References and Notes

1. V. V. Zheleznyakov, *Sov. Astron. AJ* **6**, 3 (1962); T. Kakinuma and G. Swarup, *Astrophys. J.* **136**, 975 (1962); M. R. Kundu, *Solar Radio Astronomy* (Interscience, New York, 1965); P. Lantos, *Ann. Astrophys.* **31**, 101 (1968); E. Ya. Zlotnik, *Sov. Astron. AJ* **12**, 245 (1968); *ibid.*, p. 464; V. V. Zheleznyakov, *Radio Emission from the Sun and Planets* (Pergamon, New York, 1970); A. Kruger, *Introduction to Solar Radio Astronomy and Radio Physics* (Reidel, Boston, 1979).
2. J. A. Ratcliffe, *The Magneto-Ionic Theory* (Cambridge Univ. Press, Cambridge, 1962); K. R. Lang, *Astrophysical Formulae* (Springer, New York, 1974, 1980).
3. M. R. Kundu and C. E. Alissandrakis, *Nature (London)* **257**, 465 (1975); ———, J. D. Bregman, A. C. Hin, *Astrophys. J.* **213**, 278 (1977); K. R. Lang and R. F. Willson, *Nature (London)* **278**, 24 (1979); in *Radio Physics of the Sun* (International Astronomical Union Symposium 86), M. R. Kundu and T. E. Gergely, Eds. (Reidel, Dordrecht, 1980), p. 109; M. R. Kundu, E. J. Schmahl, M. Gerassimenko, *Astron. Astrophys.* **82**, 265 (1980); M. Felli, K. R. Lang, R. F. Willson, *Astrophys. J.* **247**, 325 (1981); R. Pallavacini, T. Sakurai, G. S. Vaiana, *Astron. Astrophys.* **98**, 316 (1981).
4. M. R. Kundu, T. Velusamy, R. H. Becker, *Sol. Phys.* **34**, 217 (1974); K. R. Lang, *ibid.* **36**, 351 (1974); C. E. Alissandrakis and M. R. Kundu, *Astrophys. J.* **222**, 342 (1978); M. R. Kundu, in *Radio Physics of the Sun* (International Astronomical Union Symposium 86), M. R. Kundu and T. E. Gergely, Eds. (Reidel, Dordrecht, 1980), p. 157; K. Marsh, G. J. Hurford, H. Zirin, *ibid.*, p. 191; K. R. Lang, R. F. Willson, M. Felli, *Astrophys. J.* **247**, 338 (1981).
5. K. A. Marsh and G. J. Hurford, *Astrophys. J.* **240**, L111 (1980).
6. ———, *Annu. Rev. Astron. Astrophys.* **20**, 497 (1982); M. R. Kundu, *Rep. Prog. Phys.* **45**, 1435 (1982); ——— and L. Vlahos, *Space Sci. Rev.* **32**, 405 (1982).
7. E. B. Fomalont, *Proc. Inst. Elec. Eng.* **61**, 1211 (1973).
8. K. R. Lang and R. F. Willson, in *Advances in Space Research—Proceedings of the 24th Meeting of COSPAR* (Pergamon, London, 1983), vol. 1, p. 91; ———, V. Gaizauskas, *Astrophys. J.* **267**, 455 (1983); R. K. Shevgaonkar and M. R. Kundu, *ibid.* **283**, 413 (1984).
9. G. B. Gel'freikh and B. I. Lubyshev, *Sov. Astron. AJ* **23**, 316 (1979); C. E. Alissandrakis, M. R. Kundu, P. Lantos, *Astron. Astrophys.* **82**, 30 (1980); C. E. Alissandrakis and M. R. Kundu, *Astrophys. J.* **253**, L49 (1982); K. R. Lang and R. F. Willson, *ibid.* **255**, L111 (1982).
10. M. R. Kundu and T. Velusamy, *Astrophys. J.* **240**, L63 (1980); T. Velusamy and M. R. Kundu, *ibid.* **243**, L103 (1981); K. R. Lang, R. F. Willson, J. Rayrole, *ibid.* **258**, 384 (1982); G. A. Dulk and D. E. Gary, *Astron. Astrophys.* **124**, 103 (1983); K. R. Lang and R. F. Willson, *ibid.* **127**, 135 (1983); D. McConnell and M. R. Kundu, *Astrophys. J.* **269**, 698 (1983).
11. D. Rust, *Sol. Phys.* **47**, 21 (1976); M. R. Kundu *et al.*, *Astron. Astrophys.* **108**, 188 (1982); R. F. Willson, *Sol. Phys.* **83**, 285 (1983); K. R. Lang and R. F. Willson, in *Advances in Space Research—Proceedings of the 24th Meeting of COSPAR* (Pergamon, London, 1983), vol. 2, p. 91; M. R. Kundu, *Proc. Int. Astron. Union Symp. 107* (1984), p. 185.
12. T. Gold and F. Hoyle, *Mon. Not. R. Astron. Soc.* **120**, 89 (1960); J. Heyvaerts, E. R. Priest, D. M. Rust, *Astrophys. J.* **216**, 123 (1977); D. S. Spicer, *Sol. Phys.* **53**, 305 (1977); S. I. Syrovatskii and V. D. Kuznetsov, in *Radio Physics of the Sun* (International Astronomical Union Symposium 86), M. R. Kundu and T. E. Gergely, Eds. (Reidel, Dordrecht, 1980), p. 445; V. D. Kuznetsov and S. I. Syrovatskii, *Sol. Phys.* **69**, 361 (1981); D. S. Spicer, *ibid.* **70**, 149 (1981); V. Petrosian, *Astrophys. J.* **255**, L85 (1982); B. V. Somov and S. I. Syrovatskii, *Sol. Phys.* **75**, 237 (1982); E. R. Priest, *ibid.* **86**, 33 (1983).
13. K. R. Lang, *Sol. Phys.* **52**, 63 (1977); M. R. Kundu, in *Radio Physics of the Sun* (International Astronomical Union Symposium 86), M. R. Kundu and T. E. Gergely, Eds. (Reidel, Dordrecht, 1980), p. 157; G. Hurford and H. Zirin, *Air Force Geophys. Lab. Rep. AFGL-TR-82-0017* (1982); R. K. Shevgaonkar and M. R. Kundu, *Astrophys. J.*, in press; K. R. Lang and R. F. Willson, *Advances in Space Research—Proceedings of the 25th Meeting of COSPAR* (Pergamon, London, in press).
14. M. R. Kundu, E. J. Schmahl, T. Velusamy, *Astrophys. J.* **253**, 963 (1982); ———, L. Vlahos, *Astron. Astrophys.* **108**, 188 (1982); T. Velusamy and M. R. Kundu, *Astrophys. J.* **258**, 388 (1982); M. R. Kundu, *Sol. Phys.* **86**, 205 (1983); M. R. Kundu, in *Advances in Space Research—Proceedings of the 24th Meeting of COSPAR* (Pergamon, London, 1983), vol. 1, p. 159; M. R. Kundu, D. M. Rust, M. Bobrowsky, *Astrophys. J.* **265**, 1084 (1983); R. F. Willson and K. R. Lang, *Astrophys. J.* **279**, 427 (1984); R. F. Willson, *Sol. Phys.* **92**, 189 (1984).
15. M. R. Kundu, V. Gaizauskas, B. Woodgate, E. J. Schmahl, R. Shine, H. Jones, *Astrophys. J. Suppl. Ser.*, in press.
16. M. R. Kundu, *Proc. Int. Astron. Union Symp. 107* (1984), p. 185.
17. J. C. Brown, D. B. Melrose, D. S. Spicer, *Astrophys. J.* **228**, 592 (1979); D. F. Smith and C. G. Lilliquist, *ibid.* **232**, 582 (1979); L. Vlahos and K. Papadopoulos, *ibid.* **233**, 717 (1979); G. D. Holman, M. R. Kundu, K. Papadopoulos, *ibid.* **257**, 354 (1984).
18. D. E. Gary and J. L. Linsky, *ibid.* **250**, 284 (1981); K. Topka and K. A. Marsh, *ibid.* **254**, 641 (1982); J. L. Linsky and D. E. Gary, *ibid.* **274**, 776 (1983).
19. D. E. Gary, J. L. Linsky, G. A. Dulk, *ibid.* **263**, L79 (1982); D. B. Melrose and G. A. Dulk, *ibid.* **259**, 844 (1982); K. R. Lang, J. Bookbinder, L. Golub, M. M. Davis, *ibid.* **272**, L15 (1983).
20. C. Slottje, *Nature (London)* **275**, 520 (1978); in *Radio Physics of the Sun* (International Astronomical Union Symposium 86), M. R. Kundu and T. E. Gergely, Eds. (Reidel, Dordrecht, 1980), p. 195; R. Zhao and S. Jin, *Sci. Sin. A* **25**, 422 (1982).
21. G. D. Holman, D. S. Eichler, M. R. Kundu, in *Radio Physics of the Sun* (International Astronomical Union Symposium 86), M. R. Kundu and T. E. Gergely, Eds. (Reidel, Dordrecht, 1980), p. 457; G. D. Holman, *Adv. Space Res.* **2**, 181 (1982); R. R. Sharma, L. Vlahos, K. Papadopoulos, *Astron. Astrophys.* **112**, 337 (1982); L. Vlahos, R. R. Sharma, K. Papadopoulos, *Astrophys. J.* **275**, 374 (1983).
22. Adapted from (*5*), plate 2.

