Some appreciation of the quality of the response pattern can be gained from inspection of the quantitative data. At the intermediate intensities the average duration of stimulation for all sites was 4.3 seconds, and the average reset time was 3.2 seconds. Thus, during the 15-minute tests animals were oscillating back and forth between the onset and offset levers. At the higher stimulus intensities this oscillation rate tended to be considerably faster (average duration, 2.8 seconds; average reset time, 2.6 seconds).

Some additional qualitative observations were made. For a few animals the reset time increased at the highest intensities. In some of these cases it was noted that a motoric response (for example, jumping), associated with the offset of the stimulus, appeared to interfere with the animals' ability to turn the stimulus on again. With ventral hippocampal electrode placements there was a significantly longer reset time. These animals tended to press the offset lever repeatedly before turning the stimulus on again. We could not determine with certainty whether some effects persisted after termination of the stimulus, so that experience of an abrupt offset was lacking; or whether after hippocampal stimulation the animals were confused and did not know which lever to press.

It was suggested (*2*) that animals receiving hypothalamic stimulation terminate the stimulus earlier at higher intensities as a consequence of more rapid activation of an aversive system. In contrast, it was also hypothesized that animals with septal electrodes may not exhibit this tendency because stimulation in this area does not activate an aversive system; in such animals the stimulus would be terminated only as a result of a loss of its effectiveness resulting from adaptation. It would be expected that adaptation would occur more slowly at higher intensities. The data from the present experiment directly oppose this hypothesis. The differences in duration and reset times attributable to these two neural areas seem to indicate that animals with septal electrodes terminate the stimulus faster at higher intensities than do animals with electrodes located in the hypothalamus. For the eight hypothalamic sites studied, the average duration and reset times were 7.1 and 1.6 seconds (intermediate intensity) and 3.0 and 1.2 seconds (highest intensity), respectively; for the seven septal placements the comparable averages were 1.5 and 2.3 seconds (intermediate intensity) and 0.9 and 1.1 seconds (highest intensity), respectively.

Several conclusions may be drawn from data which suggest that most, if not all, self-stimulating animals will repetitively turn a stimulus on and off when enabled to control duration. The position that these positive reinforcing areas have neighboring aversive areas would be difficult to maintain, however, in the absence of supporting evidence. In the past, when applied only to hypothalamic areas, this argument could be justified in view of the claims that medial hypothalamic stimulation produced ambivalent or avoidance reactions (*8*). According to another, perhaps more defensible conclusion, it is assumed that most positive systems have the potential to activate an aversive system or systems located at some unknown distance from the stimulation site. If this is true it is evident that any conclusion about the location of such an assumed aversive system(s) cannot be based solely upon the act of terminating positive brain stimulation. In fact, it would not be unreasonable to postulate that aversive consequences of prolonged stimulation may result from afferent feedback from systemic effects. Animals may terminate stimulation to obtain a respite from shifts in heart rate, body temperature, respiratory rhythm, and numerous other changes in bodily states known to be produced by central stimulation.

From another vantage point it may be asked whether termination of a stimulus should be considered sufficient evidence of the existence of an aversive system. It has been reported (*9*) that animals repeatedly turn on and off any appropriate stimulus placed under their control. With respect to reinforcing brain stimulation, more recent data raise some questions about interpretations in which an activation of an aversive system is assumed. From the present experiment it is evident that, although animals terminate the stimulus sooner at high intensities, there is no resistance to turning the stimulus on again. In fact, the stimulus is reset faster at the higher intensities. We have also fixed stimulation trains many times longer than the preferred durations, but even under these conditions there was no hesitancy in turning the stimulus on again (*10*). Similarly, it has been reported that when stimulus trains longer than the preferred duration are offered as a reward, animals respond at higher rates on a variable-interval reinforcement schedule (*11*), and at higher ratios on a progressive ratio test (*12*). This strong evidence that stimulus durations longer than those determined by the animals themselves have high reinforcement value raises a critical question about the nature of the presumed aversion which results from prolonged positive stimulation.

ELLIOT S. VALENSTEIN
THELMA VALENSTEIN
Fels Research Institute,
Yellow Springs, Ohio

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Gravitational Stress: Changes in Cortical Excitability

Abstract. *Evoked responses, recorded from the visual cortex, are enhanced during positive (head to tail) acceleration, and during cerebral hypotension induced by hemorrhage. The phenomenon observed during positive acceleration may therefore be due, at least in part, to its hypotensive effect, but the increased somatic stimulation must also be considered.*

Electrical activity, recorded from the visual cortex and evoked by photic stimulation, is enhanced during mild positive acceleration. This effect is in marked contrast to the electrocortical depression which is produced by more severe acceleration.

Five cats were prepared with permanently implanted extradural electrodes,

(*1*). The electrodes, held by drilled stainless-steel screws, were oriented to the posterolateral gyrus by reference to bony landmarks. The technique preserved the integrity of the skull, essential for these studies, but did not permit a detailed exploration of the cortex for responses of maximum amplitude. The electrode placement was confirmed post mortem.

