median intake of the standard S + G solution was 251 ml. An analysis (Friedmann) (*5*) over these three groups yielded a significant F (< .01), and a subsequent test revealed that the test with the standard S + G solution and the test with 0.25 percent saccharin and 6 percent glucose were not different ($P > .05$). These results indicate that, if the rat is given 6 percent glucose in one bottle and 0.25 percent saccharin in a second bottle, the characteristic S + G synergy is apparent.

To be sure that the sequence of presentation of the solutions did not affect the outcome, eight more rats were tested. Half received the 6 percent glucose and 0.25 percent saccharin solutions first; the other half received the 3 percent glucose and 0.125 percent saccharin first. The results indicated that order of presentation had no effect on the results, and the statistical analysis of the results for these eight rats was identical to that of the original 20 subjects. Since the rats drank approximately equal quantities of the 6 percent glucose and the 0.25 percent saccharin solutions, it appears that they were mixing the two solutions and essentially receiving the standard S + G solution.

In our final experiment we attempted to observe the rat's pattern of drinking the 6 percent glucose and the 0.25 percent saccharin to see if there was any tendency for the rat to show an alternation between the glucose and saccharin during a drinking bout. Special individual home cages were constructed so that each rat had access to either one of two sipper tubes. These tubes were inserted partially in slots 3.5 cm long by 0.95 cm wide. In either slot the rat's tongue interrupted an infrared beam with each lick. The infrared receptors, in turn, through electronic circuits, operated high-speed relays which controlled the pens of two event recorders. The paper drive of one recorder operated at a speed of 7.6 cm/min and the other at 10.2 cm/sec.

More than 20 rats have been tested, and half of them demonstrate a pattern of drinking never before reported in the literature. During a drinking bout these rats alternate between the two sipper tubes, essentially mixing the standard S + G solution in the mouth. Figure 1 shows a typical drinking bout recorded at 7.6 cm/min which lasted 3 minutes 48 seconds. During this bout the rat switched from one bottle to the other 35 times. Data from the higher-speed recorder made it possible to count individual licks during these bouts. Seldom did a rat lick one tube more than 20 times (or about 4 seconds) before changing to the other tube. There is no consistent difference in lick rate on the glucose or the saccharin tubes. The rat shown in Fig. 1 consumed a slightly larger volume of glucose than saccharin (although the amounts of each solution consumed varied with each rat tested).

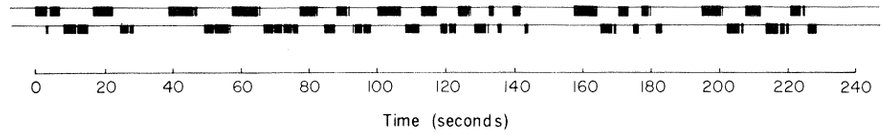

Fig. 1. Reproduction of an event recording of a drinking bout showing a pattern of rapid alternation by the rat between 0.25 percent saccharin solution (top) and 6 percent glucose solution (bottom).

Alternation appears to be a robust phenomenon. Rats which had demonstrated the alternating behavior with glucose and saccharin solutions and were then given a choice of standard S + G solution versus water in two tubes failed to exhibit the alternating pattern with the new two-bottle system. However, when the saccharin and glucose solutions were reintroduced, the alternating behavior reappeared. Since rats which develop the alternating pattern consistently drink more glucose and saccharin than nonalternators, it seems that these animals are in essence "mixing a cocktail" on their tongues.

Valenstein *et al.* have emphasized the value of the standard S + G solution for experimental purposes requiring consumption of large volumes of fluid. Our own studies have shown its additional utility in view of the fact that the animals show a marked reduction of neophobia. The alternating behavior manifests a potential for even broader application in taste research and raises many questions relative to the observation of consumption of other taste substances.

