SLEEP APNOEA SYNDROME AND ANAESTHESIA

FRANCES CHUNG AND RONALD R. CRAGO

ABSTRACT

The sleep apnoea syndrome is reviewed, defined, and classified. Particular emphasis is placed on the identification of a patient population that is prone to sleep apnoea and the diseases and syndromes that are associated with it. For anaesthetists, direct enquiry into daytime and nightime sleep abnormalities and careful examination of the upper airway are important for preoperative detection of these patients and especially patients with obstructive sleep apnoea who might present for anaesthesia and operation. A typical case is reported and details of the preoperative, peroperative and postoperative management are discussed.

KEY WORDS: VENTILATION, sleep apnoea; COMPLICATIONS, respiratory obstruction, central apnoea.

HISTORY

CHARLES DICKENS in his Pickwick Papers, described a boy who was obese and hypersomnolent.[1] In 1918 Sir William Osler described a syndrome of obesity, hypersomnolence, cyanosis, and coined the term "Pickwickian Syndrome".[2] In 1956 Burwell described a patient so somnolent that, having been dealt a poker hand of three aces and two kings, he dropped off to sleep and failed to take advantage of his opportunity. He included obesity, hypersomnolence, periodic breathing with hypoventilation, and cor pulmonale in the syndrome.[3] In 1956 Gastaut first described multiple respiratory pauses occurring during sleep in a Pickwickian patient.[4] In the past decade, there have been numerous advances in the diagnosis of and research into the sleep apnoea syndrome. It is now well established that the sleep apnoea syndrome can occur in the absence of obesity and with normal carbon dioxide responsiveness. This review is an attempt to provide an awareness of this entity and the problems in its anaesthetic management.

DEFINITION

Sleep apnoea denotes cessation of airflow at the mouth and nose for ten seconds or more.[5] Such episodes can occur in normal persons, particularly during the onset of sleep, during rapid eye movement bursts, and following body movements.[6] Sleep apnoea is diagnosed if, during seven hours of nocturnal sleep, at least thirty apnoeic episodes are observed both in rapid eye movement and non-rapid eye movement sleep, some of which must appear repeatedly in non-rapid eye movement sleep.[7] Symptomatic patients with sleep apnoea syndrome might stop breathing hundreds of times during a single night with episodes of apnoea lasting between 20 and 90 seconds. Sometimes these patients are apnoeic during 50 per cent of sleep time.

CLASSIFICATION

Sleep apnoea may be classified as obstructive, central or mixed.[4] *Obstructive apnoea* is the most common, occurring in 85–95 per cent of various series. Obstructive apnoea is characterized by persistence of diaphragmatic and chest wall movement but lack of effective airflow at the nose and mouth because of upper airway obstruction. *Central apnoea* is characterized by cessation of both airflow and respiratory movements. *Mixed apnoea* is defined by cessation of airflow and an absence of respiratory effort early in the episode followed by resumption of unsuccessful respiratory effort in the latter part of the episode.

INCIDENCE

The incidence of the sleep apnoea syndrome has not been identified. Its true occurrence is probably underestimated. Sleep apnoea can

Frances Chung, M.D., F.R.C.P. (C), Lecturer; Ronald R. Crago, M.D., F.R.C.P. (C), Assistant Professor; Department of Anaesthesia, University of Toronto, Toronto Western Hospital, Toronto, Ontario, Canada.

occur at all ages, even in infants and children. It may be more prevalent in the elderly, but its significance is unknown. Severe degrees are most often noted in men older than forty years. A marked male over female predominance is evident. Most women with sleep apnoea are postmenopausal and severe sleep apnoea is relatively unusual in women lacking aggravating factors such as morbid obesity.[8] There is a slight suggestion that obstructive sleep apnoea might have a familial basis.[9]

Diseases and Syndromes Associated With Sleep Apnoea

A number of previously separate clinical syndromes share the essential features of sleep apnoea, and may therefore be classified with it. These include the Pickwickian syndrome,[10] central alveolar hypoventilation, and Ondine's curse.[11] It has also been suggested that the sudden infant death syndrome and near-miss might be disorders related to sleep apnoea.[12]

