Postoperative sleep disturbances: mechanisms and clinical implications

S. ROSENBERG-ADAMSEN, H. KEHLET, C. DODDS AND J. ROSENBERG

Major surgery is beset by complications such as pulmonary, cardiac, thromboembolic and cerebral dysfunction, which cannot be attributed solely to inadequate surgical and anaesthetic techniques, but rather to increased organ demands caused by the endocrine metabolic response to surgical trauma [33]. Postoperative cerebral dysfunction comprises delirium, confusion and milder degrees of mental dysfunction [1, 48, 53, 62], and disturbances in the normal sleep pattern [4, 7, 9, 13, 17, 34, 39, 42, 65]. Changes in early postoperative sleep [4, 7, 8, 13, 17, 34, 39, 42, 65] and sleep after non-surgical stress [10, 20, 24, 26, 27, 29, 60] are characterized by a decrease in total sleep time, elimination of rapid eye movement (REM) sleep, a marked reduction in the amount of slow wave sleep (SWS) and increased amounts of non-REM (N-REM) sleep stage 2. Recent data have suggested that postoperative sleep disturbances may be involved in the development of altered mental function [27], postoperative episodic hypoxaemia [38, 65] and haemodynamic instability [40]. The aim of this article, therefore, is to review the incidence and duration of postoperative sleep disturbances, and to evaluate possible mechanisms and potential implications for postoperative outcome.

The normal sleep pattern

Using standardized polysomnographic recordings, which include simultaneous electroencephalographic (EEG), electro-oculographic (EOG) and electromyographic (EMG) recordings, sleep can be divided into different sleep stages (see below). By questionnaire, subjective sleep quality, which has been shown to be related to total sleep duration and number of awakenings [3], may be assessed.

Sleep is not homogeneous, but is divided into two discrete forms, REM and N-REM, by their electrophysiological characteristics [58], and each has been subdivided further. There are striking differences between these two sleep patterns. For example, arousal to effective awareness depends on the magnitude of the stimulating input and the “depth” of the sleep stage. Wakening from REM and stage 4 N-REM requires the greatest input, and this is as true for external stimuli, such as noise and light, as for internal autonomic signals, such as hypoxia or pulmonary stretch receptor reflexes. This clearly has an important influence on the hazards that may accompany deep sleep after surgery (see below).

N-REM sleep is subdivided into four stages of increasing depth or resistance to arousal, with stage 1 N-REM being light sleep and stage 4 N-REM very deep sleep (fig. 1). The physiology of N-REM sleep is based entirely on the autonomic medullary and brain stem control systems. There is a reduction in basal metabolic rate and this is associated with a progressive decrease in oxygen requirement, heart rate, arterial pressure, temperature and ventilation. The decrease in arterial pressure is largely because of peripheral vasodilatation, with cardiac output remaining relatively unchanged. Other physiological variables are generally unchanged during N-REM sleep. The tone of the anti-gravity muscles decreases with increasing depth of N-REM sleep, but the tonic drive to the diaphragm is maintained at all levels and types of sleep. There is a reduction in hypoxic and hypercapnic ventilatory responses and the response to increasing respiratory resistive loads. The maximum decrease occurs in stages 3 and 4 N-REM (also called slow wave sleep (SWS)), where for example the hypoxic drive decreases by 40%.

