and this is true even when so disastrous an event as cerebral apoplexy is the commanding fact in the clinical picture. Thus it seems safe to say that retinal haemorrhages are not produced by the "bursting" of healthy blood vessels—or for the matter of that of degenerated blood vessels—under the stress of increased intravascular pressure, even allowing—what is a disputed point—that a high reading obtained from the brachial artery means increased blood pressure within the retinal vessels. Retinal haemorrhages, as I have already argued, mean, indeed, rupture of vessels, but the essential cause of this rupture is, not increased intravascular stress, but degenerative changes in the vessel walls. Support for these propositions is found in yet another group of cases—namely, those in which there is manifest arterial degeneration without high sphygmometer readings; for in such cases retinal haemorrhages may be present—may be present, that is, when, at least so far as the sphygmometer can be trusted, the blood pressure is not abnormal. In other words, high sphygmometer readings—or, if you will, high blood pressures—do not *per se* involve vascular rupture, while vascular disease may lead to rupture even when the alleged measurement of blood pressure is well within normal limits. It is change in the value of the vessel wall that is the necessary preliminary to rupture and haemorrhage, and this change either may or may not be associated with a high sphygmometer record.

Perhaps here I may be allowed a word in appreciation of the work done by ophthalmic surgeons on the pathology and clinical evidences of degenerative disease of the blood vessels. It has helped towards the solution of not a few obscure problems.

Of retinal haemorrhages in glycosuria there is little to be said. They are found, as everyone agrees, not in the typical diabetes of the earlier years of life but in middle-aged or elderly persons with slight or moderate symptoms and non-extreme degrees of glycosuria. They may or may not be accompanied by retinitis. When, as sometimes happens, the glycosuria disappears for a more or less prolonged interval the ophthalmoscopic picture may be a very puzzling one for an observer ignorant of the patient's earlier history. Upon prognosis in glycosuria retinal haemorrhage as such exerts little or no influence; the haemorrhage is quite consistent with active life continued during many years.

In the course of our discussion we are likely to hear of the differences—the diagnostic differences, as some affirm—between retinal haemorrhages in glycosuria and retinal haemorrhages in albuminuria. I admit the interest of the comparison as a subject for ophthalmoscopic contemplation and as an opportunity for the cultivation of the sporting spirit in the art of diagnostic adventure. But the observation has little clinical value. At best the conclusion suggested is a probability, and a probability which no one will say is invariably justified by the event. Moreover, the procedure is an offence against a sound and sensible rule—important in medicine as in law—that second-rate evidence ought not to be accepted when first-rate evidence is available.

I have now briefly to refer to certain clinical situations in which the discovery of retinal haemorrhages has a high practical value, inasmuch as such a discovery may save the practitioner from error and may indicate the route along which an accurate diagnostic conclusion is to be sought. Experiences of retinal haemorrhages as the first clinical event in renal disease, in glycosuria, and in arterial degeneration, are obvious illustrations of this proposition. A few others may be mentioned.

First, a doubtful or ambiguous appearance at the optic disc will be confidently interpreted if the suggestion of a possible commencing optic neuritis is accompanied by one or more small linear haemorrhages. They may require close observation, but once detected there can be no question of their significance.

Next I mention septicaemia and septic endocarditis. Quite a number of such cases are clinically, and for a more or less prolonged period, cases of unexplained temperature—fever without physical signs; and such diagnoses as tubercle, enteric fever, paratyphoid, and *Bacillus coli* infection of the urinary passages come up for discussion; or, again, with the fever are joint pains, and acute rheumatism offers itself as an appropriate label; and once more, in a patient known to be the subject of a chronic valvular lesion occurs an outbreak of febrile temperatures, and a recrudescence of active rheumatism is a tempting explanation. Yet in every one of these instances an ophthalmoscopic examination may promptly and confidently alter both the diagnostic position and the prognostic outlook, for retinal haemorrhages are not to be expected either in tuberculosis, or in enteric fever, or in paratyphoid, or in *B. coli* infections, while in septicaemia and in septic endocarditis they are quite frequent events.

