



American Board of Craniofacial Dental Sleep Medicine

NOTES TO REVIEW IN PREPARATION FOR THE WRITTEN EXAMINATION

The American Board of Craniofacial Dental Sleep Medicine is pleased to provide candidates with these review notes, which we have compiled in an easy-to-read, bulleted format. Although they are not intended to cover 100% of the information included in the written examination, we hope that these notes –used in combination with information and resources on our website – will enable you to optimize your study time.

- Three states of consciousness – wakefulness, REM, NREM
- EEG of wakefulness – low voltage fast frequency waves, (resembles REM sleep)
- NREM SLEEP- homeostatic regulation, blood pressure gradually decreases, prevalence of parasympathetic influence, sympathetic tone decreases (increased bursts of sympathetic activity with arousals), decline in heart rate, cardiovascular quiescence, decreased myocardial workload, thermoregulatory response intact, respiratory pattern is regular, activity in brainstem reticular formations lower than wakefulness, unfocused thought, occasional short dreams,
 - Four stages of NREM
 - I typically 1-7 minutes
 - II typically 10 – 25 minutes
 - III, IV – **Slow wave sleep** – earlier in sleep cycle (**first 1/3 of night**)
- REM Sleep – poikilostasis (impaired, suppressed, homeostatic regulation) low voltage fast frequency EEG (resembling wakefulness), blood pressure fluctuates, thermoregulatory response to ambient temperature is absent, penile erections in men, dreaming with recollection, muscles atonic (except extraocular muscles and the diaphragm), initial REM period 70 – 120 minutes after sleep onset, succeeding REM periods get longer, 4 – 6 REM periods per night is typical, alcohol, and antidepressants are REM suppressors, depression, narcolepsy, and drug withdrawal shorten REM latency, **more common last ½ of night**
 - Tonic – parasympathetic tone increases, sympathetic tone reaches its nadir,
 - Phasic – increases in **sympathetic** tone, mean arterial pressure, heart rate
- Neurotransmitters promoting wakefulness
 - Norepinephrine
 - Dopamine
 - Acetylcholine
 - Histamine (*antihistamines are sedation producing*)
 - Glutamate
- Neurotransmitters promoting **sleep**
 - Serotonin (prevents its synthesis results in insomnia)
 - Adenosine (caffeine blocks adenosine receptors)

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- Gamma-amino butyric acid (GABA) (*benzodiazepines bind to GABA receptors and enhance sleep*)
- Neurotransmitters involved in REM Sleep
 - Acetylcholine
 - Adenosine
- Acetylcholine – two receptors, muscarinic and nicotinic
 - Cholinergic antagonists – atropine and belladonna cause sedation
 - Cholinergic agonists - tobacco at nicotinic receptors enhances alertness
- Acetylcholine produced in the brainstem and basal forebrain
- Cerebrospinal factors that promote sleep – enkephalin, endorphin, dynorphin, prostaglandin, D2 and interleukin
- **Blood** factors that promote sleep – insulin, cholecystokinin
- REM-on neurons - **cholinergic** rather than nicotinic
- REM-off neurons – **norepinephrine, serotonin and histamine**
- Superchiasmatic Nucleus (SCN) – paired nuclei, located in anterior hypothalamus, dorsal to optic chiasm, modulates **circadian rhythms** and influences **secretion of melatonin** from pineal gland
- **Hypocretins** are proteins that regulate sleep and wakefulness – **narcolepsy** results from **lack** of neurons for making hypocretins
- **Hypocretins** are made in the hypothalamus (produced by 1500 cells)
- **Sleep terrors** occur first 1/3 of night (**slow wave, NREM**)
- **Nightmares** more common early morning (**REM**)
- Actigraphy is of value in insomnia, circadian rhythm disturbances, restless leg (RLS), periodic limb movement (PLMD) *not indicated as primary diagnostic tool*
- **Arousal**
 - **NREM**, 3 second or longer shift in EEG frequency preceded by minimum of 10 seconds of any stage sleep
 - **REM**, 3 second or longer EEG shift + **increase in chin EMG**
- Normal arousal level: 14 +/-2/hr in **young adults (20 – 40 years of age)**, 18+/-2/hr middle age (40 – 60), 27+/-3/hr on elderly (60+)
- Epoch – 30 second screen of data
- EEG Frequencies
 - Beta 16 – 25 Hz, is not a requirement of any sleep stage
 - Alpha 8 – 12 Hz, characteristic of drowsy wakefulness with eyes closed, typically 1-7 minutes, and disappears with the onset of sleep
 - Theta 3 – 7 Hz.....Known as saw tooth wave, **seen in REM**
 - Delta <2 Hz
- **K-complex** – slow, negative, upward, diphasic, EEG waveform commonly followed by a spindle or burst of K-alpha, feature of **Stage II** sleep characterize such **arousals** as OSA, PLM
- **Sleep spindles** – frequency of 12 – 16 Hz, generated in reticular nucleus of thalamus, defining **characteristic of Stage II** sleep, thought to decrease external stimulation to the cortex, therefore **facilitate sleep**

