

READERS' COMMENTS

More About Yawning

In response to a case report of repetitive yawning associated with cardiac tamponade,¹ I wrote a Readers' Comment titled "Yawning for an Answer."² As a consequence, I recently received a very interesting and informative communication from Olivier Walusinski,³ who is a world authority on yawning and author of a book titled "Bâillements et Pandiculations."³ He enlightened me on several aspects of yawning that might be of interest. Because Walusinski's reports appeared in noncardiologic publications, cardiologists might not have seen or read them.

First, yawning is a phylogenetically old, stereotypical event that occurs in reptiles, fish, birds, and mammals.⁴ Darwin in his 1838 notebook said "... seeing a dog and horse and man yawn, makes me feel how much all animals are built on one structure."⁵ Figure 1 shows a kitten yawning, which is not very different from a human yawning (Figure 2). Incidentally, in the current concern about a world pandemic of avian influenza, yawning in infected flocks is one of the most sensitive clinical indicators to detect such an outbreak, with a sensitivity of 100% and a specificity of 79% in turkeys.⁶

Second, yawning is contagious^{7,8} (Figure 3). A famous saying states that a good yawner will induce yawning in 7 others.⁷ Ethologic studies of nonhuman primates have shown that at certain times an entire group goes about yawning, without the possibility for 1 member to be aware of another yawner, whether by sight, hearing, or smell.⁷ Although yawning occurs in reptiles, fish, birds, and mammals,⁴ yawning replication is found only in humans.^{9–11}

Third, yawning is a familiar and frequent occurrence in neonates soon after



Figure 1. Kitten yawning. (Courtesy of Olivier Walusinski.)



Figure 2. Human yawning. (Courtesy of Olivier Walusinski, whose son is the subject depicted.)

they are born. Yawning can even be detected by 4-dimensional sonography in a fetus⁴ (Figure 4). In fact, the lack of fetal yawning may be a key to predicting brain stem dysfunction after birth.⁴

Last, but not least, Charles Darwin is said to have qualified yawning as a piece of useless physiology.⁷ However, from the voluminous data on the subject (682 citations in a Medline PubMed search using yawning as the keyword as of November 14, 2005), yawning is a

very pertinent model allowing for the understanding of a principle of causality, not only through ethologic observations, but also through neurologic determinations regarding neuroanatomy, on the one hand, and neurotransmitters and hormones, on the other.⁷ Charcot (1825–1893) and Tourette (1857–1904) described yawning as a clinical sign, long since forgotten.⁷ With the recent report by Krantz et al¹ of repetitive yawning in a patient with cardiac tam-

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Figure 3. Yawning is contagious, as shown by the responses of the delegates to China's National People's Congress to a series of long-winded reports in Beijing's Great Hall of the People on March 9, 2005 (from Provine,⁸ with permission.)

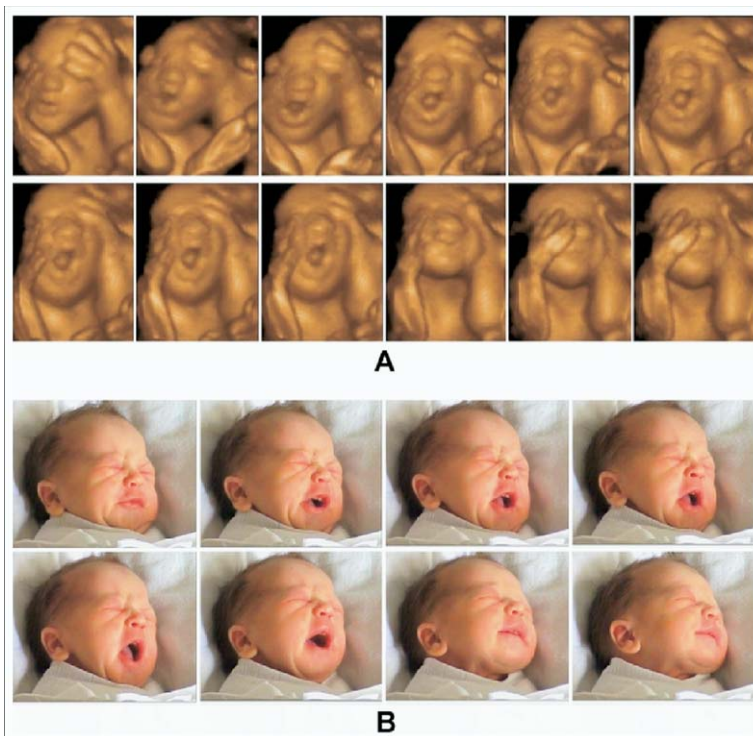


Figure 4. (A) Fetal yawning demonstrated in utero by 4-dimensional sonography similar to (B) yawning during neonatal period of same baby. (Courtesy of Olivier Walusinski.)

ponade, its importance extends even into cardiology. For those readers interested to know more about yawning, there is a Web site <http://yawning.info> with all you want to know about yawning.

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Retinal Arterial Signs and Coronary Heart Disease

The recent article by Tedeschi-Reiner and colleagues¹ on the relation of retinal arteriolar changes and coronary heart disease adds to the growing evidence that retinal vascular signs may

be markers of systemic cardiovascular disease.¹ In 109 patients aged 40 to 80 years who underwent coronary angiography, the investigators showed a statistically significant correlation of retinal atherosclerotic changes (using the Scheie classification) with the severity of coronary atherosclerosis (using the Gensini score).

Although the findings are interesting, I have some concerns with the methods and presentation of this study. First, direct ophthalmoscopy was used to define the severity of the retinal arteriolar changes. This may be problematic, because direct ophthalmoscopy has been shown to be a subjective and unreliable technique in measuring retinal arterial changes.^{2,3} If available, data on the reliability of their retinal assessment (e.g., intraobserver kappas) should be presented. Second, the investigators only present the crude correlation between the severity of retinal arterial changes and coronary atherosclerosis, without consideration for the effects of important confounders, such as age, blood pressure, presence or absence of diabetes, and cigarette smoking status. Thus, it is unclear whether this correlation is independent of the shared risk factors. It will be important for readers to see multivariate analysis results that adjust for age and other factors to determine whether an independent association exists between retinal and coronary artery disease.

Finally, the findings of the present investigation should be discussed within the context of an earlier study by Michelson and colleagues,⁴ who reported that retinal arteriolar changes were predictive of the severity of coronary stenosis on angiography in 70 nonhypertensive, nondiabetic patients. The similarity of their findings with those of the present study is striking. In addition, a substantial body of research in large population-based cohorts have examined the association of retinal vascular changes, as measured from fundus photographs, and systemic atherosclerosis disease, including incident stroke,⁵ coronary heart disease,⁶ and congestive heart failure.⁷ Data from these cohorts provide further support to the present study findings.

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Peripheral Arterial Disease Is a Marker of Risk for Abdominal Aortic Aneurysms in Patients With Coronary Artery Disease

Madaric et al¹ studied the prevalence and predicting factors for abdominal aortic aneurysms (AAAs) in 109 patients >60 year old with coronary artery disease (CAD). However, the evaluation of a variety of parameters at the moment of diagnosis of an AAA is not the best way to identify the etiologic factors that should precede the development of AAAs. We believe, nonetheless, that this study may still be useful to identify markers of AAAs, pointing to patients with a particularly high risk of AAAs. In this regard, some of their data are difficult to compare with other studies, because they did not provide a precise definition for independent variables, such as hyperlipoproteinemia, pe-