WHY FEELINGS MATTER

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"Neuropsychology, like classical neurology, aims to be entirely objective, and its great power, its advances, come from just this. But a living creature, and especially a human being, is first and last active -- a subject, not an object. It is precisely the subject, the living 'I', which is being excluded. **Neuropsychology is admirable, but it excludes the psyche** – it excludes the experiencing, active, living 'I'."

(Sacks, 1984, p. 164)

PSYCHOANALYSIS

(FEELINGS MEAN SOMETHING)

BEHAVIOURISM

(FEELINGS DON’T REALLY EXIST)

COGNITIVE SCIENCE

(FEELINGS ARE REALLY INFORMATION)

PSYCHIATRY

(FEELINGS ARE REALLY BRAIN CHEMICALS)

AFFECTIVE NEUROSCIENCE

(BRAIN CHEMICALS REALLY FEEL LIKE SOMETHING)

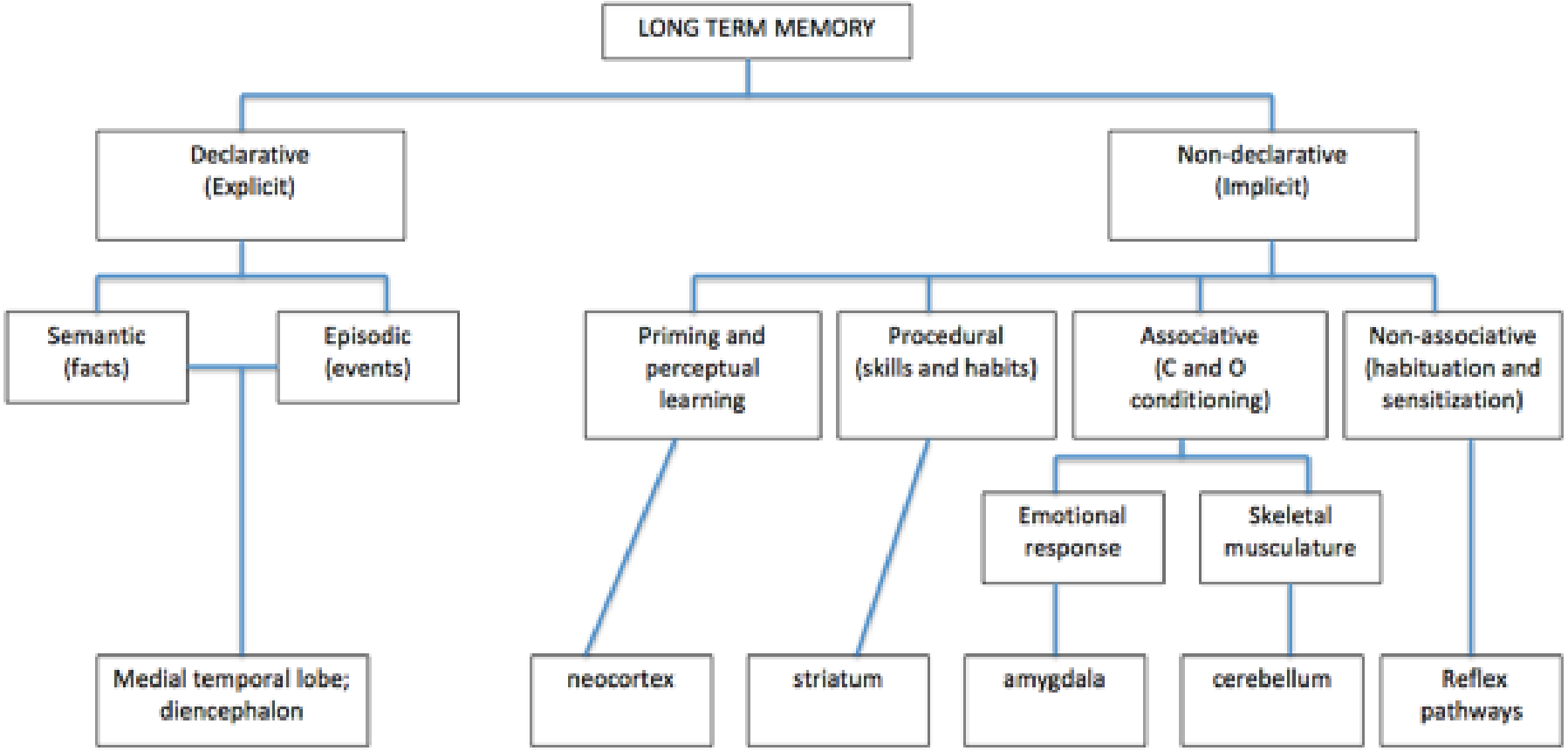
"It is undeniable that some organisms are subjects of experience. But the question of how it is that these systems are subjects of experience is perplexing. Why is it that when our cognitive systems engage in visual and auditory information-processing, we have visual or auditory experience: the quality of deep blue, the sensation of middle C? How can we explain why there is **something it is like** to entertain a mental image or experience an emotion? It is widely agreed that experience arises from a physical basis, but we have no good explanation of how and why it so arises."