NORMAL/HEALTHY	young adult (20 - 30)	middle age (30 - 60)	elderly (60+)
total sleep time (TST)	430	360	368
sleep latency (min)	20		25
sleep efficiency (%)	91		80
Stage I (%)	5	3 - 8	10
Stage II (%)	50	45 - 60	65
Stage III + IV (%)	16	15 - 20	3
REM %	26	18	20
REM Sleep latency (min)	66		58

- **Depressants** such as **benzodiazepines** result in an **increase of Sleep spindles**
- Slow wave sleep
 - Stage III 20 – 50% of an epoch must be Delta
 - Stage IV >50 % of an epoch must be Delta
- Stage II sleep
 - <20% Delta
 - K complexes
 - Sleep spindles
- Slow wave sleep suppresses the occurrence of sleep-disordered breathing.
- The quantity of Stage I sleep often directly relates to **daytime alertness and the subjective feeling of refreshment from sleep.**
- Changes in sleep architecture with pregnancy:
 - Increase TST
 - Increase daytime sleepiness
 - Increase nocturnal awakenings
 - Decrease in sleep quality
 - Decrease in SWS
 - Reduced REM in third trimester
- Changes in sleep architecture with menopause:
 - Increased prevalence of SAHS
- Alternative terms for REM Sleep: *paradoxical sleep, D-sleep, dreaming sleep*
- REM sleep generated in **pons and midbrain**
- Shortened REM latency <15 minutes
- Factors resulting in low REM Sleep
 - Fragmentation effect of OSA, RBD, nightmares
 - REM sleep suppressing medications: **amphetamines, barbiturates, MAO inhibitors, anticholinergics, alcohol, tricyclic antidepressants (withdrawal shortens REM latency)**
 - Age
 - Sleep/wake schedule disorder
 - Environmental factors: noise light, uncomfortable mattress,
 - REM rebound (= shortened REM latency)
- Factors resulting in high number of arousals:
 - Sleep disorders: *OSA, snoring, PLM, restless leg*

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- Drug withdrawal
- Depression
- Ageing
- Environmental disruption
- Alpha/Delta – alpha intermixed with delta waves, seen in such disorders as fibromyositis, chronic fatigue syndrome, and some psychiatric disorders
- Severity of OSA should be based on two components: the PSG and the complaint of daytime sleepiness
- AHI at or greater than 30 carries significant high risk of hypertension
- Severity of OSA:
 - Mild – AHI 5 -15/hr
 - Moderate – 15 – 30 AHI/hr
 - Severe - > 30 AHI/hr
- Apnea is likely to be most severe during REM and in supine position
- The highest AHI/RDI is not present during REM-sleep because REM-related apneas and hypopneas have a longer durations
- Saw tooth waves are seen during REM sleep, have theta frequency and are seen more prominently in central leads
- OSA – effort, no airflow (<80% of normal air flow)
- Central apnea – no effort, no airflow
- Hypopnea – effort, reduced air flow (20 – 80% reduction), accompanied by 4% desat or EEG arousal
- Mixed Apnea – start out as a central, followed by an OSA. The effort begins against an obstructed airway
- Prevalence of snoring: 32% men, 24%women (becomes equal post-menopause)
- SAHS adult signs and symptoms: low soft palate, large tongue, enlarged tonsils, iatrogenic micrognathia, retrognathia mouthbreathing, snoring, snorting, choking, gasping, witnessed apneas, morning headaches, impotence, nocturia, daytime sleepiness
- SAHS presenting signs and symptoms in children: retrognathia, large tonsils, mouthbreathing, long face, hyperactivity and poor school performance
- CPAP compliance defined as at least four hours use every night
- Oral appliances success defined as either
 - 50% reduction in AHI (54 – 81% success rate reported)
 - AHI less than 10 (51 – 64% success rate reported)
- Patients with systolic heart failure have increased prevalence of OSA and central apnea
- Two main treatment regimens for insomnia:
 - Pharmacologic
 - CBT (cognitive behavior therapy) sleep restriction therapy stimulus control therapy, progressive muscle relaxation
- Tetrad of narcolepsy:
 - Hypersomnolence
 - Sleep paralysis
 - Cataplexy (attacks of muscle weakness triggered by emotional stress i.e. laughter is the most common)

