side, however slight, I now invariably fix both sides at the same time.

Another cause of incomplete cure is fixation too low in the loin, so that the kidney is at an abnormal angle in relation to the ureter. This interferes with proper drainage, and places the kidney in the angle of flexion of the body which is often the cause of continued symptoms.

After fifteen years of careful study of over three hundred cases, and fitting and adapting various kinds of belts upon some two hundred, I am compelled to state that I have never once seen a cure arising from their use. There has never been invented a belt capable of keeping a dropped kidney in its place, which could be tolerated by the patient. Its pressure would ulcerate the skin in less than a week.

Operation, properly performed, is the only means whereby those who suffer from the effects of nephroptosis can be effectually cured. In 1910 Mr. Billington published his book giving the results of his work on the surgical side. His cases number hundreds, and the results leave no room for doubt as to the best method of treatment. Since that time he has operated upon many more, and has placed the surgical treatment of dropped kidney outside the realm of question or doubt. I fail to comprehend the teaching which, while recommending operation on hernia, displaced uteri, varicocele, and various abdominal conditions causing reflex dyspepsia, such as ileal kinks, intestinal bands, and chronic appendix, yet hesitates to deal with dislocated organs of such importance as the kidneys on similar lines.

Method of Operation.

It was not until Mr. Fullerton of Belfast published his method that nephropexy was placed upon a sound mechanical basis. Since its publication I have practised his method with slight modification. Formerly I used the oblique incision parallel with the last rib and about a fingerbreadth below, carrying it forward through the external and internal oblique muscles, exposing the lumbar fascia. This method damaged the important muscles of the back and side, and I now use the almost vertical incision recommended by Edebohls and divide the lumbar fascia transversely; the opening, which can be pulled apart, allows the bulging of perirenal capsule and fat. The capsule should be thoroughly separated from the muscles behind, and then clipped through deep in, and stripped off the kidney with the contained fat and pushed deep into the wound and left there. The kidney can then be lifted out quite easily and all adhesions in whatever part divided, paying particular attention to a strong band of tissue passing down from the lower pole of the kidney—the so-called reno-colic ligament. The ureter and the pelvis of the kidney should be cleared of all fat and bands, if any. Care should be taken to avoid any aberrant artery or vein; these I leave alone, unless they definitely hold the kidney down, but this is not usual.

The operation as designed by Mr. Fullerton consists of raising a tongue-shaped flap of capsule from the back of the kidney, passing this through the external arcuate ligament, and drawing the kidney up into position and stitching the suspension flap to the capsule below. Billington and Pardhy bring the flap over the last rib. The merit of this plan is that the kidney can be got higher up, according to the length of flap. In addition to this I follow the plan of Mr. Billington in using Brodel's silk-gut suspension stitches, two of which I pass through the lower portion of the capsule in the form of a letter U, bringing the free ends through the muscle and the skin, tying them over a pad of gauze, when the operation is finished. They should be left for three weeks. Care should be taken that none of the fat or loose tissue finds its way between the upper pole and the stripped psoas muscle, as this prevents strong union. Immediate risks are contusion of the gland, rupture of artery, vein, or ureter, and opening of the pleura. Later risks are shock, embolism, and sepsis.

The mortality in my whole series is two—one due to post-operative shock, with acute paresis of the bowels; the second was due to embolism caused by laceration of a large abnormal vein in the lower pole of the left kidney, which I ligatured.

Results of Treatment.

Mr. Billington, in his recent work, gives the percentage of cures as 90; Mr. Pardhy informs me that he has a

like result. In a first series of 100 cases operated upon by me, my percentage of complete cures is 73. This low percentage has arisen from the reason already mentioned—namely, that in a number of my early cases I fixed up only one kidney, and also that a number of long-standing cases were constitutionally in a condition in which, I believe, permanent changes had taken place in the autonomic nerves which no operation could completely cure. Nevertheless, in the remaining 27 cases there was great improvement, and the operation was fully justified, particularly so where there was only one kidney down. In the second series of 50 cases, the percentage of cures rose to 94, owing to the facts that most of the patients were younger subjects, and that in 45 of them I fixed both kidneys.

THE RADIAL PULSE IN INTRATHORACIC ANEURYSMS.

