6

The Autonomic Nervous System and Functional Somatic Symptoms

Abstract In this chapter we continue our exploration of the neurobiology of functional somatic symptoms by considering the autonomic nervous system. In response to stress, the autonomic system activates into defensive mode to prepare the body for protective action. And when protective action is no longer necessary, the system typically deactivates. But if the child has faced stress that is chronic, uncontrollable, unpredictable, cumulative, recurrent, or overwhelming, the autonomic system may remain activated and represent an ongoing burden on the body. In this chapter we discuss functional somatic symptoms that are the direct expression of the activation or dysregulation of the autonomic system.

We have chosen to discuss the autonomic nervous system next because the functional somatic symptoms related to the stress-related activation of this system are common both in daily life and in clinical practice. Activation and dysregulation of the autonomic system in

Electronic supplementary material The online version of this chapter (https://doi.org/10.1007/978-3-030-46184-3_6) contains supplementary material, which is available to authorized users.

children (including adolescents) has been documented across the full spectrum of functional disorders (see table in Online Supplement 6.1). Therapeutic approaches that target the autonomic system are therefore a cornerstone of treatment (see Chapter 14). More broadly, some approaches to psychotherapy—for example, Peter Levine’s Somatic Experiencing, Patricia Ogden’s Sensorimotor Psychotherapy, or Kathy Kain’s use of therapeutic touch—use tracking and settling/release of autonomic activation patterns as their primary therapeutic tools (Levine 1997; Payne et al. 2015; Ogden and Fisher 2015; Kain and Terrell 2018). And these activation patterns are so important and, to the trained therapist, so tangible that some therapists from the bodywork tradition ‘listen’ only to the story told by the body and *never* ask for the body’s story to be told in words. Understanding the autonomic system and its patterns of activation also enables clinicians to devise therapeutic approaches for infants and preverbal children (Porges et al. 2019).

What that all means for us in clinical practice is that we need to identify and understand the relevant patterns of autonomic activation if we are to make good progress in treating our patients. While this chapter focuses on the body expression of symptoms associated with the autonomic nervous system, it is important to keep in mind that the autonomic system is regulated by the brain (Craig 2005) and that changes in autonomic activation also involve changes in the brain stress systems (see Chapter 11). In the stress-system model, the interdependence between the autonomic system and brain stress systems is represented by the overlapping circles in Fig. 4.2.

On a final note, we hope that as readers make their way through this chapter, they will bring to mind the patterns of activation that their own bodies experience in the face of stress. Understanding their own patterns of activation will enable psychotherapists to be more effective in recognizing them in the children they work with.

The Autonomic Nervous System and Body Regulation

The *autonomic nervous system* relies on electrical signalling by neurons to fine-tune body state second-by-second. Its *afferent*, interoceptive nerves (from the body to the brain) carry information to the brain

about the body's physiological condition (Craig 2003). The autonomic system's *efferent* nerves (from the brain to the body) innervate all the internal organs and tissues of the body, such as the heart, liver, bladder, gut, white and brown fat, immune cells, and connective and other tissues, including the fascia and the smooth muscle within the blood vessels and body organs. All the organs and tissues from inside the body are called the *viscera*. Because the efferent nerves innervate the viscera, including heart muscles and constriction or relaxation of smooth muscle, they are sometimes referred to *visceromotor nerves*.

The autonomic nervous system has two main components: the *sympathetic system* and *parasympathetic system*. The latter includes both a *restorative* and a *defensive* component. The differentiation between the restorative and defensive components of the parasympathetic system is the work of Stephen Porges (2011) (see Online Supplements 1.2 and 6.1). The main nerve containing parasympathetic nerve fibres, both restorative and defensive, is the *vagal nerve*, and for this reason the terms referring to that nerve, including *vagal* and *vasovagal*, are often used to refer to parasympathetic activation. See Fig. 6.1 and following sections for further details.

