Is Yawing an Arousal Defense Reflex ?

The Journal of Psychology, 123(6), 609-621
published by HELDREF PUBLICATIONS,
4000 Albernarle Street, N.W., Washington, D.C. 20016.

J. J. M. ASKENASY
Sleep Research Institute; Sheba Medical Center, Israel

Requests for reprints should be sent to J. J. M. Askenasy, Sleep Research Institute, Sheba Medical Center, Tel-Hashomer 52621, Israel.

Yawning consists of an involuntary wide opening of the mouth, with maximal widening of the jaw, and long and deep air inhalation through the mouth and nose followed by a slow expiration. As a result, an increased amount of air is inspired in the lungs (Figure 1). The average duration of the yawn is 5 s, (range, 3 to 45 s).

Yawning may be associated with tears, shivering, obstruction of the eustachian canal (causing a reduction in audiologic acuity), or stretching of the limbs. During monotonous work, yawning may be preceded by looking about, changing position, or arranging one's hair. Female infants yawn more than male infants (Feldman, Brody, & Miller, 1980), a ratio that later becomes inversed.

The earliest appearance of yawning was observed in a 15-week-old embryo (De Vries, Visser, & Prechtl, 1983). Fetal yawning appears randomly, in contrast to fetal hiccups, which appear in a frequent rhythmic pattern (De Vries, Visser, & Prechtl, 1985). In newborn babies, a yawn occurs a few minutes after the first breath and the first oral cues (Gill, White, & Anderson, 1984). At 2 months of age, babies yawn when separated from their parents. The frequency of yawning decreases with maturation, and it may be speculated that connections between the limbic system and the forebrain areas are already established (Fogel, 1980).

Etiology

The causes of yawning, as described in the medical literature, can be classified in seven groups (see Table 1). Boredom is sometimes associated with the yawning reflex (Bell, 1980). Boredom occurs; when the main source of stimulation in a person's environment no longer sustains his or her attention, by content or by form. At this moment, the mind has to make an effort to maintain contact with the environment; structures involved in this process include the prefrontal area and the limbic system. Boredom induces drowsiness by stimulating the sleep generating system through the interconnection of the prefrontal area and the limbic system with the serotoninergic raphe pontine nuclei and the ascendant activatory reticular system (AARS). When boredom is associated with fatigue, the latter potentiates the drowsiness-inducing effect (Suganami, 1977). Drowsiness is the most common stimulus of the yawning reflex.

During a yawn, massive inspiration of oxygen and the exhalation of C02 serve to reverse blood hypoxia and to increase the metabolic rate. The inhalation stretches the bronchial musculature, stimulating the vagal terminals and the cholinergic pathway, which dilate arterioles, thus lowering peripheral circulation resistance and enhancing blood flow (Friedell, 1974; Lehmann, 1979; Twiest, 1974). Gaping jaws and stretched limbs have important circulatory effects. The lateral pterygoids and the soleus muscle contain rich venous plexi within their mass, which empty with the contraction and are therefore called "peripheral hearts." Through this mechanism, venous return is enhanced (Bhangoo, 1974; Last, 1963; Siegal, 1974). The diaphragm contraction associated with the deep inspirium also enhances venous return, reviving circulation. The hyperoxygenation and increased blood circulation stimulate all the cephalic structures, including the AARS (Montagu, 1962). Monitoring jugular 02 partial pressure, cerebral blood flow, and electroencephalograms (EEGs) in thrombotic patients, Karasawa et al., (1982) found that yawning appeared when the EEG showed slow frequency and the oxygen monitoring showed decreased O2 partial pressure. Thus, boredom, drowsiness, and fatigue lead to hypoxia, reduced metabolic rate, and increased secretion of hypnogenic derivatives. Yawning is the reflex answer of the AARS, whose aim is to reverse drowsiness, maintaining the brain at the level of alertness needed for wakeful activities.
It may be hypothesized that the yawning center is a complex neuronal reflex system, located at the level of the reticular brain stem close to the AARS. The reticular neuronal yawning complex is intimately interconnected with (a) the three aggregates of respiratory neurons located in the medulla, dorsal, ventral, and parabrachialis; (b) the motor nuclei of the fifth, seventh, ninth, tenth, and twelfth cranial nerves; (c) the phrenic nerve; (d) the motor roots of the intercostal nerves; and (e) the parasympathetic terminals of the lacrimal gland.

