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Discussion

DR. F. W. MEINECKE (*Germany*). I would like to thank my friend John Yeo very much for his excellent paper, and ask a question. How would you explain the increase of an incomplete lesion as a result of oedema with the results you had in your experiments?

MR. P. HARRIS (*G.B.*). I also enjoyed Dr. Yeo's paper. I would like to ask a couple of questions. What is the length of period of spinal shock in the sheep that he uses? Secondly, in the experimental study of trauma in animals, how akin is this to the situation in man? We are dealing with a different type of injury altogether. Has he tried to simulate the situation in man? I think this is lacking in many, if not most, of the experimental studies which involve animals, so that it would be quite difficult to relate this, I think, to the human situation.

And thirdly, I am not certain, although I should know this from his previous work, if the dura is opened or not, because these animals do then start off with a decompressive laminectomy, of which, of course, I personally do not approve. Does this play any part in the situation?

PROF. M. WEISS (*Poland*). I also very much admire these experiments made in relation to the clinical symptoms by Dr. Yeo. This is a beautiful model for studying different techniques of treatment. Not to compare, of course, to the human situation, but as I feel he clarified much concerning the effects of steroids and other drugs, I would like to ask him if he tried local cooling, which probably can be a factor for terminating or diminishing local swelling.

DR. MASALAWALA (*India*). I think this paper needs to clarify the difference between a compression fracture and a fracture dislocation as occurs in the human being. A very small experiment was carried out in our Unit on cervical dislocation and fractures on patients who died sometimes after 48 hours, sometimes after three days, etc., etc. They showed that the damage in the cord was not exactly locally at the site of the injury but spread up and down in a sort of spindle shape, the maximal degree of damage being opposite the fracture but extending for two inches almost coming to a pinpoint above and below, which accounts for the bizarre clinical picture. Any model, such as Dr. Yeo has shown, does not include the factor of rotation and stress on the spinal cord as a whole, and therefore does not really compare with the injuries in a human being. But I have no doubt it will be useful for judging the effects of certain drugs merely in relieving swelling.

Another point I would also like to make is that the dura is a fairly strong membrane, and any swelling of the cord coming within must cause a certain amount of compression.

SIR LUDWIG GUTTMANN (Chairman). If there are no more questions or comments, I would like to make a few comments. I think what Masalawala has just said is very apt. All these experimental studies, however detailed they are, do not give a clear picture of what happens in human injuries. The factor of rotation in most of the fracture dislocations is, I think, the main cause of the widespread damage to the spinal cord. This, as I have pointed out before, is due to a stretching and squeezing effect on the spinal cord above and below the level of the lesion. Whether you have an antero-hyperflexion or a retroflexion injury (the latter called commonly hyperextension injury), the cord at the level of the injury is fixed. But above and below, whatever the injury might be, you may get a stretching (distraction) of the spinal cord by the movement of the body, and/or the head

as a result of the impact of the violence. I feel further experimental studies without opening the dura should take this mechanism into account. The histological pictures, as shown by Yeo, were really excellent, and what we have seen, in addition to central destruction of the cord, is really the effect of the injury on the developing of the oedema. Even in the case with the destruction of the greater part of the spinal cord, the white tracts on both sides were not completely damaged. That is where, of course, the recovery, even in a very severe injury, may occur.

I would conclude that these experimental studies are very important and should be continued, and I am very glad that this has now been taken up in Australia. I hope that these studies will be taken also up in other countries. Those members of our society who have been in Phoenix will remember that we had a very interesting discussion on the experimental models shown by Professor White, Dr. Campbell and the others, but the results were still rather inconclusive.

I now ask Dr. Yeo to answer the questions and comments made.

DR. J. YEO. Dr Meinecke's question: we have not really described the true pathogenesis of the oedema. I am not sure whether I understood your question correctly—just what the relationship of oedema and swelling is to the final pathology. I don't know whether oedema is important. We are looking at a means of measuring the degree of pathology. I honestly do not know.

Mr. Harris asked how the length of spinal shock varies between four hours and one week in the sheep—how akin to man? We have looked at our 550 cases since the Unit was begun in 1954 and we have had only nine cases where we have been able to do detailed of autopsies, varying from 24 hours to 22 months after injury. There is no doubt in our minds that what we look at in man—and Sir Ludwig referred to this—is essentially Wallerian degeneration. On those slides we showed you, it would be difficult if I said that this is the section above or below the level of the lesion. Bill Payne and I cannot usually tell. We are dealing with a vascular lesion in the sheep—in man you can tell very easily because Wallerian degeneration develops above and below the cord lesion in the appropriate way. So, I am quite sure that in man there is a direct injury which is the predominant injury. But, it is the vascular element that, we think, might be important.

Let me ask this question, if I may; how much white matter does a man require? If the sheep requires a rim to be able to have a 75 per cent recovery, how much rim of white matter does man require to have significantly functional return? The dura, Mr. Harris, was, as I said, opened, and in that sense we do a decompression laminectomy on all animals, both controlled and treated.

To Professor Weiss: we have not used local cooling nor have we used saline of normal body temperature, which, of course, also has had good results. Professor Masalawala; the rotation factors are very important and again we have removed the dura from playing any significant role in the pathological process. I have referred to Sir Ludwig's remarks and I would be interested, Sir, in your comments as to how much cystic change can you have before some significant recovery in man is possible? We may only require just a few millimetres of white matter to see that recovery. Paul Dollfus just asked me a question on the side, which is very important, and that is what of the toxicity of the drug we are using. It is said to be very toxic renal-wise but we have not seen any renal toxicity in the sheep in which we have used the drug. This hasn't been reported in the literature and we will set about doing this as soon as we get home. Alphamethyl-paratyrosine caused a marked massive diuresis, and it may well be that the disturbance of the haemodynamics in the region of the spinal cord might be the important factor. It is a very intriguing point and hasn't been described to my knowledge in the literature. This may be how Alphamethyl-paratyrosine works.

DR. MASALAWALA. May I just add one comment, Sir, with your permission? We have had a large number of cases with tubercular spine and some of them come with

very acute kyphosis. This is in reference to the point made by Dr. Yeo that he does not know how much white tissue is required to recover function. We have on a number of occasions seen these patients with extreme degree of cord damage and yet a fair amount of function has been produced for years. Progressively a further function is lost and in one of these cases which came to post-mortem, we examined the cord and found that the dural tube was only present, no cord tissue had finally been left. But it takes a long time for the cord to degenerate to such an extent that funcion is totally lost. And the same thing may be shown in cervical injuries that even minor amounts of white tissue remaining intact will allow a fair amount of function.

SIR LUDWIG. There is only one snag in this, Masalawala: that is you cannot compare the gradual compression in tuberculosis and spinal cord tumours with the acute impact of injuries. There is a great difference, which I have always emphasised. With regard to recovery, this again shows how important it is to wait for all conclusions and in particular also for all legal conclusions. We cannot make conclusions immediately—perhaps after one year or even after two or three years, especially in clinically incomplete lesions.