HAEMODYNAMIC EFFECTS OF PERIODIC VENTILATION: ABOLITION WITH SUPPLEMENTARY OXYGEN

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SUMMARY
A 63-yr-old man was noted to breathe with a periodic pattern of ventilation during sleep, both before and after operation for coronary artery grafting, and 6 weeks later after aortic aneurysm repair. Periodic ventilation was associated with cyclic oxygen desaturations and increases in heart rate and arterial pressure. Administration of oxygen was found to abolish consistently the periodic pattern and the associated haemodynamic changes.

KEY WORDS

Episodic decreases in arterial oxygen saturation (\(\text{Sa}_\text{O}_2\)) during sleep are common in both the early [1] and late postoperative periods [2], and are thought to be caused by obstructive or central apnoea and hypopnoea. In patients with obstructive sleep apnoea (OSA), increases in heart rate and arterial pressure accompany arousals and the release of episodic ventilatory obstruction [3]. The present report documents heart rate and arterial pressure fluctuations occurring with episodes of central apnoea in a patient with a periodic pattern of ventilation. We also describe the effect of supplementary oxygen on ventilatory pattern and haemodynamic changes.

PATIENT AND METHODS
During an investigation of pre- and postoperative arterial oxygen saturation in patients undergoing elective major abdominal vascular surgery, the patient was studied on two occasions. He was studied initially before and after coronary artery grafting, and again 6 weeks later, before and after aortic reconstructive surgery. On both occasions, pre- and postoperative \(\text{Sp}_\text{O}_2\) and heart rate were measured and displayed once every 1 s from an Ohmeda 3700 pulse oximeter, using a computer program designed for respiratory sleep studies. Respiratory monitoring on both occasions before and after operation used an Oxford Medical MPA portable sleep and respiration recorder with an oximeter, mercury-in-rubber strain gauges, and nasal and oral airflow thermistors.

On several nights after both surgical procedures, arterial pressure was measured noninvasively with a Finapres (Ohmeda), and the digital output of the Finapres (systolic, diastolic AP) was sampled each beat by a microcomputer, simultaneously with \(\text{Sp}_\text{O}_2\) data from the oximeter.

CASE REPORT
A 63-yr-old man (weight 70 kg, height 166 cm) was admitted for repair of an abdominal aortic aneurysm. He had no respiratory history of significance and did not snore heavily, although his wife reported that for years he had breathed heavily and deeply during sleep. He was mildly hypertensive, but not receiving medication. He had symptoms of effort-dependent chest pain and nocturnal angina. A preoperative radionuclide cardiac gated blood pool (MUGA) scan indicated an ejection fraction of 31% and severe ischaemic heart disease. This was confirmed by angiography, and he underwent coronary artery grafting 6
weeks before attempted aneurysm repair. For both procedures, the patient received a standard anaesthetic technique using fentanyl during operation, with an i.v. infusion of morphine for postoperative analgesia.

RESULTS

Preoperative recordings

A pattern of nearly symmetrical de- and resaturation and modest cyclic changes in heart rate typical of central apnoea was noted, with an apnoea/hypopnoea index of 69 per hour of sleep time, with a desaturation index of 54 per hour of study. Similar results were obtained before aortic aneurysm surgery.

Postoperative respiratory patterns

Following both surgical procedures, 50% oxygen was given via a face mask. The first night was free from significant respiratory events. The following night (postoperative night 2) supplementary oxygen was discontinued. In addition to a baseline \( S_{Po_2} \) of 85%, there were episodic decreases in saturation to about 70%, associated with increases in heart rate (fig. 1). Reinstitution of oxygen therapy prevented the desaturations and abolished the periodic increases in heart rate for the remainder of the night. During the 1-h period without supplementary oxygen, the patient suffered 62 episodes of apnoea, resulting in decreases in \( S_{Po_2} \) to 70–75%. There were three episodes of apnoea during the next 6 h of oxygen therapy. Comparable findings were recorded after aortic aneurysm repair.

Arterial pressure and periodic breathing

Figure 2 shows systolic arterial pressure and \( S_{Po_2} \) on day 3 after aortic reconstructive surgery while the patient breathed room air and while he breathed oxygen 6 litre min\(^{-1}\) via face mask. Cyclic changes in arterial pressure occurred with each cycle of desaturation, with room air. With the increase in \( S_{Po_2} \) after supplementary oxygen, periodic ventilation ceased and the arterial pressure pattern became more stable. Brief discon-
tinuation of oxygen resulted in the reappearance of the previous pattern. Identical findings were recorded after coronary artery graft surgery.

DISCUSSION
Abnormal patterns of ventilation (central or obstructive apnoeas, or hypopnoeas), are seen frequently in both the early and late postoperative periods [1, 2]. In patients undergoing major abdominal vascular surgery, we have frequently observed periodic breathing in the postoperative period. The cause of these patterns of breathing may be related to opioid sedation, hypoxaemia or decreased cardiac output and prolonged circulation time [4, 5]. Our patient’s periodic ventilation before operation may have been a result of his reduced ejection fraction [4-6].

Periodic breathing occurs also during sleep in healthy individuals at high altitude [5]. In these individuals, administration of oxygen eliminates periodic breathing and is associated with a concomitant increase in $P_{\text{a}}\text{CO}_2$ [7]. Under hypoxic conditions, administration of carbon dioxide abolishes periodic breathing promptly. These chemoreceptor reflexes are likely also to become unstable if the circulation time from the lungs to the chemoreceptors is increased [5].

Arterial pressure fluctuations during obstructive sleep apnoea are well documented [8]. This case report demonstrates that, in our patient, periodic ventilation was accompanied also by cyclic increases in arterial pressure and heart rate. The mechanism for these periodic alterations in pressure is believed to arise from chemoreceptor stimuli, synchronous with those controlling respiration [9].

It is not certain if fluctuations in heart rate, arterial pressure and $S_{\text{a}}\text{O}_2$, accompanying periodic ventilation are harmful to patients with ischaemic heart disease. Cardiac work is increased in association with cyclic hypertension and it is of interest that this patient complained of nocturnal angina before coronary artery grafting.

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REFERENCES