In the design of two-bottle preference tests the results presented here point to the necessity of examining not only the consumption but also the patterns of drinking to ascertain that the mixing of solutions has not contributed to the amount consumed. Whereas it has been found that some animals develop a clear pattern of alternation as early in their exposure to saccharin and glucose as day 1, some take several days to acquire the pattern and some (less than 50 percent) fail to exhibit this behavior after 10 days of exposure. We hypothesize that a forced-learning type of design might facilitate acquisition of this unusual behavior. Clearly, the alternation phenomenon presents a model of an expressly unique pattern of drinking behavior.

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28 August 1975; revised 20 October 1975

Selective Brain Dopamine Depletion in Developing Rats: An Experimental Model of Minimal Brain Dysfunction

Abstract. *Administration of 6-hydroxydopamine to neonatal rats produces a rapid and profound depletion of brain dopamine. Total activity of treated animals is significantly greater than that of controls between 12 and 22 days of age, but then declines, an activity pattern similar to that seen in affected children. This suggests a functional deficiency of brain dopamine in the pathogenesis of minimal brain dysfunction.*

Minimal brain dysfunction (MBD) in children is one of the most common and difficult problems in present-day pediatrics. Conservative estimates indicate that it affects 5 to 10 percent of the elementary school population and is a major cause of school learning and behavior problems. Clincially the disorder is characterized by a constant involuntary hyperactivity that completely surpasses normal, short attention span, impulsivity, and a variety of cognitive and perceptual problems (*1*). We have produced an experimental model in developing rats that is strikingly similar to the clinical syndrome of MBD. The model is effected by the intracisternal administration of 6-hydroxydopamine (6-OHDA) to neonatal rat pups, resulting in a rapid and profound depletion of brain dopamine.

We demonstrate that rat pups treated with 6-OHDA as neonates are significantly more active than their littermate controls during the period of behavioral arousal that occurs between 2 and 3 weeks of

Table 1. Brain catecholamine concentrations. Dopamine and norepinephrine values are mean nanograms per gram (wet weight) of whole brain, ± standard error of the mean. The ratio of dopamine in 6-OHDA–treated rats to that in controls is 41.0 percent and the difference in the values is significant ($P < .001$). The ratio of norepinephrine in 6-OHDA–treated rats to that in controls is 91.7 percent and the difference is not significant.

Rats	No. of rats	Dopamine* (ng/g)	Norepinephrine† (ng/g)
Control‡	27	693.6 ± 15.5	314.3 ± 11.6
6-OHDA–treated‡	31	284.4 ± 34.1	288.2 ± 9.0
Maternal rats	11	722 ± 23.8	370.7 ± 16.9

*Dopamine corrected for 80 percent recovery. †Norepinephrine corrected for 72 percent recovery. ‡Rat pups were killed at 35 days of age.

age. However, as the rat pup approaches maturity (4 weeks of age) the hyperactivity initially observed in the 6-OHDA–treated animals is no longer apparent. This finding appears to correspond to that found in children with the clinical syndrome of MBD. In these affected youngsters hyperactivity is pronounced until 10 to 12 years of age but then abates, similar to the pattern seen in the experimental model of hyperactivity in rats. Moreover, in children with MBD, although the hyperactivity does indeed disappear, associated cognitive, perceptual, and emotional difficulties persist (*2*). Similarly, our rats depleted of brain catecholamines as neonates also have deficits in learning as adults, again paralleling the clinical syndrome of MBD seen in children.

