

in terms of EEG, no different from the consequence of severing ponto-mesencephalic sections in the *cerveau isolé* preparation.

Let me begin by addressing the second type of section, the one obtained about four millimeters higher than the plane of trigeminal-nerve entry. Although not as damaging as the extensive lesions that cause coma by damaging this neighborhood, such a section probably had at least three consequences: First, it damaged acetylcholine nuclei located at the level of the section and disrupted upward projections from them; second, it damaged downward cortical projections and thus precluded cortical signaling from penetrating the tegmental region of the upper pons; third, it damaged part of the parabrachial nucleus. Individually or in combination, these effects would disrupt the normal process of consciousness, for instance, by interfering with the feeding of signals to proto-self structures from both lower and higher structures. The results seen in the cat are thus consonant with the hypothesis.

Even more interesting, however, are the results of the section performed four millimeters below, at trigeminal entry level. Although we have no way of knowing what was the cats' resulting state of consciousness, their EEG profile became one of permanent wakefulness. The interpretation of this finding is as follows: First, the section precluded the sleep-producing effects of the nucleus tractus solitarius, which is located below the level of the section and is known to have hypnogenic effects; second, the section did *not* damage any of the structures which constitute the foundation of the proto-self, thus permitting signals from the cortex and thalamus to enter the critical region and alter the proto-self state. This would be possible since the animal would continue to process visual stimuli, thus activating thalamocortical and tectal regions. The visual accommodation apparatus as well as vertical eye movements would have remained intact, past memory could still be evoked from cortical structures, and all of these processes would have signaled normally to the intact brainstem region located above the section of cut. Finally, chemical infor-

mation relating to overall body state would still be relayed directly to the central nervous system via the hypothalamus and the subformal organs, and the consequences of their signaling could be brought down to the proto-self structures located above the plane of section. In short, unlike patients with coma-producing lesions, and unlike cats with sections located either slightly higher, or much higher, at the ponto-mesencephalic junction, cats with this particular section would keep intact all of the structures necessary to implement the proto-self, as well as residual means of signaling ongoing organism changes toward those structures. This situation, combined with the lack of any sleep-inducing influence from below, would explain the awake EEG, and would account for maintained wakefulness and even attention. Whether or not normal consciousness would still be possible is a question that cannot be decided on the basis of this experiment and certainly will never be answered in human beings since no natural lesion will be sufficiently circumscribed to produce such a selective defect.²²

Reconciling Facts and Interpretations

Although they ostensibly address unrelated functions, I suspect the results of the two strands of research on the reticular formation are connected at a deep level. The two strands of studies have been motivated by different questions, but in my framework their interconnectivity begins to be visible. As an example, consider my interpretation of a recent finding in an experiment by Munk, Singer, and colleagues.²³ Munk and colleagues were able to produce in cats the sort of "desynchronized" EEG with "local synchronization" characteristics that is indicative of wakeful and attentive states. They did so by aiming their electrical stimulation at the midbrain reticular formation. However, they indicated in a footnote that they *actually stimulated the parabrachial nucleus*, something revealed by the autopsy of their experimental animals (at autopsy it is possible to follow the tracks of the stimulating electrodes and they had been placed in and about the

parabrachial nucleus). In short, electrical stimulation of a nucleus of the reticular formation that has, heretofore, been associated with autonomic regulation of heart, lungs, and gut, as well as with body states such as pain, produced an electrical cortical state that is characteristic of wakefulness and attention and traditionally associated with the classical reticular nuclei.

Another experimental connection between the two strands comes from work in my laboratory in the area of emotion. In a series of studies involving healthy human subjects without neurological disease (performed in collaboration with Antoine Bechara, Thomas Grabowski, Hanna Damasio, and Josef Parvizi), we have been able to induce a variety of emotions experimentally and demonstrate, using positron emission tomography (PET), that brain-stem structures within the upper reticular formation become remarkably active with some emotions but not others.

