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Function of the thalamic reticular complex: The searchlight hypothesis

(conjunctions/rapid bursts/Malsburg synapses/vertical cell assemblies/synfire chains)

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ABSTRACT **It is suggested that in the brain the internal attentional searchlight, proposed by Treisman and others, is controlled by the reticular complex of the thalamus (including the closely related perigeniculate nucleus) and that the expression of the searchlight is the production of rapid bursts of firing in a subset of thalamic neurons. It is also suggested that the conjunctions produced by the attentional searchlight are mediated by rapidly modifiable synapses—here called Malsburg synapses—and especially by rapid bursts acting on them. The activation of Malsburg synapses is envisaged as producing transient cell assemblies, including "vertical" ones that temporarily unite neurons at different levels in the neural hierarchy.**

This paper presents a set of speculative hypotheses concerning the functions of the thalamus and, in particular, the nucleus reticularis of the thalamus and the related perigeniculate nucleus. For ease of exposition I have drawn my examples mainly from the visual system of primates, but I expect the ideas to apply to all mammals and also to other systems, such as the language system in man.

Visual System

It is now well established that in the early visual system of primates there are at least 10 distinct visual areas in the neocortex. [For a recent summary, see Van Essen and Maunsell (1).] If we include all areas whose main concern is with vision, there may be perhaps twice that number. To a good approximation, the early visual areas can be arranged in a branching hierarchy. Each of these areas has a crude "map" of (part of) the visual world. The first visual area (area 17, also called the striate cortex) on one side of the head maps one-half of the visual world in rather fine detail. Its cells can respond to relatively simple visual "features," such as orientation, spatial frequency, disparity (between the two eyes), etc. This particular area is a large one so that the connections between different parts of it are relatively local. Each part therefore responds mainly to the properties of a small local part of the visual field (2).

As one proceeds to areas higher in the hierarchy, the "mapping" becomes more diffuse. At the same time the neurons appear to respond to more complex features in the visual field. Different cortical areas specialize, to some extent, in different features, one responding mainly to motion, another more to color, etc. In the higher areas a neuron hardly knows where in the visual field the stimulus (such as a face) is arising, while the feature it responds to may be so complex that individual neurons are often difficult to characterize effectively (3, 4).

Thus, the different areas analyze the visual field in different ways. This is not, however, how we appear to see the world. Our inner visual picture of the external world has a unity. How then does the brain put together all of these different activities to produce a unified picture so that, for example, for any object the right color is associated with the right shape?

The Searchlight

The pioneer work of Treisman and her colleagues (5–8), supported more recently by the elegant experiments of Julesz (9–11), have revealed a remarkable fact. If only a very short space of time is available, especially in the presence of "distractors," the brain is unable to make these conjunctions reliably. For example, a human subject can rapidly spot an "*S*" mixed in with a randomly arranged set of green *X*s and brown *T*s—it "pops out" at him. His performance is also rapid for a blue letter mixed in with the same set. However, if he is asked to detect a green *T* (which requires that he recognize the *conjunction* of a chosen color with a chosen shape), he usually takes much more time. Moreover, the time needed increases linearly with the number of distractors (the green *X*s and brown *T*s) as if the mind were searching the letters *in series*, as if the brain had an internal attentional searchlight that moved around from one visual object to the next, with steps as fast as 70 msec in favorable cases. In this metaphor the searchlight is not supposed to light up part of a completely dark landscape but, like a searchlight at dusk, it intensifies part of a scene that is already visible to some extent.

If there is indeed a searchlight mechanism in the brain, how does it work and where is it located? To approach this problem we must study the general layout of the brain and, in particular, that of the neocortex and the thalamus. The essential facts we need at this stage are as follows.

Thalamus

The thalamus is often divided into two parts: the dorsal thalamus, which is the main bulk of it, and the ventral thalamus. [For a general account of the thalamus, see the review by Jones (12).] For the moment when I speak of the thalamus I shall mean the dorsal thalamus.

Almost all input to the cortex, with the exception of the olfactory input, passes through the thalamus. For this reason it is sometimes called the gateway to the cortex. There are some exceptions—the diffuse projections from the brainstem, the projections from the claustrum, and also some projections from the amygdala and basal forebrain—that need not concern us here.

