spectra measurements of solid XeF_4[1] show the pattern of lines characteristic of a planar configuration (*1*), but this experiment on the solid is not nearly so indicative of the molecular configuration in the gas phase as is usually the case in chemical problems because the small binding energy may be comparable to the energy difference between two closely related crystal structures. If future gas phase electron diffraction studies or other experiments prove that XeF_4 is tetrahedral rather than planar, we will be forced to treat *s* and *p* shells as a single entity of eight electrons composed of two interpenetrating four-electron tetrahedra with opposite spins. Lennard-Jones (*8*), Linnett (*9*), and Pauling (*10*) have discussed this type of electron arrangement.

Linear dihalide molecules are also predicted by the model. We start with the same octahedral *np* distribution as before, but now the two halide atoms are attached at the polar positions. In contrast to the binding to equatorial electrons, binding to the polar electrons is enhanced by angular antiparallel spin correlation. Each polar electron is left relatively isolated by two surrounding pairs of almost cancelling antiparallel spin pairs. Circular symmetry in the equatorial plane and the increase in binding produced by antiparallel spin separation constrains the *ns* electron distribution to a nearly spherical shape. Binding through the polar position may allow the halogen atom to penetrate deeper into the noble gas atom, leading to a slightly more completed shell for the halogen, a slightly larger binding energy, and a shorter bond length (*11*).

The various symmetries inherent to the model exclude the possibility of an unambiguous stable trihalide molecule. As suggested by the experiment (*1*), the observed species of this form are probably transitory. The same argument holds true for any odd number of halogen atoms. Stable hexahalides should also be excluded. One of the postulates of the model is the potentiality for binding one halogen atom for every participating antiparallel spin pair. I believe that this is essentially correct and that a hexahalide molecule is not likely to be found, but because of the possibility of contributions from inner shells and the large angular separation of antiparallel spins in the *np* shell there is a definite probability that they exist.

No detailed attempt at present has been made to understand the apparently stable $XeOF_3$ and $XeOF_4$ species. But when the asymmetry of a third type of atom is introduced, a large variety of new electronic configurations become possible, each a small perturbation of the basic di- and tetrahalide configurations. In $XeOF_3$ the oxygen atom may be a direct substitution for a fluoride, being satisfied by the partial sharing of a single electron. A more nearly complete $2p$ shell is possible for oxygen in XeF_3OF, and xenon retains four bonds.

These predictions and discussion apply equally to all inert gas halogenide molecules, but there are other standard chemical effects which make it unlikely that the whole series will be observed as readily XeF_4 and XeF_2. In addition to the electron-sharing type of binding I have hypothesized there will be attractive dispersion forces between the atoms. The two forces may well be of comparable magnitude. Since the dispersive force is directly proportional to the polarizability of the noble gas atoms and since the polarizability decreases with decreasing atomic number for the inert gases (*12*), we may expect the probability of finding these molecules to decrease in similar fashion. The other controlling factor is the relative electronegativity of the halogenide atoms. Higher electronegativity favors binding and since electronegativity decreases along the sequence F, Cl, Br, I (*7*), we expect the probability of formation for inert gas halogenide molecules also to decrease along this sequence (*13*).

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17 October 1962

Second Order Neurons in the Acoustic Nerve

Abstract. In silver-stained preparations of the rat auditory system large neurons were distributed over the whole length of the acoustic nerve. These neurons received many synaptic endings which arose from collaterals of the acoustic fibers. The axons of the neurons ascended toward the cochlear nuclei. Similar neurons were found in the mouse, but not in the bat or cat.

In a variety of mammals, including the rat, mouse, guinea pig, cat, dog, and some bats, the lower auditory system is generally represented as consisting of an acoustic nerve which terminates in the several divisions of the cochlear nucleus. While the literature contains references to nerve cells extending from the cochlear nucleus into the acoustic nerve, these cells are usually considered as belonging to the ventral cochlear nucleus and as being confined to the head of the acoustic nerve. An example of this arrangement is to be found in the distribution of the cells of the interstitial division of the cochlear nucleus in the upper part of the acoustic nerve in the cat as described by Lorente de Nó (*1*).

