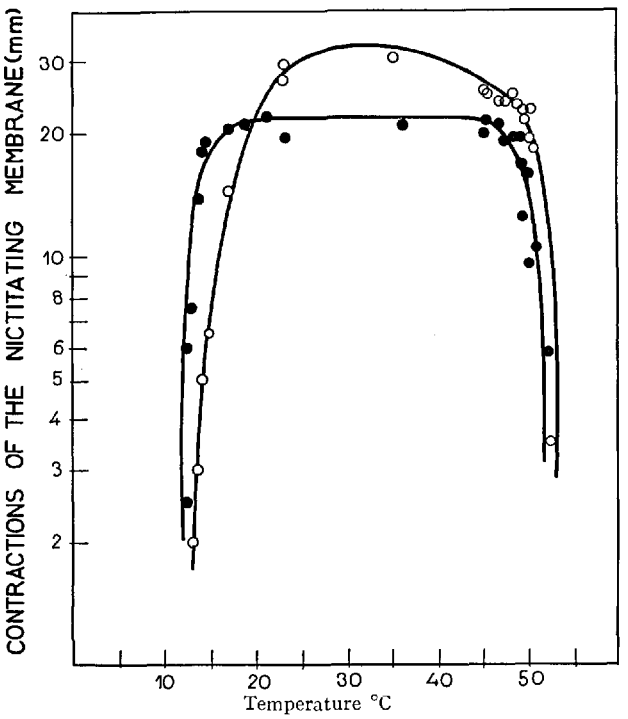

Fig. 3. – Contractions of the nictitating membrane as function of the temperature for two different frequencies of stimulation: 2/s (black dots) and 10/s (white circles).

choline was greatly increased at higher rates of stimulation. When the same experiment was repeated at 20°C an increased number of preganglionic shocks did not increase the total output of acetylcholine.

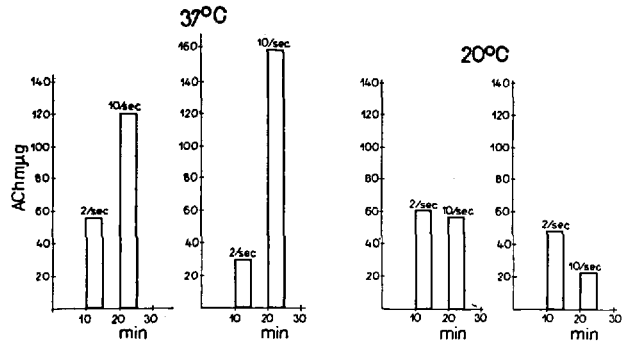

Fig. 4. – Acetylcholine output at 37°C and 20°C for different frequencies of stimulation (2 and 10/s). Each block represents the amount of acetylcholine released during 5 min stimulation of the cervical sympathetic.

These results seem to support our assumption expressed in a previous paper[3] that the disagreement between Browns results[5] and ours was due to the difference in the frequencies of stimulation used in the experiment.

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[5] G. L. BROWN, J. Physiol. *124*, 26 P (1954).

Résumé

Durant le refroidissement du ganglion cervical supérieur perfusé du chat, la réaction de la membrane nictitante est notablement plus sensible aux stimulations de haute que de basse fréquence. Aux températures élevées, c'est l'inverse.

L'influence de la fréquence des stimulations a aussi été perceptible lors des expériences, au cours desquelles le rendement de l'acétylcholine a été déterminé. A 37°C, le rendement s'accroît sensiblement lorsqu'on accélère le rythme de stimulation. A 20° C, cependant un nombre croissant de chocs préganglionnaires n'augmente pas le rendement total de l'acétylcholine.

Effects of Intense Noise on Adrenal and Plasma Cholesterol of Mice

The problem of intense noise and its relation to hearing impairment or other systemic injury in animals and man has recently received renewed emphasis. This is partly due to certain legislative measures adopted in some states where hearing loss is now listed as an occupational disease. In part it is also due to the belief that noise is a stressor agent capable of exceeding the limits of physiological hyperactivity of the pituitary-adrenocortical system.

