common laboratory animal may have a very similar though less pronounced stimulus intensity control system to that previously observed in humans and that relation of the control system to behavior may also parallel that found in people. The ability of these neurophysiological data to predict individual differences in animal "personality" was rather striking. This opens the possibility that common laboratory animals may be used for extensive experimental study of these phenomena. R. A. HALL

Santa Clara County Mental Health Services and Institute for Medical Research, San Jose, California 95128

M. RAPPAPORT, H. K. HOPKINS

R. GRIFFIN, J. SILVERMAN

Agnews State Hospital, San Jose, California 95114

Criteria of Brain Death

I suggest that it is important, in regard to the experiments of Hossmann and Sato (1), to elucidate the reasons for the recovery of brain function after presumably prolonged anoxia under their experimental conditions, since this is contrary to the experience of other investigations on the effects of cerebral anoxia, beginning with the classic experiments of Sugar and Gerard (2). Furthermore, the conclusion that this "raises serious questions about the reliability of criteria currently used for the determination of brain death" is unwarranted. Experimental anoxia must approximate the clinical situation in man to have relevance to the problem of criteria for brain death; even at that, species differences must always be considered, as well as the fact that the human condition defies precise measurement of the degree and length of anoxic insult.

The ultimate answers to the criteria for brain death must come from the human experience. The clinical criteria evolved thus far (3) are eminently conservative: totally unresponsive coma, loss of all motor function (including respiration), loss of reflexes and electrocerebral silence (defined as no electrical activity over 2 μ v when recording from scalp electrode pairs ten or more centimeters apart with interelectrode resistances under 10,000 ohms but over 100 ohms) existing for a 24-hour period, except in the instances of initial overdose of a central nervous system

References

- 1. M. Buchsbaum and J. Silverman, Psychosom.
- M. Buchsbaum and J. Shverman, *Psychosom.* Med. 30, 12 (1968).
 J. Silverman and M. Buchsbaum, Proceed-ings of an International Conference on Methodology in Psi Research (Parapsychology Methodology in Pst Research (Parapsychology Foundation Inc., New York, 1970); B. Spilker and E. Callaway, Psychophysiol. 6, 49 (1969).
 J. Silverman, Brit. J. Psychiat. 114, 1201 (1968).
 M. Buchsbaum, R. Henkin, Percept.

- M. Buchsbaum, K. Heinkin, Lorept. Mot. Skills 28, 71 (1969).
 S. Freud, The Complete Works of Sigmund Freud (Hogarth, London, 1955).
 I. P. Pavlov, Experimental Psychology and Other Essays (Philosophical Library, New Works 1957).
- Other Lissays (Liner, P. Wisecup, York, 1957).
 7. R. Fischer, L. P. Rinetine, P. Wisecup, Biol. Psychiat. 1, 209 (1969).
 8. A. Petrie, Individuality in Pain and Suffering (Univ. of Chicago Press, Chicago, 1967); E. D. Ryan and R. Foster, J. Personality Soc. Psychol. 6, 472 (1967).
- J. Ryan and R. Foster, J. Personality Soc. Psychol. 6, 472 (1967).
 9. L. I. Leushina, Fiziol. Zh. (Akad. Nauk. Ukr. RSR) 49, 1400 (1963); R. C. Dill, E. Vallecalle, M. Verzeano, Physiol. Behav. 3, 797 (1968).
- 10. M. Buchsbaum, D. Murphy, F. Goodwin, G. Borge, paper presented at the American Psychiatric Association Meetings, San Francisco, May 1970.

.

24 June 1970; revised 17 August 1970

depressant drug, or hibernation, when the observation period must be extended. Evidence is accumulating that spinal reflexes may be preserved despite brain death. Whether shorter observation periods in specific clinical situations, as has been suggested by some, are appropriate has yet to be determined by systematic research in man.

DANIEL SILVERMAN Graduate Hospital of the University of

Pennsylvania, Philadelphia 19146

References

- 1. K.-A. Hossmann and K. Sato, Science 168,
- K.-A. RUSSHAIM and A. Sare, A. Barry, S. S. (1970).
 O. Sugar and R. W. Gerard, J. Neurophysiol. 1, 558 (1938).
 D. Silverman, M. G. Saunders, R. S. Schwab, R. L. Masland, J. Amer. Med. Ass. 209, 1505 (1960) (1969).

19 May 1970

We agree that an important aspect is to elucidate why, in our experiments, neuronal recovery occurred after more than 1 hour, in contrast to earlier investigations. In most of the experiments

to which Silverman refers (1) the pneumatic cuff method was used, which produces an interruption of both the arterial blood supply to the brain and the venous outflow from the brain. Ames et al. (2) showed that this may cause an impairment of the blood recirculation (no reflow phenomenon) after a few minutes of ischemia. In our experiments, in which the venous outflow was not blocked, a "no reflow phenomenon" did not occur, and this was possibly one of the reasons for the improved recovery.

We have discussed the reliability of the criteria on brain death mainly because in our experiments the electroencephalogram (EEG) was still isoelectric when membrane excitability and synaptic transmission had already recovered for a long time. Furthermore, the reappearance of the EEG seemed to depend on a relatively high blood pressure and could be delayed at normotensive levels. We have noticed the sudden recovery of EEG activity after many hours of electrocerebral silence when the blood pressure was increased. This suggests that even prolonged electrocerebral silence does not prove the irreversible loss of neuronal function. We do not deny that the human brain is irreversibly damaged when the criteria elaborated by Silverman et al. (3) are fulfilled, but we feel that this is due to the current limitations of therapeutic measures rather than to the reliability of these criteria themselves.

K.-A. HOSSMANN, K. SATO Department of General Neurology, Max Planck Institute for Brain Research, Cologne-Merheim, Germany

References

- 1. H. Kabat and C. Dennis, Proc. Soc. Exp. Biol. Med. 38, 864 (1938); R. G. Grenell, J. Neuro-pathol. Exp. Neurol. 5, 131 (1946); H. Hirsch, K. H. Euler, M. Schneider, *Pfluegers Arch.* Ges. Physiol. 265, 281 (1957).
- A. Ames III, R. L. Wright, M. Kowada, J. M. Thurston, G. Majino, Amer. J. Pathol. 52, 437 (1968).
- 3. D. Silverman, M. G. Saunders, R. S. Schwab, R. L. Masland, J. Amer. Med. Ass. 209, 1505 (1969).

29 June 1970

Amino Acid Synthesis in Simulated Primitive Environments

In reference (10) of their report Bar-Nun et al. (1) stated: "H. R. Hulett (2), by confusing meteorites with meteoroid and micrometeorite fluxes, reached an erroneous value of 4×10^{-5} cal cm $^{-2}$ yr $^{-1}$ " for the energy flux of meteoric material on the earth. I should have used some term

other than meteorite to refer to the incoming material. However, in one of the references I cited there appears the statement: "The total mass of meteoric material that enters our atmosphere per day may be something of the order of 100 tons" (3). This refers to all incoming solid material, not just material that