In the course of a series of anatomical experiments on the auditory system of the albino rat (Sprague-Dawley) we found that the acoustic nerve contained a number of very large neurons scattered throughout its length. The general appearance of the acoustic nerve and its contained neurons is shown in Fig. 1*A*. Acoustic nerve neurons have been found in all the animals (50) we have ex-

amined. Thus, the appearance of these cells is not an isolated or freak phenomenon.

In order to establish the status of these nerve cells, the following observations and experiments were carried out. The tissue used in these experiments was embedded in paraffin, cut at 16 μ, and impregnated with silver by the silver protein method described by Bodian (*2*).

We found that the neurons of the acoustic nerve received a large number of small synaptic endings. These endings arose from fibrils of small diameter, which appeared to be collaterals of the acoustic fibers (Fig. 1*B*). Destruction of the cochlea abolished these synaptic endings while leaving the cells intact (Fig. 1*C*). Thus there is little doubt that these neurons are second order acoustic nerve cells which receive connections from collaterals of the fibers of the acoustic nerve. It is interesting to note that these collaterals arise distally to the well-known bifurcation (or trifurcation) of the acoustic nerve in the cochlear nucleus.

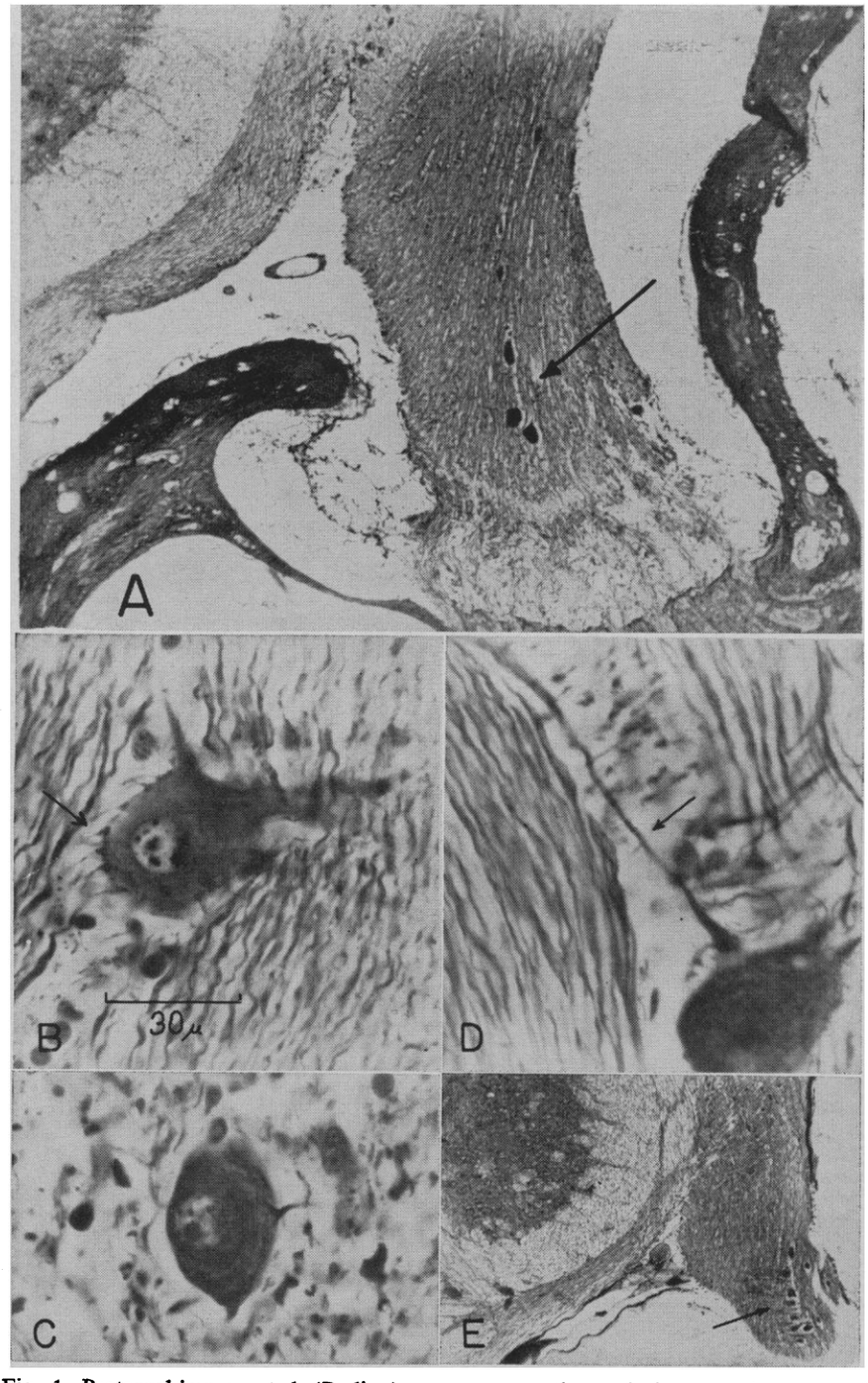

Fig. 1. Protargol-impregnated (Bodian) transverse sections of the acoustic nerve of the rat and mouse. *A*, Distribution of neurons (arrow) in the acoustic nerve of the rat, decalcified preparation (× 54). *B*, Neuron of the acoustic nerve nucleus (rat) showing synaptic endings (arrow) (× 628). *C*, Acoustic nerve neurons after destruction of the cochlea (rat). Note the absence of synaptic endings (× 628). *D*, Axon (arrow) of acoustic nerve neuron shown joining the ascending fibers of the acoustic nerve (rat) (× 628). *E*, Distribution of neurons (arrow) in the acoustic nerve of the mouse (× 37).

In some mammals the vestibular nerve contains scattered cells (Cajal's interstitial nucleus) which receive synaptic endings in a manner similar to that described here. There is some evidence (*3*) that vestibular fibers from the posterior ampulla and saccule enter the central nervous system via the acoustic nerve. Thus it would have appeared possible that the cells we observed in the acoustic nerve were actually associated with the vestibular fibers and represented displaced cells of the interstitial nucleus of the vestibular nerve. However, the foregoing observations eliminate this possibility.

The cells of the acoustic nerve were found to give rise to axons which ascend into and possibly through the cochlear nucleus (Fig. 1*D*). The termination of these axons was not determined.