This generalization is not true for projections *from* the cortex, which do not need to pass through the thalamus. Nevertheless, for each projection *from* a region of the thalamus there is a corresponding reverse projection from that part of the cortex *to* the corresponding region of the thalamus. In some cases at least this reverse projection has more axons than the forward projection.

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Most of the neurons in the thalamus are relay cells—that is, they receive an input from outside the thalamus (for example, the lateral geniculate nucleus of the thalamus gets a major input from the retina) and project directly to the cortex. Their axons form type I synapses and therefore are probably excitatory. There is a minority of small neurons in the thalamus—their exact number is somewhat controversial—that appear to form type II synapses and are therefore probably inhibitory.

While on the face of it the thalamus appears to be a mere relay, this seems highly unlikely. Its size and its strategic position make it very probable that it has some more important function.

Reticular Complex

Much of the rest of the thalamus is often referred to as the ventral thalamus. This includes the reticular complex (part of which is often called the perigeniculate nucleus), the ventral lateral geniculate nucleus, and the zona incerta. In what follows I shall, for ease of exposition, use the term reticular complex to include the perigeniculate nucleus. Again, "thalamus" means the dorsal thalamus. Although much of the following information comes from the cat or the rat, there is no reason to think that it does not also apply to the primate thalamus.

The reticular complex is a thin sheet of neurons, in most places only a few cells thick, which partly surrounds the (dorsal) thalamus (13–32) (see Fig. 1). All axons from the thalamus to the cerebral cortex pass through it, as do all of the reverse projections from the cortex to the thalamus. The intralaminar nuclei of the thalamus, which project very strongly to the striatum, also send their axons through it, as may some of the axons from the globus pallidus that project back to the thalamus.

It is believed that many of the axons that pass in both directions through the reticular complex give off collaterals that make excitatory synaptic contacts in it (15, 18, 21, 29, 30). If the thalamus is the gateway to the cortex, the reticular complex might be described as the guardian of the gateway. Its exact function is unknown.

Not only is its position remarkable, but its structure is also unusual. It consists largely (if not entirely) of neurons whose dendrites often spread rather extensively in the plane of the nucleus (29). The size of these neurons is somewhat different in different parts of the complex (31). Their axons, which project to the thalamus, give off rather extensive collaterals that ramify, sometimes for long distances, within the sheet of the reticular complex (19, 29). This is in marked contrast with most of the nuclei of the thalamus, the principal cells of which have few, if any, collaterals either within each nucleus or between nuclei. The nuclei of the thalamus (with the exception of the intralaminar nuclei) keep themselves to themselves. The neurons of the reticular complex, on the other hand, appear to communicate extensively with each other. Moreover, it is characteristic of them that they fire in long bursts at a very rapid rate (25).

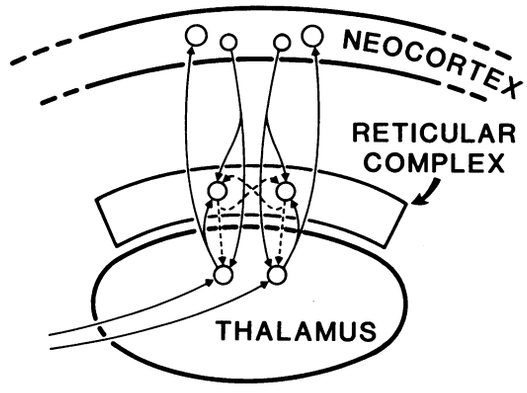

FIG. 1. The main connections of the reticular complex, highly diagramatic and not at all to scale. Solid lines represent excitatory axons. Dashed lines show GABAergic (inhibitory) axons. Arrows represent synapses.

An even more remarkable property of reticular neurons concerns their output. Whereas all of the output neurons of the thalamus make type I synapses and appear to be excitatory, many (if not all) of the neurons in the reticular complex appear to be GABAergic (GABA = γ-aminobutyric acid) and thus almost certainly inhibitory (26–28). The excitation in the complex must come almost exclusively from the activity of the various axons passing through it.