- Hypnagogic (pertaining to drowsiness) or hypnopompic hallucinations (persist after awakening)
- MSLT provides evidence for diagnosis of narcolepsy – mean sleep latency of less than 5 minutes in 2 or more sleep onset REM periods during the MSLT
- Medications used to treat daytime sleepiness in narcolepsy: modafanil, methylphenidate and dextroamphetamine
- Primary RLS (restless leg syndrome) dysfunction of the dopaminergic system. Supplementing dopamine with levodopa or dopaminergic agonists often improve the symptoms
- Iron supplementation in patients with RLS and ferritin levels less than 50mg/dl can improve symptoms
- Causes of secondary RLS: renal disease, iron deficiency anemia, pregnancy, peripheral neuropathy, antidepressant medication, and agents blocking dopamine receptors
- Neurodegenerative disorders (synucleinopathies) have a high association with RBDs i.e. Parkinson's, Lewy body disease, multiple system atrophy
- RBD typically occurs in the last ½ of the night when majority of REM sleep occurs
- RBD can be effectively treated with clonazepam (a benzodiazepine) in the majority of patients
- REM sleep is thought to be protective against seizure activity, due to mixed frequency and lack of synchronization
- Slow wave sleep is thought to promote seizure due to its high voltage and synchronization
- Side effects of dopaminergic meds: vivid dreaming, hallucinations, and insomnia
- Benzodiazepines shorten sleep latency, reduce arousals and awakenings and increase total sleep time
- PSG findings associated with benzodiazepines excessive spindling on EEG and suppressed slow wave sleep
- Acute alcohol ingestion: increased slow wave sleep. Once metabolized, REM rebound and sleep fragmentation
- Risk factors for OSA in children: enlarged tonsils and adenoids, obesity, craniofacial abnormalities, allergies, abnormal upper airway tone associated with neurological disorders
- Newborns typically spend 16 hours/day sleeping, ½ in REM
- Prone sleeping position for infants increases risk for SIDS
- Primary enuresis: child has never had a dry period for more than 3 months
- Secondary enuresis: redevelops in a child who was previously dry for 3 months
- Core body temperature reaches its nadir 1.5 – 2 hours prior to habitual morning arising. Melatonin reaches its maximum just before this time
- Functions of sleep: memory consolidation, energy conservation, brain restoration, brain and body growth, immune function regulation
- Ultradian processes repeat with a periodicity of less than 24 hours
- Infradian processes multiday periodicity i.e. menstrual cycle
- Properties of circadian rhythms:
 - Persist without environmental cues
 - Innate and not learned
 - Invariant over a wide range of temperatures
 - Can be retrained by external cues (light/dark cycle)

