

Acknowledgements

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Autonomic Nervous System

Intermediate article

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CONTENTS

Introduction

Anatomy and physiology

Functions of the ANS: homeostasis and allostasis

Emotion, anxiety, and ANS activity

Cognitive interactions with ANS activity

Conclusion

The autonomic nervous system has been viewed as a reflexive system for maintaining internal homeostasis. It is now clear, however, that the autonomic nervous system has reciprocal interactions with higher neurobehavioral substrates, influencing both autonomic control and cognitive/behavioral processes.

INTRODUCTION

The autonomic nervous system (ANS) is the designation applied by John Langley to a complex network of peripheral nerves and ganglia, together with associated regulatory systems of the brain and spinal cord, which serve to control smooth muscles and glands of the viscera (Langley, 1921). Implicit in the term ‘autonomic’ is the view that the ANS is rigidly regulated, and not subject to the

vagaries of ‘volitional’ control like the somatic nervous system. This was even more strongly implied by an earlier name: the involuntary nervous system.

The influential physiologist Walter Cannon argued that the ANS was specialized for what he termed ‘homeostasis’, or the maintenance of stability of the internal fluid matrix, necessary to sustain life (Cannon, 1939). This function was suggested to be implemented by an array of feedback-regulated autonomic reflexes responding to perturbations in visceral states with compensatory adjustments to restore homeostatic balance. An example is the baroreceptor reflexes, whereby an increase in blood pressure, signaled by baroreceptor afferent activity, triggers reflex responses including relaxation of vascular smooth muscle, as well as decreases in heart rate and myocardial contractility that reduce

cardiac output. Together, these responses serve to decrease blood pressure and compensate for the initiating perturbation (Figure 1).

Early research focused on the plethora of autonomic reflexes organized at lower brainstem and spinal cord levels. An important historical development, however, was an emerging recognition that central regulatory systems extended well above these lower levels of the neuraxis. Although many aspects of ANS regulation may be based on simple reflexes, autonomic adjustments are also closely linked to cognitive and behavioral processes that arise from higher neurobehavioral substrates, including the limbic system and cerebral cortex. Another important recognition is the fact that the ANS is as much a sensory system as a motor system. In addition to contributing to lower-level reflexive regulation, visceral afferent information now appears to bias processing in higher neural systems.

ANATOMY AND PHYSIOLOGY

Peripheral Components

In addition to the intrinsic enteric system that is sometimes considered to be part of the ANS, the

ANS consists of two major peripheral divisions, the sympathetic and parasympathetic branches. These two branches have distinct central origins, and differ in their peripheral anatomy and physiology. The sympathetic nervous system has its central origins in the intermediolateral cell column of the thoracic and lumbar divisions of the spinal cord (Figure 1), and so has also been termed the thoracolumbar division. Spinal sympathetic motor neurons give rise to preganglionic efferents which exit the spinal cord in the ventral roots and enter an interconnected set of sympathetic chain ganglia which lie along each side of the cord. On entering the chain ganglia, preganglionic fibers may ascend or descend before terminating on sympathetic ganglion cells, which give rise to postganglionic axons that in turn project to visceral organs. Because of the extensive ganglionic interconnections within the sympathetic nervous system, this division is often considered to discharge as a whole (i.e., in sympathy). We now know, however, that the sympathetic system can exert much more precise and organ-specific actions. The primary neurotransmitter at the ganglionic synapse is acetylcholine (ACh), whereas the postganglionic synapse employs the catecholamine neurotransmitter noradrenaline (norepinephrine). There are some deviations from

