

Serum dilutions—											
20	40	80	160	320	640	20	40	80	160	320	640
Culture X19; dried; formalin- ised; 2 weeks old.						Not dried; formalinised; young culture X19.					
R. H. typhus serum: 2 hours at 55° C.											
+++	+++	+++	+++	+++	++	—	—	—	—	—	—
Rabbit specific serum for X19.											
+++	+	—	—	—	—	+++	+++	+++	+++	+++	+

The addition of the formalin to the suspension of the dried cultures does not affect their sensitiveness to typhus serum agglutinins, though with undried cultures it has this effect. The drying of bacilli renders them much less agglutinable by specific agglutinins. I have found this to be the case for *B. typhosus*, *Paratyphosus A* and *B*, and *B. coli*; to this rule proteus X 19 forms no exception. The effect of desiccation in reducing the agglutinability of proteus X 19 by specific serum from a rabbit is shown below.

Dilutions of serum: 3 hours at 55° C.									
	20	40	80	160	320	640			
Living X19 .. ..	+++	+++	+++	+++	++	++			
Dried X19 .. ..	+++	++	+	—	—	—			

The effect of desiccation on proteus X 19 as regards its agglutinability by the agglutinins in typhus serum and by those in the serum of a rabbit immunised against the bacillus adds another point of distinction between these agglutinins.

So far no satisfactory explanation has been offered of the occurrence of these heterologous agglutinins in typhus serum. From November, 1920–April, 1921, I examined material from over 30 cases of typhus in Belfast. As in my former investigations, I found that in the urine of several of them coliform bacilli which were agglutinated by typhus serum, sometimes in dilutions of 1 in 640, whereas normal serum in 1 in 40 was without effect. These agglutinins developed in the course of the disease. From the urine of one case *B. pyocyaneus* was isolated in pure culture. A few of the serums as the disease progressed agglutinated this bacillus in dilutions of 1 in 160 and 1 in 320, although early in the disease even dilutions of 1 in 20 had no effect. These agglutinins for *B. pyocyaneus* were destroyed by heating for ten minutes at 65°–66° C., and absorption tests showed that they were distinct from those for proteus X 19. Kreuscher<sup>4</sup> also found *B. pyocyaneus* agglutinated by typhus serums.

When the serology of typhus is understood, the presence of agglutinins, not only for the various X strains of *B. proteus* but also for innumerable other bacilli will be accounted for. It may be noted that a strain of *B. proteus* which failed to liquefy gelatine has been found to be almost as readily and constantly agglutinated as the typical liquefying X 19. (Braun and Salomon.<sup>5</sup>) In none of the 30 cases did I succeed in isolating proteus X 19 from the urine.

Summary.

1. Proteus X 19 when rapidly dried in vacuo can preserve its agglutinability as regards typhus serum for months either at room or body temperature.
2. When emulsions are made of such desiccated bacilli they can be for some weeks preserved with 0.1 per cent. formalin without their sensitiveness being impaired.
3. These facts enable us to prepare a stable sensitive diagnosticum and to dispense with living cultures, the advantage being obvious in military service in the field and also in enabling us to use an emulsion of known sensitiveness. It is well known that proteus X 19 in the living state is apt to vary as regards its agglutinability.
4. Desiccation of bacilli renders them less agglutinable by specific serums, and to this rule proteus X 19 is no exception. It would therefore seem that the typhus serum agglutinins for X 19 are different from those produced in the blood of an animal by inoculation.
5. Coliform non-lactose fermenting bacilli are occasionally found in typhus urine and are agglutinated by the serums not only of the individual patient but of other patients.
6. On one occasion a strain of *B. pyocyaneus* was isolated which was agglutinated by a few of the typhus serums.

(Continued at foot of next column.)

THE EARLIEST STAGE OF SENILE CATARACT.

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OUR knowledge of the causation of cataract is hardly even in the stage of theory. Not one of the few theories advanced will stand the test of facts. How could it be otherwise? We know nothing of the nutrition of the lens or of the elimination of its waste in health; pathological anatomy up to the present time does not bear on this issue. Its extreme prevalence in certain localities—the plains of North-West India—and its relative absence in other parts of that country help us little. Where it prevails the diet is chiefly wheat, maize, and peas with fats; where it does not prevail to the same extent—as in the Himalayas, Bengal, Burma, &c.—the chief articles of diet are rice, maize, and peas. One might therefore say that wheat had something to do with it, but caution is required in the consideration of such a theory. Except in Burma the people chiefly belong to the Aryan race, so that there is no racial element in the matter.

