neither practice in scanning a particular series nor lengthening of the time it has been stored in memory need increase the rate at which it is scanned.

In accounting for human performance in other tasks that appear to involve multiple comparisons, theorists have occasionally proposed that the comparisons are carried out in parallel rather than serially (*13, 14*). (This perhaps corresponds to the assumption mentioned earlier that the momentary capacity of consciousness is several items rather than only one. Are the present data inconsistent with such a proposal? Parallel comparisons that begin and also end simultaneously (*14*) are excluded because the mean latency has been shown to increase with s. A process in which multiple comparisons begin simultaneously is more difficult to exclude if the comparison times are independent, their distribution has nonzero variance, and the response is initiated when the slowest comparison ends. A linear increase in mean latency cannot alone be taken as conclusive evidence against such a process. The magnitude of the latency increase that would result from a parallel process is bounded above, however (*15*); it is possible to apply the bound to these data (*16*). This was done for the negative responses in both experiments, with the results shown by the broken curves in Figs. 1 and 2. Evidently, the increase in response latency with s is too great to be attributed to a parallel process with independent comparison times (*17*).

Other experiments provide added support for the scanning theory (*16*). Two of the findings are noted here: (i) variation in the size, n, of the negative set ($n \geqslant s$) had no effect on the mean latency, indicating that stimulus confusability (*10, 18*) cannot account for the results of experiments 1 and 2; (ii) variation in the size of a response-irrelevant memory load had no effect on the latency function, implying that the increase in latency reflects the duration of retrieval and not merely the exigencies of retention.

The generality of the high-speed scanning process has yet to be determined, but there are several features of experiments 1 and 2 that should be taken into account in any comparison with other binary classification tasks (*14, 19*): (i) at least one of the classes is small; (ii) class members are assigned arbitrarily; (iii) relatively little practice is provided; (iv) high accuracy is required and errors cannot be corrected; and (v) until the response to one stimulus is completed the next stimulus cannot be viewed.

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6. For both experiments the data subjected to analysis of variance were, for each subject, the mean latency for each value of s. So that inferences might be drawn about the population of subjects, individual differences in mean and in linear-regression slope were treated as "random effects." Where quantities are stated in the form $a \pm b$, b is an estimate of the standard error of a. Such estimates were usually calculated by using variance components derived from the analysis of variance.
7. The analyses of variance for both experiments provided a means of testing the significance of differences among individual slopes. Significance levels are .07 (experiment 1) and .09 (experiment 2), suggesting true inter-subject differences in slope; the population distribution of slopes has an estimated standard deviation of 8.0 msec per symbol.
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Anxiety Levels in Dreams: Relation to Changes in Plasma Free Fatty Acids

Abstract. *Blood samples for determination of plasma free fatty acids were obtained throughout the night by means of an indwelling catheter. The first sample was drawn at the onset of rapid eye movements and a second after 15 minutes of these movements. Subjects were then awakened and asked to relate their dreams; a third sample was drawn 15 to 25 minutes later. Anxiety scores derived from 20 dreams of nine subjects had significant positive correlations with changes in free fatty acids occurring during REM sleep. No statistically significant relation was found between anxiety and the changes in free fatty acids occurring from the time just before awakening to 15 to 25 minutes later. Presumably, anxiety in dreams triggers the release of catecholamines into the circulation, and these catecholamines mobilize proportional amounts of free fatty acids from body fat.*

A previous study of the relation of emotions and blood lipids during the waking state revealed a significant positive correlation between low levels of arousal of anxiety, as determined from a 5-minute period of free associative speech (1), and concentrations of free fatty acids (FFA) in plasma. No essential correlation was found in this study between hostility and concentrations of FFA. The positive correlation between anxiety levels and FFA raised the question whether a similar relation might occur while subjects are in a dream state. Our study was undertaken to explore such a possibility.