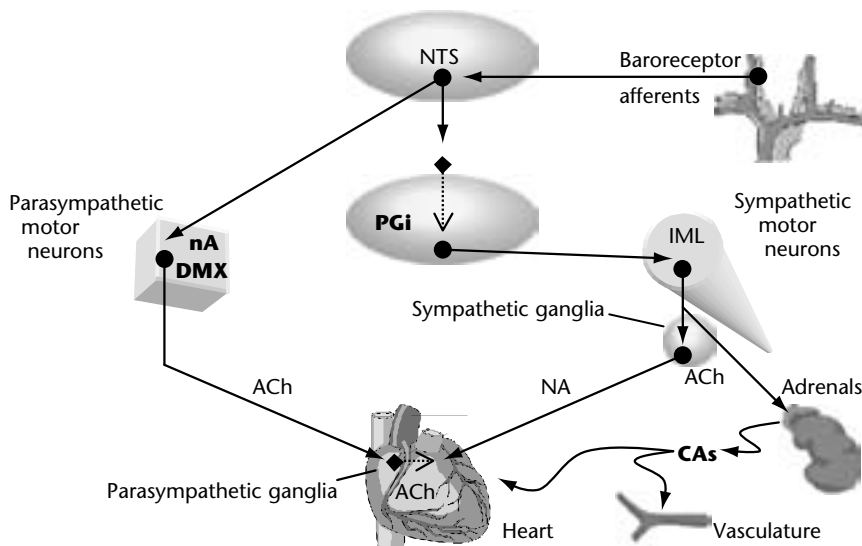


Figure 1. Some features of the baroreflex circuits and peripheral organization of the autonomic nervous system. Baroreceptor afferents project to the nucleus of the tractus solitarius (NTS), by which baroreceptor activity leads to activation of parasympathetic motor neurons in the nucleus ambiguus (nA) and dorsal motor nucleus of the vagus (DMX). The NTS also indirectly inhibits the nucleus paragigantocellularis (PGi) within the rostral ventrolateral medulla, leading to a withdrawal of excitatory drive on the sympathetic motor neurons in the intermediolateral cell column of the cord (IML). ACh, acetylcholine; CAs, catecholamines; NA, noradrenaline (norepinephrine). (Adapted from Cacioppo JT, Tassinary LG and Berntson GG (2000) *Handbook of Psychophysiology*, p. 465, with permission from Cambridge University Press.)

this general anatomical plan, as preganglionic fibers innervate the adrenal medulla directly, without synaptic interruption in the chain ganglia. Hence, sympathetic synapses onto the adrenal medulla release ACh, and the adrenal secretory cells release the catecholamines noradrenal and adrenaline (epinephrine). In contrast to postganglionic sympathetic innervation, however, these adrenomedullary catecholamines are released into the general circulation where they can act humorally on many organ systems, including some that do not receive direct innervation. Because many of the peripheral actions of the sympathetic system are activational and promote energetic metabolism, this division has been considered to be a mobilization system involved in responding to adaptive challenges.

The other branch of the ANS, the parasympathetic division, differs from the sympathetic in its central origin, peripheral anatomy, neuropharmacology, and functions. The lower central motor neurons of the parasympathetic division lie in the intermediolateral column of the sacral spinal cord, and in numerous nuclei within the brainstem (e.g. the nucleus ambiguus, dorsal motor nucleus of the vagus, and salivatory nuclei; see Figure 1). Because of this anatomy, the parasympathetic division has been termed the craniosacral branch, and is also sometimes referred to as the 'vagal' branch, after the vagus nerve (10th cranial nerve) that carries parasympathetic efferents. Strictly, however, the latter term applies only to the vagal component of the parasympathetic branch. Like the sympathetic division, the parasympathetic system includes peripheral ganglia, but these are not collected into coherent ganglionic chains, but rather are generally located in or near the visceral organs innervated. Because of this anatomical difference the parasympathetic system has been thought to be capable of more localized action, although considerable regional specificity can also be demonstrated for the sympathetic branch. As in the sympathetic system, the preganglionic axons of the parasympathetic branch employ ACh as a neurotransmitter (both divisions acting primarily via nicotinic cholinergic synapses on the ganglia); but in contrast to the sympathetic division, postganglionic axons of the parasympathetic system also employ ACh (generally acting via muscarinic cholinergic receptors). This simple distinction in neurochemical coding among the peripheral autonomic branches belies the tremendous complexity of neurotransmitter, neuromodulatory, and neurohormonal interactions within the peripheral ANS.

