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Pathologies of brain attentional networks

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Abstract

In the last decade, it has been possible to trace the areas of the human brain involved in a variety of cognitive and emotional processes by use of imaging technology. Brain networks that subserve attention have been described. It is now possible to use these networks as model systems for the exploration of symptoms arising from various forms of pathology. For example, we can use the orienting network to understand the effects of lesions that produce neglect of sensory information either by brain damage or by restricting transmitter input. Frontal attention networks may provide similar understanding of pathologies at higher levels of cognition. Evidence relating these networks to attention deficit and hyperactivity disorder (ADHD) is considered.

Keywords: Attention deficit and hyperactivity disorder; Attention; Frontal control; Caudate

1. Introduction

Attention is currently being examined in terms of three major functions: orienting to sensory stimuli, executive functions, and maintaining the alert state [1]. Although knowledge of the precise neural mechanisms responsible for these operations is still incomplete, many of the brain areas and networks involved have been identified. Moreover, there is evidence that damage to some of these networks produces similar symptoms regardless of whether the etiology is stroke, degenerative disease, psychopathology or abnormality of development [2,3]. For this reason, knowledge of the networks of attention may be of special use in examination of disorders with attentional symptoms whose cause is not well understood, such as attention deficit and hyperactivity disorder (ADHD). In this paper, we briefly examine the three networks in light of new findings that might relate to theories of ADHD.

Many attempts have been made to understand and characterize the deficits associated with ADHD. Recently, three different theoretical accounts have been proposed [4–6]. We attempt to argue that it is possible to conceptualize all three under the umbrella of pathologies of attentional networks.

2. Networks of attention

Posner and Raichle [1] summarized the three attentional networks approach, and based the localization of the different networks on the brain-imaging literature. Each attentional function is localized not in a single brain area, but as a network of interconnected brain areas.

2.1. Executive-control network

This network has been related to the control of goal directed behavior, target detection, error detection, conflict resolution and inhibition of automatic responses. The executive control network seems to include the midline frontal areas including the anterior cingulate gyrus, SMA, and portions of the basal ganglia. Neuroimaging studies have shown activity in this area during tasks that require mental effort such as in dealing with conflict, handling novelty, developing anticipations and detecting errors [7]. Recently, it has been shown that tasks involving both cognitive and emotional controls produce activation in the cingulate [8,9].

Accumulating evidence shows involvement of the basal ganglia, more specifically the caudate nucleus, in cognitive functioning [10]. Many studies show similar behavioral deficits in animals following experimental lesions of the anterior dorsolateral frontal cortex and the caudate. Moreover, the caudate and the prefrontal cortex show similar activity when recorded with depth electrodes during tasks such as lexical decision and semantic categorization that were not due to motor or premotor activity [11]. In addition to that, the basal ganglia have been considered to be

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particularly important in mediating the connection between executive attention and other attentional operations [12,13].

Lateral areas of the frontal cortex have also often been identified with executive attention. However, the bulk of the literature suggests to us that these areas involve representation of specific kinds of spatial, verbal or form information rather than more general attentional operations.

2.2. Alerting network

The alerting network is defined by a network of brain areas in the right frontal lobe (especially the superior region of Brodmann area 6), the right parietal lobe and the locus coeruleus [1,14]. This network is involved in establishing a vigilant state and maintaining readiness to react. Recent alert monkey studies have shown clearly that the readiness induced by warning signals can be blocked by drugs that reduce norepinepherine (NE) [15]. In addition, patients with right parietal lesions show difficulty in sustaining attention and in the use of warning signals to improve behavior [16].

2.3. Orienting network

A network for covert orienting to sensory, particularly visual signals has been discussed in some detail previously [17]. Neuroimaging evidence has shown that covert shifts of visual attention most strongly activate the parietal lobe. Moreover, other areas, related also to the oculo-motor system, are also activated [18]. There is strong evidence that attending to an object in a spatial location increases blood flow and electrical activity in extrastriate visual regions particularly the fusiform gyrus [19].