BY

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DIFFERENCES between the arterial pulses as these are studied at corresponding points on opposite sides of the body have an established position in the list of physical evidences suggestive of intrathoracic aneurysm. Since the introduction of the sphygmograph and the sphygmometer it has been possible more confidently to detect these differences and more precisely to define them. Indeed, it may not be unfair to say that unless the contrast between the compared pulsations is a very decided one its value as a trustworthy observation, until confirmed by instrumental evidence, would hardly be admitted. Certainly the advantage of such graphic and numerical displays is beyond dispute, though, as will here be shown, they include opportunities for error peculiar to themselves. Where their worth resides, apart from their merits as summarized and permanent records, is in the confirmation or contradiction they afford to the judgement dictated through the unaided finger. In this respect at any rate, whatever may be true in other directions, the facts of the pulse may miss an adequate recognition in the absence of registration by an apparatus which eliminates the personal factor of the individual observer. In other words, a graphic or numerical record has this merit: it speaks in terms not of a personal impression but of a physical fact.

The usual statement made on the point here under consideration is that the radial pulses in a case of intrathoracic aneurysm may differ the one from the other in force and in volume; and to this statement there is universal assent. But an alleged further difference—namely, a delay in the arrival of one pulse in relation to the arrival of its fellow—is a sharply contested issue. Everyone allows a possible sense or impression of delay as judged by the finger, and writers in pre-instrumental days—rightly or wrongly—assumed this impression to represent an actual occurrence. Graphic records, however, have shown that in some instances where delay seems to be beyond challenge it does not really exist, and that the suggestion to the finger of a unilateral pulse delay must be attributed to a more gradual rise of pressure within the artery between which and the left ventricle there is interposed the elastic sac of an aneurysm. Not unnaturally this observation has thrown doubt on the accuracy of the assertion that in intrathoracic aneurysm the radial pulse on the side corresponding to the aneurysm may in point of time fall behind its fellow. As appearances have certainly been misleading in some instances a facile conclusion is that they are misleading in all, and in the circumstances it may be admitted that the *onus probandi* now falls on those who affirm real delay—as distinct from the mere appearance of delay—to be among the possibilities. Manifestly, to secure this position graphic records are essential, for without such records there can be no effective answer to those who are able to show that the test of the finger has in particular instances undoubtedly proved deceptive; and who, further, have a ready and reasonable explanation of the source of the mistake. It is on registered tracings,

therefore, that the case for actual pulse delay must stand if it is to stand at all.

The record now submitted (Fig. 1) is claimed to be a demonstration of actual pulse delay in the left radial artery as compared with the time incidence of the right radial, and the tracings were obtained from a case of intrathoracic

FIG. 1.—Sphygmometer: Right, maximum 135, minimum 55; Left, maximum 75.

aneurysm. This diagnosis is beyond question, for a *post-mortem* examination revealed a considerable fusiform aneurysm of the aortic arch extending beyond the origin of the left subclavian artery. The wall of the aneurysm showed but little fibrinous deposit, and doubtless, therefore, during life the sac responded freely to the alternate rise and fall of blood pressure determined respectively by the systole and diastole of the left ventricle. In a word, the aneurysm was an elastic bag placed in the course of the circulation between the left ventricle and the left radial artery. Such an

explained, delay as interpreted through the finger may be apparent only, it is necessary to add that delay as registered in a graphic record falls outside this comment and must be accepted as the announcement of an actual occurrence.

While a time difference of the radial pulses, when it exists, needs careful demonstration, there are in not a few cases of aneurysm other differences that may be openly displayed in the contrasted sphygmograms. One of these is a difference in the height of the right and left waves; another is difference in form; and the full message is provided when both are present. Of the two, difference in form is much the more significant. Indeed, mere difference in the height of the contrasted waves is by itself of little moment, for this may exist with a perfectly normal cardio-vascular apparatus. Thus, Fig. 2 is taken from a healthy adult, and although the excursion of the wave in the left radial tracing is decidedly more extensive than that of the right radial, the other features of the tracings are substantially identical, and no experienced eye would read into the picture a suggestion of aneurysm. On the other hand, in Fig. 3 the waves in the two tracings are of one and the same height, but they are strikingly different in form, the right showing the sharply defined lines and angles of a normal sphygmogram, while the left suggests a swinging or pendulum-like movement—the ascent is gradual and the secondary waves proper to the descending line are absent. In Fig. 4 the contrast is a glaring one, and is exhibited both in the height and in the form of the waves of the sphygmograms. As a matter of fact, Figs. 3 and 4 were obtained each from a case of

FIG. 2.—Sphygmometer: Maximum, Right and Left, 135.