Opposing versus Synergistic Actions and Modes of Autonomic Control

Many visceral organs are dually innervated by both autonomic branches, and the two divisions are often opposing in their actions. For example, the sympathetic cardiac innervation increases heart rate via β adrenergic receptors that speed the depolarization of the sinoatrial pacemaker potential. In contrast, the parasympathetic innervation slows the beat of the heart via ACh, acting at muscarinic receptors on the sinoatrial node, which increases potassium conductance and slows the rate of pacemaker depolarization. More generally, in contrast to the mobilizing functions of the sympathetic branch, the parasympathetic system has been viewed as a conservation system that functions to promote energy intake, reduce energy expenditure, and preserve energy reserves. Historically, the autonomic branches sometimes have been considered to be subject to reciprocal central control, with increased activity of one division associated with decreased activity of the other (Berntson *et al.*, 1991). Indeed, to the extent to which branches are functionally opposed, the reciprocal mode of regulation would yield the widest dynamic range of autonomic control over target organs (Berntson *et al.*, 1991).

All organs are not dually innervated, however, and it is often the case that actions of the two branches on a given organ are not precisely opposite. Major arterioles, for example, receive only sympathetic innervation, and both sympathetic and parasympathetic activity can stimulate salivary secretion. Even when generally opposing in their actions, as in the control of heart rate, the two branches may operate by different cellular mechanisms with distinct features and temporal dynamics. These differences can lead to distinct functional states, with differing levels of activity of the two branches, which cannot be duplicated by simple variations along a reciprocal bipolar continuum extending from maximal sympathetic to maximal parasympathetic activity. Penile erection and ejaculation, for example, require the coactivation of both autonomic branches.

Higher Central Controls and Neurobehavioral Systems

Many basic autonomic homeostatic reflexes, such as baroreceptor reflexes, do display a reciprocal pattern of control over the autonomic branches. However, the ANS is far from being a simple

homeostatic mechanism controlled by reflex systems of the lower brainstem. Indeed, it is now apparent that autonomic outflow is regulated by neurobehavioral systems at the highest levels of the neuraxis, including the cerebral cortex. Rostral brain systems not only modulate lower reflex mechanisms, but issue descending projections that terminate directly on autonomic source nuclei in the brainstem and spinal cord. In accord with the general increase in flexibility and integrative capacity of higher neural systems, rostral neurobehavioral mechanisms appear to exert more variable and flexible control over autonomic outflows. Consequently, in addition to the reciprocal mode of control often seen in reflex regulation, a wider range of control modes can be observed in behavioral contexts, including independent changes in the autonomic branches as well as coactivation or coinhibition of both divisions.

Autonomic Afferents and Ascending Central Pathways

An important aspect of the autonomic nervous system is its sensory function (Dworkin, 2000). In fact, over 75% of the fibers in the largest autonomic nerve, the vagus, are afferents. Visceral afferents carry a range of information concerning the internal state of the body, from baroreceptors, chemoreceptors, and other interoceptors. Some visceral afferents enter the spinal cord via the dorsal root (along with somatic afferents) and terminate in the dorsal horn, where second- and higher-order neurons may participate in local autonomic reflexes, or relay visceral information to higher central structures. One such structure is the nucleus of the tractus solitarius (NTS), a major visceral relay station in the brainstem (Figure 1). Additional visceral afferents, such as those carried by the vagus and other cranial nerves, terminate directly in the NTS. The NTS is a key structure in brainstem autonomic reflexes and serves as an important relay in ascending pathways to higher levels of the neuraxis where they can modulate the processing of rostral neural systems. Although the functional contributions of this ascending visceral information have not been fully elucidated, it has been shown, for example, that baroreceptor activation can reduce cortical arousal, suppress spinal reflexes, and attenuate pain transmission (Dworkin, 2000). The impact of this ascending information on rostral neurobehavioral mechanisms, and its role in cognitive and behavioral processes, has become an active area of research.

