Forty-nine of the total number of strains in the series were isolated during the period 1944 to 1947—that is, during the early era of penicillin therapy. Seven of these strains were type 80/81, and of these, six were sensitive to penicillin as determined by the tube-dilution method. The one resistant strain had been isolated before the patient received penicillin, and the strain was inhibited by 6.25 units of the antibiotic. In one instance a penicillin-sensitive strain with the pattern 52/52A/80 was isolated from an osteomyelitic lesion before the start of antibiotic therapy; after the administration of penicillin for 1 month, a strain (not included in the tabulation) showing the same phage pattern was isolated from the same lesion and was found to be inhibited by 50 units of the antibiotic.

The only previous report with which our observations can reasonably be compared is that by Wilson and Atkinson (*6*) in 1945, in which are described the techniques of staphylococcal phage typing upon which current methods are based. The majority of the strains examined by Wilson and Atkinson were derived from a variety of infections during a part of the same period covered in our report. Although the typing schema of Wilson and Atkinson is rather different from that in current use, they did recognize certain broad categories which now correspond to groups I, II, and III. When their figures for the frequency distribution of "types" are rearranged to correspond to groups I, II, and III, it is found that the proportion of typable strains encountered in the three groups by Wilson and Atkinson and by us, respectively, are as follows: group I, 33.4 and 32.5 percent; group II, 19.4 and 21.6 percent; and group III, 9.4 and 8.8 percent. Type 80/81, as such, was not identified until 10 years after publication of the report of Wilson and Atkinson. Lysis in the pattern 52/52A was not mentioned specifically by Wilson and Atkinson, and it can be only a matter of conjecture whether any of the strains which they reported to be lysed by phage 52 or phage 52A actually represented type 80/81. It is of some interest, however, to note that five strains of type 80/81 in the present series had been submitted to phage typing 10 or 12 years ago and had been found to be lysed by phages 52 or 52A, or both.

The low incidence of group III strains reported by Wilson and Atkinson and encountered in this series is in striking contrast to the predominance of these strains that became apparent not long after the introduction of antibiotic therapy (*7*). A trend in this direction was seen in the present series, for group III strains, among the strains isolated from 1944 through 1947, were increasingly more numerous, and more frequently penicillin-resistant.

Although the incidence of strains in this series is reported in terms of percentage, we do not intend to imply that the figures necessarily indicate the true distribution of staphylococci among the broad groups during the 20 years in question. We feel, however, that the figures have some degree of validity, to the extent that they express the broad relationships of the several groups and correspond closely to the observations made by Wilson and Atkinson at a similar period of time. There is little question that type 80/81 was a not insignificant cause of staphylococcal disease long before its recent rise to prominence (*8*).

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Nomenclature of Devices Which Simulate Biological Functions

Abstract: The suffix *-mime* is proposed to create generic names for the general class of man-made devices which simulate biological functions. The suffix is used after the stem of the word that describes the organ or cell being simulated; for instance, artificial neurons are described as neuromimes.

With increasing knowledge of the mechanisms of many biological functions, and concomitant development of technology, it has become possible in recent years to construct (or computer-simulate) devices of one sort or another which, to a specified extent, can act like and even replace parts or organs of the living organism. Such artifices as heart-lung systems, which allow prolonged surgery on the heart and pulmonary system, artificial kidneys, which take over renal function temporarily or even semipermanently, and most recently, artificial neurons, which are providing a valuable tool in neurophysiological research, are examples. In the near future many more developments in this direction may be anticipated.

In discussing these devices authors unavoidably make comparisons between the performance of their inventions and the "real thing," draw conclusions on the basis of experiments with their analogs which they wish to apply to the living structure, and attempt to correct their artifacts as a result of apparent discrepancies between artificial and biological behavior.

In all of such activity, juxtaposition of the same names referring to the device and to the "prototype" is likely to become confusing to the reader who may be at a complete loss in trying to find out whether the "neuron" the author is discussing is the real or the simulated thing. The author, anticipating this, can find several ways to avoid confusion.