Might this activation be a consequence of the attentive state in which the subjects need to be in order to experience these emotions? If so, our finding would be interesting but not new, given what we know from the traditional studies of the reticular formation, and given that a previous study by Per Roland and colleagues revealed activation of the reticular formation during a task requiring attention.²⁴ Attention alone, however, cannot explain our findings. To begin with, the control task we used demands a comparable degree of attention to imagery. If the finding we attribute to emotion had been due to attention, the activation would have vanished during the subtraction of the control task. Moreover, the findings were different for different emotions. We found maximal brain-stem activations for emotions such as sadness and anger, and little activation for an emotion like happiness. Yet, the subjects were performing the same procedure for all emotions, and there is nothing to suggest that the demand for internal attention varied across these emotions. It is likely that the upper reticular activations were tied to the neural process required for processing some specific emotions and producing the eventual feeling of those emotions.

This finding adds to the evidence suggesting that the structures of the reticular formation, traditionally linked to the control of sleep-wakefulness cycles and attention, are also linked to emotion and feeling, as well as to the representation of internal milieu and visceral states and autonomic control. There is abundant evidence that this is the case, especially regarding the periaqueductal gray (PAG). The repertoire of body changes that define several emotions is in fact controlled by PAG.²⁵ In brief, the structures of the so-called reticular formation of the upper pons and midbrain can be credibly linked to the notion of proto-self that I have advanced previously. That may well be the fundamental reason why they can also be associated with seemingly diverse, but nonetheless closely interrelated, functions such as emotion; attention; and, ultimately, consciousness.

Another intriguing finding from my research group comes from a study carried out in collaboration with Josef Parvizi and Gary W. Van Hoesen.²⁶ The study involved a detailed mapping of reticular-formation nuclei in patients with Alzheimer's disease as well as in age-matched normal controls, and it revealed a new and surprising finding: most patients with advanced Alzheimer's disease have a severe destruction of their parabrachial nucleus, on both the left and right sides of the brain stem. The parabrachial nucleus was damaged in *all* of the patients with early-onset Alzheimer's disease, an especially severe variant of the disease, and in 80 percent of the patients with late-onset Alzheimer's.

Given that patients with advanced Alzheimer's disease have a marked impairment of consciousness (see chapter 3), it is reasonable to wonder if parabrachial damage might be related to the decline in consciousness. Certainly their decline in consciousness cannot be explained by the well-known involvement of the entorhinal cortex and nearby temporal cortices.²⁷ Unfortunately, it is not possible at this point to go beyond wondering, because there are too many sites of focal pathology in Alzheimer's disease for one to be entirely comfortable with the correlations between particular impairments and particular sites of neural degeneration. For instance, posterior cingulate

cortices and medial parietal association cortices are also heavily compromised in Alzheimer's, and they are candidate sites for second-order maps, as previously indicated.²⁸

In conclusion, I see one powerful fact emerging about the critical region of the brain stem we have been discussing: it is simultaneously engaged in processes concerning wakefulness, homeostatic regulation, emotion and feeling, attention, and consciousness. The functional overlap may appear random at first glance, but upon reflection, and in the framework developed in the previous chapters, it appears sensible. Homeostatic regulation, which includes emotion, requires periods of wakefulness (for energy gathering); periods of sleep (presumably for restoration of depleted chemicals necessary for neuronal activity²⁹); attention (for proper interaction with the environment); and consciousness (so that a high level of planning of responses concerned with the individual organism can eventually take place). The body-relatedness of all these functions and the anatomical intimacy of the nuclei subserving them are quite apparent.

This view is compatible with the classical idea that there is a device in the upper brain-stem region capable of creating special types of electrophysiological states in the thalamus and cortex. In fact, my proposal incorporates the classical idea but is distinctive in the following ways: first, it offers a biological rationale for the origin and anatomical placement of the device; and second, it posits that the actions of the device, as currently described, contribute importantly to the state of consciousness but do not produce the subjective aspect that defines consciousness.

ASSESSING STATEMENT NUMBER TWO: EVIDENCE FOR A ROLE OF SECOND-ORDER STRUCTURES IN CONSCIOUSNESS

Let us now turn to statement number two, which concerns damage to regions presumed to participate in the second-order neural pattern that subtends core consciousness: the cingulate gyrus, thalamic nuclei, and superior colliculi. As you read these comments recall, again,

my injunctions regarding phrenology. I am not suggesting that any of these regions is solely responsible for the neural pattern that is critical for consciousness to emerge. In all likelihood, the critical neural pattern is based on cross-regional interactions.