David Chalmers (1995)

“An organism has conscious mental states if and only if there is something that it is like to *be* that organism—something it is like *for* the organism"

Nagel (1974)

>> *Why is there something it is like to be an organism, for the organism, and how does this something-it-is-like-ness come about*?


LONG TERM MEMORY
Declarative (Explicit)
Non-declarative (Implicit)
Semantic (facts)
Episodic (events)
Priming and perceptual learning
Procedural (skills and habits)
Associative (C and O conditioning)
Non-associative (habituation and sensitization)
Emotional response
Skeletal musculature
Medial temporal lobe; diencephalon
neocortex
striatum
amygdala
cerebellum
Reflex pathways

“The easy problems are easy precisely because they concern the explanation of cognitive abilities and functions. To explain a cognitive function, we need only specify a mechanism that can perform the function. The methods of cognitive science are well-suited for this sort of explanation, and so are well-suited to the easy problems of consciousness. By contrast, the hard problem is hard precisely because it is not a problem about the performance of functions. The problem persists even when the performance of all the relevant functions is explained …

… What makes the hard problem hard and almost unique is that it goes beyond problems about the performance of functions. To see this, note that even when we have explained the performance of all the cognitive and behavioural functions in the vicinity of experience … there may still remain a further unanswered question: **Why is the performance of these functions accompanied by experience?** A simple explanation of the functions leaves this question open … Why doesn't all this information-processing go on 'in the dark', free of any inner feel?" Chalmers (1995)

“It is surely of the essence of an emotion that we should be aware of it, i.e. that it should become known to consciousness. Thus the possibility of the attribute of unconsciousness would be completely excluded as far as emotions, feelings and affects are concerned.”

Freud (1915)

