Finally, there are some neurofibrillae found in the neurones of birds and mammals whose histochemical nature remains undetermined. Characteristically they are absent from the neurones of wall-lizard and *Uromastix*.

Résumé. L'analyse histochimique comparée des neurones des Reptiles, des Oiseaux et des Mammifères révèle que les divers corps qui y sont inclus ont une histochimie identique. Les sphéroïdes lipides abondent dans les phospholipides des jeunes neurones qui sont transformés en corps triglycérides chez les adultes. Les corps lipides ne sont pas un produit de sécrétion de l'appareil canaliculaire de Golgi dont l'homologue a été observé seulement dans les neurones adultes. La substance de Nissl est pleine de RNA.

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Effect of Paraoxon (Diethyl 4-nitrophenyl phosphate) on Axone Reflex Sweating Produced by α-Lobeline

The physiological significance of axone reflex sweating remains obscure despite a considerable amount of information available on its characteristics, as recently reviewed by WADA[1], who, with KUNO[2], has suggested that this mechanism may play a role in local temperature regulation. As yet, however, there is no evidence that the axone reflex occurs under any physiological conditions.

Nicotinic agents, including acetylcholine, can elicit the response when injected intradermally in appropriate concentrations. This suggests that endogenous acetylcholine, normally liberated at sudomotor endings in the skin, may be capable of initiating an axone reflex. This possibility was tested by MCLAUGHLIN and SONNENSCHEIN[3] who studied the effects of a cholinesterase inhibitor, paraoxon (diethyl 4-nitrophenyl phosphate), on human sweat glands and the sympathetic axone reflex. Intradermal injection evoked local sweating which was definitely not of axone reflex nature. The failure of paraoxon to produce axone reflex sweating was thought possibly to be due to an inadequate enzyme inhibition and subsequent accumulation of a subthreshold concentration of endogenous acetylcholine. On the other hand, paraoxon did facilitate the action of injected acetylcholine in producing the axone reflex. This observation did not, however, allow direct inference of the role of endogenous acetylcholine, for it was not possible to distinguish between the effects of paraoxon on this and on the exogenous acetylcholine.

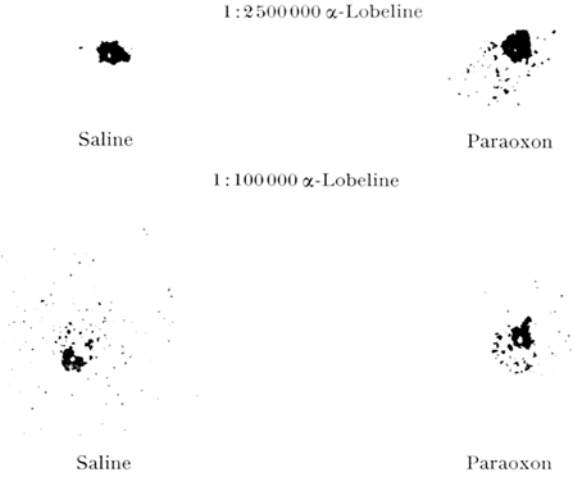
Resolution of this problem was attempted in the present study by determination of the effect of paraoxon on the action of a nicotinic agent, α-lobeline, which is not subject to enzymatic hydrolysis by acetylcholinesterase. It was reasoned that should facilitation of the axone reflex producing action of α-lobeline occur, this would be a result of the increase in local concentration of endogenous acetylcholine acting additively with α-lobeline. Such a finding would support the hypothesis that endogenous acetylcholine can act in the initiation of the axone reflex.

