

The near-death experience: a cerebellar method to protect body and soul. Lessons from the Iboga-Healing-Ceremony in Gabon

By Süster Strubelt, Dip Psych, Dip soc, MPH; Uwe Maas, MD, DrMed

Originally Published In: Alternative Therapies 2008; 14 (1): 30-34

Authors' e-mail: umaas@scientific-african.org

Abstract

The root bark of the Iboga shrub (*Tabernanthe Iboga*) is used in Gabon (Central Africa) to induce a near-death experience for spiritual and psychological purposes. The pharmacology of ibogaine, a psychoactive indole alkaloid extracted from the bark, has been extensively investigated because of its anti-addictive qualities. This current review of these studies and neuroscientific approaches to the near-death experience compared with field studies of traditional African rituals has generated new insights into the neurological correlates and the psychological effects and after-effects of the near-death experience.

Ibogaine stimulates the cerebellar fastigial nucleus in the same manner as ischemia and leads to a medium-term protection of the brain against glutamate induced neurotoxicity. At the same time, it induces changes of the autonomic nervous and the cardiovascular system, which serve the overcoming of the danger of life: Iboga-intake and ischemia both lead to slowing of EEG activity (dominance of theta- and delta- waves), a stimulation of the limbic system, and a dominance of a phylogenetically older branch of the vagus nerve, originating in the dorsal motor nucleus, which lowers the metabolic rate of the body.

As a conclusion, the near-death experience seems to be the result of a dominance of phylogenetically and ontogenetically old neurological structures and brain waves, which are allowed to show their (para) psychological abilities in absence of cortical dominance. If parts of the neocortex are still active and permit observation and memory performance, the experience can be integrated within the personality. The new learned peaceful state of vagal and subcortical dominance can be actively self-induced.

Implications of this model for alternative healing are discussed.

Introduction

In the Republic of Gabon in Central Africa, an initiation ritual is performed with the consumption of the root bark of the Iboga-shrub and is supported by an ingenious musical accompaniment. Gabonese healers expect the person to be initiated to experience a “vision” of “death and rebirth” (1-4). Reports of Europeans being initiated and Gabonese people interviewed by ethnologists show all the typical elements of a near-death experience: a life review, out-of-body experience and floating over various landscapes, an encounter with deceased, gliding through a tunnel into an ulterior world, and an encounter with a divine entity – finally the arrival at a point of no return, which is not to be surpassed by the living, and the return to life (1-4). Also, Gabonese expectations of a successful initiation coincide with the research results of the effects of near-death experiences: acceptance of responsibility and reduction of dependencies, increase in spirituality and reduced fear of death, as well as a new love for people and environment are unanimously observed as effects of the encounter with death (5-9). Lommel et al. were able to prove these effects in a longitudinal study. In this study, patients, who had a near death experience related to cardiac arrest, were compared with a control group (also with heart failure, but without recollection). The observed psychological differences between both groups in their study were more pronounced after eight years of follow-up than after two years (10).

However, the causes of these effects remain unclear. Neurological attempts to formulate an explanation hold the endorphins responsible for the exalted mood during the near-death experience (11-13). The visions are presumed to be caused by a disengagement of external stimuli (13-14) and a cerebral increase in activity (11): In particular, an over activation of the temporal lobe (11,13,15) and the hippocampus (11,14,16). Blanke et al. report they could provoke an out-of-body experience of a woman with a diagnosis of epilepsy by electric stimulation of the right angular gyrus (17). On the other hand, Woerlee holds malfunction of several brain areas (prefrontal cortex, motor cortex, parietal cortex, angular gyrus, amygdala and hippocampus) caused by ischemia and resuscitation responsible for near-death experiences during cardiac arrest (18). Reader explains the near-death experience as a parasympathetic response to an excessive sympathetic stimulation caused by the extreme stress in situations of danger (19). Jansen proposes that the near-death experience serves to protect the brain from the effects of ischemia by blocking NMDA type glutamate receptors (13).

Studies about near-death experiences have several limitations because of their unexpected occurrence, the priority of live-saving measures (18) and the impossibility to communicate with the near-death experiencer. The Iboga-vision is, on the contrary, provoked in a controlled situation with a specific musical and psychological accompaniment. In addition, ibogaine, the most important psychoactive alkaloid of the Iboga-shrub, has been extensively pharmacologically investigated in animal studies

because of its putative anti-addictive properties. Though classified as an illegal drug in the United States and most European countries, it is used by self-help groups and private clinics to cure addiction (20).

Research questions

It was the aim of our study to gain new insights into neurological correlates of near-death experiences integrating the results of a literature review of neuroscience with ethnological investigations and own field studies in the region of Lambaréné (Gabon) in the years of 1999-2005. Through a comparison of international science and traditional knowledge in a qualitative study-design we aimed at gathering a maximum of possible information about the topic. Our research questions were:

Are Iboga visions and near-death experiences based on a common neurological mechanism?