REM sleep is the phase most closely associated with dreaming. It is more akin to full awareness than sleep, in the variability and rapidity of changes in physiological state. There are marked variations in arterial pressure and heart rate, rate and depth of breathing, and metabolic rate. It appears that we act out our dreams physiologically. Major differences from N-REM sleep are also evident in respiratory control during REM sleep. The α-motor neurones are hyperpolarized during REM, with the exception of the facial muscle supply and that of the diaphragm. The hypoxic ventilatory response is reduced by up to 60% and carbon dioxide responses to between 27%
In REM sleep, atonia of the anti-gravity muscles predisposes to instability of the airway, and most obstructive apnoea episodes occur in this stage of sleep. When the airway has become obstructed, further inspiratory effort is futile because of the lack of muscle tone during deep sleep. Vigorous paradoxical ventilatory movements are easily visible but ineffective. The only route to recovery of the airway is arousal from sleep which is usually very brief (< 5 s) and not associated with recall. The cycle of sleep–obstruction / hypoxia–arousal–sleep–obstruction/hypoxia may occur more than 100 times each hour. The consequences of these episodes of obstruction are two-fold: physiological effects of the progressive hypoxia and hypercapnia, and daytime sleepiness from repeated life-saving arousals. These patients are described as suffering from obstructive sleep apnoea (OSA) syndrome [46].

The consequences of airway obstruction during sleep may be profound and include progressive hypoxia, bradycardia and hypercapnia. Pulmonary artery pressure increases by hypoxic pulmonary vasoconstriction, and there is a generalized increase in systemic peripheral resistance. During REM sleep this causes increased oxygen demands at a time when hypoxia and hypertension are marked and ischaemic injury becomes likely, particularly of the heart. If apnoes occur during N-REM, the association of hypotension and hypoxia may lead to cerebral hypoperfusion and stroke.

Function of sleep

The function of sleep is largely unknown, although several theories have been proposed. The most widely held theory is that sleep serves as a period of restoration, either total body or neurological restoration [66]. Total or selective sleep deprivation affects particularly the brain, with psychological and neurological dysfunction [30], impairment in behavioural and psychological performance [8, 23, 30] and sleepiness [8], and impaired concentration [30] and performance on psychometric tests [8, 23, 30]. Finally, mood is affected, with increased sadness and irritability, and decreased vigour [30]. Selective REM sleep deprivation produces a state of pronounced irritability and lability, and signs of confusion, anxiety and suspicion [2]. Deprivation of REM sleep is followed by a significant increase (rebound) in a recovery night [2]. SWS deprivation produces a feeling of physical discomfort [2], characterized by a depressive and hypochondriacal reaction [2]. Selective deprivation of SWS also produces a rebound in SWS during a recovery night [2, 23].

The postoperative sleep pattern

Only six studies have been performed with EEG recording of sleep in postoperative patients after non-cardiac surgery, including a total of 35 patients after major abdominal surgery [4, 17, 39, 65], 18 patients after herniorrhaphy [17, 34] and 46 patients after minor undefined surgery [42] (table 1).
After abdominal surgery all patients were sleep deprived, as shown by total sleep time, proportion of REM sleep and SWS on the first and second postoperative nights in the ICU [4] and on the ward [17, 34, 38, 39, 65]. Total sleep time was reduced by up to 80% on at least 1 of the first postoperative nights [4, 7, 13, 17, 34], with considerable inter-individual variation. Throughout the operative night and the subsequent 1 or 2 nights, sleep was highly fragmented with numerous movement arousals and spontaneous awakenings with long wake periods [17, 34, 39], preventing the inherent rhythmicity of sleep and the normal distribution of sleep stages [17, 39].

REM sleep is usually absent on the first and sometimes the second and third postoperative nights [4, 17, 34, 38, 39, 42, 65]. During the following 2–4 nights, when other sleep abnormalities recover, REM sleep reappears with increased density and duration (rebound) in most patients [34, 38, 39, 65] (fig. 2). In patients undergoing cholecystectomy and gastroplasty, Knill and colleagues showed that increments in REM sleep during rebound were primarily a result of lengthening of individual REM sleep periods rather than to an increased number of periods [31]. Thus, increased total REM sleep time, combined with increased REM density, results in a substantial rebound in total REM activity [39]. The increase in REM activity is associated with frequent reports of distressing and vivid nightmares [39]. Nightmares have been shown to be frequent (about 20%) during the first week after major non-cardiac surgery [7, 9], with the highest incidence on the fourth postoperative night [9], corresponding to the period of rebound REM sleep.