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- Zeitgebers - environmental variables that are capable of entraining circadian rhythm
- Amplitude is peak to trough difference in a rhythm
- Phase is a particular point in the cycle
- **Acrophase** is the maximum amplitude of a rhythm
- Nadir is the minimum amplitude of a rhythm
- **Slow wave** activity is driven by **homeostatic** factors, most prominent **first 1/3 of night**
- Spindle activity and REM sleep are driven by circadian factors
- **Adenosine** is the neurobiologic substrate underlying the **homeostatic** sleep need
- Adenosine acts as a direct negative feedback inhibitor of neuronal activity
- Integration of **autonomic function in sleep – NTS** - nucleus tractus solitarius
- Parasympathetic tone predominates during NREM
- Tonic REM mediated by parasympathetic tone
- Phasic REM is the result of sympathetic tone
- Pupillary **constriction** occurs during NREM sleep because of parasympathetic innervation
- Parasympathetic tone increases and sympathetic tone is decreased in NREM sleep
- In arousals sympathetic tone increases
- Pupillary **dilation** occurs during REM due to inhibition of parasympathetic output
- During tonic REM or NREM sleep parasympathetic tone is increased, sympathetic decreases
- During phasic REM, sympathetic tone transiently increases to above waking levels – leads to vasoconstriction in skeletal muscle vessels.
- Muscle tone maximum during wakefulness, decreases during NREM, decreases or is totally absent during REM.
- Atonia of REM modulated by **perilocus coeruleus**
- Upper airway dilator muscles reduced in NREM, reduced even more during REM – lead to upper airway narrowing and upper airway resistance.
- Upper airway muscles involved in reduction of airway diameter – genioglossus, palatoglossus, tensor veli palatine, levator veli palatine, hyoid muscles.
- pCO₂ increases acutely at sleep onset by 2 – 8 mmHg
- Arterial pO₂ typically decreases by less than 2% during sleep
- Slowing of metabolism during sleep results in decrease in O₂ consumption and decrease in CO₂ production.
- Hypoxic ventilatory response decreases during NREM and decreases even further during REM
- Functional residual capacity declines by about 10% during sleep or **just the supine position**
- Cough reflex decreases during sleep
- Hypoxemia is a poor stimulus for arousal
- The combination of hypoxemia and hypercapnia provides the strongest stimulation for arousal
- Heart rate decreases during NREM (due to increased parasympathetic activity) , fluctuates during REM
- Cardiac output falls during both REM and NREM progressively throughout the night
- Arterial blood pressure falls by approximately 10% during NREM – fluctuates significantly during REM, probably due to sympathetic activity
- Cerebral blood flow falls 5 -23% during NREM, increases up to 41% during REM
- NREM is thought to be resting or inactive sleep

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- REM is thought to be a highly active neurologic state
- Testosterone levels rise during sleep
- During sleep there is inhibition of secretion of thyroid stimulating hormone (TSH)
- Cortisol secretion is independent of sleep
- Growth hormone is most closely associated with slow wave sleep
- Serotonin is a precursor to melatonin
- Melatonin release peaks during sleep – length of secretion proportional to the length of sleep
- Gastric acid output is lowest in the morning hours
- Swallowing is significantly reduced during sleep – 25 swallows / hour during the day, 5 /hour during sleep
- Sleep is thought to be generated in anterior hypothalamus and preoptic areas. There is a complex neurologic pathway from brainstem to cortex to generate sleep
- Reticular activating system (RAS) in brainstem projects via the hypothalamus, thalamocortical pathways to the forebrain; system is necessary for maintenance of wakefulness
- Four main effects of medications on sleep
 - Insomnia
 - Daytime hypersomnolence
 - Suppression of REM
 - Suppression of slow wave sleep
- REM – phasic eye movement and muscle atonia on EMG, saw tooth waves and theta frequency on EEG
- Phasic REM – sympathetic, irregular heartbeat breathing and BP spikes
- Tonic REM – parasympathetic, atonia of major muscles
- Shortened REM latency – depression, narcolepsy, REM suppressing meds such as antidepressants or alcohol
- Slow wave sleep more predominant in first 1/3 of the night
- REM more dominant last 1/2 of night
- REM periods progressively increase in length and phasic activity, 4-6 REM periods per night
- Adult Stage I, 3 – 8%
- Adult stage II 45 – 55%
- Adult Stages III & IV 15 – 20%