Can ibogaine research help to detect structural changes in brain function during a near-death experience?

Are there neurological correlates for the personality changes after near-death experiences and after Iboga-visions?

Results: Pharmacology and physiology of the near-death experience under Iboga

A cerebellar emergency program

Through an unknown mechanism, ibogaine induces a rhythm of 8-12 Hz in the inferior olive. This rhythm leads to enhanced synaptic excitation in cerebellar Purkinje-cells through a glutamatergic pathway. Glutamate, the most important excitatory neurotransmitter can produce 'excitotoxicity' in Purkinje cells (21-22). Certain groups of cells, normally inhibiting the fastigial nucleus, are especially vulnerable to neurodegeneration. If inhibition through these cells fails, the fastigial nucleus induces neuroprotective mechanisms in the brain. (23-26). It could be presumed that this mechanism serves as a protection in case of ischemia.

Welsh et al discovered that ibogaine in very high concentration caused patterns of cell degeneration in rats that mimic global ischemia (21).

Although the death of Purkinje cells in rats could only be demonstrated after large doses of ibogaine (28), the protective properties are presumably effective with smaller doses. Using a model of mild ischemia in sheep fetuses, Harding et al. were able to demonstrate

that despite the loss of almost all activity in Purkinje cells, no obvious cellular damage was seen (29). This ischemic preconditioning also seems to stimulate the fastigial nucleus thereby inducing protection against subsequent ischemic insults (30). Reis et al. could reduce the infarct volume after focal ischemia by up to 50% after electrical stimulation of the fastigial nucleus, a protective effect that could be observed for weeks (26).

It can be concluded that Ibogaine induces this type of protective reaction against (prospective) oxygen deprivation in the brain. Parallels can be drawn to near-death experiences, which might occur in situations of real as well as anticipated ischemia, for example during a fall or roll-over of a car (31-32).

The model we suggest here fits well with the results of a very recent study carried out by Schutter and coworkers, who could induce an out-of-body experience by transcranial stimulation of the Cerebellum (33).

The induction of a hippocampal theta rhythm

Enhanced release of the stimulating neurotransmitter glutamate, which can be neurotoxic not only for Purkinje-cells is claimed to be the first danger in case of ischemia (34-35). Presumably as a measure of protection, higher cerebral functions are reduced under oxygen deprivation, indicated by a gradual slowing of EEG activity (18,36,37).

In case of mild or beginning ischemia, a theta rhythm of 5-6 Hz in human beings (38) is intensified (36,39). Similarly, ibogaine also increases this rhythm (40), probably through its stimulating effect on the fastigial nucleus (25). Schutter and van Honk were able to induce it by transcranial stimulation of the Cerebellum (41). During the Gabonese initiation ceremony additional augmentation of the theta rhythm results from the polyrhythmic ritual mouth bow (in male initiations) and harp music (in female initiations), which is played in an absolutely constant measure of 5-6 Hz (42).

Theta rhythms with a frequency spectrum of 4-8 Hz are generated in different parts of the hippocampus (43) and develop in humans within the first years of their life. They become dominant in the wake-EEG at the age of 6-7 years (44). Alpha activity shows a slower onset and becomes dominant in puberty (45). While theta rhythms serve the formation of episodic memories and the encoding of new information, alpha rhythms serve to memorize semantic matters (46).

Normally, slower rhythms in the brain are inhibited by faster ones. This blockage is supposed to be more distinct in fearful people (47-48). However, in the setting of ischemia this relation is reversed and normal inhibitions are removed thereby inducing neuroprotection and allowing extraordinary ways of thinking. Since it can be assumed

that the recall of memories is facilitated by the same EEG frequencies that were present during memory formation, we hypothesize that the phase of dominance of the theta rhythms is responsible for the episodic life review in near-death experiences. After consumption of ibogaine this phase lasts for several hours and enables the confrontation with childhood problems, which is psycho-therapeutically guided by the healers.

The induction of a cerebral delta-rhythm

In the course of ischemia, a transition to delta-waves can be observed (31,36), a frequency spectrum (0.5 – 4 Hz) also induced by ibogaine (49).

Gabbard was able to observe a transition from theta to delta-waves in the EEG of adults, who were able to self-induce an out-of-body experience in a motionless rest position (50). Whinnery observed the same effect in pilots, who had near-death experiences during fast accelerations (31).

Delta-rhythms, which in adults normally only occur during fatigue or sleep, dominate the wake EEG of reptiles (51,52). Wettach interprets these delta wave patterns in human`s wake EEG as a shift of the brain to evolutionary older structures (53), which are more resistant to ischemia than the neurocognitive networks of mammals (18,54).

