

It is much more difficult to ascertain whether visual dreams also persist in such cases, although one of our hemianopic patients was sure of this when he reported dreaming that his vision had fully recovered, and he was seeing the entire scenery flowing by on his (blind) left side when he was driving. Yet more intriguing are reports of after-images resulting from very strong or persistent visual stimulation, because they imply that even optical stimuli may retain some unusual and still mysterious capacity to provoke awareness. Even if these examples of non-veridical vision are of little use to the patient in everyday life, they are important in showing that even fully -formed phenomenal visual percepts can exist when V1 is destroyed.

Why is blindsight blind? Many hypotheses have been proposed: V1 provides the lion's share of retinal input to the visual cortical areas, which cannot render vision conscious on the basis of the remaining weaker signals arriving via extrageniculate-striate pathways; the visual cortical areas have to report back to V1 to instigate conscious vision; destruction of V1 interferes with phase locking of neuronal discharges or with establishing a particular frequency (gamma) band; communication with areas in prefrontal cortex or with particular thalamic structures fails as a consequence of the lesion. All of these ideas are presently under investigation, and some will need to be revised to accommodate both the return of (some) conscious vision and the system's capacity to generate non-veridical visual perceptions in the absence of V1. Transcranial magnetic stimulation on patients as well as in attempts to induce blindsight in the normal sighted may help by showing where and when disruption of normal activity interferes with particular blindsight functions.

Any related phenomena?

In view of the dissociation between stimulus awareness and visually-guided performance observed following destruction of V1, one might expect damage to

primary auditory or somatosensory cortices to produce similar phenomena, and indeed 'numb sense' is well established in patients with unilateral lesions of somatosensory cortex. As information from each ear reaches both hemispheres, unilateral lesions of auditory cortex do not cause cortical deafness. Nevertheless, a patient with total cortical deafness from bilateral damage demonstrated 'deaf hearing' when required to guess about the localization of 'unheard' sounds.

Implicit processing of visual qualities that are not consciously perceived has been elicited in patients suffering from achromatopsia, who may process and respond to different wavelengths without perceiving colour. This has also been reported for subjects with dense visual agnosia for faces, who may show different reaction times when having to respond to the identity, familiarity, or expression of faces they no longer recognize or understand.

Finally, patients with severe amnesia who cannot overtly recall recent events, and fail to remember that they have undergone prior testing, can score much better upon further testing with the same task. The wealth of evidence on implicit processes in many domains makes one wonder whether implicit processes always remain when the explicit representation is destroyed by circumscribed cortical damage, and whether rehabilitation programmes could successfully harness the remaining capacities for restitution.

Further reading

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Essay

What does the cerebellum really do?

Mitch Glickstein

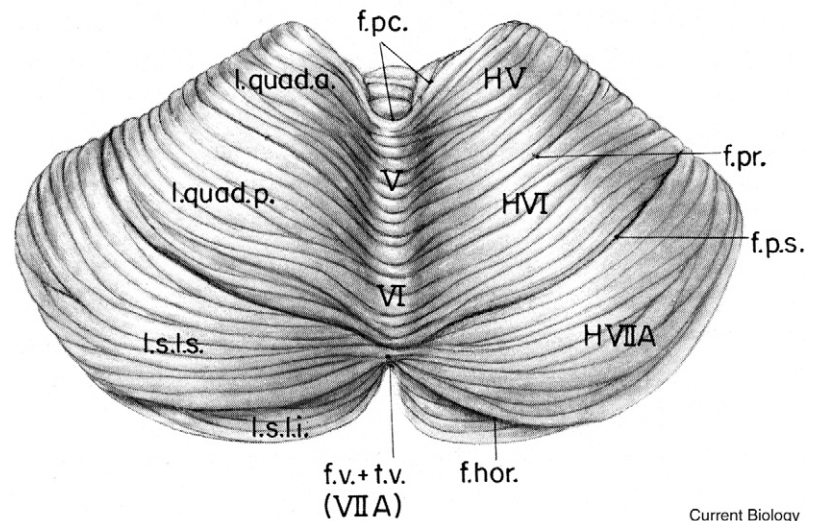
The cerebellum is a large part of the brain in all vertebrates. In humans it constitutes about ten percent of the total brain weight, but the small granule cells of the cerebellar cortex are densely packed so that the cerebellum contains more neurons than all of the rest of the brain. Although it has a uniform cellular structure in mammals and birds, there is great variability in the relative size of its parts. In mammals the cerebellum can be roughly divided into three parasagittal divisions; a midline vermis (Latin: a worm) and two lateral hemispheres. The hemispheres are large in the higher primates and they are very large in the human brain. Because the cerebellar hemispheres are particularly large in humans and the higher primates, from time to time claims have been made that in addition to its role in motor control, the cerebellum is important for cognitive functions, such as learning, attention, and language. Here I review some of the evidence for and against that claim.