My first choice for second-order structure is a vast portion of the cerebral cortex known as the cingulate cortex. Located near the midline, one cingulate cortex per hemisphere, this cortex is divided into numerous cytoarchitectonic regions. (See appendix figures A.4 and A.5.) In its anterior section, the structure is dominated by areas 24 and 25, immediately visible around the anterior part of the corpus callosum. Two other cytoarchitectonic areas, however, respectively areas 33 and 32, in spite of their remarkable size, are hardly visible because they are embedded in sulci. The posterior part of the ~~cerebral cortex~~ ^{CINGULATE} is constituted by area 23, quite visible on the large crown of the gyrus, and by areas 31, 29, and 30, which are again quite extensive but embedded in sulci and thus hidden. CING.

The easiest way to summarize the known functions of the cingulate cortex is to say that they comprise an odd combination of sensory and motor roles. The cingulate is a massively somatosensory structure which receives inputs from all the divisions of the somatosensory system described in chapter 5. This includes not only a remarkable quantity of internal milieu and visceral signals but also important signals from the musculoskeletal division. Yet the cingulate is also a motor structure involved, both directly and indirectly, in the execution of a large variety of complex movements, from those that have to do with vocalization to those that involve the limbs, alone or in synergy, and to those that involve viscera. But this is not all. The cingulate is also clearly involved in the processes of attention; it is clearly involved in processes of emotion; and it is clearly involved in *consciousness*. This overlap of functions is remarkable and reminiscent of another sector of the central nervous system: the upper brain stem.

It is reasonable to say that we know both a lot and not too much about the cingulate. In spite of a number of remarkable neuroanatomical studies, the intrinsic anatomy of the cingulate and many of its connections to other regions remain uncharted territory.³⁰ This is also

true of the neurophysiology of the cingulate, which remains somewhat mysterious, especially concerning the posterior sector. One explanation for this panorama of ignorance has to do with the paucity of naturally occurring bilateral cingulate lesions in humans. Lesions are quite rare, as far as the anterior cingulate is concerned, and exceedingly rare in the posterior cingulate as well. Consider that not one single case has ever been described of a bilateral lesion of the cingulate involving *all* the cytoarchitectonic regions I enumerated above.

Under the circumstances, we should tread prudently. We know for a fact that epileptic seizures arising in the cingulate cortex are characterized by loss of consciousness—periods of absence that are actually longer than those caused by regular, non-cingulate seizures. A number of functional neuroimaging studies have also yielded some important findings. Situations in which consciousness is suspended or diminished, such as slow-wave sleep, hypnosis, and some forms of anesthesia, are associated with *reduced activity* in the cingulate cortex; on the other hand, REM sleep, as well as myriad attention paradigms are associated with *increased activity* in the cingulate cortex.³¹

In both lesion studies as well as functional imaging studies, the cingulate has been associated with emotion, attention, and autonomic control.³² Bilateral anterior lesions of the cingulate cause the condition known as akinetic mutism. As we saw in the case of L (chapter 3), patients with bilateral damage to the cingulate cortices have impaired consciousness although they remain awake. The patients' condition is best described as suspended animation, internally as well as externally, and this is the reason why the patients are described as akinetic and mute. From the literature and from my own observations, I can say confidently that bilateral anterior damage to the cingulate disrupts both core consciousness and extended consciousness while preserving wakefulness. We should note, however, that although the affected patients do not recover an entirely normal mind, they do recover core consciousness in a matter of months. Their recovery might be due to the preservation of both posterior cingulate regions. It is possible that bilateral damage to the posterior aspect of the cingulate causes per-

manent damage, but I have only studied one convincing case. Be that as it may, it is reasonable to venture that bilateral damage to the entire cingulate is likely to disrupt consciousness remarkably, perhaps even permanently. Of the two large sectors of the cingulate, anterior and posterior, I would also venture that the posterior sector is the most indispensable, although I imagine that normal operations require both sectors to work in concert.