Delta-rhythms also dominate the wake EEG in newborns (44). Jacobs and Nadel claim that the hippocampus and the hippocampal theta-rhythm are not yet functioning in infants and therefore they are not able to memorize information about space and time (55). This could offer one explanation for the special performance of space and time during the near-death experience.

A transition from theta to delta-rhythms is also facilitated by the Gabonese ceremonial music during the induction of a possession-trance (42). Characteristic features of this trance, as observed by Bourguignon in several African cultures_ (56) are strange behavioral patterns, as if belonging to another person (interpreted as being “possessed” by a ghost), the inability to speak and a subsequent amnesia. We interpret the loss of the cortical functions of speech and memory as indication for dominance of subcortical networks during this possession-trance.

Maintaining neocortical functions

In contrast to the possession-trance, the ability to memorize and the ability to verbally communicate are preserved during the Iboga-“vision”. The access to neocortical functions also seems to be intact during near-death experiences as indicated by clearly structured and linguistically formulated memories, even though in most cases during the experience, because of the circumstances, no verbal exchange with the environment

takes place. The mechanism for the maintenance of the cortical function is unclear. Certain cerebral functions however, especially those of the prefrontal lateral cortex, seem to be responsible for the suppression of motor responses (57) which is observed under Iboga (2) and during near-death experiences (31) and seems to be reasonable in case of ischemia to save energy (18).

In Gabon the maintenance of the cortical functions is considered to be therapeutically very important. During the Iboga initiation ceremony the healers constantly demand a verbal communication about the observed matters and their possible meanings and request target-oriented activities in the "hereafter". Only a remembered vision is considered successful. Therewith new spiritual contents of the unconsciousness as well as knowledge of interior processes are opened to the consciousness, and conditions are prepared to transfer them into long-term memory. The vision symbolizes with the elements of the tunnel, divine light and "point of no return" the fight of the body for survival and the "re"birth.

That cortical functions are not always maintained, might be the reason why near-death experiences are rare. Lommel et al. found, that 80% of patients did not remember anything after cardiac arrest (10).

The acquisition of "vagal competence"

The near-death, like other spiritual experiences consists not only of images but also strong emotions that are experienced as being exceptional. These may be recalled later and felt as long-term shaping (6-9).

In emergency situations the fastigial nucleus influences the brain but also breathing, heart-rate and other autonomic parameters (58-63). Ischemia causes bradycardia, reduced respiratory rate and metabolism (64-65). Ibogaine has the same effects, additionally a decrease of body temperature and extreme slowdown of body movements could be observed (2,20,66).

According to Porges' polyvagal theory, the different emotions of higher mammals are made possible through a complicated cooperation of the two antagonists of the autonomic nervous system, the sympathetic and vagal system (67,68). Phylogenetically old reptiles have only an energy saving mode (vagal excitement) and an activity mode (sympathetic excitement) (69) whereas higher reptiles and mammals have two vagal branches: the evolutionary older one, originating in the dorsal motor nucleus of the nervus vagus and a second branch, originating in the nucleus ambiguus. This "new vagus" is connected with neuromuscular regions, for example the facial muscles and therefore allows different emotions and forms of communication (sucking and tasting,

head movement and mimicking, call-outs and the coordination of breathing and heart-rate).

The “old” energy saving vagus is dominant in mammals only in extreme situations of danger (67,68). Humans need it presumably mainly at birth.

This exceptional vagal stimulation during near-death experience might explain that the situation is perceived as completely new and beyond comparison with other “real” experiences. The similarity to ischemia during birth is reflected in the Gabonese description, that one dies and is reborn.

If the new feeling of vagal dominance is connected with cerebral networks, the initiated or near-death experienced can recall the feelings of the experience later and continuously activate a new awareness of life. We suppose that meditation initiates similar processes of conscious influence on the autonomic nervous system. Benson et al. showed that Buddhist monks were able to reduce their need for oxygen - by up to 70% (70).

This “vagal competence” could also be effective psychologically. Sahar et al. discovered that patients with a post traumatic stress disorder demonstrate a stronger sympathetic activation than a control group when presented with a mathematical problem. This demonstrates they were unable to use the vagal system adequately for their own relaxation (71).

Trauma-victims, who have experienced extreme situations of helplessness and often dissociations, even out-of-body experiences seem to generally inhibit vagal reactions. The Iboga vision might enable them to experience a massive vagal reaction in a situation of safety and therefore make it once again available for their own experience. In case of depression, which is also accompanied by a sympathetic over-reaction (72), Mash et al. were able to demonstrate an improvement after ibogaine treatment using a self-rating depression scale (73).

The induction of neurogenesis in the hippocampus

In Gabon the night of the vision is only the beginning of the healing-process. The traditional initiation may last several weeks. After the consumption of Iboga, the person to be initiated will be sheltered from the outer world for several days and is accompanied by healers. He is in a state of regression and relives childhood phases. Hereby he is continuously accompanied by healers and other initiated persons in the processing of the experiences (1).

