bers of pycnotic nuclei represent death of cells in some other mitotic stage. Thus in the vast majority of mitotic cells in these roots the current divisions were completed. Entry into another mitosis was then blocked. It is possible that the long delay in response is in part a result of slow penetration of the drug into the cells. But elongation and prevascular differentiation stop long before the mitotic index drops, so that there is a period of at least 24 hours during which actinomycin suppresses differentiation without holding up the progress of cells already in mitosis. Whether the interphase block is during the period or periods of DNA synthesis or in one of the two gaps (G_1, G_2) will have to be determined by a different sort of experiment.

The recovery of growth after removal of actinomycin is an argument against a nonspecific injury effect, unrelated to macromolecule synthesis; but the recovery is in itself somewhat surprising, in view of the strong binding of antinomycin D to DNA (*2*). A similar recovery has, however, been observed by Goldstein and coworkers (*12*), who studied actinomycin effects on HeLa cells.

That some messenger RNA and some new protein must be made for each mitosis in root meristem cells (*13*) is supported by our data. Once prophase is underway, RNA synthesis probably stops normally; presumably protein synthesis can stop without affecting a division already past prophase. Three classes of "division" protein are of importance in cells of this type, possibly requiring renewal in each cycle: the structural proteins of the mitotic spindle, enzymes required for mitotic movements and for breakdown of the nuclear membrane, and the histones of reconstituting nuclei. The observed failure of tissue to become differentiated, presumably soon after the addition of actinomycin, may reflect dependence upon messengers whose half-lives are shorter than one cell cycle (*14*).

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24 December 1962

Eye Fixation Aspect of Attention to Visual Stimuli in Infant Chimpanzees

Abstract. *Infant chimpanzees look at a visual stimulus for a regularly decreasing proportion of the time after presentation of the stimulus. Individual differences and presentation of a novel object affect the general level of fixation but do not significantly influence the slope of the curve showing the decline of fixation with time.*

Classical theories of attention (*1*) and more recent studies of the orienting reflex (*2*) have been concerned with moment-to-moment adjustments made by an organism for the efficient reception of information. An especially significant aspect of orientation to a visual stimulus involves the turning of the eyes toward potentially important stimuli.

While autonomic and electroencephalographic changes are manifestations of a general mobilization of the organism to receive stimuli, it is the direction of gaze that can often indicate which stimulus the organism is responding to at any moment. Thus, patterns of eye movement are used to analyze visual search of a complex stimulus field (*3*) and to assess which aspects of such a field are discriminated (*4*) and preferred (*5*).

By considering the shift of eye movements to a new stimulus as one component of the orienting reflex, as Sokolov (*2*) has done, it is possible to regard a change in eye fixation as a phasic psychophysiological response to the onset of a simple stimulus. The purpose of the studies we report was to consider eye fixations in this way, to examine the course of eye fixations after presentation of a visual stimulus, and to analyze certain factors that might affect the resulting curve.

Five infant chimpanzees (aged 7 to 22 months) were tested in two experiments. Testing took place in a 31-by-18-by-29-inch box from which light was excluded. The animal was strapped into a chair inside the box facing a 20¼-by-16¾-inch panel, 14½ inches away. In the panel, at the subject's eye level, were two 3¼-by-3-inch windows, 10¾ inches apart. Each window could be illuminated by a 7-watt bulb masked by frosted Plexiglas. There was a 5/16-inch peephole halfway between the two windows through which the subject could be observed from outside the box.

The general procedure involved turning on one of the lights and observing the animal's eye fixations toward that light during a number of successive 5-second intervals. An eye fixation toward the stimulus was counted if the stimulus light was reflected in the center of the pupil, and an event key connected to a pen writer was depressed for the duration of each fixation. Scoring the records involved determination of the duration of eye fixation within each 5-second interval. The median correlation between observers on this measure was .84 (range, .66 to .94).

In a first experiment it was found that duration of eye fixations toward the light was maximum immediately after the light was turned on and that there was a regular decrease in fixation of the stimulus in the following 25 seconds. There was typically at least one fixation during the first 5 seconds. It was followed by looking away and then by repeated fixations later in the trial.