Both the input and the output of the complex are arranged topographically (16, 29, 30, 32). It seems likely that if a particular group of axons going from the thalamus to the cortex passes through a small region of the reticular complex, the reverse projection probably passes through or near that same region. There may well be a rough map of the whole cortex on the reticular complex, though how precise this map may be is not known. It should be remembered, however, that the spread of the receiving dendrites of the reticular nucleus is quite large.

The projection of the reticular complex to the thalamus is also not random. Though any individual axon may spread fairly widely, there is a very crude topography in the arrangement. The projection from any one part of the reticular complex probably projects to that part of the thalamus from which it receives input as well as other neighboring parts. The exact nature of these various mappings would repay further study.

The neurons of the reticular complex project to the (dorsal) thalamus. The evidence suggests that they mainly contact the principal (relay) cells of the thalamus (22). What effect does the reticular input have on the behavior of the cells in the dorsal thalamus?

Obviously this is a crucial question. Let us consider two oversimplified but contrasting hypotheses. The first is that the main effect of the reticular complex is inhibitory. This would lead to the following general picture. The traffic passing through the reticular complex will produce excitation. Let us assume that one patch of the complex is more excited than the rest because of special activity in the thalamo-cortical pathways. The effect of this will be 2-fold. That region will tend to suppress somewhat the other parts of the reticular complex, because of the many inhibitory collaterals. It will also suppress the corresponding thalamic region. These two effects will damp down the thalamus in its most active region and have the opposite effect (since the inhibition from the reticular complex will be reduced there) on the remaining parts. The total effect will be to even out the activity of the thalamus. This is not a very exciting conclusion. The function of the reticular complex would be to act as an overall thermostat of thalamic activity, making the warm parts cooler and the cool parts warmer.

The second hypothesis is just the opposite. Let us assume that the effect of the reticular complex on the dorsal thalamus is mainly excitation in some form or other. Then we see that, once again, an active patch in the complex will tend to suppress many other parts of the complex. This time, however, the effect will be to heat up the warmer parts of the thalamus and cool down the cooler parts. We shall have positive feedback rather than negative feedback, so that "attention" will be focused on the most active thalamo-cortical regions.

How can GABAergic neurons produce some sort of excitatory effect on the relay cells of the thalamus? One possibili-

ty is that they might synapse only onto the local inhibitory neurons in the thalamus. By inhibiting these inhibitory cells they would thereby increase the effect of incoming excitation on the relay cells.

This is certainly possible but the anatomic evidence (22) suggests that in the main the neurons of the reticular complex project directly to the thalamic relay neurons. One would expect that this would inhibit these neurons. We must therefore ask if thalamic neurons show any unusual types of behavior.

Properties of Thalamic Neurons

The recent work of Llinás and Jahnsen (33–35) on thalamic slices from the guinea pig confirms that this is indeed the case. Their papers should be consulted for the detailed results, which are complicated, but, broadly, they show that all thalamic neurons display two relatively distinct modes of behavior. When the cell is near its normal resting potential (say, −60 mV) it responds to an injected current by firing (producing axonal spikes) at a fairly modest rate, usually between 25 and 100 spikes per second. The rate increases with the value of the current injected.

If, on the other hand, the negative potential of the membrane is increased somewhat (that is, if the cell is hyperpolarized) to, say, −70 mV, then a neuron responds to an injected current, after a short delay, with a spike or a *short fast burst of spikes*, firing briefly at rates nearer *300 spikes per second*. Moreover, the after-effect of this burst is that, even though the injected current is maintained constant, the cell will not produce a further burst for a time of the order of 80–150 msec. Jahnsen and Llinás (34, 35), by means of many elegant controls, have shown that this behavior depends on a number of special ion channels, including a Ca^{2+}-dependent K^+ conductance.

Thus, it is at least possible that the effect of the GABAergic neurons of the reticular complex on the thalamic relay cells is to produce a brief burst of firing in response to incoming excitations, followed by a more prolonged inhibition. Whether this is actually the effect they produce in natural circumstances remains to be seen, since it is not easy to deduce this with certainty from the results of Jahnsen and Llinás on slices.