Since Flourens [1] it has been recognized that lesions of the cerebellum produce profound deficits in coordinated movement. More recent experimental and clinical evidence has shown that the cerebellum also plays a critical role in motor learning [2] and reflex adaptation [3]. Claims for a cognitive function of the cerebellum have been raised and refuted in the past [4]. The syllogism seems to be: "monkeys and apes are clever; people are very clever; monkeys and apes have big cerebellar hemispheres; people have very big cerebellar hemispheres; therefore, the cerebellum is related to intellect"

[5] (Figure 1). But people and monkeys have other things in common: in addition to their intellect, they are good with their fingers, and they move their eyes continuously and accurately despite challenges set by fatigue [6] or injury to the extra-ocular muscles [7]. The cerebellum could equally well be related to these shared abilities.

If you go to Pub Med and type in “cerebellum AND cognition” you get 771 references. If you type in “cerebellum AND fMRI” you are offered 4,822 references, many of these tying the cerebellum to one or another cognitive function. So why am I sceptical? The clinical literature suffers from a common problem. An interesting finding is remembered; negative evidence may not be. The extreme folding of the cerebellar cortex, and the long latency of the BOLD signal detected by fMRI make the cerebellum a difficult structure for fMRI studies. Activation of the human cerebellum in fMRI scans may be related to actual or planned movement of the eyes, vocal apparatus or fingers. Anatomical evidence is interpreted on the basis of cognitive functions of the structures that receive an input from the cerebellum, but these same structures may be more related to the control of eye movements than to cognition.

Evidence in support of cognitive functions for the cerebellum come from several sources. Following cerebellar lesion in humans there are reported deficits in attention [8] and short-term memory [9]; fMRI studies have shown that the cerebellum is activated when subjects perform a wide variety of cognitive tasks [10]. Further support comes from anatomical studies: if a virus-based trans-neuronal retrograde tracer is placed into an area of the cerebral cortex known to be cognitive in function, labelled cells are found in the dentate nucleus, the main output of the cerebellar hemispheres [11]. I propose to comment on these sources of evidence.



Current Biology

Figure 1. The human cerebellum.

A drawing of the human cerebellum, cerebellar connections and cerebellar cortex from Larsell and Jansen *The Comparative Anatomy and Histology of the Cerebellum* (University of Minnesota Press). The figure shows the large size of the cerebellar hemispheres in the human brain, as compared to the vermis. V, VI and VIIA are cerebellar vermis. The rest is cerebellar hemispheres.

Some years ago there was a report of an interesting deficit in a patient with massive right cerebellar hemisphere damage [12]. When given the task of finding an appropriate verb for a noun that the experimenter recited, the patient made a number of inappropriate responses. If I were to say “car”, you might respond “drive”; the patient made some peculiar and somewhat inappropriate responses. The logic seemed clear. The right hemisphere of the cerebellum projects to left cerebral cortex by way of the thalamus. In the patient, the cortical language area would be deprived of its input from the cerebellum, so there is a resultant deficit in word finding. But although the results remain of interest, there have been several failures to replicate the same effect in other patients with cerebellar damage [13]. But it is often the original case that is remembered.