Methods. Paraoxon and α-lobeline were dissolved in sterile 0.9% sodium chloride. Intradermal injections were made into the skin of the volar surface of the forearm in normal human subjects with sterile 0.5 ml tuberculin syringes and $^1/_2$ inch No. 26 needles. 0.3 ml of α-lobeline solution was injected intradermally in the center of an area where 0.5 ml of the paraoxon solution had been injeced intradermally 5–7 min previously. This intervalt allowed for the manifestation of the local sudorific effect of paraoxon[3]. A control was made for comparison simultaneously on the opposite arm where 0.3 ml of the same concentration of α-lobeline was similarly injected into an area previously injected with 0.5 ml of 0.9% saline. Room temperature was maintained constant at 24.1–24.8°C; relative humidity was 56–62%. Sweating was detected by RANDALL's[4] method; 2% tincture of iodine was applied to the skin and allowed to dry, after which a piece of heavy bond paper, already containing starch, was held firmly on the skin for an appropriate length of time, generally 20 sec. The sweat prints so obtained were in-

Effect of paraoxon (1.5×10^{-4} *M*) on axone reflex sweating[a] produced by α-lobeline

Concentration of α-lobeline (w/v)	Number of experiments			
	Total	Facilitation	Inhibition	No effect
1:50000 to 1:125000	17	6 (1)[b]	7 (3)[c]	0
1:500000 to 1:1750000	19	7	8 (1)	3
1:2000000 to 1:2500000	34	24 (3)	3	4

[a] In 6 experiments, the sweating produced by α-lobeline was apparently local.
[b] Numbers in parentheses are experiments whose results were doubtful.
[c] Includes 2 experiments using 1.5×10^{-3} *M* paraoxon.



Effect of intradermal paraoxon (1.5×10^{-4} *M*) in facilitating the production of axone reflex sweating by a low concentration of α-lobeline and inhibiting that of a high concentration. In each case, sweat prints were taken for the interval 20–40 sec after injection of α-lobeline.

[1] M. WADA, in *Essential Problems in Climatic Physiology* (Nankodo Ltd. Co., Tokyo 1960), p. 185.
[2] Y. KUNO, *Human Perspiration* (Charles C. Thomas, Springfield 1956), p. 291.
[3] J. T. MCLAUGHLIN and R. R. SONNENSCHEIN, Acta pharmacol. toxicol. *17*, 7 (1960).
[4] W. C. RANDALL, J. clin. Invest. *25*, 761 (1946).

spected directly and were sometimes photographed for a permanent record.

Results and Discussion. The action of low concentrations of α-lobeline in producing axone reflex sweating was usually facilitated by paraoxon, while higher concentrations (≧ 1:1,750000) of α-lobeline were inhibited by paraoxon in more than one-half the trials (Table). The Table gives combined results on 11 subjects, so that dose-response characteristics for individual subjects tend to be obscured. As a rule, the results on each subjects followed the trend that low concentrations of α-lobeline were facilitated by paraoxon and high concentrations inhibited (Figure). Moreover, α-lobeline itself shows the characteristic, previously reported for the nicotinic agents[5], of producing a progressively diminishing axone reflex response at concentrations higher than an optimum. These results suggest that endogenous acetylcholine, here acting additively with α-lobeline, can participate in the initiation of axone reflex sweating. Whether in fact this occurs under physiological conditions remains unknown.

Zusammenfassung. Paraoxon beeinflusst die Wirkung von α-Lobelin über den Axonreflex, der Schwitzen menschlicher Haut auslöst. Geringe Konzentrationen von α-Lobelin werden durch Paraoxon verstärkt, während höhere gehemmt werden. Daraus wird gefolgert, dass endogenes Acetylcholin an der Auslösung des Axonreflexes beteiligt sein kann.

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Department of Physiology, University of California Medical Center, Los Angeles (Calif.), June 6, 1961.

[5] S. Rothman and J. M. Coon, J. Pharmacol. exp. Therap. *73*, 1 (1941).

[6] *Acknowledgements.* This work was supported by a contract between the Office of Naval Research, Department of the Navy, and the University of California, NR 101-385. Paraoxon was kindly supplied by Dr. R. B. March, University of California, Riverside, and spectrophotometric determination of its concentration by Dr. G. Stevenson, University of California, Los Angeles.

Qualitative Effect of Strychnine and Brucine on Spontaneous Potentials from Explants of Telencephalon

The cells of origin of the spontaneous potentials from explants[1] of brain tissue in culture have not yet been determined. Some evidence as to their identity can be obtained from their reactions to pharmacological agents with a known effect on brain tissue. The study reported below concerns the reaction of spontaneous potentials from explants of 15 day chick embryo telencephalon to the analeptic drugs strychnine and brucine.