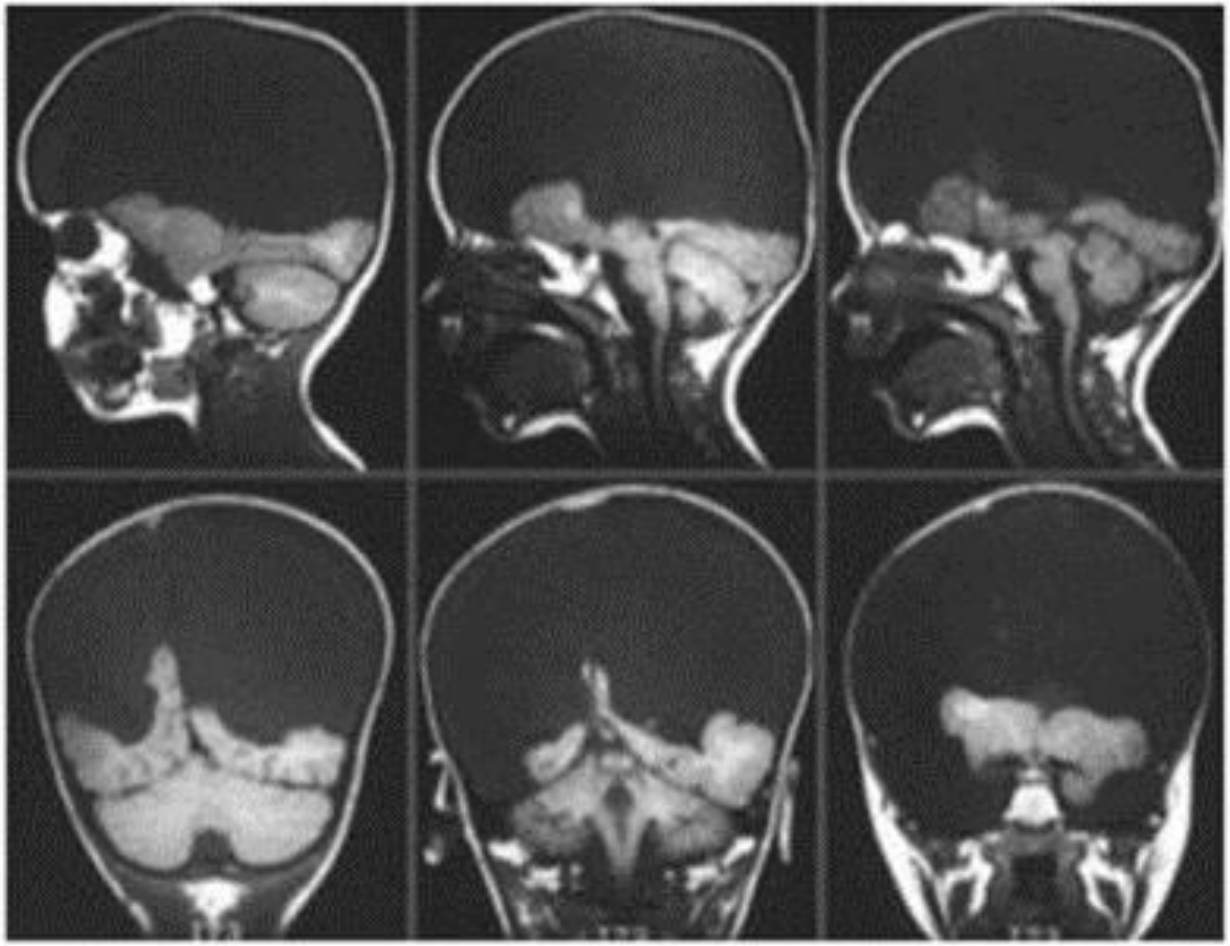
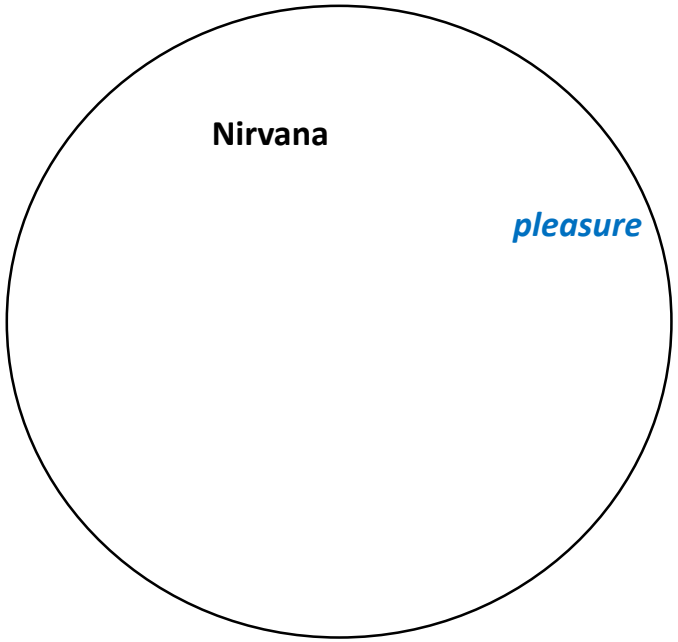
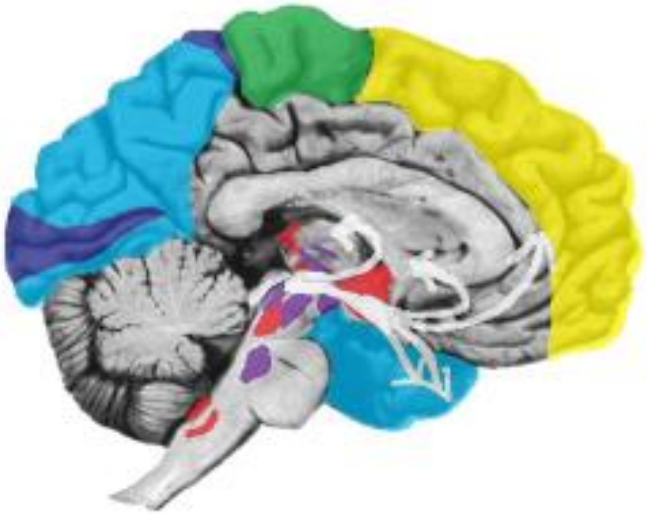