23. This review would have been impossible without the fine contributions to solar radio astronomy made by R. F. Willson at Tufts University and C. E. Alissandrakis, E. J. Schmahl, T. Velusamy, and L. Vlahos while working for the University of Maryland. Radio astronomical studies of the sun and other active stars at Tufts University are supported under Air Force Office of Scientific Research grant AFOSR-83-0019-B. Investigations of flare stars at Tufts University are also supported by NASA grant NAG 5-477; whereas comparisons of VLA and Solar Maximum Mission satellite data are supported by NASA Guest Investigator grant NAG 5-501. Solar research in the Astronomy Program at the University of Maryland is supported under NSF grant ATM 84-15388, NASA grant NGR 21-002-199, and NASA contract NSG 5320. The Very Large Array is operated by Associated Universities Inc., under contract with the National Science Foundation.

Emotion and Facial Efference: A Theory Reclaimed

R. B. Zajonc

Nearly 80 years ago a book appeared in Paris under the title *Physionomie Humaine: Son Mécanisme et son Rôle Social* (*1*). The book, written by Israel Waynbaum, a physician, offered a radical theory of emotional expression, defying all previous ones, including Darwin's dominant theory. It clarified, for the first time, the function of emotional expression in the emotional process—the foremost problem in the study of emotions at the turn of the century. Yet Waynbaum's book received no attention and it has remained unknown until now. Neither the author nor his idea are to be found in the *Science Citation Index* or in numerous reviews of research on emotion or facial expression. In this article I review Waynbaum's theory, compare it with Darwin's, bring it up to date, and show that it forms a promising basis for a comprehensive theory of the emotions.

Waynbaum's analysis led him first to question the term "emotional *expression*." He feared that referring to facial movements as "expressions," the standard term since Aristotle, reinforced by Darwin's classic work (*2*), implicitly fixed their role in the emotional process, and "solved" the problem by definition. The term "expression" specifies a priori the causal sequence among emotional correlates, placing the efferent process at the terminus. As such, therefore, "expression" cannot be the cause of any other aspect of the emotional process. The term "expression" also implies the existence of an antecedent internal state which "expression" externalizes, manifests, and displays. It implies further that the antecedent internal state seeks externalization that forces itself onto the surface. Hence, there also exists the term "suppression." In many cases the behavioral output may well "express" an internal state, and some reactions are indeed suppressed. But it is by no means established that all facial gestures that are classified as expressions are caused by internal subjective states. It may be

The author is a professor of psychology at the University of Michigan, Ann Arbor 48104.