The psychological changes could be based on neurological changes as the brain responds after ischemia (74) or epilepsy (75) with increases in neurogenesis in the hippocampus.

These changes last for several weeks, consistent with the period of retreat in the traditional initiation ceremony (74).

This could be important because long-term stress or trauma result in a reduction of hippocampal volume (76-78) which could also be shown in cases of depression (79) and alcohol dependency (80). Hippocampal neurogenesis also seems to be part of the effect of electro convulsive therapy (81) and antidepressants (82). Gould was able to confirm these effects in a study using rats: a relatively small population of immature new cells could exert a substantial functional influence over the hippocampal function (83).

Keeping the person isolated and in permanent company of healers after the initiation ceremony in Gabon in the period of neurogenesis, which lasts several weeks (74) may serve to consolidate personality changes. Gabonese healers always emphasize how important it is not to “forget” the initiation. To our knowledge, impacts of aftercare on the long-term effects of near-death experiences have not yet been investigated. However, based on extensive clinical experience, Clark emphasizes the importance of a sensitive and respectful treatment of near-death experienced in the hospital setting (84).

Conclusions

Iboga-induced visions and the near-death experience seem to be attributed to a common neuronal mechanism. They result from a protective cerebellar reaction against a real or anticipated oxygen deprivation, which can be induced pharmacologically in the fastigial nucleus. We presume that cortical influences (the belief that death is impending) may also be a possible cause.

In the resulting exceptional state the brain reduces its activity and re-activates infantile brain-rhythms and networks that are exceptionally resistant to oxygen deprivation.

The near-death experience is therefore the result of a dominance of phylogenetically and ontogenetically old neurological structures and brain waves, which are allowed to show their (para)psychological abilities in absence of cortical dominance. Our study permits the conclusion that not the neocortex, but older structures are leading in spiritual performance. Neocortical structures seem to inhibit these processes under normal circumstances.

However, only when parts of the cortex are no longer inhibiting but are still active (permitting observation and memory performance) can the experience be integrated within the personality.

Implications for alternative healing

During an ibogaine-induced or “natural” near-death experience the adult person relives a condition of massive vagal dominance that is not normally at his disposal. To experience a life threatening situation in a peaceful and beatified manner can heal basic life fears. In Gabon initiation is induced in cases where an individual has a problem with the deceased. Many people who had near-death experiences have also solved a problem with the after-life: the fear of death. Interestingly enough, in both cases, personality changes are not induced by resolving or reliving life-problems, but by a transcendental experience. This is a potential, which is neglected in Western psychotherapy.

But near-death experiences do not always cause personality changes. For African healers, aftercare is as important as the vision itself, an approach that fits with studies about neurogenesis after life-threatening situations. Near-death experiencers usually lack this period of retreat and counseling and only casually (particularly if they are hospitalized) have the time to consolidate and integrate their experiences in a comprehensive atmosphere without the demands of daily life. Considering the high number of near-death experiences in Western societies (Knoblauch found 4% NDE-reports in a representative survey in Germany (85)) aftercare should be a therapeutic concern.

Aftercare and the importance of the spiritual experience are also underestimated in ibogaine therapies against drug-addiction performed by private clinics in several industrial countries. Although drug addicts often report elements of near-death experiences after the intake of ibogaine (86), these are neglected by the pharmacological research or are considered as being disturbing and without influence on the success of the therapeutic process (20). Glick and co-workers even developed an Iboga alkaloid congener to treat addiction, which is expected not to be “hallucinogenic” (87).

We propose that in further research, physiology and spirituality should no longer be considered apart from each other. The physiology of spiritual experiences and concomitant spiritual phenomenon of physiological processes are worthy of further investigation. The results could offer new psychotherapeutic approaches in the future.

