allow one to speculate that 1) if the rate of change of arterial oxygen is great enough during apnea in active sleep, arousal may fail to occur before electrocortical signs of cerebral hypoxia and primary apnea occur (11); and that 2) if an infant is repeatedly exposed to hypoxemia—either as a result of multiple apneic episodes or hypoxemia during sleep as a result of gas exchange abnormalities—that the arousal response to apnea might be impaired (12). Furthermore, data from our present study would allow one to speculate that if carotid chemoreceptor and/or carotid baroreceptor function is impaired, arousal may fail to occur before hypoxic cerebral depression occurs. If, in addition, there is a deficit in the gasping mechanism or if the circulation fails before the onset of gassing, death could quickly ensue.

REFERENCES

5. Henderson-Smart DJ, Read DJC 1979 Ventilatory responses to hypoxemia during sleep in the newborn. J Dev Physiol 1:195-208

Erratum