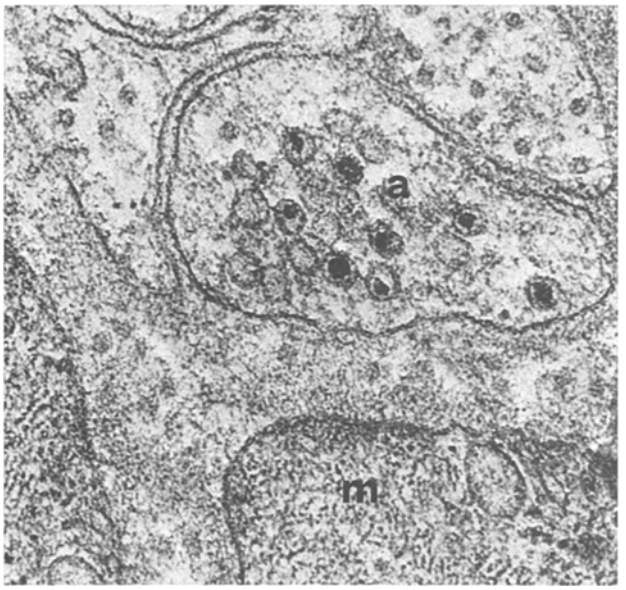

Fig. 2. Intramuscular nerve bundle in the inner muscle layer of the rat small intestine, showing a nerve process (a) with small granular vesicles (catecholamine-storage vesicles); (m) smooth muscle cell. × 83,000.

membrane (micropinocytosis vesicles) can be observed in this segment of a smooth muscle cell, as well as on any other part of its surface.

Summing up, the inner muscle layer of rat small intestine displays numerous nerve bundles; a distinction between true intramuscular and perivascular nerve fibres is not possible since each bundle type is intimately related to smooth muscle cells of the tunica muscularis. Three types of nerve processes are recognized as to their vesicular content and transition forms were not observed; this would seem to indicate that there are 3 types of nerve fibres; at present, identification extends only to the nerve fibres with small dense-core vesicles (catecholamine-storage vesicles), which are to be considered postganglionic orthosympathetic fibres of extrinsic origin. Varicosities seem to bear no constant relationship to any definite structure, nor to structures situated within a definite distance. Transmitters released by these fibres are expected to act on plasma membranes situated only 200 Å away or on membranes lying at much greater distances. Nerve processes with the same kind of vesicles are observed inside the ganglia of Auerbach's plexus[13].

Riassunto. Nella tonaca muscolare interna dell'intestino tenue di ratto si osservano numerosi fasci di fibre nervose; una distinzione tra fibre intramuscolari vere e proprie e fibre perivascolari non è possibile in quanto ogni ordine di fasci ha rapporti intimi con cellule muscolari lisce proprie della tonaca muscolare. L'osservazione di tre tipi di espansioni a vescicole distinte, senza forme di passaggio, fa ritenere verosimile l'esistenza di tre tipi differenti di fibre nervose.

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Some Characteristics of the 'Auditory Neurophonic'[1]

The cochlear microphonic (CM) is a well-known electrophysiological phenomenon which is a close replicate of the original auditory stimulus. However, the CM is not a neural phenomenon; it has no latency and is present after death, sometimes for hours. A second phenomenon, which has been studied less extensively although originally reported many years ago[2], also resembles the stimulus. Unlike the CM, it is of neural origin and is not restricted to the cochlea. We have termed this phenomenon the 'auditory neurophonic' (AN), a phrase we will attempt to justify below.

The AN has been investigated in some detail recently by BOUDREAU and TSUCHITANI[3–5], after having been virtually ignored for several years. These investigators extended observations of the AN, originally recorded in the VIIIth nerve[2], to the trapezoid body and superior olivary complex. It is mildly surprising that they did not emphasize the neural substrate of the phenomenon. We chanced upon the AN and at the time, unaware of Boudreau's work, we assumed that it was either stimulus artifact or the CM recorded by volume conduction. Control experiments to verify these assumptions revealed instead that the waveforms resembling the stimulus were indeed of neural origin. We report briefly these observations.

Materials and methods. The subjects were 6 adult cats, anesthetized with sodium pentobarbital and fixed in a conventional stereotaxic instrument. They were maintained at normal body temperature in an acoustic chamber. Auditory stimuli, consisting of tone bursts, were presented from PDR 10 earphones through hollow ear bars. Stimulus intensity was always between 80 and 90 db re: 0.0002 dyne/cm^2. Bipolar electrodes consisting of 0.010 inch stainless steel wire, insulated except for 0.5 mm at their tips and affixed side by side with their tips offset vertically by 1 mm, were used to explore the auditory system from the level of the trapezoid body to the medial geniculate nucleus. Histological controls verified intended placements. Bioelectric activity was amplified by conventional differential amplifiers and displayed on a multi-

[1] This study was supported by research grant No. 11250 from the National Institute of Mental Health to N.M.W.