I should add that patients with damage to a region just behind and around the posterior cingulate also have disturbances of consciousness. The region is medial and parietal, a combination of retrosplenial and cuneus territories. Cytoarchitectonic areas 31, 7, and 19 are part of this region. Patients with bilateral damage to this area have a profound disturbance of consciousness. Their impairments are not as marked as those seen in coma, but are comparable to the impairments I have just described for bilateral cingulate damage.

Just as is the case with patients with bilateral cingulate damage, patients with bilateral medial parietal damage are awake in the usual sense of the term: their eyes can be open, and their muscles have proper tone; they can sit or even walk with assistance; but they will not look at you or at any object with any semblance of intention; and their eyes may stare vacantly or orient toward objects with no discernible motive. These patients cannot help themselves. They volunteer nothing about their situation and they fail to respond to virtually all the examiners' requests. Attempts to engage them in conversation are rarely successful, the results being erratic at best. We can coax them into looking briefly at an object, but the request will not engender anything else in terms of productive reaction. These patients react no differently to friends and family than they do to physicians and nurses. The notion of zombie-like behavior could perfectly well have come from the descriptions of these patients, although it did not.

The most common cause of involvement in medial parietal region is Alzheimer's disease. Outside of degenerative diseases, bilateral parietal damage is not a frequent presentation of stroke. The case of bilateral parietal damage I most vividly remember was caused by fairly

symmetrical metastases from colon cancer—to picture what the patient looked like, imagine the state of absence automatism described in chapter 3 but in slow motion and without an end in sight. Head injury can cause the condition, too. The renowned British neurologist Macdonald Critchley mentioned one such case in his landmark monograph on the parietal lobes.³³

Reflection on the anatomical specifications of the cingulate cortex indicates that it is an excellent candidate for the sort of second-order structure I proposed earlier. Its different subregions and the massiveness of its somatosensory inputs can give rise to perhaps the most “integrated” view of the entire body state of an organism at any given time. But since the cingulate cortices are also privy to signals from the main sensory channels—the appearance of an object can be reported to the cingulate easily via both thalamic projections and direct projections from higher-order cortices in inferotemporal, polar temporal, and lateral parietal regions—the cingulate could help generate a neural pattern in which the relationship between the appearance of an object and the modifications undergone by the body could be mapped in the proper causal sequence. The cingulate might actually make the critical contribution to the “feeling of knowing,” the special, high-order feeling that defines core consciousness.

THE REASONS WHY the superior colliculi also qualify as a structure contributing to second-order patterns are as follows. The superior colliculi are multilayered structures which receive a multiplicity of sensory inputs from an assortment of modalities, integrate signals in a complicated fashion across their several layers, and communicate the resulting outputs to a variety of brain-stem nuclei, the thalamus, and the cerebral cortex.³⁴ For instance, the superior colliculi receives visual information directly from the retina in its top layer, and, just a few layers deeper, it also receives information from visual cortices; it receives auditory information from the inferior colliculi located just below, and massive somatosensory information (including visceral information) from varied brain-stem nuclei.

The integrative activity of the superior colliculi is aimed at orienting the eyes, the head and neck, and the ears (in creatures that move them) toward the source of a visual or auditory stimulus so that optimal object processing can take place. In the course of this activity, the superior colliculi map the temporal appearance and spatial position of an object as well as varied aspects of body state. It is conceivable that one of their seven layers of cells might be dedicated to mapping a second-order neural pattern describing the object-organism relationship based on the data they have available. The result would influence classical reticular nuclei (and subsequent cortical processing, via the intralaminar nuclei of the thalamus) as well as monoamine and acetylcholine nuclei. In species with little cortical development this might be the source of the simple form of core consciousness that may accompany the execution of attentive behaviors. I hasten to add that, in the case of humans, there is no evidence that the superior colliculi can support core consciousness in the absence of thalamic and cingulate structures, even assuming intactness of the brain-stem proto-self structures.³⁵

FINALLY, THERE IS the matter of the thalamus. Reviewing the neuroanatomy and neurophysiology of the thalamus is outside the scope of this book. Just as is the case for the cerebral cortex and brain stem, the thalamus is the subject for entire books, not paragraphs. For the sake of my argument, however, I can say that the thalamus gets first-hand “reports” of the sequential engagement of the varied structures representing both the characters and the events in the would-be primordial plot. The thalamus could signify the object-organism relationship in implicit form and follow that by creating more explicit neural patterns in cingulate cortices and somatosensory cortices. Some thalamic nuclei, such as the reticular nucleus and the pulvinar, would be critical in this process. The idea that the thalamus is related to consciousness is based on credible experimental evidence in animals, on the result of thalamic lesions, and on the likelihood that abnormal discharges in absence seizures, during which consciousness is

disrupted, originate in the thalamus.³⁶ The current evidence on the thalamus, however, is insufficient to address the hypothesis with any degree of specificity, although it is in accordance with the overall prediction. One must be content with concluding that bilateral damage to the thalamus disrupts consciousness for certain.

IN CLOSING, I will add a bit of curious and potentially relevant evidence. In the summer of 1998, my colleagues and I had a collective recognition experience when a visiting lecturer came to our department to give a talk, not about consciousness but rather about neuroimaging studies in children. In his talk the speaker included a set of images of PET scans obtained shortly after birth and within the first few months of life. Early on, the structures which are remarkably active in those newborn brains, almost as isolated islands in a sea of neuroimaging silence, are the brain stem and hypothalamus, the somatosensory cortices, and the cingulate. As you can see, the set of activated structures entirely matches those needed for the proto-self and second-order maps. The functional maturity of these structures at birth is noteworthy. Given that other brain systems have also been in full swing, e.g., auditory, the activation suggests a considerable functional precedence. The next structures to show up in PET scans, a few months later, are the ventromedial frontal lobe and the amygdala. Several of us looked at each other knowingly, and the speaker may have wondered why.³⁷

ASSESSING THE OTHER STATEMENTS

Now, let us turn to the remaining statements, which concern brain sites whose damage should not cause impairment of core consciousness: the hippocampus, the higher-order cortices of temporal and frontal lobes, and the early sensory cortices of vision and hearing.

To make a long story short: bilateral damage to any of these areas individually leaves core consciousness unscathed. Sense of self and knowing still operate efficiently regarding any object that can be

properly mapped. This fact underscores the following situation: proto-self and second-order maps depend largely on one set of paramidline structures—the brain stem, hypothalamic, basal forebrain, and the thalamic nuclei, as well as the centrally located cingulate cortices; while mapping of objects depends largely on less centrally located sensory cortices distributed over the cortical mantle. The left and right halves of “self and knowing” structures sit centrally, just across from each other, and are often damaged together by the same pathological cause; the left and right halves of the structures on which object mapping depends sit farther apart and are often damaged independently.

We can say with confidence that bilateral damage to the hippocampus, or to the entire anterior temporal lobe or to the entire lateral temporal lobe or to most of the medial and inferior temporal lobe does *not* cause impairments of core consciousness. HM and David, two patients we discussed in chapter 4, indicate this fact unequivocally. In fact, not even a combination of all these lesions disrupts core consciousness. Bilateral damage to the amygdalae also leaves core consciousness intact as patient S (chapter 2) shows so clearly. Needless to say, unilateral damage to any of these structures does not cause impairment of consciousness, either.

The cortege of impairments caused by all these lesions that leave consciousness intact is legend. Profound alterations of learning, memory, and language are the well-known results of such lesions. But in spite of those remarkable impairments, the patients remain keenly aware of self and surroundings, their core consciousness unscathed. They are perfectly conscious, and, more often than not, they are quite conscious of their own impairments. They are the very conscious owners of disrupted memories and broken language.

Likewise, bilateral or unilateral damage to auditory cortices, visual cortices, and prefrontal cortices does not impair core consciousness at all. In essence, the patients' ability to perceive and recognize stimuli along the auditory or visual channel is impaired, the ability to create internal images in those sensory modalities is also impaired, and there

are selective memory defects pertinent to the sensory channel that has been compromised. Yet core consciousness goes on normally outside of the affected sensory modality.