Summary. A theory of emotional expression, ignored since 1906, holds that facial muscles act as ligatures on facial blood vessels and thereby regulate cerebral blood flow, which, in turn, influences subjective feeling. The theory, developed by Israel Waynbaum, a French physician, hypothesizes the subjective experience of emotions as following facial expression rather than preceding it. It answers Darwin's question of why different muscles contract or relax in different emotions better than Darwin's own theory. When restated in terms of contemporary neurophysiological knowledge, it explains and organizes several ill-understood emotional processes and phenomena.

best, therefore, to refer to these phenomena as "emotional efference"—a term that has fewer a priori implications. Of course, the term "expression" is consistent with all current theories of emotion; only Lange and James (*3*) questioned the assumption that feeling precedes efference, but they failed to provide an explanatory and conceptual framework, and their argument was soon rejected. Waynbaum's first contribution was to question these 19th-century views—views that still dominate current thinking about emotion.

According to Andrew (*4*, p. 1034), facial expression has evolved "to communicate information about the probable future behavior of the displaying animal." By expressing its emotions, one animal could signal its intentions to another. But why should humans, who have such a powerful means of communication—language—be the one animal to have also developed the most complex repertoire of facial movements? Do we need, in addition to speech, some 80 facial muscles merely to broadcast our intentions?

Moreover, why should we project our intentions on our faces in the first place? It is perhaps reasonable to suppose that baring teeth and growling in rage might cause an intruder to retreat, and thus prevent violence. But what purpose might be served by displaying one's own fear to an enemy, or one's own surprise to an intruder? No doubt some evolutionary explanations might be suggested, but such explanations depend on conjecture, and it is difficult to imagine the evolutionary process whereby they could have become universal. Hardly any theory of emotion and facial expression challenges or even questions this critical aspect of Darwin's theory.

In contrast to all contemporary theories of emotion, Waynbaum offered the hypothesis that emotional responses have as their principal function the auxiliary control of cerebral blood flow (CBF). Noting that all overt emotional responses (blushing, sobbing, weeping, frowning, and so forth) are closely tied to vascular processes, Waynbaum proposed that the musculature of the face is capable of compressing veins and arteries of the face and thus controlling facial blood flow, which he thought could act as a safety valve for CBF. Blood circulation was also implicated in the actions of the diaphragm that arise in sobbing or laughing, or in tears shed when weeping or laughing. Waynbaum did not take issue with the display function of facial movements, but he considered this function to be derivative and incapable of explaining them.

To fully appreciate Waynbaum's contribution, however, one must put it in the context of Darwin's work.

Darwin's theory of emotional expression. To explain emotional expression, Darwin wrote, one must understand "why different muscles are brought into action under different emotions; why, for instance, the inner ends of the eyebrows are raised, and the corners of the mouth depressed, by a person suffering from grief or anxiety" (*2*, p. 3). However, Darwin's theory of emotional expression does not begin to answer his own question.

Darwin seems, by his empirical method, to have been less interested in explicating emotional expressions than in using these phenomena to substantiate the evolutionary hypothesis for behavior as he did for structure. He did not seek to specify the ongoing process of emotional expressions, nor to understand their mechanisms. Instead, he solicited observations about emotions from remote parts of the world to establish the universality of expressions. His book closes with the assertion that his "theory of expression confirms to a certain limited extent the conclusion that man is derived from some lower animal form" (*2*, p. 365). Most subsequent research on emotions has followed Darwin's interest in universality and taxonomy (*5*). This research supports the hypothesis of the innate basis of expression, but it does not address Darwin's "why" question.

Darwin's theory consists of three principles. The first—that of serviceable habits—holds that some actions relieve the organisms's wants, gratify its needs and desires, and guide it in reacting to sensations and to internal states. These serviceable actions will be repeated by force of habit, especially when they have done so "during a long series of generations" (*2*, *6*). The second principle holds that for many actions there exist antithetical actions elicited by the opposite stimulus conditions. Darwin gives the example of a dog mistaking a familiar person for a stranger, and upon discovery of its error changing from a threatening posture to the antithetical posture of submission and affection. The third principle—that of the direct action of the nervous system—concerns behavior that occurs independently "of the Will, and independently to a certain extent of Habit" (*2*). Tears, trembling, rage, and changes in heart rate are such direct products of the nervous system.

Thus, Darwin's three basic principles are a heuristic schema that directs the researcher interested in knowing "why different muscles are brought into action under different emotions" to conjure up any reasonable function that the given expression could have played at some point of evolution. If such a function cannot be hypothesized, then the researcher must look for a function of an opposite expression. If both fail, then the expression must be elicited by the direct action of the nervous system. But we are not told by Darwin how or where to look for these functions, and it is not obvious what are "opposite" actions. Thus, the baring of teeth is viewed as a threat signal, even though teeth are also displayed in the grin (*4*). Therefore, Darwin's principles cannot be falsified. Darwin revolutionized the thinking on emotional expression by placing it in the context of adaptive processes, and he was an outstanding observer who amassed a wealth of data. But it would be false to credit him with a general theory of emotional expression.

Waynbaum's vascular theory of emotional expression. In contrast to Darwin, Waynbaum refused to view muscular movements as the terminal stage of the emotional process. He attributed to them a much more significant internal regulatory role. Waynbaum's thinking was based on a number of converging facts. (i) The supply of blood to the brain and to the face derives from one source, the common carotid artery. (ii) The supply of cerebral blood must be stable. There can be no sudden changes even if the rest of the circulatory system undergoes violent variations. (iii) Although the bony

structure of the face is mostly rigid, the face has an inordinate number of muscles. Why? (iv) The facial artery, a major branch of the external carotid, is rich in muscular tissue. Again, why?

Waynbaum argued that all emotional reactions produce circulatory perturbations. Either they mobilize considerable energy, or, as in depression, they remove energy demands. They may thus produce a disequilibrium in CBF. Since the external and internal carotid have a common origin, equilibrium in the CBF can be maintained by directing more blood to the face and skull or by diverting it toward the brain.