The Searchlight Hypothesis

What do we require of a searchlight? It should be able to sample the activity in the cortex and/or the thalamus and decide "where the action is." It should then be able to intensify the thalamic input to that region of the cortex, probably by making the active thalamic neurons in that region fire more rapidly than usual. It must then be able to turn off its beam, move to the next place demanding attention, and repeat the process.

It seems remarkable, to say the least, that the nature of the reticular complex and the behavior of the thalamic neurons fit this requirement so neatly. The extensive inhibitory collaterals in the reticular complex may allow it to select a small region that corresponds to the most active part of the thalamo-cortical maps. Its inhibitory output, by making more negative the membrane potential of the relevant thalamic neurons, could allow them to produce a very rapid, short burst and also effectively turns them off for 100 msec or so. This means that the reticular complex will no longer respond at that patch and its activity can thus move to the next most active patch. We are thus led to two plausible hypotheses:

(*i*) *The searchlight is controlled by the reticular complex of the thalamus.*

(*ii*) *The expression of the searchlight is the production of rapid firing in a subset of active thalamic neurons.*

So far I have lumped the perigeniculate nucleus (17–24) in with the reticular nucleus proper which adjoins it. It seems probable that the lateral geniculate nucleus (which in primates projects mainly to the first visual area of the cortex) sends collaterals of its output to the perigeniculate nucleus, while the rest of the dorsal thalamus sends collaterals to the reticular nucleus proper (20). This suggests that there may be at least two searchlights: one for the first visual area and another for all of the rest. Indeed, there may be several separate searchlights. Their number will depend in part on the range and strength of the inhibitory collaterals within the reticular complex. Clearly, much more needs to be known about both the neuroanatomy and the neurophysiology of the various parts of the reticular complex.

Malsburg Synapses

We must now ask: what could the searchlight usefully do? Treisman's results (5–8) suggest that what we want it to do is to form *temporary* "conjunctions" of neurons. One possibility is that the conjunction is expressed merely by the relevant neurons firing simultaneously, or at least in a highly correlated manner. In artificial intelligence the problem would be solved by "creating a line" between the units. There is no way that the searchlight can rapidly produce new dendrites, new axons, or even new axon terminals in the brain. The only plausible way to create a line in a short time is to strengthen an existing synapse in some way. This is the essence of the idea put forward in 1981 by von der Malsburg in a little known but very suggestive paper.* After describing the conjunction problem in general terms he proposed that a synapse could alter its synaptic weight (roughly speaking, the weight is the effect a presynaptic spike has on the potential at the axon hillock of the postsynaptic cell) on a fast time scale ("fractions of a second"). He proposed that when there was a strong correlation between presynaptic and postsynaptic activity, the strength of the synapse was temporarily increased—a dynamic version of Hebb's well-known rule (36)—and that with *un*correlated pre- and postsynaptic signals the strength would be temporarily decreased below its normal resting value.

Notice that we are not concerned here with *long-term* alterations in weight, as we would if we were considering learning, but very short-term *transient* alterations that would occur during the act of visual perception. The idea is not, however, limited to the visual system but is supposed to apply to all parts of the neocortex and possibly to other parts of the brain as well.

Most previous theoretical work on neural nets does not use this idea, though there are exceptions (37, 38). The usual convention is that while a net, or set of nets, is *performing*, the synaptic weights are kept constant. They are only allowed to alter when *learning* is being studied. Thus, von der Malsburg's idea represents a rather radical alteration to the usual assumptions. I propose that such (hypothetical) synapses be called Malsburg synapses. Notice that in the cortex the number of synapses exceeds the number of neurons by at least three orders of magnitude.

Let us then accept for the moment that Malsburg synapses are at least plausible. We are still a long way from knowing the exact rules for their behavior—How much can their strength be increased? What exactly determines this increase (or decrease) of strength? How rapidly can this happen? How does this temporary alteration decay?—to say nothing of the molecular mechanisms underlying such changes.

*von der Malsburg, C. (1981) Internal Report 81-2 (Department of Neurobiology, Max-Planck-Institute for Biophysical Chemistry, Goettingen, F.R.G.).

