The Brainstem Begs the Question: *"Petitio Principii"*

Commentary by Heather Berlin

Mark Solms proposes that the upper brainstem is intrinsically conscious and that the cortex is intrinsically unconscious and is only permeated with consciousness from the brainstem. His theory relies heavily on studies of hydranencephalic children, who appear to have emotional reactions to outside stimuli despite the fact that they are missing a cerebral cortex. Solms uses this as his main evidence that consciousness is not a function of the cortex. However, I explain in this commentary, based on years of accumulated neuroscientific evidence, why Solms is making two unsupported assumptions: (1) that without cortex you have affective consciousness, and (2) that without brainstem you lose consciousness. It is important not to confuse "consciousness as such" (i.e., wakefulness) with the "content of consciousness" (i.e., awareness). There is excellent converging evidence for the cortical basis of the *contents* of consciousness.

Keywords: consciousness; cortico-thalamo-cortical pathways; frontoparietal network; hydranencephaly; neuroscience; persistent vegetative state

In his eloquent article "The Conscious Id," Mark Solms proposes several groundbreaking ideas, which, if substantiated, could potentially turn the fields of neuroscience and psychoanalysis on their heads. He first suggests that "affective consciousness" is derived from brainstem mechanisms that control and receive input from the autonomic body, and that "cognitive consciousness" is derived from cortical mechanisms that receive and send information to and from the sensorimotor body. This in itself is not so radical, but he goes on to propose that all of our cortically-based sensory and perceptual experiences are imbued with consciousness only by the affective processes that exist to govern our internal bodily needs. Solms therefore makes the radical claim that consciousness is a function of the upper brainstem. He proposes that the upper brainstem is intrinsically conscious and the cortex is intrinsically unconscious and is only permeated with consciousness from the brainstem. I have several major points of contention with this proposal, based on the accumulation of years of neuroscientific evidence.

First and foremost, Solms's theory relies heavily on one piece of evidence: Bjorn Merker's 2007 study of hydranencephalic children—that is, children born without a cortex. In Merker's study, hydranencephalic children appear to have emotional reactions to outside stimuli despite the fact that they are missing a cerebral cortex. Although they have no cortex, Solms reports that they clearly display signs of feeling pleasure and unpleasure and an extensive capacity for emotional learning. But emotional learning and processing rewards and punishments does not require consciousness (Berlin, 2011; Esteves, Parra, Dimberg, & Ohman, 1994; Fischman, 1989; Lamb et al., 1991; Pessiglione et al., 2007). He uses this as his main evidence that consciousness is not a function of the cortex—that is, that you can have consciousness in the absence of a cortex. However, we cannot assume that expressions of emotion equate with consciousness, when they may just be reflexive. Changes in vigilance and expression of emotion do not equal consciousness—consider, for example, persistent vegetative state (PVS) patients. Solms makes two unsupported assumptions: (1) that without cortex you have affective consciousness, and (2) that without brainstem you lose consciousness.

Early behaviors are highly reflexive, and specific arousal effects, as Pfaff (2006) has catalogued, are most of a lower organism's behavioral repertoire, but whether there is a conscious phenomenal aspect of these phenomena is unknown. The use of the hydranencephalic infant model (Merker, 2007; Shewmon, Holmse, & Byrne, 1999) begs the question entirely: showing that strongly conserved emotional facial displays and conditioned responses from a brainstem/spinal-cord system can be developed over time says nothing about whether conscious emotional states attach to these observable phenomena.

Therefore, Solms's primary assumption that hydranencephalic children are conscious is unwarranted. We cannot assume that having a sleep–wake cycle and expressions of emotion (laughter, rage, etc.) necessitates consciousness. For example, we can reproduce similar pseudo-emotional reactions in nonconscious machines (e.g., affective computing—such as the Siri application on Apple devices). Affective behaviors do not equate with consciousness—for example, decorticate rats, sleepwalkers, and people with conversion disorders and hysterical blindness can all display what look like meaningful affective behaviors without being

Heather Berlin: Department of Psychiatry, Mount Sinai School of Medicine, New York, U.S.A.

conscious of them. While it is true that they may in fact be consciousness, we cannot assume that they are. Unconscious processes can be quite sophisticated and complex (Berlin, 2011).