Because facial muscles, when contracting, push against the bony structure, they can act as tourniquets on arteries and veins and thus regulate facial blood flow. By modulating flow in the external carotid and in the facial veins, they regulate CBF, reducing or complementing it. The same is true of other expressive acts, such as those controlled by glandular secretion or the diaphragm. For example, the lachrymal gland is supplied by the lachrymal artery which branches off from the internal carotid by way of the ophthalmic artery. Therefore, an augmented blood flow in the lachrymal artery should reduce the flow in the internal carotid, decreasing CBF, and hence causing momentary anesthesia of the corresponding regions of the nervous system.

If Waynbaum's theory is correct, it can answer Darwin's "why" question more directly than the evolutionary theory of expression. Why are smiling and laughing the major gestures of happiness and joy? Waynbaum asserted that moderate brain hyperemia is associated with healthy and positive affective tonus, whereas the opposite—temporary brain anemia or ischemia—is associated with negative affect, depressive moods, and unsound physical condition. But he also asserted that the subjective experience of elation follows the smile, not the other way around. The laughing person approaches a state of congestion. A hard laugh makes the face quite red, and sometimes even violet. The return circulation is impeded by the contracted cervical skin muscles that press on the jugular veins. Thus, more blood remains in the brain. The function of tears that are often shed during hard laughter is to relieve the rising pressure of cerebral blood. These tears always come at the very end of a laughing bout, and as in crying they lead to a local anesthesia.

Why is the zygomatic muscle involved in smiling and in happiness? The contraction of the major zygomatic muscle,

Israel Waynbaum

asserted Waynbaum, has a congestive cerebral circulatory function. The proof is simple. Pull the corners of your mouth apart by contracting the major zygomatic muscle, as if in intense exaggerated smile. After several seconds, the frontal vein will be gorged with blood. Thus, claims Waynbaum, the zygomatic muscle acts as a ligature on the branches of the external carotid and the slave action of the corrugator blocks the return blood. Cerebral blood is thus momentarily retained causing temporary intracerebral hyperemia, which in turn leads to a surge of subjectively felt positive effect.

Waynbaum offered the opinion that, given its beneficial circulatory effects, laughing must be healthy. It is like taking an oxygen bath. The cells and tissues receive an increased supply of oxygen, causing a feeling of exuberance. The conversion of venous blood into arterial blood is accelerated and, because of the spasmodic action of the diaphragm, the lungs oxygenate at a more rapid pace. In contrast, sadness produces disoxygenation of tissues and attenuates vital processes. Happiness leaves the face young because it involves only one major muscle—the zygomatic. In sadness, many muscles are contracted: the elevators, orbicularis oculi, orbicularis oris, corrugator, frontalis, pyramidal, and others. Hence, frequent crying results in a prematurely wrinkled face.

Why do we furrow our forehead when we concentrate? The frontalis contracts and the forehead becomes furrowed by transverse wrinkles. The eyeball is swollen and pupils dilated. The eyes are often closed, and the orbicularis oris as well as the masseter are contracted to make the jawbone project forward. By putting a tourniquet on the external carotid and on facial veins, all these actions, at the cost of facial circulation, send more blood to the brain. More cerebral blood means better brain work. After 60 years, this hypothesis received empirical support (*7*). A host of other mannerisms, universal in all cultures, are recognized as revealing internal mental states, such as thinking, problem-solving, trying to remember, or making decisions. There are rubbing one's chin, scratching one's head, licking lips, pressing one's ocular region, passing the hand over the forehead, frowning, biting fingernails or pencils, and pulling one's earlobes or eyebrows. If facial musculature can to some extent control CBF, we would have an explanation for phenomena that seem otherwise bizarre and for which no explanation has thus far been offered. While these efferents are peripheral to the emotional process, they show the pervasive role of the vascular system in mental phenomena.

Had Waynbaum Worked Today

Waynbaum developed his theory nearly 80 years ago, and he necessarily made some assumptions now known to be false. Had Waynbaum worked in a modern laboratory he would have known, for example, that the blood supply to the brain does not derive from a single source but that it is distributed through the circle of Willis, which is supplied by a large network of vessels, and that the interdependence between the internal and facial flow is much more limited than he suspected. He would also have known that neural circulatory controls, the profusion of arteries and veins, and the presence of resistance beds create a redundancy in blood supply that can keep CBF constant in a variety of ways, in spite of various muscular acts. But muscle action could be more significant with respect to return circulation because veins are more readily controlled by muscles. (Hence one notes the gorged frontal veins when straining excessively or when in rage.) Because blood from the brain drains through the forehead and face, the muscular action in the face can briefly delay the outflow of blood from the brain. Thus, Waynbaum might have focused more on the influence of muscles on veins than on arterial flow, especially since arteries abound in neural circulatory controls (vasoconstrictors and vasodilators), while veins do not. This new knowledge about the vascular system, nevertheless, does not preclude

Waynbaum's basic hypothesis. The stability of CBF, being of crucial importance, may well be maintained by partially redundant systems. Alterations in CBF that are under different time constraints may be controlled by more than one mechanism. Facial action also has a direct effect on CBF. CBF supplies blood to those regions that are active, and those areas of the motor cortex that correspond to a particular group of facial muscles that are activated receive increased flow. Thus CBF can be regulated by facial musculature, but not necessarily through facial blood flow.

Had Waynbaum worked today, he would not have suspected CBF changes to be the direct antecedents of mood and emotional effects. Rather, he may have drawn a connection between emotion and brain temperature. Before entering the brain, the internal carotid passes through the cavernous sinus. One function of this structure, the only one in the body in which the artery is located inside a vein, is thermoregulation of arterial blood before it enters the brain. In desert ungulates such as the camel, for example, the internal artery is divided into numerous small vessels, creating a radiator pattern. The brain, which in the resting adult organism produces one-fifth of the body's heat, is cooled by arterial blood (*8*).

Temperature is important because it is likely to influence the biochemical action of neurotransmitters and enzymatic pathways. Thus a particular pattern of regional blood flow, together with local rise and fall of brain temperature, might enhance or impair the release and synthesis of different neurotransmitters and thereby produce different subjectively felt states. These subjective states are distinctive because different emotional muscular patterns can alter temperature in different brain regions in different ways. Not only different brain regions and different temperatures may be produced by different muscular patterns, but different quantities of neurotransmitters (serotonin, enkephalin, and so forth) may be synthesized and released selectively.