The yawning neuronal complex enables concomitant massive inspiration, with opening of the mouth accomplished by the digastric and milohyoid muscles and retraction of the jaw achieved by the posterior fibers of the temporal muscles and the geniohyoid muscles. Yawning sometimes provokes a mass response in which stretching of the four limbs appears together with tears or shivering (Bhangoo, 1974). The following arguments can be presented in favor of the location of the yawning neuronal complex in the brainstem reticular system close to the AARS. A tetraplegic patient with a Pontine glioma was able to yawn despite the fact that he could not open his mouth at will (Geschwend, 1977). Opening the mouth through a motor cortex command was impossible, but reflexogen yawning was still possible because of the short interconnections between the reticular neurons and the motor axons of the fifth and seventh cranial nerves.

Another tetraplegie and "locked-in" syndrome patient, suffering from an anterior pons glioma, could yawn but couldn't open his mouth at will (Bauer, Gerstenbrand, & Hengl, 1980). The preservation of the reticular complex center and of the AARS in this patient made yawning possible. Yawning in a schizophrenic patient was observed to be related to increased vigilance (Beckmann & Zimmer, 1981). Yawning persists in the vegetative state with spontaneous breathing. In these patients, the reticular system and AARS are preserved (Braunwald et al., 1987). Jurko and Andy (1982) suggested that the persistence of yawning following thalamotomy is due to the preservation of the hindbrainstern reticular system.

Pimozide, a neuroleptic, abolished valproate-induced yawning through its inhibitory effect on the AARS (Rollinson & Gilligan, 1979). Bombardment of the AARS by transcutaneous application of a high frequency electric current of 300 mA for 30 min decreased yawning significantly in 11 heroin addicts aged 18 to 31 by slowing down the threshold of the defense reflex (Daulouede, 1980).

The yawning reticular neuronal complex bas close connections with a large number of subcortical and cortical structures (Figure 2). The link with the hypothalamo-hypophysary formation is suggested by the capacity of ACTH and MSH (see Table 1) to provoke yawning and by the sensitivity of this area to sleep-promoting factors and drowsiness-inducing states. The specificity of boredom as a stimulus of yawning shows the close interrelationship between the limbic system and the brain stem areas. A conversation or a bad movie or book are potent yawn releasing stimuli, showing the role of the cortex, especially the occipital and associative areas. Auditory perceptions of a monotonous voice, music, and periodic noise are potent yawn releasing stimuli, showing temporal area involvement in yawning. Thinking about yawning is a potent stimulus, which reaches the motor cortex through the prefrontal associative area and affects the brain stem yawning center, thus provoking the reflex (Provine & Hamemik, 1986; Small, 1977). The infectiousness of yawning among subjects who are not bored, drowsy, or fatigued proves the existence of efférent connections between the motor cortex and the brain stem reticular center.

The Biochemistry of Yawning

Yawning was suppressed by the opiate antagonist naloxone but was provoked by administration of naloxone in drug addicted patients and in normal cigarette smokers (Flechter, Cohen, Borenstein, Regev, & Vardi, 1982; NemethCoslett & Griffiths, 1986; Paroli, Nencini, & Moscucci, 1984). The hypothalamic peptides ACTH and MSH, given intracranially, induced a peculiar stretching and yawning syndrome associated with penile erection and ejaculation (Donovan, 1978). Despite the fact that the intimate mechanism, of the endorphines in yawning remains unknown, there are several empirical observations of endorphine involvement in the yawning reflex (Donovan, 1978; Fletcher et al., 1982; Nemeth-Coslett & Griffiths, 1986; Paroli et al., 1984).

