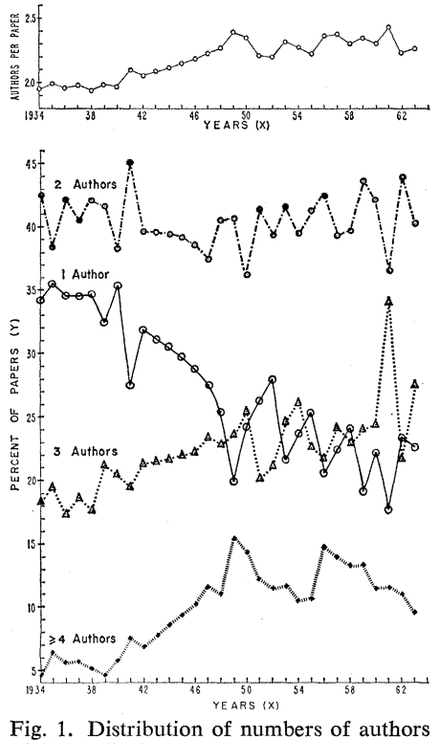

Fig. 1. Distribution of numbers of authors of biomedical papers.

As estimated from the Price curves (*1*) extrapolated (he publishes no data), papers cited in *Chemical Abstracts* by four or more authors increased from about 2.7 percent in 1946 to about 9.5 percent in 1963; even if our biomedical curve does show a slight trend, it is minuscule compared to this. Price writes (*1*): ". . . if the trend holds . . . by 1980 . . . we shall move steadily towards an infinity of authors per paper. It is one of the most violent transitions that can be measured in recent trends of scientific manpower and literature." At present this may be true of the chemists, but if the *lack* of trend reported here continues, planners of biomedical journals can expect that the average number of authors per paper will still be about 2.3 in the year 1980.

This striking contrast between chemists and biomedical scientists is puzzling. An explanation was sought in the fact that *Chemical Abstracts* is a heterogeneous universe compared with *Federation Proceedings,* since the former cites many papers in nonchemical disciplines. To test this idea, the program for the fall 1963 meeting of the American Chemical Society (*6*), which might be expected to represent chemists speaking to chemists, was analyzed for authorship distribution (a 50-percent sample). In each authorship category the correspondence with the Price curve extrapolated to 1963 (Table 3) was good, and thus there is no significant difference in authorship distribution between the "diluted" and the "pure" chemist universes.

It is interesting, also, that when these percentages for the chemists are compared with those for the 1963 biomedical papers (Tables 1 and 3), there is, in the four-or-more-author category, no significant difference. This, however, is apparently a coincidental crossing of two curves with different slopes; for the Price study shows that the distribution among chemical papers continues to change exponentially toward multiple authorship, whereas that of biomedical papers has reached an almost steady state.

As with all growth curves, this presently exponential curve for chemical papers must at some time pass through a point of inflection and eventually become asymptotic to some line parallel to the time axis. It appears that biomedical papers reached this inflection point almost two decades ago.

It is an attractive hypothesis that the multiple authorship trend among biomedical workers during the war years was caused by heavy pressure to get the research done, with forced emphasis on the team approach. But this trend did not hold for the chemists; and I have not been able to contrive an explanation.

Another speculation is that the difference between the authorship habits of the Federation members and those of the American Chemical Society may lie in the much higher qualifications for membership in the Federation. Perhaps the more mature and seasoned scientists who make up the Federation find less need for multiple research collaboration than do the chemical writers who are, *on the average,* less well established as independent investigators.

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2. Abstracts of annual meeting papers are recorded in *Federation Proceedings* since 1942, and in the meeting programs or the journals of the constituent societies prior to that. The number of meeting papers ranged from 459 in 1934 to 3284 in 1963.
3. W. J. Youden, *Statistical Methods for Chemists* (Wiley, New York, 1951), chap. 5.
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5. W. J. Youden, *Science* **120**, 627 (1954).
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7. I am indebted to W. J. Youden, consultant in applied statistics, National Bureau of Standards, for advice and assistance. Price wrote me, in a personal communication: ". . . sample counts taken for physics literature in general and for mathematics show just the same behavior as chemistry . . . so that one might have a good case for saying that the peculiarity you find is a significant particular property of social structures that occur perhaps in medical schools and not in university or industrial research establishments."

