the biology of attention*

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The possibility of a biological understanding of the processes we call "attention" seemed so remote a few years ago that, were this article to have been attempted, it would, like the Cheshire cat, have faded away, leaving only its title. While there are not yet volumes to fill, there are some observations about the biology of attention which are definitely worth reporting.

One of the most impressive advances is the discovery by Mountcastle (1975 and 1976), while studying the firing rates of single neurons in the awake, behaving monkey, of units in the parietal cortex which "increase their discharge rates abruptly when the animal fixates with his eyes objects of interest in his immediate surround" (Mountcastle 1975, p. 128). The firing rate changes only if the object of fixation has motivational significance; the mere process of gazing at a visual stimulus is not sufficient. For example, fixation of a food object leads to an increase in firing rate only when the animal is hungry. These cells are by no means rare; in the inferior parietal lobule, as many as half the neurons observed are of this type. Other neurons in this area fire just before visual saccades, evoked by interesting objects, but not before spontaneous eye movements. An interpretation in terms of selective attention seems very plausible. Ingle (1975) has made similar observations in the tectum of the frog.

Certain neurons in the somatosensory cortex of the monkey acquire a differential response to tactile stimuli which have significance as cues. These neurons also show short-term memory, responding as if the cue stimuli were present after they have disappeared. In experiments by Werner and Roppolo (Werner, personal communication), the monkey faces a red light and his hand is stroked with a fine brush in one of two directions. If the light is on and the brushing is in one direction, he may obtain a reward (using his other hand). The firing pattern changes when that specific stimulus combination is presented, and the firing pattern remains altered during the intertrial period. Werner suggests that "new stimulus-instruction conjunctions are evaluated against some residual information as to the last stimulus-instruction combination which was held over in the form of 'buffer memory.'" These persistent acquired changes in firing pattern may be involved in the formation of sensory set.

Motor set has been investigated by Tanji and Evarts (1976), who used monkeys trained to push or pull a lever depending on the combination of cues given. The color of a light determined whether to push or pull, and a slight movement of the lever indicated when to push or pull. Motor cortex neurons were observed which changed firing rate in specific ways to the two cues, before the monkey began to move—in fact, before the appearance of electromyographic activity. The instruction-related changes persisted for several seconds before the cue to move was given. In cases when the "wrong" response to the visual cue was made in the motor cortex, a wrong behavioral response also occurred when the cue to move was given. If the cue to move was given before the effect of the instruction was detectable in the motor cortex, the monkey's motor response was delayed or was incorrect. These neurons seem to reflect "intention" or "motor set."

Human neurological cases also shed some light on the biology of attention. Mesulam and Geschwind (in press), reviewing the literature, conclude that distur-

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ances in attention are a significant complication of strokes in the occipito-temporal, parietal, or frontal areas. "The ability to direct and maintain vigilance is impaired; distractibility by irrelevant stimuli is augmented; and the ability to maintain a coherent stream of thought or to perform a sequence of goal directed behavior is severely compromised." In these cases damage is to the right hemisphere; similar lesions of the left hemisphere produce language disorders without interfering with attention. Lesions in either parietal or frontal lobes can also produce neglect of the sensory field opposite the lesion. "In extreme cases, these patients may act as if one half of the universe did not exist at all. One patient may shave only one half of his face; another may only dress one half of his body; still another may read only half of each sentence on a page." Primary sensation on the unattended side may be intact in these patients, so the defect is attentional. Since the lesions in these cases are also primarily in the right hemisphere, Mesulam and Geschwind postulate that the right hemisphere is predominant in regulating attention, just as the left hemisphere is specialized for language. It is especially interesting that the attention-related neurons which Mountcastle’s laboratory has described lie in the inferior parietal lobule, one of the regions where lesions give rise to inattention syndromes in man.

One may even begin to speak of a neurochemistry and pharmacology of attention. Intriguing correlations have been found between plasma dopamine-beta-hydroxylase (DBH) and measures of selective attention. Buchsbaum et al. (in press) report that, among normal subjects, high DBH is associated with a greater enhancement of the evoked response when subjects direct attention toward the stimulus channel. Subjects with high DBH also make fewer commission errors on the continuous performance test. These differences are especially marked in a subgroup with low platelet monoamine oxidase. The meaning of these correlations is not yet known.

Excessive fixation of attention is commonly observed in amphetamine overdose. Ellinwood, Sudilovsky, and Nelson (1973) described "engagement in tasks that primarily involve small bits or minutiae and a marked enhancement of perceptual acuity directed toward these minute objects" (p. 1088). Very similar states ("stereotyped behavior") can also be provoked by amphetamine and apomorphine in animals. At least in the monkey, the stereotypy seems to be mental rather than motor, since the animal will make use of a variety of motor behaviors in order to pursue a fixed objective, such as persistently following another monkey and positioning himself at a vantage point from which he can stare at his cagemate, much to the cagemate’s distress. L-Dopa sometimes causes similar states of attentional perseveration, exemplified by this narrative account by Sacks (1973) of a Parkinsonian patient treated with L-dopa:

Miss D. would lift a tea-cup to her mouth, and find herself unable to put it down; she would reach for the sugar-bowl, and find her hand "stuck" to the bowl; when doing crosswords, she would find herself staring at a particular word, and be unable to shift either her gaze or her attention from it; and, most disquietingly (not only for herself but for others), she would at times feel "compelled" to gaze into someone else’s eyes. . . . [p. 42]

Just as dopaminergic agonists have a tendency to produce attentional perseveration, dopamine antagonists such as the neuroleptics seem to weaken the grip of delusional obsessions or fixed ideas. Indeed, this may be one of the reasons for their therapeutic value (Matthysse, in press).

Attention is not so mysterious as it once was; we now have the beginnings of an understanding at neurophysiological, neuroanatomical, and neurochemical levels. If not yet a whole cat, it is more than a grin.

References


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**erratum**

In Péter H. Venables’ article “The Electrodermal Psychophysiology of Schizophrenics and Children at Risk for Schizophrenia: Controversies and Developments” (*Schizophrenia Bulletin*, 3:28-48, 1977), the sentence beginning on the seventh line of the second column on p. 41 should be corrected to read as follows: “In confirmation of earlier findings, Zahn, Carpenter, and McGlashan (1975) showed the recovery time of the SCR to 72-dB, 500-Hz tones of two groups of schizophrenics, who did or did not improve on treatment, to be significantly shorter [instead of slower] than normal and not to differ between each other.”