SUMMARY.

1. *Bacillus typhosus* is present in the blood in all cases of typhoid fever in the second and third weeks, when the temperature is 102 or over.

2. Bacilli of the "enteric group" of bacteria (*Bacillus alcaligenes*) are present in a considerable percentage of the cases after the second week, at first accompanying *Bacillus typhosus*, then supplanting it.

3. Later in the disease cocci may be in the blood.

4. *Bacillus coli communis* is never present in the blood of typhoid patients.

METHYL ALCOHOL AMBLYOPIA, WITH SPECIAL REFERENCE TO OPTIC NERVE.

REPORT OF CASE.*

C. S. G. NAGEL, M.D.
SAN FRANCISCO.

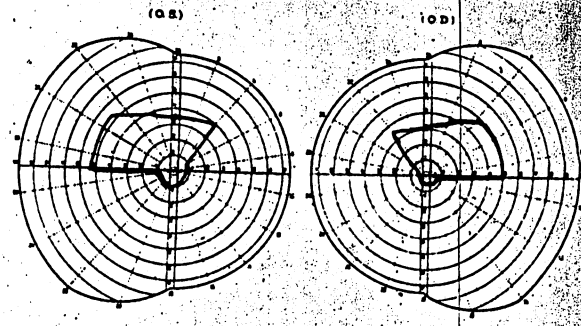
The very meritorious report of Drs. Buller and Wood[1] has absolutely established the specifically deleterious effect of wood alcohol on the optic nerve. Until anatomic findings may give us a better insight into the pathology, cases offering special clinical features seem to me to be worth recording.

Previous History.—It has been my good fortune to come across the patient in Case 80 of the report referred to and to have had this individual under observation for about a year. It is only necessary to recall here that early in October, 1901, this patient became absolutely blind for a number of hours through acute wood alcohol intoxication from imbibing; then a very considerable improvement had been present for some hours, after which blindness had set in again lasting for several days. After improving for a few months, vision had been, on a whole, getting worse ever since. It is noteworthy to quote from Dr. J. F. Van Kirk's report as follows: "This case was characterized by frequent transient changes. The vision would be markedly improved for a few hours and then it would suddenly fail. Often the morning vision would be good, while exercise seemed to increase the amblyopia." The patient further stated to me that he always used to see much worse temporarily whenever discouraged about failure to secure food. He had also had occasionally "fullness of head" and attacks of pain over the head, which felt "tight" from forehead to top, but not farther back.

Examination.—When I first had occasion to examine him (March, 1904) vision was R. 5/15; L. fingers in 0.65 m. Anterior chambers were normal o. u. Left pupil was dilated. T. normal o. u. In each pupil there was slow minimum reaction to light. R. F. V. extremely contracted, from 50 degrees outward over ca. 50 degrees outward and upward, 30 degrees upward and 35 degrees inward—up to 5 degrees for the remaining 5/8 of the field. In other words, over ½ of total F. V. is missing, below and inward, and upward and outward F. V. is contracted. There are small fields for blue, red and green and no central scotoma. L. F. V. is analogous, though uniformly a very little wider, but color perception for blue only. The striking point regarding the fields is the similarity as to the position in each of the parts intact and missing.

Corpus vitreum normal o. u. Ophthalmoscopically, the disc in both eyes is white (atrophic), with well-marked lamina cribrosa, outlines sharp, arteries a very little contracted. There is a halo around each papilla. Each disc shows glaucomatous cupping upward. To do away with one other point, I will say that for some time, during the beginning of my observation, there were apparent, at times, two or three discolored small roundish spots in the macular region of right eye which had created in the mind of another casual observer the impression of impending anatomic changes. During my long observation I have become convinced that such is not the case, and that the changes are confined in each eye to the disc, to its immediate neighborhood and to the vessels.[2]

Treatment and Result.—For reasons which will become clear from the epicrisis, I promptly performed a broad peripheral iridectomy in both eyes. For a short time only (ca. 2 weeks) after operation both pupils became a little smaller. The ophthalmometer showed R. + 1.0 axis 60 temp., L. + 3.0 ax. 75 nas. In the right eye vision remained about the same, in the left eye there was a decided improvement. Fingers were now counted (one week after operation) in 1.0 m., and 3 weeks p. o. in 1.25 m. These results of repeated examinations became the more convincing (as generally in cases of extreme amblyopia) because the patient, in trustworthy statements, had repeatedly spontaneously maintained improvement of vision from his observations in general. As a result of repeated perimetric examinations there also seemed to be a slight improvement in both F. F. V. peripherally. Such was the status to about the middle of April, 1904. Soon afterward, however, and gradually more and more pronouncedly, a decided deterioration set in again. In R. F. V. there soon remained but a small field for blue (only), and besides a perception of green could be found occasionally paracentrally. The left eye has kept a still smaller field for blue up to the end of my observation. F. F. V. could only be taken with finger fixation from about August, 1904. In the beginning of November, 1904, the patient was taken to the City and County Hospital, where a careful general examination by Dr. Schmoll revealed nothing abnormal except induration on one pulmonary apex. The patellary reflexes were rather exaggerated, no Romberg. Urine was normal. While, unfortunately, I find nothing before in my notebook regarding the point, the corneæ are now of subnormal sensibility to touch. Only



fingers are counted in each eye in about 1.0 m. Left pupil was dilated. T. normal o. u.