Several conditions might contribute to anatomical upper airway obstruction and result in excessive daytime somnolence. Among these conditions are micrognathia with bird-like facies,[13,14] temporomandibular joint disturbances,[15] deviated nasal septum,[16] acromegaly with macroglossia,[17,18] enlarged tonsils and adenoids,[19,20] laryngeal stenosis, anterior laryngeal web,[21] vocal cord paralysis,[22] supraglottic oedema following irradiation of the hypopharynx,[23] nasopharyngeal carcinoma[24] or lymphoma involving Waldeyer's ring.[25]

Sleep apnoea might be seen in patients with damaged respiratory centres as in cranial trauma, cerebral tumour,[26,27] encephalitis,[28] brain stem infarct, carotid body chemodectoma, bilateral cordotomy,[29,30] and bulbar poliomyelitis.[7] It might occur with abnormality of the breathing apparatus as in myotonic dystrophy,[31] kyphoscoliosis,[32] chronic obstructive pulmonary disease.[33] It might be associated with Shy-Drager syndrome,[34] Down's syndrome,[35] myxoedema,[36] and diabetes mellitus with autonomic polyneuropathy.[37]

Mechanism of Obstructive Sleep Apnoea

The site of obstruction has been shown to occur at the level of the oropharynx. During inspiration, the pressure within the extrathoracic airway is less than the atmospheric pressure without, leading to a tendency for that portion of the airway to collapse. In normal individuals respiratory neurones activate the oropharyngeal muscles, which dilate the oropharynx and maintain patency. However, in obstructive sleep apnoea, electromyographic measurements of oropharyngeal muscles show a disappearance of activity.[38] As apnoea continues, hypoxaemia and hypercapnia stimulate the inspiratory muscles, diaphragm and intercostals, which leads to greater negative pharyngeal pressure and greater collapse of the upper airway. When electroencephalographic arousal occurs, genio-glossus muscle activity increases, pulls the tongue forward, and terminates the apnoea.[39]

Pathophysiology

Pulmonary artery pressure increases with the vigorous inspiratory efforts made against a closed airway during upper airway sleep apnoea episodes. Very large intrathoracic pressure swings develop. At the onset of ventilation, intrathoracic pressure immediately drops to a normal range, but increases in pulmonary artery pressure are typically sustained.[7]

Pulmonary hypertension also results from protracted hypoxia and acidosis which induce vasoconstriction in the pulmonary vascular bed, with eventual onset of medial thickening and secondary right heart failure.[40]

Systemic hypertension is present in many cases. Abnormalities which contribute to both systemic hypertension and myocardial dysfunction include hypoxia and increased circulating catecholamines.

Continuous electrocardiographic monitoring shows a variety of arrhythmias during the night in association with repeated sleep apnoea.[41] The early part of the apnoeic periods are often accompanied by sinus bradycardia, atrioventricular block and prolonged asystoles. Increasing efforts are associated with tachycardia, ventricular extrasystoles and runs of ventricular tachycardia. The typical brady-tachycardia cycle offers strong presumptive evidence on Holter monitor for a diagnosis of sleep apnoea.[42] Arrhythmias have been the possible cause of sudden death during sleep in such patients.

Medical History

Direct inquiry into the possible daytime and nocturnal clues is essential. The history is by far the most important feature in making a diagnosis.

Sleep apnoea patients present with loud pharyngeal snoring, associated with snorting and interrupted by silences. The patient's struggling against respiratory obstruction can precipitate abrupt flinging of the arms and sudden sitting up or standing up during sleep. Other nocturnal symptoms might include restless sleep, somnambulism, nocturnal enuresis, insomnia and nightmares. Daytime hypersomnolence is the usual reason why patients seek medical treatment. It might be simple fatigue or sleepiness, so severe that the patient is unable to drive a car or carry on conversation without falling asleep. Patients might also complain of early morning headache, decreased performance levels, poor memory and poor judgment.[5]

Physical Examination

The majority of sleep apnoea patients have the ex-football-player habitus of stocky frame and short thick neck. Obesity is common but not invariably present.

On otolaryngological examination, the gag reflex is hyperactive in the majority of patients. Small ecchymoses or petechiae in the soft palate, or uvula are often present. There might be macroglossia, macrouvula, micrognathia, increased lymphoid tissue or glottic deformities.

Cardiovascular examination might show systemic or pulmonary hypertension or there could be signs of right or left heart failure.

Neurological examination might show signs of such associated conditions as myotonic dystrophy, or structural lesions of the brain or cervical spinal cord.

