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Discussion to papers of Dr Sahgal & Subramani, and Dr Ravichandran

Dr HARDY (*Chairman*)

DR EL TORAEI (U.S.A.). How do you explain then the work of Dr Osterholme with the catecholamine blocking agents helping regeneration or whatever?

DR SAHGAL (U.S.A.). First of all let me say I wish you hadn't asked that question. I don't think Dr Osterholme's work on inhibitors of catecholamines help in the spinal cord injured dogs is reproducable. We have tried reproducing in our laboratory and any other laboratory work which have tried to reproduce it, it doesn't pan out. So the best contribution that I can say Dr Osterholme made in the field of spinal cord injury is the fact that he drew attention to a system of neuro-transmitters that we were hitherto not aware of. His conclusions were certainly erroneous, they were a little simplified to his way of thinking, and his methodology was incorrect because he had mostly formaldehyde fixed tissues that he did fluorescence on.

DR HARDY. One observation from the chair: Dr Sahgal, one question. We are aware as clinicians and pathologists that the injury in most of our patients is a longitudinal one involving many segments, sometimes four or five, but very often at least three. Now you are using a surgical section do you have longitudinal lesions as well?

DR SAGHAL. Yes, no matter how cleanly you cut that spinal cord the spinal cord lesion is always longitudinal, it almost reminds you of a syrinx, it is small at one level and then unexpectedly you see it large at another level and that is why I think you have to be very cautious in the interpretation of our clinical findings, that is when they do not fit a level when you see multi-level lesions; always keep in mind that it is a longitudinal lesion, and this has been borne out in many pathological reports, especially in observations of Brian Kakulas in the Handbook of Neurology a chapter written with Sir George Bedbrook.

DR YEO (*Australia*). Mr Chairman. May I ask our speaker just one question and also make one comment. Our experimental animal is the sheep and we have in fact just completed a series doing radio-active microsphere studies in which we have shown that laminectomy in fact increases the blood flow in the grey matter in sheep within the first hour following operation. Now, that increase only occurs when you are using cautery. If you do a laminectomy without using cautery with, we suspect, reflex vaso-dilatation, there isn't that increased blood flow. So your statement that laminectomy may be an unwise thing, I think may need some qualification. The question I would like to ask you, in view of the fact that you are suggesting a selective vascular hypoxia of the cord would you consider the possibility that if hyperbaric oxygen therapy was available that that could be indicated in the onset of neurological dysfunction in Paget's disease?

DR RAVICHANDRAN (*G.B.*). Thank you. The information on decreased bone perfusion was reported by Ducker in 1978 in Surgical Neurology and his experimental models were rats. As regards the use of hyperbaric oxygen I think that perhaps it is indicated in the cases you mentioned. But I am not sure whether hyperbaric oxygen will carry adequate oxygen to the affected tissue since we know from the work at Wootton that the bone perfusion is increased about four times and the spinal segmental artery is comparatively very much narrower. I'm not sure how much it will contribute.

DR EL TORAEI (*U.S.A.*). I would like to ask you did you, compare the results or calcitonin with the dydronil or the diphosphonate?

DR RAVICHANDRAN. Not it is not a series. The results of the use of calcitonin and diphosphonate (EDTA) have been done by Sadar and more recently Avromedes and I think the blood perfusion has now been measured by Wootton. I am basically interested in the improvement of perfusion of the spinal cord and that work has not been done. I do not know whether EDTA produces an improvement in bone perfusion.