SLEEP APNOEA AND ANAESTHESIA

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History

Sleep apnoea was first described by William Shakespeare in his play "King Henry IV". Another description appeared in "The Posthumous Papers of the Pick wick Club" by Dickens. In 1918, Sir William Osler described a syndrome of obesity, hypersomnolence, cyanosis, and coined the term "Pickwickian Syndrome". 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. In 1956, Gastaut first described multiple respiratory pauses occuring during sleep in a Pickwickian patient.

Introduction

In relation to anaesthesia sleep apnoea is described as below in three sections:

1. Sleep-related breathing disorders.
2. Sleep and anaesthesia-their nature and effects on ventilation.
3. Anaesthesia and sleep disordered breathing.

Sleep-related breathing disorders

Definitions

1. Apnoea is defined as a cessation of oronasal airflow for more than 10 seconds
2. Hypopnoea is defined as a reduction in airflow of respiratory effort for more than 10 seconds plus a desaturation of 3% or more and/or EEG evidence of arousal.
3. Apnoea Hypopnea Index (respiratory disturbance index) is the number apnoeas and hypopnoeas per hour of sleep.

Classsification of Aponea

a) Obstructive (OSAS)
: There is persistent respiratory effort without airflow

b) Central (CSAS)
: Respiratory effort is absent due to transient withdrawal of central drive to the respiratory muscles. cause may be known or unknown, like in primary alveolar hypoventilation (Ondine's curse).

c) Mixed
: It is combination of obstructive and central sleep apnoca.

DIFFERENCE BETWEEN CSAS AND OSAS

CSAS	OSAS
Normal body habitus	Commonly Obese.
Insomnia, Hypersomnolence rare	Day time Hypersomnolence
Awaken during sleep	Rarely awaken during sleep
Snoring mild and intermittent	Loud snoring
Depression	Intellectual deteoriation
Minimal Sexual dysfunction	Sexual dysfunction more
	Morning headache.
	Nocturnal enuresis

Obstructive Sleep Apnonea Syndrome (OSAS)

Clinical Features

	Adult	Child
1. Snoring	Alternating with pauses	Continuous
2. Excessive day time sleeping	Main Symptom	Infrequent
3. Obesity	Very common	Infrequent
4. Failure to thrive/underweight	Not reported	Common
5. Mouth breathing	Usually not	Common
6. Male preponderance	Yes (8-10:1)	No (1:1 prepubertal)
7. Enlarge tonsils & adenoids	Uncommon	Most common
8. Most common obstructive pattern	Apnea	Hypopnea
9. Arousal on apnea termination	Very common	Uncommon
10. Sleep pattern disruption	Very common	Uncommon
11. Sleep pattern common during	REM stage	REM and NREM
12. Complication	Cardiopulmonary, EDS.	Cardio-pulmonary, FTT, behaviour
13. Surgery as a treatment	Selected cases, minority UP3	Most cases, T&A, UP3
14. CPAP treatment	Most common treatment	Selected case, minority
15. Mortality	Death during sleep, CVS	Usually preoperative.

CPAP = continuous positive airway pressure; CVS = cardiovascular; EDS = excessive daytime sleepiness; FTT = failure to thrive; T & A = tonsillectomy and adenoidectomy; UP3 = uvulopalatopharyngoplasty

Upper Airway Resistance Syndrome (UARS) is condition where habitual snores have recurrent arousals from sleep resulting from increase in upper airway resistance not sufficient to cause apnoeas or hypopnoeas.

Predisposing conditions: Obstructive sleep apnoea

Condition	Examples	Contribution
Obesity, body fat distribution.	Adult obesity,	
	Prader-Willi syndrome	Complex and ill defined
Race/genetics		Anatomical similarity
Age		Tissue laxity
Male gender		Unclear
Alcohol, sedatives,		Muscle relaxation, depressed arousal
analgesics, an aesthetics		
Smoking		Chronic nasal congestion, pharyngeal oedema
Nasal obstruction	Septal deviation, chronic nasal congestion	increased pharyngeal negative pressure
Pharyngeal obstruction	Tonsillar and adenoidal hypertrophy	increased pharyngeal nagative pressure
Cranio-facial abnormality	Down's, Pierre-Robin,	Mid-face hypoplasia, macroglossia or micrognathia
	Treacher-Collins,	
	Apert's, Crouzon's,	
	Beckwith-Wiedemann,	
	achondroplasia, acromegaly,	
	Fragile-X	
Laryngeal obstruction	Laryngomalacia, tracheomalacia	Laryngeal collapse.
Endocrine/Metabolic	Hypothyroidism, androgen	Upper airway infiltration or myopathy, obesity
	therapy, Cushing's	
Neuromuscuiar disorders	Stroke, cerebral palsy, head injury,	Disordered pharyngeal neuromuscular function
	Shy-Drager, poliomyelitis,	
	myotonicdystrophy,	
	dysautonomia, tetraplegia	
Connective tissue disorders	Marfan's	Abnormal upper airway connective tissue
Storage diseases	Mucopolysacchardoses	Macroglossia.
Chromic renal failure		Unclear