FUNCTIONS OF THE ANS: HOMEOSTASIS AND ALLOSTASIS

A historically recognized role of the ANS is in the maintenance of internal homeostasis. Central and ganglionic autonomic reflex circuits react to perturbations in internal states and generate responses that compensate for these perturbations and restore internal conditions. On standing up from a sitting position, for example, gravitational forces result in a pooling of blood in the legs, which could lead to a dangerous drop in blood pressure and circulatory compromise. This orthostatic challenge becomes a serious problem in autonomic failure and other conditions of impaired autonomic function, where it may lead to syncope (fainting). In healthy individuals, however, this postural maneuver results in the unloading of the carotid baroreceptors and an associated decrease in baroreceptor afferent activity. Baroreceptor reflexes then trigger a compensatory increase in sympathetic outflow and a reciprocal decrease in parasympathetic activity. The resulting increase in heart rate and cardiac output, together with sympathetically mediated vasoconstriction, serves to restore normal blood pressure. This illustrates a classical feedback-regulated (servocontrolled) homeostatic reflex. However, the homeostatic model of autonomic function is overly restrictive, and does not adequately reflect the adaptability and flexibility of autonomic regulation. Feedback-regulated systems, for example, can respond only after a disturbance has taken place, and hence do not effectively prevent these disturbances. Fortunately, central autonomic control systems provide for a broader range of regulatory adjustments, including anticipatory responses.

The early work of Pavlov, demonstrating the conditioning of autonomic responses, foreshadowed current models of autonomic regulation that extend well beyond simple feedback-regulated control mechanisms. Through central associative processes, both exteroceptive and interoceptive stimuli can come to control ANS activity in an anticipatory fashion, and can effectively prevent or minimize perturbations prior to their occurrence (Dworkin, 2000). Conditioned anticipatory autonomic responses, for example, contribute to cardiovascular adjustments to orthostatic stress prior to severe blood pressure perturbations, and trigger anticipatory insulin release prior to the onset of a meal. Specific associations between an innocuous stimulus (conditioned stimulus, CS) that is paired with an aversive event (unconditioned stimulus,

US) can result in conditioned fear reactions to the CS in which preparatory somatic and autonomic responses can be emitted in anticipation of the aversive stimulus or event (Ohman *et al.*, 2000). Similarly, the exaggerated affective and autonomic reactions to a wider (and sometimes poorly defined) range of stimuli and contexts in anxiety states probably reflects a pattern of hyperattentional processing of threat-related stimuli based on a broader and less specific associative structure (Berntson *et al.*, 1998).

Moreover, although central autonomic regulatory systems do contribute to homeostasis, steady state conditions are not always optimal, and central regulatory systems may also promote explicit deviations from homeostasis. Psychological stress, for example, can lead to an inhibition of the baroreceptor heart rate reflex, allowing an increase in both heart rate and blood pressure that might contribute to an adaptive response (see Berntson and Cacioppo, 2000). This is indicative of what has been termed 'allostasis' (McEwen, 1999), rather than homeostasis, and represents a class of autonomic control by which central mechanisms can actively adjust internal states in accord with adaptive demands (see McEwen, 1999; Berntson and Cacioppo, 2000). This is important, as it is not always optimal to maintain homeostatic, steady state conditions. During physical exercise or in the face of a survival threat, for example, there would be considerable adaptive advantage to increasing cardiac output and blood pressure, to enhance blood perfusion of muscles. In such instances, integrative neural systems shift the regulatory set point to a new level (allostasis), rather than maintaining a single homeostatic state. In fact, regulation of autonomic activity is often more dynamic than static, especially in behavioral contexts, so central neurobehavioral systems could more appropriately be considered to exert a pattern of allodynamic regulation over autonomic states (Berntson and Cacioppo, 2000). The construct of allodynamic regulation more appropriately encompasses the complexity and flexibility of central autonomic control and its integration with neurobehavioral function.

EMOTION, ANXIETY, AND ANS ACTIVITY

Autonomic reactions have long been recognized as integral aspects of emotional states such as fear and anxiety, and it is now apparent that there is no clear distinction between higher neural systems underlying behavioral, autonomic, and neuroendocrine

regulation. Increasingly, a close integration of these diverse response domains is apparent, so that behavioral responses, for example, are accompanied by the requisite autonomic, and neuroendocrine support. An illustration of this comes from the broad actions of corticotrophin releasing hormone systems at many levels of the neuraxis, which appear to coordinate and integrate the affective, behavioral, autonomic, and neuroendocrine states underlying stress reactions.