Nine paid volunteer male subjects, ranging in ages from 19 to 25, were asked to sleep overnight in a dream laboratory, arranged to appear as a hospital room. Subjects slept 1 or 2 nights in the laboratory but were not given preliminary periods in which to become accustomed to sleeping in this room. They were told that we were investigating sleep, dreams, and changes in body chemistry. Subjects were instructed not to eat after their evening meal at 6:00 p.m. and to report to the laboratory at 11:00 p.m. At this time a venipuncture was performed in the left antecubital vein with a No. 18 thin-

walled needle. A fixed-core stainless steel wire (diameter, 0.8 cm) was inserted into the vein, the needle was removed, and the tip of a 122-cm thin-walled polyethylene catheter (inside diameter, 0.18 cm) was inserted over the wire into the vein. The wire was then removed. The catheter was attached to a three-way stopcock so that normal saline could be slowly infused to prevent clotting and the subject could move comfortably throughout the night. Blood samples were drawn through the stopcock, and since only 1.5 ml of catheter dead space was present, the first 2 ml of each sample was discarded to eliminate possible dilution by the saline drip. These samples could be obtained without awakening the subject.

The rapid eye movement (REM) dream state was detected by means of electrodes attached to the outer angles of the eyes, and ocular movements were recorded on a Grass polygraph, model 5, at a paper speed of 0.5 mm/sec according to the method of Aserinsky and Kleitman (*2, 3*).

The initial sample of blood was drawn at the time of venipuncture, and samples for determination of FFA were drawn at onset of the REM state and 15 minutes afterwards. Subjects were then awakened and asked to report any dreams; all verbal responses were recorded on electronic tape. Subjects were then allowed to go back to sleep. Another sample of blood was drawn 15 to 25 minutes after this brief awakening. In all instances the subjects had presumably resumed sleeping by this time, but no REM's were occurring. This procedure was repeated for all REM states during the night. Subjects were not permitted to sit up or eat during the experimental period.

The reports of dreams were independently analyzed and scored by two technicians for anxiety, hostility out, hostility in, and ambivalent hostility by the method of Gottschalk and Gleser (*4, 5*). Reports having 70 or more words were the only ones used for analysis because the reliability of smaller samples has been found to be relatively poor. The two sets of scores were averaged on 20 scorable dreams occurring on 14 nights in all subjects. Concentrations of FFA were determined by the method of Dole (*6*) as modified by Trout *et al.* (*7*).

Figure 1 shows a curve of the average concentration of FFA obtained throughout the 14 nights of sleeping and dreaming of the nine subjects. A small average increase in the concentration was found to occur and reach its peak 2 to 3 hours after retiring at 11:00 p.m. Free fatty acids returned to the bed-time (5-hour, postprandial) level about 5 hours after going to sleep. These findings have not been previously reported, and they are important to note as a nocturnal base-line rhythm in the concentration of FFA on which is superimposed the more transient changes in FFA associated with dream anxiety.

The rank-order correlation of anxiety scores derived from 20 dreams and the associated change in FFA, occurring between the onset of REM activity and 15 minutes of REM sleep, was 0.62 ($P < .01$). The range of changes in FFA during the 15-minute REM periods was +14 to −11 meq/liter, which was +28 to −21 percent of the FFA level at the onset of REM periods. None of the correlations between these specific changes in FFA and the hostility scores derived from the dreams was significant. Furthermore, there were no significant correlations between the dream anxiety or hostility scores and changes in FFA that occurred between the time just before awakening the subject to tell his dream and 15 to 25 minutes later (see Table 1).

Figure 2 shows the average concentrations of FFA during REM sleep and 15 to 25 minutes after the interruption of REM sleep for the nine subjects over the 1 or 2 nights they slept in the laboratory. With these nine subjects, the concentration of FFA tended to increase during the first 15 minutes of REM sleep, in the instance of the first and third REM periods, and to decrease during the comparable interval of the second REM period. None of the variations in the average concentration of FFA during each of the REM pe-