Symptoms associated with sleep apnoea

Adults	Children
Heavy snoring	Snoring
Excessive daytime sleepiness	Restless sleeping
Witnessed apnoeas	Somnolence/Aggression/behavioural problems
Sudden awakenings with 'chocking'	Hyperactivity
Accidents related to sleepiness	Odd sleeping postures
Poor memory/concentration	Frequent coughs/colds
Delirium	

Gastro-oesophageal reflux
Mood/personality changes
Nocturia
Enuresis (uncommon)
Dry month on awakening
Nocturnal or morning headache
Impotence
Nocturnal epilepsy

Signs associated with sleep apnoea

Oedematous soft palate of uvula
Long soft palate and uvula
Decreased oropharyngeal dimensions
Nasal obstruction
Maxillary hypoplasia
Retrognathia
Central adiposity/increased neck circumference
Hypertension and other cardiovasular consequences
Conditions/syndromes (already mentioned above)

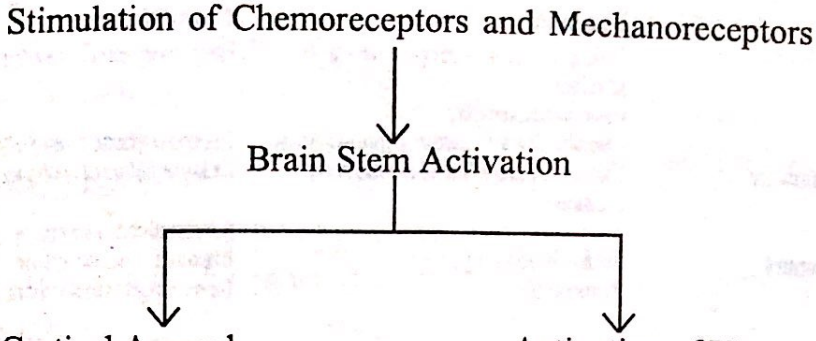
Potential sequelae of sleep apnoea

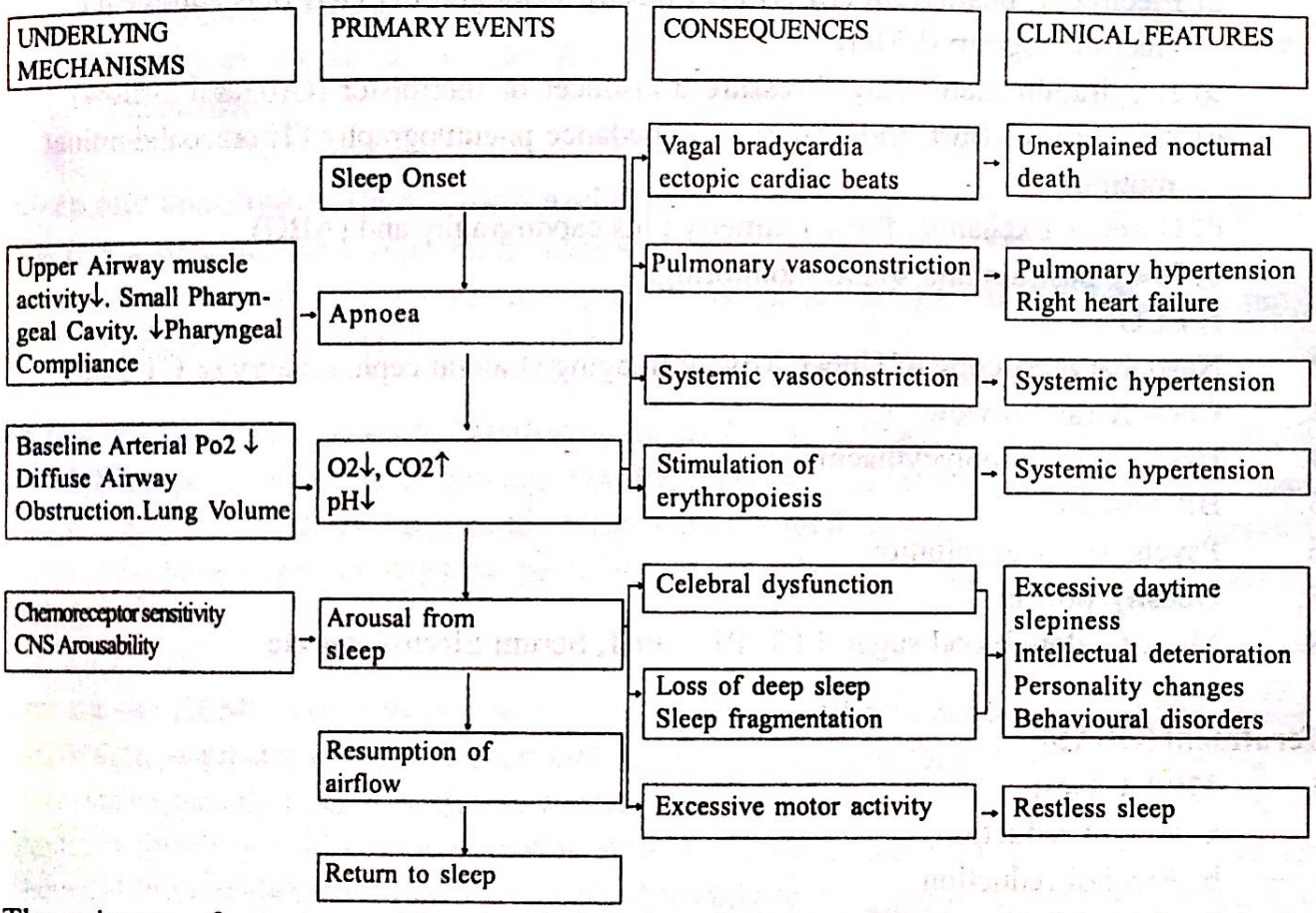
Neuropsychological	Sleepiness, impaired memory and cognition, decreased vigilance, increased accident risk, anxiety and depression, chronic headache, intracranial hyperstension
Cardiovascular	Hypertension, ischaemic heart disease, cerebrovascular disease, right heart failure.
Pulmonary	Hypoxaemia, hypercapnia, pulmonary hypertension.
Endocrine	Decreased growth hormone and testosterone levels, diabetic instability.
GIT	Gastro-oeaophageal reflux.

Site of obstruction in obstructive sleep apnoea syndrome.

Type	Site of obstruction
1.	Anterioposterior displacement of the tongue against the posterior pharynx.
2.	Posterior displacement of the soft palate by the tongue against the posterior pharynx
3.	Opposition of the lateral pharyngeal walls.
4.	Circular closure of the pharynx.

Sequence of events in the termination of OSAS





The primary of events resulting in obstructive sleep apnoeas, the resulting physiologic response and clinical features.

Central Sleep Apnoea Syndrome (CSAS)