The Autonomic Nervous System and Attachment Figures

The baseline balance between these three components of the autonomic nervous system is substantially influenced by attachment figures, who function as biopsychosocial regulators (for references see Online Supplement 1.3). In this capacity, sensitive attachment figures help children develop good autonomic regulation characterized by higher restorative parasympathetic activation and lower sympathetic activation. By contrast, attachment figures who are themselves dysregulated are unable to help their children regulate. Stress—whether physical (illness or injury) or psychological (worry, fear, conflict in interpersonal relationships)—has an adverse effect on the autonomic system balance in both attachment figures and their children. Interventions that promote

Fig. 6.1 A simplified functional visual representation of the autonomic nervous system. Afferent signals from the body to the brain provide the brain with interoceptive information about the state of the body (figure on the left). Efferent signals from the brain to the body—involving both the *sympathetic* and *restorative parasympathetic* systems—provide second-by-second fine-tuning of body state (figure on the right). In addition, when needed (as in response to threat), the *sympathetic nerves* (depicted in red) *up* body arousal by increasing heart rate, activating the secretion of adrenalin (from the adrenal glands), adjusting vascular tone, and so on. Likewise, *restorative parasympathetic nerves* (depicted in blue) *down* body arousal (e.g., by decreasing heart rate). The *defensive parasympathetic* nerves (depicted in purple) work alongside the sympathetic system in response to threat by activating defensive programs in the gut and in the heart. Online Supplement 4.4 includes a version of the functional visual representation of the autonomic nervous system that can be printed out (© Kasia Kozłowska 2013)

restorative parasympathetic activation via touch, vocal connection, eye-to-eye contact, and emotional communicating serve to support autonomic regulation in young children (Porges et al. 2019) (Fig. 6.2).

The Autonomic Nervous System Under Conditions of Low Stress and Safety

Under conditions of safety and low stress, the sympathetic and parasympathetic systems work together continually, and in a complementary manner, to regulate body state. For example, in the heart the sympathetic system functions like an accelerator (increasing heart rate when needed), and the parasympathetic system functions like a brake (decreasing heart rate). Similarly, in most situations the sympathetic nerves activate immune-inflammatory cells into defensive mode (when needed), and parasympathetic nerves down-regulate immune-inflammatory cells into restorative mode.

Sympathetic efferent nerves to body organs and tissues innervate, and with the exception of the gut and bladder, usually activate those organs on a second-by-second basis to maintain ongoing bodily functions. Via activation of the adrenal medulla, the sympathetic system also facilitates the release of adrenaline and noradrenaline, thereby enabling the catabolic, energy-expending responses that are needed to maintain body functions.

Restorative parasympathetic efferent nerves also innervate body organs and tissues to regulate body state, but in a manner that complements, and sometimes counteracts, the sympathetic system. Their priority, however, is to efficiently manage key life processes such as digestion (to appropriate energy from food), elimination of waste products, energy conservation, and tissue regeneration and repair, as well as to maintain a body state that facilitates close emotional connection with significant others (Porges 2011; Porges and Carter 2011). For a detailed account of specifically which nerves innervate which organs and tissues, see Wehrwein and colleagues (2016).

Fig. 6.2 Mother and child co-regulating. This figure depicts a mother figure (and her autonomic nervous system), a child figure (and her autonomic nervous system), and the co-regulation between them (© Kasia Kozłowska 2019)

With the exception of an ongoing role in *orienting* to sudden changes in the environment that may represent potential danger—which results in a decreased heart rate—*defensive* parasympathetic efferents are largely offline, waiting in the background to activate in response to threat.

Thus, overall—under conditions of safety and low stress—the sympathetic system has a catabolic effect, causes the release of energy, and is associated with arousal, whereas the parasympathetic system has an anabolic effect, increases saving and storing of energy, and is associated with rest and restoration. Under such conditions, the child is rarely

aware of body changes mediated by the autonomic nervous system. She may notice when her stomach rumbles with hunger, or when her heart thumps and skin sweats after she has been running, or when she blushes and her face heats up. But most of the time the autonomic system just chugs along in the background.