18 November 1963

Antidromic Inhibition Accompanied by Ventral Root Positivities

Abstract. *Ventral-root positivities exhibiting the time course of antidromic inhibition were recorded in the cat. Hyperpolarization of the motoneuron occurs concomitant to antidromic inhibition without damage to the motoneuron. Under optimal conditions L7 and S1 ventral-root filaments show electrotonic potentials of positive sign with the time course of antidromic inhibition. Conditions predisposing to membrane depolarization are not responsible for this hyperpolarization. Antidromic inhibition was not found in roots caudal to S1.*

It is generally held that the inhibitory postsynaptic potentials (IPSP) of cat's motoneurons are responsible for much of the inhibition seen segmentally (*1*). Recent criticisms have been based on apparent temporal discrepancies between recordings made from the ventral roots and those made from the motoneuron pool (*2*). The critics hold that penetration of the motoneuron with a microelectrode produces depolarization, driving the resting potential away from the more slowly equilibrating inhibitory equilibrium potential. They have implied that such depolarization may even result from the poor condition of the animal.

Recently Araki *et al.* (*3*) showed the difference in timing between ventral-root and motoneuron recordings can be accounted for by conduction time and asynchrony in the ventral-root volley. They also recorded ventral-root positivities with the time course of inhibition in sacral segments using high gain amplification from electrodes placed at the point of exit of the root from the spinal cord. These ventral-root positivities were associated with the two-neuron so-called "direct" inhibition, and other forms of segmental inhibition were not described.

I have recorded ventral-root posi-

tivities associated with orthodromic inhibition in segments S2 and caudally, but I could find no antidromically evoked ventral-root positivities (*4*). This negative finding may not be surprising in view of the fact that antidromic inhibition had been demonstrated in previous studies only in roots rostral to S2, namely L6 and L7. In fact, I have been unable to demonstrate antidromic inhibition in S2 and caudally. Therefore, examination of more rostral ventral roots was made.

Since the synaptic potentials of motoneurons are conducted electrotonically from the cell soma to the ventral root, exponential decrement of the potentials with distance occurs. Ventral-root recording of synaptic potentials is therefore more difficult in segments rostral to S2 than in more caudal segments because of the longer intraspinal course of the motor axons in the higher segments. This is caused by the increasing bulk of the white matter in rostral segments. The small size of the intracellularly recorded IPSP and the great shunting of external potentials also contribute to recording difficulties.

Nevertheless it was possible to record ventral-root positivities associated with antidromic inhibition. Small ventral-root filaments in segments lumbar 6 and lumbar 7 and the first sacral segment were dissected for lengths of at least 20 mm; the spinal cord was gently rotated, and a small silver hook was placed within 0.5 mm of the exit of the root from the cord. The potential difference between this hook and an electrode placed on a crushed segment of the root filament at least 2 cm from the proximal electrode was recorded without allowing the filament

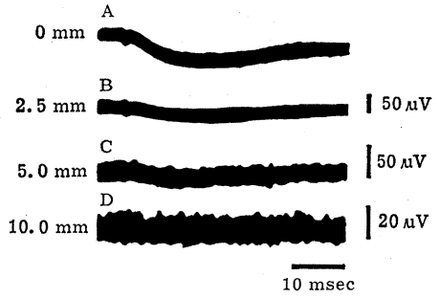

Fig. 1. Ventral-root potentials accompanying antidromic inhibition in a single filament of L7 are shown at various distances from the exit of the root from the spinal cord. Note the different gain settings; the gain in *A* and *B* are the same. The findings in this rootlet are consistent with a length constant of 1.7 mm. Recordings were made by superimposing many faint traces. Positive is down.