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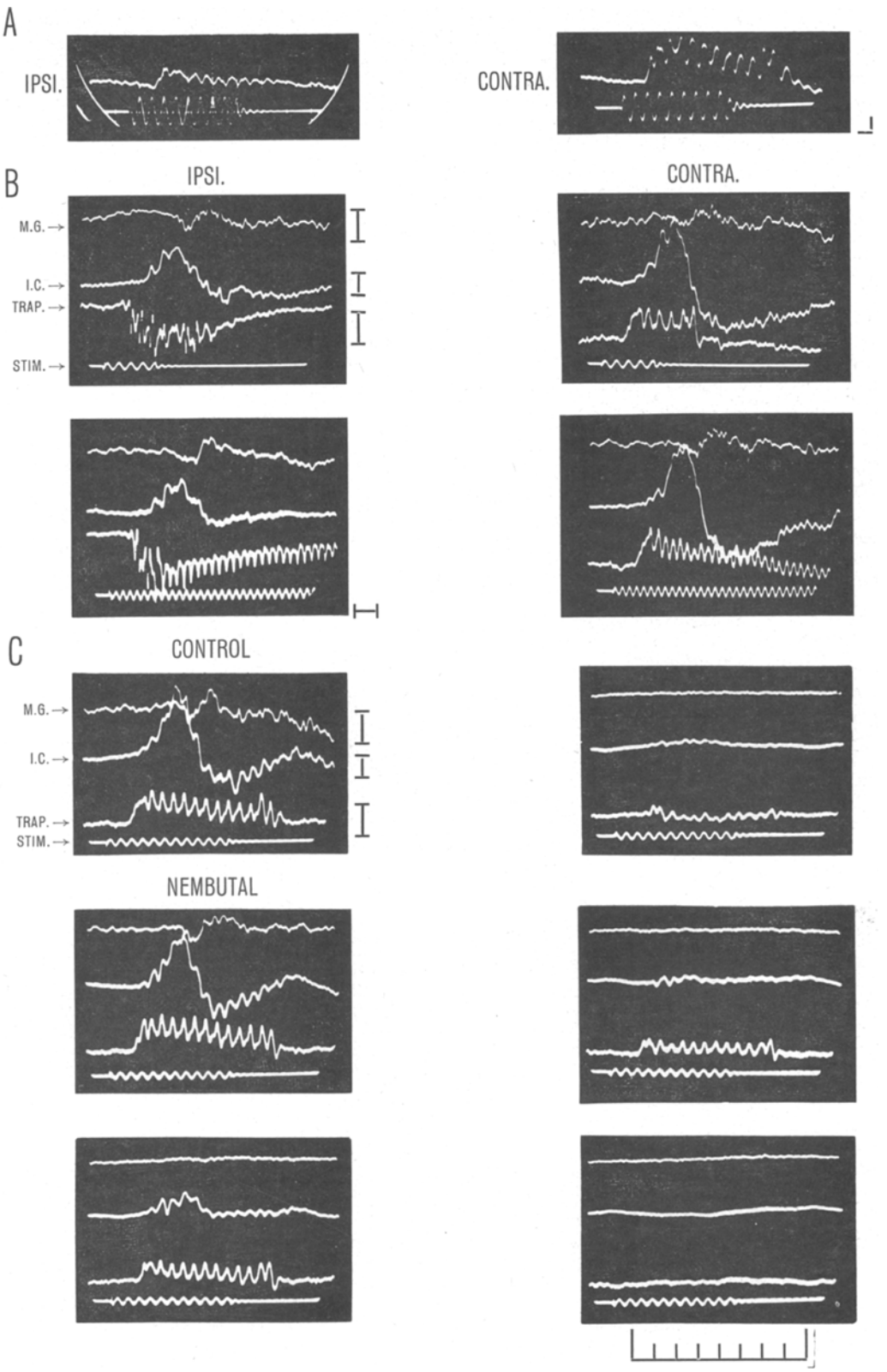

Fig. 1. (A) The AN recorded from the lateral superior olive in response to a tone burst of 1000 Hz. Note that AN amplitude is greater for contralateral than ipsilateral stimulation, and the close correspondence between the stimulus and the AN, particularly on the contralateral side which reflects the damped stimulus at tone burst offset. (B) Simultaneous recordings from the trapezoid body, inferior colliculus and medial geniculate body to 1 kHz (upper) and 1.8 kHz (lower) stimulation. The AN is less clearly seen as the auditory system is ascended, and its onset latency is longer at the colliculus than trapezoid body. (C) Recordings from 3 levels of the auditory system prior to (Control) and at 5 min intervals following (Nembutal) lethal injection (i.p.) of sodium pentobarbital. Note the gradual diminution of the AN, paralleled by that of the evoked potential, and its attenuation first at the medial geniculate, than the inferior colliculus and finally at the trapezoid body. Calibrations: 2 msec and 100 μV.

beam oscilloscope. A bandpass of 0.8 Hz to 10 kHz was employed in order to visualize both gross slow wave evoked potentials and the auditory neurophonic.