It is clearly not a starvation disease in the ordinary sense of the term, because, in the areas most affected, the peasantry are physically the finest in the Indian Empire; there is no question of malnutrition or starvation amongst the peasantry of the Punjab. Undue exposure to glare (actinic rays) has been advanced as a cause, but this influence, again, is as great in non-cataractous as in cataractous areas.

Failing Distant Vision the Earliest Symptom.

The earliest symptom of senile cataract is *failing distant vision*. I think that I was the first to advance this observation. The patient's near vision with spectacles is good for ordinary purposes when his distant vision is reduced to less than one-half. This is a very noticeable fact to anyone in the Punjab who examines railway employees once a year. These men require vision of 6/6. Normal vision is much better than this. When distant vision falls to 6/9, 6/10, or even 6/12, nothing may be observed in the lens further than that it does not transmit light as well as a normal lens. You can observe this fact by having a normal case on one stool and one of these cases on another. On examination of the eye, including the retina of each patient, with an ophthalmoscope in the same light, it will be found that, while in the normal case every structure is clear, the structure of the retina in the early cataract case is easily made out, but the retina appears in the one as though the examination were in a good light and in the other as though the light were poor.

In these early cases black sand-like particles in the periphery of the lens are occasionally seen. In other cases you may see a few opaque striæ making their way from the periphery towards the centre. The centre in either case usually remains clear. These appearances are unusual unless the vision has been reduced below 6/10.

In subjects under 30 years of age the appearance of small oil droplets replaces the above-mentioned appearances. It is evident that the old descriptions of cataract as mature and immature refer only to the later stages of cataract—*immature* implying extensive partial opacity of the lens, *mature* implying complete opacity.

Treatment of the Early Stage of Cataract.

There must evidently be an earlier stage, in that cataract does not develop all at once. As regards treatment of this early stage, many years ago I had

(Continued from previous column.)

References.—1. Wilson, W. J.: Jour. of Hygiene, 1920, xix., i., 115. 2. Russ and Kirschner: Zeitschr. für Hygiene u. Infektionskrank., 1921, xcii., i., S. 38. 3. Vide review by H. da Rocha-Lima: Ergebnisse der Allgem. Path., 1919, xix., S. 159. 4. Kreuscher, A.: Berl. klin. Woch., April 22nd, 1918, S. 374. 5. Braun, H., and Salomon, R.: Centralbl. f. Bakt. Orig., 1918, lxxxi., S. 20

an Anglo-Indian patient, aged about 50, who had a very faint nebula in the centre of each cornea due to a recent ophthalmia. Ophthalmoscopic examination showed that she was also suffering from cataract at a very early stage. She told me that her vision had become useless for anything distant or fine. I gave her a subconjunctival injection of cyanide of mercury in each eye, which I believed would clear up the corneal nebulae, and asked her to write and let me know after some weeks the condition of both near and distant vision. She wrote to say that both had recovered and were as good as they had ever been.

It struck me, on thinking over the case, that this was impossible unless the hyperæmia (which I had induced) had also influenced the nutrition of the lens. In the railway class of patient and others who came to me I was able to see many cases of early cataract, and I treated them on the same lines, being agreeably surprised at my success. I tried cases at different stages and found that up to 6/10 we might expect to be successful in almost every case. Even 6/12 may recover to 6/6. But while the result in 6/10 and less lasts for years 6/12 or more has a tendency to recur. (I do not wish it to be imagined that dead tissue, such as sand-like opacities, or minute opaque striæ, when present, disappear.) I have had many cases under observation for years and am satisfied that the treatment is eminently satisfactory, both for the patient and for the surgeon.

#### *The Danger-Signal.*

As regards the future of the question, I hope that the general practitioner will learn to recognise that failing distant vision in people over 40 is the first sign of cataract, and that he would do well to send the patient to an ophthalmologist for investigation. If this is done in every case it seems to me that operations for senile cataract will in the future be less frequently necessary, and that it will be found that the early stage of cataract is more frequent in people over 50 years of age than is usually supposed.

Since I wrote my first paper on this subject I have learnt that eye-baths containing iodide of potassium and other agents have been tried over long periods with success. I have tried iodide of potassium subconjunctivally and have found that from 15 to 18 gr. to the ounce give an efficient hyperæmia, giving the same result as 1 in 4000 cyanide of mercury injection. My opinion is that the agent (dionin included) does not matter; the result depends not on the agent but on the hyperæmia. From my experience of malingerers who use nicotine to produce an ophthalmia I think that drops of nicotine solution would be as effective as dionin or more so.