Bilateral damage to early visual cortices is generally restricted to a subsector and causes visual loss either in part of the visual fields or in the entirety of the visual fields. Often it also creates one of many astonishing conditions in which visual processing is disrupted. For instance, the ability to see color may be lost across the entire visual field or in a part of it, while the ability to see movement, depth, and shape remains intact (a condition known as achromatopsia); or the ability to recognize previously familiar objects may be lost, although appreciation of the physical structure of the object remains intact (the condition known as agnosia, which we discussed previously); or the ability to survey the visual field in a harmonious and attentive manner may vanish (in what is known as Balint's syndrome).³⁸ In all of these instances, core consciousness remains intact; the patient is able to process normally any aspect of cognition except for the selectively disrupted aspects of visual processing. That the patients are keenly aware of what they can no longer do indicates that the "general" process of core consciousness has been spared. Of equal interest is the fact that some of these patients may retain certain aspects of nonconscious processing relative to stimuli that they can no longer either perceive or recognize. A strong example of the former occurs in the condition known as blindsight.³⁹ In some patients who have lost vision altogether, as a result of what is often termed cortical blindness, the patients may claim, quite truthfully, not to see any object in their visual field and yet be able, when asked to hazard a pointing finger at the possible location of the object, to move their arm and finger in the correct direction. This indicates that some correct processing is taking place such that the structures in charge of movement can guide the arm and finger in the appropriate direction even if part of the information underlying that process is not made available to the process of consciousness making.

Something along the same lines can happen in similarly blind patients when the damage to visual cortices is especially extensive, in a situation known as Anton's syndrome. The patients may deny, in the manner previously described in anosognosia, that they are blind, but the bizarre claim may have a partial explanation. The patients' eyes remain capable of veering toward objects that are attractive to a visual organism and remain capable of focusing on them. The results of the efforts of that now useless visual-perceptual machinery are of no consequence to the visual cortices themselves but are conveyed nonetheless to structures such as the superior colliculi and the parietal cortices. The brain is still informed of an ongoing set of perceptually related adjustments, probably not unlike those that would occur should the brain still be capable of visual processing.

In a situation in which visual processing is completely absent, the brain constructs a reasonably appropriate account for those perceptual adjustments that are being perceived in consciousness; an account that says, in fact, that seeing an object is in progress. The account is not adequate, of course, but is not entirely irrational, either. In the cases I have seen, such a belief generally wanes within hours, as one might expect. I am persuaded that the complete absence of visual images, actual or recalled, that occurs during the first hours of the event, explains why the patient is fooled. The profound defect in visual imagery impedes the construction of a counterargument.

I have devoted many studies, as well as *Descartes' Error*, to the situation of patients with bilateral damage to the ventromedial prefrontal lobe. I can say confidently that although their ability to decide advantageously and to resonate emotionally with certain issues is impaired, their core consciousness is not. Even bilateral damage to the dorsolateral prefrontal cortices, including the frontal pole, does not cause impairments of core consciousness.⁴⁰ Such damage does alter working memory and consequently affects extended consciousness, but these impairments leave core consciousness intact.

The "negative" evidence reported above is as important to identifying

the brain territories from which consciousness can arise as the “positive” evidence concerning territories that lead to an unequivocal impairment of consciousness. Of the negative evidence just mentioned, I would like to emphasize the facts that bilateral damage to the hippocampus does not impair core consciousness, and that neither does bilateral damage to visual or auditory cortices.

The importance of the negative evidence is as follows: The hippocampus is a recipient of information from several sensory modalities and its circuitry is such that its signals can probably construct, in some fashion, an *n*-order map of the “scene” that results, at each moment, from the organism’s multiple image-making devices. It might be conceived, then, that the hippocampus would be an ideal structure to generate the second-order map I proposed as a basis for core consciousness. This cannot be the case, however, as many studies of patients in whom the hippocampal region is damaged on both sides indicate. A profound learning and memory defect can always be found in those cases, but no impairment of core consciousness ever ensues.

CONCLUSIONS

The foregoing assessment of the available evidence allows us to draw a number of provisional conclusions.

1. Damage to the brain regions presumed to support either the proto-self or the second-order account of the organism-object relationship disrupts core consciousness. Extended consciousness is disrupted as well.
2. The regions which support either the proto-self or the second-order maps have special anatomical characteristics: (a) they are among the phylogenetically older structures of the brain; (b) they are largely located near the midline; (c) none is located on the external surface of the cerebral cortex; and (d) all are involved in some aspect of body regulation or representation.