3. Leading theories of ADHD and their predictions

As mentioned in Section 1, three theoretical accounts explaining ADHD have been recently proposed. Swanson et al. [6] characterized ADHD as a combination of executive attention and alerting deficits. This approach focuses on two of the attentional networks and predicts brain pathology in areas related to these networks, i.e. midline frontal cortex (cingulate and SMA), basal ganglia (especially caudate), anterior prefrontal cortex, anterior right parietal cortex. According to Barkley [4], the core of ADHD pathology is executive functions. By executive functions he referred to working memory, internalization of self-directed speech, control of emotion arousal and motivation. Barkley suggests that during normal development children switch from external cues to internal guidance of behavior. Although the definition of executive functions varies somewhat in he literature, most of the functions included by Barkley haven been conceptualized, as being part of the executive network. Activation of both emotional and cognitive tasks has been shown to occur in the anterior cingulate [8]. Posner and Rothbart [9] have argued that emotional control mechanisms arise in early infancy and this same general system is later used for control of conflict and other executive functions. Barkley, too, although using a different nomenclature, ties the disinhibition behavioral symptoms of ADHD as being secondary to the main executive control deficit. Sergeant et al. [5], on the other hand, emphasize the energetic factors, as the most critical deficit in ADHD. According to their framework, ADHD suffer from a deficit in the energetic maintenance and allocation of resources (which leads to the secondary symptoms of disinhibitory behavior). This approach identifies the activation pool with the basal ganglia and corpus striatum, and suggests it operate mainly through shifts in the criterion for responding. According to an attentional network framework, this frontal activation could, also, be part of the executive control network.

4. Brain pathologies in ADHD

Filipek [20,21] found that despite similar hemispheric volumes, ADHD subjects had smaller volumes of left caudate and caudate head, with reverse asymmetry than controls and right anterior–superior frontal region en bloc and white matter. Moreover, possible structural correlates of ADHD response to stimulants were found. Castellanos et al. [22] found smaller right anterior frontal, caudate and globus pallidus regions in ADHD compared to control. Aylward et al. [23], too, report volumetric differences in caudate and globus pallidus, between ADHD children and control (for review, see Ref. [24]).

Behavioral studies support the basic idea that ADHD is a deficit in executive control and regulation that extends to emotional and cognitive processes [4]. There is evidence of difficulty in controlling the activation state [5]. This result may relate to basal ganglia and frontal control system deficits, rather than more direct NE or parietal abnormalities. There is little empirical support of the involvement of the orienting network in ADHD pathology. Although a few studies have tested endogenous and exogenous orienting of attention, in ADHD, the results are ambiguous as to abnormalities in the basal ganglia and frontal control structures, versus parietal structures.

5. Conclusions

For many years, psychiatrists and psychologists have been trying to define and understand the deficits that underlie ADHD. Evidence is consistent with difficulties in two of the attentional networks: executive functions/effortful control, and vigilance and alerting regulation. We believe that a better understanding of the functions and neuroanatomical bases of attentional networks in normal development will be helpful in understanding ADHD.

References

[1] Posner MI, Raichle M. (Revised). Images of mind, Washington, DC: Scientific American Books, 1996.

[2] Fernandez-Duque D, Posner MI. Imaging attentional networks in normal and brain injured populations. Journal of Experimental and Clinical Neuropsychology, in press.

[3] DiGirolamo GJ, Posner MI. Attention and schzophrenia: a view from cognitive neuroscience. Cognitive Neuropsychiatry 1996;1(2):95–102.

[4] Barkley RA. Attention-deficit hyperactivity disorder. Scientific American 1998;279:66–71.

[5] Sergeant J, Oosterlann J, van der Meere J. Information processing and energetic factors in attention-deficit hyperactivity. In: Quay HC, Hogan AE, editors. Handbook of disruptive behavior disorders, New York: Plenum, 1999, in press.