Pimozide, a haloperidol-like dopaminergic antagonist, abolished valproate-induced yawning (Rollinson & Gilligan, 1979). This observation, together with die observation that individuals suffering from, Parkinson's disease yawn less, suggests that dopaminergic receptors may be involved in the complex yawning reflex.

Experiments on rats have shown that increased serotoninergic activity induces yawning (Holmgren, Urba-Holmgren, & Rodriguez, 1980). Bombardment with hypnagogic derivatives of the pontine serotoninergic raphe nuclei induces yawning before the onset of sleep. Serotonin is considered to be a positive modulator of the cholinergic activity that sets off the yawning reflex (Holmgren et al.). Serotoninergic-induced. yawning increases cholinergic activity, and through an inhibitory feedback mechanism, decreases serotoninergic activity. As long as yawning continues, sleep initiation is avoided (Friedell, 1974; Lehmann, 1979; Twiest, 1974). The end of yawning is associated with a feeling of comfort, the result of a temporary victory over the onset of sleep.

A certain constellation of neurotransmitters may favor the yawning reflex. This constellation includes increased serotoninergic activity and changed cholinergic, peptidergic, and dopaminergic receptor activity. This pattern suggests that yawning has an antisleep effect.

The Pathology of Yawning

Yawning may occur not only because of boredom, drowsiness, or fatigue but also in association with various pathologies. For example. psychoneurotic patients can present a state of repetitive yawning with a rhythmic periodicity lasting for 9 to 12 min. The rate of yawning is usually 2 to 4 yawns per min (personal observation; Marcus, 1973).

The Fatal Yawn

When boredom, fatigue, and drowsiness overcome a driver during a long and fast journey, a noisy yawn may be a sign of imminent sleep and, possibly, a fatal road accident. Nakanishi, Hinoki, Ito, Izumikawa, & Baron (1980) found this fatal yawn to be related to an autonomic nervous system disturbance. The fatal yawn must be differentiated, however, from. car-sickness yawning, which appears in scoliotic females and is associated with nausea and vertigo during driving.

Recurrent Cerebral Ischemic Yawning

Yawning was responsible for transient ischemic attacks in a patient who underwent a temporal artery-middle cerebral artery bypass operation (Handa, Nakasu, & Kidooka, 1983). Yawning provoked recurrent cerebral ischemia by kinking the donor artery with each wide mouth opening. Yawning associated with a slow EEG due to borderline hypoxia was present in occlusive cerebrovascular patients (Karasawa et al., 1982).

Synkinetic Yawning

Mulley (1982) found yawning with synkinetic movements in the hemiplegic arm in 80% of 40 hemiplegic patients. Much less frequent synkinesias were observed during stretching, coughing, and laughing. Voluntary movements diminish synkinesias. Sinkinetic yawning indicates poor prognosis for hemiplegics (Mulley).

Yawning Epilepsy

Yawning associated with head extension and occipital headaches showed general dysrhythmic activity on the EEG record of a 35-year-old woman, who was diagnosed as having diencephalic epilepsy. The administration of naloxone hydrochloride suppressed the attacks, suggesting disturbed endorphine receptor activity (Flechter et al., 1982). This case resembles the patient described by Jacorne, McLain, and Fitzgerald (1980) whose volitional head and neck extension precipitated laughing epilepsy associated with orgasmolepsy. Ratner and Kaisarova (1981) observed 12 children in whorn a prolonged inclined position of the head or prolonged stress provoked yawning, headache, and neck pain, followed by unconsciousness lasting 20 to 30 min. The unconsciousness was followed by several hours of muscle atonia. Ratner and Kaisarova narned these attacks "syncopal vertebral syndrome."

Yawning and Orgasm

Yawning associated with orgasm appeared as a side effect of clomiprarnine treatment in a depressed patient and disappeared when treatment ceased (McLean, Forsythe, & Kapkin, 1983).

Drug Withdrawal

One of the most frequently encountered pathologic associations of yawning is withdrawal from opiates. Yawning associated with sneezing, profuse sweating, and vomiting in infants 2 to 3 days old is a withdrawal reaction to maternal. heroin, morphine, or methadone addiction (Rudolph, Barnett, & Einhorn, 1977).