- Berlin Questionnaire – four categories: persistent snoring, daytime sleepiness, hypertension, body mass index. Predict AHI >5 sensitivity of 86%, specificity 77%, positive predictive value >.89

- Pharyngeal dilator muscles have increased daytime activity in patients with SAHS compared to controls, but this activity diminishes after sleep onset.
- Neuromuscular stimulation applied intraorally to the hypoglossal nerve improves upper airway patency by increasing tonus of upper airway muscles – genioglossus, levator veli palatine, tensor veli palatine, palatoglossus, palatopharyngeus, and superior pharyngeal constrictors
- Upper airway in SAHS patients is narrowed in the lateral dimension, essentially normal in the antero-posterior dimension.
- Classification of severity of OSA:

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- ≤ Normal
- 5-15 Mild
- 15 – 30 Moderate
- >30 Severe
- Protryptiline has been shown to moderately reduce AHI and may be used in OSA that is REM related.
- The physiologic effect of CPAP is as a pneumatic splint
- Low O₂ saturation despite correction of OSA implies underlying cardiac, pulmonary or neuromuscular disease ABCDSM Craniofacial Dental Sleep Medicine Credentialing
- CPAP at higher levels increases intrathoracic pressure and has the potential to induce hypotension with decreased cardiac output. Rare effect however.
- Minimum usage time per night considered **compliant** is 4 hours.
- Studies have shown comparable compliance rates between CPAP and BiPAP.
- BiPAP lowers air flow pressure during exhalation – too low of a pressure may induce continued airway collapse
- Oral appliances – results depend on definition of success
 - 54 – 81% reduce AHI by 50%
 - 51 – 64% reduce AHI to ≤10
- Predictors of success using oral appliances:
 - Younger age
 - Lower BMI
 - Positional OSA
 - Increased protrusion by appliance
- Presence of a bed partner doubles the reported rate of snoring.
- Most common cause of Central Sleep Apnea: congestive heart failure. Others: Neurologic disorders, Medications, Pregnancy
- Less than 5 CSAs is generally considered normal
- In wakefulness compensatory responses exist for the changes that cause OSA. These mechanisms are lost in sleep.
- Homeostatic control (closed loop) in NREM sleep allows reflexive control of somatic and autonomic functions
- REM sleep (open loop operation) further impedes and suppresses reactions to compensatory responses. OSA should be worse
- Alterations in cardiac rhythm in REM originate in ponto-geniculooccipital activity
- Patients with heart disease and OSA are at more risk during REM sleep because of the challenge of dual control by the cardiovascular and respiratory centers (**poikilostasis**)
- REM sleep characterized by surges in sympathetic and vagal nerve activity may result in cardiac arrhythmias, myocardial ischemia or myocardial infarction in patients with heart disease.
- OSA patients have higher sympathetic tone, that can trigger cardiac arrhythmias and myocardial infarction in individuals with heart disease.
- Autonomic stability in NREM sleep is regulated by vagal nerve
- Depressed heart rate variability is caused by loss of vagal nerve function
- OSA in children is accompanied by increased heart rate variability