Diminished or absent respiratory effort may occur in association with disorders of ventilatory control of neuromuscular function or where the respiratory musculature is excessively loaded. These conditions may result in diminished ventilatory capacity insufficient for their needs during wakefulness leading to hypoventilation during sleep and failure of compensatory mechanism. Consequences include hypoxaemia, hypercarbia, sleep disruption and daytime somnolence. Unrecognized and untreated, polycythaemia and/or respiratory and right heart failure may supervene if sleep related hypoventilation is sufficiently severe. Similar consequences can accompany hypoventilation due to severe OSA.

Predisposing conditions for central sleep apnoea

Condition	Examples	Contribution
Neuromuscular disorders	Poliomyelitis, amyotrophic lateral sclerosis muscular dysterophy	Respiratory muscles weakness
Excessive respiratory load	Obesity, airways disease, kyphoscoliosis	Excessive elastic, resistive of threshold loading of muscles
Disordered peripheral chemosensitivity	Cardiac failure, bilateral carotid body excision	Delay or failure of ventilatory feedback from peripheral chemoreceptors
Disordered central ventilatory control	Stroke, head injury	Impaired ventilatory drive
Endocrine/metabolic	Acromegaly	Increased growth hormone and insulin like growth factor

Investigation

1. Polysomnography (PSG)
   a) Electroencephalogram (EEG) plus electro-oculogram (EOG) plus sub-mental electromyogram (EMG)
   b) Respiration monitoring: Pressure transducer or thermistor (Oronasal airtlow).
   c) Respiratory effort: Inductance or impedance pneumography (Thoracoabdominal motion).
   d) Gaseous Exchange: Pulse oximetry plus capnograghy and (ABG).
   e) Body position and sound monitoring.
   f) ECG
2. Nasopharyngoscopy of Upper Airway Imaging (Lateral cephalometry or CT Scan)
3. Chest X-ray PA view
4. HB to rule out polycythaemia
5. BP Monitoring
6. Psychiatric Consultation
7. Obesity profile
8. Misc: random blood sugar, LFT, RFT, PFT, Serum Electrolytes etc.