The crux of Solms's theory relies on a projection of the existence of consciousness based on what look like meaningful emotional behaviors, an example of the "moralistic fallacy" (arguing that something must be true because it would make us feel good to believe it). Humans have a natural desire to assume that consciousness exists (Shermer, 2011), but it is a misconception to uncritically equate vigilance, eye movements, and expression of emotion with consciousness. For example, PVS patients can open and close their eyes, but some do not have sleep–wake cycles as shown with EEG (Landsness et al., 2011). Take, for example, the well-known case of the PVS patient Terri Schiavo. Video clips of Terri showed spontaneous movements and reflexes (e.g., grimacing, crying, eye tracking for brief periods), but no evidence of awareness. In fact, according to the medical examiner's report, she was cortically blind. We must at least consider the possibility that the "emotional behaviors" displayed by hydranencephalic children are simply reflexes. Reflexes can be mediated without consciousness (e.g., classic spinal reflexes in frogs to pain; the withdrawal reflex can be accompanied by consciousness if the cortex is intact). We could just as easily use enteric nervous-system reactions to measure consciousness. Behavioral responses of the enteric nervous system to external stimuli would probably be accurate and reliable, but that does not mean that the enteric nervous system is conscious. What we really need is a theory of consciousness that will enable us to quantify consciousness with an objective, independent measure.

Furthermore, subcortical mediation of consciousness has been described so far only in congenital brain malformations, so developmental plasticity may play a role. Hydranencephalic children's abilities may reflect "vertical" plasticity of brainstem and diencephalic structures. "Vertical plasticity" is subcortical plasticity for supposedly cortical functions, whereas "horizontal" plasticity is cortical plasticity for cortical functions or subcortical plasticity for subcortical functions (Shewmon, Holmse, & Byrne, 1999). In fully formed adult brains, losing cortical function results in loss of the *content* of consciousness. Discrete cortical lesions give rise to specific pathologies of consciousness, such as blindsight, neglect, amnesia, anosognosia, and changes in personality and emotion (e.g., Phineas Gage; Harlow, 1848).

A distinction must be made between "consciousness as such" (i.e., wakefulness) and the "content of consciousness" (i.e., awareness). Enabling factors are necessary for any form of "consciousness as such" (wakefulness) to occur. These enabling factors include the mesencephalic reticular formation (a.k.a. ascending reticular activating system), cholinergic pathways from the brainstem and basal forebrain, and the intralaminar nucleus of the thalamus (Koch, 2004). One could perhaps think of these enabling factors as the power supply to the brain, as distinct from its processing center. However, specific factors are required for any one particular conscious percept—that is, "content." Experiments show that various cognitive tasks that require awareness are accompanied by short-term temporal correlations among distributed populations of neurons in the thalamocortical system. Hence, we need enabling factors as well as a dominant neuronal coalition in the cortex and thalamus for consciousness to occur.

There is no reason to believe that loss of the upper brainstem alone produces permanent unconsciousness unless the lesions are extensive, bilateral, and extend rostrally—and even in these cases the contribution of functional alteration of the rest of the cerebrum is unclear (N. Schiff, personal communication). It is likely that an intact corticothalamic system could in fact recover consciousness without the brainstem. For example, studies in cats show that brainstem lesions can decrease activation, but if you wait long enough their vigilance can recover; they can eventually recover activation and deactivation patterns and slow-wave sleep patterns. Studies by Villablanca (2004) show that you can cut and isolate thalamus and cortex from the brainstem of cats and keep them alive. At first this induces a coma, but after about a month the cortex reactivates and they show sleep–wake cycles. So animals with brainstem lesions can come out of a coma, but if they have no cortex activation they cannot. A cortex without a brainstem can potentially become conscious.

People in a PVS are "awake" (presence of sleep–wake cycles or eyes opening and closing), but not "aware" (no evidence of awareness of self or environment, and an inability to interact with others). The brainstem is mostly spared while the grey and white matter of both cerebral hemispheres are widely and severely damaged. Overall cortical metabolism is about 40–50% of the normal range (Laureys, 2005; Laureys, Lemaire, Maquet, Phillips, & Franck, 1999; Laureys et al., 1999; Schiff et al., 2002). PVS patients usually have either diffuse cortex or thalamic lesions. However, lesions in the brainstem of PVS patients can resolve, but lesions in cortex or paramedical thalamus cause loss of consciousness (Schiff, 2004, 2008). Upper-brainstem lesions can lead to coma, but patients

can recover as long as cortex and thalamus are intact (Schiff, 2004, 2008).