- NREM sleep causes **metabolic restoration** based on autonomic stability – hypotension (caused by **reduction in sympathetic vasomotor tone**), bradycardia (caused by **increased vagal activity**), **reduction in cardiac output** and systemic vascular resistance.
- Disruption of integrated control of peripheral vascular beds is unique to REM sleep and puts chronic heart failure patients at greater risk.
- In REM sleep, the loss of excitation mediated by serotonin and norepinephrine contributes to the hypotonia of motoneurons that innervate genioglossus and other upper airway muscles.
- The decrease in medullary respiratory neuronal activity in sleep is **not** because of a loss of state-dependent tonic excitatory inputs. The neuronal activity is not lost, it becomes subthreshold.
- **Sleep affects primarily neurons that receive large amounts of nonrespiratory inputs**
- Stimulation of the **reticular formation** preferentially facilitates the activity of the **muscles of the upper airway** rather than the muscles of the diaphragm
- From wakefulness to NREM sleep the upper airway muscles lose their tonic excitatory impulses to a greater extent than the diaphragm.
- Upper airway motor neurons are more sensitive than diaphragm motor neurons
 - To depressive effects of
 - Alcohol
 - Diazepam
 - Barbiturates
 - Hypocapnia
 - To the stimulative effects of
 - Protryptiline
 - Cyanide
 - Strychnine
- In patients with OSA the obstructive episodes are longest and SpO₂ desaturations are the most severe during REM sleep
- Cells throughout the nervous system are more active in REM than NREM
- **In REM, suppression of the activity of hypoglossal motor neurons results primarily from withdrawal of the excitation mediated by serotonin and norepinephrine**
- During sleep there is a loss of voluntary control and a decrease in the usual ventilatory response to both low oxygen and high carbon dioxide levels
- **Both the hypoxemic and hypercapnic responses are most depressed in REM**
- The major goal of the respiratory control system is homeostasis
- Respiratory muscles **do not** have a built-in pacemaker.
- Medulla is the respiratory center – it responds to three types of information CHEMICAL, MECHANICAL, BEHAVIORAL
- Carotid body senses PaO₂. Glossopharyngeal (IX) sends impulses to the medulla CHEMICAL
- CO₂ is sensed in carotid body *and central chemoreceptor in the medulla* CHEMICAL
- Drugs that depress CNS function may profoundly depress the chemical drive to breathe
- MECHANICAL – receptors in the lung that respond to **irritation, inflation, deflation and congestion of blood vessels** info carried on Vagus (X)
- BEHAVIORAL – respiratory system linked to functions other than breathing – singing laughing crying, speaking – controlled by higher brain centers, **linked only to wakefulness**

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- NREM hypoxia greater in men than in women
- IN OSA there is a defective compensation for upper airway resistance in sleep; ventilatory mechanics are not impaired in NREM
- In REM, hypoxemia results from hypoventilation
- The upper airway includes:
 - Extrathoracic trachea
 - Larynx
 - Pharynx
 - Nose
- The upper airway is a common pathway for digestion, phonation and respiration
- Structures surrounding the pharyngeal airway:
 - Close the airway during deglutition
 - Valve and shape the airway during phonation
 - Conduit for airflow during respiration
- Pharynx is the only collapsible segment of the respiratory tract
- Retroglossal region – caudal margin of the soft palate to the base of the epiglottis
- Hypopharynx – base of the tongue to the larynx
- Majority of OSA patients manifest upper airway closure or narrowing during sleep in the retropalatal and retroglossal regions
- The anterior wall of the oropharynx is formed by the soft palate and tongue
- The posterior wall of the oropharynx is comprised of the superior, middle and inferior constrictor muscles
- During nasal breathing the tongue is in apposition with the tongue
- Oral breathing with the mouth open and tongue in the floor of the mouth destabilizes the airway by allowing the soft palate to move posteriorly
- Posterior movement of the genu of the mandible with mouth opening causes the tongue and hyoid apparatus to move posteriorly and thereby narrow the pharynx
- Neck flexion has the same effect without a change in the maxilla/mandible relationship
- Increases in nasal resistance produce greater pharyngeal intraluminal pressure and reduced pharyngeal cross-sectional area
- Narrowing at the retropalatal region is associated with a further decline in intraluminal pressure during inspiration

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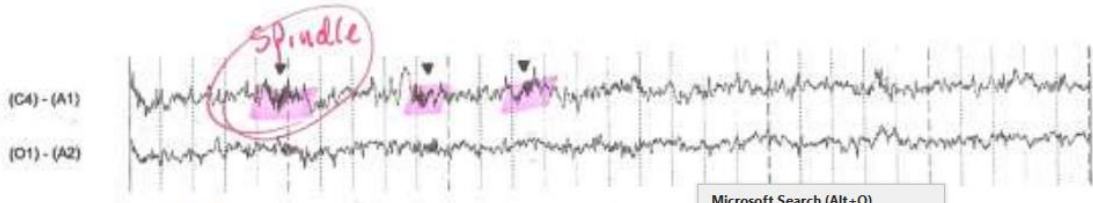


Figure 8. Typical spindle activity, designated by arrows.

