Can stroke localisation be used to map out the neural network for yawning behaviour?

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What are the neuroanatomical structures involved in repetitive yawning during stroke?

Since the 19th century, cases of pathological yawning have occasionally been published in medical journals. In this issue of *J Neurol Neurosurg Psychiatry*, Singer *et al*¹ present the first study to focus specifically on yawning during acute stroke affecting the middle cerebral artery territory (*see page 1253*).

None of seven patients suffering from abnormal repetitive yawning had diencephalic lesions. Classically, yawning is thought to originate in archaic brain structures common to all vertebrates. It appears to be a powerful muscular stretch which recruits specific control systems, particularly the paraventricular nucleus of the hypothalamus, locus coeruleus and reticular activating system; these structures explain its ability to increase arousal. A persistent vestige of the past, yawning has survived evolution with little variation.² Singer *et al*¹ suggest that neocortical brain areas have an inhibitory effect on the paraventricular nucleus of the hypothalamus, and that in certain middle cerebral artery strokes this region is liberated, provoking repetitive yawning. This hypothesis merits discussion.

According to Lapresle,³ palatal myoclonus is the human homologue of a primitive respiratory reflex in gill breathing vertebrates, submerged but not lost, reappearing when the inhibitory system is damaged by lesions to the dentato-olivary pathway.

We coined the term "parakinesia brachialis oscitans"⁴ to describe cases of hemiplegia where the onset of yawning coincides with involuntary raising of the paralysed arm. We argued that a lesion in the internal capsule affecting an inhibitory pathway liberates certain subcortical structures that coordinate the massive inspiration of yawning and the motor control associated with quadrupedal locomotion.

In these examples, loss of cortical inhibition following stroke releases a hidden function, phylogenetically more primitive. Singer *et al*¹ do not provide evidence of such an event.

Face scratching, nose-face rubbing, yawning and sighs are automatisms frequently reported after epileptic seizures. These behaviours are also considered a characteristic pattern in healthy subjects on waking. Movement speed

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and repetition are the factors that vary, based on whether the context is physiological (sleep, arousal) or pathological (epileptic seizure, stroke). These behaviours are related to the brainstem and diencephalic activation that occurs when the cortex is disconnected from these areas (where the "central pattern generators" are located) by an epileptic discharge or a stroke.⁵

Adaptive behaviours depend on interactions between neural networks at various levels, requiring continuous mutual feedback. Yawning is an exterior manifestation of the tonic stimulation of the cortex by subcortical structures, particularly when the brainstem does not receive appropriate feedback from the cortex.

I agree with the conclusion reached by Singer *et al*¹: further studies are necessary to determine the exact neuroanatomical structures involved in repetitive yawning during stroke and the pathophysiological role of this behaviour.

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