While it is true that the normal conscious state depends intimately on the activity of the upper brainstem/central thalamus and related centrencephalic components, lesions that produce initial coma give way to varying patterns of recovery that emphasize difference in the contribution of these structures and opportunities for the conscious state to reconstitute, despite lesions that initially wipe it out. Several papers by Nicholas Schiff address the misconception that the lesion literature localizes consciousness to the upper brainstem and/or central thalamus (Schiff, 2004, 2008). Lesions restricted to the rostral pons and mesencephalon producing coma have roughly dichotomous outcomes—death due to malignant hypotension and cardiopulmonary dysregulation, or recovery of consciousness typically in about 7 days. In sum, there is currently little neurological evidence for any local area to be absolutely critical for consciousness. Instead, many important hubs can alter the critical dynamic processes needed across the cerebrum (primarily corticothalamic systems) to maintain the awake, intentional conscious state (N. Schiff, personal communication).

Functional imaging in transient dissociations of wakefulness and awareness show decreased blood flow in the frontoparietal network in patients with complex partial seizures, absence seizures, and sleepwalking (Laureys, 2005). Medial posterior cortex (including the precuneus and posterior cingulate cortex) is the most active brain region in healthy controls and patients with locked-in syndrome (fully conscious, but paralyzed and thus not behaviorally responsive). In PVS patients, this same brain area is the least active region; patients in a minimal conscious state show an intermediate metabolism here, less than PVS patients, but more than healthy controls. These brain regions are among the most active in conscious waking and among the least active in altered states of consciousness such as general anesthesia, sleep, hypnotic state, dementia, and Korsakoff's or postanoxic amnesia. So this richly connected association area may be part of the neural network subserving awareness/consciousness (Laureys, Owen, & Schiff, 2004).

Neuroimaging of vegetative state (VS) patients identified brain areas that still show activation during external stimulation, but this activation is limited to subcortical and "low-level" primary cortical areas, disconnected from the frontoparietal network necessary for awareness/consciousness (Laureys, 2005). Electrical stimulation (painful in controls) of 15 VS patients activated midbrain, thalamus, and primary somatosensory cortex (S1), but not higher order areas of the pain matrix (secondary somatosensory areas, insular, posterior parietal, anterior cingulate cortex). Also, activated S1 was isolated from the frontoparietal network thought to be required for consciousness perception (Laureys et al., 2002). Similarly, auditory stimulation in VS patients activated primary auditory cortex, but not higher order multimodal areas from which they were disconnected (Boly et al., 2004; Laureys et al., 2000). The activation in primary cortices in these awake, but unaware, patients confirms Crick and Koch's (1995) early hypothesis (based on visual perception and monkey histological connectivity) that neural activity in primary cortices is necessary, but not sufficient, for awareness/consciousness.

In a recent study, Boly et al. (2012) found that decreased backward corticocortical connectivity from frontal to parietal cortices was associated with loss of consciousness under the anesthetic propofol, but thalamocortical connectivity was not. Thus, corticocortical communication appears to be important in the maintenance of consciousness and propofol seems to directly affect these cortical dynamics. What matters is cortex. When cortex comes back, so does consciousness; everything else is doubtful. In line with this, Velly et al. (2007) took intracranial recordings from subthalamic nuclei (thalamus) and cortex in Parkinson's disorder patients during anesthesia (sevoflurane or propofol). When the thalamus was "asleep," there was low-frequency activity for several minutes before the patients became unconscious. Patients only became unconscious when cortex started showing slow waves. Patients remained conscious as long as their cortex was activated, which suggests that consciousness mainly involves the cortex and we may not even need thalamus activation for consciousness.

Summary

There is excellent converging evidence for the cortical basis of conscious contents from lesions and nonspecific cortical damage, direct brain stimulation and recording, and functional brain-imaging methods that compare conscious vs. unconscious stimulation, like binocular rivalry, which is especially clear in single-cell work in human epileptics (Cerf et al., 2010; Kreiman, Fried, Koch, & 2002; Kreiman, Koch, & Fried, 2000a, 2000b; Reddy, Quiroga, Wilken, Koch, & Fried, 2006). Evidence for brain mechanisms corresponding to unconscious ("id") impulses (e.g., activation of subcortical structures like the amygdala and basal ganglia) and top-down control struggles involving prefrontal regions (e.g., anterior cingulate cortex, dorsolateral prefrontal

cortex, orbitofrontal cortex) is very strong. Clinical observation *and* direct evidence strongly support the neural basis for a range of defense mechanisms, so in this respect Freud was on the right track (Berlin, 2011).