One obvious way is to label his device, whenever he is talking about it, "artificial"; but this becomes clumsy, and after a few papers on the subject, authors tend to assume that by now everybody knows what they are writing about, and drop the adjective. Another method is to baptize the device with some arbitrary name, for example, ARKID for artificial kidney, or CARDIOTRON for an artificial heart-lung system; the -tron suffix seems to be especially popular. The trouble is, again, that the author (usually after the first paper) neglects to clarify the name, or at most refers to it in a footnote.

There seems to be reason, therefore, to make a case for universal nomenclature specifically designed for artificial devices which simulate to some extent biological functions. Such a nomenclature should meet several criteria: (i) It is desirable, for purposes of orientation, that the stem of the word which refers to the original cell or organ be retained; so, in artificial kidney systems the stem *nephr-* or *ren-* should occur, in heart-lung systems *card-* or *pulm-* should be present, and artificial neurons should have *neur-* or *nerv-* in their names; (ii) For convenience in writing and speaking, a single word of not more than three or four syllables should suffice. This leads immediately to the necessity of using prefixes or suffixes; (iii) The affix, then, should be easily recognized for meaning: -like, simulating, analog of, behaving as if, and so forth; (iv) Affixes already much in use, such as *para-*, *meta-*, *-oid* and *-id* should, where possible, be avoided, so as not to evade one confusion by creating another.

The choice now narrows down to a few relatively little-used affixes, of which the prefix *sim-* or *simu-* (from

the Latin stem: *simul-* = -like, as in *simulare* = simulate), and the suffix *-mime* (from Greek stem: $\mu\iota\mu\eta$ = imitate, as in pantomime), appear best suited. Of these two, *sim-* (or *simu-*) is the more difficult to identify in formations such as: simuneuron, simunephron or simoculus (artificial eye).

I would, therefore, propose that the suffix *-mime* be adopted to designate artificial devices simulating biological functions. The suffix should be understood to mean that the device under discussion belongs to this general class of artifices. Thus, the *-mime* ending can be considered to yield a generic name, whereas authors are, of course, free to name their inventions by any specific name they please. So ARKID is a species of *nephromime*, cardiotron is a *cardiomime*, and the Perceptron (a mathematical model) is an *oculomime* or *neuromime*, depending on one's emphasis. The various types of transistorized artificial neurons are all neuromimes, and so are nets of computer-simulated models.

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Acetylcholinesterase Regeneration in Peripheral Nerve after Irreversible Inactivation

Abstract. The return of acetylcholinesterase activity was studied in several cholinergic structures in the cat after irreversible inactivation by di*iso*propylfluorophosphate. It was found that enzymic activity returned uniformly along the hypoglossal and cervical sympathetic nerve trunks. No evidence for somatoaxonal convection of enzyme was obtained.

It is widely held that axonal protein is formed in the perikaryon of the neuron and conveyed peripherally by centrifugal displacement of neurocytoplasm or axoplasmatic currents. This conception developed first as a result of the comprehensive studies of Weiss and Hiscoe (*1*), which suggest the phenomenon of somatoaxonal convection of axoplasm. Secondly, when the essential role of ribonucleic acid in protein synthesis became appreciated, its absence from the axon in the form of visible aggregates of ribonucleoprotein (variously termed Nissl substance, granular endoplasmic reticulum, and ribosomes) lent further credence to the concept of dependence on the perikaryon for biosynthesis of protein for the remainder of the neuron. However, it might be pointed out that the complete absence of ribonucleic acid from the normal, mature axon has never been proven, and that ribonucleic acid is certainly not absent from embryonic nerve (*2*).

The evidence that enzymes, specifically cholineacetylase and acetylcholinesterase, migrate distad in the axon after their formation in the cytoplasm has been suggestive, albeit somewhat tenuous. Thus, Hebb and Waites (*3*) and Sawyer (*4*) demonstrated an increase in enzymic activity of cholineacetylase and acetylcholinesterase, respectively, in the regenerating proximal stump of peripheral nerve after sectioning. In addition, Lewis and Hughes (*5*), using a nonspecific histochemical method, demonstrated that cholinesterase is present in the outgrowing embryonic nerve of the toad *Xenopus laevis*. However, a histochemical study by Schwarzacher (*6*) of acetylcholinesterase content in neuronal somata during peripheral nerve regeneration indicated that this enzyme is not present in the perikarya during the outgrowth period. The last-mentioned finding controverts the validity of the conclusion, generally drawn in previous studies by many workers in the field, of somatoaxonal migration of acetylcholinesterase. The observations of Sawyer (*4*) and Schwarzacher (*6*), considered together, suggest rather that the biosynthetic systems for acetylcholinesterase may migrate only during growth (that is, during regeneration and embryogenesis). If the dissolution of Nissl substance (chromotolysis) which invariably follows axonal sectioning is regarded as the mobilization of ribonucleic acid for its axonal passage, this phenomenon lends further support to the latter hypothesis.

