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Functional Somatic Symptoms in Children and Adolescents: The Stress-System Approach to Assessment and Treatment

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Online Supplement 7.1

The Skeletomotor System, Cellular Respiration, Hyperventilation, and Fascia

In this supplement to Chapter 7, we provide the reader with additional information about the skeletomotor system: the names of nerves innervating different muscles in the body; non-medical names used when referring to activation of muscle; and muscle spindles. We also discuss the respiratory motor system and cellular respiration, the neurobiology of hyperventilation, the trapezius muscle as a highly stress-responsive muscle, and the mechanics of belching, rumination/regurgitation, and globus sensation. Finally, we provide the reader with interesting references pertaining to fascia.

Terminology Pertaining to the Innervation of Different Muscles in the Body: Somatomotor Nerves, Brachiomotor Nerves, and Visceromotor Nerves

Somatomotor and Branchiomotor Nerves

As we saw in Chapter 7, anatomists refer to the motor nerves that innervate the skeletomotor muscles as *somatomotor nerves*. The word *soma* comes from

the Greek and means *body* (or *body proper*). In this way anatomists distinguish nerves that innervate skeletal muscle (somatomotor nerves) from nerves that innervate the viscera (visceromotor nerves).

In actual fact the anatomy of how skeletomotor muscles are innervated is more complicated than this, and we provide a brief summary here. *Somatomotor nerve fibres* target striated muscles derived from somites in the embryo. These striated muscles are often referred to as skeletal muscles. They typically attach to bone and enable us to move the body. Somatomotor innervation of skeletal muscles is direct from the central nervous system to the muscle (no ganglion). The tongue muscles and oculomotor muscles of the eye are also derived from somites.

Branchiomotor nerve fibres target other striated muscles found in the head, middle ear, larynx, pharynx, and neck (sternocleidomastoid muscles and trapezius) – muscles controlling facial expression, mastication, and head movements. Branchiomotor nerves are derived from the branchial arches in the embryo. Branchiomotor innervation of muscles controlling facial expression and head movements is direct from the central nervous system to the muscle (no ganglion). This distinction is important in clinical practice because *somatomotor nerves* are involved in striated muscles that move the body proper, whereas *branchiomotor nerves* are involved in the movement of the head and neck, as indicated above, and play a key role in interpersonal communication (Porges 2011).

Visceromotor Nerves

From the simple functional perspective used across all chapter of the book, the motor nerves of the autonomic system include sympathetic, restorative parasympathetic, and defensive parasympathetic nerves (see Chapter 6). These nerves innervate the internal organs of the body – the viscera – including both the smooth muscles that line the blood vessels that regulate blood flow to skeletal muscles and the inner part of the adrenal gland that releases hormones (adrenalin and noradrenalin) to increase body arousal.

The word *viscera* comes from the Latin word *viscus/viscera*, meaning organ(s). The key muscle type innervated by the autonomic system within

the viscera is smooth muscle. The only exception is heart muscle – a special type of striated muscle (see below). Anatomists refer to the motor nerves that innervate the viscera as *visceromotor nerves*. *Visceromotor nerve fibres* target all smooth muscle in the viscera, which derive from the mesoderm (though some smooth muscles also come from ectoderm). *Visceromotor nerves* include a relay in an autonomic ganglion (both sympathetic and parasympathetic). The ganglion lies between the central nervous system and the muscle itself. In this way, the term *visceromotor system* refers to the efferent component of the autonomic nervous system – *efferent sympathetic and parasympathetic nerves* – and the term *visceromotor activation* refers to activation of efferent *sympathetic and parasympathetic nerves*.

The *heart muscle tissue* – which is a unique type of muscle – looks striated under the microscope like skeletal muscles. Like smooth muscle, however, its innervation is autonomic (with a ganglion lying between the central nervous system and the muscle itself), and it derives from the mesoderm.