In addition, cerebral cortical areas, including the medial prefrontal cortex, have been implicated in the cognitive aspects of affective states, and have been shown to have relatively direct projections to lower autonomic mechanisms (Berntson *et al.*, 1998). These descending pathways (Figure 2) represent important routes by which rostral neural systems can modulate and regulate ANS activity.

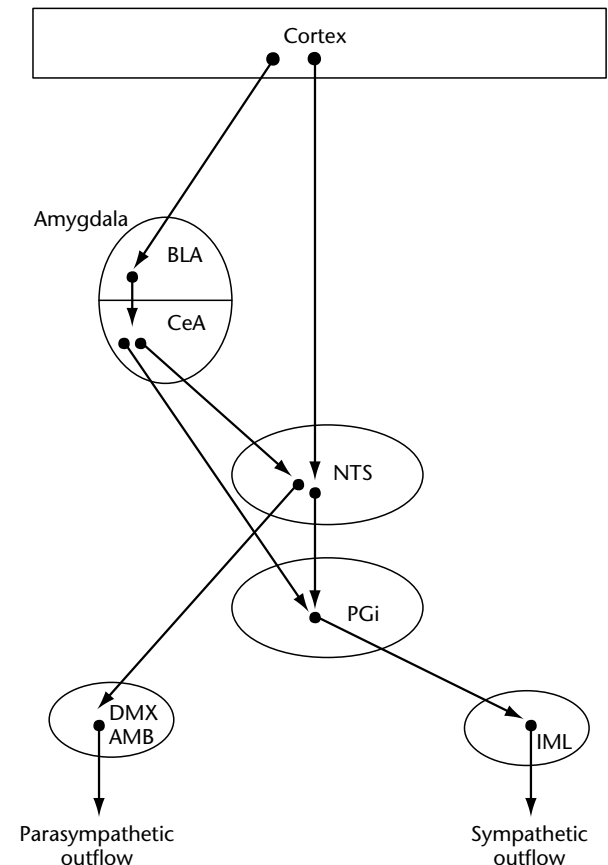


Figure 2. Descending neural systems that affect fear, anxiety, and autonomic control. AMB, nucleus ambiguus; BLA, basolateral amygdala; CeA, central nucleus of the amygdala; DMX, dorsal motor nucleus of the vagus; IML, intermediolateral cell column of the cord; NTS, nucleus of the tractus solitarius; PGI, nucleus paragigantocellularis.

Indeed, autonomic adjustments are ubiquitous in cognitive and behavioral contexts, and these adjustments frequently do not adhere to expectations based on simple metabolic demands. Rather, these autonomic adjustments are likely to reflect an integrated pattern of central control over cognitive, behavioral, and autonomic functions. Consequently, it is not surprising that psychological dysfunctions are often associated with altered autonomic control.

The fourth edition of the *Diagnostic and Statistical Manual of Mental Disorders* (DSM-IV) published by the American Psychiatric Association recognizes altered autonomic activity as a frequent feature of anxiety disorders. However, there is no single autonomic feature that uniformly characterizes anxiety states; indeed, there appear to be considerable differences between the anxiety disorders, and even between individuals within a given diagnostic category (Berntson *et al.*, 1998). This variability attests to the flexibility of autonomic control by higher neural substrates. It remains the case that altered autonomic function is a common accompaniment of anxiety disorders, and may reflect both cause and consequence of these conditions.

The pathways considered above (Figure 2) provide ample routes by which cognitive and behavioral processes can influence autonomic states. Autonomic and neuroendocrine activation in response to stressors serves to mobilize visceral resources in support of the requirements of 'fight or flight', and of the attentional and cognitive demands of adaptive challenges. The stressors of contemporary society, however, do not require – or even allow – a behavioral response of fight or flight, and hence this visceral mobilization may not be readily resolved. Indeed, the metabolic demand for somatic activation imposed by chronic stressors has diminished at the very time that the requirement for visceral support of attentional, cognitive, and behavioral adaptation strategies has increased. Thus, the physiological response to stress that worked well in human evolution may have maladaptive aspects, which become more evident as urban societies develop and life expectancy increases well beyond the reproductive years. Because the somatovisceral mobilizing functions of the autonomic nervous system appear to have evolved to deal with transient challenges (Dworkin, 2000), the prolonged somatovisceral activation associated with chronic psychological stressors in today's society may have long-term health costs (McEwen, 1999). Consequently, the cortical and cognitive processes that underlie this chronic activation are especially important to understand.