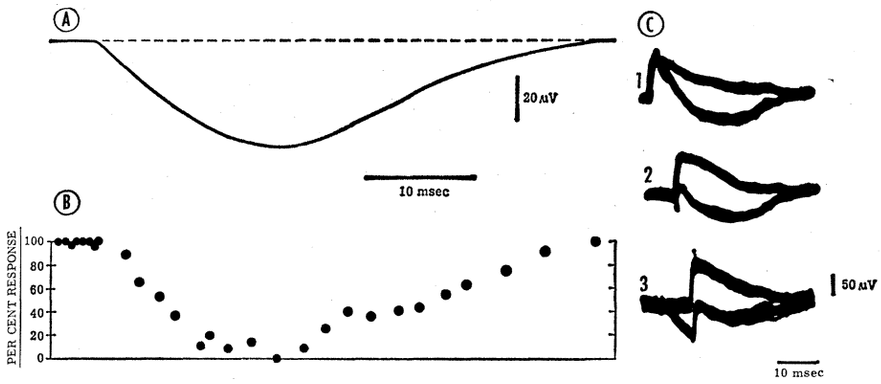

Fig. 2. A tracing of the potential recorded from an L7 rootlet and the percentage of inhibition of an EPSP conditioned by antidromic inhibition are shown in *A* and *B*, respectively, on the same time scale. Records of EPSP's evoked by dorsal root stimulation and their interaction with antidromic stimuli are shown in *C*. Positive is down.

to touch any other structure. The remainder of the root was then placed on stimulating electrodes. Single shocks of appropriate intensity were used to condition a dorsal-root volley. Two conditions were necessary before successfully recording antidromically evoked ventral-root positivities in any filament. These were (i) the presence in that filament of unequivocal antidromic inhibition of reflexes, and (ii) the presence in that filament of an EPSP (excitatory postsynaptic potential) greater than 100 μv in amplitude.

Under these conditions antidromic inhibition was found in filaments from the lower two lumbar ventral roots and at times in the rostral fibers of the first sacral ventral root. Antidromic inhibition was found to be accompanied by ventral-root positivities only in caudal filaments of the L7 ventral root and in rostral filaments of the S1 ventral root (Fig. 1). The potentials were clearly electrotonic and decreased with length constants of 1.5 to 2.9 mm. Potentials as large as 80 μv were recorded but most of the potentials were less than half this amplitude.

These positivities had long time courses that agreed remarkably well with those of the antidromic inhibition found in that filament. This is illustrated in Fig. 2*A* which shows the tracing of the mean potential elicited by an antidromic stimulus in that root. The potential has the same time course as that of the antidromic inhibition of the EPSP evoked by dorsal root stimuli (Fig. 2*B*). Figure 2*C* illustrates superimposed EPSP's with and without conditioning ventral-root positivities at various delays. Monosynaptic reflex spikes were inhibited with the same time course after antidromic stimuli but did not show 100 percent inhibition.

These results provide further evidence for the presence of hyperpolarization associated with inhibition even in the absence of motoneuron impalement. Thus, at least some unpenetrated cells have inhibitory equilibrium potentials greater than the resting potential. The healthiest and least deeply anesthetized animals were those most likely to exhibit antidromic inhibition and its concomitant ventral-root positivity. The vigor of the animal was judged by the amount of spontaneous tonic activity recorded in ventral roots and the size of EPSP's. It is not possible, unfortunately, to estimate from these data the actual proportion of unpenetrated cells with inhibitory equilibrium potentials greater than the resting potential. Nor can the values of IPSP's in unpenetrated cells be adequately estimated.

Finally, in the company of two-neuron orthodromic inhibition, antidromic inhibition is seen to involve IPSP's and their associated conductance change as a primary mechanism. Neither poor "condition" of the animal nor intracellular penetration is necessary for production of the IPSP.

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31 October 1963