Results. The AN is seen as a waveform whose basic frequency is identical to that of the stimulus. It may appear to be as pure a sinusoid as the original stimulus and is easily observed at frequencies between 500 Hz and 2500 Hz. This particular characteristic does not differentiate the AN from the CM. Evidence that the AN is of neural origin comes from 3 other observations.

First, it has a latency (Figure, A). The latency is appropriate for the level of the auditory system from which it is recorded. The AN appears immediately following the gross evoked potential at the site in question (Figure, B). For example, in simultaneous recordings, the AN can be observed to occur with a latency of approximately 2 msec at the trapezoid body and 3.5 msec at the inferior colliculus. Second, the amplitude of the AN is generally greater to contralateral than ipsilateral stimulation above the level of the decussation of the trapezoid body (Figure, A and B). This finding, which is well known for evoked potentials also, cannot be explained on the basis of presumed volume conduction from the cochlea, for such volume conduction would produce greater amplitudes on the ipsilateral side. Thirdly, the AN decreases in amplitude gradually as barbiturate anesthesia is deepened, until death, when it disappears. This amplitude reduction is paralleled by that of the evoked potential. Additional evidence to support the contention that the AN is synaptically transmitted is provided by the finding that its susceptibility to the action of barbiturates is greater at successively higher levels of the auditory system (Figure, C).

Sites from which we have successfully recorded the AN include the trapezoid body, superior olivary complex (including both the medial and lateral superior olivary nuclei), and the lateral lemiscus. The AN may be seen in the inferior colliculus, but cannot be definitively differentiated from pre-synaptic lemniscal activity. We have observed the AN in the medial geniculate body, but it is of small amplitude. Further, it is seen only in the ventral portions of this nucleus, in the region of the entrance of the fibers from the brachium of the inferior colliculus. We are inclined to believe that the AN at this high level of the auditory system is generated by lateral lemniscal fibers which are known to bypass the inferior colliculus.

Discussion. Some characteristics of the AN have been noted in order to emphasize its neural origin. Previous studies have carefully investigated the phenomenon, particularly with regard to its upper limits and relationships to stimulus intensity and frequency[3–5]. None of our observations are at variance with these reports. Some other observations consonant with the findings of BOUDREAU also have been reported[6–8].

The AN has been referred to as 'wave activity'[3], the 'following response'[6], and even the 'frequency following response'[7,8]. We consider none of these terms to be satisfactory because they fail to indicate that the phenomenon is of neural origin. Additionally, 'frequency following response' is restrictive and may be misleading because the phenomenon 'follows' amplitude as well as frequency[5]. We have chosen the term 'auditory neurophonic' because it indicates that the phenomenon is similar to the CM in closely reproducing the actual physical stimulus but is of neural origin and is not restricted to one locus in the auditory system. Finally, the terminology 'auditory neurophonic' (AN) parallels the phrase 'cochlear microphonic' (CM), as does the phenomenon itself[9].

Résumé. Nous avons remarqué dans le système auditif central des ondes qui ressemblent aux ondes microphoniques cochléaires (MC) en imitant étroitement le stimulus. Notre étude montre qu'elles sont d'origine neurale parce que: 1°) l'amplitude est souvent plus grande en réponse à la stimulation contralatérale au niveau du corps trapézoïde; 2°) il y a une latence en rapport avec le système auditif examiné; 3°) une anesthésie progressive cause la dépression de l'onde et finalement sa disparition avec la mort. Nous proposons le terme de phénomène «neurophonique auditif» (NA).

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The Effect of Catecholamines and Nicotine on the Transmembranal Potential of Frog Liver Cells

The pharmacological effects of neuroamines and other drugs on excitable tissues (neurons, muscles, glands) are associated with changes in their membrane potential. We have observed that the resting potential of the liver cell is sensitive to the action of exogenously administered drugs such as epinephrine for which the liver is one of the target organs.

Materials and methods. The liver of unfed, 25–35 g frogs (*R. pipiens*), anesthetized with urethane (4 g/kg) was exposed and kept moist with Ringer's solution. Intracellular recordings were made using KCl micropipettes. 10–100 cells were sampled before and at various intervals after drug administration. 4–8 frogs were used at each dose level. The monoamine oxidase inhibitor (MAOI), pargyline and urethane were administered via the ventral lymphatic sac; other drugs were injected i.m. Results were analyzed using the 't'-test.

Results. Typical potentials for impalements of 5 min or longer varied between 30 and 60 mV. L-*Epinephrine* increased the resting potential at 0.1–0.5 mg and reduced it at even larger doses (Figure 1). The dose response curve was shifted to the left in fall (October and November) in