3. Proto-self and second-order structures constitute a central resource, and their dysfunction causes a general disruption of consciousness for any object. Early sensory structures are involved in processing separate aspects of objects, and thus the disabling of one of those structures, even if extensive, does not affect consciousness in general.
4. The regions whose damage does not cause a disruption of core consciousness constitute, in the aggregate, a larger proportion of the central nervous system than the ensemble of those that do disrupt consciousness.
5. Those same regions (e.g., early sensory cortices, higher-order cortices) are primarily involved in: (a) signaling the objects and the events which come to be known because of core consciousness; (b) holding records pertaining to their experience; and (c) manipulating those records in reasoning and creative thinking.
6. The early sensory structures are also involved in the process of making consciousness. They do so in a different manner—there is only *one set* of structures to support proto-self and second-order maps, while there are *several sets* of early sensory structures, one per sensory modality. The participation of early sensory structures includes: (a) initiating the process by influencing the proto-self structures; (b) signaling to second-order structures; and (c) being the recipients of the modulatory influences consequent to the second-order neural patterns. It is because of the latter influence that the enhancement of the neural patterns which support the object does occur and varied components of the object to be known become integrated.

In short, core consciousness depends most critically on the activity of a restricted number of phylogenetically old brain structures, beginning in the brain stem and ending with the somatosensory and cingulate cortices. The interaction among the structures in this set: (1) supports the creation of the proto-self; (2) engenders the second-order

neural pattern which describes the relationship between the organism (proto-self) and the object; and (3) modulates the activity of object-processing regions which are not part of the set.

The specificity with which I am identifying these critical candidate sites should not be interpreted to mean that I regard any one of them as *the* basis for consciousness. None of the functions outlined above is executed at the level of a single neural site or center, but rather, these functions emerge as a result of cross-regional integrations of neural activity. I envision the sense of self and the enhancement of the object as arising out of the interactions among this set of neural sites and the set of neural sites directly involved in the construction of the object.

The neural pattern which underlies core consciousness for an object—the sense of self in the act of knowing a particular thing—is thus a large-scale neural pattern involving activity in two interrelated sets of structures: the set whose cross-regional activity generates proto-self and second-order maps, and the set whose cross-regional activity generates the representation of the object.

A Remarkable Overlap of Functions

There is a remarkable overlap of biological functions within the structures which support the proto-self and the second-order mappings. Taken individually, these structures are involved in most of the following five functions: (1) regulating homeostasis and signaling body structure and state, including the processing of signals related to pain, pleasure, and drives; (2) participating in the processes of emotion and feeling; (3) participating in processes of attention; (4) participating in the processes of wakefulness and sleep; and (5) participating in the learning process.

The entire quintet of overlaps applies fully to the brain stem and cingulate cortices, and applies in large part to the other structures. The overlaps identified here are a matter of fact, and yet they have not previously been emphasized for several reasons. Perhaps the main reason is that knowledge about one of these brain regions, the brain stem, has been segregated along two distinct strands of research, one

related to the problem of homeostatic regulation and the other related to mechanisms of sleep and attention. The problems and the investigators have been kept apart. Another reason is that the neglect of emotion by neuroscience has retarded the realization that all these regions, from the brain stem to the somatosensory cortices, are critical for the processes of emotion.

It is reasonable to conclude, then, that beyond the above quintet of functions, these areas participate in one additional function: the construction of core consciousness.

The functional overlaps revealed by this survey may appear counterintuitive at first glance, and yet, after reflection on the relevant data they become transparently sensible. First, the overlaps probably result from the function of distinct “families” of contiguous nuclei. Second, notwithstanding their anatomical distinctiveness, the varied families of nuclei are highly interrelated by anatomical connections. Third, the contiguousness and anatomical interrelations which give rise to the functional overlaps are not a mere accident and probably are indicative of the overriding functional roles for the regions.