Nerves Can Carry Fibres Originating from Different Parts of the Brain

The anatomy of nerves themselves is also complex; nerve fibres originating from different parts of the brain can run within the same nerve. For example, the vagus nerve is a mixed nerve that carries both visceromotor and branchiomotor nerve fibres. Consequently, just because a structure is innervated by the vagal nerve does not mean it is innervated by vagal visceromotor fibres only. In other words, vagal innervation does not equal parasympathetic innervation, a mistake that is often made when anatomical ideas are oversimplified for non-medical audiences.

Likewise, many cranial nerves are mixed nerves that carry both branchiomotor and visceromotor nerve fibres. For example, the facial nerve carries branchiomotor nerve fibres that provide motor innervation of facial muscles responsible for facial expression, sensory nerve fibres carrying taste sensation from the anterior two-thirds of the tongue, and visceromotor

(parasympathetic) nerve fibres to the salivary glands of the oral cavity and lacrimal gland.

What is important from the clinical perspective is that the skeletomotor (both somatomotor and branchiomotor) and the visceromotor (autonomic) systems – are coupled systems that work together in a coordinated way. The somatomotor and visceromotor systems work together to enable the body to move and to address, through action, the challenges of daily life. The branchiomotor and visceromotor systems work together to enable movements that support feeding, vocalizing, and interpersonal communication via facial expressions of emotion (Porges 2011; Pace-Schott et al. 2019). When therapists who work with the body (Levine 1997; Payne et al. 2015; Ogden and Fisher 2015; Kain and Terrell 2018) notice activation, they are usually noting interconnected changes in arousal/visceromotor/autonomic activation, in skeletomotor/branchiomotor activation, and in fascia tension.

Non-medical Terminologies Used for Activation of Muscles: Bracing, Tension Patterns, Constriction, and Contraction

The terms *bracing*, *tension pattern*, *constriction*, and *contraction* are often used by therapists working from bottom-up therapy models such as Peter Levine's somatic experiencing. For example, Levine writes that 'most people can duplicate the physical posture, the muscle contractions, and the movements that accompany hyper-arousal to some degree, though generally not with the same level of coordination and synchronicity that accompanies the real thing' (Levine 1997, p. 134). Likewise, Levine and Maggie Phillips (2012, p. 19) write, 'Once the fear response is activated, the body begins to brace to protect itself against the threat. For example, our arms may instinctively rise up to protect our heads. Bracing can trigger chronic constriction if it is not released. And if this constriction persists, it creates pain.'

In working with the breath, a *constricted breath* or *constricted breathing pattern* would be one in which the patient does not fully utilize the diaphragm and depends mainly on the intercostal and supraclavicular muscles. This

terminology for different ways of breathing is actually metaphorical, however; it reflects subjective experience, or the patient's felt sense, and not the anatomical process itself, which may involve contraction in some muscles and relaxation in others. For example, while the patient who takes a big full breath feels a sense of *opening*, the actual anatomical process involves constriction of the diaphragm, which results in its centre moving downward and its edges moving upward. Other patterns of constriction are sometimes more closely aligned with anatomical increases in muscle tone or the contraction of certain muscle groups. For example, bracing often involves activations of muscles involved in posture. That said, contraction patterns may involve complex patterns of muscle contraction and relaxation (see, e.g., section on rumination, below).

Muscle Spindles (Stretch Receptors or Proprioceptors)

Overactivation of muscles spindles (also known as *stretch receptors* or *proprioceptors*), which detect the amount and rate of stretch in skeletomotor muscle, has been implicated in chronic pain presentations involving musculoskeletal pain. Muscle spindles are made up of special muscle fibres (called *intrafusal muscle fibres*) that sit in a sheath made of fascia. They are also hypothesized to play an important role in pain associated with myofascial trigger points, taut bands of muscle that are painful on palpation (Partanen 2017). Alternatively, it may be that pain receptors located in the fascia surrounding the muscle spindles play an important role in musculoskeletal – or musculo-facial-skeletal – pain (see section on fascia, below).

Additional references about muscle spindles include Lund and colleagues (2010) and Ribot-Ciscar and colleagues (2000).