COGNITIVE INTERACTIONS WITH ANS ACTIVITY

The descending pathways of Figure 2 represent important substrates for 'top down' regulation of the ANS, whereby cognitive and behavioral states can affect autonomic function. Indeed, cognitive imagery alone is sufficient to trigger autonomic responses. Because cognitive and attentional biases lie at the core of anxiety disorders such as post-traumatic stress disorder and generalized anxiety disorders, these descending pathways probably contribute to the altered autonomic function in anxiety states. The interactions between cognitive and autonomic functions are not one-way, however, and the sensory functions of the ANS may allow autonomic states to bias cognitive processing.

The nineteenth-century psychologist William James proposed that sensory feedback from physiological responses might constitute emotional experience (see Cacioppo *et al.*, 1992). Although it now appears that somatovisceral feedback is not essential for emotional reactivity, this feedback may importantly bias cognitive and emotional processing (see Berntson *et al.*, 1998). Thus, visceral afference, carried by vagal afferents with a relay in the NTS, has been shown to enhance emotional memory in animals (McGaugh *et al.*, 2000), and feedback from autonomic responses may be sufficient to trigger panic attacks in people with panic disorder. More generally, somatovisceral and environmental feedback may have a crucial role in organizing and regulating broad aspects of behavior, affect, and cognition (see, for example, Bechara *et al.*, 2000).

One anatomical route by which this type of 'bottom up' priming may be effected is suggested by work on the nucleus paragigantocellularis (PGi) and the locus ceruleus (LC) in the brainstem (Aston-Jones *et al.*, 1996). These structures could be considered as parts of an ascending visceral afferent system. The PGi receives direct input from the NTS, and modulates sympathetic outflow via descending projections and LC activity via ascending projections (Figure 3). Thus, activity of LC neurons would be expected to be highly responsive to the state of activity of the sympathetic branch. Noradrenergic neurons of the LC in turn issue excitatory projections to the basal forebrain cortical cholinergic system, as well as to the cortex directly.

The ascending pathways of Figure 3 represent a potentially important route by which autonomic activity and associated visceral afference might modulate cognitive and emotional processing. In

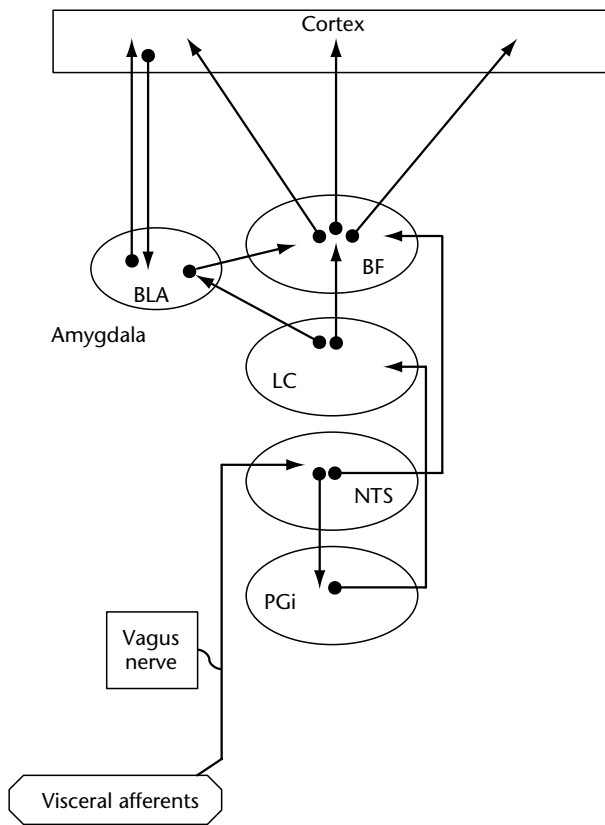


Figure 3. Ascending neural systems that may affect cognitive and emotional processing. BF, basal forebrain; BLA, basolateral amygdala; LC, locus ceruleus; NTS, nucleus of the tractus solitarius; PGi, nucleus paragigantocellularis.

