Visceral sensory and cognitive-affective neuroscience: towards integration?

Lukas Van Oudenhove

*Gut* 2010 59: 431-432
doi: 10.1136/gut.2009.192658

These include:

**References**

This article cites 11 articles, 6 of which can be accessed free at:
http://gut.bmj.com/content/59/4/431.full.html#ref-list-1

**Email alerting service**

Receive free email alerts when new articles cite this article. Sign up in the box at the top right corner of the online article.

To order reprints of this article go to:
http://gut.bmj.com/cgi/reprintform

To subscribe to *Gut* go to:
http://gut.bmj.com/subscriptions
Further to acid reflux by changing the nature of the refluxate itself. The enlarged proximal acid pocket in subjects with hiatus hernia means that they have a larger reservoir of highly acidic juice available to reflux into the oesophagus whenever the lower oesophageal sphincter spontaneously relaxes or fails under pressure.

Competing interests None.

Provenance and peer review Commissioned; not externally peer reviewed.

Gut 2010;59:430–431. doi:10.1136/gut.2009.192922

REFERENCES


Visceral sensory and cognitive-affective neuroscience: towards integration?

Lukas Van Oudenhove

Despite rapidly growing evidence supporting a link between psychological processes—both cognitive and affective—and visceral sensation in health as well as functional gastrointestinal disorders (FGIDs), the neural mechanisms underlying these interactions remain infrequently studied within the field of ‘neurogastroenterology’ and therefore rather poorly understood.1 The somatic pain field, in contrast, has already made considerable progress in unravelling these complex brain mechanisms by which emotion (eg, anxiety) and cognition (eg, attention) influence the processing and perception of bodily signals.2 More specifically, the amygdala, insula, and cingulate and prefrontal subregions have been shown to be involved in pain—emotion interactions. This knowledge is the result of a fruitful integration between different branches of science within the somatic pain field over the past decades, including psychology, psychiatry, anaesthesiology and the affective, cognitive and sensory branches of neuroscience.

Such a degree of integration has not been achieved yet within ‘visceral sensory neuroscience’ but is, in my opinion, much needed if we really want to move the field forward. This may be especially critical if we want to make progress in the understanding of the multifactorial pathophysiology of complex, symptom-based disorders including FGIDs. Although rather sparse, recent attempts towards such integration have been made, and I will try to situate these against a long tradition of studying the interactions between mind, brain and body underlying visceral sensation.

In the present issue of Gut, Elsenbruch and colleagues (see page 489) report on a functional MRI study showing that, in irritable bowel syndrome (IBS), anxiety and depression scores correlate with subjective pain ratings during rectal distension.3 Furthermore, within the IBS group, anxiety scores correlate with pain-induced activation of the anterior midcingulate cortex (aMCC) and the pregenual anterior cingulate cortex (pACC); depression scores show a correlation with activity in the dorsomedial prefrontal cortex (PFC) and cerebellar areas. Finally, when pain-induced brain activity in patients with IBS was compared with that of healthy volunteers, (small) differences were found in the ventromedial PFC and the anterior insula (aINS). However, when anxiety and depression scores were controlled for in the analysis, these differences disappeared. I believe this article is important because it is one of the first to show the neural mechanisms underlying the interaction between anxiety and depression on the one hand and visceral sensation on the other in patients with IBS. However, it may be seen as the continuation of a long tradition of (neuro)scientific research on this type of interaction in healthy volunteers. Most of this work comes from a variety of distinct research traditions and may therefore be largely unknown to most people in the field of ‘neuro-gastroenterology’.

In the 1880s, James and Lange provided the first theoretical account of an influence of bodily, especially visceral, signals on emotions. The core idea of their theory is that emotional stimuli automatically induce bodily changes and that the feedback of these bodily changes to the brain is constitutive of the feeling of the emotion.4 Neurobiological support for this theory was recently provided, most notably by Damasio.5 In the 1920s, Cannon formulated an influential alternative theory, stating that emotions are generated in subcortical brain regions, especially the hypothalamus, which influences the viscera through the autonomic nervous system.6 The importance of Cannon’s work for the present issue is twofold. First, he was the first to formulate the idea of reciprocal interaction between gastrointestinal function and emotions. Secondly, Cannon put firm emphasis on physiological study of the influence of (unconscious) psychological

Correspondence to Dr Lukas Van Oudenhove, Translational Research Center for Gastrointestinal Diseases (TARGID), University of Leuven, University Hospital Gasthuisberg, Herestraat 49, B-3000 Leuven, Belgium, lukas.vanoudenhove@med.kuleuven.be

Gut April 2010 Vol 59 No 4 431

Commentaries

Downloaded from gut.bmj.com on March 27, 2010 - Published by group.bmj.com
processes on the viscera, and the role of autonomic, involuntary responses in mediating this interaction.