The Respiratory Motor System and Cellular Respiration

Cellular respiration uses oxygen and produces carbon dioxide. Oxygen (O₂) is needed for cellular respiration, a process by which carbohydrates, fats, and amino acids are metabolized to produce energy. Carbon dioxide (CO₂), a waste product of cellular metabolism, diffuses into the blood and is transported to the lungs, where it diffuses into the air in the lungs and is expelled from the body by the breath (expiration). In this way, respiratory rate is tied in with energy consumption, and the body uses respiration to maintain the blood level of CO₂ in homeostatic limits. When energy demands increase, O₂ demand increases, CO₂ production increases, and respiratory rate increases, to meet the increase in O₂ demand and to eliminate more CO₂ via the lungs. When energy demands decrease, O₂ demand decreases, CO₂ production decreases, and respiratory rate decreases. In this way, the arterial levels of both O₂ and CO₂ are kept within a homeostatic range, a range that supports life and that is associated with optimal function in the body and brain. CO₂ levels drive the respiration rate via changes in blood pH (acidity/alkalinity level), which is sensed by chemoreceptors in the brain (Brinkman and Sharma 2018). High levels of CO₂ make the blood acidic, and low levels make the blood alkaline.

The Respiratory Motor System and the Neurophysiology of Hyperventilation

This material pertaining to *the respiratory motor system and the neurophysiology of hyperventilation* was originally published in a text box that made up the online materials to an open access article entitled ‘The Respiratory Control of Carbon Dioxide in Children and Adolescents Referred for Treatment of Psychogenic Non-epileptic Seizures,’ in the journal *European Child & Adolescent Psychiatry* (Kozłowska et al. 2017).

Hyperventilation, breathing in excess of metabolic demands, causes a broad range of neurophysiological changes, in both the central and peripheral nervous systems.

Central Changes

Phase 1

Hyperventilation involves an initial excitatory phase, with an increase in cortical excitability, that is followed in some individuals by a hypoxic phase, with a decrease in cortical function. Excitatory changes include increased cell membrane permeability, increased metabolism and oxygen consumption, hypo-polarization of neurons, increased cortical excitability in widely distributed networks (including motor and visual cortex) (Sparing et al. 2007; Jensen et al. 2002; Carbon et al. 2000; Stenkamp et al. 2001), and decreases in intracortical inhibition (Sparing et al. 2007). These initial changes that occur with hyperventilation are similar to those seen with sympathetic nervous system stimulation and mild-moderate activation of cortical arousal systems.

Phase 2

In some individuals, hyperventilation may continue, producing an increase in cerebral hypoxia and consequent decrease in cerebral function. The relevant cascade of changes include the following: lowered arterial CO₂ and an increase in arterial pH (alkalosis) (Blinn and Noell 1949; Engel et al. 1947); cerebral artery vasoconstriction (Gibbs et al. 1942; Hauge et al. 1980; Kraaier et al. 1988) and decreased blood flow (Yamaguchi et al. 1979; Gibbs 1992; Yamatani et al. 1994; Ball and Shekhar 1997); increased binding of oxygen to haemoglobin (Bohr effect) (Bohr et al. 1904); decreased brain tissue oxygenation and cerebral oxygen metabolism (Yang et al. 2015; Meng et al. 2012); and increased glycolysis with production of lactic acid by neurons (Siesjo and Kjallquist 1969). The cerebral cortex and basal ganglia – which mediate changes in consciousness – are the brain regions most sensitive to hypoxia, followed by the hypothalamus and midbrain, with the medulla and pons being most resistant, leaving their programs available for activation even in states of hypoxia (Gastaut 1974). With hyperventilation, and in contrast to adults, children and adolescents show more pronounced decreases in cerebral blood flow (Yamatani et al. 1994; Yamaguchi et al.

1979) and more pronounced hypoxia-related EEG slowing (Gotoh et al. 1965; Gibbs et al. 1943; Son et al. 2012). The latter is associated with changes in cognitive processing and with altered awareness and responsiveness including states of presyncope or syncope (Okel and Hurst 1961; Allen and Agus 1968; North et al. 1990; Epstein et al. 1994; Barker et al. 2012). In healthy adults, central symptoms are more likely if PCO₂ falls to 20 mm HG (range, 14–29 mm HG; SD, 3–4 mm HG) (Rafferty et al. 1992).