Figure 9. The reaction of a three year old girl with hydranencephaly in a social situation in which her baby brother has been placed in her arms by her parents, who face her attentively and help support the baby while photographing.



Homeostasis




pcpt.-cs.
SUPER-EGO
preconscious
repressed
EGO
unconscious
ID

The Depressing News About Antidepressants

Newsweek, Jan 28, 2010 7:00 PM EST

Studies suggest that the popular drugs are no more effective than a placebo. In fact, they may be worse.

Although the year is young, it has already brought my first moral dilemma. In early January a friend mentioned that his New Year's resolution was to beat his chronic depression once and for all. Over the years he had tried a medicine chest's worth of antidepressants, but none had really helped in any enduring way, and when the side effects became so unpleasant that he stopped taking them, the withdrawal symptoms (cramps, dizziness, headaches) were torture. Did I know of any research that might help him decide whether a new antidepressant his doctor recommended might finally lift his chronic darkness at noon?

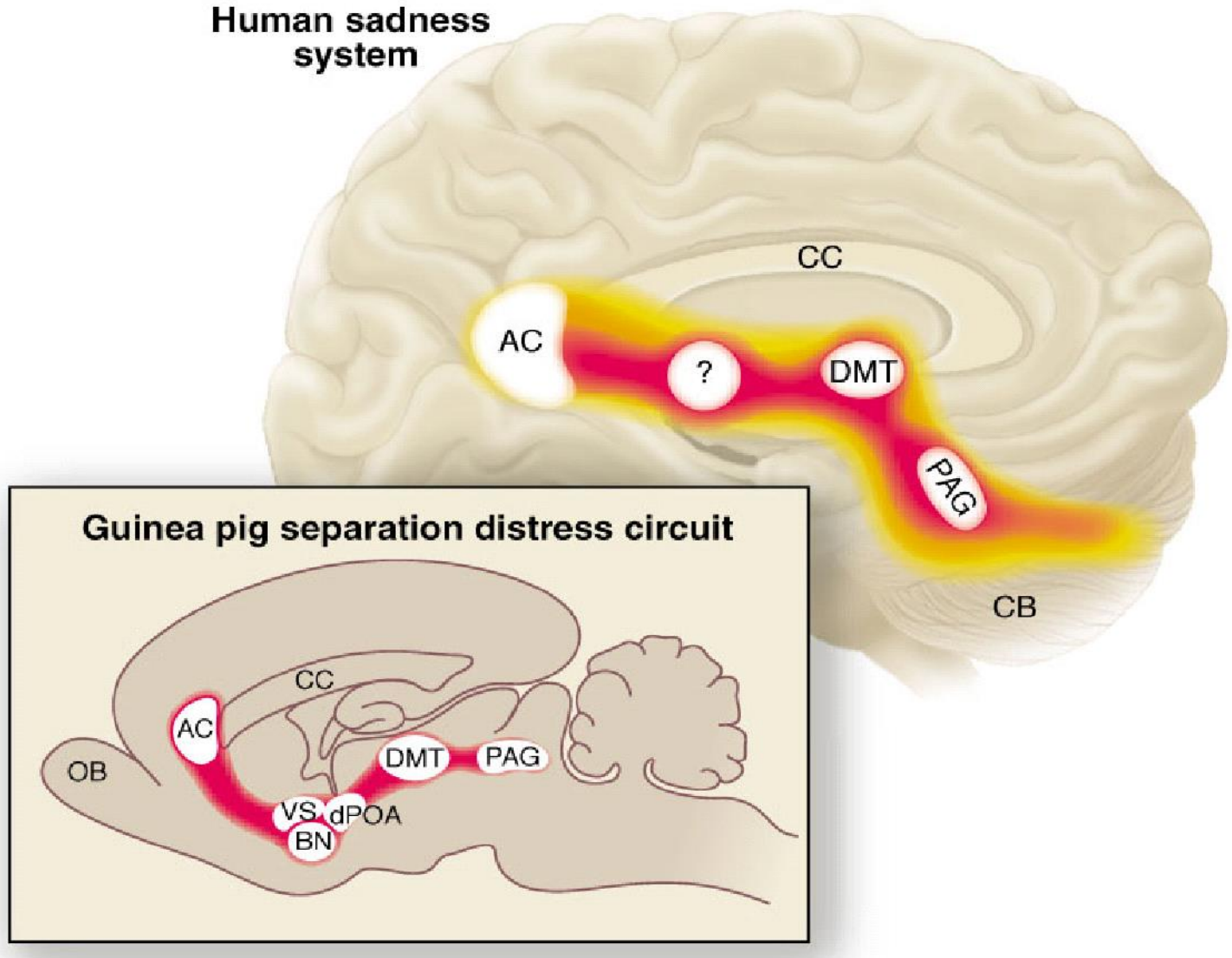
…

Major Depressive Disorder Diagnostic Criteria according to DSM-IV-TR

A	Five (or more) of the following symptoms have been present during the same 2-week period and represent a change from previous functioning. At least one of the symptoms is (1) depressed mood or (2) loss of interest or pleasure. (1) Depressed mood most of the day, nearly every day, as indicated by either subjective report or observation