Another disease in which the presence of retinal haemorrhages affords much help is pernicious anaemia. Here again, though rarely I admit, the problem presents itself in the form of unexplained pyrexia, and the anaemia and weakness are readily regarded as consequences of the high temperature, and for this an explanation is sought in the directions already indicated; the detection of retinal haemorrhages in such circumstances is of great practical value. On the other hand, a more or less severe and prolonged anaemia may be attributed to chlorosis, to dyspepsia, to malnutrition, or to some other more or less indefinite influence. The recognition of retinal haemorrhages here ought to sound a note of alarm, for such haemorrhages are frequent in pernicious anaemia, whereas in chlorosis and in secondary anaemias they are either absent or, if occasionally present, are slight in degree, few in number, and confined to extreme and advanced cases. Even a wider generalization may be ventured—namely, that retinal haemorrhages in the absence of renal disease, of glycosuria, and of arterial degeneration mean either sepsis or one of the so-called blood diseases, and particularly pernicious anaemia and leukaemia. Rarely they are due to malaria, and very rarely to syphilis; personally, I have not seen them in haemophilia or in Hodgkin's disease, and only once in purpura haemorrhagica. Retinal embolism and thrombosis of the retinal vessels perhaps ought to be mentioned, but in each of these there are in the history and in the ophthalmoscopic examination characteristic facts which reduce the attendant haemorrhages to a purely subordinate position. In addition, the generalization just proposed is qualified by the "unexplained" haemorrhages to which reference has already been made, and by the necessary remark that propositions which pretend to absolute values in the field of clinical medicine, however serviceable for general guidance, are apt now and again to suffer in their dignity by the arrival of events and combinations which contradict them.

In a congress of ophthalmic surgeons it would be gratuitous to insist on the importance of examining each fundus oculi, and yet it may be well to remark that even in so generalized a disease as pernicious anaemia retinal haemorrhages may have, at any rate for a time, a purely unilateral distribution.

CONCLUSIONS.

Finally, I submit the following practical conclusions—namely:

1. That retinal haemorrhages may exist without recognized prejudice to vision, and ophthalmoscopic examination is therefore a necessary part of every clinical examination.
2. That such haemorrhages may be the first objective signs of serious disease, and a discovery of them, therefore, demands a complete examination of the patient.
3. That the recognition of retinal haemorrhage is often of high value in directing the observer to a correct interpretation of the clinical facts, while the prognostic significance of the observation *per se* is indeterminate.
4. That retinal haemorrhages do occasionally exist as isolated clinical facts, and when so existing are comparable to haemorrhages in other parts of the body (haematemesis, haematuria, haemoptysis, etc.), for which no ready explanation is at hand.

A METHOD OF REDUCING CONGENITAL DISLOCATION OF THE HIP.

BY

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REDUCTION of a congenital dislocation, except in very young children, is not always the simple matter described in the textbooks. When the ordinary methods of pulling, lifting, and levering are employed the operator works under mechanical difficulties, and, unless well equipped physically, is likely to fail. So much energy is expended in pulling that proper control over the movements of the head of the femur is lost, and if the help of an assistant be utilized there is often want of complete co-ordination between the manipulators. In any case much energy is wasted. The method here described appears to me to avoid misdirected effort and

to put the movements of the head of the femur completely under the control of the operator. I can find no procedure exactly like it mentioned in the literature, except perhaps Hoffa's "pump-handle" method, but the idea seems so simple that it must have been tried before. Even if it is not new, I can claim to have discovered it for myself.

It requires some strength to pull the head down to the level of the acetabulum, and more strength to maintain it there. It occurred to me, therefore, that it might be much easier to push than to pull, and that if I could, by downward pressure of one hand on the fully flexed knee, push the head below the ill-marked lower rim of the acetabulum, I might with the other hand guide the head into the socket. I was surprised at the control so gained, and at the amount of downward pressure possible.

The method adopted for the left hip is as follows: The patient's sacrum may be placed on a sandbag; the pelvis on the right side must be steadied by an assistant. The knee and hip are flexed, the latter as fully as possible till the knee lies as nearly as can be managed beside the trunk. The right hand of the operator is put upon the patient's left knee and has two functions—first, by downward pressure to push the head towards the acetabulum, and, secondly, by rotation at the knee to direct the movements of the femoral head. The palm of the operator's left hand is placed against the great trochanter with the thumb in front and the fingers on the neck of the femur behind. While downward pressure is maintained with the right hand to keep the head of the femur as low as possible an attempt is made, by lifting with the fingers of the left hand, and depressing the right hand with or without rotation, to put the head below the acetabulum. A little time and some preliminary stretching of the tissues below the acetabulum may be needed, but no great force. The next manœuvre consists in maintaining the forward position of the head while gradually relaxing the pressure on the knee, when the head sinks noiselessly into the acetabulum, often at the first attempt. This done, external rotation at the hip-joint brings the limb into the usual Lorenz position. The thigh should finally be approximately at right angles to the body, and the knee more dorsally placed than the pubic symphysis, so that the head of the femur is fairly prominent in the groin. Reduction should be repeated a few times, noting the degree of prominence of the posterior and superior margin of the acetabulum by the "shock" of redislocation, and the amount of movement possible at the hip-joint without producing redislocation. It will often be found at this stage that reduction has become possible by the more usual method of lifting the head of the femur over the posterior lip of the acetabulum.