FIG. 3.—Sphygmometer: Right, 120; Left, 80.

FIG. 4.—Sphygmometer: Right, 160; Left, 50.

arrangement might on the face of it suggest a delay in the arrival of the pulse wave at the left wrist as compared with the time occupied by the passage of the wave along the arterial trunks of the opposite limb. That it did, as a matter of fact, effect this delay is apparent from the contrasted tracings. These were taken simultaneously on a polygraph, a wrist splint being fixed on each forearm; and it is obvious that the commencement of the rise in pressure in the left radial falls behind the corresponding movement in the right radial by an appreciable interval of time—approximately one-fifth of a second. Thus even if, as is alleged, there is normally a difference of one to two one-hundredths of a second in favour of the right radial pulse, the present record is an announcement of pathological delay in the radial artery on the side corresponding to the situation of the aneurysm. It is a proof that in the individual case here reported the delay was a real happening and not merely an apparent one. Other cases must, of course, be tested by a similar order of inquiry, but if the above record is accepted it is necessary to admit that among the possible evidences of intrathoracic aneurysm a place must be found for a true asynchronism of the radial pulses.

This evidence does not stand alone. Dr. Leonard Findlay¹ has published tracings which tell the same story, and has demonstrated by an ingenious apparatus the delaying effect of the introduction of a distensible sac on pulsations produced in a system of elastic tubes. His paper includes a very full discussion of the whole subject, and he rests the case for real delay in one or other radial pulse both on clinical and on experimental evidence. Hence even when the admission is freely made that, for reasons already

aneurysm, and, as can well be understood, gave definite aid to the diagnosis. Yet such sphygmograms cannot be quoted as compelling confidently a diagnosis of aneurysm; they necessarily mean some unilateral interference with the course of the arterial pulse, but it does not follow that this interference is provided by an aneurysm. Fig. 5, for example, shows facts essentially identical with those of Fig. 4; yet the case was not one of aneurysm. It was, indeed, a quite manifest case of chronic interstitial nephritis, and as no evidences of aneurysm could be discovered the differences between the radial pulses provided an opportunity for clinical speculation. The riddle was read at the *post-mortem* examination, for this revealed in the right subclavian artery a firm clot adherent to the arterial wall for a matter of two inches and largely obstructing the lumen of the vessel.

One other possible difference between the circulatory conditions existing in the right and left upper limbs in intrathoracic aneurysm may be noted—namely, a difference in the sphygmometer measurements. In the cases from which Figs. 1, 3, and 4 were taken the difference was a considerable one, the records being: in the first, right 135, left 75; in the second, right 120, left 80; and in the third, right 160, left 50. In each case it was observed that the auditory method applied to the left brachial artery detected no sound, so that the maximum reading had to be fixed solely by the finger, and no minimum reading was possible. Some degree of difference between the readings in the right and left limbs is not uncommon, but nothing like the contrast exhibited here can be passed as normal. On the basis of numerous measurements, Dr. O. K. Williamson² came

to the conclusion that a difference of 30 mm. or upwards in the sphygmometer readings obtained from the two upper limbs speaks strongly in favour of aneurysm; that a difference of 20 mm. may mean either aneurysm or dilatation of the aorta; and that with a still slighter difference may be found either of the conditions just mentioned, or arteriosclerosis, or mediastinal tumour. Broadly, the position is that to be of diagnostic value the inequality between the measurements must be a pronounced one, and even then, as

FIG. 5.—Sphygmometer: Right, 40; Left, 160.

shown by the case from which Fig. 5 was taken, the possibility of a unilateral interference with the arterial circulation other than aneurysm must be remembered. Further, such differences as are illustrated by the readings recorded in Figs. 1, 3, and 4 are unusual even in cases of aneurysm, and frequently—probably in the majority of cases—the readings are identical or approximately identical. A parallel remark applies to the other pulse differences noted in this paper. Neither unilateral delay in the pulse wave, nor modification in the height or in the form of the wave, is to be observed as a frequent clinical experience in thoracic aneurysm. The absence of such conditions from an individual case is therefore of little diagnostic moment, and while their positive values, when present, may be considerable, even these are subject to such qualifications as are here described and illustrated.