Variations in the external flow cause corresponding complementary variations in the internal flow (*9*), effects achieved by direct ligatures on the external carotid. It has not been shown that facial musculature, by action on external flow, can affect CBF. If it can, the effect is likely to be limited. Nevertheless, Waynbaum's hypothesis of facial efference having subjective pleasurable consequences by affecting CBF, might be true for reasons that he could not have suspected. For if facial action instigates localized regional CBF, not by peripheral but central processes, and if there are concomitant effects on venous flow, the obtained temperature change may act on the release and synthesis of neurotransmitters (*10*).

Migraine headaches are caused by a vascular dysfunction, migraine sufferers make a variety of unusual mouth movements, such as licking lips and biting the inside of their cheeks, and the external carotid flow rises during headache (*11*). These previously ignored facial movements may therefore play some role in reducing external vasodilation.

Although Waynbaum was right in many ways, his theory was necessarily incomplete because he lacked knowledge that became available only decades later. He constructed his theory piece by piece, creating a coherent and elegant structure of assumed processes, about which he could no more than make intelligent guesses. Waynbaum's theory is a product of a superior imagination that conceptualized the nature of emotional processes almost entirely by the force of logical necessity, for Waynbaum had no special training other than in medicine and never did research in a laboratory.

Tests of the vascular theory of expression. The vascular theory answers Darwin's "why" question of emotional expression directly and it is directly falsifiable. Modern methods of measuring blood flow, for example after xenon-133 is inhaled (*12*), or thermography can be applied to determine whether emotional expressions influence blood flow and CBF. It can now also be determined whether changes in CBF and brain temperature have the effects on emotion and mood that Waynbaum hypothesized. Many hypertensive drugs that produce significant circulatory changes often have serious depressive side effects, and profound mood changes are associated with cerebral vascular disease and with stroke (*13*).

The stability of CBF, suspected by Waynbaum nearly 80 years ago, has also been demonstrated. Globus *et al.* (*14*) found increases in heart rate, breathing, and blood pressure during heavy 10-minute exercise, yet there was no change in CBF over the same time interval. These findings need not contradict the vascular theory because different measures of time are probably involved. The sort of stability found in CBF by Globus *et al.* is obtained by comparing intervals of several minutes. Therefore, these data could conceal local changes in CBF that occur within seconds and dissipate rapidly. Little is known today about the effects of brain temperature on the release of neurotransmitters, although it would be surprising if the optimum temperature for their synthesis and release were not confined to a narrow range, perhaps a fraction of a degree.

Toward an Integrated Theory of Emotional Expression

Waynbaum's most significant contribution was to examine the role of facial movements apart from their expressive consequences. His analysis provides some answers to the controversial problem raised by Lange and James (*3*) about the relative positions of muscular output and subjective feeling in the causal structure of the emotional process. Both Waynbaum and Lange however, begged the question of what causes bodily emotional movements in the first place. The antecedents of facial muscular movements, glandular secretions, arrested breathing, or diaphragmatic spasms are numerous.

1) Since several of these actions can be produced at will, whatever moves a person to perform them—desire to please or frighten another individual, craving for affection, need for help, playful imitation, caprice—is a candidate for an antecedent condition for what might be perceived as "emotional expression." These voluntary acts can be performed even though no emotion or an emotion other than the one displayed is experienced by the individual.

2) Among the involuntary antecedents are fixed action patterns—instinctual reactions that occur spontaneously, such as fear reactions to strangers, withdrawal of an injured limb, or retreat from a rapidly looming object.

3) Expressive movements might be elicited as conditioned responses that have been acquired in the past. A dog that withdraws its leg in response to a shock that had been preceded by a bell comes to withdraw the leg on hearing the bell alone.

4) Expressive acts can arise in mimicry either as instinctive reactions or as responses established by previous reinforcement. For example, if a child's smile is a response to the father's smile and the child, as a result, receives affection, the child's likelihood of returning smiles (and other expressions) is heightened.

5) A class of antecedent conditions for emotional bodily movements derives

from the organisms's orienting reactions. The theories of Piderit and of Gratiolet (*15*) specified these conditions. According to Piderit, emotional expressions derive from the sensory and peripheral activity elicited by emotional stimuli and from the hedonic reactions to these stimuli. Piderit assumed that sensory events give rise not only to peripheral orienting muscular acts but also to consequent acts that are occasioned by their hedonic effects. Orienting acts, such as squinting, drawing up one's nostrils, or licking one's lips, optimize sensory receptivity. The consequent reactions, however, depend on the hedonic nature of the stimulation. When a bitter solution is placed in the mouth, a person tends to make expelling motions. Piderit thus assumed that some sensory events are in themselves pleasurable, and so are the representations that derive from these events. These pleasurable events evoke approaching and accepting actions. Sensations and representations that are disagreeable evoke rejecting actions.

