Neuropsychology of Emotion and Emotion-Regulation

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In classic neuropsychological terms, emotion regulation is a higher cortical function that depends on the concerted work of widespread cortical, subcortical, and deep subcortical brain areas. This suggests that we should not only consider the historically relevant question of hemispheric laterality, but also the contribution of specific cognitive skills and brain regions. Thus far, there is emerging evidence to support the link between particular emotion-regulation strategies (e.g., reappraisal and suppression) and well-known basic neuropsychological processes (e.g., inhibition and verbal fluency).

Keywords: emotion ; emotion regulation ; process model of emotion regulation ; reappraisal ; suppression ; laterality

1. Introduction

For more than half a century, there has been a debate in neuropsychology on the issue of hemispheric asymmetry in emotion, linked to a broader discussion about the role of cortical brain regions in emotion. The debate has brought data from a wide range of sources: most notably human lesion work [1][2], and electrical stimulation work in non-human animals [3][4]. A key issue in the debate has been whether there is hemispheric asymmetry in the way that the brain processes emotional information in general, a broad question that can be interpreted in several ways. After extensive investigation and discussion, the field now appears to have some resolution to this larger issue. Essentially, there is evidence for hemispheric asymmetry in some elements of emotional life, but not in others. Indeed, the cortex itself is clearly important only for some elements of the broad phenomenon of emotion, for example the way that emotions are perceived and expressed, which often show strong effects of hemispheric asymmetry. As we discuss below, there is also emergent literature describing laterality effects in emotion regulation, closely linked to specific neuropsychological skills. On the other hand, the role of deep subcortical structures in the generation of emotion has become increasingly clear, particularly as regards the experience of powerful feelings, or affect [3][5].

2. Anosognosia as the ‘Absence’ of Emotion?

There are various limitations to this prototype hemispheric asymmetry model of anosognosia (see [6] for a review). The first is that it fails to account for various forms of emotional complexity in the neurological patient group, including emotion selectivity, and variability across time. For example, a disruption of negative emotions would explain only the absence of emotion in relation to paresis in patients, not why patients might actively deny their paresis [7]. In addition (see [8] for review), the low mood seen in patients with left-sided lesions is likely to result from an emotionally appropriate response to their substantial levels of disability, which typically involves hemiparesis and non-fluent aphasia.

Research on emotion and laterality has also neglected the dynamic character of emotion, particularly emotional experience. During development, human beings learn to manage feelings, particularly painful ones, using either automatic or cognitively controlled regulatory strategies [9][10]. Thus, emotional experience, or emotion generation, cannot be separated from emotion regulation [11], posing important methodological challenges. This fluctuating element is seen in patients with frontal lesions in whom, due to impairments in cognitive control, the dynamics of emotional experience change substantially (e.g., rise time, magnitude, decay rate; see [12][13].

Perhaps an even more complicated question is whether we can even speak of an ‘absence’ of emotion after brain injury. This approach is heavily influenced by how neurology and neuropsychology have historically portrayed cognitive impairments, (correctly) offering accounts of functions that are genuinely lost: a-phasia, a-praxia, a-mnesia, etc. But emotions are not abolished after brain damage. Studies exploring emotional changes after unilateral lesions often report a disruption of specific emotional processes, but not their complete collapse or absence. There are, of course, rare cases in which the processing of a specific emotion can be heavily compromised in all modalities, of which the best known is fear, after bilateral damage to the amygdala [14][15]. However, even in these cases, the loss is not complete, and certainly does not produce an absence of emotional life—not least because other negative emotions are preserved in experience. Case studies that report a preservation of emotional life after extensive bilateral damage to the limbic system support this point,

suggesting that neither cortical nor subcortical damage can completely abolish emotional experience [16][17][18][15][19][20][21][22][23][24][25]. Indeed, data from children with hydranencephalic brains, and non-human animals in which the cortex was removed, suggest that the cortex is certainly not the neural substrate of emotional experience, since emotional experience in these cases was preserved and even amplified [4][26][27][28]. Only deep subcortical lesions, especially to the upper brain stem, appear to completely abolish emotional experience, but in this case due to a complete loss of consciousness [29].