Claims for an immediate memory deficit similarly are often rather weak, and they may not be permanent. In one study [9] a cerebellar patient was asked to repeat a series of digits recited by the experimenter either in the

same or reverse order. An initial deficit was entirely recovered in a few months. There are examples of failure to replicate findings of deficits in short-term memory, attention and other cognitive functions in such patients. In those cases in which deficits have been seen, the cause may often be the inevitable motor deficits associated with cerebellar lesion, or damage to brain structures outside of the cerebellum.

For the execution of accurate steering of the arm, and adjusting the wrist and fingers in relation to the size and orientation of a target, there must be circuits that link visual to motor areas of the brain. The cerebellum is probably the single most important link between visual and motor areas of the cerebral cortex. What sort of visual information reaches the cerebellum? In monkeys there are two groups of cortical areas that process visual information beyond the primary visual cortex. A dorsal, medially located group that is dominated by cells that are responsive primarily to moving stimuli, and a ventral group involved in visual recognition. Ungerleider and Mishkin [14]

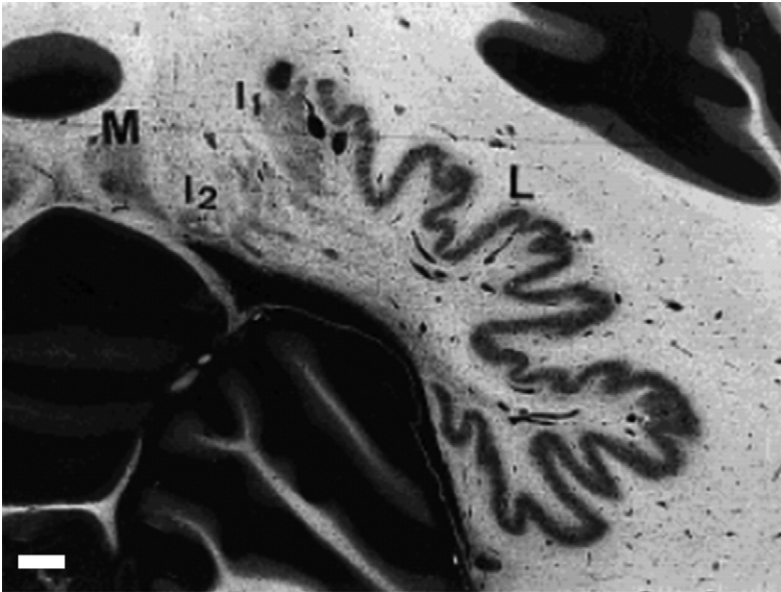


Figure 2. The dentate nucleus.
The figure shows the large size of the dentate nucleus in the human brain. (From Matano and Hirasaki [20].)

based their suggested grouping of visual areas — “where is it?” versus “what is it?” — on the behavioural effects of lesions. Jack May and I [15] based our distinction between a medial and a lateral group on the difference in the pontine projections of the two groups. The dorsal group is connected to the cerebellum by way of the pons; the lateral, more ventrally placed group is not. Information on movement of objects and of the body in relation to its surroundings is sent continuously to the cerebellum by cells in the dorsal cortical visual areas. Lesions of the dorsal group, including the cortex within the angular gyrus in monkeys [16] and a similar cortical area in humans [17] produce a lasting deficit in visual guidance of movement of the arms and fingers. Lesions of the ventral group do not.

The output from the cerebellum is by way of the cerebellar nuclei. The cerebellar hemispheres project to the lateral, dentate nucleus (Figure 2). Connections from the cerebellar nuclei to the cerebral cortex are by way of the thalamus. The great majority of the cerebellar output is to ventral areas of the thalamus that project to motor cortex. There

is also a smaller projection to a region of medio-dorsal nucleus of the thalamus which projects to prefrontal cortex. In every study in which the output of the cerebellar nuclei has been mapped (for example [18]), the connections to medio-dorsal thalamic nucleus are to a region at its lateral edge, adjacent to the internal medullary lamina of the thalamus, an area whose cells relay to cortical areas controlling eye movement. A region of prefrontal cortex rostral to the frontal eye fields which receives a relayed input from the dentate nucleus also contains cells active in regulating eye movements [19].