In a lefetamine-addicted adult, who was using this opiate drug at a dose of 0.9 to 1.8 g/day, the withdrawal syndrome appeared when 140 mg of naloxone per day was administered. The syndrome consisted of yawning accompanied by a sympathicornimetic reaction of midriasis, piloerection, and arterial hypertension (Paroli et al., 1984).

Among heroin addicts, withdrawal consists of yawning, profuse sweating, digestive disorders, and agitation (Daulouede, 1980). A withdrawal reaction to pentazocin, a morphine-nalorphine-like narcotic, consists of prominent yawning associated with general epileptic seizures. Greater drowsiness and respiratory depression différentiate the effects of pentazocin from those of morphine, and consequently the defense arousal reaction to borderline hypoxia resulting from pentazocin's withdrawal is made more active (Krupp & Chatton, 1977).

Withdrawal frorn methadone consists of yawning accompanied by lacrimation, rhinorrhea, coughing, gooseflesh, tremor, hot and cold flashes, abdominal cramps, muscle aches, nausea, vomiting, and diarrhea (Lett, Wilson, & Gambrell, 1976). Naloxone (0.2 mg/kg) provokes yawning in opiateaddicted adults (Paroli et al., 1984; Nemeth-Coslett & Griffiths, 1986).

The Psychoanalysis of Yawning

Boredom lowers concentration capacity, which stimulates yawning (Suganami, 1977). Sartre, whose work Nausea was analyzed by Bell (1980) suggested that boredorn and yawning are associated with existence and being, and not with freedom and transcendence. According to Sartre, yawning plays a role in the ethics of authenticity. Bell induced yawning in undergraduate subjects by instructing them to think about it, showing that yawning is a stereotyped action pattern. A nonverbal stereotyped pattern may be encountered as passive violent behavior, expressing refusal to participate in dialogue.

Prognostic Aspects

Clicks or crepitations or myofacial pain during yawning may indicate a temporomandibular joint disturbance requiring X-ray examination (Heloe & Heloe, 1979). Yawning in drivers is a dangerous sign, showing the need for an immediate stop and rest prior to the resumption of driving. Yawning is such a stable feature in withdrawal that it could be used as an objective component of a conditioned abstinence response prior to its spontaneous appearance (O'Brien, O'Brien, Mintz, & Brady, 1975). Yawning associated with synkinesias in hemiplegics indicates poor prognosis (Mulley, 1982). In early schizophrenia, yawning indicates good prognosis, but in chronic schizophrenia it indicates poor prognosis (Lehmann, 1979). During coma, yawning predicts the approach of wakefulness (Braunwald et al., 1987).

Yawning is a sui generis neuropsychological field of inquiry, till now neglected by medicine. I hope that, by delineating this phenornenon, I have stimulated interest in further scientific research on this topic.

References

1. Arai, K., Kita, K., Komiyama, A., Saeki, N., & Nagao, K. 1. (1986). Progressive dysautonomia in hemangioblastoma of the fourth ventricle region. Brain-Nerve, 38, 195-200.

2. Ase, Y, Arai, T., Iida, R., & Okada, S. (1985). Positive middle ear pressure on tympanogram in sleeping children. Audiology Japan, 28, 772-777.

3. Bauer, G., Gerstenbrand, F., & Hengl, W. (1980). Involuntary motor phenomena in the locked-in syndrome. Journal of Neurology, 223, 191-198.

4. Beckmann, H., & Zimmer, R. (198 1). An ethological interpretation of stereotypy induced by environmental stimulus. Archiveffir Psychiatry und Nervenkrank, 230, 81-89.

5. Beeson, P. B., McDermott, W., & Wyngaarden, J. B. (Eds). (1979). Cecil textbook of medicine (15th ed.). Philadelphia: W. B. Saunders, Co.