The Autonomic System Under Conditions of Acute Stress, and Episodic Functional Somatic Symptoms

Under conditions of stress or in response to signals of danger, the sympathetic and parasympathetic systems shift into their respective defensive modes:

Sympathetic system (see Fig. 6.3). In response to threat or danger—whether physical, psychological, or both—the sympathetic system revs up. In the body as a whole, increased sympathetic activation raises energy consumption, heart rate, and vascular resistance, all in order to prepare the body for defensive action. Children may consequently experience what they describe as a thumping heart (a high heart rate), sweatiness (activation of sweat glands), and sudden changes in body temperature (changes in dilation and constriction of blood vessels near the skin). In the mouth and gut—as a means of making energy available for more urgent purposes—sympathetic activation disrupts, and potentially entirely shuts down, salivary gland, gastric, and colonic function. Because salivary glands are affected, dry mouth is a common symptom of sympathetic arousal. In some cases of chronic sympathetic arousal, the child feels that the food she swallows is just sitting in the stomach (sometimes described as having what feels to be a rock in the stomach) or that she ‘just can’t eat’. In other cases the child experiences symptoms of constipation. Increased sympathetic activation also affects sleep, with children describing difficulties with sleep initiation and increased arousals in the early hours of the morning (see also Chapter 5).

Parasympathetic system. Stress triggers a response in both the restorative and defensive parasympathetic systems.

Fig. 6.3 A girl in a state of high arousal. The state of high arousal, in which sympathetic activation, including that of the sweat glands in the skin and smooth muscles of the pupils and eyelids, is coupled with skeletomotor activation. The child often describes a thumping heart, sweatiness, feelings of heat on the inside, tightness in the muscles of the body, and an increased breathing rate or a sensation of not getting enough air. The observer may be aware of dilated pupils and a widening of the eyes (© Kasia Kozłowska 2019)

The activity of the *restorative parasympathetic system* decreases. Most importantly, withdrawing the parasympathetic break on the heart increases the heart rate immediately and also frees the sympathetic system to increase the heart rate, if necessary, even more. In addition, withdrawal of restorative parasympathetic activity and activation of the sympathetic activity is associated with lower pain threshold in the gut (i.e., more susceptibility to pain), which creates a risk for developing hyperalgesia (pain responses to stimuli that do not trigger pain in healthy controls) (see Online Supplement 1.3). This is probably why abdominal pain is such a common symptom in young children who experience significant stress and also why adults experiencing severe

stress or anxiety sometimes report that something makes them ‘feel sick to the stomach’.

Altan was a 10-year-old boy in year 4 of primary school. His family had moved house in the middle of the academic year, and he was now attending a new school, where he was the target of teasing from boys in the year above him. Altan began to worry about going to school. In particular, he was worried about being waylaid by the boys at the front gate and teased. At night he had trouble falling asleep (decreased restorative parasympathetic [vagal] activation, increased sympathetic activation, and increased hypothalamic-pituitary-adrenal [HPA] axis activation [see Chapter 8]). In the mornings he was unable to eat and complained of abdominal pain and nausea, and sometimes he vomited (decreased restorative parasympathetic [vagal] activity and activation of defensive parasympathetic [vagal] programs to the gut). Altan’s symptoms melted away when a neighbour suggested that Altan walk to school with her twin boys, who were in year 6. Altan now happily went to go to school feeling safe—and with a settled autonomic nervous system—in the presence of his new buddies.

Alongside the withdrawal of the restorative parasympathetic system, the *defensive parasympathetic system* lowers its set-point, which enables this system to activate its defensive programs for the heart and for the gut and bladder more readily, thereby working in tandem with the sympathetic system to defend the child’s body from threat or danger. Again, it is important to note that these defensive parasympathetic programs are usually offline; they are activated only when the sympathetic system reaches a certain threshold involving severe or imminent threats or danger or when defensive parasympathetic fibres are (reflexly) activated by threat stimuli.

In response to a threat or danger that arises suddenly (including sudden pain or shock), activation of defensive parasympathetic (vagal) nerve fibres to the heart (see Fig. 6.4) can suddenly slow it down (bradycardia) or briefly stop it (resulting in asystole)—which is, in extreme circumstances, itself a defence response (Roelofs et al. 2010). Possible manifestations include dizziness and giddiness (symptoms that occur before a faint) or actual fainting. The most well-recognized expression of this response is blood phobia—that is, fainting in response to the sight of blood (Ost et al. 1984).