In spite of all of these uncertainties it seems not unreasonable to assume that the effect of the searchlight is to activate Malsburg synapses. We are thus led to a third hypothesis.

(iii) The conjunctions produced by the searchlight are mediated by Malsburg synapses, especially by rapid bursts acting on them.

We still have to explain exactly how activated Malsburg synapses form associations of neurons. This is discussed by von der Malsburg in his paper in some detail but most readers may find his discussion hard to follow. His argument depends on the assumption that the system needs to have more than one such association active at about the same time. He describes at some length how correlations, acting on Malsburg synapses, can link cells into groups and thus form what he calls topological networks. What characterizes one such cell assembly is that the neurons in it fire "simultaneously," an idea that goes back to Hebb (36). von der Malsburg suggests that two kinds of signal patterns can exist in a topological network: waves running through the network or groups of cells switching synchronously between an active and a silent state. He next discusses how a set of cells rather than a single cell might form what he calls a "network element." Finally, after an elaborate development of this theme he broaches the "bandwidth problem." In simple terms, how can we avoid these various groups of cells interfering with each other?

Cell Assemblies

The cell assembly idea is a powerful one. Since a neuron can usually be made to fire by several different combinations of its inputs, the *significance* of its firing is necessarily ambiguous. It is thus a reasonable deduction that this ambiguity can be removed, at least in part, by the firing pattern of an *assembly* of cells. This arrangement is more economical than having many distinct neurons, each with very high specificity. This type of argument goes back to Young (39) in 1802.

There has been much theoretical work on what we may loosely describe as associative nets. The nets are usually considered to consist of neurons of a similar type, receiving input, in most cases, from similar sources and sending their output mainly to similar places. If we regard neurons (in, say, the visual system) as being arranged in some sort of hierarchy, then we can usefully refer to such an assembly as a *horizontal* assembly.

von der Malsburg's ideas, however, permit another type of assembly. In his theory a cell at a higher level is associated with one at a lower level (we are here ignoring the direction of the connection), and these, in turn, may be associated with those at a still lower level. (By "associated with" I mean that the cells fire approximately simultaneously.) For example, a cell at a higher level that signified the general idea "face" would be temporarily associated, by Malsburg synapses, with cells that signified the parts of the face, and, in turn, perhaps with their parts. Such an assembly might usefully be called a *vertical* assembly. It is these vertical assemblies that have to be constructed anew for each different visual scene, or for each sentence, etc. Without them it would be a difficult job to unite the higher level concepts with their low level details in a rather short time. This idea is reminiscent of the K lines of Minsky.†

The idea of *transient* vertical assemblies is a very powerful one. It solves in one blow the combinational problem—that is, how the brain can respond to an almost unlimited number of distinct sentences, passages of music, visual scenes, etc. The solution is to use *temporary* combinations of a subset of a much more limited number of units (the 10^{12} or so neurons in the central nervous system), each new combination being brought into action as the circumstances demand and then largely discarded. Without this device the brain would either require vastly more neurons to do the job or its ability to perceive, think, and act would be very severely restricted. This is the thrust of von der Malsburg's arguments.

A somewhat similar set of ideas about simultaneous firing has been put forward by Abeles. His monograph (40) should be consulted for details. He proposes the concept of "synfire chains"—sets of cells, each set firing synchronously, connected in chains, which fire sequentially. He gives a plausible numerical argument, based mainly on anatomical connectivity, which suggests that to establish a functioning synfire chain only a few (perhaps five or so) synapses would be available at any one neuron. Since this is such a small number he deduces that the individual synapses must be strengthened (if these five synapses by themselves are to fire the cell) perhaps by a factor of 5 or so. However, he gives no indication as to how this strengthening might be done.

Abeles' argument stresses the importance to the system of the *exact time of firing* of each spike, rather than the *average* rate of firing, which is often taken to be the more relevant variable. This exact timing is also an important aspect of von der Malsburg's ideas. These arguments can also be supported by considering the probable values of the passive cable constants of cortical dendrites. Very rough estimates (for example, $\tau = 8$ msec, $X = \lambda/5$) suggest that inputs will not add satisfactorily unless they arrive within a few milliseconds of each other (see figure 3.18 in ref. 41).