made by others. (2) Markedly diminished interest or pleasure in all, or almost all, activities most of the day, nearly every day. (3) Significant weight loss when not dieting or significant gain, or decrease or increase in appetite nearly every day. (4) Insomnia or hypersomnia nearly every day. (5) Psychomotor agitation or retardation nearly every day. (6) Fatigue or loss of energy nearly every day. (7) Feelings of worthlessness or excessive or inappropriate guilt (which may be delusional) nearly every day (not merely self-reproach or guilt about being sick). (8) Diminished ability to think or concentrate, or indecisiveness, nearly every day (either by subjective account or as observed by others). (9) Recurrent thoughts of death (not just fear of dying), recurrent suicidal ideation without a specific plan, or a suicide attempt or specific plan for committing suicide.
B	The symptoms do not meet the criteria for a mixed episode
C	The symptoms cause clinically significant distress or impairment in social, occupational or other important areas of functioning.
D	The symptoms are not due to the direct physiological effects of a substance (for example, a drug of abuse, a medication), or a general medical condition (for example, hyperthyroidism).
E	The symptoms are not better accounted for by bereavement, i.e. after the loss of a loved one, the symptoms persist for longer than 2 months or are characterised by marked functional impairment, morbid preoccupation with worthlessness, suicidal ideation, psychotic symptoms or psychomotor retardation.

