Association of airway obstruction, sleep, and phasic abdominal muscle activity after upper abdominal surgery

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We recorded nasal gas flow, sleep stage, and abdominal muscle EMG pattern in 11 patients throughout the night after abdominal surgery, to examine the association between phasic activity of the abdominal muscles, sleep stage, and flow disturbance. We used a miniaturized data logging system, and obtained satisfactory records in eight patients. The data were divided into 30-s epochs. Each epoch was classified as either awake or asleep. The epochs were also classified for the presence of phasic activity in the external oblique abdominal muscle, and for evidence of airway obstruction. Association between these features was tested by a quasi likelihood log linear model. Values given are median (quartiles) for the eight subjects. Sleep occurred for 62 (46–69)% of the study time. During sleep, inspiratory flow was normal for 69 (48–81)% of the time, whereas during wakefulness, the flow pattern was normal for 51 (28–77)% of the time. Phasic activity was present 16 (12–25)% of the time during sleep and 24 (19–37)% of the time during wakefulness (P<0.001). In the awake state, when breathing was normal, phasic activity was present 16 (11–30)% of the time. When breathing was obstructed, phasic activity was present 38 (25–44)% of the time (P<0.001). These surprising findings suggest that sleep may be seriously disturbed by airway obstruction, so that a stable sleep state is not reached. We could not confirm previous findings that disturbed breathing in post-operative patients only occurs during sleep.

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In patients who have had abdominal surgery, frequent episodes of hypoxaemia have been reported during sleep in the night after surgery.¹ These episodes were associated with abnormal chest wall movements consistent with the presence of airway obstruction. We observed abdominal muscle activity in patients after upper abdominal surgery, with a phasic pattern of greater activity during expiration.² This activity is significantly associated with airway obstruction and abnormal chest wall movements,³ has a marked effect on abdominal pressure,⁴ and is likely to reduce functional residual capacity and worsen gas exchange. However, this pattern of activity is not constant, and periods occur when the muscles are tonically active. We considered that because airway obstruction has been associated with both episodic hypoxaemia and sleep, there should be an association between airway obstruction, sleep, and phasic muscle activity. We designed an observational study to examine the relationship, considering it likely that during sleep, as during anaesthesia,⁵ abdominal muscle activity would be phasic.

Methods

The study was approved by the relevant subcommittee of the Health Board Ethics Committee. On the day before surgery, we asked patients to participate and obtained written informed consent. After surgery, the patients were seen in the recovery area and the following electrodes and sensors were applied. Sleep was monitored by electroencephalography (EEG) with silver/silver chloride electrodes applied at scalp sites Cz-Pz and C3-C4, by electrooculography (EOG) using disposable gel electrodes above and lateral to the eyes, and by submental electromyography (EMG). We used a pair of similar disposable gel electrodes placed in the right upper quadrant of the abdomen to measure external oblique abdominal muscle EMG, another pair of gel electrodes to measure ECG, and placed a strain gauge sensor round the lower rib cage to measure chest wall excursions. The strain gauge also carried a

We placed a nasal pressure cannula (ProTek airflow sensor) (similar to a very narrow nasal oxygen cannula) at the nostrils and connected this to a miniature differential pressure transducer (Gaeltec 3EA). All the devices were taped firmly in place with adhesive dressings, and the cables were led away in a single bundle from the patient’s shoulder to a miniature data logging harness at the head of the bed. The submental EMG was filtered between 22 and 90 Hz with a 50 Hz notch, and the other sleep stage signals between 0.3 and 35 Hz. After the patient had returned to the ward, and was stable and comfortable, the recording was started with a portable polysomnographic recording system (Embla), and continued for 14 h.

The patients were nursed in a multi-bedded high dependency unit with a staff provision of one nurse for two patients. No special precautions were taken to ensure that the ward was quiet or to adjust the level of lighting, but the ward lights are kept low during the night hours. We did not have ethical permission to modify clinical practice in any way, and anaesthesia during surgery, and analgesia after the operation, were according to the preference of the anaesthetist involved. However, in all cases anaesthesia involved premedication with temazepam orally, i.v. induction of anaesthesia with propofol, neuromuscular block with either atracurium or vecuronium, and nitrous oxide and isoflurane to maintain anaesthesia. In five patients, a thoracic epidural catheter was placed at the start of anaesthesia so that post-operative analgesia could be provided using a continuous infusion of a mixture of bupivacaine (0.125%) with either fentanyl or diamorphine. In the other eight patients, analgesia was provided with a patient controlled injection device programmed to deliver morphine 2 mg i.v. with a 5 min lockout period between demands.