The considerations just given appeared at first sufficient in themselves to account for the success of the manœuvre, but an additional explanation of the comparative ease of reduction by this method was afforded by a short but important paper by Mr. A. H. Tubby, published in the *Lancet* of December 20th, 1919, describing an open operation for refractory cases. He emphasized the fact that in older children the ilio-psoas tendon is, after the adductors, the greatest bar to reduction, because it produces a virtual, and in late cases an actual, narrowing of the capsule of the hip-joint where the tendon crosses. This constriction prevents the head from passing through the "button-hole" in the capsule and lodging in the acetabulum. The tendon divides the capsule of the dislocated hip-joint into two communicating cavities—an inner one, of which the acetabulum is a part, and an outer one containing the head of the femur. Full flexion in my method relaxes the grip of the tendon; downward pressure puts the head of the bone to the acetabular side of the relaxed ilio-psoas, and, unless there is real narrowing, relaxation of downward pressure allows the head to enter the acetabulum without the intervention of the folded capsule of the hip-joint. It is important to remember that the success of reduction by this method does not necessarily obviate an open operation, because when the leg is extended the tendon again becomes tight, perhaps sufficiently so to cause redislocation. But that point does not apply to the more limited purpose of this paper.

I have found this method of the greatest value in neglected cases of congenital dislocation. It renders unnecessary long-continued extension of the limb and exsection of portions of the adductors and other muscles. In younger children it may avoid a preliminary division of the adductors. Hence I commend it to all who have encountered difficulties in reduction by the well-established methods.

FRAGILITAS OSSIUM ASSOCIATED WITH BLUE SCLEROTICS.

BY

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[*With Special Plate.*]

THE publication of Dr. J. B. Alexander's paper on "Fragilitas ossium associated with blue sclerotics" in the BRITISH MEDICAL JOURNAL of April 29th, 1922, leads me to report the following series of cases:

Case 1.—L. G., a girl with blue sclerotics, aged 7 years, has been treated in this hospital on different occasions for ten complete and two greenstick fractures. She was first admitted in 1917 when 2 years old, suffering from a fracture of her left femur, and since then has been treated for the following fractures: right femur, six complete and one greenstick; right humerus, two complete; right tibia, one complete and one greenstick.

Case 2.—T. McV., a boy with blue sclerotics, aged 2 years, half-brother of L. G. (Case 1), is at present in hospital suffering from a fracture of his right femur.

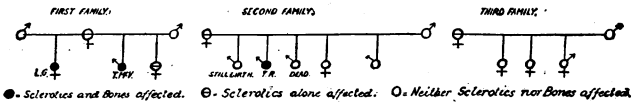
Case 3.—T. R., a boy with blue sclerotics, aged 7 years (cousin of L. G. and T. McV.), has had six fractures of his leg bones. In 1917, when 2 years old, he was admitted to this hospital with his right tibia and fibula broken. Since that time he has been treated for the following fractures: right femur, two complete; right tibia, one complete; left tibia, one complete; right fibula, one greenstick.

These three children are poorly developed and have prominent foreheads. The only other member of the family to which Cases 1 and 2 belong is a female infant, aged 5 months. She also has blue sclerotics.

The broken bones united at about the normal rate. Union was firm, though callus formation was scanty. *X*-ray photographs showed the cortex of the bones to be excessively thin and the medulla somewhat diffuse. The fathers of these children showed no apparent abnormality.

The mother of L. G. and T. McV. (Cases 1 and 2) has blue sclerotics and has suffered from partial deafness since childhood. She gives a history of having had her right femur fractured on two occasions. The first fracture occurred when 2 years old, and the second two months later. She has seven sisters and two brothers, all younger than herself. None of them suffer from undue fragility of their bones. The three eldest of these are females, and have blue sclerotics. The second member of the family is the mother of T. R. (Case 3).

The relation of these individuals to each other is shown in the following tree:



The points of interest in this series of cases are:

1. The four eldest members of the family have blue sclerotics; the children of the two eldest suffer from fragilitas ossium and blue sclerotics, while the children of the third are apparently normal. The fourth sister, who died some years ago, was unmarried.
2. The great preponderance of right-sided fractures.
3. The leg bones are chiefly affected.
4. The first fracture in each case occurred at 2 years of age.
5. Only one case of deficient hearing.
6. All cases with brittle bones have blue sclerotics, but several cases with blue sclerotics have apparently normal bones.

I am indebted for access to these cases to Mr. S. T. Irwin and Dr. R. J. McConnell, under whose care they have been.

BEFORE the war the birth rate in Germany and the natural increase—that is, the excess of births over deaths—were greater in Germany than in this country. The death rate also was higher. The *Morning Post* has recently published some interesting statistics of the German rates in the second quarter of this year. For the forty-six great towns of Germany the birth rate was 18.5, whereas in England and Wales it was 22.2. For the same period the death rate in Germany was 13.5, and in this country 12.7. The rate of annual increase in England and Wales in the period 1904–13 was 11.2; in the second quarter of this year it was 9.5. In Germany before the war the rate was 15.1, but in the second quarter of this year it was only 5.