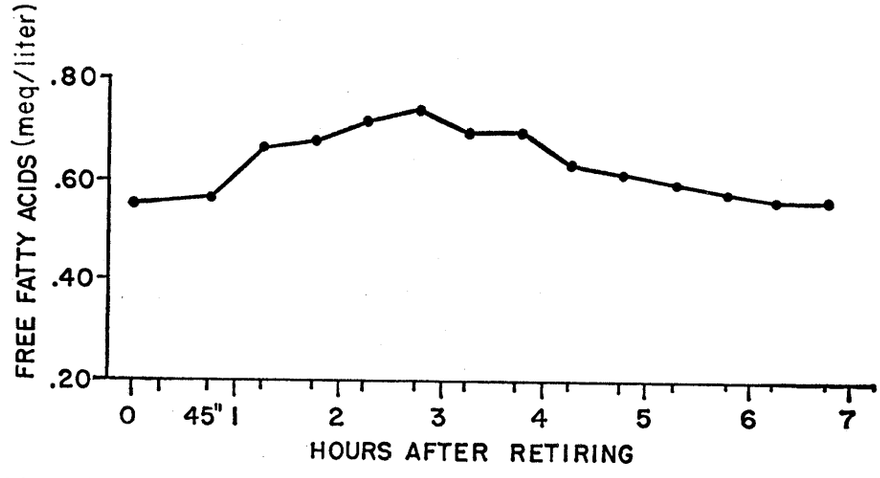

Fig. 1. Average concentrations of free fatty acids of nine males. Determinations were made at ½-hour intervals while subjects were sleeping and dreaming for a period of 14 nights.

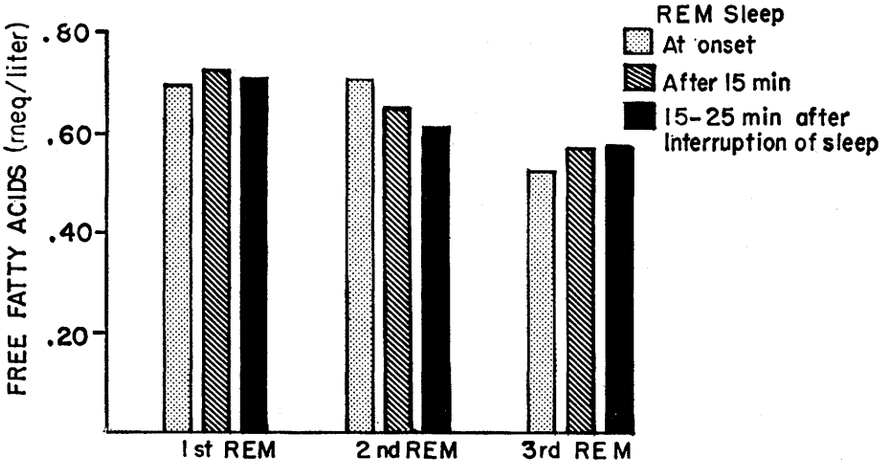

Fig. 2. Average concentrations of free fatty acids of nine males during REM sleep and 15 to 25 minutes after the interruption of REM sleep. The first REM period represents 13 nights; the second, 12 nights; and the third, 8 nights.

Table 1. Rank-order correlations between levels of anxiety and hostility and changes in free fatty acids (FFA) during sleeping and dreaming.

Anxiety	Hostility out	Hostility in	Ambivalent hostility
FFA changes during first 15 minutes of REM			
+.62*	+.31	−.24	+.13
FFA changes from preawakening level to 15 to 25 minutes later			
+.11	+.02	−.37	+.27

* Significant at $P < .01$.

riods and the corresponding non-REM period immediately thereafter were statistically significant.

Further analysis of our data reveals that most of our subjects reported scorable dreams (that is, 70 words or more) after awakening from the second or third REM periods. Eight awakenings after the first REM period elicited either reports of no dreams at all (on five occasions), "feelings and thoughts" (on two occasions), or dreams with too few words (on one occasion); most of these first REM's occurred before 3 hours of sleep had elapsed. Four out of five of the first REM's that were followed by scorable dreams on awakening occurred three or more hours after going to sleep.

Finding of a positive correlation between magnitude of the anxiety content of dreams (using the Gottschalk-Gleser anxiety scale) and changes in plasma FFA that occur during the first 15 minutes of REM sleep has not been previously pinpointed. This finding strongly suggests that the anxiety content of dreams is capable of triggering adrenergic discharges.