After overnight recording, the data from the portable logging system were downloaded onto a personal computer for analysis. For each patient, three separate passes through the system were needed. These passes were to determine the presence or absence of sleep, of phasic or tonic abdominal muscle activity, and of airway obstruction. Each of the three passes through the data was performed with the other traces hidden so that each trace was scored independently. The scorer who had been in contact with the patient was blinded to the identity of the data. Each coded tracing was analysed using the same synchronized 30 s epochs. In all cases, the operator classified each epoch if the feature was either present, absent, or could indicate ‘unsure’ if a clear distinction between the states could not be drawn. These features were sleep, phasic activity, and airway obstruction, depending on the variable being inspected. Sleep was determined by standard criteria.Each epoch was considered either wake or sleep if either state occupied more than 15 s (Fig. 1). Abdominal muscle activity was classified as phasic if a recognizable phasic pattern was present, with a frequency between 0.1 and 0.4 Hz, at any time during the 30 s (Fig. 2). Respiration was considered abnormal if there was any change in the clear inspiratory and expiratory pattern, or if the smooth domed appearance of the inspiratory wave was reduced in amplitude with a ‘flow limitation’ pattern (Fig. 3). After these sets of signals had been separately classified, the code was broken and the association between sleep stage, phasic activity, and abnormal nasal flow analysed. Each patient’s time was successively categorized in three ways: first, according to sleep state (awake/asleep); then according to the pattern of breathing (clear/obstructed); and finally according to abdominal muscle action (phasic/tonic).
The association between these categories was studied using a quasi likelihood log linear model.\textsuperscript{9} Other two-way associations were explored by the chi-squared test, with Minitab version\textsuperscript{10} run on a personal computer. All values for the patient data are given as median and quartiles for the eight subjects, unless otherwise stated.

**Results**

We recruited 13 patients. The planned surgery was changed in two of them, and in the remaining 11 patients, the recording of the nasal flow signal was technically inadequate in two, and in one subject both EEG leads became disconnected. We, therefore, obtained technically satisfactory recordings in eight patients with 14.2 (13.4–14.6) h of recording, and 95 (93–98)\% of these recordings were classifiable for all three channels. In the remainder of the recording, one or more signals had to be classified as ‘uncertain’. The most frequently unclassifiable signal was the abdominal EMG, which was classified as uncertain for nearly 3\% of the recording time.

The mean age of the patients was 39 yr (range 24–58), height 173 (SD 14) cm and weight 77 (15) kg. Four of the patients were male and five patients had thoracic epidurals. The other patients had analgesia with patient controlled i.v. morphine. The median total dosage of morphine in these patients, during and after surgery, up to the end of the study period, was 51 (15–90) mg. Most of the patients had operations for partial hepatic resection or pancreatic surgery.

The relative duration of each study period, spent in the different states, is shown in Table 1. The interactions between these states that could be studied are given in Table 2. The first two lines of this table present and test the \emph{a priori} hypothesis, but we first present some of the more general results.

Patients were awake for 38 (31–58)\% of the study period, and breathed normally for 54 (46–68)\% of the time. While the patients were awake, the breathing was normal for 51 (28–77)\% of the time.

Only one patient showed slow wave sleep (stage 3 or 4) and none showed any REM sleep. When the patients were asleep, breathing was normal for 69 (48–81)\% of the time. In six of the patients, breathing was normal for more of the time during sleep than during wakefulness.

Phasic abdominal muscle activity could be detected in all except one of the patients at some time in the recording period, and was not clearly altered in those patients who had epidural anaesthesia although there could have been a decrease in the amplitude of the action potentials in these patients. Phasic abdominal muscle activity was present for 25 (18–31)\% of the duration of the recordings. Phasic activity was less common when the patients were asleep. During sleep, activity was phasic for only 16 (12–25)\% of the time, whereas during wakefulness, phasic activity was present for 24 (19–37)\% of the time.

