Supporting Information

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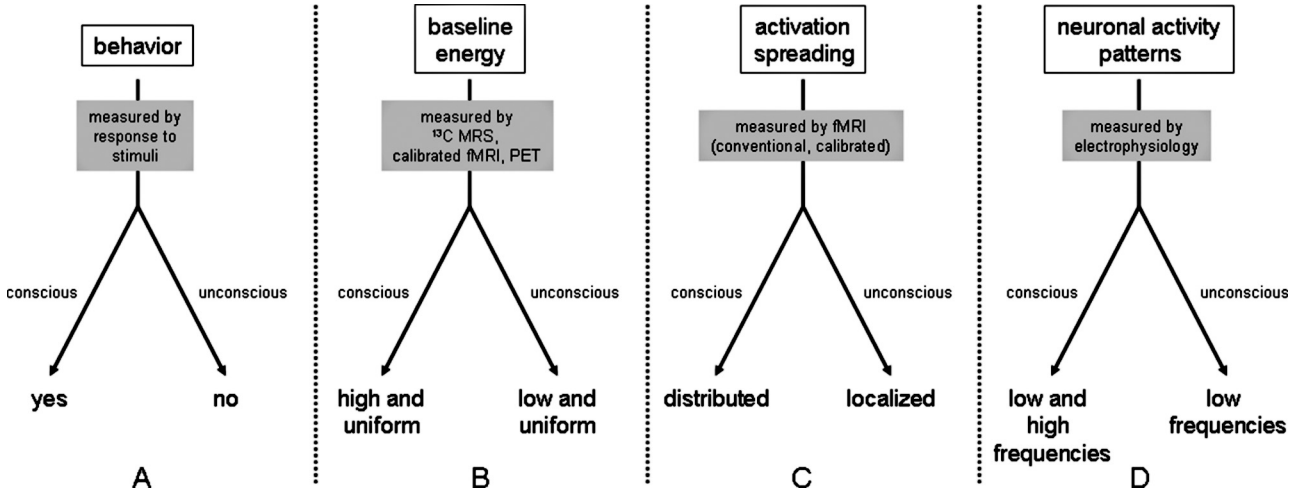

Fig. S1. Measurable properties for studying consciousness and its loss. (*A*) Behavioral output measured by response to stimuli. (*B*) Brain property of baseline energy measured by neurophysiological methods like ^{13}C magnetic resonance spectroscopy (MRS), calibrated fMRI, and PET. (*C*) Brain property of activation spreading measured by fMRI, both conventional and calibrated. (*D*) Brain property of neuronal activity patterns measured by electrophysiology.

Table S1. Anesthesia-induced decrease in glucose metabolism measured by PET

Area	ΔCMR_{O2} Sevoflurane, % (1)	ΔCMR_{O2} Propofol, % (1)	ΔCMR_{O2} Propofol, % (2)	ΔCMR_{O2} Halothane, % (3)	ΔCMR_{O2} Isoflurane, % (3)
Frontal cortex	29	38	58	47	52
Parietal cortex	31	42	53	41	55
Temporal cortex	26	36	52	48	47
Occipital cortex	43	50	59	50	40
Anterior cingulate	—	—	53	46	48
Caudate	30	33	—	—	—
Putamen	31	32	—	—	—
Thalamus	41	43	55	52	53
Basal ganglia	—	—	52	48	44
Hippocampus	—	—	42	50	45
Midbrain	—	—	44	54	50
Cerebellum	44	43	56	54	55

Values for anesthesia-induced decrease in metabolism were approximations made from graphical representations of data in the original papers (1–3). Decreased metabolism in whole brain, neocortex, and subcortex were 40–50%, 45–50%, and 35–45%, respectively. Therefore, brain metabolism of gray matter regions in anesthetized humans is uniformly suppressed.

1. Kaisti KK, et al. (2003) Effects of sevoflurane, propofol, and adjunct nitrous oxide on regional cerebral blood flow, oxygen consumption, and blood volume in humans. *Anesthesiology* 99:603–613.
2. Alkire MT, et al. (1995) Cerebral metabolism during propofol anesthesia in humans studied with positron emission tomography. *Anesthesiology* 82:393–403; discussion 327A.
3. Alkire MT, et al. (1999) Functional brain imaging during anesthesia in humans: Effects of halothane on global and regional cerebral glucose metabolism. *Anesthesiology* 90:701–709.

Table S2. Awake values of glucose metabolism measured by PET

Area	Awake CMR_{glc}, μmol/g/min
Frontal cortex	0.39 ± 0.05
Parietal cortex	0.37 ± 0.01
Temporal cortex	0.35 ± 0.07
Occipital cortex	0.37 ± 0.04
Anterior cingulate	0.34 ± 0.01
Caudate	0.32 ± 0.06*
Putamen	0.40 ± 0.07*
Thalamus	0.34 ± 0.01
Basal ganglia	0.36 ± 0.03
Hippocampus	0.29 ± 0.08
Midbrain	0.15 ± 0.01
Cerebellum	0.26 ± 0.06

Awake CMR_{glc} values show that the average of the whole brain (0.33 ± 0.04 μmol/g/min) is within 10 and 3% of the gray matter regions in the neocortex (0.36 ± 0.03 μmol/g/min) and subcortex (0.34 ± 0.05 μmol/g/min), respectively. Therefore, brain metabolism of gray matter regions in the awake human is quite high and uniform. Awake CMR_{glc} values were mainly derived form averaged value in refs. 1 and 2, except for caudate and putamen.
*Awake CMR_{glc} values were derived from CMR_{O2} values in ref. 3.

1. Alkire MT, et al. (1995) Cerebral metabolism during propofol anesthesia in humans studied with positron emission tomography. *Anesthesiology* 82:393–403; discussion 327A.
2. Alkire MT, et al. (1999) Functional brain imaging during anesthesia in humans: Effects of halothane on global and regional cerebral glucose metabolism. *Anesthesiology* 90:701–709.
3. Kaisti KK, et al. (2003) Effects of sevoflurane, propofol, and adjunct nitrous oxide on regional cerebral blood flow, oxygen consumption, and blood volume in humans. *Anesthesiology* 99:603–613.