

Re: Some brain questions i need help with

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From: John Hasenkam (johnh_at_faraway.)

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"dan michaels" <feedbackdroids@yahoo.com> wrote in message
news:8d8494cf.0409240735.3206463c@posting.google.com...

> *"John Hasenkam"* <johnh@faraway.> wrote in message
news:<41536e36@dnews.tpgi.com.au>...

>

>

> > *About 20 years ago I had a rare nightmare where I woke up in the early
> > morning hours drenched in sweat and quite terrified, in the dream I was
just*

> > *about to be killed. This dream obsessed me for many months, somehow I
knew*

> > *it carried an important message and it was very symbolic in nature.*

>

>

> *Once, 20 years ago ???*

That's it, even when I tried writing down dreams to look for patterns the emotional content was nearly absent. For the most part I'm just grateful my brain can generate entertainment for me while asleep. Even last night I nearly got killed twice through reckless driving but no sweat I just kept driving like a maniac (in my younger days I nearly got killed several times on the bike). In one situation it was physically impossible for me to avoid the accident but somehow I managed to retrieve control yet thought nothing of it. Last night was a typical example of my current dreams, lots of exploration with an old friend as we explored some new territory and figured out how to make it work to our advantage. In dreams I am typically on the move, usually on some adventure or solving some practical problem (never conceptual problems). No-one dies in my dreams, I'm not even sure really bad things happen to anyone. However, I am a freak so I am not surprised that my dreaming experience is different from others.

What might be interesting is to contrast dreams that occur during REM and NREM sleep. NREM dreams are much harder to assess but there is more research being conducted in this area. The Hobson et al text I am reading states that considerable controversy still exists in regard to these two dreaming states. (What about day dreams, where we still are monitoring the environment? Not nearly as bizarre, if bizarre at all.) The authors also

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make this important point:
Hobson et al ...

The sleep laboratory itself constitutes a second major methodological problem. Anyone who has ever slept in a sleep laboratory (as all of us have!) knows that it is an inhospitable and unnatural setting which makes sleep more difficult and less deep than is possible in more naturalistic settings.

Additionally, how people report their dreams is contingent upon certain features of their cognition. Eg. Vivid images usually require more words to be described, so verbal reports of such accounts are much longer than accounts of mundane images in dreams and may reflect a reporting bias. REM dreams are more vivid because of greater cortical activation, reporting of NREM sleep may be hindered by failure to remember the same. NREM dreaming is active but we remain largely unaware of it. There are important differences in these types of dreams but is this a matter of reporting or of actual differences in these dream states:

Hobson et al ...

Therefore, we conclude that

while some NREM dreams approach REM dreams in length, vividness, dreaminess and bizarreness (Cicogna et al., 1998; Foulkes & Schmidt 1983; Herman et al. 1978; Nielsen, 1999) and while "dream-like" versus "thought-like" mentation may predominate in some NREM reports (Foulkes 1962; Nielsen 1999; Rechtschaffen et al. 1963a; Zimmerman 1970), NREM reports are far more likely than REM reports to be short, dull and undreamlike (Nielsen 1999; Rechtschaffen et al. 1963).

Some studies have indicated the exercise of being asked to recall dreams changes reports over time. Thus people interested in their dreams may often generate differing reports than those who simply remember some dreams but are not too interested in the exercise. The conscious attention to dreaming changes the reporting, I suspect it also changes the dreaming. Now that's a problematic confounder.

Another problem is that memory is severely degraded in sleep and just prior to sleep, thus making verbal recall problematic. What is remembered and reported may at best be a very fragmentary and misleading account of dreams:

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All of the above findings can be regarded as being caused by the failure of recent episodic memory (as defined by Tulving 1994) in sleep. And as we have noted, recent episodic memory is weak across wake-sleep and sleep-wake transitions as well as within sleep itself (Pace-Schott et al. 1997a). We believe that a deficiency of memory in dreaming may go a long way toward explaining such distinctive and robust dream phenomena as orientational instability, loss of self-reflective awareness, and failure of directed thought and attention.

With many caveats regarding the technologies employed, the authors offer these neurophysiological observations:

...

Instead of the global, regionally nonspecific picture of forebrain activation that has been suggested by EEG studies, all of these new imaging studies indicate a preferential activation of limbic and paralimbic regions of the forebrain in REM compared to waking (Braun et al. 1997, 1998; Maquet et al. 1996; Nofzinger et al. 1997). One implication of these discoveries is that dream emotion may be a primary shaper of dream plots rather than playing a DREAMING and the BRAIN: Toward a Cognitive Neuroscience of Conscious States <http://home.earthlink.net/~sleeplab/bbs/BBS.html> (40 of 222) [1/6/2000

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secondary role in dream plot instigation.

This validates Dan's remark that dreams have high emotional content. What am I then, cloned Spock?! Lester is probably correct in asserting I am sleeping lightly at present, hence not much REM sleep, which may explain lethargy, poor cognition, and easy awakenings; especially since I have been taking the SSRI for sometime.