Fig. 6.4 Activation of defensive parasympathetic programs to the gut and heart. *Frame A.* Activation of defensive parasympathetic programs to the gut causes activation of nausea and vomiting programs in this child. *Frame B.* Activation of defensive parasympathetic programs (vagal nerve fibres) to the heart causes threat-induced fainting in this child (© Kasia Kozłowska 2018)

In the following vignette of Jean-Luc, we see a man whose fainting episodes started as a child and recurred well into middle age (with potentially still more to come). Jean-Luc, like his mother, suffered from fainting events mediated by activation of the defensive parasympathetic (vagal) nerve fibres to the heart—an activation that was usually, but not always, induced by acute pain.

At 12 years of age, after his mother tapped him on the elbow—on the ulnar nerve—as a reminder of good table manners, Jean-Luc’s eyes rolled back, and he fell off his chair to the floor. At 20, in a science lab, he fainted when his procedure on a live frog (in preparation for dissection) went badly amiss. At 44, he fainted in response to an intense stomach pain in a pub, hitting his head on the counter. Because he was bleeding, and because the pub’s bouncers assumed that he had been causing trouble, he was thrown out onto the street with a warning. At 54, he twisted his leg and fractured his fibula while playing ball with a group of children

at a party. During the sudden sharp intense pain, he fell to the ground, aware that he was losing consciousness. At 55, Jean-Luc was asleep in a hotel in Europe. In the middle of the night he woke up with a growing pain in his stomach from food poisoning or a gastrointestinal bug. As the pain got worse, he got up to go the bathroom to vomit (defensive parasympathetic program). The next part of the story is given in Jean-Luc's own voice: 'I vomit all my guts in the toilet, and it seems to get better. Then a second wave comes, very sharp and intense, and I lean again over the toilet bowl. I don't remember passing out. Next I wake up. My body is stuck between the toilet and the wall in a weird position. Luckily, I did not fall from high and did not hit anything. I don't know how long I have been there, but most probably not more than a few minutes.'

Activation of defensive parasympathetic programs for the gut and bladder manifest as nausea and vomiting (see Fig. 6.4) and as fear-induced faecal or urinary incontinence or simply as an increase in (sudden) bowel motions or urinary frequency, with the consequence that the child complains that she repeatedly needs to run urgently to the toilet to defecate or urinate. Common examples of this mechanism—which many readers will be familiar with—include the feeling of nausea in response to an aversive stimulus/memory or the need to run to the toilet to empty the bladder before public speaking. These defensive programs were developed (via evolution) to empty the gut in case of poisoning or infection, or to rid the body of unnecessary weight when flight was required. The defensive parasympathetic programs to empty the gut can operate at the same time as the sympathetic programs that tend to shut down the gut, so that the child can experience alternating bouts of diarrhoea and constipation.

Chronic Activation or Dysregulation of the Autonomic Nervous System in Children with Functional Somatic Symptoms

Chronic activation or dysregulation of autonomic function is a pervasive feature of functional presentations. It contributes to, and helps maintain, dysregulation of the stress system as a whole and the

associated, nonspecific symptoms of fatigue, nausea, disrupted sleep, sensation of a beating heart, and so on. But in some cases this chronic activation/dysregulation of autonomic function expresses itself in stable, repeating symptom patterns in one or more body systems. Because these symptom patterns are both recognizable and common, they have been given medical names such as irritable bowel syndrome, irritable bladder, and orthostatic intolerance (also known as postural orthostatic tachycardia syndrome [POTS]). For historical terminologies pertaining to autonomic dysregulation—for example, in soldiers from the American Civil War—see Online Supplement 1.1.

The following vignette of Barbara highlights how the concomitant increase in sympathetic activity, decrease in restorative parasympathetic activity, and increase in defensive parasympathetic activity can manifest in chronic gut symptoms.

