain as a cause of postoperative sleep disturbance that can be improved with opioid analgesics are drawn from a questionnaire study.[7] Those subjective findings are supported by the subjective data in this study which reveal a weak association between increased pain with coughing and decreased subjective evaluation of sleep quality as well as a higher subjective sense of well-being in the opioid group.

An explanation for the lack of an association between pain and polygraphically defined sleep disturbance in this study is that pain was at least moderately controlled in all of our patients. If pain had been poorly controlled, the sleep disturbance could potentially have been more extreme.

We were only partially successful in separating the influences of opioids and pain since the bupivacaine group tended to have slightly more pain than the opioid group. Conceivably, opioids could have caused the sleep disturbance in the fentanyl group, and pain could have caused the sleep disturbance in the bupivacaine group. However, we do not support this interpretation since the difference in pain between the groups was small (and not statistically different on the night of the largest sleep disturbance), and because several patients in the bupivacaine group reported excellent pain control yet still had a profound sleep disturbance.

Randomization was successful in balancing the surgical duration, estimated blood loss, and intraoperative fluid requirements between the treatment groups. Although crude measures of the magnitude of the surgical insult, they suggest that the groups were similar in this respect. Any potential small difference would be unlikely to cause this degree of sleep disturbance in either group.

Rather than eliminating the consideration of pain or opioids as a cause of postoperative sleep disturbance, the results from this study point to the importance of additional unidentified influences profoundly disturbing sleep in postoperative patients. While these sleep disturbing influences have not been identified, several have been proposed.[1] The effect of general anesthesia on nocturnal sleep has been studied in normal volunteers.[17] Nocturnal sleep following three hours of isoflurane anesthesia during the day had a moderate decrease in SWS (16% to 6%), but no change in REM sleep. Following surgery with inhalational (halothane) or intravenous (dehydrobenzperidol) anesthesia, REM sleep and SWS on the first postoperative night were inhibited in both groups equally.[8] The surgical endocrine stress response with elevation of cortisol levels could inhibit REM sleep since administration of cortisol to normal volunteers decreases REM sleep.[18,19] However, the inhibition of REM sleep by cortisol is much smaller in magnitude than the eradication of REM sleep seen in postoperative patients, and the cortisol sleep effect includes an increase in SWS. Finally, the cytokine response to tissue trauma includes elevation of interleukin-6 levels.[20] Administration of interleukin-6 to human volunteers decreased REM sleep and SWS, so it is a possible contributor to postoperative sleep disturbance.[21]

Patients on general medical wards and in intensive care units develop a sleep disturbance. This is associated with decreased strength of the circadian rhythm as revealed by decreased amplitude of the circadian secretion of melatonin.[22] Postoperative patients also suffer derangement of the circadian rhythm as demonstrated by temperature and heart rate cycles[23] as well as the sleep/wake cycle.[9] Although not tested by this study, postoperative sleep disturbance might, in some part, be a manifestation of the underlying circadian rhythm disturbance.

Possibly contributing to the loss of circadian rhythm and poor sleep in the hospital is nighttime noise and nursing interventions. In this study the environmental conditions were controlled to remain relatively stable throughout the four nights. Nursing disturbances to check vital signs were reduced to once during each postoperative night. The hourly blood draws were probably slightly disruptive; however, the same routine was observed on all three postoperative nights and patients rarely awoke during the blood draws. Despite the attempts to create environmental conditions conducive to undisturbed sleep, patients still suffered a loss of SWS and REM sleep in the early postoperative period, and returned toward normal by the third postoperative night.

Limitations and future directions: This is the first prospective study of the etiology of postoperative sleep disturbance using polysomnography for more than one night in surgical patients. The inference available from this study is limited by the small sample size. Larger studies are required in patients undergoing "smaller" surgical procedures with a less extreme sleep disturbance in order to have the power to detect subtle influences of opioids or pain on postoperative sleep. Because the cumbersome polysomnography instrumentation interfered with recovery activities and would potentially be dangerous in a confused patient, monitoring was performed only during the night. Continuous monitoring using alternative techniques in future studies will permit accurate measurement of daytime sleep and evaluation of the degree of circadian rhythm disturbance.

Conclusion

REM sleep and SWS were reduced in the early postoperative period. SWS was slightly more reduced in the opioid group, but REM sleep percentage was not different between the treatment groups. Pain was adequately controlled in all patients and was not found to be a significant influence on objective measurement of postoperative sleep. Despite the absence of opioids or significant pain, postoperative patients experienced a profound sleep disturbance.

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