![Fig 3 Examples of nasal flow pattern during (A) normal breathing and (B) obstructed inspiration showing absence of expiratory flow and flow limitation during inspiration. Vertical lines indicate 5 s.](image_url)

<table>
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<th>Abdominal EMG subject</th>
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<th>No. 2</th>
<th>No. 3</th>
<th>No. 4</th>
<th>No. 5</th>
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<td>17</td>
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<tr>
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Table 1: Percentage of classifiable recording spent in different conditions.

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There were significant interactions between sleep state, breathing pattern, and phasic activity of the abdominal muscles. When awake, the patients had significantly more phasic abdominal muscle activity when the nasal airflow was obstructed ($P<0.001$), but there was no significant interaction between the pattern of abdominal activity and obstruction during sleep. When the breathing was normal, there was less phasic abdominal muscle action during sleep ($P<0.001$) (Fig. 4). One patient (number 8) had an unusually large proportion of phasic activity present during obstructed breathing while asleep. This patient was receiving an epidural infusion of 0.125% bupivacaine, which contained clonidine 0.6 \( \mu \text{g ml}^{-1} \).

### Discussion

This observational study was intended to answer a single question and help direct further investigation of postoperative breathing difficulties. We found a highly significant relationship between airway obstruction and phasic abdominal activity, but we were surprised to find that this relationship was only present during wakefulness. In addition, we were surprised to find that abnormal breathing was more frequent during wakefulness. When Catley and colleagues\(^1\) examined the relationship between episodes of hypoxaemia, breathing abnormalities, and sleep, they found that these episodes only occurred during sleep. On the basis of this past study and our clinical impressions, we expected to find abnormal breathing during periods of sleep. However, few workers, other than Catley and colleagues, have studied sleep state at the same time as respiratory disturbances in patients after surgery.

We used standard methods for measurement and ensured that the association we found was not affected by the possibility of bias, by conducting separate blinded classification of the three features. We have previously found measurement of respiratory flow with a nasal cannula system\(^10\) to be simple and effective for periods of several hours\(^11\) and the method has now been applied successfully for assessment of sleep disturbed breathing.\(^8\ 12\ 13\)

The standard method for determination of sleep stage that we used has been defined in criteria that were accepted by consensus as standard in 1968. These criteria are the current standard in sleep labs and are well known and widely used. The transition between wake and stage 2 is relatively unequivocal, defined by simultaneous changes in EEG and EMG signals. There are few other criteria used for sleep definition, so the validity of these criteria has not been widely tested but they are reliable and generally accepted. Unfortunately, sleep classification is a skilled and 'labour intensive' task, needing individual inspection and classification of successive 30-s epochs of recording.

Our patients often appeared asleep after surgery and the nurses considered that they were asleep. Analysis of the EEG and submental EMG showed, however, that a large proportion of this time was in fact wakefulness. Data on the extent of sleep and the validity of different methods of assessment are scanty, but our observations support those of

### Table 2

<table>
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<tr>
<th>Interaction between:</th>
<th>Quasi likelihood ratio test</th>
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others. In a study using the same criteria, patients similar to ours had an average nightly sleep duration of 2.5 h, which was estimated by the attendant nurses to be 7 h. Trained intensive care nurses are more accurate in this estimate, and assess the sleep state correctly on 73.5% of occasions, when observing medical ICU patients undergoing mechanical ventilation.15

These criteria for sleep may not be appropriate for patients receiving opioids. For example, the EEG frequencies that we noted during what was classified as wakefulness were often low. However, as there are no alternative widely used methods of staging, we chose to use the standard criteria. In addition, it is possible that we did not detect rapid and transient arousals, which are considered to be responsible for some of the symptoms found in patients with sleep-disordered breathing.

Finally, the recording of external oblique EMG is a simple and robust technique that we have used successfully in many post-operative patients. In the present study, it may have been affected by the presence of subclinical epidural motor block in some patients, although our patients showed no clinical evidence of abdominal weakness. Stimuli such as hypercapnia and elastic loads activate different abdominal muscles to different degrees. The external muscles, such as the external oblique, are less readiness activated than inner muscles such as the transversus abdominis.16 However, recording the activity of that muscle requires needle or wire electrodes, so we chose to study a muscle that can be assessed by surface electrodes, even though it gives a less sensitive indication of general expiratory abdominal activation. Despite this disadvantage, we still found a highly significant relationship.