Barbara was a 13-year-old girl with a two-year history of chronic abdominal pain and nausea, punctuated by episodes of recurrent vomiting that led to dehydration and that brought her repeatedly to hospital. Barbara and her family were refugees. Many of Barbara's relatives in her home village—including her father and older brother—had been threatened by armed militia, sometimes at gun point. Barbara's immediate family fled the village, but the rest of her relatives stayed behind. On arrival to Australia, Barbara had nightmares about the well-being of her aunt, who had helped raise Barbara and her siblings. At night Barbara often woke from her nightmares sweaty and with her heart racing (sympathetic activation). She also began to suffer from recurrent episodes of abdominal pain (changed pain thresholds associated with increased sympathetic, and decreased restorative parasympathetic, activity), leading to frequent visits to the local hospital's emergency department. A year after arrival, she and other members of her family experienced a bout of gastritis that required, in her case, admission to hospital. After the gastritis resolved, she continued to feel nauseous, had trouble eating, and suffered from vomiting episodes (activation of defensive gut programs mediated by the defensive parasympathetic system). Her abdominal pain also spread: she now also suffered from headache and pain in the hip (central pain sensitization/activation of pain maps; see Chapter 11). Her nausea and retching were triggered on a daily basis, whenever it was time to eat a meal (activation of defensive

parasympathetic system and defensive gut programs). She had difficulty maintaining her weight. She missed a lot of school. When Barbara experienced any emotional stress or distress—for example, when she had a falling out with a friend, when her father was unwell, or when there was conflict with a teacher—her symptoms (pain, nausea, and vomiting) would be triggered, and she would present to the emergency department in a dehydrated state. (For other factors that could have contributed to Barbara's presentation, see Chapter 10 about the microbiota-gut-brain axis.)

The following vignette of Carmen highlights how the loss of a parent can lead to chronic autonomic dysregulation presenting as fainting episodes. We can hypothesize that the sudden death of Carmen's father led to significant distress and concomitant autonomic activation—a withdrawal of restorative vagal activity, plus sympathetic arousal—as well as a changed set-point for activation of the defensive parasympathetic system (defensive vagus), resulting in more frequent activation of the defensive parasympathetic system (defensive vagus) and frequent fainting (vasovagal) events. Previously for Carmen, such events had been episodic—for example, in response to the sight of blood.

Carmen was a 14-year-old girl from a middle-class family. Prior to the death of her father in a motor vehicle accident, Carmen had occasionally fainted when standing in the heat. Having her blood taken or seeing the needles used for vaccination also made her queasy, and in a few instances she had fainted. Following her father's death, Carmen's distress was extreme, and she thought of her father frequently. At school she began to have almost daily episodes of fainting (activation of the defensive parasympathetic fibres [defensive vagus] to the heart). Her friends reported that she would turn white, drop to the ground, and recover herself soon after. On several occasions she sustained significant injuries to her arms and head. Carmen has no memory of the faints.

Autonomic dysregulation manifesting as orthostatic intolerance (or POTS) expresses itself via physical symptoms of dizziness, giddiness, palpitations, lightheadedness, near-fainting, and fainting on standing up from a reclined or sitting position. It involves too little restorative parasympathetic (vagal) activity (allowing heart rate to increase), too

much sympathetic activity (through which heart rate increases even more), and no change in blood pressure (Stewart 2012; Wells et al. 2018) (for details about POTS and additional references, see Online Supplement 6.1). Orthostatic intolerance is often accompanied by nausea—or even abdominal pain or vomiting—reflecting concomitant activation of defensive gut programs by the defensive parasympathetic system. It is frequently comorbid with other functional somatic symptoms or syndromes. Because the autonomic nervous system is very sensitive to stress, orthostatic intolerance is a common consequence of physical or psychological stress, such as surgery, a viral illness, gravitational deconditioning (too much bed rest), puberty (growth spurt or, for girls, the onset of menstruation), a traumatic event, cumulative adverse life events, or ongoing distress in the context of family conflict.

In the following vignette of Ines, we see orthostatic intolerance in a young girl with non-epileptic seizures and social anxiety. The standing test mentioned as part of the vignette is done on waking. The child stands for ten minutes with heart rate and blood pressure being taken at one-minute intervals. A heart rate increase of ≥ 40 beats per minute (accompanied by symptoms) without a significant lowering of blood pressure gives a good indication of orthostatic intolerance.