Each explant was taken in the usual way from the telencephalon of 15 day chick embryos and placed onto the upper aspect of a piece of cellulose sponge on a coverglass in such way as to lie between the sponge and a 36 gauge platinum electrode. The reference electrode (also of 36 gauge platinum wire) was cemented onto the side of a Kahn tube into which the coverglass with the sponge, explant and electrode were placed. A supernatant made from balanced salt solution TDL1[1] and 0.25% human serum protein at 37°C was added to the Kahn tube in such a way as to come half way up the cellulose sponge and immerse the lower end of the reference electrode without touching the explant itself (except insofar as the supernatant permeated the cellulose sponge). The Kahn tube was sealed with a serum stopper containing an air filter to prevent any change in the pressure inside the

[1] A. W. B. Cunningham, M. Dougherty, and B. J. Rylander, Nature *186*, 477 (1960).

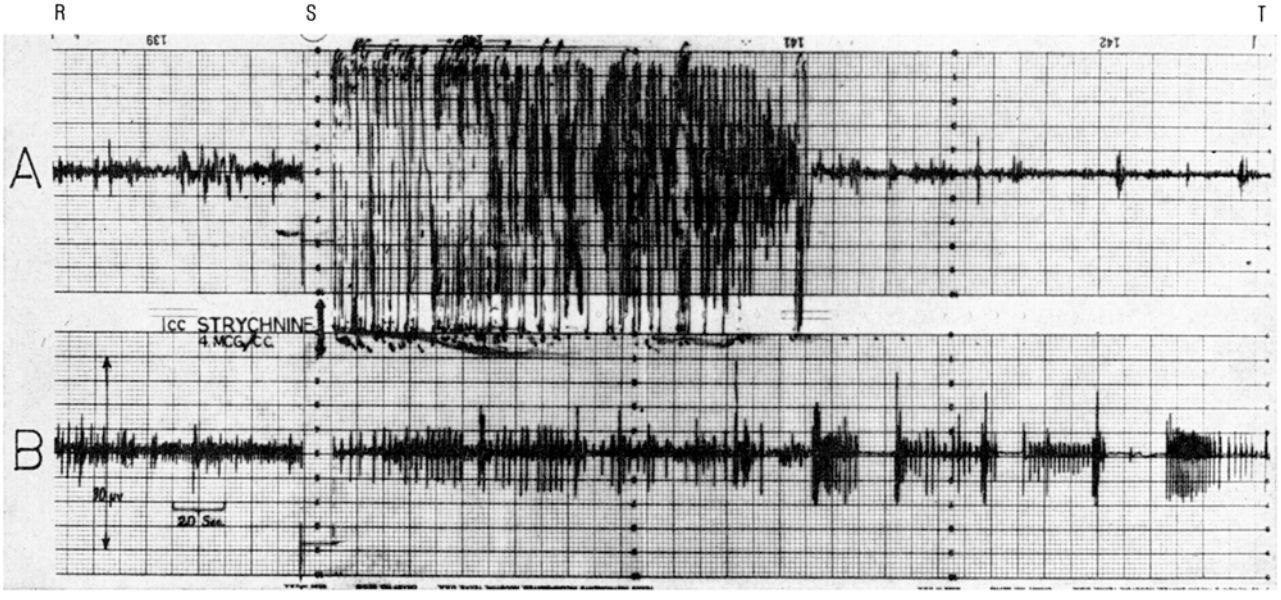

Fig. 1. Simultaneous effect of mcg/cm³ strychnine sulphate on the spontaneous potentials from two explants of 15 day chick embryo telencephalon. In both explants the section R to S represents the normal potential production from the explants. The strychnine is added at the point S and the section S to T represents the effect of the strychnine. The increase in magnitude of the potentials in both explants is obvious as is the subsequent inhibition of the potentials in Explant A. X-axis each major division represents 10 sec. Y-axis eight major division represents 30 μV.