Lack of a significant correlation between anxiety scores from dreams and the changes that occur in the concentration of plasma FFA from the preawakening level to that observed 15 to 25 minutes later may be due to any one of a number of factors, or a combination of these factors. Our previous work (*1*) indicated that in waking subjects changes in concentration of FFA reach their peak within 15 to 20 minutes after an anxiety stress, such as from a venipuncture, and then tend to return to the prestimulus level; it is likely that, for most people, any changes in FFA due to the anxiety content of the dream largely disappear 30 to 40 minutes after onset of the dreaming state. In addition, the gradual nocturnal rhythm that occurs in concentrations of FFA after fasting from 6:00 p.m. (Fig. 1) may tend to obscure other more transient changes in plasma FFA that occur during and immediately after REM sleep. Still another possibility is that shifts from REM sleep to the waking state and then to non-REM sleep may be accompanied by either mild changes in metabolic rate or the level of catecholamine secretion, or both, which can affect the concentration of FFA and hence mask possible prolonged or delayed reactions to the level of anxiety in dreams.

Scott (*8*) has reported two major patterns of change in FFA during REM sleep—one in which the concentration rises (pattern II) and which occurs more frequently early in the night; the other in which the concentration drops (pattern I). He speculated that these changes in the concentration of FFA correspond to changes in "sympathetic tonus," but he carried out no independent measure of sympathetic nervous system arousal. Our findings with respect to the direction of changes in FFA during REM sleep (Figs. 1 and 2) correspond roughly to Scott's observations except that a small average rise in the concentration was observed later in the night on those nights during which there was a third REM period. Our subjects probably did not have more than two or three REM periods per night because, as previously noted (*9*), they were not adapted to the dream laboratory before collection of data began.

Other kinds of evidence strongly suggest that arousal of anxiety associated with stimulation of the sympathetic nervous system accounts for the changes in FFA that we observed. Gottschalk *et al.* (*1*) found significant correlations between anxiety scores derived from verbal behavior and concentrations of FFA in two groups ($N = 24$ and 20) of fasting males. Gottlieb *et al.* (*10*) found a significant correlation between anxiety scores measured by verbal behavior and decreases in skin temperature in 12 boys 16 to 17 years old. Karacan (*11*) found that the penile erections that commonly occur in male subjects during REM sleep are inhibited when dreams have high anxiety levels, measured by the Gottschalk-Gleser anxiety scale. Fisher (*12*), using a clinical method of assessing anxiety, reported a similar finding. Snyder (*13*, p. 382) reported "a marked correlation between REM periods and episodes of nocturnal angina." Nowlin *et al.* (*14*) also found a high degree of association with nocturnal angina, and, in addition, he found a significant depression of the ST segment in the electrocardiogram after onset of REM activity. Although the patients of Nowlin *et al.* were not asked the content of dreams when they awakened with nocturnal chest pain, they were questioned for any dream recall on the morning after each study; generally, dreams which preceded an awakening with chest pain involved either strenuous physical activity or the emotions of fear or anger. We speculate that the angina occurs principally with REM dreams that have a high anxiety content and that otherwise the angina is minimal or absent.

There is substantial evidence that epinephrine and norepinephrine (*6, 15*) can increase concentrations of FFA and that this effect on FFA is greatest with primary and secondary catecholamines that are hydroxylated on the β-carbon of the side chain (*16*). Furthermore, Bogdonoff and Estes (*17*) and Fishman *et al.* (*18*) found that emotionally stressful situations induce elevation of FFA in human subjects. These findings strongly suggest that the affect of anxiety, measurable from the dream content, leads to adrenergic stimulation and consequent transient elevation of FFA in plasma during sleep.

It has long been common knowledge to every troubled sleeper that nightmares may awaken him and that he may find himself in a state of high autonomic arousal with a great deal of associated physiological activation; but there has been no satisfactory evidence that mild to moderate emotional experiences during dreams are capable of activating physiological and biochemical activities. Our study, with the studies of Karacan and Fisher, begins to provide some of the missing evidence.