If the great expansion of the cerebellum is not related to increased cognitive ability, what does it do? Everyone who studies the cerebellum knows how it works, but no two people seem to agree. Theories range from those that view the cerebellum as a purely sensory structure, the function of which is to interpret the constant stream of sensory information, to those that view it as a motor structure that uses sensory information to guide movements. A possible role for the cerebellum in cognition does not rule out either of these interpretations.

I would propose an alternative to the syllogism of big cerebellum, hence cognitive function. Monkeys, apes and people all have remarkably similar retinal structures with an area of exquisitely high acuity, and two eyes that are normally positioned precisely for single vision. Monkeys and apes are partially bipedal; people are completely bipedal. As Matano and Hirasaki [20] point out, the hands and fingers are freed for independent use in humans. Interestingly, the gibbon *Hylobates* has a dentate nucleus that is almost as complexly folded as that of humans. People or monkeys with cerebellar lesions fail to adapt to challenges posed by deviation of one eye [7], fatigue [6] or vestibular damage [3]. There are deficits in finger use in patients with cerebellar lesions [21]. I would therefore argue that the cerebellum is related to skilled use of the fingers and accurate direction of the eyes.

The cerebellum is busy planning, adjusting and executing movements of the body, the limbs and the eyes. It plays a major role in several forms of motor learning. The evidence for a role for the cerebellum in cognitive functions is rather weak. The anatomical data are more consistent with its function in motor control and adaptation.

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Primer

Adaptive radiation of cichlid fish

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How do new species arise? What is the genetic basis for adaptive morphological change and reproductive isolation? How can closely related species co-exist in the same place? What makes some groups diversify faster and more extensively than others? These are some of the questions that evolutionary biologists would like to answer by investigating the extraordinary tendency of cichlid fishes to diversify in tropical lakes. In addition, their diverse suite of mating and parental care strategies, and the importance of the tilapias in human nutrition through aquaculture, have placed cichlid fishes at the centre of an important confluence of research areas and led to the recent decision to fund the sequencing of a cichlid fish genome.

Cichlid origins and relationships
Cichlids (pronounced SICK-lids) are a family of fishes (Cichlidae) found mainly in tropical

freshwaters. Mitochondrial genome sequences have indicated that cichlids are closely related to the marine surperches (Embiotocidae) and damselfishes (Pomacentridae), but not, as previously thought, to wrasse and parrotfishes (Labridae and related families). Many fishes have pharyngeal teeth in the throat for processing their food, but fishes of all of these families have a similar and unusually structured set of pharyngeal jaws which are believed to be particularly powerful and flexible in the processing of food items. This is thought to have allowed the oral jaws to become more specialised for food capture, as they have in the cichlids and wrasse.

The natural distribution of the cichlids is centred on Africa, Latin America and Madagascar, with a few species native to south Asia and the middle east, suggesting that cichlids were already widespread throughout the great southern supercontinent Gondwana when it started to split up around 120–160 million years ago. The African cichlids have been the focus of most research, both because of their great adaptive radiations and their importance as food fish (Table 1). The African cichlids are classed into a number of ‘tribes’, of which the haplochromines are the most species-rich and ecologically

Table 1. Natural distribution of cichlid species.

Location	Number of known species	Estimated Age of basin (Myrs)	Major radiating lineages
African Lakes	1,400		
Malawi	600	8.6	Haplochromine
Victoria	>500	0.4	Haplochromine
Tanganyika	180	~20	Several
Edward	60	2.0	Haplochromine
Kivu	16	5.0	Haplochromine
Barombi Mbo	11	~1	Tilapiine
Kyoga	>10	0.4	Haplochromine
Albert	10	2.0	Haplochromine
Bermin	9	0.8	Tilapiine
Ejagham	7	0.01	Tilapiine
Mweru	6	0.35	Haplochromine
Natron	5	1.0	Tilapiine
Africa, Rest of	175		
Asia (South)	3		
Middle-East	7		
South America	450		
Mesoamerica	110		
Madagascar	32		
Total	2,200		