6. Bell, L. A. (1980). Boredom and the yawn. Review of Existential Psychology and Psychiatry, 17, 91-100.

7. Bhangoo, K. S. (1974). Letter. New England Journal of Medicine, 290, 1440.

8. Braunwald, E., Isselbacher, K. J., Petersdorf, R. G., Wilson, J. D., Martin, J. B., & Fauci, A. S. (Eds). (1987). Harrisons principles of internal medicine (I Ith ed). New York: McGraw-Hill.

9. Daulouede, J. P. (1980). Une nouvelle methode de sevrage des toxicomanes par utilisation du courant de Limoge [A new method of eliminating drug addiction using Limoge's current]. Annales Medico-Psychologigues, 138, 359-370.

10. De Vries, J. 1. P., Visser, G. H. A., & Prechtl, H. F. R. (1983). The emergence of fêtal behaviour: I. Qualitative aspects. Early Human Development, 7, 301-322.

11. De Vries, J. I. P., Visser, G. H. A., & Prechtl, H. F. R. (1985). The emergence of fetal behaviour: II. Quantitative aspects. Early Human Development, 12, 99-120.

12. Donovan, B. T. (1978). The behavioral actions of the hypothalamic peptides: A teview. Psychological Medicine, 8, 305-316.

13. Feldman, J. F., Brody, N., & Miller, S. A. (1980). Sex différences in non-elicited neonatal behaviors. Merril Palmer Quarterly, 26, 63-73.

14. Flechter, S., Cohen, F., Borenstein, F., Regev, I., & Vardi, J. (1982). Yawning as a

paroxysmal sign of diencephalic seizures. Archives Psychology Psychiatry Neurology, 43, 45-54.

15. Fogel, A. (1980). The effect of brief separations on 2-month-old infants. Infant Behavioral Development, 3, 315-330.

16. Friedell, A. (1974). Letter. New England Journal of Medicine, 290, 1439-1440.

17. Geschwend, J. (1977). Yawning in a case with transecting glioma of the pons. Fortschrift Neurologie und Psychiatry Grenzgeb, 45, 652-655.

18. Gill, N. E., White, M. A., & Anderson, G. C. (1984). Transitional newbom infants in a hospital nursery: From first oral eue to first sustained cry. Nursing Research, 33, 213-217.

19. Goldberg, R. L. (1983). Sustained yawning as a side effect of imipramine. International Journal of Psychiatry and Medicine, 13, 277-280.

20. Handa, J., Nakasu, Y., & Kidooka, M. (1983). Transient cerebral ischemia evoked

21. Rollinson, R. D., & Gilligan, B. S. (1979). Post anoxic action myoclonus Lance Adams Syndrome responding to valproate. Archives of Neurology, 36, 44-45.

22. Rudolph, A. M., Bamett, H. L., & Einhom, A. H. (Eds.). (1977). Pediatrics (16th ed.). New York: ACC.

23. Sandyk, R. (1987). Excessive yawning and progressive supranuclear palsy. International Journal of Neuroscience, 34, 123-124.

24. Siegal, S. (1974). Why the yawn? New England Journal of Medicine, 290, 1027.

25. Small, M. M. (1977). Covert discrimination training: An experimental test of the assumed functional equivalence between covert and overt conditioning procedures. Psychological Reports, 41, 715-720.

26. Suganami, S. (1977). Study on subjective symptoms of fatigue of senior high school students: Part 2. Study on physical load of senior high school students. Okayama Iqakkai Zasshi, 89, 195-218.