The most direct evidence for axonal migration of protein was obtained by H. Koenig (*7*) and Waelsch (*8*), employing intrathecal administration of isotopically labeled amino acids in the cat and frog, respectively. "Peaks" of radioactivity in peripheral neuronal protein were observed by Koenig to be displaced peripherad in the ulnar and sciatic nerves at rates ranging from 2 to 11 mm/day.

Fukuda and Koelle (*9*), in a histochemical study, using the thiocholine method of Koelle (*10*), showed that the cytoplasmic acetylcholinesterase of the neurons of the cat ciliary ganglion has a distribution pattern closely resembling that of the Nissl substance. In addition, following inactivation by di*iso*propylfluorophosphate, the regenerating acetylcholinesterase appeared in the cytoplasm before its presence in the surrounding preganglionic axonal terminals could be detected. Such observations support the general contention of somatic synthesis of the enzyme prior to its transit to the periphery.

On the assumption that the "somatoaxonal convection hypothesis" is true for acetylcholinesterase, it should be possible to demonstrate a proximodistal "activity" gradient of the enzyme along the nerve after its irreversible inactivation. In order to test this hypothesis, a high dose (40 μmole/kg, given intravenously) of the irreversible anticholinesterase agent, di*iso*propylfluorophosphate, was administered to anesthetized cats previously treated with atropine (5 mg/kg), and the regeneration of acetylcholinesterase was followed over a period of 1 to 15 days in selected cholinergic structures. The enzyme activity was assayed by a modification of the method of Bonting and Featherstone (*11*), with methacholine as substrate. The following structures were studied: (i) hypoglossal nucleus; (ii) three successive segments of peripheral hypoglossal trunk; (iii) two successive segments of the distal portion of the cervical sympathetic trunk; (iv) superior cervical ganglion (acetylcholinesterase localized chiefly in cholinergic axonal terminals); (v) ciliary ganglion (acetylcholinesterase localized chiefly in neuronal cell bodies); and (vi) the inferior oblique muscle of the eye. The structures were selected to yield as complete a picture as possible of the regeneration of acetylcholinesterase in cholinergic somata, axons and axonal terminals, and a nonneuronal tissue.

Figure 1 shows the patterns of acetylcholinesterase regeneration observed in the structures studied. Of primary

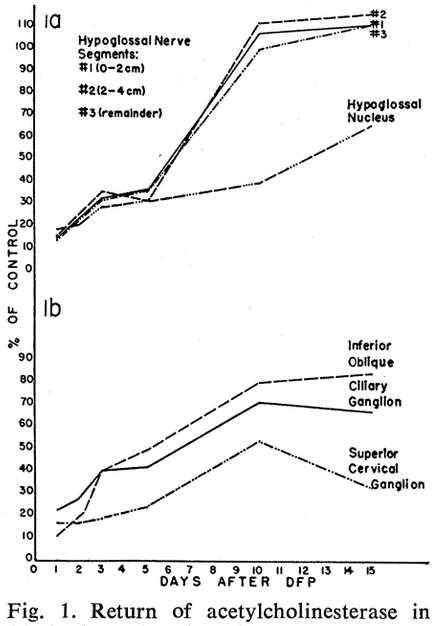

Fig. 1. Return of acetylcholinesterase in (*a*) the hypoglossal nucleus and nerve, and (*b*) the superior cervical and ciliary ganglia and inferior oblique muscle, after irreversible inactivation by di*iso*propylfluorophosphate. The time intervals of the study were 1, 2, 3, 5, 10, and 15 days. Each point represents the average for three animals with the exception of the point for the 2-day period, which represents the average for two animals.