(*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.

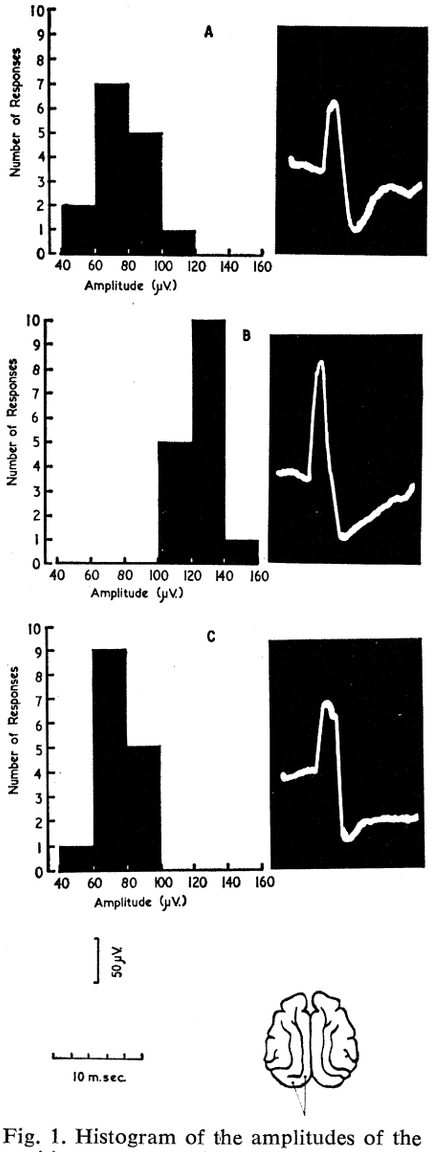

Fig. 1. Histogram of the amplitudes of the positive wave and the median wave form (*5*) for responses during certain 15-second intervals in one cat. (*A*) Preacceleration control interval. (*B*) First interval after peak acceleration. (*C*) Postacceleration control interval. Calibration: Each interval 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

References and Notes

1. P. B. Bradley and J. Elkes, *Electroencephalog. Clin. Neurophysiol.* **5**, 451 (1953); A. N. Nicholson, *Med. Electron. Biol. Eng.*, in press.
2. F. Latham, *Nature* **175**, 1057 (1955).
3. A. Iggo, *Acta Neuroveg.* **24**, 225 (1962).
4. G. D. Dawson, V. P. Podachin, S. W. Schatz, *J. Physiol.* **166**, 363 (1963); A. Angel and G. D. Dawson, *ibid.*, p. 587. 587 (1963).
5. A. L. Berman, *J. Neurophysiol.* **24**, 608 (1961).
6. I am indebted to the Director General of Medical Services, Royal Air Force, for permission to submit this report.

16 July 1964

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-

sults: the absence of a rise in blood pressure following carotid occlusion or sectioning of the buffer nerves in the decerebrate cat. Since we obtained equally active vasomotor reflexes in the intact and in the mid-collicular decerebrate cat, the conclusion is inescapable that the basic control mechanism for baroreceptor reflexes must reside in the brainstem and that the influence exerted by suprapontine structures is not essential.

R. L. KATZ, C. Y. CHAI, N. KAHN
S. H. NGAI, N. N. SHARE, S. C. WANG
Departments of Pharmacology and Anesthesiology, College of Physicians and Surgeons, Columbia University, New York

References

1. D. J. Reis and M. Cuénod, *Science* **145**, 64 (1964).
2. S. C. Wang and C. Y. Chai, *Am. J. Physiol.* **202**, 31 (1962); C. Y. Chai, N. N. Share, S. C. Wang, *ibid.* **205**, 749 (1963).