Diagnosis

Some patients may be screened by clinical observation during sleep, by nocturnal ear oximetry, or by demonstration of obstruction during sleep by fluroscopy of the neck or fiberoptic pharyngoscopy,[43] or intra-oesophageal pressure measurements. The presence of saw-toothing in the flow volume loop during inspiration might indicate that there is a great likelihood of significant upper airway obstruction associated with sleep.[44] The definitive laboratory study for diagnostic confirmation of sleep apnoea is polysomnography which involves polygraphic monitoring of sleep stages, air exchange, ventilatory effort, electrocardiogram, and arterial oxygen saturation.

Management

Although there is an increase in clinical awareness of sleep apnoea, medical treatment may not be very effective. Conservative therapy for this syndrome includes weight loss, removal of obstructing tissues, and avoidance of sedation and alcohol. Agents that enhance ventilatory responses such as medroxyprogesterone,[45] theophylline,[46] or doxapram hydrochloride, are not very effective. Recent studies show protriptyline hydrochloride[47] and strychnine sulphate[48] might be of some value.

It has been suggested that insertion of a nasopharyngeal airway at night might prevent obstructive sleep apnoea.[49] Continuous positive airway pressure applied through the nares has been shown recently to abolish obstructive sleep apnoea.[50] Its practical value has to be further evaluated. Sometimes the obstructive condition can be relieved by an operation such as tonsillectomy and adenoidectomy,[20] septoplasty or mandibular osteotomy.[51]

In some cases the only effective treatment available is tracheostomy, which is left open at night. There is immediate disappearance of daytime hypersomnolence, and the life threatening complications of hypoxaemia such as arrhythmias and cor pulmonale improve within days.[52] Diaphragmatic pacing may be lifesaving in central apnoea.[7]

Case Report

The patient was a fifty-six year old male who had a history of easy tiredness for years. He had a tendency to fall asleep during the daytime, at work, while eating, or while socializing with friends. He was involved in a car accident three years before admission due to falling asleep at the wheel. His wife had noticed that he snored heavily, was a restless sleeper at night, and stopped breathing at times during his sleep.

He had a past history of angina on exertion and of hypothyroidism. Medications taken were levothyroxin (Eltroxin) 0.15 mg o.d. Isosorbide dinitrate (Isordil) 40 mg q.i.d and propranolol (Inderal) 40 mg q.i.d.

On physical examination, he was an obese, plethoric man, 178 cm tall and weighing 100 kg. Head and neck examination showed a large tongue, large tonsils, large uvula and an abundant amount of soft tissue in the pharynx. Cardiovascular examination revealed a blood pressure of 130/90 mm Hg and pulse rate of

84/min. Heart sounds were normal. Respiratory examination showed an increase in anterior-posterior diameter of chest wall with slight decrease in air entry throughout. Abdominal and neurological examinations were normal.

Laboratory examination showed a haemoglobin of 16 g per cent. Blood urea, blood sugar, electrolytes and thyroid indices were normal. Arterial blood gases were $[H^+]$ 40 nmol/l (pH 7.40), Pa_{O_2} 9.84 kPa (74 mm Hg), Pa_{CO_2} 4.65 kPa (35 mm Hg), HCO_3 22 mmol/l. His electrocardiogram showed ST-T wave changes in the anterior precordial leads. Chest X-ray was normal. Pulmonary function tests were normal. Soft tissue lateral neck X-rays and tomogram showed evidence of bilateral nasopharyngeal masses causing encroachment on the air space. A sleep study revealed numerous episodes of obstructive sleep apnoea. He had a total of 401 apnoeic periods over 10 seconds in length during 6½ hours, the longest lasting 82 seconds. It was decided to examine him under anaesthesia, and to carry out a tonsillectomy.

Verbal reassurance and explanation was given during the preoperative visit. No preoperative sedation was given. On arrival in the operating room, intravenous and arterial lines were installed under local analgesia. The lungs were pre-oxygenated, pancuronium 1.0 mg was given, and fentanyl 140 μg was added slowly. This was followed by thiopentone 250 mg and, when a patent airway was assured, suxamethonium 100 mg was given. The trachea was intubated under cricoid pressure and the lungs were ventilated. Anaesthesia was maintained with nitrous oxide-oxygen, enflurane 0.5 per cent and pancuronium.

Tonsillectomy, uvulectomy and biopsy of the nasopharynx were done. Relaxation was reversed with atropine and neostigmine at the end of the operation, the trachea was extubated and he was given oxygen 6 l/min by nasal prongs. Half an hour after the anaesthesia, his arterial blood gases were $[H^+]$ 47 nmol/l (pH 7.33), Pa_{O_2} 15 kPa (113 mm Hg), Pa_{CO_2} 6.65 kPa (50 mm Hg), and HCO_3 26 mmol/l. He was monitored with ear oximetry for twenty-four hours postoperatively. No sleep apnoea was documented. After discharge from hospital, the daytime hypersomnolence had disappeared. An apnoea study confirmed the marked improvement.

Discussion

Anecdotal accounts of postoperative respiratory arrests in the recovery room and in the ward after patients had apparently recovered from the effects of anaesthetics are common. Did some of these patients belong to the group of unidentified sleep apnoea syndromes who were prone to upper airway obstruction and sensitive to hypnotics and narcotics? In fact, a higher incidence than usual of unexpected cardiorespiratory arrests in association with anaesthesia was reported in diabetic patients with autonomic neuropathy.[53,54] This suggests that abnormal ventilatory control was present and a recent study did show that these patients had a higher incidence of sleep apnoea.[37] There are two reported cases in the medical literature of death linking anaesthesia and sleep apnoea.[11,55]

It might be prudent for anaesthetists to inquire into a patient's pattern of sleep during the preoperative visit. They should familiarize themselves with the pattern of the patient population that is at higher risk such as the exfootballer status, micrognathia, myotonic dystrophy, kyphoscoliosis etc.

Preoperative Visit

An assessment of the airway is essential during the preoperative visit. An adequate assessment of the cardiovascular status is needed to ensure that there is no evidence of heart failure and that systemic hypertension is well controlled.

Our patient was given no preoperative medication. He arrived in the operating room calm and cooperative. Preoperative sedation is contra-indicated in sleep apnoea patients, as these patients are much more susceptible to respiratory depression and obstruction after premedication. Two patients with sleep apnoea and micrognathia, scheduled for operation, developed respiratory arrest after being given a small dose of sedative drugs.[14,56] Three patients have been reported who developed upper airway obstruction while awake, after intramuscular premedication with diazepam[16,33] and one after a combination of morphine, hydroxyzine and atropine.[57]

Induction

Induction in sleep apnoea patients can provoke a life-threatening situation. These patients may develop respiratory obstruction during induction, which is usually relieved by elevation of the jaw or insertion of a naso-pharyngeal or oropharyngeal airway. The trachea can be difficult to intubate because of the short thick neck,

obesity or associated anatomical anomalies such as micrognathia.[58] If there is any suspicion of the patency of the upper airway or of difficult intubation, awake intubation is indicated with the assistance of fiberoptic laryngoscopy, or induction with an inhalation anaesthetic should be considered with avoidance of muscle relaxants.

A tray for difficult intubations should be available with different sizes of laryngoscope blades, smaller sizes of tracheal tubes and introducer. Emergency cricothyrotomy might be necessary in a desperate situation.

Anaesthetic management of our patient was complicated by angina, sleep apnoea, and a short surgical procedure involving the upper airway. Awake intubation and inhalation induction were avoided in this case because of his cardiovascular status. Induction with a large dose of narcotic would protect his heart but might aggravate the sleep apnoea postoperatively. Small doses of thiopentone, fentanyl and pancuronium were used so that there was minimal effect on the cardiovascular and respiratory status.

Maintenance of Anaesthesia

It may be better to ventilate the lungs during operation because patients might develop apnoea. A patient with obstructive sleep apnoea has been reported who developed prolonged periods of central apnoea with halothane while the upper airway obstruction due to enlarged tonsils had been eliminated by tracheal tube.[59] The use of inhalation agents may be beneficial in order to decrease the amount of narcotics and sedatives required to maintain anaesthesia. If muscle relaxants are used, total reversal should be ensured.

Postoperative Management

An obstructive sleep apnoea patient has been reported who required mechanical ventilation for six days after tonsillectomy because of prolonged periods of central apnoea.[57] Therefore relief of airway obstruction might unmask a degree of central hypoventilation which had not been detected before operation.

Central sleep apnoea can occur after cervical cordotomy,[29] after anterior cervical spinal surgery[30] and in certain craniovertebral and hindbrain anomalies.[26]

Therefore, sleep apnoea patients should be carefully monitored for periods of apnoea and arrhythmia and should be kept in the recovery room for longer periods than usual. Ear oximetry, as in our patient, provides an easy method of continuous monitoring.

It has been shown that administration of high concentrations of oxygen prolongs sleep apnoea during REM and non-REM sleep.[7] Increases in carbon dioxide and acidosis were noted after oxygen administration. Presumably one of the reasons to resume breathing is severe oxygen desaturation and, if this does not occur, apnoea could be prolonged. It might be, therefore, that oxygen therapy in the recovery room should be well controlled to prevent hyperoxia.

References

1. Dickens, C. Pickwick Papers, 1837.
2. Osler, W. The Principles and Practice of Medicine, New York, Appleton, 8th Edit. 1918.
3. Burwell, C.S., Robin, E.D., Whaley, R.R. *et al.* Extreme obesity associated with alveolar hypoventilation – a Pickwickian syndrome. Am. J. Med. *21*: 811–818 (1956).
4. Gastaut, H., Tassinari, C.A. & Duron, B. Polygraphic study of the episodic diurnal and nocturnal manifestations of the Pickwickian syndrome. Brain Res. *2*: 167–186 (1966).
5. Guilleminault, C., Tilkan, A. & Dement, W.C. The Sleep Apnoea Syndromes. Ann. Rev. Med. *27*: 465–484 (1976).
6. Aserinsky, E. Periodic respiratory pattern occurring in conjunction with eye movements during sleep. Science *150*: 763–66 (1965).
7. Guilleminault, C. & Dement, W.C. Sleep apnoea syndromes, Liss, New York, 1978.
8. Clark, R.W. Sleep apnoea, Primary Care 6 (3): 653–679 (1979).
9. Kingman, P.S., Saunders, N.A., Feldman, N.T. & Hallett, M. Obstructive sleep apnoea in family members, N.E.J.M. 299 (18): 969–973 (1978).
10. Castaut, H., Lugaresi, E., Berti-Ceroni, G. & Coccagna, G. The abnormalities of sleep in man. Bologna, Gaggi, 1968.
11. Fruhmann, G. Hypersomnia with primary hypoventilation syndrome and following cor pulmonale (Ondine's Curse Syndrome). Bull Physiopathol. Respir. *8*: 1173–1179 (1972).
12. Guilleminault, C., Ariagno, R., Korobkin, R. *et al.* Mixed and obstructive sleep apnoea and near miss for sudden infant death syndrome: 2, Comparison of near miss and normal control infants by age. Pediatrics 64: (6): 882–891 (1979).
13. Tammeling, G.J., Blokzijl, E.J., Boonstra, S. & Sluiter, H.J. Micrognathia, hypersomnia and periodic breathing. Bull. Physio-path Resp. *8*: 1229–1238 (1972).
14. Coccagna, G., di-Donato, G., Verucchi, P. *et al.* Hypersomnia with periodic apnoea

in acquired micrognathia; a bird-like face syndrome. Arch. Neurol. *33*: 769–766 (1976).
15. WEITZMAN, E.D., POLLACK, C.P. & BOROWIECKI, B. Hypersomnia – sleep apnoea due to micrognathia – reversal by trachoplasty. Arch. Neurol. *35*: 392–395 (1978).
16. SIMMONS, F.B. & HILL, M.W. Hypersomnia caused by upper airway obstruction. Ann. Otol. *83*: 670–673 (1974).
17. PERKS, W.H., HORROCKS, P.M., COOPER, R.A. *et al.* Sleep Apnoea in acromegaly. Brit. Med. Journ. *29*: 894–897 (1980).
18. MEZON, B.J., WEST, P., MACLEAN, J.P. & KRYGER, M.H. Sleep apnoea in acromegaly. Am. J. of Med. *69*: 615–618 (1980).
19. MANGAT, D., ORR, W.C. & SMITH, R.O. Sleep apnoea hypersomnolence, and upper airway obstruction secondary to adenotonsillar enlargement. Arch. Otolaryngol. *103*: 383–386 (1977).
20. ORR, W.C. & MARTIN, R.J. Obstructive sleep apnoea associated with tonsillar hypertrophy in adults. Arch. Int. Med. *141*: 990–992 (1981).
21. SIMMONS, F.B., GUILLEMINAULT, C., DEMENT, W.C. *et al.* Surgical management of airway obstruction during sleep. Laryngoscope, *87*: 326–338 (1977).
22. WESTERMAN, D.E. & FERGUSON, A.D. Obstructive sleep-induced apnoea due to bilateral recurrent laryngeal nerve palsies. S. Afr. Med. J. *54*: 825–827 (1978).
23. BAKER, S.R. & ROSS, J. Sleep apnoea syndrome and supraglottic oedema. Arch. Otolaryngol. *106*: 486–491 (1980).
24. MOSES, F.M. & BUSCEMI, J.H. Obstructive sleep apnoea syndrome associated with nasopharyngeal carcinoma. West, J. of Med. 134 (1): 69–70 (1981).
25. ZORICK, F., ROTH, T., DRAMER, M. *et al.* Intensification of excessive daytime sleepiness by lymphoma. Sleep Res. *6*: 199 (1977).
26. KREIGER, A.J. Respiratory failure as a surgical risk in patients with hindbrain anomalies. Heart lung *2*: 546–551 (1973).
27. KELLY, D.H., KRISHNAMOORTHY, K.S. & SHANNON, D.C. Astrocytoma in an infant with prolonged apnoea. Paediatrics 66 (3): 429–431 (1980).
28. EFRON, R. & KENT, C.D. Chronic respiratory acidosis due to brain disease. Arch. Neurol. Psychol. *77*: 575–587 (1957).
29. KREIGER, A.J. & ROSOMOFF, H.L. Sleep-induced apnoea, part I: A respiratory and autonomic dysfunction following bilateral percutaneous cervical cordotomy: J. Neurosurg. *40*: 168–180 (1974).
30. KREIGER, A.J. & ROSOMOFF, H.L. Sleep-induced apnoea, part 2: Respiratory failure after anterior spinal surgery. J. Neurosurg. *40*: 181–185 (1974).
31. COCCAGNA, G., MANTOVANI, M., PARCHI, C., MIRONI, F. & LUGARESI, E. Alveolar Hypoventilation and hypersomnia in myotonic dystrophy. J. of Neurol. Neurosurgery & Psychol. *38*: 977–984 (1975).
32. GUILLEMINAULT, C., KARLAND, G., WINKLE, R. & MILES, L.E. Severe kyphoscoliosis, breathing and sleep. Chest: 79 (6): 626–630 (1981).
33. GUILLEMINAULT, C., CUMMISKEY, J. & DEMENT, W.C. Sleep apnoea syndrome: Recent advances. Adv. Int. Med. *26*: 347–72 (1980).
34. LOCKWOOD, A.H. Shy-Drager syndrome with abnormal respiration and antidiuretic hormone release. Arch. Neurol. 33: 292, 1976.
35. LOUGHLIN, G.M., WYNNE, J.W. & VICTORICA, B.E. Sleep apnoea as a possible cause of pulmonary hypertension in Down syndrome. J. of Paed. 98 (3): 435–437 (1981).
36. ORR, W.C., MALES, J.L. & IMES, N.K. Myxoedema and obstructive sleep apnoea. Am. J. of Med. *70*: 1061–66 (1981).
37. REES, P.J., PRIOR, J.G., COCHRANE, G.M. & CLARK, T.J.H. Sleep apnoea in diabetic patient with autonomic neuropathy. J. of Roy. Soc. of Med. *74*: 192–195 (1981).
38. GUILLEMINAULT, C. *et al.* Obstructive sleep apnoea: Electromyographic and fiberoptic studies. Exp. Neurol. *62*: 48 (1978).
39. REMMERS, J.E. *et al.* Pathogenesis of upper airway occlusions during sleep. J. Appl. Physiol. *44*: 931–938 (1978).
40. TILKAN, A.G., GUILLEMINAULT, C., SCHROEDER, J.S., LEHRMAN, K.L., SIMMONS, F.B. & DEMENT, W.C. Haemodynamics in sleep-induced apnoea. Ann. of Int. Med. *85*: 714–719 (1976).
41. TILKAN, A.G., GUILLEMINAULT, C., SCHROEDER, J.S., LEHRMAN, K.L., SIMMONS, F.B. & DEMENT, W.C. Sleep induced apnoea syndrome, prevalence of cardiac arrhythmias and their reversal after tracheostomy. Am. J. Med. *63*: 348–358 (1977).
42. IMAIZUMI, T. Arrhythmias in sleep apnoea. Am. Ht. Journ. 100(4): 513–516 (1980).
43. BOROWIECKI, B. *et al.* Fibre optic study of pharyngeal airway during sleep in patients with hypersomnia obstructive sleep apnoea syndrome. Laryngoscope, *88*: 1310–1313 (1978).
44. SANDERS, M.H., MARTIN, R.J., PENNOCK, B.E. & ROGERS, R.M. The detection of sleep apnoea in the awake patient. JAMA 245 (23): 2414–2418 (1981).
45. STROHL, K.P., HENSLEY, M.J., SAUNDERS, N.A., SCHARF, S.M., BROWN, R. & INGRAM, R.H. Progesterone administration and progressive sleep apnoea. JAMA *245*: 1230–1232 (1981).
46. HUDGEL, D.W. & SHUCARD, D.W. Coexistence of sleep apnoea and asthma resulting in severe nightime hypoxaemia. JAMA *242*: 2789–2790 (1979).
47. CLARK, R.W., SCHMIDT, H.S., SCHAOL, S.F. *et al.* Sleep apnoea: Treatment with protriptyline. Neurology, *29*: 1287–1292 (1979).
48. REMMERS, J.E. & ANCH, A.M. Sleeping and breathing. What to do for a lack of both. Arch. Int., Med. *141*: 989 (1981).
49. AFZELIUS, L.E., ELMQUIST, D., HOUGARRD, K., LAURIN, S., NILSSON, B. & RISBERG, A.M. Sleep apnoea syndrome – an alternative treatment to tracheostomy. Laryngoscope, *91*: 285–290 (1981).

