BREATHING DURING SLEEP IN NORMAL SUBJECTS

It is important to know normal breathing patterns during sleep to appreciate further abnormal breathing during sleep such as sleep apnoea or any other type of breathing. Many factors such as increasing age male sex, excess weight and snoring (which are considered normal by non medicals or even by medicals that are not exposed to study of sleep physiology) may influence the normal breathing.

Breathing during NREM Sleep

The oscillations in breathing amplitude or periodic breathing at the onset of sleep have been reported. The duration of periodic breathing at sleep onset lasts from 10 to 20 min or even unto 60 minutes. Periodic breathing persists as long as sleep oscillates between arousal and stage 1 or 2. It disappears when stable stage 2 or deeper sleep is reached. Periodic breathing at sleep onset may also be associated with sleep apnoea of 10 second to 40 seconds duration. This apnoea is of central in origin. Breathing instability at sleep onset is explained on the basis of two factors:

a) The set point of regulation of ventilation is different in wakefulness and sleep, with a higher PCO_2 level and lower ventilation during sleep.

b) Sleep onset is not immediate but rather oscillate between arousal, stage 1 and stage 2 sleep before stable stage 2 or deep sleep (SWS) is reached.

When person is trying to fall sleep, it results in decreased ventilation and higher PC02 adjusted to the sleep point. On subsequent arousal, the increased PCO_2 above the wakefulness set point constitute an error signal that provokes hyperventilation until the wakefulness set point is reached. When the subject subsequently falls asleep, the level of PC02 will in turn be below the sleep set point, with resultant hypoventilation and so forth till stable sleep is reached. Sleep instability and intrinsic control mechanisms together create a vicious cycle of breathing instability at sleep onset. Stable sleep can reached only if this vicious cycle is somehow broken. It is likely that dampening systems more likely neurogenic than chemical in nature have a major role allowing ventilation and sleep to reach a steady state.

Thus, breathing at sleep onset is physiologically unstable, but the magnitude of the instability is variable ranging from small fluctuations in breathing amplitude to severe periodic breathing with repeated apnoeas. During stable NREM

sleep, ventilation is lower. Minute ventilation is low though there is increase in PCO_2 with deeper stages of sleep, which indicates that decrease in minute ventilation during NREM sleep is linked to active sleep mechanism. Mean inspiratory flow is decreased but there is no consistent change in inspiratory duration and cycle duration/ result is decreased tidal volume.

Other changes seen during sleep are as follows:

a) Increased respiratory EMG activity of intercostals muscles.

b) Increase in upper airway resistance two fold during NREM sleep. The commonest site is palatal or hypopharyngeal. Respiratory muscle activity is of two types: – Phasic Type : which occur periodically in phase with a component of the respiratory cycle generally inspiration, other type is tonic type which is background activity on which phasic activity is superimposed. Regulation of phasic activity is clearly related to respiratory regulation whereas tonic activity is independent of respiratory regulation and more related to general muscle tone. Changes in tonic activity are more consistent during sleep or sleep apnoea. It is decreased in the tensor veli palatine (cause fro increase in nasopharyngeal resistance), genioglossus, geniohyoid and posterior cricoartytenoid. Phasic activity is seen to increased in genioglossus and decreased in the posterior cricoaryenoid and no change in geniohyoid. There is continual discrete decrease in the EMG activity of laryngeal muscles.

c) Decrease in thoracic lung volume.

d) Alveolar ventilation decreases and PC02 increase by 3 to 7 mmHg during NREM sleep and fall in SaO_2 by 2 to 3%. This occurs in spite of decrease in metabolic rate by 10 to 20% and thus reduction in O_2 consumption and CO_2 production.

e) Pulmonary artery pressure both systolic and diastolic increases by 4 to 5% during NREM sleep.

Breathing during REM Sleep

Breathing during REM sleep is irregular, with sudden changes in amplitude and rate, at times interrupted with by central apnoea of 10 to 30 seconds. This breathing pattern is linked to burst of rapid eye movements and produced by the behavioural respiratory control system by REM sleep processes. Chemical regulation of ventilation is

not abolished in REM sleep. Most of the studies conclude that minute ventilation, tidal volume and respiratory rate during REM sleep differ little from those observed in NREM sleep.

In contrast to the increased rib cage contribution to the breathing during NREM sleep, rib cage contribution during REM sleep is found to be decreased owing to marked reduction in intercostals muscle activity. This occurs due to REM related supraspinal inhibition of alpha motor neuron drive and the specific depression of fusimotor function. This is the reason diaphragm is spared during REM sleep as this muscles fusiform innervations is low.

Upper airway resistance is expected to be increased during REM sleep because of the muscle atonia but most of the workers have found it same as during NREM sleep. On average, SaO_2 values have been found to be lower during REM sleep and end tidal PCO_2 does not yield and approximation of alveolar PCO_2 because end expiratory plateaus are seldom reached during the rapid, shallow breathing pattern characteristic of REM sleep. Cause of hypoxaemia during REM sleep could be due to reduced functional residual capacity, which leads to airway closure, or simply due to hypoventilation. The breathing pattern during sleep can be summarized as follows:

Breathing Pattern	**Type of Sleep**
Periodic Breathing	Unsteady NREM sleep
Regular	Steady NREM sleep
Mostly regular	Tonic REM sleep
Irregular, central apnoea common	Phasic REM sleep

Respiratory related events during sleep like sighs occur in all sleep stages and snoring may be seen as normal in many of the subjects. Snoring may be considered to be part of a continuum including normal, silent, unobstructed breathing, occasional snoring, and habitual snoring with occasional obstructed sleep apnoea, an overt obstructive sleep apnoea syndrome.

Some factors like sex where more desaturation seen in males than in females, age where apnoea of both type central and obstructive are commonly seen with apparently no effect on health, similarly during pregnancy due to reduced functional residual activity, one would expect more hypoxemia, apnoea but seen less frequently due to respiratory stimulant effects of progesterone.

Sleep is associated with define changes in respiration function in normal humans. Whether these changes serve a specific function remains unclear and will remain till the functions of sleep itself are better understood. Voluntarily controlled ventilation gives rise to a conflict when consciousness is periodically abolished and motor control is subjected to central nervous system influences specific to sleep. This conflict is incompletely solved in humans. A possible solution would be to sleep like Dolphins, alternately and independently with either cerebral hemisphere.