Sleep quantity and quality were poor in our subjects, confirming previous studies of patients after surgery. Factors such as noise, frequent measurement of arterial pressure and other observations, pain, and some hormonol changes can reduce the capacity to attain deeper sleep.17 There is some evidence in man that morphine can reduce the amounts of both slow wave and REM sleep.18 Studies in cats suggest that this action involves a cholinergic pathway in the pontine reticular formation.19 20 Despite the evidence that opioid analgesics disturb sleep, most empirical evidence is that patients receiving opioid analgesia are drowsy, have evidence of increased airway resistance or frank obstruction, and have repeated episodes of hypoxaemia attributable to obstructive as well as central apnoea.21 22

The role of impaired airway control in post-operative sleep disruption, and the contribution of opioids to this impairment, is not clear. In patients with sleep disordered breathing, repetitive episodes of airway narrowing or obstruction can cause severe loss of sleep.23 Our patients showed more airway obstruction during wakefulness, than when sleeping. Airway obstruction can cause rapid arousal24 25 and it is possible that airway obstruction within a few moments of sleep onset was acting to rouse these patients. We classified an epoch when either obstruction or phasic abdominal activity was present, if there was any evidence of these features in the epoch. However, we only classified a 30-s epoch of recording as ‘sleep’ if more than half of this period was sleep pattern. This is a standard definition6 and periods of sleep shorter than this would be considered the waking state. It is possible that airway obstruction at sleep onset could lead to arousal, allowing an apparent association between airway obstruction and wakefulness. This would also explain why there was less airway obstruction in the sleeping patients: those patients who were able to sleep would be those patients with less difficulty breathing. To confirm this possibility it would be necessary to assess these features at the same time, to observe the exact time relationships within the epoch. We could not do this and at the same time make an unbiased assessment of the relationships between the features being studied, but are conducting further studies to address this question. Catley and colleagues analysed their data in epochs of 5 min, so the relationship between sleep and abnormal breathing movements in their study may have been very different.1

Abdominal muscle activation is prominent in patients receiving opioids for analgesia after surgery.2 Factors that may cause this activation include hypercapnia, partial airway obstruction,26 and increased respiratory drive.27 28 During sleep, respiratory drive is less, and this decrease in drive, and the reduced response to positive airway pressure,29 may result in less frequent phasic activity during sleep. In animals, however, abdominal muscles are more active during slow wave sleep.30 31 In the present study, the more frequent airway obstruction in the waking state could explain why phasic abdominal activity was likely during wakefulness. This increase in activity may aid ventilation, but at the expense of potentially reducing lung volume. This will impair gas exchange and reduce pulmonary oxygen stores making desaturation more rapid in the event of obstructive apnoea.32 Episodic obstruction33 and hypoxia34 can cause hypertension and tachycardia, by a range of possible neural and humoral mechanisms, which may be harmful in patients after surgery.

Our findings suggest that airway obstruction can cause sleep disturbance and augment abdominal muscle activity. Satisfactory methods that can reduce these features after surgery remain to be defined, but could include non-opioid analgesics that have less influence on airway control than morphine.

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References
with ventilatory pattern and analgesic regimen. Anesthesiology 1985; 63: 20–8
18 Kay DC, Eisenstein RB, Jasinski DR. Morphone effects on human REM state, waking state and NREM sleep. Psychopharmacology 1969; 14: 404–10
19 Keifer JC, Baghdoyan HA, Lydic R. Sleep disruption and increased apneas after pontine microinjection of morphine. Anesthesiology 1992; 77: 973–82
21 Jones JG, Sapsford DJ, Wheatley RG. Postoperative hypoxemia—mechanisms and time course. Anesthesia 1990; 45: 566–73
22 Stone JG, Cozine KA, Wald A. Nocturnal oxygenation during patient-controlled analgesia. Anesthesiology 1999; 89: 104–10
34 Fletcher EC. Effect of episodic hypoxia on sympathetic activity and blood pressure. Respir Physiol 2000; 119: 189–97