Stress-induced increases in catecholamines in the brain of the young chick

The suggestion that stimulation of turnover of 5-hydroxytryptamine (5-HT) with sedation as the behavioural correlate (Hanig, Aiello & Seifter, 1970) prompted this short report. Several other investigators have implicated the catecholamines in sedation or depression, based upon direct intracerebral administration in subjects that have mature blood-brain barriers (Feldberg & Fleischhauer, 1965) and in studies with the newly hatched or young chick which, because of its lack of a blood-brain barrier to these biogenic amines (Waelsch, 1955; Lajtha, 1957; Key and Marley, 1962), becomes sedated upon their peripheral administration (Mandell & Spooner, 1968).

We have been studying the effects of reserpine, administered during embryogenesis of the domestic chicken, upon some biochemical and behavioural parameters post-natally (Sparber & Shideman, 1968, 1969a). The possibility that lower levels of brainstem catecholamines in chicks hatched from eggs injected with drug compared to eggs injected with vehicle might be due to an attenuated effect of stress by reserpine. If moderate stress could increase catecholaimine levels in the brain, perhaps disproportionately greater differences between two groups might result after more severe stress.

Six, twenty-three day-old male chickens hatched from eggs injected before incubation, with the vehicle in which reserpine was dissolved in an earlier study (Sparber & Shideman, 1968), were used. Three were subjected to a stressful situation which consisted of being placed within a box attached to a mechanical shaker (Eberbach Corp., Ann Arbor, Michigan) and shaken vigorously for 2 min. At the end of this time they were killed by decapitation and whole brain catecholamines or adrenaline and noradrenaline were determined by a semi-micro method (Sparber & Shideman, 1969b). The 3 chicks not stressed were taken directly from their brooder and killed.

The catecholamines are significantly elevated as a result of having been in a stressful situation for this short time. The net fluorescence of non-stressed chicks was 50 ± 8 (s.e.) and of stressed chicks 75 ± 7 (s.e.). The difference is significant at $P < 0{\cdot}05$. Since we did not measure catecholamines in peripheral blood, the source of their increase in brain is uncertain. No doubt, sympathetic activation with concomitant adrenal medullary release could account for at least some of the amines. It is an intriguing notion that the lack of a blood-barrier to these naturally occurring depressant compounds in immature organisms might have survival benefit in species which exhibit "freezing" under stressful situations or where sedation would allow greater resistance to environmental changes.

Department of Pharmacology, SHELDON B. SPARBER
University of Minnesota,
Minneapolis, Minnesota 55455, *U.S.A.*

July 9, 1970

REFERENCES

FELDBERG, W. & FLEISCHHAUER, K. (1965). *Br. med. Bull.*, **21**, 36–43.
HANIG, J. P., AIELLO, E. L. & SEIFTER, J. (1970). *J. Pharm. Pharmac.*, **22**, 317–318.
KEY, B. J. & MARLEY, E. (1962). *Electroenceph. clin. Neurophysiol.*, **14**, 90–105.
LAJTHA, A. (1957). *J. Neurochem.*, **1**, 216–227.
MANDELL, A. J. & SPOONER, C. (1968). *Science, N.Y.*, **162**, 1442–1453.
SPARBER, S. B. & SHIDEMAN, F. E. (1968). *Develop. Psychobiol.*, **1**, 236–244.
SPARBER, S. B. & SHIDEMAN, F. E. (1969a). *Ibid.*, **2**, 56–59.
SPARBER, S. B. & SHIDEMAN, F. E. (1969b). *Ibid.*, **2**, 115–119.
WAELSCH, H. (1955). *Biochemistry of the Developmental Nervous System*, pp. 187–207. New York: Academic Press.