The plausibility of this idea is strengthened by considering the nature of the functional overlaps at the level of the brain stem. Regarding emotion and attention, the rationale for the functional overlap would be as follows. Emotion is critical for the appropriate direction of attention since it provides an automated signal about the organism’s past experience with given objects and thus provides a basis for assigning or withholding attention relative to a given object. Simple organisms initiate wakeful behavior by having basic image-making capabilities and minimal attention, as a result of which the following happens: first, processing of objects can take place; second, emotion can ensue; third, further enhancement and focusing of attention can occur, or not occur, under the direction of emotion. In organisms capable of consciousness, the above list of events still applies, but the second step would read as follows: “Emotion can ensue and become known to the individual having it.”

It makes expedient, if not necessarily tidy, housekeeping sense that

structures governing attention and structures processing emotion should be in the vicinity of one another. For certain components of these processes, the structures might even be the same, although operating in slightly different modes. Moreover, it also makes good housekeeping sense that all of these structures should be in the vicinity of those which regulate and signal body state. This is because the consequences of having emotion and attention are entirely related to the fundamental business of managing life within the organism, while, on the other hand, it is not possible to manage life and maintain homeostatic balance without data on the current state of the organism's body proper.

How sensible is it for emotion and attention to overlap with core consciousness? The answer is that it is sensible, if we regard consciousness as the most sophisticated means at our disposal to regulate homeostasis and manage life. Nature is an expedient tinkerer and since consciousness is a latter-day means of achieving homeostasis, it would have been convenient for nature to evolve the machinery of consciousness *within, from, and in the vicinity of* the previously available machinery involved in basic homeostasis, in other words, the machinery of emotion, attention, and regulation of body states.

A New Context for Reticular Formation and Thalamus

The above conclusions do not deny, in any way, that some brain-stem structures are involved in wakefulness and attention, and that they modulate the activity of the cerebral cortex via the intralaminar thalamic nuclei, via the non-thalamic cortical projections of monoamines, and via the thalamic projections of acetylcholine nuclei. The issue is that nearby brain-stem structures and perhaps even some of the very same structures have *other* activities, namely, managing body states and representing current body states. Those activities are not incidental to the brain stem's well-established activation role: *they may be the reason why such an activation role has been maintained evolutionarily and why it is primarily operated from that region.*

In short, I have no problem with the roles that have been traditionally assigned to the brain stem's "ascending reticular activating system," and to its extension in the thalamus. On the contrary, I have no doubt that the activity of those regions contributes to creating the selective, integrated, and unified contents of the conscious mind. I simply doubt that such a contribution is sufficient to explain consciousness comprehensively. That is why I focus on a set of different, albeit related, questions: What drives those regions to perform the tasks they perform? What is the purpose of their labors? How much does the result of those labors account for what I believe consciousness is, mentally speaking?

A Counterintuitive Fact?

The above conclusions underscore an important fact: although even the simplest core consciousness requires ensemble activity that involves regions of every tier and quarter of the brain, consciousness does depend most critically on regions that are evolutionarily older, rather than more recent, and are located in the depth of the brain, rather than on its surface. In a curious way, the "second-order" processes I propose here are anchored on ancient neural structures, intimately associated with the regulation of life, rather than on the modern neural achievements of the neocortex, those which permit fine perception, language, and high reason. The apparent "more" of consciousness depends on "less," and the second-order is, in the end, a deep and low order. The light of consciousness is carefully hidden and venerably ancient.

Let me note that this is a fact, not a hypothesis—whether my hypotheses turn out to be correct or not, the fact remains that damage to these sites impairs consciousness, while damage elsewhere does not. The least that can be said about this fact is that it seems counterintuitive. We rightly think of consciousness as a significant biological advancement, even when we grant consciousness to nonhuman creatures. Well, the advancement is certainly significant, but it may be

older than usually thought. What is not so old, evolutionarily speaking, is the extension of consciousness that has been allowed by memory, first, by permitting us to establish an autobiographical record; second, by giving us a broad record of other facts; and third, by endowing us with the holding power of working memory. Surely enough, these extensions of consciousness, which have blossomed so powerfully in humans, are based on the evolutionarily modern aspects of the brain, namely those of the neocortex. In the end, however, none of those astounding new features of consciousness occur independently of the modest feats of core consciousness.

