

Head retraction and respiratory disorders in infancy

Head retraction in infancy is a physical sign that is usually associated with central nervous system pathology. It is not generally recognised that head retraction can occur as a consequence of respiratory system abnormality, particularly in younger infants.

Head retraction may be seen in any respiratory condition where decreased lung compliance or airway obstruction causes increased work of breathing. Mechanisms by which head retraction can reduce work of breathing include: (1) increasing the efficiency of accessory muscles of respiration and (2) reducing resistance in large airways by: (a) 'splinting' the airway and (b) preventing upper airway obstruction.

(1) Increasing the efficiency of accessory muscles of respiration

The efficiency of accessory muscles of respiration can be maximised by fixing their origin as rigidly as possible. In the case of sternocleidomastoid and scalene muscles this means holding the mastoid process and the cervical spine rigid. This is best achieved by anchoring the head in hyperextension. The resulting muscle action expands the rib cage along its anteroposterior and transverse diameters.¹ The efficacy of this mechanism can be demonstrated in older ventilator dependent quadriplegic children where 'neck' breathing can be life saving.² Maximising the efficiency of these accessory muscles becomes essential in infants with respiratory fatigue in order to help intercostal muscles stabilise and expand a pliable chest wall.³

Therefore head retraction may be seen in primary or secondary lung disease—for example, pulmonary infection, aspiration syndromes, chronic lung disease of prematurity, or pulmonary oedema. We have recently observed a premature infant whose appreciable head retraction coincided with the onset of florid signs of cardiac decompensation secondary to a patent ductus arteriosus. Head retraction dramatically improved after duct ligation.

(2) Reducing resistance in large airways

The trachea and main bronchi in neonates are readily collapsible⁴ probably because of deficient cartilagenous support. Extension of the neck by stretching these floppy airways provides a splinting action allowing a more even airflow, lower airway resistance, and decreased work of breathing.

Upper airway obstruction causing apnoea in the preterm infant would be expected to occur in association with the muscle hypotonia of active (rapid eye movement) sleep; recent studies however, do not support a particular sleep phase.⁵ Inhibition of muscle tone interferes with the vital role of the genioglossus and geniohyoid muscles in maintaining upper airway patency.⁶ Extension of the head and neck increases resting muscle tone thus preventing the tongue from occluding the lower pharynx.

In artificially ventilated infants, head retraction may indicate obstruction of the endotracheal tube or inadequate ventilatory support.

When assessing the significance of head retraction in infancy, one needs to consider neurological and maturational causes in addition to the respiratory ones mentioned above.

The presence of seizures, signs of raised intracranial pressure (for example, a full fontanelle) or corticospinal tract damage (for example, persistent clonus) indicate central nervous system pathology. Head retraction has been associated with meningeal inflammation, posterior fossa haemorrhage or tumours, toxic cerebral insults (for example, kernicterus and hypoxic-ischaemic encephalopathy), or cerebral irritation caused by drug withdrawal and overdose.

Clinical features include head and spine extension and a general increase in muscle tone, adduction and internal rotation of the shoulders, as well as fisting and flexion of the wrist. Elbows may be either flexed or extended, usually the former. Hips and knees are extended and the feet flexed.

This clinical picture of hyperextension is thought to arise from the unopposed action of the medial subcorticospinal pathway. Corticospinal tract damage interferes with its normal inhibitory action of flexion and adduction.⁷

An additional consideration in preterm infants is the 'normal' extensor posture and tone, which results from differential myelination. This process is complete at 34 weeks in the medial subcorticospinal pathway allowing its extensor action to predominate until inhibitory pathways of the lateral subcorticospinal and corticospinal tracts are fully myelinated at term or even later.⁷

Differentiating between respiratory, neurological, and maturational causes of head retraction will therefore not always be straightforward, especially in artificially ventilated infants. Careful physical examination and selective investigation will indicate the correct aetiology.

We thank Dr A Bye and Professor DJ Henderson-Smith for their helpful comments and L Webster for help in preparing the manuscript.

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