## DISCUSSION

*Hancock, D.* (G.B.). I was most interested in Dr. Yeo's paper, especially in the first patient. I wonder whether he has considered the possibility of pituitary infarction in severe head injury or perhaps not even that severe. I believe the first patient had quite a severe brain injury from his history, at least he finished up with epileptic fits. It is true, that the majority of these quadriplegics do have an accompanying head injury, and I wonder if we are not dealing with, at least in some cases, a traumatic pituitary infarction.

*Silver, J.* (G.B.). Studies were carried out at Stoke Mandeville Centre by Robinson, who showed that the urinary hydroxy steroids were lowest and well below normal in the cervical patients and were normal in low lesions. The one possibility is that the nervous pathway may be influencing the adrenal gland. The other possibility is that the quadriplegics are so extensively paralysed that they may be putting out less adrenal steroids. In support of this is the very low oxygen consumption of these patients and the very low lactic acid production of these patients. We found at Stoke Mandeville, oxygen consumption of these patients about 50 per cent. of normal and similarly with the lactic acid levels. I wonder what Dr. Yeo thinks of that possibility?

*Frankel, H. L.* (G.B.). I congratulate Dr. Yeo on some of his observations. Robinson also found many years ago a significant eosinophilia in all complete cervical lesions. This may be of some interest to him. If one sees a large number of tetraplegic patients over the years one can find in them a number of abnormalities of hormonal origin, such as myxoedema which one sees quite clearly and yet is unable to confirm by laboratory tests. Most commonly a Cushingoid appearance is shown by these patients, including striae and the typical appearance of the upper part of the body of someone with Cushing's Syndrome. There is obviously something going on there, although laboratory findings are within normal limits. How do these patients mimic these diseases, perhaps Dr. Yeo's investigations will give us the answer.

*Wolman, L.* (G.B.). There are just two questions I would like to ask Dr. Yeo: I wondered if, in the cervical injuries, vertebral angiography had been carried out subsequently to see the state of one or other of the vertebral arteries, because the subsequent ischaemia that may result from blocking of one of these may have been a factor in damaging the rostral part of the brain stem and the hypothalamus. The other point concerns the observation made by a previous questioner who wondered about damage to the pituitary as the result of an associated trauma to the head when the cervical spine was fractured. I think this is a definite possibility but I would like to widen it even more and wonder whether there was any evidence of damage to the hypothalamus in addition to or instead of damage to the pituitary or even to the mid brain at the level of the incisuria of the tentorium, because when the cervical spine is fractured the shake up inside the head especially at a narrowed orifice like the incisure of the tentorium may well inflict small petechial haemorrhages on the rostral mid brain or the posterior hypothalamus.

*Hardy, A.* (G.B.). Dr. Wolman, my colleague, is hiding his light under a bushel. I think there is a very great need for controls, I think in the studies of quadriplegics, these investigations that Mr. Yeo describes, must have controls on a bigger group. In regard to Dr. Wolman, I happen to know that he has collected hundreds of pituitaries, and here we have one source of control, I am sure.

*Harris, P.* (G.B.). Regarding Mr. Yeo's paper and the comments which have been made about this, I think this topic and this problem would lend itself to experimental study and I wonder if he is doing this or knows of work in this respect.

*Young, A.* (U.S.A.). I think it is unfortunate that one of the cases that Dr. Yeo reported had fits. The syndrome he is describing is much more common and the fits I

think were a red herring in this one particular case. What he is describing is a syndrome, as he labels it, but I am sure we have all seen in the tetraplegic patient a depression. If it is a paraplegic you will have vague gastro-intenstinal complaints, a gradual lowering of blood pressure. This person does not respond well to stress, he is certainly a surgical risk, and as you commented, the laboratory is no help at all. These people do not have enough steroid, and being rather unscientific in our programme we say to hell with that, we're going to give him steroid anyway. For a number of years now we have given these people steroid, even in the face of infection. They have got over their depressions within 24 hours or 48 hours, they combat their infections better. You are not giving them excess steroid you are really giving them replacement steroid. The spinal man has something going on in his hormonal system, particularly with the adrenal cortex. I have a hunch we are faced with a chronic marginal functioning adrenal. It incidentally can be whipped up in stress and you will get increased plasma sterol, but somehow or other he operates at a very low level. We are in the process of studying over a period of two years plasma sterol when the person is on a Stryker frame in the immobilisation period, when he first gets up for his activity, wheel-chair, brace crutch ambulation, and then during the pre-discharge period when his stress level is certainly the highest. We will complete this study in another year and I will be more than happy to report it to this Society.

*Yeo, J.* (Australia). Mr. Hancock mentioned Sheeham's syndrome. In the pathogenesis we have considered this first. This is why I stressed the hypotension in the original case whether this was a Sheeham's syndrome. We have only gone as far as having a look at histopathologically in one specimen of the hypothalamus. The next problem is of course, who can interpret this. This is an extraordinarily difficult area, if we are going to find infarction it would be nice to know whom to send the slides to, and this goes for some of our spinal cord sections as well. I was grateful for Dr. Frankel's comments. If you read a book like Williams' you find that isolated deficiencies of ACTH and its effect on the adrenal cortex are very rare, and they quote something like two cases. If it was a total pituitary or hypothalamic knock-out, whether it be head trauma or vascular, maybe we should be seeing other signs before we see the syndrome that we are describing. In replay to Dr. Wolman we have not done vertebral angiography, but, of course, we have thought of Basilar Artery insufficiency. Head trauma is, as you know, very hard to exclude, in the history of cervical spinal cord injury. In reply to Mr. Harris, we have not yet done any experimental work, but it is very encouraging to know, that this work has been of interest and the discussion has given me some guidance and help for the future in this regard.