Preferential depletion of brain dopamine was accomplished by the method described by Breese and associates (*3*). Five-day-old rat pups were pretreated with desmethylimipramine (20 mg/kg, intraperitoneally) followed 1 hour later by the intracisternal administration of 25 μl of 6-OHDA (100 μg per 25 μl, calculated as free base). Littermate controls received 0.9 percent saline intraperitoneally followed 1 hour later by intracisternal administration of vehicle solution (0.9 percent saline plus 0.4 mg of L-ascorbic acid per milliliter). Behavior was evaluated with a time sample measure of general activity modified after methods described by Gray and by Russell (*4*). Transparent Plexiglas rat cages containing a water bottle and food were arranged in three rows on a steel cage rack, allowing the simultaneous observation of an entire litter at each session. Observations were made on nine occasions during the first month of postnatal life, always between 1300 and 1600 hours in order to minimize the variability due to circadian periodicity. Rat pups were randomized and placed one to a cage, and observations were begun immediately. At the beginning of every minute the cages were scanned and a written note was made of the behavior of each rat at that particular instant, within one of the following exhaustive and mutually exclusive categories—sleeping: lying motionless, body resting on floor, head often tucked under body; inactive: standing or sitting motionless; ambulating: walking or running about cage; climbing: forepaws climbing on side of cage or about water bottle; rearing: both forepaws clear of floor; eating: gnawing at food in hopper or at pieces on cage floor or holding food in forepaws; drinking: mouth contact with nozzle of water bottle; sniffing: sniffing at any part of cage or in the air; grooming: any self-washing or licking movement; scratching: scratching body with hind legs.

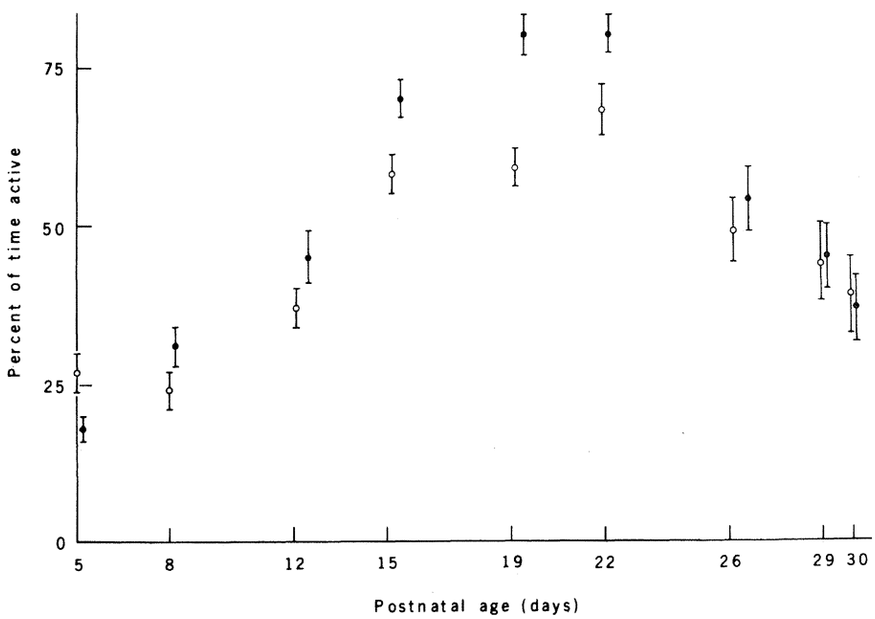

Fig. 1. Mean total activity in normal (control) rat pups (open circles) and rat pups treated with 6-OHDA during the first month of postnatal life (closed circles). Bars represent ± standard error of the mean.

The observations were always performed by the same observer, "blind" to which rat pups were treated or controls. Observations continued for 1 hour, and thus 60 recordings were made for each rat pup at each experimental session. Activity was determined on eight litters composed of 27 control and 31 6-OHDA–treated rat pups, with approximately equal numbers of males and females in each group.

Rats were killed by decapitation at 0900 to 1100 hours at 35 days of age, and their brains were rapidly removed and frozen on Dry Ice within 1 minute after death. Frozen brains were stored at −80°C, and biochemical determinations were performed within 2 weeks after killing. Brain dopamine and norepinephrine were extracted with trichloroacetic acid, isolated on alumina columns, and analyzed fluorometrically with procedures described by Roth and Stone and by Boadle-Biber *et al.* (*5*).

Activity in 6-OHDA–treated rat pups and littermate controls during the first month of postnatal life was determined by recording the frequency of each behavioral parameter, and differences were analyzed by *t*-test. Each parameter of activity was calculated separately; in addition, various parameters were combined to determine total inactivity (sleeping and inactivity); total very active behavior (ambulating, climbing, eating, rearing, drinking); total slight activity (sniffing, grooming, scratching); and total activity (ambulating, climbing, eating, rearing, drinking, sniffing, grooming, scratching).