On November 16, sympathectomy of the left superior ganglion was performed by Dr. Stillman, under chloroform-anesthesia. In taking up the ganglion and at the moment of cutting, a slight pupillary play was noted, there was no marked dilatation. Both pupils were somewhat contracted while the patient was fully under the influence of the anesthetic. Postoperative tension was a little subnormal. Ten hours after operation both conjunctivæ bulbi were congested. L. T. is still subnormal (decidedly less than R. T.). Patient complained of some trouble in swallowing.

L. Pu. < R. Pu. (before operation opposite!).

Nov. 17, 1904; L. conjunctiva bulbi injected (not R.). L. Pu. < R. Pu. L. T. < R. T.

Nov. 19, 1904: L. T. < R. T. though a little higher than so far since operation. L. Pu. < R. Pu.

Nov. 24, 1904: L. T. < R. T. L. Pu. < R. Pu.

Nov. 28, 1904: Stat. id. left eye better sensibility than R.

Dec. 9, 1904: L. Pu. < R. Pu. L. T. Pu. < R. Pu. both physiologic. L. F. V. somewhat wider.

Dec. 12, 1904: Sympathectomy of superior ganglion o. d. (Dr. Stillman); chloroform narcosis. There was no change in pupil during operation. Pupil contracts with excision of ganglion. Some hours afterward R. T. < L. T. R. Pu. < L. Pu.

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1. Buller and Wood: "Poisoning by Wood Alcohol," The Journal A. M. A., Oct. 1, 8, 15, 22, 29, 1904.

2. Koller in Med. Record, July 1, 1905. In a report on wood alcohol poisoning states the same observation, comparing this evanescent feature to *chorioiditis guttata*.

Jan. 7, 1905: T. normal o. u., R. a little < L. R. Pu. a little < L. Pu. Vision to patient seems slightly better," measurably stat. id. Ophthalmoscopically, stat. id. R. F. V. somewhat better.

Whether this improvement (real or apparent) has kept up, I am unable to say, having been on a trip abroad since.

Before entering on a discussion of the special points of interest in the foregoing history, I should like to express briefly my belief that wood alcohol amblyopia sets in at first through a defective local blood supply consequent on disturbances in the general circulation, as they find their clinical expression in weak pulse, slow respiration, sweating, coma, etc. This, at least, seems to me the inference to be drawn from those cases in which a very prompt great improvement of vision had taken place, after the first, often very sudden attack of blindnes, to be followed again by fresh failure of sight gradually leading to optic atrophy. As we know from the experiments of Pohl,[3] who found in the urine the maximum of formic acid, as the product of oxydation of wood alcohol, only after from three to four days, a cumulative effect of the poison takes place the reason, I take it, for the second (eventual) attack of grave amblyopia. The first attack of blindness would then be due anatomically to what Alfred Graefe[4] has described as *Ischæmia retinæ*. It would be of great interest, of course, if ophthalmoscopically contraction of blood vessels could be found during the attack.

Since the prognosis is so very bad, I should advise prompt performance of iridectomy or at least keratomy, in order to lower the intraocular pressure, in acordance with the good results obtained by thus increasing the intraocular circulation in Graefe's and Knapp's[5] cases of ischemia of the retina.

Neither do we know at present how to discriminate between cases in which the first attack of blindness might be overcome spontaneously, and those in which it will not, nor can anyone doubt the lowering effect on the optic nerve of the lessened blood supply, thus predisposing it for the specific effects of methyl alcohol.

If there is any foundation for the theoretic reasoning, I now beg to submit, in conclusion, that a course of procedure, as advocated, might also have a preventive effect in that regard. It is, of course, scarcely necessary to point out expressly that I did not perform iridectomy with any lingering thought for therapeutic effects as considerd so far. As those I have been able to interest sufficiently will have noticed, I had become convinced that in this case a simple glaucoma had developed. My reasons therefor are as follows:

1. The great similarity in both F V, which would be very unusual in optic atrophy pure and simple, and the missing parts being the inner ones.
2. Glaucomatous cupping of discs.
3. Insensibility of corneæ.
4. Indication of halos around papillæ.
5. History of pain (cf. above).

Neither would the absence of (observed) increase of tension necessarily be against the diagnosis of glaucoma simplex, nor am I, on the other hand, indeed, unmindful that the positive symptoms enumerated may not be absolutely convincing to every one, though they have tended to become so to me in this case from the extended observation. I should like to add that the late appearance of central color scotoma in R. E. seems to be rather in favor of the idea of a glaucomatous process complicating the pathologic changes in the optic nerves. Regarding the glaucomatous cupping, I have to add that Leber[6] states that exceptionally the atrophic excavation may assume a form that can not be differentiated from the pressure excavation in glaucoma simplex. Also Stelwag[7] makes the statement that in atrophy of the optic nerve the lamina cribrosa sometimes loses in resistance to such a degree as to recede (*nach hinten weichen*) and thus a deep cupping comes about, in exceptional cases an excavation even with steeper margins, such as develops constantly in glaumoca. Of greatest interest is the history given by Schmidt-Rimpler,[8] which supplies the anatomic proof for such views.