In the second experiment, three conditions were presented to determine whether a retinal adaptation process alone could account for this short-term decrement in eye fixation. In this experiment a 3¾-by-3¾-by-3¼-inch lighted white box into which the subject could see was placed behind each window. A hinged door on the top of each box permitted the experimenter to insert objects into it. A guillotine door between the window and the box

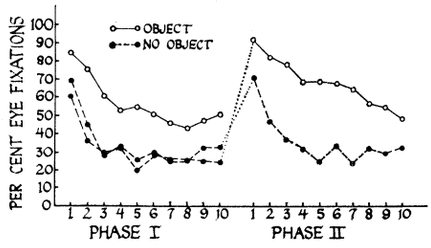

Fig. 1. Duration of eye fixations in successive 5-second intervals.

could be lowered to prevent the subject from seeing into the box. There were three experimental conditions. In the first of these conditions the door was raised and the subject's eye fixations on the empty box were recorded for 1 minute (phase 1). The door was then lowered for 2 seconds and the box remained empty. The door was again raised and the eye fixations to the stimulus were observed for another minute (phase 2). In a second condition, phase 1 was the same as in condition 1, but now when the door was lowered for 2 seconds a novel object (for example, a piece of a broken toy) was placed in the box. As in condition 1, the eye fixations to the stimulus window were observed for another minute. The third condition was restricted to phase 1 only, with an object in the stimulus box.

Three trials were run for each subject in each condition on each of 3 days. Trials were separated by 1-minute periods in which the light in the box to be presented on the next trial was turned on behind the closed guillotine door. Since light leaked around the sides of the guillotine door, the subject could see which window would be presented on the following trial. Position of the window and order of the experimental conditions were randomized.

Curves averaged over subjects are presented in Fig. 1. Analysis of variance was performed for the effects of ten 5-second intervals, the five curves, and subjects. The decrement in eye fixations over 5-second intervals was again observed ($p < .001$). Neither of the other variables interacted with intervals. There was a significant difference among the five curves ($p < .001$), with all subjects showing the highest scores in the Object conditions. The curve for the Object condition of phase 2 was significantly higher than each of the No Object curves. None of the other comparisons between curves was significant. The difference between subjects was significant ($p < .001$).

The results indicate that when a single visual stimulus is presented, the amount of eye fixation on the stimulus is maximum immediately after its presentation, and then it decreases. The orienting response of the eyes to the onset of a stimulus is thus not merely a single shift of the gaze but a series of fixations whose occurrence is graded over a short period of time. This function (*6*), together with other psychophysiological responses (for example, galvanic skin response, alpha blocking), may reflect a general phasic mobilization of the organism for the identification of a stimulus. This process is initiated by the presentation of the stimulus and probably involves arousal mechanisms in the reticular formation of the midbrain. Since the light remains on, a short-term habituation of the eye fixations to the light may also be involved. Retinal adaptation is probably not an important factor. The shape of the function is not significantly affected by individual differences or by characteristics of the stimulus, such as novelty. However, these variables appear to affect the total duration of fixation on the stimulus which is reflected in a variation of the level of the curve (*7*).

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5 December 1962

N-Methylmetanephrine: Excretion by Juvenile Psychotics

Abstract. *Three out of 18 psychotic children excreted* N-*methylmetanephrine, a metabolite of* N-*methylepinephrine. It is not clear whether this tertiary amine plays a part in causing some forms of psychosis or is merely a secondary result of mental dysfunction. Urinary excretion of bufotenin and of 3,4-dimethoxyphenylethylamine, each of which has been reported elevated in some adult schizophrenics, was not unusual in these children.*

In recent years there has been an intensive search for biochemical causes of mental illness, stimulated in part by an awareness that an increasing number of diseases involving enzyme deficiency are associated with mental deficiency. The possibility that some forms of mental illness may be due to abnormalities in amine metabolism is an attractive one. Biogenic amines are believed to participate in synaptic functions in the central nervous system. Three tertiary amines, bufotenin, *N*,*N*-dimethyltryptamine, and psilocin, as well as mescaline, produce psychotomimetic effects in man (*1*). Recent reports state that some schizophrenic patients, in contrast to normal subjects, excrete bufotenin (*2*), or 3,4-dimethoxyphenylethylamine (*3*) in their urine. The latter amine, and other *p*-methoxylated phenylethylamines, produce striking neurological effects in animals (*4*). Mammalian tissues contain an enzyme (*5*) which converts, by *N*-methylation, serotonin and tryptamine to their psychotomimetic metabolites, bufotenin and *N*,*N*-dimethyltryptamine. Finally, a recent study by Pollin *et al.* (*6*) has shown that administration of tryptophan and methionine to schizophrenics maintained on a monoamine oxidase inhibitor in some cases provokes an exacerbation of mental symptoms. Tryptophan and methionine, since the latter might act as a methyl donor, could serve as precursors to some of the psychotomimetic amines listed above.

The present report is a preliminary survey of the amines present in the urine of 18 juvenile psychotics. The likelihood of success in detecting biochemical abnormalities causally related to mental illness seemed greater in children than in adults, both since the early onset of mental illness would favor a genetic determination and since psychotic children are less likely than psychotic adults to show the secondary