Table 2. Mistakes on the GDSA are compared, by the Wilcoxon matched-pairs signed-ranks test (two-tailed), with respect to the condition under which they occurred (nondrug versus drug) and with respect to the point at which they occurred—that is, early (before subject reached the number 80 in the test) compared to late (after subject passed 80). Mistakes made during 48 nondrug trials (placebo and base line) are compared to those made during 48 drug trials (all doses of THC at 1½ and 3½ hours after ingestion). Not significant, N.S.

Trial	Serial immediate memory mistakes			Miscalculations		
	Early	Late	*P*	Early	Late	*P*
Nondrug	5	3	N.S.	16	18	N.S.
Drug	20	65	.01	26	23	N.S.
P	.05	.01		N.S.	N.S.	

er doses. This suggests that factors other than the span of short-term memory may be involved in temporal disintegration.

Analysis of types of mistakes made during performance of the GDSA showed that, with increasing doses of THC, there were progressively more errors in the serial or "working" functions of immediate memory ($P < .035$, Wilcoxon matched-pairs signed-ranks test). Serial immediate memory errors include loss of place, failure to alternate between subtraction and addition, and blocking (*3*). Compared to the short-term retention of a list of items, as measured by the digit spans, serial immediate memory requires the subject to keep track of inputs at the same time that he manipulates them. Difficulties in this process appear to be involved in temporal disintegration, because during GDSA performance THC induced significantly more serial immediate memory errors, as opposed to miscalculations, when subjects got nearer to their goals—that is, when they had to begin making adjustments to reach their goals exactly (Table 2). In switching back and forth between their most recent operation and the goal, in the attempt to temporally link the means to the end, the subjects frequently lost track of where they were in the serial process. To quote one subject, "I'd pick out a number now and then go ahead. . . . Coming back, I'd forget which number I just did or what I was supposed to do next."

Furthermore, with increasing doses of THC, there were progressively more errors in reaching the goal. Of particular relevance to temporal disintegration is the mistake of disregarding the goal, which occurred only under drug conditions. Here, subjects kept going right past the goal until they later checked themselves when they realized they had not brought the goal, which they remembered accurately, into awareness at the right time.

This temporal incoordination of recent memories with intentions may account, in part, for the disorganization of speech patterns that occurs under marihuana intoxication (*8*). As one subject remarked, "I can't follow what I'm saying . . . can't stay on the same subject . . . I can't remember what I just said or what I want to say . . . because there are just so many thoughts that are broken in time, one chunk there and one chunk here." These difficulties are similar to the breakdown of goal-directed serial operations during performance of the GDSA. In this regard, the construction of meaningful speech requires that words and phrases be hierarchically ordered in a goal-directed fashion (*9*). If there is a deficiency in immediate memory, the components of speech become poorly interconnected over time, and the person is apt to lose his train of thought (*10*). Hence, "loose associations" emerge, since ideas are determined primarily by very recent or current stimuli rather than by a more extended temporal context of what has already been said and what is intended. Lack of goal-directedness and loose associations were common in the speech patterns of our subjects when they were under the influence of THC.

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2. Temporal disintegration was also measured by a subjective inventory that tapped the subjects' appraisal of how well they could plan ahead without confusing elements of the past, present, and future. Changes in this inventory correlated highly with changes in GDSA overall performance ($r = .719$, $P < .0001$). Both the inventory and the GDSA are rooted in the conceptual framework presented by G. A. Miller, E. H. Galenter, and K. H. Pribram [*Plans and the Structure of Behavior* (Holt, New York, 1960)] and by K. H. Pribram and F. T. Melges [in *Handbook of Clinical Neurology*, P. J. Vinken and G. W. Bruyn, Eds. (Wiley, New York, 1969)].
3. Overall performance on GDSA is measured as the time (seconds) + *C* (1 + number of mistakes), with *C* equal to 39, which was the average number of seconds associated with errorless performance for all subjects on their base-line testing. Types of mistakes, which were scored and checked by two observers without knowledge of dose, are as follows. Forgetting instructions was the inability to repeat back out loud the required procedure before starting the task. Miscalculations were errors in arithmetic, involving the long-term memory operations of subtraction and addition. Serial immediate memory errors consisted of loss of place; no alternation between subtraction and addition; and blocking—that is, having to restart or skipping at least 20 numbers. Mistakes in reaching the goal consisted of forgetting the goal; disregarding the goal; and misadjustment to the goal, stemming from inadequate planning. Since the GDSA is a goal-directed task, failure to reach the goal exactly was weighted as three mistakes for the overall GDSA performance score.
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11. We thank Dr. H. Kraemer, C. Owen, and S. Thomasson for statistical advice and data analysis, and Mr. S. Kanter and his coworkers for biochemical work. Supported in part by PHS grants MH-03030 and MH-29163.

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Metastable Oxygen: Origin of Atmospheric Absorption near 50 Kilometers

Krueger (*1*) has reported unexpectedly large atmospheric absorption (not due to ozone) near 3000 Å from observations made above an altitude of 30 km. He concludes that the column abundance of the new absorbing species above 50 km is about 5×10^{16} cm^{-2}, assuming a fully allowed, absorbing transition with a cross section equal to $\sim 10^{-17}$ cm^2. As possible candidates for the absorber he considers the metastable oxygen $O_2(b^1\Sigma_g{}^+)$, $O_2(a^1\Delta_g)$, and vibrationally excited O_2 near $v = 11$ in the ground state $O_2(X^3\Sigma_g{}^-)$.

