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AUTONOMIC NEWS

Developments in autonomic research: a review of the latest literature

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Abstract In addition to its homeostatic role, the autonomic nervous system is responsible for generating many of the outward signs of our emotions: our tears, our racing heart, our cold sweat. While mental stress causes an increase in heart rate and sweat release, viewing emotionally charged images—especially negative images—causes a decrease in heart rate. Children with autism spectrum disorder show increases in skin conductance (sweat release) when presented with images of faces, but their capacity to recognize faces is compromised. Conversely, children with Williams syndrome, who are very interested in faces and are socially very engaging, show blunted sudomotor responses to faces and show decreases in heart rate. Many studies have used emotionally charged visual stimuli to induce autonomic responses, and changes in electrical skin conductance as a convenient marker of autonomic arousal, to examine the brain structures involved in emotional processes. Such studies have shown that the amygdala is strongly activated during exposure to fearful images, whereas the nucleus accumbens responds strongly to erotic or romantic images (and is deactivated when exposed to negative images). Given that the nucleus accumbens forms part of the reward circuitry, it is perhaps not surprising that it is activated during that most pleasurable of autonomic acts, orgasm.

Keywords Amygdala · Emotion · Fear · Nucleus accumbens · Orgasm · Pleasure · Skin conductance · Ventromedial prefrontal cortex

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It's just emotion taking me over

We all know that the autonomic nervous system provides many of the physiological markers of emotion, and that certain homeostatic functions have been commandeered for the sake of emotional expression. Apart from the production of tears by the parasympathetic nervous system, a good example is thermoregulation: in many mammals, homeothermic control is brought about by sympathetically-mediated changes in skin blood flow, piloerection and, in horses and humans, sweating. An angry cat will erect its fur; a person in fear will show cutaneous vasoconstriction, sweat release and also (albeit minor) piloerection. Emotional arousal plays an important role in survival, as discussed in the September 2008 issue of *Psychological Science* by Löw and colleagues, from the Center for the Study of Emotion and Attention at the University of Florida, USA [14]. This group examined the changes in heart rate and sweat release while simulating survival in the predator–prey context by using cues that signalled possible monetary rewards or losses: the observed anticipatory response patterns of heightened vigilance led them to conclude that "the physiology of emotion is founded on action dispositions that evolved in mammals to facilitate survival by dealing with threats or capturing life-sustaining rewards." Emotion utilises the same effector organs as used in the classic "fight or flight" sympathetic responses: for example, there is an increase in sweat release (as measured by changes in electrical skin conductance) and an increase in pupil diameter. In the July 2008 issue of *Psychophysiology*, Bradley and colleagues [1], from the same institution, point out that affective changes in pupil diameter when subjects viewed emotionally arousing images paralleled the changes in skin conductance, irrespective of whether the pictures were pleasant or unpleasant, supporting the idea that the sympathetic nervous system modulates these changes in the context of affective picture viewing. What does differ between the arousal and emotional response is the nature of the changes in heart rate: arousal induces an increase, but emotion a decrease. In this same article, the authors point out that viewing pleasant, neutral or unpleasant images causes a decrease in heart rate, but that the cardiodeceleration is more pronounced when viewing unpleasant images. This is further explored by Codispoti and colleagues [5], from the Department of Psychology at the University of Bologna, Italy, in the August 2008 issue of the *International Journal of Psychophysiology*. These authors controlled for the level of arousal and, rather than showing static images, showed films to the participants. They demonstrated that sustained decreases in heart rate, and increases in skin conductance, were induced by viewing pleasant as well as unpleasant films, and concluded that sustained exposure to pleasant and unpleasant stimuli elicit similar cardiac orienting when stimuli are equated for the subjective report of emotional arousal. And in the June 2008 issue of the same journal, Rohrman and Hopp [17], from the Department of Psychology, University of Frankfurt am Main, Germany, examined the nature of disgust by showing films either of an amputation of a person's arm or of a person vomiting. While both films induced strong subjective reports of disgust, increases in skin conductance and cardiovascular reactions, the nature of the cardiovascular responses to the two films differed. Viewing an amputation is a disease-related stimulus of disgust, whereas viewing a person vomiting is a food-related disgust stimulus: only the former induced cardiodeceleration. So, while sweat release is common to different types of emotional reaction, the cardiovascular response varies according to context. Moreover, emotional stress differs from mental stress. This was explored in the December 2008 issue of the *American Journal of Physiology* by Carter and colleagues, from the Department of Exercise Science at Michigan Technological University, USA [2]. They recorded muscle sympathetic nerve activity (MSNA), heart rate and blood pressure in subjects exposed to neutral or negative images, or to mental stress. Only mental stress caused sustained increases in heart rate and blood pressure, but neither mental stress nor viewing emotionally charged images changed MSNA. Evidently, the increase in blood pressure during mental stress was brought about by an increase in cardiac sympathetic nerve activity.