On postoperative nights 1–4 there was a marked reduction in the amount of SWS [4, 17, 34, 39, 42, 65]. In one study SWS was absent in all 10 patients studied on the first and second nights after major abdominal surgery [65]. An increased duration of SWS (rebound) in the middle of the first postoperative week may occur after initial deprivation of SWS [65].

The duration of the abnormal postoperative sleep pattern has been described only poorly. No studies with EEG monitoring have been performed later than the sixth night after abdominal surgery. Total sleep time is gradually normalized to the pre-operative level within the first postoperative week [34]. REM sleep was still increased in nine of 12

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**Table 1** Summary of clinical studies with EEG measured sleep after non-cardiac surgery.

<table>
<thead>
<tr>
<th>Type of surgery</th>
<th>Postoperative nights</th>
<th>REM sleep</th>
<th>SWS</th>
<th>Stage 2</th>
<th>Arousal</th>
<th>Rhythmicity</th>
<th>Patients</th>
</tr>
</thead>
<tbody>
<tr>
<td>Aurell &amp; Elmqvist [4]</td>
<td>1–4</td>
<td>↓</td>
<td>↓</td>
<td>↓</td>
<td>↑</td>
<td>0</td>
<td>9</td>
</tr>
<tr>
<td>Ellis &amp; Dudley [17]</td>
<td>1, 2</td>
<td>↓</td>
<td>↓</td>
<td>↓</td>
<td>↑</td>
<td>↑</td>
<td>8</td>
</tr>
<tr>
<td>Upper abdominal surgery</td>
<td>1, 2</td>
<td>↓</td>
<td>↓</td>
<td>↓</td>
<td>↑</td>
<td>↑</td>
<td>4</td>
</tr>
<tr>
<td>Cholecystectomy</td>
<td>1, 2</td>
<td>↓</td>
<td>↓</td>
<td>↓</td>
<td>↑</td>
<td>↑</td>
<td>6</td>
</tr>
<tr>
<td>Gastroplasty</td>
<td>1, 2, (3)</td>
<td>↓</td>
<td>↓</td>
<td>↓</td>
<td>↑</td>
<td>↑</td>
<td>3 (6)</td>
</tr>
<tr>
<td>Major abdominal surgery</td>
<td>1, 2</td>
<td>↓</td>
<td>↓</td>
<td>↓</td>
<td>↑</td>
<td>↑</td>
<td>1 (2)</td>
</tr>
<tr>
<td>Herniorrhaphy</td>
<td>1, 2</td>
<td>↓</td>
<td>↓</td>
<td>↓</td>
<td>↑</td>
<td>↑</td>
<td>4 (4)</td>
</tr>
<tr>
<td>Major non-cardiac surgery</td>
<td>1–4</td>
<td>↓</td>
<td>↓</td>
<td>↓</td>
<td>↑</td>
<td>↑</td>
<td>7 (10)</td>
</tr>
<tr>
<td>Herniorrhaphy</td>
<td>1, 2</td>
<td>↓</td>
<td>↓</td>
<td>↓</td>
<td>↑</td>
<td>↑</td>
<td>6 (4)</td>
</tr>
<tr>
<td>Herniorrhaphy</td>
<td>1, 2</td>
<td>↓</td>
<td>↓</td>
<td>↓</td>
<td>↑</td>
<td>↑</td>
<td>6 (4)</td>
</tr>
<tr>
<td>Herniorrhaphy</td>
<td>1, 2</td>
<td>↓</td>
<td>↓</td>
<td>↓</td>
<td>↑</td>
<td>↑</td>
<td>6 (4)</td>
</tr>
<tr>
<td>Herniorrhaphy</td>
<td>1, 2</td>
<td>↓</td>
<td>↓</td>
<td>↓</td>
<td>↑</td>
<td>↑</td>
<td>6 (4)</td>
</tr>
<tr>
<td>Herniorrhaphy</td>
<td>1, 2</td>
<td>↓</td>
<td>↓</td>
<td>↓</td>
<td>↑</td>
<td>↑</td>
<td>6 (4)</td>
</tr>
<tr>
<td>Herniorrhaphy</td>
<td>1, 2</td>
<td>↓</td>
<td>↓</td>
<td>↓</td>
<td>↑</td>
<td>↑</td>
<td>6 (4)</td>
</tr>
<tr>
<td>Herniorrhaphy</td>
<td>1, 2</td>
<td>↓</td>
<td>↓</td>
<td>↓</td>
<td>↑</td>
<td>↑</td>
<td>6 (4)</td>
</tr>
</tbody>
</table>