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Figure 9. Typical alpha activity. Note that the activity is more prominent in the occipital lead than in the central lead.



Figure 10. Typical saw-tooth waves as seen during REM sleep. Note that they are more prominent in the central lead than in the occipital lead.

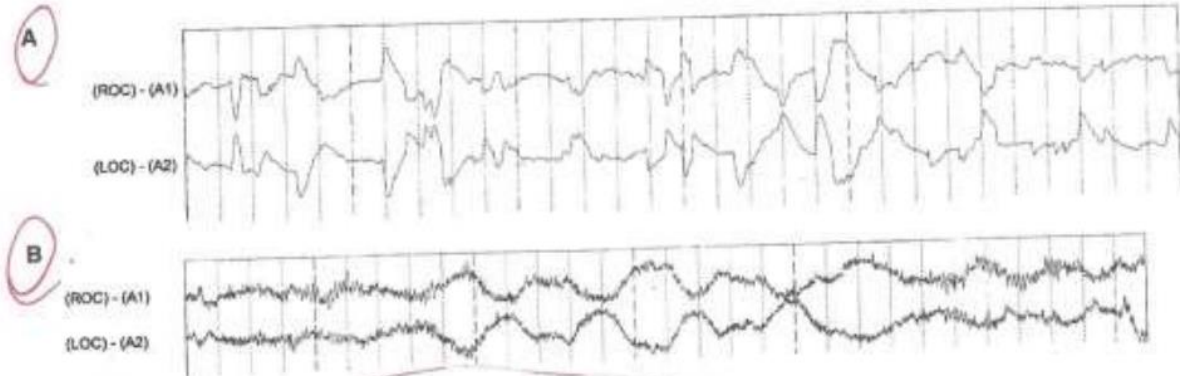


Figure 6. A. Typical rapid eye movements (REMs). B. Slow, rolling eye movements.

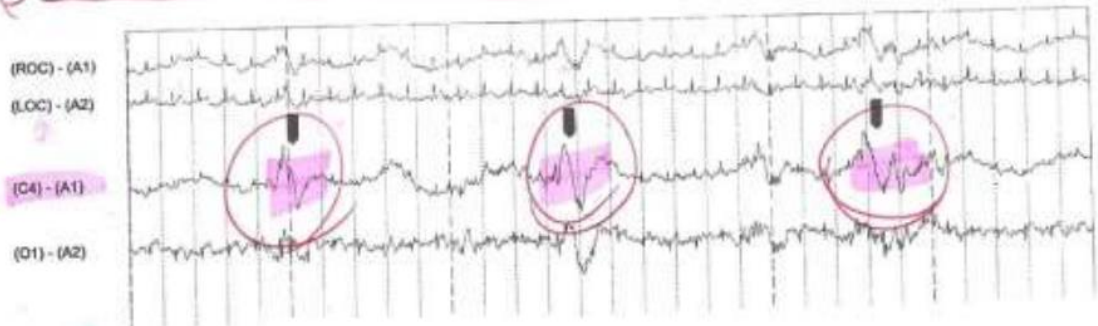


Figure 7. Typical K complexes, designated by arrows.

- Increase in Class II fast-twitch fibers (more susceptible to fatigue than Type I fibers in normals) in Genioglossus of apneic patients
- Myopathy of genioglossus is a secondary phenomenon in apneics – reversible with CPAP
- MADs increase airway size more in the retroglottal area than in the retropalatal because they pull mandible and tongue forward
- In awake patients with OSA supraglottic resistance is elevated and pharyngeal lumen is somewhat narrowed.
- Loss of pharyngeal muscle activity at sleep onset induces pharyngeal narrowing
- There is a compromise in the response of the genioglossus to increases in hypercapnic stimulus in NORMALS
- ***Pharyngeal airway patency is maintained by a balance between the upper airway dilating muscles and negative intraluminal pressure***
- The main clinical significance of snoring is as a marker of UARS and OSA
- Common side effects of oral appliances – mucosal dryness (86%), tooth discomfort (59%), excessive salivation (55%), jaw pain (41%) – all described as minor
- Increased protrusion is a predictor of success of oral appliances