In earlier studies[1] we found that exposure of mice to moderately intense noise (110 db, 10–20 kc) did not result in excessive adrenocortical activation and consequently noise did not appear to constitute a 'harmful' nonspecific stressor stimulus. Criteria used to measure adrenal activation were: adrenal hypertrophy, eosinopenia, thymicolymphatic involution and the appearance of gastric ulcers. Since several workers have reported that noise acts in a fashion similar to other stress agents[2] we decided to extend our studies to include much higher noise levels in an effort to elicit physiological changes characteristic of a stress response.

The sound apparatus used in the present study[3] consisted of a noise generator, a 700 watt University B-24 loudspeaker (having 24 separate 30 watt speakers), a Bogen amplifier and a Navy Beach amplifier. Sound levels attained were about 140 db with most of the energy concentrated in the 150–4800 cy/s frequency range. Such levels approximate those put out by turbo-jet engines and are near the maximal limit one can obtain with conventional diaphragm type loudspeaker systems. Individually isolated mice were exposed to noise for 5 min and autopsied immediately after exposure and at 3 and 6 h intervals for analysis of changes in adrenal weight and in adrenal content of cholesterol and serum Na, K, and Ca levels following noise exposure. Preliminary analyses for total cholesterol were made using the SPERRY-WEBB technique[4]. This was later abandoned in

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[2] E. DAY, D. FLETCHER, G. NAIMARK, and W. MOSHER, J. Aviation Med. *22*, 316 (1951). – R. MILINE and O. KOCHAK, C. R. Ass. Anat., 38e réunion, Nancy *1951*, 1. – C. FORTIER, Endocrinology *49*, 782 (1951).

[3] Authorized for publication as paper No. 2183 in the Journal Series of The Pennsylvania Agricultural Experiment Station. This work was sponsored in part by the Aero Medical Laboratory, Wright Patterson Air Force Base, Dayton, Ohio, under contract No. AF 33(616)-2505.

[4] W. M. SPERRY and M. WEBB, J. biol. Chem. *187*, 97 (1950).

Table I

Cholesterol levels in adrenals and plasma of mice exposed to intense noise

Group	Sex (*n*)	Treatment	0 h		3 h		6 h	
			Adrenal *	Plasma **	Adrenal	Plasma	Adrenal	Plasma
I	M (9)	controls	3·0 ± 0·8	149 ± 9	4·7 ± 0·4	148 ± 4	3·0 ± 0·9	161 ± 10
II	M (15)	sound (114 db, 10–20 kc)	3·5 ± 0·6	146 ± 27	3·7 ± 0·5	146 ± 9	4·5 ± 0·7	150 ± 17
III	M (12)	controls	4·6 ± 1·6	63 ± 7	5·1 ± 0·7	62 ± 13	4·5 ± 0·9	50 ± 16
IV	M (12)	sound (149 db, 150–4800 cy/s)	5·1 ± 1·2	69 ± 2	4·6 ± 1·4	59 ± 8	5·0 ± 1·8	45 ± 11
V	F (12)	controls	7·2 ± 1·9	49 ± 6	6·8 ± 0·4	49 ± 6	8·3 ± 1·8	54 ± 18
VI	F (12)	sound (149 db, 150–4800 cy/s)	7·4 ± 0·7	66 ± 12	6·9 ± 1·0	72 ± 15	8·2 ± 1·0	49 ± 8

* mg cholesterol/100 mg adrenal ** mg cholesterol/100 ml plasma

favor of a simpler method of analysis recommended by ZLATKIS, ZAK, and BOYLE[5].

Cholesterol levels in adrenals and plasma of control mice and mice exposed to intense noise are summarized in Table I. Two noise levels were used to stimulate different animal groups. The first noise stimulus (114 db, 10–20 kc) may be likened in approximate magnitude to the noise produced next to a riveter but at a higher frequency. The other noise source approaches the levels produced by jet motors. It appears from Table I that neither of the noise stimuli caused any appreciable change in adrenal or plasma cholesterol levels. The mean adrenal and plasma cholesterol values of groups I and II differ from groups IV through VI since the first two groups were analyzed by the SPERRY-WEBB technique and the others by the method of ZLATKIS.