Notice the idea that a cell assembly consists of neurons firing simultaneously (or at least in a highly correlated manner) is a very natural one, since this means that the impact of their joint firing on *other* neurons, elsewhere in the system, will be large. The content of the cell assembly—the "meaning" of all of the neurons so linked together—can in this way be impressed on the rest of the system in a manner that would not be possible if all of the neurons in it fired at random times, unless they were firing very rapidly indeed. Therefore, our fourth hypothesis follows.

(iv) Conjunctions are expressed by cell assemblies, especially assemblies of cells in different cortical regions.

It should not be assumed that cell assemblies can only be formed by the searchlight mechanism. Some important ones may well be laid down, or partly laid down, genetically (e.g., faces?) or be formed by prolonged learning (e.g., reading letters or words?).

It is clear that much further theoretical work is needed to develop these ideas and make them more precise. If the members of a vertical assembly fire approximately synchronously, exactly how regular and how close together in time do these firings have to be? Are there special pathways or devices to promote more simultaneous firing? Are dendritic spikes involved? How does one avoid confusion between different cell assemblies? Do neurons in *different* cell assemblies briefly inhibit each other, so that accidental synchrony is made more difficult? Etc.

The idea that the dorsal thalamus and the reticular complex are concerned with attention is not novel (19, 42, 43). What is novel (as far as I know) is the suggestion that they control and express the internal attentional searchlight proposed by Treisman (5–8), Julesz (9, 10), Posner (44, 45), and others. For this searchlight at least two features are required. The first is the rapid movement of the searchlight from place to place while the eyes remain in one position, as discussed above. There is, however, another aspect. The brain must know what it is searching *for* (the green *T* in the example given earlier) so that it may know when its hunt is successful. In other words, the brain must know *what* to attend to. That aspect, which may involve other cortical areas

†Minsky, M. (1979) Artificial Intelligence Memo No. 516 (Artificial Intelligence Laboratory, Massachusetts Institute of Technology, Cambridge, MA).

such as the frontal cortex, has not been discussed here. The basic searchlight mechanism may depend on several parts of the reticular complex, but these may be influenced by top-down pathways, or by other searchlights in other parts of the reticular complex, which may be partly controlled by which ideas are receiving attention. An important function of the reticular complex may be to limit the number of subjects the thalamus can pay attention to at any one time.

Experimental Tests

These will not be discussed here in detail. It suffices to say that many of the suggestions, such as the behavior of the dorsal thalamus and the reticular complex, are susceptible to fairly direct tests. The exact behavior of reticular neurons and thalamic neurons is difficult to predict with confidence, since they contain a number of very different ion channels. Experiments on slices should therefore be complemented by experiments on animals. Obviously, most of such experiments should be done on alert, behaving animals, if possible with natural stimuli. An animal under an anesthetic can hardly be expected to display all aspects of attention. Various psychophysical tests are also possible.

Other aspects of these ideas, such as the behavior of Malsburg synapses, may be more difficult to test in the immediate future. It seems more than likely that dendritic spines are involved, both the spines themselves and the synapses on them (46, 47).

The existence and the importance of rapid bursts of firing can also be tested. Such bursts, followed by a quiet interval, have been seen in neurons in the visual cortex of a curarized, unanesthetized and artificially respired cat when they respond to an optimal visual signal [see figure 1 in Morrell (48)]. It is unlikely that the two systems—the rapid-burst system and the slow-firing system—will be quite as distinct as implied here. In fact, as von der Malsburg has pointed out, one would expect them to interact.

Thus, all of these ideas, plausible though they may be, must be regarded at the moment as speculative until supported by much stronger experimental evidence. In spite of this, they appear as if they might begin to form a useful bridge between certain parts of cognitive psychology, on the one hand, and the world of neuroanatomy and neurophysiology on the other.

Note Added in Proof. Recent unpublished experimental work suggests that the reticular complex may produce bursts of firing in some thalamic neurons but merely an increase of firing rate in others.

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