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A NOTE ON THE NATURE OF THE BLOOD SUGAR.

BY

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IN a recent paper¹ we gave the results of an investigation into the nature of the blood sugar by a new method. Hewitt² has lately criticized both the method employed and also the interpretation placed thereon. He states that we "reached the conclusion that the normal blood sugar is γ glucose." In discussing the experimental evidence which we brought forward in our original paper we stated: "The nature of the normal blood sugar we leave for the present undecided." Later, in view of the fact that Hewitt and Pryde³ had shown "with some certainty"² that γ glucose is formed from α , β glucose in equilibrium of the intestine, we suggested that the unstable sugar which was found to be present might be γ glucose. We stated further that "The enzyme responsible for the conversion being apparently absent from the blood, and the reaction of the blood being alkaline, suggests that it is impossible for so reactive a sugar to exist free in the blood."

It is obvious therefore that our only reasons for ever suggesting the presence of γ glucose were based on the work of Hewitt and his collaborators. They state⁴:

"It may be presumed on *a priori* grounds that a structural change of the nature referred to has some fundamental reason." The reason was not clear, but a possible explanation was afforded by the differences we showed to exist between the normal and diabetic blood sugars, and more recently by the fact that the nature of the diabetic blood sugar becomes similar to that of normal persons after injections of insulin.⁵

Hewitt² objects: "Polarimetric evidence alone, therefore, must exclude rigorously the presence of β glucose in amount greater than that found normally in aqueous solution of glucose in equilibrium." Control experiments showed that the equilibrium of α , β glucose was hardly affected by the experimental technique; further, after the alcoholic extraction no trace of copper-reducing substance was found to be left behind. Preferential extraction by alcohol or alteration in equilibrium of α , β glucose is therefore definitely excluded. In some experiments the initial rotation was below that which would be given by β glucose alone, and in the case of diabetics above that of α glucose alone. The suggestion that the normal blood sugar contains β glucose, and diabetic blood α glucose, is therefore not valid. Further, in one experiment a laevo-rotation was observed, but as this has not been repeated no stress has been laid on it; in this connexion it is of interest to note that Hewitt and Pryde attach considerable importance to the one laevo-rotation observed in their own experiments.

The fact that we obtained a glucosazone from normal blood may be due to the fact that some of the normal blood sugar is the stable α , β form. It seems probable that normal blood sugar is an equilibrium mixture of α , β glucose, and a reactive sugar.⁵

The question of sterility is disposed of by the following facts: (a) the polarimeter tubes were sterilized; (b) the copper-reducing value was unaltered at the end of three or four days; (c) the fluid was perfectly clear throughout, and no sign of any growth was observed.

With regard to the criticisms on the general technique adopted, though admittedly it is not perfect, we maintain that by it we have been able to obtain a clear and concentrated protein-free filtrate from blood by which we are able to show with ease the very wide differences in the nature of the blood sugar under different conditions. We may merely mention here that the nature of the diabetic blood sugar differs from normal, and that after administration of insulin the blood sugar of the diabetic approaches normal; and further, that after administration of adrenaline the blood sugar differs again from normal. A most striking difference is observed in the blood sugar of normal animals after insulin (unpublished experiments); the blood sugar in this condition, though dextro-rotatory, has no copper-reducing value, as determined by the Wood-Ost method.

The downward change in optical values of the diabetic blood sugar is not always observed, and it would appear that in this pathological condition the blood sugar may be very complex.⁶

Hewitt criticizes the experiments in which we endeavoured to test whether there is present in the blood an enzyme capable of converting added α , β glucose or *D*-fructose into a reactive sugar. The experiments of Cooper and Walker,⁷ directed to the same end, are also not allowed to pass unchallenged. The first point he makes is that the blood was extravascular; criticism on this line is purely destructive and would invalidate a vast amount of work which has been done with blood. A more legitimate objection which he makes against the experiments of Cooper and Walker is that an enzyme, if present, was probably unable to pass through the membrane of the dialyser owing to its physical properties. Our own experiments he dismisses on the ground that the method employed was in every way suitable to destroy γ glucose; he omits to notice that if differences in the blood sugar could be demonstrated by this method, the same method would probably be capable of showing a difference had any change taken place in the nature of the sugar which had been added to the blood. He agrees, however, "that no such enzyme can be demonstrated in the blood,"² and