Without any other convincingly glaucomatous symptoms there was found in his case a typical pressure excavation in both eyes. Won Graefe's diagnosis was glaucoma simplex.

Before iridectomy, which had been decided on, could be done, the patient died of pneumonia. Postmortem examination showed both optic nerves throughout their whole course attenuated and flattened. Microscopically there was pronounced atrophy of the nervous fibers, which could be traced still beyond the chiasma into the tractus. The left *papilla optica* (hardened in Mueller's solution) was found to be pushed backward together with the lamina cribrosa. "Here, then, was an excavation showing absolutely the picture of glaucoma and yet being solely the result of the atrophy of the nerve."

Irrespective of the true place of T. P.'s case in reference to the excavation, it is of some value, I think, insofar as it is apt to make us ask whether so-called glaucoma simplex might not come about sometimes under normal intraocular tension if the optic nerve or lamina cribrosa has lessened resistancy. This question has repeatedly suggested itself to my mind when dealing with cases of glaucoma simplex in which there were absolutely no typically glaucomatous symptoms except a "pressure" excavation. In conversing about the point with my former chief, the late Professor Pflüger, whose mind was particularly trained to approach ophthalmologic questions from the standpoint of the physicist, he readily conceded its feasibility. Although Schmidt-Rimpler, as well as Leber,[9] refer in passing to the importance of the relative resisting power of the lamina cribrosa in the formation of a glaucomatous cupping, I know of only one reference in literature having a direct bearing on the point raised.

"Impossible, however, it is not that there are cases where, under the influence of the normal pressure, together with primary inflammation or weakness of opticus, the excavation takes place."[10]

The anatomic discoverer of the glaucomatous cupping of the disc, Heinrich Mueller, discusses in full in his classical treatise[11] the several possible modes of origin for the various forms of excavation of the disc, without, however, referring specifically to the formation of a glaucomatous cupping through normal pressure. He states that normal tension with lessened resistance may "modify the place of entrance." Mueller then refers to Donders as having verbally laid great stress on this

3. "Ueber die Oxydation von Methyl u. Aethylalkohol im Tierkoerper," Arch. f. exper. Pathol. etc., vol. xxxi, p. 281.
4. Arch. f. Ophth, viii, 1, p. 142.
5. "Erblindlng durch Netzhaut-ischaemie, etc." Arch. f. Augen u. Ohrenhlkd., v. p. 203-207.
6. Graefe-Saemisch Handbuch d. Augenhlkd., 1. ed. vol. viii, pp. 853-4.
7. Lehrb. d. prakt. Augenhlkd., 4. Aufl., p. 228.
8. Arch. f. Oph. xvii, 1, p. 117.
9. S. Graefe Saemisch, v. 1. ed.
10. Schonte u. Koster: Ergebnisse d. Allgem. Pathol. etc. d. Auges von Lubarsch u. Ostertag, p. 403, 1903.
11. "Glaucom und Excavation der Sehnerven, in Gesammelte Schriften, etc.," edited by Otto Becker, Leipzig, 1872.

point. Von Graefe[12] says "if we imagine the substance of the optic nerve loosened (*gelockert*) through some internal process the papilla will give way to the normal pressure in an anomalous way." Later it was Schweigger who in 1891 (cf. Horstmann) insisted that cupping of the disc to the margin may exist without the least demonstrable increase of tension, and that "such a condition might arise from any atrophic disease of the optic nerve when there was previously a physiologic cup." Neither Schweigger nor Horstmann[13] refer to the normal intraocular pressure as the direct cause for such "glaucomatous" cupping in apparently non-glaucomatous eyes.

Schnabel's[14] views regarding true glaucomatous cupping as due to disintegration (Zerfall) of the optic fibers with formation of caverns, and "the glaucomatous excavation is one of these caverns"—are beside the point raised and are merely quoted for completeness' sake,[15] so are also Mueller's[11] conclusions that excavation—in general—might sometimes also be brought about by traction from without through shrinking exudate, etc.

Clearly the point is not of merely anatomic interest; if we could discriminate in glaucoma simplex without increase of tension between cases in which the "glaucomatous" cupping is due solely to simple atrophy, i. e., of fibrillæ and finer septa, and those in which it is the result directly of normal pressure on diseased or congenitally less resistant tissue, it would seem that the vexed question concerning the therapeutic effect of operative measures in glaucoma simplex would become clearer.

DISCUSSION.

DR. E. V. L. BROWN, Chicago, said that the pathogenesis of glaucomatous cupping in optic atrophy was much discussed at last year's meeting of the Vienna Ophthalmologic Society. The position was taken that this does occur in a great many cases without any tension whatever, and is due to the atrophy in the nerve head. Just back of it are found extensive areas of atrophic nerve fibers. Schmidt-Rimpler has also reported some observations on this point.