50. SULLIVAN, C.E., ISSA, F.G., BERTHEN-JONES, M. & EVES, L. Reversal of obstructive sleep apnoea by continuous positive airway pressure applied through the nares. Lancet: 862–865 (1981).
51. KUO, P.C., WEST, R.A., BLOOMQUIST, D.S. & MCNEIL, R.W. The effect of mandibular osteotomy in three patients with hypersomnia sleep apnoea. Oral Surgery. *48*: 385–392 (1978).
52. GUILLEMINAULT, C., SIMMONS, F.B., MOTTA, J., CUMMINSKY, J., ROSEKIND, M., SCHROEDER, J.S. & DEMENT, W.C. Obstructive sleep apnoea syndrome and tracheostomy. Long term follow up experience. Arch. Intern. Med. *141*: 985–988 (1981).
53. PAGE, M. McB. & WATKINS, P.J. Cardiopulmonary arrest and diabetic autonomic neuropathy. Lancet *1*: 14–16 (1978).
54. GARCIA-BUNUEL, L. Cardiorespiratory arrest in diabetic autonomic neuropathy. Lancet *1*: 935 (1978).
55. TSEUDA, K., SHIBUTANI, K. & LETKOWITZ, M. Postoperative ventilatory failure in an obese myopathic woman with periodic somnolence, a case report. Anaes. & Analg. 54: (4): 523–526 (1975).
56. HISHIKAWA, Y., FURUYA, E. & WAKAMATSU, H. Hypersomnia and periodic respiration – presentation of two cases, and comment on the physiopathogenesis of the Pickwickian syndrome. Folia Psych. Neurol. Jap. *24*: (3), 163–173 (1970).
57. RAFFERTY, T.D., RUSKIS, A., SASAKI, C. & GEE, J.B. Perioperative considerations in the management of tracheostomy for the obstructive sleep apnoea patient. B. J. Anaes. *52*: 619–621 (1980).
58. SIMMONS, F.B. Tracheostomy in obstructive sleep apnoea patients. Laryngoscope *89*: 1702–1703 (1979).
59. ALIVERIO, R., FERMAN, C. & ABRAMOWITZ, R. Periodic apnoea during adenotonsillectomy. Anaes. & Anal. *58*: 50–52 (1979).

RÉSUMÉ

Le syndrome d'apnée du sommeil est passé en revue, défini et classifié. Les auteurs insistent sur l'identification de la population à risque et les maladies et syndromes qui s'associent à l'apnée du sommeil. L'anesthésiste doit s'enquérir de toute anomalie du sommeil et un examen poussé des voies respiratoires supérieures est indiqué spécialement pour les candidats à l'anesthésie et à la chirurgie qui présentent la forme obstructive. Un cas typique est rapporté et la conduite à tenir aux périodes pré-opératoire, per-opératoire et post-opératoire est discutée.