[6] Swanson J, Posner MI, Cantwell D, Wigal S, Crinella F, Filipek PA, Emerson J, Tucker D, Nalcioglu O. Attention-deficit hyperactivity disorder: symptom domain, cognitive processes and neural networks. In: Parasuraman R, editor. The attentive brain, Cambridge, MA: MIT Press, 1998, in press.

[7] Posner MI, DiGirolamo GJ. Executive attention: Conflict, target detection, and cognitive control. In: Parasuraman R, editor. The attentive brain, Cambridge, MA: MIT press, 1998.

[8] Bush G, Whalen PJ, Rosen BR, Jenike MA, McInerey SC, Rauch SL. The counting Stroop: an interference task specialized for functional neuroimaging-validation study with functional MRI. Human Brain Mapping 1998;6(4):270–82.

[9] Posner MI, Rothbart MK. Attention, self regulation and consciousness. Philosophical Transactions of the Royal Society London 1998;353:1–13.

[10] Beiser DG, Hua SE, C HJ. Network models of the basal gangila. Current Opinion in Neurobiology 1997;7:185–90.

[11] Abdullaev YG, Bechtereva NP, Melnichuk KV. Neuronal activity of the human caudate nucleus and prefrontal cortex in cognitive tasks. Behavioral Brain Research 1998;97:159–77.

[12] Jackson SR, Marrocco R, Posner MI. Networks of anatomical areas controlling visuospatial attention. Neural Networks 1994;7:925–44.

[13] LaBerge D. Thalamic and cortical mechanisms of attention suggested by recent positron emission tomographic experiments. Journal of Cognitive Neuroscience 1990;2:358–72.

[14] Posner MI, Petersen SE. The attention system of the human brain. Annual Reviews of Neurology 1990;13:25–42.

[15] Marrocco RT, Davidson MC. Neurochemistry of attention. In: Parasuraman R, editor. The attentive brain, Cambridge, MA: MIT Press, 1998.

[16] Robertson IH, Tegner R, Tham K, Lo A, et al. Sustained attention training for unilateral neglect: Theoretical and rehabilitation implications. Journal of Clinical and Experimental Neuropsychology 1995;17(3):416–30.

[17] Posner MI, Dehaene S. Attentional networks. Trends in Neurosciences 1994;17(2):75–9.

[18] Corbetta M. Fronto-parietal cortical networks for directing attention and eye to visual locations: Identical, independent or overlapping neural systems. Proceedings of the National Academy of Sciences of the USA 1998;95:831–8.

[19] Mangun GR. Integrating imaging and electrophysiology (Revised). In: Gazzaniga M, editor. The cognitive neurosciences, 1998, in press.

[20] Filipek PA, Semrud-Clikeman M, Steingard RJ, Renshaw PF, Kennedy DN, Biederman J. Volumetric MRI analysis comparing subjects having attention-deficit hyperactivity disorder with normal controls. Neurology 1997;48:589–601.

[21] Filipek PA. Structural variations in measures in the developmental disorders. In: Thatcher RW, Lyon GR, editors. Developmental neuroimaging: mapping the development of brain and behavior, San Diego, CA: Academic Press, 1996.

[22] Castellanos FX, Giedd JN, March WL, Hamburger SD, Vaituzis AC, Dickstein DP, Sarfatti SE, Vauss YC, Snell JW, Lange N. Quantitative brain magnetic resonance imaging in attention-deficit hyperactivity disorder. Arch General Psychiatry 1996;53:607–16.

[23] Aylward EH, Reiss AL, Reader MJ, Singer HS, Brown JE, Denkla MB. Basal ganglia volumes in children with attention-deficit hyperactivity disorder. Journal of Child Neurology 1996;11:112–5.

[24] Swanson J, Castellanos FX, Murias M, LaHoste G, Kennedy J. Cognitive neuroscience of attention deficit hyperactivity disorder and hyperkinetic disorder. Current Opinion in Neurobiology 1998;8(2):263–71.