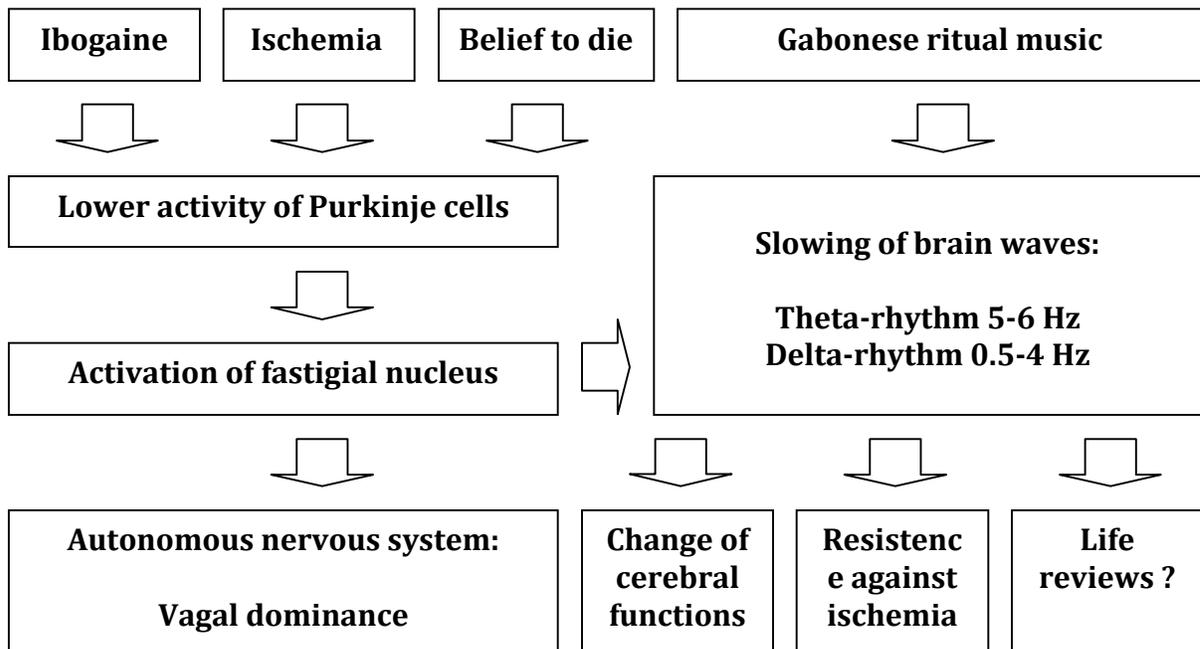


Figure: Neurogenic pathways of the near-death experience

References

1. Strubelt S, Maas U. The Iboga-healing ceremony in Gabon. Organic alterations in the brain during a controlled near-death-experience. In: Gottschalk-Batschkus CE, Green JC, eds. *Ethnotherapies in the cycle of life*. München: Ethnomed; 2005:221-227.
2. Samorini G. The initiation rite in the Bwiti religion. *Jahrbuch für Ethnomedizin und Bewusstseinsforschung*. 2000;6-7:39-56
3. Goutarel R, Gollnhofer O, Sillans R. Pharmacodynamics and therapeutic applications of Iboga and Ibogaine. *Psychedelic Monographs and Essays*. 1998;6:70-111
4. Fernandez J. *Bwiti. An ethnography of the religious imagination of Africa*. Princetown: University Press; 1982:470-493
5. Strubelt S, Maas U. *Die Iboga-Heilungszeremonie im Gabun: Gelenkte Nah-Todes-Erfahrung als Psychotherapie. Ein Vergleich afrikanischen Wissens mit internationalen Forschungsergebnissen*. Lage: Jacobs-Verlag; 2006:52-57

6. Ewald G. *Nahtoderfahrungen*. Kevelaer: Matthias-Grünewald-Verlag; 2006
7. Groth-Marnat G, Summers R. Altered beliefs, attitudes and behaviors following near-death experiences. *Journal of humanistic psychology*. 1998;38(3):110-125
8. Bonenfant R. A comparative study of near-death experience and non-near-death experience outcomes in 56 survivors of clinical death. *Journal of Near-Death Studies*. 2006;22(3):155-178
9. Schröter-Kunhardt M. Nah-Todeserfahrung – Grundlage neuer Sinnfindung. In: Kick, H, ed. *Ethisches Handeln in den Grenzbereichen von Medizin und Psychologie*. Münster: Lit-Verlag; 2002:59-70
10. Lommel van P, Wees van R, Meyers V, Elfferich I. Near-death experience in survivors of cardiac arrest: a prospective study in the Netherlands. *Lancet*. 2001;358:2039-2015
11. Schröter-Kunhardt M. Nah-Todeserfahrungen aus psychiatrisch-neurologischer Sicht. In: Knoblauch H, Soeffner H, eds. *Todesnähe. Wissenschaftliche Zugänge zu einem außergewöhnlichen Phänomen*. Konstanz: Universitätsverlag; 1999:65-97
12. Blackmore S. Neurophysiologische Erklärungen der Nah-Todeserfahrung. In: Knoblauch H, Soeffner H, eds. *Todesnähe. Wissenschaftliche Zugänge zu einem außergewöhnlichen Phänomen*. Konstanz: Universitätsverlag; 1999:32-67
13. Jansen KL. *The Ketamine model of the near-death experience: a central role for the N-Methyl-D-Aspartate receptor*. *Journal of Near-Death Studies*. 1997;16(1):5-26
14. Jourdan J. *Near-death and transcendental experiences: neurophysiological correlates of mystical traditions*. *Journal of Near-Death Studies*. 1994;12(3):177-200
15. Munro C, Persinger MA. Relative right temporal-lobe theta activity correlates with Vigniano`s hemispheric quotient and the “sensed presence”. *Percept Mot Skills*. 1992;75(3 Pt 1):899-903
16. Lempert T, Bauer M, Schmidt D. *Syncope: A videometric analysis of 56 episodes of transient cerebral ischemia*. *Ann Neurol*. 1994;36(2):233-237
17. Blanke O, Ortigue, S, Landis T, Seeck M. *Stimulating illusory own-body perceptions. The part of the brain that can induce out-of-body experiences has been located*. *Nature*. 2002;419:269