Additional data included taking adrenal weights and measuring serum electrolytes at 0, 3, 6 h after noise exposure. The data in Table II reveal that noise likewise has no effect on adrenal weight or serum electrolyte concentrations. Although the failure to obtain adrenal hypertrophy or adrenal cholesterol depletion in the present study would seem to indicate that noise exposure was not followed by excessive adrenocortical activation, there always remains the possibility that chronic or continuous noise exposures could result in a breakdown of normal defense mechanism in the animal. Recently we conducted several studies of possible stress effects of prolonged exposure of mice, rats and guinea pigs to intense noise for periods ranging from one to four weeks duration. No evidence was obtained which would suggest an overactivation of the pituitary-adrenocortical system. This would seem to lend further credence to the assertion that homeostatic adjustment mechanisms of animals are not taxed to dangerous limits by intense sound stimulation.

One still left with the problem of explaining phenomena such as 'ultrasonic sickness' which have been described in men working around jet planes[6]. The major complaints associated with this disorder range from simple subjective expressions of weakness, fatigue and irritability to claims of impotence, infertility and loss of libido. Further studies are needed to evaluate the action of intense noise as a psycho-physiological stress since it has long been recognized that disturbances in reproductive capacity sometimes arise from anxiety situations without any observable evidence of functional impairments in the reproductive system.

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Résumé

Nos expériences sur l'effet du bruit intense sur l'activité des surrénales des souris ont donné les résultats suivants: 1° la cholestérine des surrénales et la cholestérine sanguine demeurent inchangées, 2° nous n'avons pas pu obtenir les signes d'hypertrophie de la surrénale ou d'atrophie du thymus après 1 à 4 semaines d'excitations par le bruit (5 min par jour). On constate donc que chez les souris périodiquement soumises à un bruit intense mais de courte durée, le cortex surrénalien ne subit pas d'augmentation excessive.

[5] A. ZLATKIS, B. ZAK, and A. BOYLE, J. Lab. clin. Med. *41*, 486 (1953).

[6] P. BUGARD, H. SOUVRAS, P. VALADE, E. COSTE, and J. SALLE, Sem. Hôp. Paris *29*, 1 (1953).

Table II

Adrenal weights and serum ion levels of mice exposed to intense noise (143 db, 150–4800 cy/s)

Animal	Sex	*n*	(g) Body weight	h after exposure	(mg) adrenal	Na	meq/l Ca	K
Control	M	4	29 ± 3	0	31 ± 4	160 ± 2	4·8 ± 0·5	4·6 ± 0·4
Experiment	M	4	28 ± 1	0	34 ± 6	161 ± 9	4·8 ± 0·4	4·5 ± 0·7
Control	F	4	30 ± 6	0	55 ± 4	154 ± 2	4·2 ± 0·4	4·4 ± 0·5
Experiment	F	4	27 ± 1	0	53 ± 6	159 ± 3	4·7 ± 0·2	4·2 ± 0·1
Control	M	4	30 ± 2	3	32 ± 2	158 ± 4	4·6 ± 0·5	5·3 ± 1·0
Experiment	M	4	31 ± 2	3	31 ± 2	151 ± 8	4·6 ± 0·2	4·2 ± 0·4
Control	F	4	28 ± 1	3	61 ± 7	161 ± 4	4·8 ± 0·5	4·5 ± 0·4
Experiment	F	4	24 ± 4	3	60 ± 11	159 ± 3	4·6 ± 0·2	4·5 ± 0·2
Control	M	4	28 ± 2	6	34 ± 6	153 ± 7	4·4 ± 0·1	3·8 ± 0·6
Experiment	M	4	29 ± 1	6	32 ± 7	148 ± 11	4·3 ± 0·7	3·5 ± 0·4
Control	F	4	23 ± 3	6	57 ± 6	162 ± 0	4·5 ± 0·6	4·7 ± 0·4
Experiment	F	4	26 ± 1	6	57 ± 6	162 ± 0	4·8 ± 0·2	4·9 ± 0·7