**Figure 2** Changes in total REM sleep on pre- and postoperative nights (based on [4, 17, 34, 39, 42, 65]).
Postoperative sleep disturbances

Table 2: Summary of studies of postoperative sleep quality (measured by questionnaire).

<table>
<thead>
<tr>
<th>Type of surgery</th>
<th>No. of patients</th>
<th>Postoperative nights</th>
<th>Total sleep time</th>
<th>Arousals/awakenings</th>
<th>Subjective sleep quality</th>
<th>Nightmares</th>
<th>Patients</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ellis &amp; Dudley [17]</td>
<td>8</td>
<td>1, 2</td>
<td>↓ (in all)</td>
<td>↑ (in all)</td>
<td>↓ (in all)</td>
<td>0</td>
<td>8</td>
</tr>
<tr>
<td>Upper abdominal surgery</td>
<td>100</td>
<td>1, 2</td>
<td>↑ (in 25%)</td>
<td>↓ (in 12%)</td>
<td>↓ (in 25%)</td>
<td>0</td>
<td>100</td>
</tr>
<tr>
<td>Abdominal surgery</td>
<td>15</td>
<td>3, 4</td>
<td>↓ (in 5%)</td>
<td>↓ (in 5%)</td>
<td>↓ (in 14%)</td>
<td>0</td>
<td>15</td>
</tr>
<tr>
<td>Elective orthopaedic surgery</td>
<td>57</td>
<td>1–7</td>
<td>↓ (in 30%)</td>
<td>▲ (in 14%)</td>
<td>↓ (in 30%)</td>
<td>0</td>
<td>57</td>
</tr>
<tr>
<td>Day surgery (not specified)</td>
<td>53</td>
<td>1–7</td>
<td>▲ (in 30%)</td>
<td>↓ (in 12%)</td>
<td>▲ (in 14%)</td>
<td>0</td>
<td>53</td>
</tr>
<tr>
<td>Major non-cardiac surgery</td>
<td>104</td>
<td>1–7</td>
<td>▲ (in 30%)</td>
<td>↓ (in 12%)</td>
<td>▲ (in 14%)</td>
<td>0</td>
<td>104</td>
</tr>
<tr>
<td>Major orthopaedic surgery</td>
<td>57</td>
<td>1–7</td>
<td>▲ (in 30%)</td>
<td>↓ (in 12%)</td>
<td>▲ (in 14%)</td>
<td>0</td>
<td>57</td>
</tr>
</tbody>
</table>

Several factors may contribute to the disturbed sleep pattern in the surgical patient (fig. 3). The magnitude of surgery is important, as the reduction in REM sleep, SWS and the lack of inherent rhythmicity are more pronounced after major (gastrectomy or vagotomy) than minor (hernia repair) surgery [17]. In questionnaire studies, the greatest incidence of sleep disturbances was also found after the more major surgical procedures [13, 17, 27]. The duration of the operative procedure was related to the duration of postoperative sleep disturbance [17], probably as a result of more extensive surgical trauma.

In studies of patients receiving general or regional anaesthesia for herniorrhaphy [34], minor orthopaedic procedures [9] or minor undefined surgery [42], a similar degree of sleep disturbance was found, regardless of anaesthetic technique, suggesting that general anaesthesia per se is not an important pathogenic factor. Furthermore, general anaesthesia of 3 h duration in non-surgical volunteers produced only a modest reduction in SWS for 1 night and no changes in REM sleep [51]. Furthermore, similar changes in sleep pattern as are seen in postoperative patients have been found in non-surgical patients with acute ischaemic stroke [24], acute myocardial infarction [10], congestive heart failure [26] and medical and trauma ICU patients [20, 29, 60]. Thus general anaesthesia per se must be of minor importance in the pathogenesis of postoperative sleep disturbance, compared with the physical stress response caused by the surgical trauma.

The injury of surgery is followed by a complex...
stress response involving hormones and humoral mediators of the endocrine–metabolic system, immunosuppression and an inflammatory response [6, 35]. The inflammatory reaction constitutes the local response, whereas endocrine–metabolic activation with increased plasma concentrations of catabolic hormones and reduced concentrations of anabolic hormones, leads to hypermetabolism with acceleration of most biochemical reactions, including muscle protein breakdown, resulting in a negative nitrogen balance [35]. The response may last for a few days or even weeks, depending on the magnitude of the surgical insult and occurrence of complications.

The increased postoperative sympathetic activity with increased catecholamines may contribute to postoperative sleep disturbances, as high levels of noradrenergic activity maintain wakefulness [28]. The postoperative influence of surgical trauma on insulin is biphasic, with an impaired insulin response to glucose during the initial phase followed by an increased insulin response but with concomitant increased peripheral insulin resistance (post-receptor defect) [35]. Cortisol, another of the key mediators in the endocrine response to surgery, causes reduction in REM sleep and increases non-REM sleep when administered to healthy volunteers [19, 21]. Furthermore, corticotrophin-releasing hormone, which is increased in surgical stress, decreases non-REM sleep and increases wakefulness in a dose-dependent manner in rabbits and rats [55, 67]. In contrast, growth hormone releasing hormone (GHRH) may have a sleep inducing effect [69], and is probably increased during the postoperative period.

Administration of one of the key mediators of injury, interleukin-1, into the lateral ventricle of rabbits results in hyperthermia and increased non-REM sleep, with suppression of REM sleep, thus resembling the postoperative situation [54]. Pre-treatment with an interleukin-1 receptor antagonist prevented interleukin-1-induced sleep disturbances [54]. Endotoxin administration to healthy volunteers [70] and intracerebroventricular injection of tumour necrosis factor in rabbits [33] also caused deprivation of REM sleep and an increase in non-REM sleep, with no changes in SWS. As REM sleep is controlled by many regions in the brain and there is a dynamic interaction between cortex and other subcortical systems [32], the postoperative REM sleep disturbance may represent a global excitatory effect of the entire surgical stress response on the brain, rather than being caused by specific mediators of the surgical stress response.

Fever, which is seen commonly in postoperative patients, is associated with decreased REM sleep and increased SWS in rats with fungal infections [36]. It is not known if the fever-induced sleep disturbance is mediated by cytokines only or if it is induced by the increased temperature per se. Morphine 0.1 and 0.2 mg kg$^{-1}$ i.m., administered to healthy volunteers without pain, disrupted sleep in a dose-dependent manner [52]. After a dose of 0.1 mg kg$^{-1}$, only SWS was reduced, whereas a larger dose of 0.2 mg kg$^{-1}$ produced a marked reduction in both REM sleep and SWS with an increase in nocturnal awakenings [52]. There are no data on the effect of systemic or extradural morphine on sleep in postoperative patients.

After operation, pain is the most commonly reported cause of night-time awakenings [13], and based on questionnaires, provision of analgesia is the most helpful intervention to improve sleep [13]. No comparisons have been made between opioid and non-opioid drugs. In patients with pain from active rheumatoid arthritis, sleep disturbances similar to the postoperative period have been demonstrated [49]. The role of pain per se, that is nociceptive stimulation vs concomitant release of inflammatory mediators (cytokines) and stress hormones, has not been evaluated.

The noise level in an ICU is usually greater than is internationally recommended at all times of the
day and night [5]. Acoustically disturbed sleep has a decreased subjective quality, with less SWS than undisturbed sleep [23]. Patients have reported noise as one of the most disturbing factors of sleep in the postoperative period on the ward [7, 13] and in the ICU [29]. In addition, other “external” factors such as frequent observations and therapeutic procedures [29] and a high room temperature [43] may have a negative effect on sleep pattern in the surgical patient. An i.v. catheter did not affect sleep in volunteers [37], but starvation, as seen in most patients after major procedures, may be associated with decreased REM sleep and increased SWS, as shown in volunteers [45].

Obstructive sleep apnoea (OSA) syndrome

Special mention should be made of patients with the sleep apnoea syndrome. Sleep apnoea is defined as a pause of more than 10 s in breathing during sleep, and OSA is defined as at least five apnoeas per hour [48]. OSA is a common finding in non-surgical patients, with an incidence of 1–9% [48], although the more severe forms are less common. It has several major implications for anaesthesia and surgery throughout the operative period. These patients have repetitive episodes of often severe hypoxia and may have pulmonary hypertension. They may have impaired reflex responses to hypoxia, hypercapnia and inspiratory loads, and may also have altered autonomic cardiovascular reflexes [14, 71]. The treatment of OSA with nasal continuous positive airway pressure (CPAP) is highly effective at preserving the airway during sleep and over several weeks can improve the diminished reflex responses to hypoxia and hypercapnia [44]. However, it is more prudent to assume that they do not respond normally to any of these challenges during anaesthesia and in the postoperative period. They may be vulnerable to depressant medication such as benzodiazepines and opioids [12], and respiratory arrest can occur if these are given. All common anaesthetic induction agents, volatile and i.v., reduce upper airway tone in an identical manner to deep sleep. Some of the predisposing causes of sleep apnoea, such as a large tongue and recessive mandible, are also associated with difficulty in tracheal intubation and maintenance of a patent airway. Recovery from surgery and anaesthesia, while the normal protective reflexes are returning, may be a hazardous time, and obstruction of the airway can be difficult to overcome. This must be borne in mind after return to the ward if sedation or opioid analgesia is given. Regional methods of pain relief are believed to be safer than systemic opioid analgesia [18]. If patients are treated with CPAP, it is important that they bring their machines into hospital as they can be used in the recovery unit to preserve the airway until full recovery occurs and on the ward while their sleep returns to normal.

No data are available about postoperative sleep in patients with sleep apnoea syndrome, but extreme episodic oxygen desaturation in the late postoperative period has been described in a patient with the sleep apnoea syndrome [61], which may therefore be a potential surgical risk factor.

Clinical implications of postoperative sleep disturbances

The clinical consequences of suppression of REM sleep and SWS with subsequent rebound in the postoperative period remain unknown, although there are several potentially important implications. The postoperative rebound of REM sleep in the middle of the first postoperative week may contribute to the development of sleep-disordered breathing and nocturnal hypoxaemia [38, 65]. Episodic hypoxaemia is more frequent during periods of REM rebound than during other sleep stages in the postoperative period [65].

REM sleep is associated with profound sympathetic activation on nights with postoperative rebound [41] and in normal subjects [46, 68]. REM sleep in normal subjects is associated with haemodynamic instability [40, 47, 68], and increased mean arterial pressure [47, 68]. In the postoperative period these haemodynamic changes may occur with the hypoxaemic episodes [38, 65], and following uncomplicated abdominal surgery, episodic hypoxaemia was associated with cardiac ischaemia [22, 59, 64]. Sleep apnoea and episodic oxygen desaturation may induce life-threatening cardiac arrhythmias in non-surgical patients [25]. As the amount of REM sleep and the intensity of its phasic events increase in the postoperative period with simultaneous episodic hypoxaemia and haemodynamic instability, postoperative REM sleep rebound may be particularly dangerous and lead to postoperative myocardial ischaemia, infarction and eventually to unexpected postoperative death. This hypothesis warrants further study, but is supported by the finding that the majority of unexpected postoperative deaths occurred at night [63].