1. PROTEST:

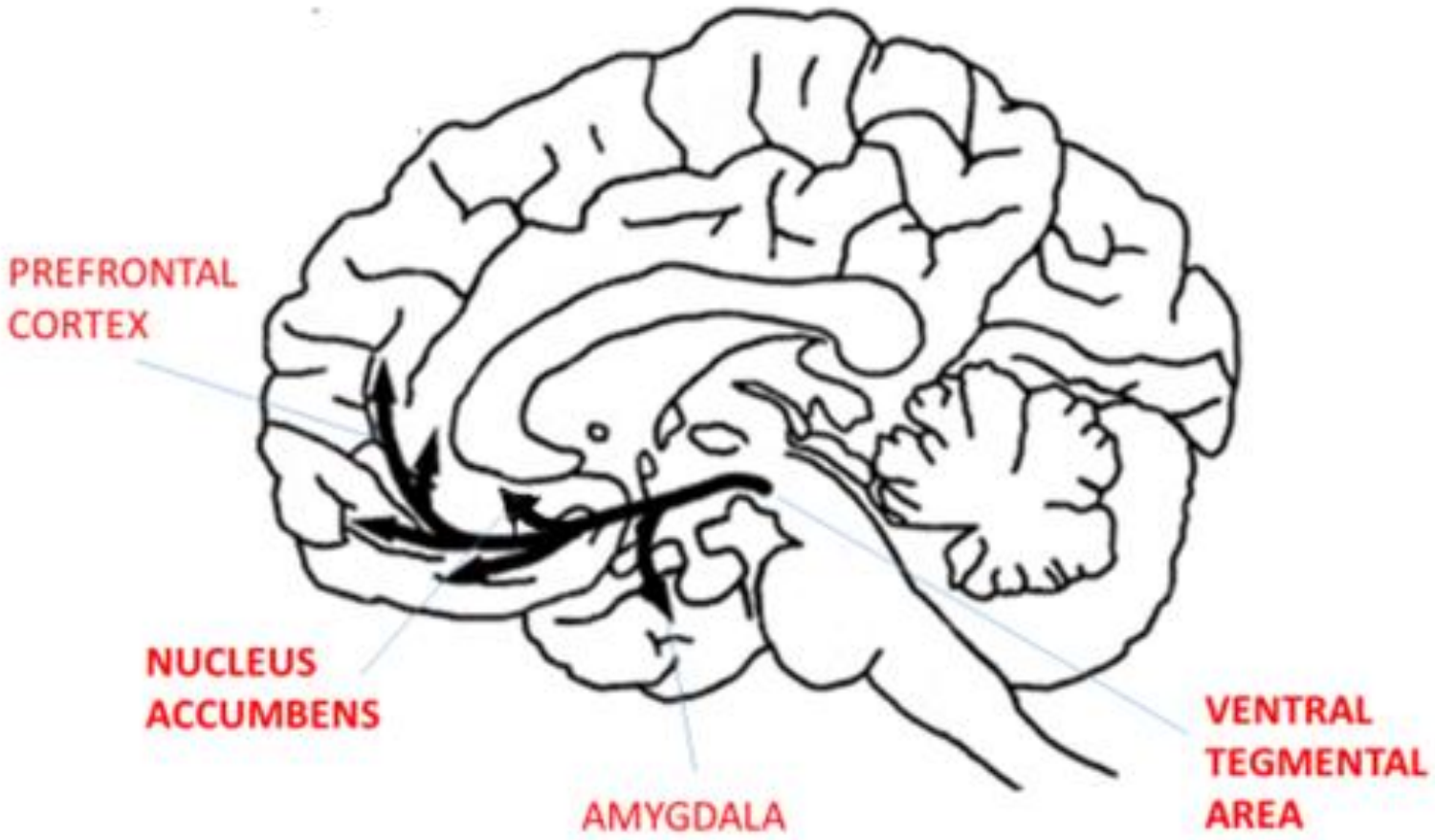
"… may begin immediately or may be delayed; it lasts from a few hours to a week or more. During it the young child appears acutely distressed at having lost his mother and seeks to recapture her by the full exercise of his limited resources. He will often cry loudly, shake his cot, throw himself about, and look eagerly towards any sight or sound which might prove to be his missing mother. All his behaviour suggests strong expectation that she will return. Meanwhile he is apt to reject all alternative figures who offer to do things for him, though some children will cling desperately to a nurse."