Avoidance learning was investigated in a single unit enclosed shuttle box consisting of two compartments each 15 cm high by 20 cm long by 18 cm deep and separated by a 6-cm-high hurdle. Rats were allowed 5 seconds to avoid a 2.5-ma shock by jumping over the hurdle into the "safe" compartment, and were tested until they avoided the shock for five consecutive trials. The numbers of trials necessary to meet this criterion in controls and 6-OHDA–treated rats were compared and analyzed by *t*-test. Four litters of rat pups (16 controls and 20 6-OHDA–treated) were investigated.

Our findings confirm previous observations that during the first month of postnatal life the normal rat pup undergoes a distinct pattern of activity which Campbell and associates (*6*) have termed the development of behavioral arousal. For the first week of life the normal rat pup moves very little. However, beginning at 12 days and continuing throughout the next 10 days his total activity increases dramatically, so that at 22 days it is two to three times the activity at day 8, comprising 68 percent of his time. By 26 days, total activity has declined again to the level it was prior to the increase (Fig. 1).

Rat pups treated with 6-OHDA as neonates also demonstrate this pattern of activity as they mature. However, 6-OHDA–treated rat pups appear to develop increased activity earlier and to a significantly greater degree than do their littermate controls. Initially, treated rat pups are less active than controls ($P < .05$). We believe that this reflects the acute effects of the treatment, since total activity at 5 days was measured just 2 hours after the intracisternal injection of 6-OHDA. By 15 days total activity of treated rat pups is significantly greater than that of controls ($P < .01$), and these differences continue at 19 days ($P < .001$) and 22 days ($P < .05$). At 26 days, total activity in treated rats has decreased to levels comparable to those of controls and remains similar to those of controls thereafter.

Shuttle box avoidance at 27 days of age required 7.6 ± 1.2 trials (mean ± standard error of the mean) in controls compared to 16.5 ± 3.2 trials in 6-OHDA–treated animals ($t = 2.35$, $P < .05$). Similar significant learning difficulties in dopamine-depleted rats have been reported previously by Howard *et al.* and Nyakas *et al.* (*7*).

Brain dopamine concentrations in the 6-OHDA–treated rat pups were significantly less than those in their littermates, averaging 41.0 percent of controls of a comparable age, while norepinephrine was reduced by only 8.3 percent (Table 1). Furthermore, in nine 6-OHDA–treated rat pups brain dopamine was depleted to values no lower than 45 percent of controls, and if these "high dopamine" animals are omitted from the calculations the remainder were reduced to 25.6 percent of control values.

Evaluation of the behavior in these "high dopamine" rat pups indicated that if brain dopamine were reduced below 55 percent (two rats), behavior was comparable to that in the usual "low dopamine" 6-OHDA–treated rat pups. If brain dopamine were greater than 55 percent, behavior was similar to that in controls (seven animals). This suggests that brain dopamine concentrations of 50 to 55 percent of controls are a threshold range for the production of hyperactivity. Rats depleted of greater amounts of dopamine will behave as hyperactive rats, while lesser depletion results in a behavior indistinguishable from that of controls.