However, Paul MacLean was the first to formulate a comprehensive theory on brain mechanisms linking emotion and visceral function, elaborating on earlier work by James and Cannon as well as on the influential Papez theory of emotion. MacLean proposed that emotion results from the association of internal and external stimuli in the phylogenetically old brain (‘visceral brain’, later called ‘limbic system’). The ‘visceral brain’ was defined by MacLean based on its anatomical location on the interface between interoceptive and exteroceptive systems. It consisted, among others, of the amygdala, hippocampus, cingulate gyrus, brainstem and hypothalamus, regions that have recently been confirmed to be involved in visceral sensation and emotions using functional brain imaging. The Hungarian physician—physiologist—psychologist György Adám may be regarded as the prototype integratory researcher in the field of visceral perception. One of its central points is that visceral afferent signals, besides being firmly rooted in organ homeostasis, also profoundly influence higher human biological and psychological functions, without having to reach conscious awareness. He stated, as early as the 1960s: ‘...we were already aware of the manifold means by which viscerosensory input could initiate or modify emotional reactions and even of the possibility that ongoing emotional behaviour could alter the efficiency of viscerosensory signals’ (cited in Adám’s highly recommended overview book.)

Interest in these issues, however, remained rather limited until the arrival of functional brain imaging in the 1990s provided us with a powerful new tool for studying brain mechanisms underlying interactions between visceral sensation and emotion/cognition in vivo. However, to date, only a few studies have investigated these interactions in a direct way (ie, studying the effect of manipulating cognitive/affective factors on brain responses to visceral stimulation). The work of the Manchester/London group has been seminal in this respect, but is limited to healthy volunteers. They showed that perceiving a non-painful visceral stimulus in a negative emotional context is associated with higher activation of the aMCC and the aINS. Selective attention towards a similar stimulus was found to be associated with higher activation of the aMCC as well as somatosensory cortex. The same group elaborated on these earlier results in two elegant recent studies. Coen et al found that the reduction in pain ratings during distraction from a painful oesophageal stimulus was paralleled by a reduction in neural activity in the right MCC and the right dorsolateral and ventrolateral PFC. The same authors demonstrated that experimentally induced sadness was associated with higher activity in the right aMCC, aINS and ventrolateral PFC during painful oesophageal distension, but not with higher pain scores.

The study by Eisenbruch et al may be regarded as one of the first applications of these earlier integratory efforts to patients with FGIDs and, therefore, a small but potentially important step further on the road towards integration of visceral sensory and affective/cognitive neuroscience, which is, in my opinion, the way ahead for our field. Thinking along similar lines, our own group has shown a correlation between state anxiety and activity in the pACC/aMCC during painful gastric distension in functional dyspepsia. Taken together, the body of evidence described here points towards a key role for the aMCC, as well as the aINS and prefrontal subregions, in emotion/cognition—visceral pain interactions.

We have, however, still a long way to go. The Eisenbruch study also suffers from some limitations, including a fairly simple experimental design and the use of a rather limited screening tool for general anxiety and depression. It is my opinion that, if we want to overcome such limitations and make further progress along the integratory road, we should try to attract people with various backgrounds (psychology, psychiatry, affective/cognitive neuroscience, neuropathology, brain imaging) to our fascinating field, whether we call it ‘visceral sensory neuroscience’, ‘enteric neuroscience’ or ‘neurogastroenterology’.

Acknowledgements The author is a postdoctoral research fellow of the Research Foundation—Flanders.

Competing interest None.

Provenance and peer review Commissioned; not externally peer reviewed.

Gut 2010;59:431—432. doi:10.1136/gut.2009.192658

REFERENCES