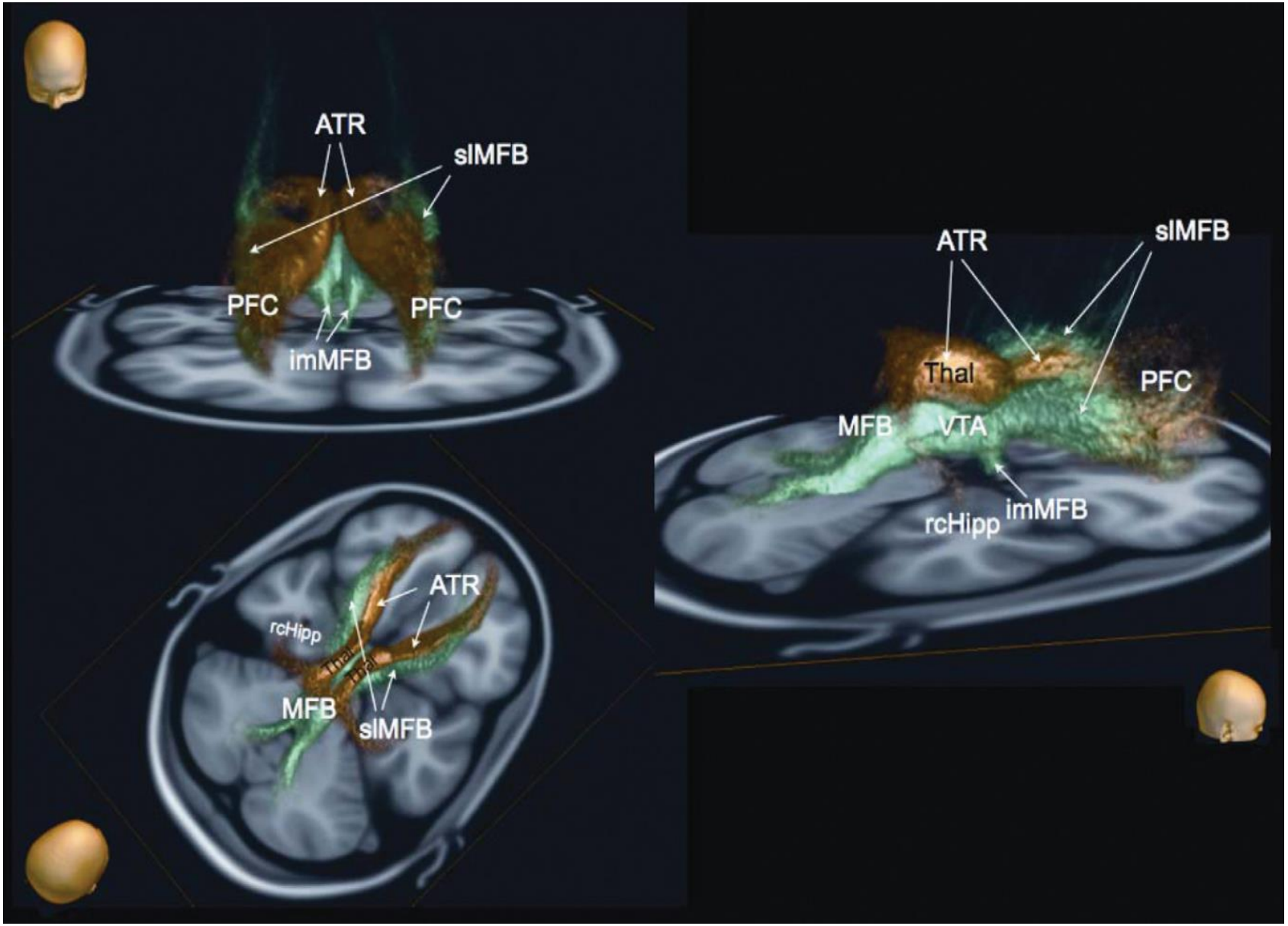


Panksepp 2003, Zubietta 2003

2. DESPAIR:

"… succeeds protest, the child's preoccupation with his missing mother is still evident, though his behaviour suggests increasing hopelessness. The active physical movements diminish or come to an end, and he may cry monotonously or intermittently. He is withdrawn and inactive, makes no demands on people in the environment, and appears to be in a state of deep mourning."