I think, therefore I feel

So, there are differences in the emotional responses to pleasant (positive) and unpleasant (negative) stimuli, but where are the emotional reactions generated in the brain? The amygdala has long been associated with emotional responses and in the December 2006 issue of *Behavioural Neuroscience*, Cheng and colleagues [4], from the Department of Psychology at the University of Milwaukee, USA, used functional magnetic resonance imaging (fMRI) to explore the role of the amygdala in conditioned fear. The authors found increases in signal intensity (reflecting increases in neuronal activity) only in trials when subjects predicted the delivery of an electric shock; these trials were also associated with conditioned increases in skin conductance. While this supports a role for the amygdala in the generation of the autonomic reactions to conditioned fear, a recent meta-analysis of functional imaging studies by Sergerie and colleagues [20] of the Douglas Mental Health University Institute at McGill University, Canada, published in the June 2008 issue of *Neuroscience and Biobehavioural Reviews*, indicates that the amygdala responds to positive as well as negative stimuli, with a preference for faces depicting emotional expressions. Sabatinelli and colleagues [19], from the Center for the Study of Emotion and Attention (University of Florida), report in the September 2007 issue of the *Journal of Neurophysiology* the results of an fMRI study in which subjects viewed images of differing emotional valence. Showing pleasant images of erotic and romantic couples caused robust increases in signal intensity in the nucleus accumbens and medial prefrontal cortex, whereas negative images caused deactivations in these regions. As reviewed by Hänsel and Känel [8], of the Department of General Internal Medicine at University Hospital Berne, Switzerland, in the November 2008 issue of *Biopsychosocial Medicine*, the ventromedial prefrontal cortex plays an important role in the generation of autonomic and affective responses to emotional stimuli. Damage to the ventromedial prefrontal cortex leads to deficits in decision making, and in a review in the December 2008 issue of *Cognition, Affect and Behavioural Neuroscience*, Rudebeck and colleagues [18], from the Department of Experimental Psychology at the University of Oxford, UK, highlight recent research indicating that the orbitofrontal cortex and the anterior cingulate cortex may play distinct but complementary roles in mediating normal patterns of emotion and social behaviour: the orbitofrontal cortex appears to be more involved in simple emotional responses (e.g. fear and aggression) while the anterior cingulate cortex may contribute more to complex aspects of emotion, such as social interaction. Indeed, even mimicking the facial expressions associated with different emotional states (anger, sadness, happiness) can engage these areas. Lee and colleagues [13], from the Institute of Neurology at University College London, UK, report in the September 2006 issue of *Social Cognitive and Affective Neuroscience* that imitating emotional expressions enhanced activity within right inferior prefrontal cortex. Enhanced activity in ventromedial prefrontal cortex and frontal pole was observed during imitation of anger, in ventromedial prefrontal and rostral anterior cingulate cortices during imitation of sadness and in the amygdala, striatum and occipitotemporal cortex during imitation of happiness; these changes did not occur when subjects passively viewed the faces. The insula has been shown to be involved in interoceptive processes and in the integration of sensory, visceral, and affective information, and is believed to contribute to the total emotional experience. In the February 2009 issue of *Brain and Cognition,* Mériau and colleagues [15], from the Berlin Neuroimaging Center at the Charité Hospital in Berlin, Germany, showed that subjects exhibiting a high state negative affect exhibited a greater engagement of the left insula and increases in skin conductance when viewing aversive images, suggesting an increased processing of salient aversive stimuli in these subjects.