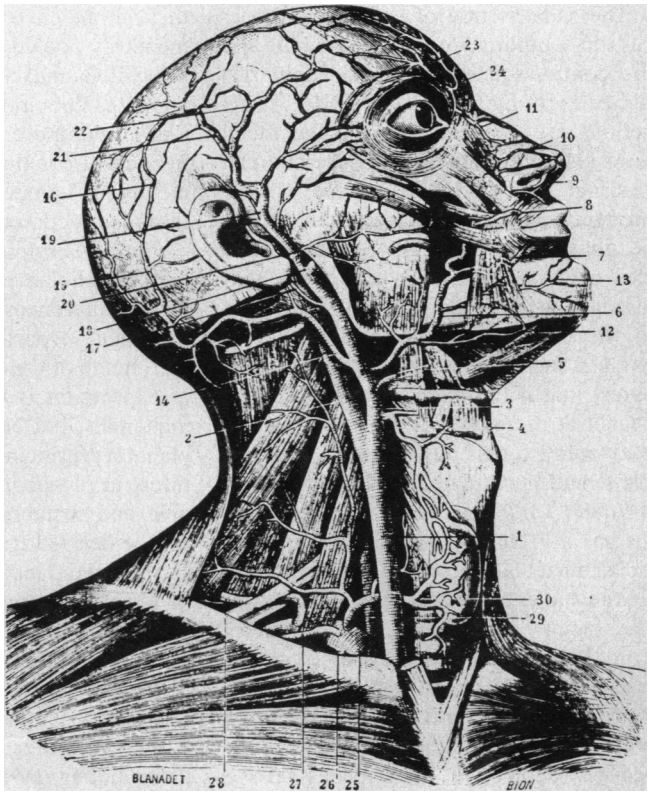
Muscular reactions can occur not only to sensory events but to the memories of these events, to imaginary events, and, through generalization, to emotional excitation. When we imagine biting into a lemon, we may form a rejecting buccal motion, and similar facial gestures express disdain for a silly idea. Gratiolet noted that bowlers contort their bodies to "correct" the paths of their bowling balls. We smile in joy because, by generalization, the mouth makes movements that are homologous to those when the individual tastes a savory morsel. Language is replete with sensation-emotion metaphors. We speak of "bitter enemies," "dark thoughts," and the "pain of separation." And there are "honey," "sweetie," "sugar," "tootsie," "buttercup," among endearments whose etymology lies in gustatory pleasures.

Whatever the antecedents of these facial and bodily movements, they are critical for Waynbaum in affecting CBF and thereby changing the affective tonus and subjective hedonic experience of the individual. A subjective state might exist immediately before what came to be known as expressive movements, or these movements could also be executed automatically and without a prior subjectively felt experience. And if Waynbaum is correct, there is necessarily one afterward. For the most part, for example, the onset of withdrawal and alarm reactions often occurs before pain is actually felt. Writhing, weeping, and thrashing of limbs bring relief. Frey (*16*) reported finding enkephalin in tears, which suggests that tears may act to relieve pain. The entire emotional process can therefore be conceptualized as being triggered by an internal sensory or cognitive event that leads to peripheral muscular, glandular, or vascular action that in turn results in a change of the subjective hedonic tone.

In this sense, the facial movements associated with emotion are no different from sneezing, coughing, and yawning. The universality of emotional expression is therefore no more surprising than the universality of yawning and sneezing. All have clear biological bases and can be ascribed to corresponding neuroanatomical structures and neurophysiological processes. What distinguishes them from emotions is that they are regarded as having no psychological instigating causes (although this cannot be always said of yawning). Moreover, being constant and universal, they can readily acquire communicative and symbolic significance.

Empathy. The expressions of basic emotions can be universally recognized by conspecifics through an empathic process. In case of fear expressions, for example, "the eyes and mouth are widely opened, and the eyebrows raised" (*2*, p. 289). Since opening of the eyes and mouth allows greater blood flow into the facial artery, lower CBF, and a more vigorous draining from the brain, a temporary rapid rise in brain temperature is allowed. As a consequence, the organism may experience momentary dysphoria. An animal in close proximity will notice the terrified neighbor because its change in posture is conspicuous. The attention of the neighbor turns into mimicry by virtue of the empathic process, and the neighbor, too, experiences some dysphoria in association with its own altered brain temperature.

Empathy is an important social mechanism whereby the feelings of one individual are transmitted and partially experienced by another. It is not entirely clear how such transmission takes place (*17*). The vascular theory, however, suggests a hypothesis that makes the puzzle of empathy less mystifying. If muscular movements of the face, by virtue of their effects on CBF and on the release of particular neurotransmitters, are sufficient to induce changes in hedonic tone and result in changed subjective states, then reproducing the expression of another may well produce in the onlooker a similar emotional state. Of course, the feeling is not experienced equally. But this might be so only because the movements are not all faithfully reproduced. The question of why should one imitate others' emotional expressions remains, and the answer may come from the theory of evolution. Mimicry has been shown in 11-day-old infants (*18*), and it is generally viewed as part of the innate behavioral repertoire of many species.



Circulatory system of the head. The arteries of interest for Waynbaum's theory are (1) the common carotid, (2) the internal carotid, and (3) the external carotid. [Reproduced from P. Sappey, *Traité d'Anatomie Descriptive* (Delahaye Lecrosnier, Paris, 1888/89)].

Theoretical Consequences of an Expanded Vascular Theory

Once Waynbaum's hypothesis that expressive acts have positive and negative hedonic and subjective consequences is verified, a number of phenomena associated with emotion can be explicated and the theory of emotional processes enriched.

Blushing and pallor. Blushing and pallor are emotional phenomena that are relatively involuntary and uncontrollable. Noting that "women blush much more than men" (*2*, p. 310), Darwin concluded that blushing occurs because attention is focused on part of one's own body. Since blushing occurs "in relation to the opinion of others" (*2*, p. 325), it affects primarily the exposed parts of the body. These vascular phenomena had a different meaning for Waynbaum. Blushing occurs in the case of emotions that are intense, often sending a sudden surge of blood through the main artery. Hence a great deal of energy is deployed without a possibility of efferent output. The person is ashamed, wishes to flee, hide, conceal a previous act, or confound someone's possible deprecating attribution. But in situations that cause blushing, there is little that the individual can usefully do. The mobilized energy has no outlet and, as in suppressed rage, facial blood flow takes up the surplus. Thus, blushing relieves CBF. The face blushes, according to Waynbaum, not because it is exposed but because the facial artery is a branch of the external carotid.

The same is true of pallor. Pallor occurs in temporary cerebral anemia, and the contraction of the facial capillaries remedies it by increasing CBF. These actions are possible because the facial artery is rich in muscular tissue and can contract and expand rapidly. Pallor brought about in pain is caused by the peculiar pain grimace of the major muscles around the mouth and eyes that diminish facial blood flow, and redirect it to the brain to ease suffering. Waynbaum's faith in his theory led him to assert that if the main carotid branched off not at the neck but at the shoulders, we would express our emotions with our arms, and blush with our shoulders.

Facial feedback. The facial feedback theory (*19*) holds that feedback from facial musculature provides information for the subjective experience of emotion. The facial feedback hypothesis differs from Waynbaum's theory in that this feedback only furnishes information about what the individual must have previously felt. Feedback does not cause hedonic subjective experiences—those are assumed to have taken place beforehand.

