localization of the DNase activity in the electrophoretic range of post-albumin α_1-globulins.

G. Riou
C. Paoletti

Unité de Biochimie,
Institut G. Roussy,
Villejuif, France.

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PHYSIOLOGY

Identification of Inhibitory Neurones in the Hippocampus

In their recent article in *Nature*, Andersen, Eccles and Løyning[1] claim to have unequivocally identified the basket cells of the hippocampus as the inhibitory neurones of this brain structure. They claim that "there is no alternative" to this interpretation and that it therefore "seems possible for the first time to give an example from the mammalian nervous system of a recurrent inhibitory pathway where both the inhibitory neurone and its synapses are histologically identifiable".

The conclusions reached by the authors are based on three main premises. The correctness of none of these is fully substantiated by the findings presented in their article.

Premise (1) postulates that the inhibitory neurone should possess "an axon with extensive ramifications . . . distributed to a large number of pyramidal cells". Quite apart from the fact that cells other than basket cells located in the stratum oriens fulfil these criteria (for example, the neurone No. 1 in Fig. 8 of Lorente de Nó's[2] paper), the observations made could just as easily be explained by assuming a rather large number of inhibitory neurones distributing their axons to a moderately small number of pyramidal cells.

It is, however, premise (2) which rests on the shakiest foundations. The authors assume that the synaptic terminals of the inhibitory neurone end on the soma of the pyramidal cell. So far as can be judged from the findings presented, this assumption is based on the laminar profiles of the responses evoked by commissural, septal and 'local' stimulation (Fig. 1*D* of ref. 1). This profile shows peak positivity of a late response component in the pyramidal layer. However, the same profile would result if excitatory post-synaptic potentials (EPSP's) were developed on the apical dendrites. In fact, this explanation would better agree with the laminar profile observed. As Fig. 1*F* of Andersen's *et al.* paper[1] correctly shows, a 'source' developed by a hyperpolarizing potential at the soma level should give rise to two 'sinks', one in the apical dendrites, the other near the alveus. The experimental findings, however, show only one 'sink' in the apical dendrites.

Furthermore, no evidence is given to indicate that the positive wave recorded extracellularly in the pyramidal layer is associated with inhibition of unit discharge and thus may safely be equated with the intracellularly recorded IPSP's. That the two are identical is an assumption for which there exists no proof. The authors themselves note a discrepancy in the duration of these two potentials which they try to resolve by assuming that the shorter duration of the extracellularly recorded potential is due to the fact that the extracellular current flow lasts for a shorter time than the intracellularly recorded hyperpolarization.

However, inhibitory potentials of the same duration as the intracellularly recorded inhibitory post-synaptic potentials (IPSP's) can be observed extracellularly with responses evoked by entorhinal stimulation, as we have shown in our own experiments[3,4]. These potentials are clearly associated with inhibition of pyramidal cell discharge (ref. 4, Figs. 8 and 9). The laminar profile of this extracellularly recorded inhibitory potential showed a peak in the apical dendritic layer (ref. 4, Fig. 5) whereas the cell layer often acted as a 'sink' (ref. 4, Figs. 8 and 9). The synapses producing this inhibitory potential therefore seem to be located predominantly on the apical dendrite and not on the soma. This excludes the basket cells from consideration and makes it likely that other hippocampal interneurons with axons ramifying mainly in the apical dendrites are responsible for these inhibitory potentials. Candidates for this role can be found in the stratum oriens as well as in the apical dendritic layer (for example, ref. 2, cell No. 5 in Fig. 6, cells No. 1, 3 and 6 in Fig. 7 and cell No. 1 in Fig. 8). We have, furthermore, given evidence suggesting that axo-somatic synapses on pyramidal cells may be excitatory rather than inhibitory[4], although we do not claim that our observations provide incontrovertible proof for this interpretation. Nevertheless, one can certainly say that the question whether the basket cells are excitatory or inhibitory neurones still remains unresolved.

Premise (3) assumes that the responses analysed are mediated by three separate inputs. This is very questionable. 'Local' stimulation must have activated the terminals of the commissural pathways and therefore we are dealing at the most with two different afferent inputs, rather than three as claimed.

There seems, therefore, to be no basis for the claim that experiences by Andersen *et al.*[1] have unequivocally identified the basket cells as the inhibitory neurones of the hippocampus. Such a conclusion is merely one among several possible, but unproved, interpretations to be considered, especially since all the known experimental evidence cannot be reconciled with this view.

P. Gloor

Montreal Neurological Institute and the
Department of Neurology and Neurosurgery,
McGill University, Montreal.

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We welcome this critical comment by Dr. Gloor because it gives the opportunity of clearing up some misunderstandings and of refuting Dr. Gloor's statement that there is "no basis for the claim that experiences by Andersen *et al.* have unequivocally identified the basket cells as [the] inhibitory neurones of the hippocampus". The word [the] is in brackets because we made no claim of exclusiveness; otherwise we accept Dr. Gloor's formulation of our position.

We deny Dr. Gloor's statement that premise (2) "rests on the shakiest foundations". Any appearance of insecurity derives from a failure to appreciate the degree of assurance which can be attained, under favourable geometric conditions, when interpreting an assemblage of extracellular potentials together with the associated