The experiments were carried out on the Royal Air Force man-carrying centrifuge (*2*). The centrifuge consists of a horizontal rotating beam carrying at each end a gondola mounted on gimbals. When rotated the gondolas swing out and maintain the direction of the resultant centrifugal force along the longitudinal axis of the subject. The recording leads run along each centrifuge arm and ascend a central duct to the top of the building. From this duct the electrical signals are led through silver-graphite slip rings to the recording apparatus. The rate of onset, maximum acceleration, and duration of maximum acceleration are controlled independently. In the present experiments an acceleration of $3g$ was applied at the rate of $1g$/sec for periods of up to a minute.

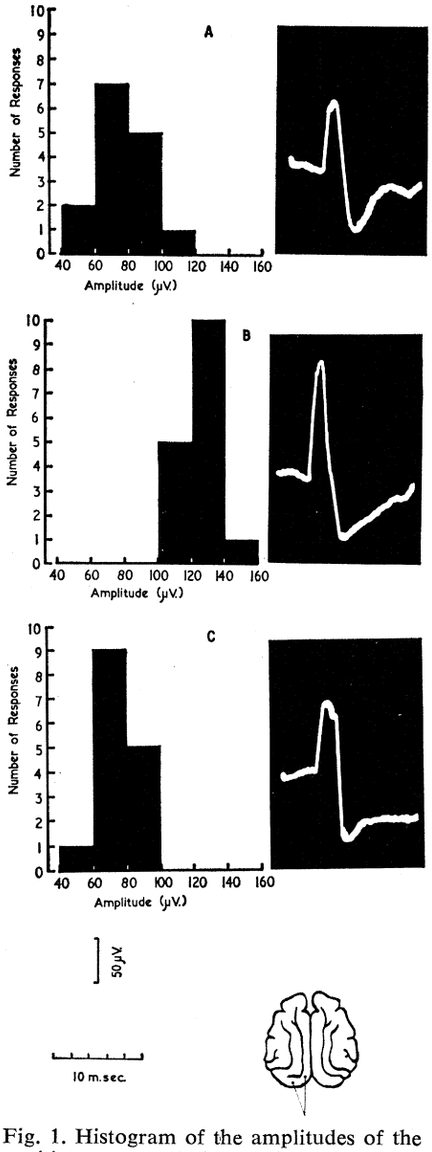

Fig. 1. Histogram of the amplitudes of the positive wave and the median wave form (*5*) for responses during certain 15-second intervals in one cat. (*A*) Preacceleration control interval. (*B*) First interval after peak acceleration. (*C*) Postacceleration control interval. Calibration: Each interval is 10 msec and the vertical bar is 50 μv. Placement of bipolar electrodes is shown in the diagram of the brain.

The animals were anesthetized by an intraperitoneal injection of pentobarbitone-sodium (30 mg/kg) and were supported in an upright contoured case. The responses, recorded from a bipolar arrangement of electrodes, were evoked once a second by photic stimulation of the ipsilateral eye. In animals that gave responses of low amplitude, the electrocortical activity was superimposed to define the waveform.

The results of such an experiment are shown in Fig. 1. The mean amplitudes of the initial positive wave of the 15 control responses recorded immediately before and after acceleration were 76 μv and 77 μv, and the mean amplitudes of the responses recorded during successive 15-second intervals from peak acceleration were 123 μv, 113 μv, 113 μv, and 110 μv. The magnitude of the enhancement was similar in the other animals.

The part which may be played by the cerebral hypotension of positive acceleration in producing these changes was further investigated by observations during the sudden reduction in blood volume (15 ml/kg) induced by the withdrawal of blood from the aorta. These experiments showed that the postsynaptic events of the initial positive wave of the cortical response, evoked by stimulation of the optic radiation, are enhanced particularly during the phase of falling blood pressure.

The phenomenon observed during positive acceleration may arise, therefore, from cerebral hypotension, but it is possible that the central effect of the excitation of ampullary receptors and slowly adapting mechanoreceptors (*3*) may also play a part. It would, however, appear unlikely that changes in angular velocity modified these responses, because no enhancement was associated with deceleration. Nevertheless, the excitation of somatic mechanoreceptors may be important: Dawson and his colleagues (*4*) have shown that, in the anesthetized rat, pressure applied anywhere on the body surface facilitates postsynaptic events at the cortical level in the dorsal column sensory pathway.

ANTHONY N. NICHOLSON
Royal Air Force Institute of Aviation Medicine, Farnborough, Hampshire, England

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Brainstem Mechanisms Subserving Baroreceptor Reflexes

In a recent report (*1*) Reis and Cuénod concluded that "rostral brain structures principally influence tonically brainstem mechanisms subserving baroreceptor reflex excitability rather than those maintaining normal blood pressure." To a large extent this conclusion was based on the observation that sectioning of the buffer nerves in the intact cat resulted in a prompt and significant rise in mean blood pressure, while sectioning of the buffer nerves in the decerebrate cat produced no change or a slight fall in blood pressure. As supporting evidence it was stated that the reflex pressor response to carotid occlusion was "inhibited" in the decerebrate cat.

In our continuing studies of the decerebrate cat, we have observed that sectioning of the buffer nerves in the decerebrate as well as in the intact animal results in a prompt and significant rise in blood pressure. In addition, the carotid occlusion response was not reduced after decerebration (*2*). We observed these results as early as 1937 and as late as July 1964. The anesthetic agents used were similar to those of Reis and Cuénod.

The conclusions reached by Reis and Cuénod are based on negative re-