Osler, to which reference will be made later. Of trophic and spasmodic affections I wish only to speak for a moment.

Certain pneumonias have been observed to follow section of, or destruction of the vagus by tumor or other cause, and were supposed to be trophic in character, the vagus possibly containing trophic fibres; although by others they are thought to have been inspiratory pneumonias, from laryngeal paralysis.

The spasmodic affections are evident neuroses as seen in hysteria and epilepsy. The ordinary spasmodic croup, while there is a primary catarrhal irritation as the determining factor, is reflex, relieved usually by emesis or rectal flushing. Laryngismus stridulus, while commonly associated with rickets, is in no way a part of the disease, but dependent upon the mal-nutrition and irritability of the nerve centres, found in this and similar conditions; and all such spasmodic affections are simply localized manifestations of the condition of nerve instability which might result in general convulsive seizures in each individual case.

Respiratory *rhythmic* affections are essentially neurotic. The Cheyne-Stokes breathing in its three varieties is the most striking, and whether occurring in cardiac disease, uremia, cerebral disease, or meningitis, or as it occasionally does in the acute infectious diseases especially in children, is a central nerve affection, and due to the lessened excitability of the respiratory centre from toxemia or from pressure, the accelerated breathing being due to the over-stimulating qualities of the asphyxial blood; the centre maintaining its rhythmic function, only acting in a larger rhythm, working under altered conditions (Gowers).

The significance of the phenomenon is of more fatal import in cerebral lesions than in toxic ones.

A slow respiration is sometimes seen in pneumonia as in other toxic conditions, where toxemia is profound, from paralysis of the respiratory centre.

I well remember the case of a little child suffering from a sub-acute ileocolitis, who, although not apparently ill enough to cause her parents alarm, showed respiratory difficulty coming on in the early morning, during sleep. She was noticed to be breathing slowly and with visible effort, slight dilation of the alae nasi and slight heaving of the shoulders. She played about the nursery that day and made no complaint, but the respiratory difficulty persisted. The following day she went into collapse and died in the early morning of the third day after the onset of the respiratory distress. Physical examination of the nasal passages, the larynx and the lungs was negative. This was, I believe, due to the over-powering of the respiratory centre from acute ptomaine poisoning.

On the other hand we often see in children suffering from