The most parsimonious assumption concerning the acoustic nerve neurons, in view of the foregoing, is that they represent nerve cells of the interstitial division of the cochlear nucleus which have migrated down the acoustic nerve. However, comparison of the cells of the acoustic nerve with those of the interstitial division failed to confirm this hypothesis. In particular, the cells of the acoustic nerve are (i) much larger and (ii) have much smaller synaptic endings than the cells of the interstitial division.

Comparison of the cells of the acoustic nerve with the cells of the other divisions of the cochlear nucleus failed to reveal any similarities. Thus the acoustic nerve cells form an entity separate from the divisions of the cochlear nucleus. We call this entity the acoustic nerve nucleus. Further data on the status of the acoustic nerve nucleus will be published elsewhere (*4*).

The acoustic nerve nucleus was found to be present in the albino mouse. In this animal the arrangement is identical to that in the rat (Fig. 1*E*). The nucleus was not found in the three bats

(*Myotis*) which have been examined. Neither has the nucleus been observed in the cat.

Galambos and Davis (*5*) have reported the presence of nerve cells in the acoustic nerve of the cat between the internal auditory meatus and the medulla. In our investigations of the cat such cells were found but were quite unlike the cells of the acoustic nerve nucleus. The cells described by Galambos and Davis are probably those which comprise the interstitial nucleus of Lorente de Nó (*1*; *6*).

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Membrane Potential of Toad Ventricle: Changes Produced by Vagal Stimulation and Acetylcholine

Abstract. Reduction produced by vagal stimulation and application of acetylcholine in the duration of the membrane action potential in the toad is clearly demonstrated in the ventricular fiber as well as in the atrium. Vagal innervation of the ventricle is thus demonstrated. Vagal innervation of the ventricle is considered to be less extensive than vagal innervation of the atrium.

The experiments reported here were made to investigate the effects of vagal stimulation and of acetylcholine on the membrane potential of the ventricular fiber in the toad.

The heart was excised, with its nerve supply, and cut open along the midline to expose the endocardial surfaces. The ventricle was pinned on a cork plate in a bath, with the epicardium underneath, and driven with electric stimuli of a fixed strength and rate (24 or 40 per minute). The vagus was stimulated with rectangular pulses of 1-msec duration, at 20 cy/sec and maximal strength for 2.5 seconds.