Peripheral Changes

Peripheral neurological effects of hyperventilation include hypopolarization of neurons and increased excitability of sensory and motor axons in the peripheral nervous system – which can cause increased muscle excitability, paraesthesias, and carpopedal spasms (Macefield and Burke 1991). Cardiac effects include increased contractility and increased oxygen extraction (Laffey and Kavanagh 2002), as well as vasoconstriction of coronary arteries, potentially inducing hyperventilation-induced chest pain (see Kozłowska [2013] for review). When hyperventilation continues without inducing significant hypoxia, some individuals experience paraesthesias and carpopedal spasms (anaesthesia and tetany seem not to develop once hypoxia sets in) (Engel et al. 1947; North et al. 1990). For more detail about hyperventilation-induced NES, see Kozłowska and colleagues (2018a,b).

The Trapezius as Another Highly Stress-Responsive (Postural) Muscle

A body of work has identified that ‘the trapezius muscle increases activation in the face of a stressor and is considered to be a highly stress-responsive muscle’ (Helou et al. 2018, p. 1526; Lundberg et al. 2002). A significant number of individuals respond to stress with an increase in sympathetic arousal coupled with activation of the laryngeal muscles, of the postural muscles in the neck, shoulders, back, and legs (e.g., activation of the

trapezius and the anterior tibialis in the calf), or of various combinations from these muscle groups (Helou et al. 2018; Kozłowska et al. 2015).

The Mechanics of Belching, Rumination/ Regurgitation, and Globus Sensation

Belching is the act of bringing up air from the stomach. When we belch, the upper oesophageal sphincter (striated muscle), lower oesophageal sphincter (smooth muscle), and crural diaphragm (striated diaphragm muscle around the lower oesophageal sphincter) all relax, and the costal diaphragm (striated muscle toward the ribs) and abdominal wall muscles (striated muscle) contract repetitively to increase pressure within the stomach, resulting in the expulsion of gas from the stomach through the oesophagus. In some individuals this action pattern can be activated habitually in response to a physical or emotional stimulus.

A similar pattern of skeleto- and visceromotor activation can be mobilized to bring up food back into the mouth – termed *rumination* or *regurgitation*. Rumination is thought to involve a belch-like response pattern in which gastric pressure is increased from repetitive diaphragmatic contractions while the lower and upper oesophageal sphincters are relaxed, to enable regurgitation of food from the stomach into the mouth (for video, see <http://bit.ly/2HxCcS2>) (O'Brien et al. 1995; Murray et al. 2019). In some individuals this action pattern can be activated habitually and begins to interfere with their daily lives.

Recent studies suggest that globus sensation unrelated to organic pathology may likewise involve changes in the tone of the oesophageal muscle (striated muscle in the upper part of the oesophagus, and smooth muscles at the lower part) (Manabe et al. 2014). In other words, in states of high arousal (visceromotor activation), some children may be prone to an action pattern that involves increased tone or dysregulation of skeletal and smooth muscle function – innervated by skeletomotor and visceromotor nerves, respectively – in the oesophagus, accompanied by a globus sensation. This action pattern produces an uncomfortable feeling in the throat.

For references on for belching disorders, see Sun and colleagues (2015); for rumination syndrome, see Murray and colleagues (2019); and for globus sensation, see Manabe and colleagues (2014).

Fascia

For a popular article in the *Washington Post* about fascia, see ‘Everywhere in your body is tissue called fascia. Scientists are unlocking its secrets’, by Rachel Damiani and Ted Spiker (2019).

For an atlas of the human fascial system – some of which atlas is available online – see Stecco (2015).

For a discussion about *dermatome* versus *fasciatome* – relevant to chronic/complex pain presentations – see Stecco and colleagues (2019).

Other interesting articles include Jantos (2011, forthcoming 2020), Stecco (2016), Bordoni and Simonelli (2018), Bordoni and colleagues (2018), and Jantos and Stecco (forthcoming). The second edition of *Fascia: The Tensional Network of the Human Body* (2012), edited by Robert Schleip and colleagues, is scheduled to be published in 2021.

References

- Allen, T. E. & Agus, B. (1968). ‘Hyperventilation Leading to Hallucinations’. *American Journal of Psychiatry*, 125, 632–637.
- Ball, S. & Shekhar, A. (1997). ‘Basilar Artery Response to Hyperventilation in Panic Disorder’. *American Journal of Psychiatry*, 154, 1603–1604.
- Barker, A., Ng, J., Rittey, C. D., Kandler, R. H. & Mordekar, S. R. (2012). ‘Outcome of Children with Hyperventilation-Induced High-Amplitude Rhythmic Slow Activity with Altered Awareness’. *Developmental Medicine and Child Neurology*, 54, 1001–1005.
- Blinn, K. A. & Noell, W. K. (1949). ‘Continuous Measurement of Alveolar CO₂ Tension During the Hyperventilation Test in Routine Electroencephalography’. *Electroencephalography and Clinical Neurophysiology*, 1, 333–342.

- Bohr, C., Hasselbalch, K. & Krogh, A. (1904). 'Über Einen in Biologischer Beziehung Wichtigen Einfluss, den die Kohlendioxidspannung des Blutes auf Dessen Sauerstoffbindung Übt [Concerning a Biologically Important Relationship – the Influence of the Carbon Dioxide Content of Blood on Its Oxygen Binding]'. *Skandinavisches Archiv für Physiologie*, 16, 401–412.
- Bordoni, B., Marelli, F., Morabito, B., Castagna, R., Sacconi, B. & Mazzucco, P. (2018). 'New Proposal to Define the Fascial System'. *Complementary Medicine Research*, 25, 257–262.
- Bordoni, B. & Simonelli, M. (2018). 'The Awareness of the Fascial System'. *Cureus*, 10, e3397.
- Brinkman, J. E. & Sharma, S. (2019). 'Physiology, Respiratory Drive'. In: *Statpearls* [Internet], Treasure Island, Florida, Statpearls Publishing. <https://www.ncbi.nlm.nih.gov/books/nbk482414/>
- Carbon, M., Wübbeler, G., Trahms, L. & Curio, G. (2000). 'Hyperventilation-Induced Human Cerebral Magnetic Fields Noninvasively Monitored by Multichannel "Direct Current" Magnetoencephalography'. *Neuroscience Letters*, 287, 227–230.
- Damiani, R. & Spiker, T. (Jan. 27, 2019). 'Everywhere in Your Body Is Tissue Called Fascia. Scientists Are Unlocking Its Secrets'. *Health & Science, Washington Post*.
- Engel, G. L., Ferris, E. B. & Logan, M. (1947). 'Hyperventilation: Analysis of Clinical Symptomatology'. *Annals of Internal Medicine*, 27, 683–704.
- Epstein, M. A., Duchowny, M., Jayakar, P., Resnick, T. J. & Alvarez, L. A. (1994). 'Altered Responsiveness During Hyperventilation-Induced EEG Slowing: A Non-epileptic Phenomenon in Normal Children'. *Epilepsia*, 35, 1204–1207.
- Gastaut, H. (1974). Syncope: Generalised Anoxic Cerebral Seizures. In: Magnus, O. & Lorentz De Hoos, A. M. (eds.) *The Epilepsies. Handbook of Clinical Neurology*, Amsterdam, New Holland.
- Gibbs, D. M. (1992). 'Hyperventilation-Induced Cerebral Ischemia in Panic Disorder and Effect of Nimodipine'. *American Journal of Psychiatry*, 149, 1589–1591.

- Gibbs, E. L., Gibbs, F. A., Lennox, W. G. & Nims, L. F. (1942). 'Regulation of Cerebral Carbon Dioxide'. *Archives of Neurology and Psychiatry*, 47, 879.
- Gibbs, F. A., Gibbs, E. L. & Lennox, W. G. (1943). 'Electroencephalographic Response to Overventilation and Its Relations to Age'. *Journal of Pediatrics*, 23, 497–505.
- Gotoh, F., Meyer, J. S. & Takagi, Y. (1965). 'Cerebral Effects of Hyperventilation in Man'. *Archives of Neurology*, 12, 410–423.
- Hauge, A., Thoresen, M. & Walloe, L. (1980). 'Changes in Cerebral Blood Flow During Hyperventilation and CO₂-Breathing Measured Transcutaneously in Humans by a Bidirectional, Pulsed, Ultrasound Doppler Blood Velocitymeter'. *Acta Physiologica Scandinavica*, 110, 167–173.
- Helou, L. B., Rosen, C. A., Wang, W. & Verdolini Abbott, K. (2018). 'Intrinsic Laryngeal Muscle Response to a Public Speech Preparation Stressor'. *Journal of Speech, Language, and Hearing Research*, 61, 1525–1543.
- Jantos, M. (2011). 'Fascia, the New Frontier in Anatomy'. *Pelvipерineology*, 35, 2.
- Jantos, M. (forthcoming 2020). A Myofascial Perspective on Chronic Urogenital Pain in Women. In: Santoro, G., Wiczorek, P. & Bartram, C. I. (eds.) *Pelvic Floor Disorders: A Multidisciplinary Textbook*, Springer.
- Jantos, M. & Stecco, C. (forthcoming). Fascia of the Pelvic Floor. In: Schleip, R., Huijing, P. A., Stecco, C. & Driscoll, M. (eds.) *Fascia: The Tensional Network of the Human Body*, 2nd ed., London, Churchill Livingstone Elsevier.
- Jensen, O., Hari, R. & Kaila, K. (2002). 'Visually Evoked Gamma Responses in the Human Brain Are Enhanced During Voluntary Hyperventilation'. *Neuroimage*, 15, 575–586.
- Kain, K. L. & Terrell, S. J. (2018). *Nurturing Resilience. Helping Clients Move Forward from Developmental Trauma. An Integrative Somatic Approach*, Berkeley, California, North Atlanta Books.
- Kozłowska, K. (2013). 'Stress, Distress, and Bodytalk: Co-constructing Formulations with Patients Who Present with Somatic Symptoms'. *Harvard Review of Psychiatry*, 21, 314–333.

- Kozłowska, K., Chudleigh, C., Cruz, C., Lim, M., McClure, G., Savage, B., Shah, U., Cook, A., Scher, S., Carrive, P. & Gill, D. (2018a). 'Psychogenic Non-epileptic Seizures in Children and Adolescents: Part I – Diagnostic Formulations'. *Clinical Child Psychology and Psychiatry*, 23, 140–159.
- Kozłowska, K., Chudleigh, C., Cruz, C., Lim, M., McClure, G., Savage, B., Shah, U., Cook, A., Scher, S., Carrive, P. & Gill, D. (2018b). 'Psychogenic Non-epileptic Seizures in Children and Adolescents: Part II – Explanations to Families, Treatment, and Group Outcomes'. *Clinical Child Psychology and Psychiatry*, 23, 160–176.
- Kozłowska, K., Rampersad, R., Cruz, C., Shah, U., Chudleigh, C., Soe, S., Gill, D., Scher, S. & Carrive, P. (2017). 'The Respiratory Control of Carbon Dioxide in Children and Adolescents Referred for Treatment of Psychogenic Non-epileptic Seizures'. *European Child and Adolescent Psychiatry*, 26, 1207–1217.
- Kozłowska, K., Walker, P., McLean, L. & Carrive, P. (2015). 'Fear and the Defense Cascade: Clinical Implications and Management'. *Harvard Review of Psychiatry*, 23, 263–287.
- Kraaier, V., Van Huffelen, A. C. & Wieneke, G. H. (1988). 'Changes in Quantitative EEG and Blood Flow Velocity Due to Standardized Hyperventilation; a Model of Transient Ischaemia in Young Human Subjects'. *Electroencephalography and Clinical Neurophysiology*, 70, 377–387.
- Laffey, J. G. & Kavanagh, B. P. (2002). 'Hypocapnia'. *New England Journal of Medicine*, 347, 43–53.
- Levine, P. (1997). *Waking the Tiger: Healing Trauma*, Berkeley, California, North Atlantic Books.
- Levine, P. A. & Phillips, M. (2012). *Freedom from Pain*, Boulder, Colorado, Sounds True.
- Lund, J. P., Sadeghi, S., Athanassiadis, T., Caram Salas, N., Auclair, F., Thivierge, B., Arsenault, I., Rompre, P., Westberg, K. G. & Kolta, A. (2010). 'Assessment of the Potential Role of Muscle Spindle Mechanoreceptor Afferents in Chronic Muscle Pain in the Rat Masseter Muscle'. *PLoS One*, 5, e11131.