Postoperative impairment of mental function is a common complication in the elderly, with an incidence of 7–77% after major surgery and a peak incidence in the middle of the first week after operation [1, 48, 53, 62]. The pathogenic mechanisms are not completely understood [1, 48, 53, 62], but factors such as hypoxaemia [1, 62] and sleep disturbances [27] may be important. After sleep deprivation in non-surgical volunteers, impaired mental performance has been demonstrated on a simple reaction time test [8, 23], as decreased ability for creative thinking [31], and by impaired ability to perform other cognitive functions [31]. The performance deficit is related to age [30] and to loss of sleep per se rather than to changes in the composition of sleep [8, 23]. Mood is affected also by sleep deprivation with decreased vigour and an increase in the subjective feelings of sleepiness [2, 23, 30] and fatigue [30]. As the postoperative sleep disturbances are pronounced and of the same magnitude as in the above-mentioned studies in volunteers, postoperative sleep disturbance may be an important contributing factor in the development of postoperative cerebral dysfunction. Furthermore, postoperative sleep disturbance could be a con-
tributing factor in the phenomenon of postoperative fatigue [11], although a direct relationship between postoperative fatigue and sleep abnormalities remains to be demonstrated.

Prevention and treatment of postoperative sleep disturbances

As the magnitude and duration of the surgical procedure determines the degree of postoperative sleep disturbance [9, 17], a reduction in the surgical stress response by minimally invasive surgery may be effective. So far, there are no comparative studies between sleep patterns after the same operation performed either as a traditional open procedure or by minimally invasive surgical techniques. Stress reduction by neural block with local anaesthetics [35] may be expected to improve postoperative sleep also, but no data are available from studies using continuous nerve block techniques. As previous studies have suggested that pain [13, 49] and morphine [52] disturb postoperative sleep, continuous analgesic techniques with neural block using local anaesthetics without morphine should be evaluated, and the effect of NSAID because of their well-documented opioid-sparing effects. Preliminary studies with ketorolac after lower abdominal surgery have shown decreased opioid requirements and increased quality of sleep on the first postoperative night [56].

Noise [7, 13, 20], nocturnal nursing procedures [29], starvation [45] and increased room temperature [43] are sleep disturbing factors that could be eliminated in the postoperative period.

It has been reported that about 30% of medical and 88% of surgical patients receive sedative drugs during hospitalization [57]. Some of the most commonly used drugs are benzodiazepines, which have an important influence on sleep architecture with attenuated REM sleep and SWS, and increased stage 2 sleep [16]. Recent studies have suggested that the newer hypnotics zopiclone and zolpidem are less likely to alter sleep architecture [16] and that they may increase REM sleep and SWS to a normal level [18]. The effect of these drugs on the newer hypnotics zopiclone and zolpidem are less likely to alter sleep architecture [16] and that they may increase REM sleep and SWS to a normal level [18]. The effect of these drugs on the newer hypnotics zopiclone and zolpidem are less likely to alter sleep architecture [16] and that they may increase REM sleep and SWS to a normal level [18]. The effect of these drugs on the newer hypnotics zopiclone and zolpidem are less likely to alter sleep architecture [16] and that they may increase REM sleep and SWS to a normal level [18]. The effect of these drugs on the newer hypnotics zopiclone and zolpidem are less likely to alter sleep architecture [16] and that they may increase REM sleep and SWS to a normal level [18].

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As previously mentioned, patients with sleep apnoea syndrome are more susceptible to respiratory depression after sedative and narcotic administration [12] and the significance of the sleep apnoea syndrome as a surgical risk factor [61] should therefore be explored in future studies.

In summary, postoperative sleep disturbances represent an important research field, as they may have a significant negative impact on postoperative outcome.

References

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