18. Woerlee G. Cardiac arrest and near-death experience. *Journal of near-death-studies* 2004;22(4):235-249
19. Reader A. The internal mystery plays: the role and physiology of the visual system in contemplative practices. *Altern Ther Health Med.* 1995;1(4):54-63
20. Alper KR. Ibogaine: a review. *Alkaloids.* 2001;56:1-37
21. Welsh JP, Yuen G, Placantonakis DG et al. Why do Purkinje cells die so easily after global brain ischemia? Aldolase C, EAAT4, and the cerebellar contribution to posthypoxic myoclonus. *Adv Neurol.* 2002;89:331-359
22. O'Hearn E, Molliver ME. The olivocerebellar projection mediates Ibogaine-induced degeneration of purkinje cells: A model of indirect, transsynaptic excitotoxicity. *J neurosci.* 1997;17(22):8828-8841
23. Zhou P, Qian L, Glickstein SB, Golanov EV, Pickel VM, Reis DJ. Electrical stimulation of cerebellar fastigial nucleus protects rat brain, in vitro, from staurosporine-induced apoptosis. *J neurochem.* 2001;79:328-338
24. Galea E, Golanov EV, Feinstein DL, Kobylarz KA, Glickstein SR, Reis DJ. Cerebellar stimulation reduces inducible nitric oxide synthase expression and protects brain from ischemia. *Am J physiol.* 1998;274:2035-2045
25. Golanov EV, Reis DJ. Neuroprotective electrical stimulation of cerebellar fastigial nucleus attenuates expression of periinfarction depolarizing waves (PIDs) and inhibits cortical spreading depression. *Brain Res.* 1998;818:304-315
26. Reis DJ, Feinstein DL, Galea E, Golanov EV. Central neurogenic neuroprotection: protection of brain from focal ischemia by cerebellar stimulation. *Fundam Clin Pharmacol.* 1997;11(Suppl.1):39-43
27. Culic M, Saponjic J, Jankovic B, Rakic L. Effect of cerebellar stimulation on EEG power spectra in the acute model of epilepsy. *Indian J Med Res.* 1994;100:135-139
28. Molinari HH, Maisonneuve IM, Glick SD. Ibogaine neurotoxicity: a reevaluation. *Brain Res.* 1996;37:255-262
29. Harding R, Rawson JA, Griffiths PA, Thorburn GD. The influence of acute hypoxia and sleep states on the electrical activity of the cerebellum in the sheep fetus. *Electroencephalogr Clin Neurophysiol.* 1983;57:166-173
30. Liu Y, Kato H, Nakata N, Kogure K. Protection of rat hippocampus against ischemic neuronal damage by pretreatment with sublethal ischemia. *Brain Res.* 1992;586:121-124

31. Whinnery JE. Psychophysiologic correlates of unconsciousness and near-death experiences. *Journal of Near-Death Studies*. 1997;15(4):231-258
32. Noyes R, Kletti R. Depersonalization in the face of life-threatening danger: A description. In: Lundahl C, ed. *A Collection of Near-Death Research Readings*. Chicago: Nelson-Hall Inc. Publishers; 1982:51-64
33. Schutter, DJ, Kammers MP, Enter D, van Honk J. A case of illusory own-body perceptions after transcranial magnetic stimulation of the cerebellum. *Cerebellum*. 2006; 5(3):238-40
34. Choi D, Maulucci-Gedde M, Kriegstein A. Glutamate neurotoxicity in cortical cell culture. *J neurosci*. 1987;7(2):357-368
35. Rothman SM, Olney JW. Glutamate and the pathophysiology of hypoxic-ischemic brain damage. *Ann Neurol*. 1986;19:105-111
36. Parnia S, Fenwick P. Near death experiences in cardiac arrest: visions of a dying brain or visions of a new science of consciousness. *Resuscitation*. 2002;52:5-11
37. Nagata K. Topographic EEG in brain ischemia – correlation with blood flow and metabolism. *Brain topogr*. 1988;1 (2):97-106
38. Basar E, Schürmann M, Sakowitz O. The selectively distributed theta-system functions. *Int J Psychophysiol*. 2001;39:197-212
39. Kasaba T, Saito Y, Sakura S, Kosaka Y. The effects of thiamylal, ketamine and nicardipine on the hippocampal theta waves produced by cerebral ischemia in cats. *Masui*. 1990;39(7):870-876
40. Depoortere H. Neocortical rhythmic slow activity during wakefulness and paradoxical sleep in rats. *Neuropsychobiology*. 1987;18(3):160-168
41. Schutter DJ, van Honk J. An electrophysiological link between the cerebellum, cognition and emotion. Frontal theta EEG activity to single-pulse cerebellar TMS. *Neuroimage*. 2006; 33 (4):1227-31
42. Maas U, Strubelt S. Polyrythms supporting a pharmacotherapy. Music in the Iboga Initiation Ceremony in Gabon. In: Aldridge D, Fachner J, eds. Music and Altered States. Consciousness, Transcendence, Therapy and Addiction. London, Philadelphia: Jessica Kingsley Publishers; 2006:101-124.
43. Kocsis B, Thinschmidt JS, Kinney GG, Vertes RP. Separation of hippocampal theta dipoles by partial coherence analysis in the rat. *Brain Res*. 1999;660:341-345