As regards the emotional consequences of ideas, we have reported several instances of preservation of negative emotional states, including the full range of emotional experience, in anosognosic patients with right sided-lesions (see [30] for review). An interesting element of these emotional states was their selective nature; for example, a tendency to produce the same magnitude of emotion as controls, but directed at an external object, rather than towards the self, thus suggesting the influence of dynamic or regulatory variables ([31][6][32][33][34], see [30] for review).

3. The Brain Basis of Emotion Generation

Firstly, as touched on above, it has become clear that cortical lesions do not disrupt the ability to generate emotional experiences. Such cortical lesions clearly produce any number of distortions in the emotional lives of neurological patients. These represent a change in the emotional 'landscape' (as we might call it): such as an increased threshold to trigger emotional reactions [8], inappropriately positive responses to hemiparesis [35][8], failure to correctly interpret emotions [1], disinhibition of emotional responses [36][37], incorrect use of emotion for decision making [38], or failure to appropriately regulate emotions [39]. However, these cortically lesioned patients preserve the full range of emotional experience: from happiness through anger [30]. The literature has increasingly suggested that the source of emotional experience is deeply subcortical [4][40][41][42].

A parallel research strand has long identified many subcortical emotion-related brain areas, such as the amygdala, insula, hypothalamus, and anterior cingulate (see, for example [43][44][45][46][47]. These seem critical for other elements of emotional life, such as emotion-memory [46][47], the integration of internally generated experienced states with externally facing senses [45], or the role of loss in decision making [44]. However, as described above, lesions to these subcortical sites do not obliterate the emotional experience itself (e.g., [48]). Instead, the core of emotional experience appears to be closely tied to systems underpinning consciousness, in the dorsal regions of the mid-brain [4][40][41], especially the PAG [3][49]. In part, this conclusion is based on evidence that all the primary emotion systems (which include the various subcortical regions named above) terminate in the PAG. In addition, it is in the PAG that one appears to find the maximal emotional outcome (pleasurable or aversive) for the smallest electrical current [3]. Stimulation of the amygdala, striatum, insula, hypothalamus, or anterior cingulate produce fewer substantial effects [3][50][51][52], and lesions to those brain areas produce some, but not overwhelming, changes in global emotional experience [53][54][48][55][56][57][58].

This subcortical source is not the central goal of this review, but it provides a much-needed context for understanding the hierarchical organization of emotional life, in all its potential complexity. Critically, this expands the debate on the neural basis of emotion beyond the problem of hemispheric asymmetry, to the 'vertical' dimension of hierarchy. In evolutionary terms, these higher-order cognitive functions have emerged not only to help us successfully deal with demands from the external world, but most importantly to successfully manage internal states of the body (the internal world) in the light of contextual constraints: to manage feelings in an adaptive fashion, in the light of environmental and social limitations. These tools allow us to use emotions to fuel and direct behaviour, to inhibit emotional responses when they are not adequate to our long-term goals, to predict the future based in relevant past experiences, and to read or hide emotional expressions when necessary.

We now further develop this idea, with a focus on the concept of emotion regulation, a complex higher-order psychological process, that has been defined as a mechanism to manage elementary emotional experiences. Below, we describe emerging evidence regarding the neuropsychological and neuroanatomical basis of different emotion regulation strategies, paying special attention to issues of laterality.

4. Emotion Regulation

A critical distinction in neuropsychology has been the difference between having feelings (emotion generation) and successfully managing those feelings (emotion regulation). For well over a century, neuropsychologists have noted that brain injury can change the ability to manage feelings (see [39] for review). The Phineas Gage case [36][37] is a commonly cited early example, reporting that the 'balance' between Gage's intellectual faculties and his 'animal propensities' had been disrupted. Hughlings Jackson also described the phenomenon as one of alteration of 'balance' between cognition and emotion [59] p. 113. The modern literature usually defines these regulation skills as involving a wide set of processes,

by which we influence which emotions we have, when we have them, and how they are experienced and expressed [60]. Outside the field of neuropsychology, emotion regulation has been a popular research topic only in the last few decades, and is increasingly linked to a remarkably wide range of mental health disorders [61][62][63][64][65].