Ekman *et al.* (*20*) have thrown a light on the problem. Subjects (some of them professional actors) were instructed to form their muscles, one by one, into six classical emotional expressions. The results showed striking changes in heart rate associated with the various expressions, even though the connection between their muscular movements and emotions was not disclosed to the subjects. The muscular pattern that conveys disgust, for example, reduced heart rate, whereas that related to sadness accelerated it. Ekman *et al.* suggested that the activity of the facial muscles triggers the autonomic reactions directly, either by means of facial feedback or through the direct excitation of the hypothalamus by the motor cortex.

Neither the evolutionary theory of emotional expression nor the sensory theory have much to contribute to the facial feedback hypothesis or to the understanding of the Ekman *et al.* data. But the vascular theory may be useful here. To the extent that facial muscles modulate arterial and venous facial blood flow, thereby controlling CBF, heart rate may have to respond to these changes. In expressions of disgust, cerebral blood supply is augmented by increased flow through the internal carotid and reduced draining because the instructed subject places a "tourniquet" on the branches of the facial artery and on the veins around the mouth and forehead. Brain blood, receiving new supply that passed through the cavernous sinus, is thus momentarily cooled. In sad expression, the same facial muscles are relaxed allowing a greater flow in the facial artery, lesser CBF, and more profuse return. Brain temperature is thus allowed to rise since metabolic processes continue while there is no cooling (*21*).

Physiognomics. Since Aristotle, numerous scholars attempted to relate individual differences in temperament to appearance, especially the face (*22*). In fact, much of the early work on emotional expression is closely allied to physiognomics, but because no plausible explanatory principles were proposed, interest in physiognomics died. A reasonable and testable physiognomic theory can be derived from Waynbaum's ideas.

Substantial individual differences exist in facial musculature. For example, in some people the risorius muscle, which extends the angle of the mouth, is absent altogether. If the facial muscles play a significant role in CBF and the resulting neurotransmitter effects, and if muscles differ among people, then individual differences in expression may emerge. If some facial muscle configurations can modulate brain temperature to optimize the action of neurotransmitters that are differentially involved in moods and emotions, and others cannot, then different affective dispositions correlated with facial expression should be found.

This conjecture is strengthened if we consider that voluntary and spontaneous emotional expressions are controlled by different neural pathways (*23*). Spontaneous emotional expressions use the extrapyramidal motor system, whereas "acted" facial expressions receive their impulses from the cortical motor strip, passing through the corticobulbar projections. The social value of a well-functioning voluntary expressive control is as important as spontaneous expression, since both are powerful communicators of internal states and both influence the behavior of others. Inability to smile "sincerely" invites, throughout one's lifetime, social reactions that produce a different personality predisposition than the capacity to break into a "sparkling smile."

Emotional expression and mental health. Since emotions are central phenomena of mental health, the vascular theory of expression can be examined for its usefulness. For example, if brain temperature can influence moods by facilitating the release of particular neurotransmitters, and if facial musculature can modify brain temperature, then thermal biofeedback should be useful in teaching anxious or depressed patients to control the appropriate facial muscles to induce more favorable affective states. Yoga and various forms of meditation are based in part on such an assumption (*24*). The effectiveness of such treatments, however, depends on whether voluntary movements of facial muscles have the same CBF effects as spontaneous ones and if patients can be trained to reproduce the spontaneous ones sufficiently well. Perhaps the study of other affective disorders and vascular diseases may draw upon Waynbaum's theory for new research directions.

Postscript. It remains for historians of science to discover why Waynbaum's original ideas have been so completely ignored for so long. Of course his theory, ostensibly implausible, is not congruent with what is known today about the vascular system, and it was already doubted when Waynbaum first presented it at a meeting of the *Societé de Psychologie Expérimentale et Comparée*. He was then struck down by such rising luminaries as Henri Piéron and Georges Dumas (*25*), the former alluding

to serious physiological difficulties, and both arguing his functional explanations. Yet his basic idea is ingenious and rich in consequences; when modified in the light of what is known and assumed today about these processes, it organizes an array of divergent facts. It therefore deserves to be reclaimed.