Evidence from several lines of investigation suggests that the increase in activity seen between 12 and 22 days in the normal rat pup is related to the simultaneous development of catecholaminergic mechanisms. Both dopamine and norepinephrine are present in the brains of newborn rats in concentrations of 20 to 30 percent of those in adult animals. Dopamine concentration attains adult values by 50 days of age and norepinephrine at 40 days of age, with the greatest increase in the brain concentration of monoamines occurring between 7 and 18 days of age. Tyrosine hydroxylase, dopa-decarboxylase, and dopamine β-hydroxylase, the enzymes involved in the synthesis of the catecholamines, increase in a parallel fashion. Similarly, the enzymes concerned with the metabolism of the catecholamines, monoamine oxidase, and catechol-*O*-methyltransferase also increase in a fashion similar to the amines and their synthetic enzymes (*8*). The administration of amphetamine or L-3,4-dihydroxyphenylalanine (agents known to increase brain catecholamines) to developing rats results in an increase in activity. Conversely, administration of α-methyl-*p*-tyrosine (AMPT), the competitive inhibitor of tyrosine hydroxylase, decreases the spontaneous motor activity of rats from 15 to 25 days, and reserpine, an agent which interferes with the storage of catecholamines, also causes a decrease in spontaneous motor activity between 15 and 25 days of age. Normal motor activity could be reinstated in the 15- to 25-day-old rats by administration of L-3,4-dihydroxyphenylalanine, immediately reversing the depressive effects of either AMPT or reserpine (*9*).

In contrast to normal rat pups, 6-OHDA–treated, 12- to 22-day-old rats exhibit significantly increased activity. In our view this apparent paradoxical response to brain dopamine depletion may be explained by considering dopamine to act as a modulator of excitatory noradrenergic activity. Thus, in normal rats, activity is restrained by adequate concentrations of brain dopamine. Reduction of brain dopamine, as produced in our experimental model, removes the constraints and allows activity to occur unchecked.

Such a mechanism would explain the apparent paradoxical response to amphetamine in children with MBD. In normal children and adults administration of amphetamine results in an increase in motor activity. However, it has been known for many years that amphetamine reduces hyperactivity when given to children with MBD. It is well established that the central actions of amphetamine are mediated via central catecholaminergic mechanisms. This agent is known to increase the amount of dopamine and norepinephrine at the synaptic cleft by a variety of mechanisms (*10*) (decreased neuronal uptake, increased granular release of catecholamines, decreased monoamine oxidase activity). Recent evidence suggests that the postsynaptic actions of dopamine are mediated via a mechanism dependent on cyclic adenosine monophosphate (*11*). Furthermore, the adenylate cyclase response to dopamine is significantly increased after denervation of striatal tissue, a neurochemical correlate of the phenomenon of the enhanced behavioral response elicited by dopamine-stimulating agents after denervation and termed supersensitivity (disuse supersensitivity) (*12*). Thus the presumed deficit of dopamine in MBD (*13*) may result in a denervated supersensitive postsynaptic dopaminergic neuron. Administration of amphetamine results in an increase of both dopamine and norepinephrine, but the supersensitive postsynaptic dopamine receptor responds to a greater extent than does the undamaged noradrenergic receptor, and the overall effect of such treatment is decreased activity. That brain dopamine concentration is indeed reduced in the syndrome of MBD is suggested by the recent findings of a significantly reduced turnover of homovanillic acid, the major metabolite of dopamine, in the cerebrospinal fluid of children with MBD (*14*).

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15. Supported by research grants NS12384 and a grant from the Charles H. Hood Foundation (to B.A.S.).

12 September 1975

Sleep During Transcendental Meditation

Abstract. *Five experienced practitioners of transcendental meditation spent appreciable parts of meditation sessions in sleep stages 2, 3, and 4. Time spent in each sleep stage varied both between sessions for a given subject and between subjects. In addition, we compare electroencephalogram records made during meditation with those made during naps taken at the same time of day. The range of states observed during meditation does not support the view that meditation produces a single, unique state of consciousness.*

In 1970, Wallace reported several physiological changes observed during transcendental meditation (TM) (*1*). His results were replicated and extended by Wallace, Benson, and Wilson (*2*) and they were subsequently made available to a wider audience (*3*). They found, in meditating subjects, reduced oxygen consumption, increased skin resistance, increased alpha activity in the electroencephalogram (EEG), decreased heart rate, and decreased blood lactate. Although many of these changes take place in ordinary relaxed wakefulness and in sleep, Wallace and his co-workers postulated that, during most of the meditation period, experienced practitioners of TM enter a single, unique state of consciousness, a "wakeful hypometabolic state," that differs from ordinary relaxed or sleep states.