PREFRONTAL CORTEX
NUCLEUS ACCUMBENS
AMYGDALA
VENTRAL TEGMENTAL AREA


ATR
slMFB
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Ultra-Low-Dose Buprenorphine as a Time-Limited Treatment for Severe Suicidal Ideation: A Randomized Controlled Trial

Yoram Yovell, M.D., Ph.D., Gal Bar, Ph.D., Moti Mashiah, M.D., Yehuda Baruch, M.D., Irina Briskman, M.D., Jack Asherov, M.D., Amit Lotan, M.D., Amihai Rigbi, Ph.D., Jaak Panksepp, Ph.D.

Objective: Suicidal ideation and behavior currently have no quick-acting pharmacological treatments that are suitable for independent outpatient use. Suicidality is linked to mental pain, which is modulated by the separation distress system through endogenous opioids. The authors tested the efficacy and safety of very low dosages of sublingual buprenorphine as a time-limited treatment for severe suicidal ideation.

Method: This was a multisite randomized double-blind placebo-controlled trial of ultra-low-dose sublingual buprenorphine as an adjunctive treatment. Severely suicidal patients without substance abuse were randomly assigned to receive either buprenorphine or placebo (in a 2:1 ratio) in addition to their ongoing individual treatments. The primary outcome measure was change in suicidal ideation, as assessed by the Beck Suicidal Ideation Scale at the end of each of 4 weeks of treatment.

Results: Patients who received ultra-low-dose buprenorphine (initial dosage, 0.1 mg once or twice daily; mean final dosage=0.44 mg/day; N=40) had a greater reduction in Beck Suicide Ideation Scale scores than patients who received placebo (N=22), both after 2 weeks (mean difference −4.3, 95% CI=−8.5, −0.2) and after 4 weeks (mean difference=−7.1, 95% CI=−12.0, −2.3). Concurrent use of antidepressants and a diagnosis of borderline personality disorder did not affect the response to buprenorphine. No withdrawal symptoms were reported after treatment discontinuation at the end of the trial.

Conclusions: The time-limited, short-term use of very low dosages of sublingual buprenorphine was associated with decreased suicidal ideation in severely suicidal patients without substance abuse. Further research is needed to establish the efficacy, safety, dosing, and appropriate patient populations for this experimental treatment.

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Suicide, with a worldwide annual mortality approaching 1 million, is anteceded by suicidal ideation—thoughts and wishes to kill oneself (1, 2). Although suicidal ideation leads to completed suicide only rarely (2, 3), it cannot be ignored clinically (1, 2), and the management of severely suicidal outpatients remains an enormous clinical challenge (2, 4). Standard antidepressants relieve suicidal ideation, but this may take several weeks, and not all patients respond adequately (1, 4–7). Atypical antipsychotics (8, 9) and lithium (10) have been found to decrease suicidal ideation and/or suicidal behavior in specific patient populations. Ketamine is effective as a quick-acting treatment for suicidal ideation and depression, but necessitates repeated administration under medical supervision (11, 12). Thus, no short-term pharmacological treatments that are suitable for independent outpatient use are currently available for suicidal ideation.

Depression, which is often accompanied by suicidal ideation, shares neurobiological and psychological characteristics with separation distress—the innate, painfully anxious, dysphoric response of young animals and humans to separation from their attachment figures (13–17). Remarkably low, nonsedating doses of opioid analgesics have been found to inhibit separation distress in every animal species tested (15–17). The neuroanatomy of the separation distress system overlaps with the brain's "pain matrix" and shares some of its neurotransmitter substrates (16, 17). It has been suggested that abrupt cessation of opioid release upon separation from attachment figures contributes to painful separation feelings in animals and humans (16, 17).

Patients with borderline personality disorder, who are exquisitely sensitive to separations, often become suicidal after interpersonal rejections (4, 18), and this patient population has been found to have abnormalities in their endogenous opioid systems (19). Among patients in psychotherapy, most suicidal acts occur after interpersonal losses or rejections (20), and analgesic treatment has been shown to decrease social pain in rejected lovers (21). Social rejection activates the endogenous


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