**Mr. D. O. Hancock's paper; 'Congenital Narrowing of the Spinal Canal' was published in *Paraplegia*, 1967, 5, p. 89. This paper was discussed at the present meeting.**

*Sarrias, M.* (Spain). I would like to ask Mr. Hancock a question. He has shown us A.P. views of the lumbar spine and lateral views of the cervical spine. I should like to ask whether in the A.P. views of the cervical and the lateral views of the lumbar spine there were also some changes that may show this funnelling of the spinal canal.

*Harris, P.* (G.B.). I was very interested to hear Mr. Hancock's paper. I think he brought out many important points, and I would agree with him about the importance of the congenitally narrowed spinal canal. I would like to know more about his ideas of the aetiology of this. One can understand it in achondroplasia. I have had the same experience like himself in requiring sometimes to decompress the cord in these patients. He mentioned two types of cervical spondylosis and mentioned that sometimes an anterior operation might be indicated and sometimes a laminectomy. I would agree and would add that in certain of these patients a third type of operation is sometimes indicated —that is the situation where there is a diffuse cervical spondylosis and in particular the

patient with the syndrome of vertebro-basilar insufficiency. In this situation, I think the third operation I would suggest is one of simple fusion or indeed even simpler an acrylic fixation of the cervical spine.

*Hancock, D.* (G.B.). With regard to the question about radiography in spinal canal narrowing. Certain views were not shown in this paper because they do not demonstrate the condition as clearly. Yes, there are changes and they are a little bit more difficult to pick out. There is shortening of the pedicles in the lumbar region in the lateral view—again, to my eyes I find it very hard to find. And, of course, as the narrowing in the neck is, as it were, saggitally orientated the lateral view of the neck would give you a much better appreciation of this. So far as the aetiology of the condition is concerned, I think, it is due to premature fusion of laminae to pedicles to bodies, very similar to the aetiology of achondroplasia. Somebody asked me the question about intermittent claudication. Perhaps I should make it clear that we think at the moment that there are two sorts of this: the vascular one which everybody knows about and the neurogenic one which I think has a limited audience, but it is well to be aware of this neurogenic one in cases of intermittent claudication which are primarily referred to see a vascular surgeon but who finds no evidence of ischaemia. Then I think it is important to look into a neurogenic possibility.

*Sir Ludwig Guttmann* (G.B.). Dr. Wolman's paper on chronic ischaemia producing myelopathy is a very important subject indeed. It was mentioned last year in cervical injuries, when we discussed amongst other problems the point that in high cervical injuries, in particular as a result of compression of the cord in the area of the foramen magnum, we are sometimes surprised to find atrophy of the interossei which might lead to diagnostic errors of the site of the pathological process. The reason for this is that the blood supply of the spinal arteries is interfered with which might produce a more distal cervical syndrome. I would like to remind you that this has already been discussed quite a number of years ago in a very interesting and important paper by C. P. Symonds and Meadows.

Mr. Hancock's paper indicates a new problem in neurology and neurosurgery, and I think the follow up of Verbeist's works on claudicatio-like symptoms due to narrowing of the spinal canal is quite important. The X-rays Hancock has shown to us are really of great interest and I am sure that in future one will have to examine more carefully, when people come with rather diffuse symptoms, similar to claudicatio or otherwise, whether this is not produced by narrowing of the spinal canal.

The papers of Dr. Price and Dr. Yeo are also of very great interest. We are considering more and more the hormonal sequelae of spinal cord injuries, in particular in cases above T5 and, especially in cervical transection, where the whole splanchnic control is crippled. I think we have to consider here two stages of hormonal changes. There is one in the acute stage, and there is no doubt from systematic studies I have carried out with my colleagues here that cervical cases show very interesting biochemical changes. The other stage concerns the more long term observations in those patients with dysfunction of the cord bladder, sometimes erroneously called the neurogenic bladder. The distension syndrome of the bladder which I published in *Brain* in 1947, in co-operation with David Whitteridge, consists in most interesting effects of bladder distension on the whole autonomic system, resulting in a complete change of the whole vascular circulation in the body, initiated, reflexly by the vasoconstriction in the paralysed areas. This work was continued throughout the years, in co-operation with other colleagues, and the results were published in a series of papers, amongst them the effects on adrenal glands activity. In this connection the question arose whether the acute hypertension due to distension of the bladder or other internal organs (rectum, uterus) is a pure reflex phenomenon or whether it is primarily the result of irritation of the adrenal glands. There is no doubt whatsoever that in cervical spinal cord transection during the maximal effect of bladder distension there is a considerable increase of both adrenalin and nor-adrenalin in

the blood, but I have not come to the conclusion that the increase of these catecholamines is the primary cause of the hypertension. It just represents another effect of the reflex activity of the isolated cord. We have also examined the effect of posture on catecholamines in cervical cases—and here again we found changes, but they are not as clear as found due to bladder distension. There are definite changes when raising the cervical man from the horizontal to the upright position and these are particularly conspicuous when the hypotension produced ischaemia and the individual fainted. On returning the patient to the horizontal position there are opposite effects such as bradycardia as compared with tachycardia when the patient was vertical and also some slight rise in blood pressure and again changes of adrenalin and nor-adrenalin. These are points which need further examination, and here, of course, the study on the pituitary-adrenal relationship is of particular interest.