Despite the clinical importance of disorders of emotion regulation, the field was relatively under-investigated in neuropsychology for many decades [66]. For example, from 1990 to 2016, only 41 articles were published (roughly 1.5 per annum) that directly addressed the problem of emotion regulation after brain damage [39]. However, the few years since have seen considerable progress in understanding the neurobiological basis of emotion regulation, and in linking this to a robust theoretical framework, namely the well-established Process Model of Emotion Regulation [60]. This model proposes that human beings manage feelings (in a range of ways, from voluntarily to automatic) by using a wide range of regulatory strategies that depend on diverse neuropsychological functions. These are: situation selection, situation modification, attentional deployment, cognitive change, and response modulation. There has been limited investigation of these in neurological populations, and the available evidence is not conclusive regarding laterality effects. Nevertheless, the field is progressively offering more clarity on the issue, and the model offers fertile ground to systematically explore hemispheric asymmetries in the regulation of affect.

In sum, there is a small, but rapidly growing, body of literature on the brain basis of emotion regulation. Neuroimaging studies with neurotypical subjects have offered relevant insights, but they are limited in establishing the causal role of this association (see [67]). Research programs such as that of Damasio and colleagues on the role of the vmPFC in decision making are a clear example of the benefits of a multi-method approach [68]. Lesion studies can greatly contribute to this endeavour, and complement neuroimaging data, since they allow us to explore how damage to discrete brain areas are related to specific changes in cognition, emotion, and indeed behaviour. The in-depth study of patients with focal lesions also allows us to capture the subjective experience of those changes, addressing the difficult-to-tackle first- and third-person perspective problem in neuroscience [69]. Importantly, patients with focal lesions can be observed and studied in natural settings, where emotion is at its most powerful, and where emotion regulation is most needed (e.g., [15]). Thus, the field is clearly making progress, but there are many opportunities for improvement, especially as regards integration across methods, and especially in better establishing the role of particular psychological abilities.

As one might expect for such an evolutionarily critical skill, emotion regulation relies on a number of foundational cognitive abilities, and is distributed across wide range of brain areas. In classic neuropsychological terms, emotion regulation is a higher cortical function that depends on the concerted work of widespread cortical, subcortical, and deep subcortical brain areas [70]. This suggests that we should not only consider the historically relevant question of hemispheric laterality, but also the contribution of specific cognitive skills and brain regions. Thus far, there is emerging evidence to support the link between particular emotion-regulation strategies (e.g., reappraisal and suppression) and well-known basic neuropsychological processes (e.g., inhibition and verbal fluency). There are strong theoretical arguments to assume that other less-studied emotion-regulation strategies, such as situation selection and attention deployment, also rely on basic neuropsychological processes (e.g., episodic future thinking and attentional control). This is a clear limitation for the field, but also one that can be remedied by additional work, of the sort that has been successful with other strategies (see [39] for review).

5. Discussion: Three Aspirations

What then are we to make of the current state of our understanding of how we regulate feelings, and the neural basis of this process? Firstly, it is now clear that the core of emotional experience is closely tied to evolutionarily ancient brain systems underpinning consciousness, in the upper brain stem and associated subcortical structures. A range of complex cognitive processes have emerged to help mammals and other vertebrates manage these basic emotional states, in the context of environmental and social constraints (e.g., attaching emotional valence to future events, suppressing or amplifying emotions, or using emotions to frame decisions). Our review has focused on emotion regulation, which represents a diverse set of cognitive control systems to manage these elementary emotional experiences. Given the diversity of psychological processes that we can use to regulate feelings, we find that a diverse set of brain regions are necessary to support this process. These are widely distributed, and suggest hemispheric asymmetry consistent with the lateralization of the basic cognitive processes they rely upon.