Especially given:

...

9) There is near universal suppression of REM sleep in humans by acute dosage of serotonin and norepinephrine reuptake-inhibiting antidepressants (Gaillard et al. 1994; Nicholson et al. 1989; Vogel 1975; Vogel et al. 1990).

The SSRI I am taking is also a nore reuptake inhibitor

...

Continuing with Hobson et al ...

In REM compared individually to delta NREM and to pre- and post-sleep waking (see Table 2), these authors showed relative activation of the pons, midbrain, anterior hypothalamus, hippocampus, caudate, and medial prefrontal, caudal orbital, anterior cingulate, parahippocampal and inferior temporal cortices (Braun et al. 1997). Based on their observations, the Braun group then offered the following speculations which are relevant to the neurology of dreaming:

- 1) Ascending reticular activation during REM as compared to waking may favor a more ventral cholinergic route leading from the brainstem to the basal forebrain over a more dorsal route via the thalamus.
- 2) Activation of the cerebellar vermis in REM may reflect input to this structure from the brainstem vestibular nuclei. We note that these nuclei also constitute an important potential source of neuronal activation causing the unique vestibular features of fictive movement in dreams (Hobson et al. 1997; Leslie & Ogilvie 1996; Sauvageau et al. 1998).
- 3) Noting both a particularly strong REM sleep-related activation of the basal ganglia and the known connectivity of these subcortical structures, Braun et al. suggest that the basal ganglia may play an important role in an ascending thalamocortical activation network. They suggest that this network extends successively from the brainstem to the intralaminar thalamic nuclei, then to the basal ganglia, and back to the ventral anterior and ventromedial thalamic nuclei, and thence to the cortex.

...

The role of the basal ganglia in the initiation of motor activity may, in turn, be related to the ubiquity of motion in dreams (Hobson 1988b; Porte & Hobson 1996).

...

5) The prominent decrease in the executive portions of the frontal cortex (dorsolateral and orbital prefrontal cortices) contrasts with the REM-associated increase in activation of the limbic associated medial prefrontal area. This medial area region has the most abundant limbic connections in the prefrontal cortex, has been associated with arousal and attention, and disruption of this area has been shown to cause confabulatory syndromes formally similar to dreaming. (Note also the dream-wake confusional syndrome associated with anterior limbic cortical lesions reported by Solms 1997.)

Nevertheless, it seems likely that considerable portions of executive and association cortex active in waking may be far less active in REM leading Braun et al. (1997) to speculate that "...REM sleep may constitute a state of generalized brain activity with the specific exclusion of executive systems which normally participate in the highest order analysis and integration of neural information" (p. 1190).

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Not only is REM sleep chemically biased but the preferential cholinergic neuromodulation is associated with selective activation of the subcortical and cortical limbic structures (which mediate emotion) and with relative inactivation of the frontal cortex (which mediates directed thought). These findings greatly enrich and inform the integrated picture of REM sleep dreaming as emotion-driven cognition with deficient memory, orientation, volition and analytic thinking.

...

In addition to prominent thalamic deactivation, all three recent studies have found regional deactivation during NREM in the pontine brain stem, orbitofrontal cortex and anterior cingulate cortex (Braun et al.

...

In a second syndrome, "visual anoneria," bilateral medial occipito-temporal lesions produce full or partial loss of dream visual imagery (again with normal waking vision).

This suggests an important difference in Dream visual imagery and ordinary seeing?

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A second concern is the often reported lack of emotion-related physiological arousal accompanying dream events (e.g., violence) which would easily elicit such arousal in waking (Perlis & Nielsen 1993). Such emotional "numbing" in dreams could result both from a sleep-related dissociation of peripheral and central autonomic activity (as with peripheral arousal in Stage 4) combined with REM-related blockade of central readout to the periphery and peripheral sensory feedback to the CNS.

Above touches on what I mentioned before about reduced affect being the result of agents external to the CNS.

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The left dorsolateral prefrontal cortex has been shown to be selectively activated during human reasoning tasks (Goel et al. 1998). Its deactivation could account for the illogical ad-hoc explanations offered for bizarre occurrences (Williams et al. 1992). Similarly, the prefrontal cortices have been consistently shown to activate during episodic and working memory tasks (Brewer et al. 1998; Courtney et al. 1997; Cohen et al. 1997; Fletcher et al. 1997; Tulving et al. 1996; Wagner et al. 1998); their deactivation in REM may contribute to the prominent mnemonic deficits in dreaming noted above in Section II.C.4.

> In fact, I suspect that highly-emotional dreams and sweating happens
> very frequently. At least it does to me. Most of the time you just
> don't wake up, or else you wake up too late to remember the dream.
> Occasionally, if there is a lot of emotional content, I wake up
> "quickly" enuf to remember it.
> Dreams have different levels of emotional content, rarely do I have
> what you have described as a nitemare, but many dreams do involve
> plenty of emotion, people dying, etc. I just assume it's one part of
> the dream phenomenon, and don't get rattled about it. It's just
> made-up.