There is some variation on how consciousness per se is defined, but consensus from most experts in the field of consciousness research is that consciousness is simply "first-person subjective experience." With this definition in mind, I agree that the hydranencephalic children cited in Solms's article are displaying *some* behaviors that appear to be in direct response to environmental stimuli, but we have no way of knowing whether those behaviors are simply reflexive or whether they are imbued with consciousness. We cannot simply make the assumption that they are conscious. Solms may be arbitrarily labeling unconscious emotions as conscious. Alternatively, due to neuroplasticity as a result of having no cortex *in utero*, the brains of these hydranencephalic children may have reorganized in such a way that some subcortical structures have taken on cortical functions. So what the evidence might show (assuming my distinction between reflex-like behavior and conscious awareness is met) is that consciousness can develop in the absence of much of the forebrain. But it does *not* show that, in a normal brain, consciousness originates anywhere other than in the corticothalamic system.

If Solms's radical theory is correct, it would have an enormous impact on the way we view the brain. We would be forced to assume that people on life support with no cortical activity—that is, brain dead, but with their brainstem intact—are still conscious. If losing awareness and certain cortical functions does not mean losing consciousness, it would necessitate keeping PVS patients alive indefinitely. Since acceptance of Solms's theory as fact would have major practical implications, we must tread lightly and only take on such assumptions as fact once the balance of the evidence is in its favor, which is currently not the case. Solms's article, although provocative, runs afoul of an important scientific dictum: "extraordinary claims require extraordinary evidence" (Sagan, 1980).

REFERENCES

Berlin, H. A. (2011). The neural basis of the dynamic unconscious. *Neuropsychoanalysis,* 13 (1): 5–31.

Boly, M., Faymonville, M. E., Peigneux, P., Lambermont, B., Damas, P., Del Fiore, G., et al. (2004). Auditory processing in severely brain injured patients: Differences between the minimally conscious state and the persistent vegetative state. *Archives of Neurology,* 61 (2): 233–238.

Boly, M., Moran, R., Murphy, M., Boveroux, P., Bruno, M. A., Noirhomme, Q., et al. (2012). Connectivity changes underlying spectral EEG changes during propofol-induced loss of consciousness. *Journal of Neuroscience,* 32 (20): 7082–7090.

Cerf, M., Thiruvengadam, N., Mormann, F., Kraskov, A., Quiroga, R. Q., Koch, C., et al. (2010). On-line, voluntary control of human temporal lobe neurons. *Nature*, 467 (7319): 1104–1108.

Crick, F., & Koch, C. (1995). Are we aware of neural activity in primary visual cortex? *Nature*, 375: 121–123.

Esteves, F., Parra, C., Dimberg, U., & Ohman, A. (1994). Nonconscious associative learning: Pavlovian conditioning of skin conductance responses to masked fear-relevant facial stimuli. *Psychophysiology,* 31: 375–385.

Fischman, W. M. (1989). Relationship between self-reported drug effects and their reinforcing effects: Studies with stimulant drugs. *NIDA Research Monograph*, 92: 211–230.

Harlow, J. M. (1848). Passage of an iron rod through the head. *Boston Surgical and Medical Journal*, 39: 389–393.

Koch, C. (2004). *The Quest for Consciousness: A Neurobiological Approach*. Englewood, CO: Roberts & Company.

Kreiman, G., Koch, C., & Fried, I. (2000a). Imagery neurons in the human brain. *Nature,* 408 (6810): 357–361.

Kreiman, G., Koch, C., & Fried, I. (2000b). Category-specific visual responses of single neurons in the human medial temporal lobe. *Nature Neuroscience*, 3 (9): 946–953.

Kreiman, G., Fried, I., & Koch, C. (2002). Single-neuron correlates of subjective vision in the human medial temporal lobe. *Proceedings of the National Academy of Sciences USA*, 99 (12): 8378–8383.

Lamb, R. J., Preston, K. L., Schindler, C. W., Meisch, R. A., Davis, F., Katz, J. L., et al. (1991). The reinforcing and subjective effects of morphine in post-addicts: A dose-response study. *Journal of Pharmacology and Experimental Therapeutics,* 259: 1165–1173.