The Stanford Research Institute estimates that, from a few hundred in 1965, the number of practitioners of TM has increased to more than 240,000 as of June 1973. Estimates from the TM organization indicate that this number now exceeds 900,000 (*4*). The findings of Wallace and his co-workers are often cited to prospective meditators and may have played an important role in producing this increase.

We have found that meditators spend considerable time in sleep stages 2, 3, and 4 during meditation; their subjective reports of sleep confirm our analysis of the EEG records. Further, our data suggest that the meditation period is not spent in a single, unique, wakeful, hypometabolic state.

The five subjects we observed had at least 2.5 years of experience with TM, and four of them were teachers of the technique. All were male Caucasians between the ages of 20 and 30, accustomed to meditating for 40-minute periods twice each day, and not in the habit of napping. Subjects reported, on the average, 7.8 hours of sleep per night.

Psychophysiological measures were made on each subject during ten sessions, each of which lasted 40 minutes. During five of these sessions, the subjects were asked to meditate in their accustomed sitting position, and in the other five sessions, they were asked to nap lying down on a bed. The first nap and the first meditation were scheduled on the first observation day. The data collected on this day are not included for analysis here because initial unfamiliarity with the laboratory situation produces atypical sleeping patterns (*5*). On eight subsequent days, subjects were asked either to meditate or to nap. These sessions were all conducted in the afternoon within 2 hours of the same time each day. The order in which the two types of sessions were scheduled followed an irregular pattern, and subjects were not told whether they would be asked to meditate or to nap on a particular day until they arrived in the laboratory. If a subject reported that his previous night's sleep was more than 30 minutes shorter than normal, he did not take part on that day. Subjects were asked not to consume food, coffee, or tea for at least 2 hours before each session.

At the beginning of each session, electrodes were applied so that occipital, central, and frontal EEG responses, eye movements, submental (below the chin) muscle potentials, and skin resistance level could be measured (*6*). The subject then moved to the room where he was to meditate or nap. A 45-db white noise partially masked any disturbance from the adjoining apparatus room (*7*). The room in which the subject sat during meditation was dimly illuminated, but the room was dark when the subject lay down to nap. Once the recording was proceeding smoothly, the subject was asked to relax for 5 minutes with his eyes closed, and then a signal was given to begin meditation or napping. After 40 minutes, an identical signal required the subject to stop meditating or napping and to relax with his eyes closed for an additional 5 minutes before leaving the recording room. At the end of the session, the subject filled out a questionnaire on his subjective impressions of what had transpired and stated whether he had slept or become drowsy during the meditation or nap.

The most striking feature of our data is that meditators spent appreciable amounts of time in EEG sleep stages 2, 3, and 4 while they were meditating (Fig. 1). Averaged over meditation sessions, we found that 39 percent of the time was spent in wakefulness (stage W), 19 percent in stage 1, 23 percent in stage 2, and 17 percent in stages 3 or 4. More than a quarter of the meditation time was spent in stages 2, 3, or 4 in 13 out of the 20 meditation sessions (Table 1). It is customary to identify stages 2, 3, and 4 as sleep and stage 1 as drowsiness (*8*); according to these conventional designations, our subjects were asleep dur-

Table 1. Percentage of time spent in stages 2, 3, or 4 during each session.

Subject	Meditation session				Nap session			
	1	2	3	4	1	2	3	4
1	51	90	59	78	37	41	59	62
2	0	0	0	26	78	92	79	58
3	49	0	0	78	86	31	83	89
4	0	90	59	74	18	38	95	88
5	37	0	86	31	95	95	93	78

Table 2. Percentage of time spent in each stage, averaged over sessions.*

Subject	Meditation				Nap			
	W	1	2	3, 4	W	1	2	3, 4
1	19	12	42	27	32	17	40	10
2	44	46	6	0	7	14	62	14
3	53	15	16	15	15	12	31	41
4	37	6	28	27	31	8	51	9
5	43	17	23	15	1	7	54	36

*These percentages do not sum to 100 because some epochs were scored as movement time.