- Lundberg, U., Forsman, M., Zachau, G., Eklöf, M., Palmerud, G., Melin, B. & Kadefors, R. (2002). 'Effects of Experimentally Induced Mental and Physical Stress on Trapezius Motor Unit Recruitment'. *Work and Stress*, 16, 166–178.
- Macefield, G. & Burke, D. (1991). 'Paraesthesiae and Tetany Induced by Voluntary Hyperventilation. Increased Excitability of Human Cutaneous and Motor Axons'. *Brain*, 114 (Pt 1B), 527–540.
- Manabe, N., Tsutsui, H., Kusunoki, H., Hata, J. & Haruma, K. (2014). 'Pathophysiology and Treatment of Patients with Globus Sensation – from the Viewpoint of Esophageal Motility Dysfunction'. *Journal of Smooth Muscle Research*, 50, 66–77.
- Meng, L., Mantulin, W. W., Alexander, B. S., Cerussi, A. E., Tromberg, B. J., Yu, Z., Laning, K., Kain, Z. N., Cannesson, M. & Gelb, A. W. (2012). 'Head-Up Tilt and Hyperventilation Produce Similar Changes in Cerebral Oxygenation and Blood Volume: An Observational Comparison Study Using Frequency-Domain Near-Infrared Spectroscopy'. *Canadian Journal of Anaesthesia*, 59, 357–365.
- Murray, H. B., Juarascio, A. S., Di Lorenzo, C., Drossman, D. A. & Thomas, J. J. (2019). 'Diagnosis and Treatment of Rumination Syndrome: A Critical Review'. *American Journal of Gastroenterology*, 114, 562–578.
- North, K. N., Ouvrier, R. A. & Nugent, M. (1990). 'Pseudoseizures Caused by Hyperventilation Resembling Absence Epilepsy'. *Journal of Child Neurology*, 5, 288–294.
- O'Brien, M. D., Bruce, B. K. & Camilleri, M. (1995). 'The Rumination Syndrome: Clinical Features Rather Than Manometric Diagnosis'. *Gastroenterology*, 108, 1024–1029.
- Ogden, P. & Fisher, J. (2015). *Sensorimotor Psychotherapy: Interventions for Trauma and Attachment*, New York, Norton.
- Okel, B. B. & Hurst, J. W. (1961). 'Prolonged Hyperventilation in Man. Associated Electrolyte Changes and Subjective Symptoms'. *Archives of Internal Medicine*, 108, 757–762.
- Pace-Schott, E. F., Amole, M. C., Aue, T., Balconi, M., Bylsma, L. M., Critchley, H., Demaree, H. A., Friedman, B. H., Gooding, A. E. K.,