27. Twiest, M. (1974). Why the yawn? New England Journal of Medicine, 290, 14391440.

TABLE 1

Yawning Etiology

Cause	References
Psychological	
Boredom	Bell (1980); Kishida (1973)
Drowsiness	Suganami (1977)
Fatigue	Suganami (1977)
Separation	Fogel(1980)
Iriùtation	Provine (1986)
Autosuggestion	Small (1977)
Hormonal	
Adrenocorticotropic hormone (ACTH)	Donovan(1978)
Melanocyte-stimulating hormone (MSH)	Donovan (1978)
Neurological	
Coma and vegetative state	Braunwald et al. (1987)
Encephalitis	Beeson et al. (1979)
Brain hypoxia	Karasawa et al. (1982)
Intracranial hypertension	Beeson et al. (1979)
Hemiplegia	Mulley (1982)
Diencephalic epileptic seizures	Fletcher et al. (1982)
Thalamotomy	Jurko & Andy (1982)
Fourth ventricle tumors	Arai, Kita, Komiyama, Saeki &Nagao (1986)
Progressive supranuclear palsy	Sandyk (1987)
Pontine tumors or lesions	Bauer et al. (1980), Geschwend (1977)
Syncopal vertebral syndrome	Ratner & Kaisarova (1981)
Psychiatric	
Schizophrenia	Beckmann & Zimmer (1981)
Organic psychosis	Lehmann (1979)
Involutional depression	Lehmann (1979)
Withdrawal syndrome	Daulouede (1980); Maeda (1975); Lett et al. (1976)
General pathologic	
Increased ear cavity pressure	Ase, Arai, Iida, & Okada (1985)
Gastric dyspepsy	Braunwald et al. (1987)
Biliar dyskinesia	Braunwald et al. (1987)
Car sickness in scoliotics	Nakanishi et al. (1980)
Drug overdose	
Naloxone	Paroli et al. (1984); Nemeth-Coslett & Griffiths (1986); O'Brien et al.(1975)
Valproate	Rollinson & Gilligan (1979)
Imipramine	Goldberg (1983)
Clomipramine	McLean et al. (1983)
Seratonin	Holmgren et al. (1980)
Pentobarbital	Lehmann (1979)
Drug withdrawal	
Heroin	Rudolph et al. (1977)
Morphine	Rudolph et al. (1977)

Lefétamine	Paroli et al. (1984)
Methadone	Lett et al. (1976)
Pentazocin	Maeda (1975)

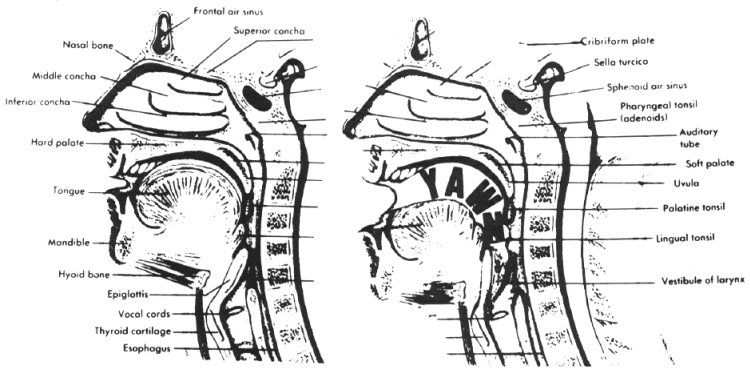

FIGURE 1. A comparative presentation of the tongue, bucopharynx, and epiglote at rest and while yawning, explaining the massive air inhalation.

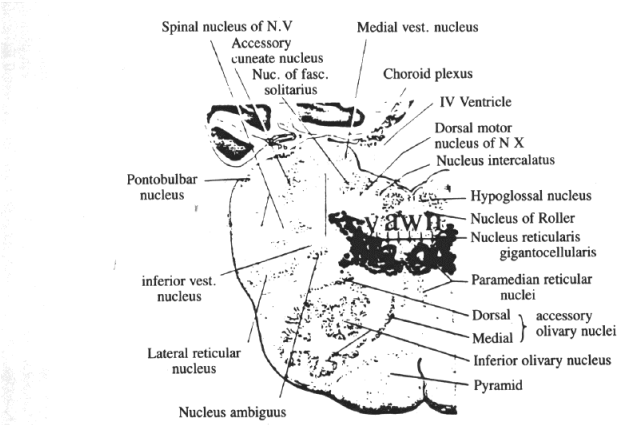

FIGURE 2. A brain stem section at the pontine level showing the location of the reticular neuronal yawning complex.