addition to the known descending projections from rostral neurobehavioral systems, this ascending pathway suggests an additional class of interactions between autonomic activity and cortical/cognitive processes. Moreover, the potential for both 'top down' (Figure 2) and 'bottom up' (Figure 3) influences may represent the substrate for a vicious circle of reciprocal cognitive/autonomic priming that might account for the apparent irrationality and exaggerated hyperattentional processing in anxiety states.

Anxiety, Anxiolytics, and the Basal Forebrain Cortical Cholinergic System

Because cognitive processes appear to contribute substantially to the clinical features of anxiety, including the hyperattentional processing of threat-related stimuli, it is not surprising that cortical systems have been implicated in anxiety disorders. An illustration comes from studies on anxiety and the basal forebrain cholinergic system (Berntson

et al., 1998). The widespread corticopetal projections of the basal forebrain provide the primary source of cortical ACh, and ACh appears to enhance cortical processing generally (Sarter and Bruno, 2000). This is illustrated by the global dementia of Alzheimer disease, which is associated with degeneration of the basal forebrain cholinergic system. In view of these considerations, cortical cholinergic activity would be expected to exaggerate the cognitive processing underlying anxiety. This is consistent with the finding that selective immunotoxic lesions of the cholinergic neurons of the basal forebrain in the rat attenuate the exaggerated behavioral and autonomic reactions in anxiogenic contexts (Berntson *et al.*, 1998).

Additional evidence supports this view and explains the antianxiety actions of benzodiazepine receptor agonists such as chlordiazepoxide and diazepam (for a review, see Berntson *et al.*, 1998). The activity of basal forebrain cholinergic neurons is regulated in part by an inhibitory input mediated by γ -aminobutyric acid (GABA), which in turn is bidirectionally modulated through a benzodiazepine binding site on the GABA receptor complex. The anxiolytic benzodiazepine receptor agonists enhance GABA-mediated inhibition and reduce basal forebrain cholinergic activity. Conversely, benzodiazepine receptor inverse or partial inverse agonists, which decrease GABA-mediated inhibition and enhance basal forebrain cholinergic activity, have notable anxiogenic properties (see Berntson *et al.*, 1998). Especially relevant in the link between anxiety and autonomic function may be the medial prefrontal cortex, an area that has been repeatedly implicated in both affect and autonomic control. Selective immunotoxic lesions of the basal forebrain cholinergic projections to the medial prefrontal cortex, like lesions of the basal forebrain itself, eliminate the exaggerated autonomic reactions in anxiety-related contexts (see Berntson *et al.*, 1998).

The ascending pathways in Figure 3 are intended to be representative rather than exhaustive, and there are many important questions that remain in this area. It is increasingly apparent, however, that comprehensive models of cognitive processing or autonomic functions will require a deeper understanding of the reciprocal interactions between these functional domains. In contrast to the Jamesian view that somatovisceral feedback constitutes emotion, the ascending pathway of Figure 3 provides a route by which visceral afference may more subtly trigger or modulate cognitive/emotional processing, even in the absence of conscious perception of the visceral stimulus.

CONCLUSION

Classical views of the autonomic nervous system often focused on brainstem reflexes and the autonomic regulation of internal homeostasis. With the elucidation of higher neurobehavioral influences over autonomic outflow, and the important sensory functions of the ANS, these limited perspectives are no longer tenable. Beyond lower sensory and motor neurons, there is no clear distinction between central systems that regulate behavioral, autonomic, neuroendocrine, and immune processes. Moreover, it appears that the higher the level of neural organization, the greater is the integration among these response domains, culminating in the supreme integrative functions of cerebral cortical systems. Consequently, an understanding of cortical/cognitive processes and the systems and mechanisms that regulate this processing is a prerequisite for a comprehensive model of the autonomic nervous system and its central regulation.

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