I wear my heart on my sleeve

In the June 2008 issue of this column, I reviewed recent research examining the deleterious effects of mental stress on the heart. In the January 2009 issue of *Anxiety, Stress and Coping* Lang and McTeague [12], from the Center for the Study of Emotion and Attention (University of Florida), review recent research that assesses psychophysiological reactivity to fear imagery in anxiety disorder patients. When presented in a fear context a startle stimulus generates a potentiated response, thereby providing a quantitative measure of fearful arousal. When people with specific or social phobia view pictures or imagine the fear-inducing object (a spider, for example) their startle responses are augmented, yet anxious patients with long-lasting comorbid depression show a blunted response, suggesting that normal defensive reactivity may be compromised by long-term mental stress. Social interactions are key to our success, yet a lack of social engagement is the hallmark of people with autism spectrum disorder (ASD). In the November 2008 issue of the *Journal of the International Neuropsychological Society*, Joseph and colleagues [10], from the Department of Anatomy and Neurobiology at Boston University School of Medicine, studied skin conductance responses in children and adolescents with ASD and IQ-matched controls while exposing them to images of faces. Although there were no differences in the skin conductance responses, the amplitude of the responses was inversely related to face-recognition accuracy in the ASD subjects, suggesting that autonomic reactivity to eye contact may interfere with face identity processing in children with ASD. Unlike people with ASD, those with Williams syndrome, exhibit a marked interest in social interaction; they are particularly interested in faces and typically lack social inhibitions, but do show deficits in emotion recognition tasks. In the March 2009 issue of *Social Cognition and Affect Neuroscience*, Plesa Skwerer and colleagues [16], from the same department at Boston University, investigated skin conductance and heart rate responses to images of facial expressions of emotion in adolescents and adults with Williams Syndrome. These subjects were less electrodermally responsive to face stimuli than the age- and IQ-matched learning and intellectual disability group, and showed more cardiac deceleration when viewing emotional faces than the controls. These two examples featured states in which autonomic processing of emotional stimuli was affected by a developmental disorder of the brain, but what about when the autonomic outflow itself is affected, such that the normal markers of autonomic arousal do not occur? This was addressed by Chauhan and colleagues [3], from the Royal Free and University College London Medical School, UK, and reported in the June 2008 issue of *Autonomic Neuroscience*. Going on the premise that empathy for the emotions of others may require "simulatory engagement of corresponding autonomic arousal states" the authors showed that patients with primary autonomic failure showed attenuated scores on an emotional empathy scale, and concluded that disruption of autonomic control does impair the ability to empathize emotionally with others.

La petite mort

No one can deny that orgasm, "the little death", is pleasurable and is a highly coordinated set of autonomic behaviours. A recent series of brain imaging studies have explored changes in brain activity associated with orgasm. Clearly, there are several methodological issues that determine how these studies can be done; not the least being that the bore of a scanner cannot accommodate two people in the throes of passion. Anyway, even with self-induced or partner-induced genital stimulation, orgasm can be induced in these less-than-romantic settings. In the April 2007 issue of *Neuroreport*, Georgiadis and colleagues [7], from the Department of Anatomy and Embryology at the University of Groningen in The Netherlands, used positron emission tomography to examine sites involved in the male orgasm, produced by manual penile stimulation by the subject's partner. The group, extending their 2003 study [9], found activation within the ventral tegmental area, which forms part of the reward circuitry, and ejaculation-related activations in the deep cerebellar nuclei and vermis, parts of the limbic system and ejaculation-related deactivations in the amygdala and throughout the prefrontal cortex; the latter perhaps contributing to the emotional release associated with orgasm. And in the December 2006 issue of the *European Journal of Neuroscience*, Georgiadis and colleagues [6] examined changes in the brain during clitorally-induced orgasm in women. Increases in regional cerebral blood flow occurred in the deep cerebellar nuclei and right caudate nucleus, with profound decreases occurring in the left lateral orbitofrontal cortex, inferior temporal gyrus and anterior temporal pole. The authors suggest that this deactivation in the left lateral orbitofrontal cortex reflects behavioural disinhibition, and that deactivation of the temporal lobe is directly related to high sexual arousal. Interestingly, women with complete spinal cord injury—and loss of somatosensory information from below the lesion—can experience orgasm and, as reported in the 2005 issue of the *Annual Review of Sex Research*, it has been suggested that the intact vagus nerve provides the afferent pathway [11]. Komisaruk and Whipple, of the Department of Psychology at the State University of New Jersey, used fMRI to show that the region of the medulla to which the vagus nerves project (the nucleus tractus solitarius) is activated during mechanical vaginal–cervical stimulation. During orgasm, the authors observed activation within the paraventricular nucleus of the hypothalamus, the amygdala, nucleus accumbens (part of the reward circuitry), hippocampus, basal ganglia and cerebellum, as well as within the anterior cingulate, insular, parietal and frontal cortices. Clearly, the longest parasympathetic nerve, the vagus nerve, has even more talents than we had perhaps given it credit for.

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