5 August 1964

Our conclusions were based on four observations: the two well-established facts that (i) decerebration by itself does not result in a fall of blood pressure, and (ii) section of the buffer nerves results in a sustained elevation of blood pressure; and (iii) our positive result that decerebration in animals with three or four severed buffer nerves results in an immediate and sustained fall of blood pressure (which Katz *et al.* appear also to have observed in their vagotomized animals, as their records in the cited references indicate), and (iv) our "negative result" that section of the buffer nerves in decerebrated animals fails to result in a sustained rise of blood pressure, although a transient rise immediately following nerve section has been observed. It is not clear from the correspondents' comments whether the blood pressure rise which they observed after buffer nerve section persists after the minimal 30 minutes interval which we used as our criteria. Without this essential information, a true difference between our results and theirs cannot be established.

The "negative result" used in support of our conclusions, and published elsewhere, was that the pressor response to occlusion of one carotid artery proximal to the only innervated carotid sinus was inhibited. Since the pressor responses which Wang and his associates clearly found unchanged before and after mid-collicular decerebration were elicited by bilateral carotid occlusion, the experiments are not comparable.

Finally, we do not claim that the mechanism of baroreceptor reflexes does not reside in the lower brainstem, that is, in the pons and medulla. Hence, we are not in disagreement with Wang and his colleagues on this point. It is our contention, supported by our facts, that the excitability of these reflexes may be modified by suprapontine structures and that this reflex excitability may be changed without changing the resting mean blood pressure. It is through a modulation of this reflex mechanism that we propose that rostral brain structures exert some tonic control of blood pressure. We have not addressed ourselves to the essentiality of this control. We have merely pointed out its presence.

DONALD J. REIS
MICHAEL CUÉNOD
Department of Neurology, New York Hospital–Cornell Medical Center, New York

19 August 1964

"Cytoplasmic" Sterility

The report on "cytoplasmic" sterility by Ehrman (10 July, p. 159) has some fascinating implications for the field of gene-cytoplasm interactions in general. One possible explanation of his results would be that the Mesitas and Santa Marta cytoplasms have some common structures which interact with genes affecting male fertility; further, that these structures occur in different proportions in the two cytoplasms, the particular ratio in either one being a response to natural selection for effective interaction with the genome. Cytoplasmic structures do not seem to replicate by the same system as the nuclear genes, and it is not necessary to assume that only two kinds of cytoplasmic "alleles" can be present for any one genetic locus, that equal distribution must occur at mitosis or meiosis, or that all of them necessarily multiply at the same rate under all conditions.

VESTA G. MEYER
Delta Branch Experiment Station, Stoneville, Mississippi

3 August 1964

Meyer's explanation is very close to a working hypothesis which I am planning to test. The Mesitas strain consistently carries a heavy infection of microsporidia, while the Santa Marta strain is free of them. It is, of course, possible that other symbionts or parasites of various kinds (protozoans, bacteria, viruses) may also be discovered in these flies. Suppose, then, that each of the six morphologically indistinguishable races or incipient species of the *Drosophila paulistorum* complex carries a symbiont to which it is adapted, and that this hereditary "infection" is transmitted via the egg cytoplasm. The nonhybrid genome keeps the infection under control so that it does not interfere with male fertility. The genotype of the hybrid disrupts this control, and the male hybrids are sterile. The symbionts are controlled by the genotype of the race in which they occur, but they may get out of control in individuals of hybrid genotypes. This may, then, be a causative factor which brings about the reproductive isolation between these incipient species.

LEE EHRMAN
Rockefeller Institute, New York City

2 September 1964

Wild and Domestic Animals as Subjects in Behavior Experiments

In a recent report, Kavanau (*1*) sets forth several generalizations which he says "have important bearings on the rationale and design of experiments on learning and reinforcement." Two of these generalizations seem especially likely to mislead those readers who are not actively engaged in behavioral research. They imply that a new era has arrived in which wild animals must wholly replace domestic animals as subjects in learning experiments.

I would agree that there certainly are differences between wild and domestic animals—differences in rearing and living conditions, in structure, in physiology, and in underlying genetic factors—and that, as a consequence, there are behavioral differences as well (*2*). Granting these does not concede Kavanau's position.

Consider first his statement concerning evolutionary processes: