In the earlier experiments (three in number) the lymphocytes increased somewhat after the onset of rickets, while in the later ones (five in number) the polymorphs underwent the increase, but in two of these latter cases a bronchopneumonia was discovered after death, and probably accounted for the leucocytosis.

Thus experimental rickets is not necessarily accompanied by any anaemia, and when anaemia does appear it is, as a rule, of minor degree, and may frequently be accounted for by some complication. There may or may not be a leucocytosis, and this also when it occurs may be the result of some complication. It can at least be truly said that in the blood of animals suffering from rickets there is no marked pathological change and absolutely nothing characteristic.

It is thus seen that these experimental results are in entire accordance with those obtained in the cases of spontaneous rickets, and which I have briefly reviewed above. Of 30 children suffering from active and uncomplicated rickets, only 9 presented anaemia, in not one of which was the number of red cells less than 4,100,000 c.mm. As a rule, however, the blood estimations were abnormally high. Nucleated red cells were exceedingly rare. In only 2 of the cases was there an unequivocal leucocytosis, 9 presented a leucopenia, and 19 a slight increase in the number of white cells.

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THE TREATMENT OF SLEEPING SICKNESS:

A SUGGESTION.

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I HAVE read with much interest the account of the discussion on cerebro-spinal meningitis at the Sheffield meeting last August. More especially have I noticed the results obtained by Drs. McKenzie and Martin by their method of introducing locally by spinal puncture a highly immune serum which they obtained from the blood of patients who had recently recovered from the disease or, as in two successful acute cases, who were still running a course. These latter were injected with their own serum taken on the sixth and seventh day of their illness respectively.

It has occurred to me that a somewhat similar method of serum-therapy might be tried in cases of trypanosomiasis and sleeping sickness.

Although cerebro-spinal fever and trypanosomiasis differ very markedly—the one being a bacterial, the other a protozoal disease—yet there are certain points of resemblance between them. In both the organism producing the disease is found in the blood and in the cerebro-spinal fluid; at least, this is so in advanced cases of sleeping sickness. In both there is that "lymphatism" described by Westenhoeffer and Symmers in cerebro-spinal fever; and in both the mesenteric glands may show marked hyperaemia and haemorrhage. In both also the final stress of the disease falls on the cerebro-spinal system, and, though the one is an acute, the other a chronic disease, cerebro-spinal fever may run a very chronic course, producing an extreme marasmus which cannot be surpassed even in the closing scenes of typical sleeping sickness. At the same time this resemblance is of minor importance. The question is, If in sleeping sickness the cerebro-spinal canal is a *locus minimae resistentiae*, does the cerebro-spinal fluid show a marked lack of trypanocidal substances? So far as I can ascertain, this matter has not been fully investigated. We know that the blood of patients suffering from trypanosomiasis possesses trypanocidal bodies, and blood serum has been used therapeutically both by subcutaneous and intravenous injection. Why should it not be tried intraspinally?

In the past great difficulty has been experienced in attacking the trypanosomes present in the cerebro-spinal canal, and Kopke has made special efforts to overcome the difficulty when treating patients with atoxyl and other chemical substances. It is true that comparatively few cases of human trypanosomiasis recover, but a suitable serum might be obtained from the blood of patients subjected in the first instance to chemo-therapy. A combined treatment might prove useful, attacking the trypanosomes in the blood by some of the more promising of our present drugs, and attacking those in the cerebro-spinal fluid by performing lumbar puncture and injecting the patient's own serum, or the serum from a case which is improving, obtained by defibrinating and centrifuging the blood. The latest work on the cerebro-spinal fluid in sleeping sickness is that by Broden and Rodhain, but they do not approach the subject from this aspect. The cellular content of the fluid seems to vary, and Nattan-Larrier has reported a very severe case with cerebro-spinal symptoms in which the fluid was very poor in cellular elements. This may point to the canal being a *locus minimae resistentiae*, to employ once more the phraseology of Drs. McKenzie and Martin.

It would seem that, so far as the cellular contents go, the fluid in human trypanosomiasis may approximate to the conditions found in cerebro-spinal fever, a fact which is rather in favour of adopting the line of treatment indicated. I admit it is merely a suggestion, and one possibly based on none too stable a foundation; but in the present state of our knowledge, everything likely to prove beneficial should be tried in cases of sleeping sickness. I am sending a copy of this note to Sir David Bruce in Uganda for his consideration, and I propose to carry out experimental work in this direction here as soon as possible.

If the idea has been voiced beforehand, I can only plead the difficulty of getting at the full literature on the subject, and apologize for having, as one may say, "carried coals to Newcastle."

INJURY TO THE VESSELS IN DISLOCATION OF THE SHOULDER.

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THIS short account of a case of injury to the axillary artery and vein possesses considerable interest, serious injury to the larger vessels of the axilla in ordinary simple dislocation of the shoulder-joint being fortunately rare. When the vessels are torn local signs vary a good deal. In the less common variety a swelling appears a few days or weeks after the injury, and increases rapidly with pulsation; in the second kind a diffused fluctuating swelling forms immediately or within a few hours, without bruit or pulsation, and reaches a large size, pushing forward the pectoral muscles. If this tumour pulsates, the diagnosis of a ruptured artery may be made; if in addition the radial pulse is present, it is extremely probable that the injured vessel is not the main artery; beyond this it is not possible to go (Stimson).

E. M., a stout woman of 70 years of age, was admitted to St. Thomas's Hospital on November 26th, 1908. She stated that on the day before admission she had fallen down and hurt her left shoulder; previously she had been in good health. When examined in the casualty department, it was discovered that she had sustained a sub-coracoid dislocation of the shoulder. This was reduced by Kocher's method without anaesthetic. Immediately after reduction, a swelling appeared in the axilla, which increased somewhat rapidly in size; it was also noticed that the radial pulse was absent on that side. When seen in the ward on the same evening, 9 p.m., the swelling in the axilla was said to be larger, and the left radial beat was still absent. She was suffering from shock; the right pulse was small and rapid, 124, and the temperature sub-normal. There was, however, no proof that the swelling was rapidly increasing. On the following day the patient was placed under an anaesthetic and the axilla opened, both pectoral muscles being divided. A large tear in the axillary vein was found under the lower border of the pectoralis minor muscle. No wound was present in the artery, but all pulsation had ceased at a