- Gosseries, O., Jovanovic, T., Kirby, L. a. J., Kozłowska, K., Laureys, S., Lowe, L., Magee, K., Marin, M. F., Merner, A., Robinson, J. L., Smith, R. C., Spangler, D. P., Van Overveld, M. & VanElzakker, M. B. (2019). 'Physiological Feelings'. *Neuroscience and Biobehavioral Reviews*, 103, 267–304.
- Partanen, J. V. (2017). Muscle Spindles and Beta Motor Units in Trigger Point and Taut Band Formation. *In: Watkins, M. & Hsüeh, L. (eds.) Trigger Points: Etiology, Pathophysiology and Clinical Management*, Hauppauge, New York, Nova Science Publishers.
- Payne, P., Levine, P. A. & Crane-Godreau, M. A. (2015). 'Somatic Experiencing: Using Interoception and Proprioception as Core Elements of Trauma Therapy'. *Frontiers in Psychology*, 6, 93.
- Porges, S. W. (2011). *The Polyvagal Theory: Neurophysiological Foundations of Emotions, Attachment, Communication, and Self-Regulation*, New York, Norton.
- Rafferty, G. F., Saisch, S. G. & Gardner, W. N. (1992). 'Relation of Hypocapnic Symptoms to Rate of Fall of End-Tidal PCO₂ in Normal Subjects'. *Respiratory Medicine*, 86, 335–340.
- Ribot-Ciscar, E., Rossi-Durand, C. & Roll, J. P. (2000). 'Increased Muscle Spindle Sensitivity to Movement During Reinforcement Manoeuvres in Relaxed Human Subjects'. *Journal of Physiology*, 523 Pt 1, 271–282.
- Schleip, R., Findley, T. W., Chaitow, L. & Huijing, P. A. (eds.). (2012). *Fascia: The Tensional Network of the Human Body*, London, Churchill Livingstone Elsevier.
- Siesjo, B. K. & Kjallquist, A. (1969). 'A New Theory for the Regulation of the Extracellular pH in the Brain'. *Scandinavian Journal of Clinical and Laboratory Investigation*, 24, 1–9.
- Son, S., Kwon, O. Y., Jung, S., Kim, Y. S., Kim, S. K., Kang, H., Park, K. J., Choi, N. C. & Lim, B. H. (2012). 'Relationship Between Hyperventilation-Induced Electroencephalographic Changes and PCO₂ Level'. *Journal of Epilepsy Research*, 2, 5–9.
- Sparing, R., Dafotakis, M., Buelte, D., Meister, I. G. & Noth, J. (2007). 'Excitability of Human Motor and Visual Cortex Before, During, and

- After Hyperventilation'. *Journal of Applied Physiology* (1985), 102, 406–411.
- Stecco, A., Stern, R., Fantoni, I., De Caro, R. & Stecco, C. (2016). 'Fascial Disorders: Implications for Treatment'. *Physical Medicine and Rehabilitation*, 8, 161–168.
- Stecco, C. (2015). *Functional Atlas of the Human Fascial System*, Edinburgh, Elsevier.
- Stecco, C., Pirri, C., Fede, C., Fan, C., Giordani, F., Stecco, L., Foti, C. & De Caro, R. (2019). 'Dermatome and Fasciatome'. *Clinical Anatomy*, 32, 896–902.
- Stenkamp, K., Palva, J. M., Uusisaari, M., Schuchmann, S., Schmitz, D., Heinemann, U. & Kaila, K. (2001). 'Enhanced Temporal Stability of Cholinergic Hippocampal Gamma Oscillations Following Respiratory Alkalosis in Vitro'. *Journal of Neurophysiology*, 85, 2063–2069.
- Sun, X., Ke, M. & Wang, Z. (2015). 'Clinical Features and Pathophysiology of Belching Disorders'. *International Journal of Clinical and Experimental Medicine*, 8, 21906–21914.
- Yamaguchi, F., Meyer, J. S., Sakai, F. & Yamamoto, M. (1979). 'Normal Human Aging and Cerebral Vasoconstrictive Responses to Hypocapnia'. *Journal of the Neurological Sciences*, 44, 87–94.
- Yamatani, M., Konishi, T., Murakami, M. & Okuda, T. (1994). 'Hyperventilation Activation on EEG Recording in Childhood'. *Epilepsia*, 35, 1199–1203.
- Yang, R., Brugniaux, J., Dhaliwal, H., Beaudin, A. E., Eliasziw, M., Poulin, M. J. & Dunn, J. F. (2015). 'Studying Cerebral Hemodynamics and Metabolism Using Simultaneous Near-Infrared Spectroscopy and Transcranial Doppler Ultrasound: A Hyperventilation and Caffeine Study'. *Physiological Reports*, 3(4).