Ines was a 13-year-old girl living with her parents and her older sister. Ines was bullied throughout primary school, which she had managed by avoiding close friendships. She also had a history of fainting if she stood too long in the heat. When Ines was in high school, her sister was assaulted. In the aftermath of the assault, the sister became depressed and began to self-harm. Ines worried about her sister: her mind produced vivid images of her sister being assaulted, being carved up with a knife, and dying from suicide. Ines presented with non-epileptic seizures—zoning-out events lasting minutes, plus periods of collapse and unresponsiveness lasting minutes to hours. She also reported nausea, wobbliness, fatigue, and dizziness. The latter cluster of symptoms typically manifested on standing. Sometimes she also had diarrhoea. During a ten-minute standing test, Ines was found to have a high baseline heart rate of 90 (>75th percentile) that increased to 160 during the test, with no drop in blood pressure, all consistent with orthostatic intolerance and a diagnosis of POTS.

Orthostatic intolerance is also frequently encountered in chronic pain patients secondary to a loss of physical conditioning that has resulted from a lack of exercise:

Orthostatic intolerance was confirmed in Paula, the bed-bound 15-year-old girl with chronic pain and recent onset of functional neurological symptoms (leg weakness and functional blindness), whom we met in Chapters 2 and 3. Immediately upon waking in her hospital bed, Paula would begin catastrophizing about her pain, about feeling sick, and about going to the hospital school. Her heart rate would range around 85, and her respiratory rate around 25. On a standing test, Paula's heart rate rose from 84 beats per minute to 146 beats over six minutes, with no significant drop in blood pressure (see Table 3.1). Paula felt sick and lightheaded; her respiratory rate increased; and her panic increased. It increased again when she was taken to school, culminating in a panic attack.

Another pattern of autonomic dysregulation is overactive bladder, manifesting as urinary frequency or urgency, which is thought to involve too much (defensive) parasympathetic activity coupled with too little sympathetic activity (Aydognmus et al. 2017). (For a discussion of urinary retention, see Chapter 7.)

Ghani was a 12-year-old girl who spent one month in hospital, confined to her bed with pneumonia. When Ghani was discharged home, she continued to experience symptoms of fatigue, dizziness, and intermittent headaches, and she had difficulties concentrating on her schoolwork and could manage only half days at school. She also developed urinary frequency and felt like she needed to go to the toilet all the time. All urology tests were normal. A standing test showed that when Ghani stood up from a resting position, her blood pressure was stable, but her heart rate increased by 69 (90 to 159) beats per minute (reflecting significant sympathetic activation on standing). A hyperventilation challenge showed chronic hyperventilation (see Chapter 7). The diagram of the autonomic nervous system (see Fig. 6.1) was used to explain that the long period in bed—because of the pneumonia—had contributed to dysregulation of the autonomic system, that this dysregulation was causing Ghani's irritable bladder symptoms and her dizziness on standing, and that it was

contributing to her symptom of fatigue. To help regulate her autonomic nervous system and switch down her respiratory motor system, Ghani added breath training to her home rehabilitation/recovery program.

As noted earlier, Online Supplement 6.1 presents a table summarizing paediatric studies showing autonomic dysregulation in relation to wide-ranging functional problems, including chronic pain/chronic abdominal pain, irritable bowel, chronic fatigue, functional neurological disorder, and disturbed sleep.

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We hope that after reading this chapter, the mental health clinician working with children will be better able to identify and track patterns of activation in the body—both by asking the child and family better questions, and by observing activation patterns in the body itself. We are confident that the clinician will discover that a large number of child patients suffer from symptoms that pertain to activation or dysregulation of the autonomic nervous system (see vignettes scattered through this chapter). The child, in turn, will find it very reassuring to know that the clinician understands the language spoken by the body, understands the manner in which the body is causing the symptoms, and knows how to implement strategies that can help the child settle her body and, as in Chapter 3's vignette of Paula, enable the symptoms to melt away.

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