Dr. Bader introduces the correspondence by an interesting essay of 82 pages on the development of ophthalmology during the 18th and 19th century. He stresses in particular the obstructionist attitude of the surgical clinics in the struggle of ophthalmology for independence during the first half of the nineteenth century. Some interesting sidelights on Swiss ophthalmic history are also brought out.

CORRESPONDENCE

OCULAR CONDITIONS IN DIABETES MELLITUS

To the Editors of THE BRITISH JOURNAL OF OPHTHALMOLOGY.

SIRS,—I have read with great interest Dr. Gray's paper on Ocular Conditions in Diabetes Mellitus and I hasten to congratulate him on the wealth of material, the minute and careful examination, and on the result achieved. As a fellow worker in the same field, may I be allowed to make some remarks?

In spite of the tremendous advance of the Insulin Era, the real aetiology of Diabetes Mellitus is, in my opinion, still unknown. One important factor is missing, that of the disturbance of cellular metabolism. As pointed out by Dr. Gray and many other writers, this disorder of cell metabolism would explain the fact that in many cases the retinal exudate is not accompanied ophthalmoscopically by degeneration of the retinal vessels. The predominance of capillary degeneration is sufficient to explain the peculiarities of diabetic exudates and haemorrhages. The same, or perhaps another missing factor, eventually produces, as a rule, an arterial hypertension. It is seldom as pronounced, or as advanced as in essential hyperpiesis and correspondingly the sclerosis of the retinal vessels is not so marked. In diabetes the sclerosis of retinal vessels is a phase intermediate between senile and hyperpietic changes. Neither Dr. Gray nor myself have yet seen diabetic retinitis in young persons or children.

I should like to make some criticism on the following three points in this important paper.

1. Historically, Marcus Gunn was the first to describe sclerotic retinal vessels and, although Dr. Gray follows Gunn's enumeration of symptoms, he omits to mention a remark of the utmost importance: "That the loss of translucency is the first and earliest pathological change." After Gunn, chronologically follows, not Foster Moore (much as I admire his work), but Röelman and particularly Hertel.

2. Some mistakes, probably printer's errors, have been made in the tables. All figures of retinal arterial pressure are printed in reverse order, *i.e.*, diastolic instead of systolic pressure and *vice versa*.

3. It is rather a pity that in cases of changes in the peripheral

vessels (presumably of the lower limbs) with gangrene, no oscillometric measurement was done. If the oscillometric index were normal as near to the affected part as possible, probably the gangrene would be due to capillary degeneration. If the index were minute or there was no movement of the needle at all, it would mean obliteration of the main arterial branch. These particulars would be of special importance, as Gray rightly states, in the absence of any definite retinal vascular sclerosis. A point always difficult to decide is the distinction between a congenital and a pathological anomaly. This especially applies to arterio-venous compression in its protean forms. With certain technical means this difficulty, in my opinion, might be overcome if the observer paid special attention to the very fine white lines accompanying the artery and crossing the vein at the point of intersection of both vessels. These white lines increase with the degree of vascular degeneration and are best seen in the "hour glass" compression of the vein. Gray mentions that Gunn first described them. Quite so, but only in normal vessels. (Adams Frost describes this also.) As an important pathological sign they were not described, as far as I know, until 1929.

The translucency of the retinal vessels is of such a delicate quality that it may sometimes disappear within a few hours, as from any infectious disease, such as, severe tonsillitis. Therefore one cannot say that in diabetes there is a "loss of translucency and perhaps some cutting of one of the lesser veins by the super-imposed artery." This is seen in 99 per cent. middle-aged normal people. This is why one sometimes wonders what is meant when in clinical histories some fundi are described as "normal," in which case the translucency of the retinal vessels should still be present. The description of arteries as "somewhat straighter than normal," especially in patients with high blood pressure, seems to me rather unusual, as the description of all other writers points rather to increased tortuosity. Magitot-Baillart's ingenious method is not easy to master. Dr. Gray has certainly mastered it to perfection, but he states that the first pulsations indicate diastolic pressure. It would take us too far to go fully into retinal arterial pressure, but the first pulsation probably indicates not the diastolic, but the mean pressure. Lately, Magitot expressed this opinion also. Measurements of mean brachial pressure are completely absent in Gray's paper.

In 36 cases declared to be hyperpetics the systolic blood pressure alone is noted. The term "intra-ocular pressure," as measured by Schiötz tonometer, instead of "tension" is probably a *lapsus linguae*, because "pressure" can be measured only by the method of Magitot and Baillart.

Yours faithfully,

N. PINES.