Landsness, E., Bruno, M. A., Noirhomme, Q., Riedner, B., Gosseries, O., Schnakers, C., et al. (2011). Electrophysiological correlates of behavioral changes in vigilance in vegetative state and minimally conscious state. *Brain*, 134 (8): 2222–2232.

Laureys, S. (2005). The neural correlate of (un)awareness: Lessons from the vegetative state. *Trends in Cognitive Sciences*, 9 (12): 556–559.

Laureys, S., Faymonville, M. E., Degueldre, C., Fiore, G. D., Damas, P., Lambermont, B., et al. (2000). Auditory processing in the vegetative state. *Brain*, 123 (8): 1589–1601.

Laureys, S., Faymonville, M. E., Peigneux, P., Damas, P., Lambermont, B., Del Fiore, G., et al. (2002). Cortical processing of noxious somatosensory stimuli in the persistent vegetative state. *NeuroImage*, 17 (2): 732–741.

Laureys, S., Goldman, S., Phillips, C., Van Bogaert, P., Aerts, J., Luxen, A., et al. (1999). Impaired effective cortical connectivity in vegetative state: Preliminary investigation using PET. *NeuroImage*, 9 (4): 377–382.

Laureys, S., Lemaire, C., Maquet, P., Phillips, C., & Franck, G. (1999). Cerebral metabolism during vegetative state and after recovery to consciousness. *Journal of Neurology, Neurosurgery, and Psychiatry*, 67 (1): 121.

Laureys, S., Owen, A. M., & Schiff, N. D. (2004). Brain function in coma, vegetative state, and related disorders. *Lancet Neurology,* 3 (9): 537–546.

Merker, B. (2007) Consciousness without a cerebral cortex: A challenge for neuroscience and medicine. *Behavioral and Brain Sciences.,* 30: 63–134.

Pessiglione, M., Schmidt, L., Draganski, B., Kalisch, R., Lau, H., Dolan, R. J., et al. (2007). How the brain translates money into force: A neuroimaging study of subliminal motivation. *Science*, 11;316 (5826): 904–906.

Pfaff, D. (2006). *Brain Arousal and Information Theory: Neural and Genetic Mechanisms*. Cambridge, MA: Harvard University Press.

Reddy, L., Quiroga, R. Q., Wilken, P., Koch, C., & Fried, I. (2006). A single-neuron correlate of change detection and change blindness in the human medial temporal lobe. *Current Biology*, 16 (20): 2066–2072.

Sagan, C. (1980). Encyclopaedia Galactica. *Cosmos: A Personal Voyage* [TV series, episode 12]. PBS, 14 December.

Schiff, N. D. (2004). The neurology of impaired consciousness: Challenges for cognitive neuroscience. In: *The Cognitive Neurosciences III*, ed. M. S. Gazzaniga. Cambridge, MA: Bradford Books, pp. 1121–1142.

Schiff, N. D. (2008). Central thalamic contributions to arousal regulation and neurological disorders of consciousness. *Annals of the New York Academy of Sciences,* 1129: 105–118.

Schiff, N. D., Ribary, U., Moreno, D. R., Beattie, B., Kronberg, E., Blasberg, R., et al. (2002). Residual cerebral activity and behavioural fragments can remain in the persistently vegetative brain. *Brain*, 125 (6): 1210–1234.

Shermer, M. (2011). *The Believing Brain: From Ghosts and Gods to Politics and Conspiracies—How We Construct Beliefs and Reinforce Them as Truths*. New York: Holt.

Shewmon, D., Holmse, D., & Byrne, P. (1999). Consciousness in congenitally decorticate children: Developmental vegetative state as a self-fulfilling prophecy. *Developmental Medicine and Child Neurology,* 41: 364–374.

Velly, L. J., Rey, M. F., Bruder, N. J., Gouvitsos, F. A., Witjas, T., Regis, J. M., et al. (2007). Differential dynamic of action on cortical and subcortical structures of anesthetic agents during induction of anesthesia. *Anesthesiology*, 107 (2): 202–212.

Villablanca, J. R. (2004). Counterpointing the functional role of the forebrain and of the brainstem in the control of the sleep-waking system. *Journal of Sleep Research*, 13 (3): 179–208.