44. Schmid RS, Tirsch WS. *Klinische Elektroenzephalographie des Kindes- und Jugendalters*. Berlin: Springer; 1995:56
45. Schmid RS, Tirsch WS. *Klinische Elektroenzephalographie des Kindes- und Jugendalters*. Berlin: Springer; 1995:70-78
46. Klimesch W. *EEG alpha and theta oscillations reflect cognitive and memory performance: a review and analysis*. *Brain Res Brain Res Rev.* 1999;29:169-195
47. Knyazev GG, Slobodskaya HR. Personality trait of behavioral inhibition is associated with oscillatory systems reciprocal relationships. *Int J Psychophysiol.* 2003;48:247-261
48. Inanaga K. Frontal midline theta rhythm and mental activity. *Psychiatry Clin Neurosci.* 1988;52:555-566
49. Binienda Z, Beaudoin M, Thorn B et al. Alteration in electroencephalogram and monoamine concentrations in rat brain following Ibogaine treatment. *Ann NY Acad Sci.* 1998;844:264-272
50. Gabbard G. *With the eyes of the mind*. New York: Praeger Publishers; 1984:203
51. Gonzalez J, Gamundi A, Rial R, Nicolau MC, de Vera L, Pereda E. Nonlinear, fractal and spectral analysis of the EEG of lizard, *Gallotia galloti*. *Am J Physiol.* 1999;277:R86-93
52. De Vera L, Gonzalez J, Rial RV. Reptilian waking EEG: slow waves, spindles and evoked potentials. *Electroencephalogr Clin Neurophysiol.* 1994;90:298-303
53. Wettach G. The near death experience as a product of isolated subcortical brain function. *Journal of Near-Death Studies.* 2000;19(2):71-90
54. Else PL, Hulbert AJ. Comparison of the „mammal machine” and the „reptile machine“: energy production. *Am J Physiol.* 1981;240(1):R 3-9
55. Jacobs WJ, Nadel L. Stress-induced recovery of fears and phobias. *Psychol Rev.* 1985;92(4):512-531
56. Bourguignon E. *Possession*. Ohio: Chandler and Sharp; 1976
57. Anderson MC, Ochsner KN, Kuhl B et al. Neural systems underlying the suppression of unwanted memories. *Science.* 2004;303:232-235
58. Xu F, Frazier DT. Role of the cerebellar deep nuclei in respiratory modulation. *Cerebellum.* 2002;1:35-40
59. Xu F, Owen J, Frazier DT. Cerebellar modulation of ventilatory response to progressive hypercapnia. *J appl Physiol.* 1994;77:1073-1080