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Binocular Disappearance of Monocular Symmetry

Abstract. *In an earlier demonstration binocular shapes were produced from monocularly shapeless, random-dot stereo images. A reversal of this phenomenon is demonstrated. A stereo image is devised in which the monocularly apparent shapes of bilateral symmetry disappear when stereoscopically viewed. This phenomenon sharpens the implications of the earlier one.*

In 1960 a perceptual phenomenon was reported which demonstrated that binocular shapes can be perceived from monocularly shapeless and contourless, random-dot stereo images (*1*). The finding that correlated areas in the left and right images could give rise to stereoscopic depth perception, regardless of the fact that these areas were completely disguised when viewed by one eye, has several theoretical and practical implications. An obvious implication is that no monocular shape recognition is necessary for stereopsis. A possible reversal of this phenomenon might be also instructive. Would it be possible to generate monocular shapes which disappear when viewed binocularly?

In order to achieve such a goal the left and right images have to differ from each other, since identical or similar images yield a binocular percept which closely resembles the monocular constituents. On the other hand, different images can give rise to binocular rivalry and cannot be fused. The way out of this dilemma is the realization that it is not the physical images which have to be dissimilar but their monocular *percepts*. The nonlinearity of the perceptual processes can result in great differences in the perception of stimuli which differ only slightly.

In the reported experiment the technique of random-dot stereo images was employed. The only departure from randomness was introduced in the form of bilateral symmetry. The upper half of the left field of Fig. 1 was randomly dotted; the lower half field was its mirror image reflected across the central horizontal axis. This bilateral symmetry can be perceived spontaneously without scrutinizing the stimulus. The right image of Fig. 1 is identical to the left image except for horizontal translations. To achieve this, the image was subdivided into twenty horizontal stripes, each five picture-elements wide. The stripes were horizontally shifted alternatively to the left and to the right, by two picture-elements. These alternate shifts were arranged such that if the *n*th stripe above the symmetry axis was shifted to the right, then the *n*th stripe under the symmetry axis was shifted in the opposite direction. This procedure achieved two results. First, when viewed monocularly, the left image can be perceived as a "one-axis kaleidoscope" while the right image lacks the impression of bilateral symmetry. Second, when stereoscopically viewed, the right image can be perceived as an ordinary random-dot stereo image having alternate stripes at two depth levels.

When the stereo pair of Fig. 1 is presented in a stereoscope to subjects with normal stereoscopic vision, fusion occurs immediately and the horizontal stripes are perceived in vivid depth. Provided the subjects have no strong dominance of one eye, or provided the *symmetric* pattern is viewed with the *nondominant* eye, the bilateral symmetry cannot be perceived in the fused binocular image. Even when the images are first viewed monocularly and the bilateral symmetry becomes apparent in one of them, the binocular percept does not give the impression of a kaleidoscope (*2*). In the binocular percept, those horizontal stripes which are symmetrical belong to different depth planes and also seem to be shifted horizontally relative to each other. It is probably this alternate shift which obscures the symmetry. Indeed, when subjects with a strongly dominant eye fuse the stereo pair, such that the *dominant* eye views the symmetric image, the stripes are seen in depth but without the horizontal shifts. In this case, the bilateral symmetry can be detected in the binocular image, but is still not as spontaneously apparent as in the monocular image.

It might be argued that the disappearance of symmetry under stereopsis results from two factors: first, the symmetry is hard to detect even monocularly; and second, the nonsymmetric display for one eye is competing with the other. It is true that bilateral symmetry of random patterns across a horizontal axis is not as easily detectable as if the axis were vertical or the pattern contained several symmetries. Nevertheless, when the bilateral symmetry becomes apparent it gives a strong and stable impression. The second argument seems convincing, but actually the opposite is true. The nonsymmetric display is not uncorrelated "noise" which masks the symmetric pattern, but a totally correlated pattern which gives

Fig. 1. Stereo pair which, when monocularly viewed, contains an image of bilateral symmetry. When viewed stereoscopically, horizontal stripes are perceived in depth and the bilateral symmetry disappears.