References and Notes

1. I. Waynbaum, *La Physionomie Humaine: Son Mécanisme et son Rôle Social* (Alcan, Paris, 1907).
2. C. R. Darwin, *The Expression of the Emotions in Man and Animals* (Univ. of Chicago Press, Chicago, 1965).
3. C. G. Lange and W. James, *The Emotions* (Williams & Wilkins, Baltimore, 1922).
4. R. J. Andrew, *Science* **142**, 1034 (1963). Andrew's interpretation of Darwin's principles has to be taken with care in the light of Darwin's passing disclaimer that there is any muscle that "has been developed or even modified exclusively for the sake of expression" (*2*, p. 354).
5. See, for example, R. Plutchik and H. Kellerman, Eds., *Emotion: Theory, Research, and Experience* (Academic Press. New York, 1980).
6. Note the Lamarckian touch in this interest of Darwin.
7. D. H. Ingvar and J. Risberg, *Exp. Brain Res.* **3**, 195 (1967).
8. D. Minard and L. Copman, in *Temperature: Its Measurement and Control in Science and Industry*, C. M. Herzfeld, Ed. (Reinhold, New York, 1963), vol. 3, part 3, p. 527.
9. K. M. A. Welch, P. J. Spira, L. Knowles, J. W. Lance, *Arch. Neurobiol.* **37**, 253 (1974).
10. R. W. Ross Russell, Ed., *Brain and Blood Flow* (Pitman, London, 1971). Studies that demonstrated increased regional CBF in the motor cortex that corresponds to the action of particular muscle groups used measures that register at best within 6 millimeters of the brain surface; deeper flow dynamics escape these measures. In emotional facial movement, CBF effects may reach deeper into the brain and perhaps touch upon structures such as the limbic system that are involved in emotional processes.
11. M. D. O'Brien, *Headache* **10**, 139 (1971).
12. W. D. Obrist, H. K. Thompson, C. H. King, H. S. Wang, *Circ. Res.* **20**, 124 (1967).
13. D. S. Bell, *Med. J. Aust.* **2**, 829 (1966); R. G. Robinson *et al.*, *Stroke* **14**, 736 (1983).
14. M. Globus *et al.*, *J. Cereb. Blood Flow Metab.* **3**, 287 (1983).
15. T. Piderit, *Grundzuege der Mimik und Physiognomik* (Vieweg, Braunschweig, 1858); P. Gratiolet, *De la Physionomie et des Mouvements d'Expression* (Hetzel, Paris, 1865).
16. W. H. Frey, II, D. DeSota-Johnson, C. Hoffman, *J. Ophthalmol.* **92**, 559 (1981).
17. M. L. Hoffman, in *Emotions, Cognition, and Behavior*, C. E. Izard *et al.*, Eds. (Cambridge Univ. Press, Cambridge, 1984), p. 103.
18. A. N. Meltzoff and M. K. Moore, *Science* **198**, 75 (1977).
19. C. E. Izard, *Human Emotions* (Plenum, New York, 1977); S. S. Tomkins, *Affect, Imagery, Consciousness*, vol. 1, *The Positive Affects* (Springer, New York, 1962); J. D. Laird, *J. Pers. Soc. Psychol.* **24**, 475 (1974); J. T. Lanzetta and S. P. Orr, *ibid.* **39**, 1081 (1980).
20. P. Ekman, R. W. Levenson, W. V. Friesen, *Science* **221**, 1208 (1983). A similar, more extensive experiment, was carried out by M. N. Rusalova, C. E. Izard, and P. V. Simonov [*Aviat. Space Environ. Med.* **46**, 1132 (1975)].
21. Heart rate is not reduced to avoid flooding the internal artery, but rather to decrease arterial pressure outside the brain. When cerebral spinal fluid pressure reaches that of arterial pressure, CBF is generally decreased if that arterial pressure remains constant. With freely varying arterial pressure, the increase of intracranial pressure triggers the Cushing reflex, that is, a conspicuous increase in arterial blood pressure. As a consequence, heart rate is reduced to attenuate arterial blood pressure outside of the brain.
22. M. Stanton, *The Encyclopaedia of Face and Form Reading* (Davis, Philadelphia, 1924).
23. G. H. Monrad-Krohn, *Brain* **47**, 22 (1924).
24. G. E. Schwartz, *Am. Sci.* **3**, 314 (1975).
25. I. Waynbaum, *J. Psychol. Norm. Pathol.* **3**, 467 (1906).

26. Supported by NSF grant BS-8117977. I thank P. Adelmann, L. G. D'Alecy, J. F. Greden, J. T. Hoff, C. E. Izard, and H. Markus for their valuable suggestions, and M. Waynbaum for information about his father.

RESEARCH ARTICLE

Expression, Glycosylation, and Secretion of an *Aspergillus* Glucoamylase by *Saccharomyces cerevisiae*

M. A. Innis, M. J. Holland, P. C. McCabe, G. E. Cole
V. P. Wittman, R. Tal, K. W. K. Watt, D. H. Gelfand
J. P. Holland, J. H. Meade

Glucoamylases (E.C. 3.2.1.3) catalyze the release of glucose from starch and related malto-oligosaccharides (*1*). *Aspergillus awamori* and the related species *A. niger* secrete two glycosylated forms of glucoamylase designated GAI and GAII (*2–4*). Both forms are encoded by a single gene, are immunologically related, and have the same amino terminal amino acid sequence (*4–7*). The two forms differ in the primary structure and length of their carboxyl terminal sequences, and recent evidence suggests that these differences arise as a consequence of differential RNA splicing (*8*). Both enzymes hydrolyze α(1,4)- and α(1,6)-glycosidic linkages, and the larger (GAI) form differs functionally from the smaller (GAII) form only by its capacity to degrade raw starch (*9*).

We have reported the molecular cloning and DNA sequence of the glucoamylase structural gene and a complementary DNA (cDNA) clone for GAI messenger RNA (mRNA) from *A. awamori* (*5*, *6*). The primary structure of the glucoamylase gene and of cDNA clones for GAI and GAII from *A. niger* have also been reported (*7*, *8*). Comparison of the primary structure of the glucoamylase gene with the cDNA clones has established that four intervening sequences are spliced from both the GAI and GAII mRNA's. A fifth intervening sequence is spliced from the GAII mRNA. Analyses of putative regulatory sequences within the 5′- and 3′-flanking regions, as well as within the intervening sequences, of the glucoamylase gene revealed striking similarities to consensus sequences defined for structural genes of *S. cerevisiae* (*6*). Despite these structural homologies, we report here that we were unable to detect either initiation or termination of transcription or proper splicing of the intervening sequences of the *A. awamori* glucoamylase gene in *S. cerevisiae*.

Construction of a yeast expression plasmid and modifications of the glucoamylase gene. A 15-kilobase (kb) Hind III fragment that includes the entire glucoamylase structural gene was introduced into laboratory strains of yeast on an autonomously replicating plasmid, YEp13 (*10*). These transformants had no detectable enzymatic activity, immunoreactive peptide, or glucoamylase mRNA sequences (*11*). Because glucoamylase transcripts were not detected in yeast with the native *Aspergillus* gene, a vector containing yeast regulatory signals was used for the expression of glucoamylase in yeast (Fig. 1). This expression vector (pAC1) contains an *Escherichia coli* origin of replication, the *bla* gene from pBR322, the yeast 2μ origin of replication, and a yeast *LEU2* structural gene. These features of the plasmid permit autonomous replication and selection in *E. coli* and yeast. The vector also contains the promoter and termination regions of one of the yeast enolase

M. A. Innis, P. C. McCabe, G. E. Cole, V. P. Wittman, R. Tal, K. W. K. Watt, D. H. Gelfand, and J. H. Meade are in the Division of Molecular and Biological Research, Cetus Corporation, Emeryville, California 94608. M. J. Holland and J. P. Holland are in the Department of Biological Chemistry, School of Medicine, University of California, Davis 95616.