In order to avoid the influence of acetylcholine liberated in the atrium by the stimulation, an experimental arrangement was set up as follows. A bath of Ringer solution, in which the preparation was placed, was divided into two parts by a partition with a semicircular notch. The partition was set in such a manner that the atrioventricular ring of the preparation lay across the notch. The space between the preparation and the partition was compactly plugged with vaseline-soaked cotton. The Ringer solution in the ventricular side of the bath was thus completely separated from that in the atrial side, so that the acetylcholine liberated in the atrium could no longer diffuse to the ventricular surface through the environmental solution. That it could not do so was ascertained by the fact that no appreciable changes in ventricular membrane potential were produced even several minutes after the introduction of acetylcholine at a high concentration (10^{-4} g/ml) into the atrial side of the chamber. Thus, if any changes occur in the ventricular membrane potential within a few minutes after vagal stimulation, they cannot be attributed to the effect of acetylcholine liberated at the atrium. The membrane potential of a ventricular fiber of the apical area was recorded by means of a glass-pipet microelectrode.

The effects of vagal stimulation are shown in Fig. 1. An acceleration of the repolarization process was observed a few seconds after stimulation (Fig. 1, middle) and attained the maximum value after 1 minute (Fig. 1, bottom). The duration of the action potential was thus reduced from 990 to 457 msec, or by 54 percent. The amplitude of overshoot decreased in 1 minute from 32 to 14 mvolt, or by 56 percent, without any changes occurring in the resting potential. The effects of acetylcholine application were quite similar to those of vagal stimulation. In the example shown in Fig. 2, the duration of the action potential was reduced from 242 to 73.5 msec, or by 70 percent, when acetylcholine in a terminal concentration of 10^{-6} g/ml was applied. The degree of reduction in the duration of the ventricular action potential brought about by these procedures was smaller than that observed in atrial fibers, which are considered to be less developed than the ventricular fiber.

It is well known that the mammalian ventricle has very little innervation and that its membrane potential is unresponsive to the action of acetylcholine (*1*). In cold-blooded animals, on the other hand, the contractile force (*2*) and electrocardiogram (*3*) of the ventricle have been reported by some authors to be affected by vagal stimulation. But the findings of these authors cannot be regarded as direct evidence of vagal innervation of the ventricular muscle because the possibility that acetylcholine

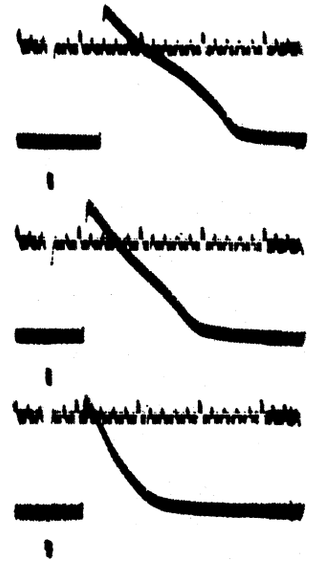

Fig. 1. Changes in membrane potential brought about by vagal stimulation in a ventricular fiber. (Top) Control; (middle) a few seconds after stimulation; (bottom) 1 minute after stimulation. The time is indicated in 100-msec periods (small pips) and 500-msec periods (large pips). Calibration of voltage on the left base:—incates 100 mvolt from the extracellular potential (time base) level.

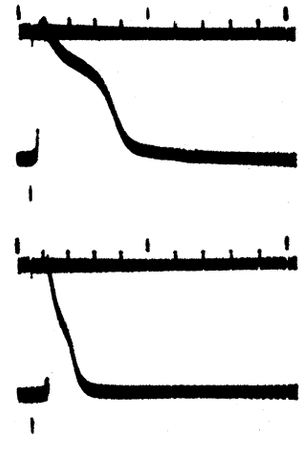

Fig. 2. Changes in membrane potential brought about by the application of acetylcholine (10^{-6} g/ml) in a ventricular fiber. (Top) Control; (bottom) 2.5 minutes after the application of acetylcholine. The time is indicated in 100-msec periods (small pips) and 500-msec periods (large pips). Calibration of voltage on the left base: dash indicates 100 mvolt from the extracellular potential (time base) level.