Teratment (OSAS)

1. **Mild Cases:**
   a. Weight reduction,
   b. Alcohol reduction
   c. Sedative consumption reduction

2. **Moderate and severe cases:**
   The above attempts from an adjunct to the aggressive therapy, with may include
   a. Nasal continuous positive airway pressure (nCPAP)
   b. Bilevel positive airway pressure (BiPAP)
   c. Intermittent positive pressure ventilation (IPPV)
   These therapies can be delivered by means of special masks or through endotracheal tubes.

3. **Surgical Therapy:**
   a. Tonsillectomy and adenoidectomy

b. Uvulopalatopharyngoplasty
c. Septoplasty
d. Hyoid advancement/expansion
e. Tongue reduction
f. Lingual suspension
g. Lingual Suspension
h. Sliding genioplasty
i. Maxillary mandibular surgery
j. Tracheostomy

Treatment (CSAS)

1. Respiratory stimulants
2. Non-invasive ventilation (IPPV & CPAP via mask)
3. Tracheostomy

Sleep and anaesthesia-their nature and dffects on ventilation

Sleep is a state of rousable unconsciousness.
Electrophysiology of sleep shows non rapid eye movement (stage 1 to 4 NREM) and rapid eye movement stage (REM).

In young adults stage 1 is brief, followed by stages 2, 3, & 4. Stage 3 & 4 predominates in the first NREM period and after 70 minutes, first REM period tend to lengthen as sleep progresses while the cycle length shortens as the NERM period tend to lengthen as sleep progresses while the cycle length shortens as the NERM period decreases more than the increase in REM.

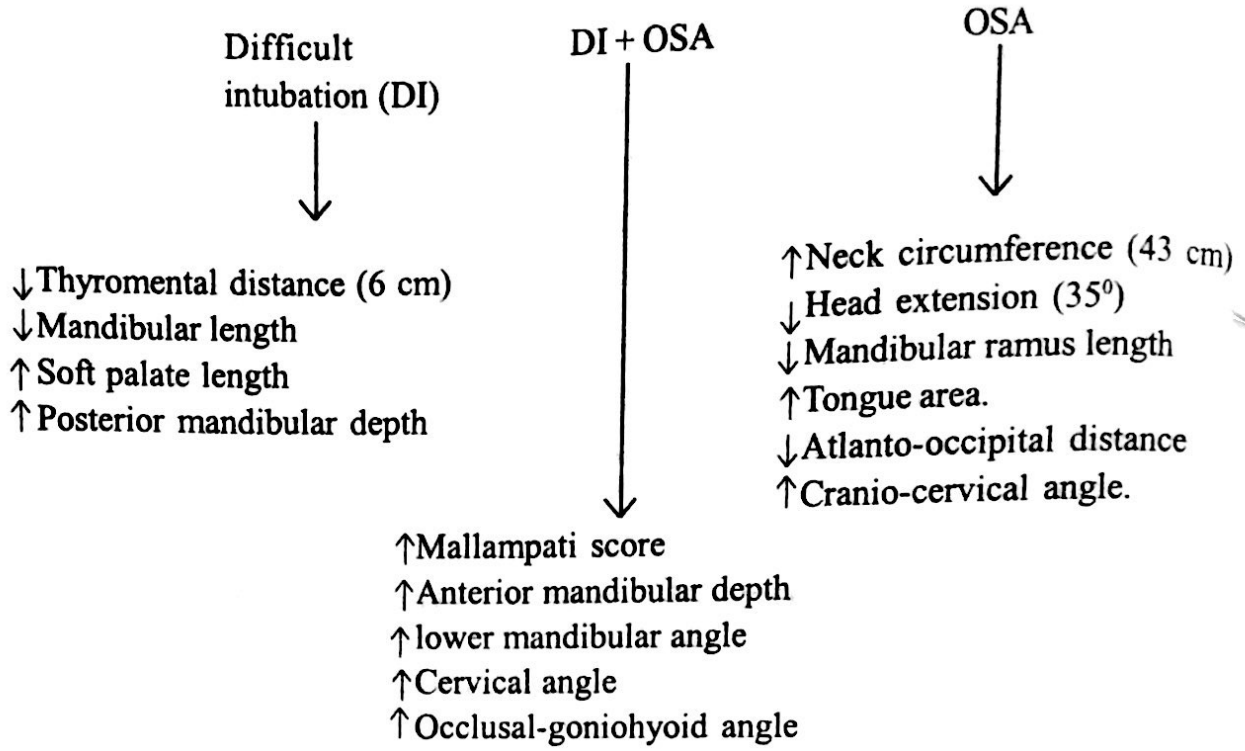
During the REM, tonic and phasic activity of chest wall and accessory muscles except diaphragm is greatly reduced. Functional residual capacity (FRC) is reduced resulting in atelectasis specially in case of obesity and chromic lung disease. During NREM, resistive or elastic respiratory load compensation is slow and incomplete with increased reliance on chemical drive which itself may be depressed leading to a degree of hypoxemia and carbon dioxide retention.