Also, since the appearance of my first paper on this subject I have learnt that many men have tried this treatment; some have not found it successful, whilst others have found it satisfactory. Diagnosis must be carefully made; failing distant vision from other conditions than cataract is to be considered, and I suspect that the failures have been due to mistaken diagnosis.

By what mechanism does this treatment affect the nutrition of the lens, or the elimination of its waste, or both? If it be assumed that an atheromatous contraction of the channels conveying nutriment to the lens is the cause of cataract then results such as have been obtained for years—from mere dilation of these channels caused by a few weeks' hyperæmia—would not be expected. One would expect the atheromatous condition to recur rapidly, as it does in other structures. It seems to me that to explain these facts we must assume that a special pabulum for the nutrition of the lens is metabolised by some of the cells in the ciliary region, and that cataract is due to a pathological condition of these cells and to the pabulum issued by them, this pabulum being non-physiological and incapable of maintaining physiological nutrition of the lens.

#### *Conclusion.*

The results of the treatment would imply that the induced hyperæmia re-establishes the physiological

condition of the metabolising mechanism. I think the existence of such a mechanism will not be disputed; it is, of course, a matter of speculation, but the speculation has facts behind it. The influence of a hyperæmia in other regions of the body is beyond the domain of doubt, as is the influence of local blood-letting, which is very closely allied to that of artificial hyperæmia. The influence of local blood-letting over a painful and acutely congested liver is very marked. I have explored with a trocar a large and painful liver for abscess a number of times and failed to find one. Four or five ounces of blood escaped and I was agreeably surprised to find that the patient was quickly relieved of all his trouble and continued to be so.

The question of cataract opens up a wide field for the physiologist, and I hope that physiologists will devote more attention to the nutrition of the lens and vitreous than they have done in the past.

## Clinical Notes:

### MEDICAL, SURGICAL, OBSTETRICAL, AND THERAPEUTICAL.

#### A CASE OF

#### LOSS OF SPEECH, MEMORY, AND HEARING FOLLOWING INJURY: RECOVERY AFTER OPERATION.

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THE following case of loss of speech, loss of memory, and loss of hearing following an injury is of interest. The patient recovered after operation.

W. S. G., aged 21, was admitted to the Whittlesea Workhouse Infirmary as a certified lunatic on Oct. 21st, 1921. When I first saw him he was sitting in a chair and was perfectly quiet. I was unable to make him hear, but could converse with him in writing. He did not know his own name or the name of his father or the name of the street where he had lived in the town, and when his mother visited him he did not recognise her. On visiting days, however, he would write that he expected a friend; up to the time of his injury his memory was a blank. On his arrival at Whittlesea he did not recognise the town, the road, the workhouse, or any person. His vision was unaffected. He had some scars on his scalp and a definitely tender spot 2½ in. behind the right external angle of the frontal bone, 4 in. above the zygoma. I also learned that he had an injury preventing him using his right arm and was taught to sew and write left-handed. This injury cleared up and he was able to resume the use of his right arm.

I am indebted to Dr. J. H. Skeen, of the Fife and Kinross District Asylum, for the following communication dated Dec. 18th, 1921.

"While he was in the poorhouse here he was, I understand, X rayed, and the report, which I did not see, but heard of, was to the effect that there was a definite fracture at the point you mention, with depression. I mention this with some diffidence as I neither saw the plates nor the original report. I sent him to Dundee to be operated on, but as they insisted on an X ray report there, and as this took some time owing to their X ray expert being away for a few days, patient got rusty, kicked up a row, and, of course, the hospital would not keep him any longer, considering him a dangerous lunatic."

#### *Operation.*

On Nov. 5th I operated, turning down a flap making the tender spot the centre, and trephined the skull with a ½ inch trephine. I saw no sign of the fracture or depression which I had thought I could feel through the scalp. On removing the disc of bone a large branch of the middle meningeal artery crossed the opening which was occluded; no pulsation was visible or perceptible on pressing the dura. I next incised the dura with a free incision and was met with a gush of blood-stained fluid which I allowed to escape; the brain underneath was plainly visible, pulsating and normal. I closed the wound by continuous suture without replacing the bone, and no drainage was inserted.