With this perspective in mind, we offer three aspirations for the field over the next decade or two. Each is achievable, but of course requires a concerted effort—though fortunately the field has been steadily growing in size and influence.

Firstly, the field needs a more comprehensive model of emotion. There are well-developed elements of emotion science that deal with (say) the experience/generation, perception/ expression, memory, and regulation of emotion. Clearly, these elements operate simultaneously, to try and understand the rounded and complex phenomenon that is emotion. However,

research on the specific components operates in largely independent silos. Again, brain-injured patients offer examples to prove that these theoretical silos are artificial constructs. Take, for example, the issue of emotion regulation and emotional reactivity/generation. Both processes tend to be studied separately, despite authors claiming that they are strongly intertwined [71]. It has been widely reported that lesions, particularly to the frontal cortex, can generate an increase in the intensity and magnitude of the emotional response, and a decreased ability to regulate feelings, often referred to as emotional lability, impulsivity, or increased irritability. Here, as noted by Jackson, over a century ago, damage to areas related to cognitive control lower the threshold of emotional reactivity, the well-known 'short-fuse' phenomenon that our patients often report. Similarly, patients with damage to brain areas related to the energization system present a decrease in emotional reactivity, in the form of flat affect, apathy, or abulia [72]. In these cases, the threshold to produce an emotional response is too high, so that the down-regulation of emotion is less required, and the amplification of emotion is too taxing. Thus, one key aspiration for the field would be to develop a more solid suite of studies devoted to the interaction between the emotional drivers and the various cognitive components seeking to regulate them.

Secondly, as regards emotion regulation itself, and the issue of laterality, there is a clear need for the field to be more systematic. As discussed above, some emotion-regulation strategies, such as reappraisal and response modulation, have been appropriately investigated after focal brain lesions, using well-designed experimental tasks and self-reporting. This research has identified which neuropsychological processes might underpin each strategy, and it has also shown that there are substantial differences in the brain regions that underpin the strategies. However, the Process Model identifies a wide range of strategies to achieve emotion regulation. As we review above, other relevant emotion-regulation strategies, such as situation selection and attentional deployment, do not have well-designed experimental tasks, and/or have not been investigated in patients with neurological lesions. Here, our efforts should aim at firstly developing the right tools to explore these processes. Insight from neuropsychology itself could prove valuable, since tasks designed to assess specific neuropsychological components could be modified to measure emotion-regulation strategies. The case of episodic future thinking is here a paradigmatic example, with several studies proposing experimental designs to tap this ability [73][74][75][76][77].

Finally, a fully developed model needs to combine both laterality and hierarchy. The field spent many decades framing the emotion question around the cerebral cortex, and around hemispheric asymmetry in particular. As we review in the Introduction, hemispheric asymmetry is an entirely appropriate question, but only in the context of some facets of emotion. Other facets, such as emotional experience or generation, are probably not cortical phenomena at all. In sum, we need a model that encompasses not only the left–right, but also the up–down dimensions of anatomy. This synthesis will be all the easier through work with non-human animals [4]. As discussed above, some facets of emotion (such as core emotional experience, and even emotion memory) are clearly evolutionarily older, and distributed across subcortical areas. Other skills (such as emotion regulation) are evolutionarily newer and achieved by cortical brain regions.

The neuropsychology of emotion is a field that has taken an enormous journey in the last half-century. The study of emotions (as our opening Luria quote suggests) was often absent, or existed as an 'after-thought' chapter towards the end of a textbook, based on a modest number of papers, published by a few far-sighted specialists. However, the field was never destined to continue this way, because of the enormous importance of emotions in human mental life, and the critical way that disorders of emotion and their management lie at the heart of mental illness. On this basis, we fully expect that the field will rapidly grow, in both size and influence, and we especially hope that the field moves towards greater precision: to better understand the complex component parts that underpin emotion and emotion regulation.

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