There is strong evidence against all three candidates, which is based on rocket measurements of the dayglow. Although a theoretical estimate, quoted by Krueger, suggested that the abundance of $O_2(^1\Sigma_g)$ might be adequate, dayglow measurements of emission from this state (*2*) show that the actual abundance above 50 km is less than 10^{13} cm^{-2}. Even with fully allowed absorption, the abundance is much too small to yield the observed effect.

As Krueger points out, measurement of the $O_2(^1\Delta_g)$ dayglow at 1.27 μ (*3*) shows that the abundance of this excited state is compatible with a 10^{-17} cm^2 cross section. But if absorption by $O_2(^1\Delta_g)$ were as strong as this, then certain unacceptable consequences arise. The absorption appears between 2900 and 3100 Å and includes the threshold wavelength for dissociation, 3037 Å; it is thus energetically possible for the process to destroy $O_2(^1\Delta_g)$. Where dissociation does not occur, the process will lead to fluorescent scattering and a "dayglow" whose intensity may be estimated. The incident solar flux over a band 100 Å wide at 3000 Å is about 10^{15} photons cm^{-2} sec^{-1} (*4*), and an absorption coefficient of 10^{-17} cm^2 implies a scattering of 10^{-2} photon molecule^{-1} sec^{-1}. This probability of scattering is about 30 times larger than the probability of emission (at 1.27 μ) from $O_2(^1\Delta_g)$ (*5*). It follows from the dayglow abundance (*3*) that the total scattered intensity above 50 km would be about 600 megarayleighs; above 80 km the intensity would be still about 30 megarayleighs. As an extreme case we assume that the fluorescent scattering is uniformly distributed over the visible region; the sky brightness at 50 km would then be about 200 kr/Å and at 80 km it would be 10 kr/Å. In both cases the predicted intensity is roughly 100 times larger than that expected for Rayleigh scattering above the same altitude. Rocket measurements (*6*) at several wavelengths between 4000 and 7000 Å make it clear that above 70 km the sky intensity is in fact close to the Rayleigh value. If the scattering were confined to the same wavelength region as the absorption, the sky intensity at 3000 Å would be as strong at 50 km as it is at ground level. The intensity at 80 km would be orders of magnitude larger than that measured by Barth (*7*) in this wavelength region.

Scattering would have to be important for all wavelengths in the absorption band which do not lead to dissociation. If the scattering process reforms $O_2(^1\Delta_g)$ it is reasonable to expect scattered wavelengths somewhere near 3000 Å or, possibly, at somewhat longer wavelength in the visible or near infrared. Since the scattered intensity appears to be unacceptably high we may consider the alternative process which destroys $O_2(^1\Delta_g)$. The foregoing computation shows that, if destruction were the sole outcome of the absorption process, the effective lifetime of $O_2(^1\Delta_g)$ would be 30 times smaller than the radiative lifetime. A discussion of the daytime $O_2(^1\Delta_g)$ measurements (*3*) indicates that no production mechanism yet conceived is capable of accounting for the measured abundance of $O_2(^1\Delta_g)$ above 70 km if the state is destroyed more rapidly than the rate set by spontaneous emission at 1.27 μ. It is therefore difficult to avoid the conclusion that the species responsible for the absorption at 3000 Å cannot be $O_2(^1\Delta_g)$; neither fluorescent scattering by the state nor significant destruction of it can be tolerated.

Rather similar arguments may be advanced against Krueger's third candidate. The argument against fluorescent scattering may be carried over if, as Krueger suggests, the absorption is in the Schumann-Runge band system, from O_2 molecules near $v = 11$ in the ground state. There is also a major problem in accounting for the required abundance of such highly vibrating O_2. Such molecules will doubtless be destroyed by collision-induced relaxation at a much faster rate than is $O_2(^1\Delta_g)$ which can withstand about 10^9 collisions with "air" molecules (*8*). It would therefore be necessary that a process exist which can create vibrationally excited O_2 at a rate that is orders of magnitude greater than the rate for $O_2(^1\Delta_g)$. The energy problems associated with such a requirement appear insurmountable when one considers that the creation of $O_2(^1\Delta_g)$ itself requires efficient use of the entire solar spectrum between 2000 and 3000 Å.

The measurements reported by Krueger resemble some described by Tohmatsu (*9*) in which rocket photometer results at 3000 Å implied a larger ozone abundance above 50 km than those from a photometer at 2500 Å; this appears to weaken the possibility of an instrumental error since the photometric methods were quite different. In both sets of measurements, the solar zenith angle was small and the optical depth of the excess absorption near 3000 Å was less than 1. Unless the absorber disappears before sunset one would expect that measurements of the solar spectrum at large zenith angle should reveal almost complete extinction in the vicinity of 3000 Å even up to 60 km. There appears to be no such anomalous absorption present in the early solar spectra taken near sunset at such heights (*10*) many years ago. It would be of interest to have additional measurements of the phenomenon at large solar zenith angle.

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Bladder Tumorigenesis

Bladder tumors have been induced in rats by a number of industrial chemicals and by cyclamate. There is, however, one factor implicated in bladder tumorigenesis in rats which is frequently ignored. We refer to the possible parasitism among the rats used in these studies by the bladder nematode *Trichosomoides crassicauda* and to the possible potentiative effect of this parasite on the chemical induction of bladder tumors.

The adult *T. crassicauda* resides in the bladder, renal pelvis, and ureter, usually stimulating little tissue reaction. Parasitism with this nematode is endemic in wild rats (*1*). In 1964 Chapman (*2*) examined rats from 19 commercial American sources and found that approximately one-half of the groups contained at least one parasitized animal. The incidence of infection in these groups varied from 4 to 91 percent (11 to 24 rats per group). Although a few cesarian-delivered rats were free of parasites, one cannot dismiss the possibility of transplacental infection or subsequent postnatal infection. Dissemination of infection occurs readily since embryo-