Wakefulenss has a stimulatory effect on ventilation. Sleep does unmask 'apnoeic threshold' not seen in wakefulness.

Anaesthesia is a state of unrousable unconsciousness. Most anaesthetic and sedative drugs produce a dose dependent depression of consciousness and other vital function related to respiration.

Anaesthesia and sleep disordered breathing

Unique and shared features of (DI) and OSA



Perioperative risks for Sleep Aponea

Anaesthetics sedatives and analgesics aggravate or precipitate OSA by decreasing pharyngeal tone, depressing ventilatory responses to hypoxia and hypercapnia and inhibiting arousal responses to obstruction, hypoxia and hypercapnia, resulting in varying degrees of central respiratory depression.

Surgery of thorax and upper abdomen compromises ventilatory function, potentially compounding the effects of OSA of centrally mediated hypoventilation. Upper airway surgery may cause swelling and may worsen or precipitate obstruction. The same is true when the nose is packed or a nasogastric tube is inserted. These may make nCPAP difficult. The anaesthetic management plan is determined by the severity of sleep apnoea.

a. Mild OSA: To nurse in lateral posture during the recovery.

b. Moderate to severe OSA: supervision in high dependency unit postoperatively may be required due to substantial analgesic need. Nasal CPAP may also be required. Therefore most patient benefit from regional anaesthesia with light GA.

Use of CPAP mask should be taught to the patient and nursing staff. It does not prevent aspiration. Its prophylaxis may be required. O2 is added to CPAP therapy. Nasopharyngeal and Oropharyngeal airways may aid during emergence.

Short acting neuromuscular relaxant (Vecuronium, Atracurium), opioids (Fentany) and volatile like isoflurane may be required. Non-steroidal analgesics may be more effective postoperatively than the sedatives.

Conclusion

Sleep apnoea syndrome (SAS) patents are exquisitely to all central depressant drugs, with upper airway obstruction of respiratory arrest occurring even with minimal doses. Thus sedative and opioid premedication should be omitted as should the intra and postoperative use of opiods be limited or avoided. All anaesthetic drugs should be administered by titration to desired effect, preferably using short-acting drugs. When possible nonopioid analgesics of local anesthetics should be used for postoperative analgesia. Perioperative monitoring for apnoea, desaturation and dysrhythmias is essential. SAS patients have a potentially difficult airway. Awake intubation is the safest approach to airway control. Extubation should only be tried in the fully conscious patient with intact upper airway.

References

1. Hanning, Obstructive Sleep Aponea, Br. J. Anaesth. (1989). 63
2. Boushra, Anaesthetic Management of patients with Sleep Aponea Syndrome, Canadian J. Anaesth 1996/43:6
3. Bower and Gungor, Pediatric Obstructive Sleep Apnea Syndrome, Otolarygologic Clinics of North America, Vol. 33 No 1(2000)
4. Loadsman & Hillman, Anaesthesia and Sleep Apnoea, Br. J. Anaesth 2001:86
5. Chung and Crago, Sleep Apnoea Syndrome and Anaesthesia, Can. Anaesth. Soc. J., Vol 29, No. 5(1982)
6. Davis and J. R. Stradling: Acute Effects of Obstructive Sleep Apnoea, Br. Journal of Anaesthesia 71 (1993).
7. Hiremath & Hillman et. al: Relaltionship Between Difficult Tracheal Intubation and Obstructive Sleep Apnoea: 80 (1998).
8. Warwick & Mason, Obstructive Sleep Aponea Syndrome in Children: Anaesthesia: 53 (1998)
9. Connolly, Anesthetic Management of Obstructive Sleep Aponea Patients: J. Clin. Anaethesia: 3(1991).
10. Symposium on Sleep Apnoea disorders: The Medical Clinics of North America: Vol. 69 (1985)