60. Parry TJ, McElligott JG. Kainic acid administration in the fastigial nucleus produces differential cardiovascular effects in awake and anesthetized rats. *Brain Res.* 1994;635:27-36
61. Chida K, Iadecola C, Underwood MD, Reis DJ. A novel vasodepressor response elicited from the rat cerebellar fastigial nucleus: the fastigial depressor response. *Brain Res.* 1986;370(2):378-82
62. Lutherer LO, Lutherer BC, Dormer KJ, Janssen HF, Barnes CD. Bilateral lesions of the fastigial nucleus prevent the recovery of blood pressure following hypotension induced by hemorrhage or administration of endotoxin. *Brain Res.* 1983;269:251-257
63. Martner J. Influences on the defecation and micturition reflexes by the cerebellar fastigial nucleus. *Acta Physiol Scand.* 1975;94:95-104
64. Kanwisher JW, Gabrielsen G, Kanwisher N. Free and forced diving in birds. *Science.* 1981;211:717-719
65. Wolf S, Schneider RA, Groover ME. Further studies on the circulatory and metabolic alterations of the oxygen-conserving (diving) reflex in man. *Trans Assoc Am Physicians.* 1965;78:242-254
66. Strubelt S. *Die Iboga-Heilungszeremonie im Gabun: Gelenkte Nah-Todes-Erfahrung als Psychotherapie. Ein Vergleich afrikanischen Wissens mit internationalen Forschungsergebnissen.* University of Bielefeld, Germany: Thesis of Master of Public Health; 2004:95
67. Porges SW. Love: an emergent property of the mammalian autonomic nervous system. *Psychoneuroendocrinology.* 1998;23(8):837-861
68. Porges SW. Orienting in a defensive world: Mammalian modifications of our evolutionary heritage. A Polyvagal Theory. *Psychophysiology.* 1995;32:301-318
69. Wang T, Warburton S, Abe A, Taylor T. Vagal control of heart rate and cardiac shunts in reptiles. Relation to metabolic state. *Exp physiol.* 2001;86(6):777-784
70. Benson H, Malhotra MS, Goldman RF, Jacobs GD, Hopkins PJ. Three case reports of the metabolic and electroencephalographic changes during advanced buddhist meditation techniques. *Behav Med.* 1990;16(2):90-95
71. Sahar T, Shalev AY, Porges SW. Vagal modulation of responses to mental challenge in posttraumatic stress disorder. *Biol psychiatry.* 2001;49(7):637-643
72. Hughes JW, Stoney M. Depressed mood is related to high-frequency heart rate variability during stressors. *Psychosom Med.* 2001;62:796-803

73. Mash DC, Kovera CA, Pablo J. et al. Ibogaine: Complex pharmacokinetics, concerns for safety, and preliminary efficacy measures. *Ann N Y Acad Sci.* 2000;914:394-401
74. Takasawa K, Kitagawa K, Yagita Y et al. Increased proliferation of neural progenitor cells. *J cereb Blood Flow Metab.* 2002;22:299-307
75. Praag H van, Zhao X, Gage FH. Neurogenesis in the adult mammalian brain. In: Gazzaniga MS, ed. *The new cognitive neurosciences.* Massachusetts: Bradford; 2000:127-137
76. Vythilingam M, Heim C, Newport J et al. Childhood trauma associated with smaller hippocampal volume in women with major depression. *Am J psychiatry.* 2002;159(12):2072-2080
77. Bremner J, Narayan M. The effects of stress on memory and the hippocampus throughout the life cycle: implications for childhood development and aging. *Dev Psychopathol.* 1998;10(4):871-885
78. Driessen M, Hermann J, Stahl K et al. Magnetic resonance imaging volumes of the hippocampus and the amygdala in women with borderline personality disorder and early traumatization. *Arch Gen Psychiatry.* 2000;57(12):1115-1122
79. Frodl T, Meisenzahl EM, Zetsche T et al. Hippocampal changes in patients with a first episode of major depression. *Am J Psychiatry.* 2002;159(7):1112-1128.
80. De Bellis MD, Clark DB, Beers SR et al. Hippocampal volume in adolescent-onset alcohol use disorders. *Am J Psychiatry.* 2000;157(5):737-344
81. Madsen TM, Treschow A, Bengzon J, Bolwig TG, Lindvall O, Tingström A. Increased neurogenesis in a model of electroconvulsive therapy. *Biol Psychiatry.* 2000;47:1043-1049
82. Malberg JE, Eisch AJ, Nestler EJ, Duman RS. Chronic antidepressant treatment increases neurogenesis in adult rat hippocampus. *J Neurosci.* 2000;20(24):9104-9110
83. Gould E. Stress, deprivation, and adult neurogenesis. In: Gazzaniga MS, ed. *The new cognitive neurosciences.* Massachusetts: Bradford; 2000:139-148.
84. Clark, C. Clinical interventions with near-death experiencers. In: Greyson B, Flynn BC, eds. *The Near-Death Experience.* Springfield: Charles C. Thomas Publisher; 1984:242-255
85. Knoblauch H. Different kinds of near-death experience: a report on a survey of near-death experiences in Germany. *Journal of near-death studies.* 2001; 20(1):15-29
86. Strubelt S and Maas, U. *Die Iboga-Heilungszeremonie im Gabun: Gelenkte Nah-Todes-Erfahrung als Psychotherapie. Ein Vergleich afrikanischen Wissens mit internationalen Forschungsergebnissen.* Lage: Jacobs-Verlag; 2006:44-52

87. Glick SD, Maissonneuve IM, Szumlinski, KK. Mechanisms of action of Ibogaine: Relevance to putative therapeutic effects and development of a safer Iboga alkaloid congener. *The Alkaloids*. 2001;56:39-62

Acknowledgements

We would like to thank Antoine Makondo and Jeannette Moussouma, traditional healers in Mitoné/Lambaréné (Gabon) for their confidence, extensive support and willingness to share their knowledge with us.