39 BEHAVIOR AND STRUCTURE OF RAT PRIMARY AND SECONDARY SCHWANN CELLS IN VITRO
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Schwann (S) cells have a remarkable remyelinating potential in PNS and CNS. Therefore it is of great interest that numerous primary (I) and secondary (II) rat S cells can be obtained by dissociation of neonatal sciatic nerve and by subculture in the presence of mitotic factors, cholera toxin and a pituitary extract (Brain Research, 165, 105, 1979). Freshly dissociated I S cells contain myelin fragments and specific immunostain for the major protein of PNS myelin (Po). During the first days in vitro these myelin components progressively disappear in cells grown in medium containing fetal calf serum and high glucose concentration but not in synthetic medium. Video-intensification microscopy revealed that I S cells form groups and have typical rhythmic pulsations and side-to-side movements. Processes of bipolar S cells extend and retract, sometimes leading to cell migration. In contrast, II S cells are multipolar, show less pulsation and migration but extend longer exploratory processes than I S cells. They become flat and almost confluent before intense mitotic activity occurs. Withdrawal of growth factors results in partial return to bipolar shape. I and II S cells show numerous intermediate filaments and no basement membrane. All cultures contain only 5% Thy I-I positive fibroblasts which show distinct variable shape, edge ruffling and rapid migration under the S cell layer. II S cells mixed with rat brain cells in suspension form aggregates which show myelination three weeks later by immunocytochemical staining for Po protein. Thus, dissociated S cells display characteristic motility, can be grown in large quantities and can reassociate with neurons.

40 ELECTRON MICROSCOPE-MICROPROBE STUDIES OF ALUMINUM IN THE BRAINS OF CASES OF ALZHEIMER'S DISEASE AND AGEING PATIENTS. Serge Duckett * and P. Galle, Faculte de Médecine de Paris (Créteil), Jefferson Medical College of the Thomas Jefferson University, Philadelphia, PA.

The debate concerning the role of Al in Alzheimer's disease goes on and there is still no actual proof that Al does -or does not- cause this disease. We examined with an electron-microscope-microprobe (EMM Camebax) the cerebral cortex of 18 cases of Alzheimer's disease + 4 additional cases (1 bismuth intoxication + 3 ageing brains (62-73 years old). We identified, we did not quantitate but we registered on graph paper the proportional amounts of Al in largest concentrations in lipofuscin granules in senile plaques and in degenerative cells in all 22 brains and smaller amounts of Al in the neuropil and sometimes in plaque cores and in the few neurofibrillary tangles present in Alzheimer's brains. Phosphorus was also present in the lipofuscin, but not in neuropil. The presence of silicium also noted in all brains is being analyzed at present. We eliminated a number of Al artifacts by searching for and not finding Al in the various fluids used in the preparation of the tissues for EMM. We had previously reported (cautiously) the presence of Al in plaques in Alzheimer's disease (C.R. Acad. Scien. (D) : 282:2115,1976). Our observations at this time concur with the quantitative result of Mc Dermott et al (Neurology,29:809,1979)which suggest that the presence of Al increases with advancing age in any human brain. This increase may be due to geriatric atrophy which could give higher levels of Al per gram of tissue. We are not convinced, at this time, of the importance of Al in the pathogenesis of Alzheimer's disease.

41 CENTRONUCLEAR MYOPATHY - A UNIFYING CONCEPT A.W. Dudley, Jr.*, B.J. Shaw, M.K. Bodden, and F.A. La Cour. University of South Alabama, Mobile, AL.

Centronuclear myopathy (CM) has been compared to myotonic dystrophy because of shared age of onset, selective Type 1 atrophy, and central nuclei. A recent report describes myotonia in two CM patients as if to provide a link between the two disorders. However, there remain many distinguishing features due to scattered reports of CM patients with fiber type disproportion, absence of 2 B fibers, aortic valve disease requiring prostheses, and widely varying genetic penetrance, both clinically and histologically. The reader is left perplexed as if looking at families with different disorders.

We biopsied a 52 yo mother and five children, ages 12,17,21,25 and 27 years. All but the 27 yo daughter had CM with Type 1 atrophy. The incidence of central nuclei increased with age, ranging from 20% in the 12 yo son to 100% in the mother. Fatty substitution was subtle in the 21 yo daughter, moderate in the 25 yo son, and advanced in the mother. Fiber type disproportion was mild in the 12 and 25 yo, moderate in the 21 yo daughter, advanced in the 17 yo son (92% Type 1), and complete (100% Type 1) in the mother. Type grouping was present in all biopsies but was mild in the 27 yo. All six patients had excessive intramuscular lipids increasing with age except for only mild increase in the 27 yo. The 12 yo had a superimposed myositis and the mother has a prosthetic aortic valve, insulin dependent diabetes, and requires a walker.

We conclude that CM is slowly progressive and autosomal dominant with 100% penetrance but widely varying degrees of expression. Previously reported superficially disparate cases are in fact recording a single, multifaceted disorder.