seems to be no reason for excluding in exceptional cases the thyroid from all participation in the disease. For the thyroid secretion is the great stimulator of the metabolic processes.

If these views on the pathogenesis of rickets are well founded, it seems illogical to expect that there can be one cure for the condition; each case, in accordance with its own particular etiology, must be treated on specific lines. It would be unreasonable, for instance, to expect good results from treating a case of rickets due to carbohydrate excess on the same basis as one due to a chronic infection or vitamin starvation. The specific treatment of rickets lies in the specific treatment of the contributory factors, although the symptomatic treatment of the resulting mineral depletion certainly lies in the adequate supply of available calcium salts, and in alkali therapy.

I have drawn up a schematic diagram (Fig. 3) to show the common etiological factors of rickets in such a way that their contributions to the common end-result may be easily visualized. These various factors are arranged in

FIG. 3.

groups. Like a vast watershed they afford their respective contributions of acid products to the common reservoir—the blood—which is indicated in the centre of the figure.

The clinical picture of any given case of rickets must be just as composite as the etiological factors which are responsible for its production. The symptoms and the general clinical picture of a case which is due to vitamin starvation, or calcium inanition, will be quite different from a case due to carbohydrate excess or a chronic infection, although they may have certain symptoms in common—namely, those which are due to the resulting acidosis.

Since almost every variety of malnutrition, either acting alone or in combination with others, can lead to one and the same end-result, we can easily understand why there has been so much confusion between the symptoms proper to the rickets condition and those due to the contributory causes—a confusion which is really one between cause and effect.

In conclusion, I would once again repeat that the essential basis, or the *causa vera*, of rickets is most probably a relative excess of acid bodies produced in the system by a disproportion between the intake and the output of energy—a condition which results either from an innate inefficiency of the body machine itself, or from defects in the environment, the latter being regarded as including the quantity and the quality of the food as well as the character of the stimuli afforded by the hygienic surroundings.

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NEPHROPTOSIS: ITS CAUSATION, SYMPTOMS, AND RADICAL CURE.

BY

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ABOUT thirty years ago Dr. Suckling of Birmingham was led to the conclusion that, as many cases complaining of nervous dyspepsia, neurasthenia, and depression had also movable kidney, there might be in these associations more than a casual relationship. His further investigations convinced him that the renal displacement and the nervous disturbances stood in the relation of cause and effect. When my attention was first drawn to Dr. Suckling's work, published in 1907, I had already seen and treated many cases in the usual way. In consequence of the striking results disclosed in his work, I decided to operate on all suitable cases.

CAUSATION.

Movable kidney may be classed under two heads: (1) accidental; (2) constitutional or congenital. Cases of the first class are usually due to accident or injury of a crushing nature, and are easily diagnosed by the history and acute symptoms. The second class comprises the vast majority of all cases, varying from the least increase of the normal mobility to the complete dislocation of the organ from its anatomical site.

To account for the fact that many more women than men suffer from nephroptosis various explanations have been advanced—such as frequent pregnancy, loss of flesh, loss of intra-abdominal pressure, chronic constipation, chronic cough, etc.; but these causes do not account for more than a small percentage of the cases I have seen. I am convinced that there is something in the anatomical formation of women which is the primary physical origin of the condition. I suggest that there is a congenital or family defect of shape of the renal pouch, which predisposes to an easy descent of the organ, and that there is some inherent weakness in the suspensory tissues which disposes them to stretch easily. Can we deny the hereditary influence, in tone of tissue, while admitting it in type of outline of body, in temperament, and in nervous responses? I am strongly disposed to consider the larger proportion of cases as directly due to some transmitted congenital defect. In support of this I would cite the instance of a father with double nephroptosis and two daughters with a similar condition; another of a mother and two daughters; and at least twenty cases of sisters. Suckling states that he has often found movable kidney in children, and that he is inclined to the view that it is hereditary and congenital. Billington and Pardhy confirm this observation.

Volkow and Dilitzin state that the paravertebral niches in which the kidneys rest are shallower and more open below in women than in men. This observation is supported by Morris, who examined one hundred men and found movable kidney in two only, and in a series of operations by him for this condition ninety-six were upon women and only two upon men.

In addition to the congenital tendency and anatomical defect in the formation of the renal pouch, there are contributory causes, of which the most important are nutrition and occupation. A large proportion of my cases in the Samaritan Hospital have been operatives from half-time age. Inquiries from doctors in large practices in other manufacturing towns confirm my view that the too early employment of girls in occupations causing long standing and weight carrying is an important causative factor. Another contributory cause which has been suggested is repeated pregnancy, but I believe it to be a negligible factor, for over 50 per cent. of my cases were unmarried or non-parous, and I have examined many mothers of large families and found no displacement. Loss of intra-abdominal pressure may, in some cases, aggravate the condition, but is never its cause. Other alleged causes are tumours of the kidney and calculi, but I do not attach much importance to them. Were nephroptosis the result of accident, physical strain, etc., we

should expect the larger proportion of cases to be found in them, whereas I have only seen it in ten men as against three hundred women.

Examination of the Patient for Nephroptosis.

Many cases of nephroptosis have been missed by examining the patient in the recumbent position. It is a common experience to find that the movable organ goes back on lying down, and deep breathing or coughing may not cause it to descend.

There are three ways of examining for a movable kidney. The first and most important one is with the patient standing, with back toward the examiner. The body being uncovered from breast to above symphysis, the patient is instructed to breathe deeply, slightly leaning towards the side examined. The thumb of the right or left hand, according to the side examined, is pressed deeply into the back below the last rib, the fingers grasping the front of the body and compressing the structures, alternately pressing and releasing until all rigidity passes off. The kidney, if movable, can then be felt; usually it slips up suddenly from the grasp, and renal tenderness is generally shown by the patient wincing.

In the second method the patient is placed upon a couch, in the sitting position, knees drawn up and body slightly bending forward, and then examined as in the standing position. In fat patients it is often necessary to try a third position. Place the patient on right or left side, as the case may be, with the knees drawn high up; this causes the abdominal contents to fall away from the flank, and the freely movable kidney can be palpated. A point of importance in each method is to begin the compression of the anterior wall over the iliac fossa, gradually ascending. If this is not done one can easily miss a kidney low down.

DIFFERENTIAL DIAGNOSIS.

In my experience only one condition has given any real difficulty—an elongated right lobe of the liver. When this is thin and freely movable it may easily be mistaken for movable kidney. It is, however, more anterior and does not slip from the compressing fingers in the same way as the kidney does. Nevertheless pressure upon this lobe may push it backwards, and give the impression that it is a kidney slipping back into its fossa. The two conditions are often present in the same subject. I need only mention tumours of gall bladder and ascending colon, and tumours of kidney, as possible sources of error.

A patient may present a typical history and symptoms of movable kidney, but on careful examination no kidney can be felt; on this account mistakes are sometimes made and patients are told that there is no displacement. I have often had to examine several times before finding displacement.

Another difficulty arises where the kidneys move downwards and inwards under the rectus, or when from their thinness we compress over them. Another point to note is the excitability of the muscles of the body wall. This is especially likely to arise if pressure be made too rapidly or too deeply. A further difficulty met with is rotation of the lower pole inwards: in such cases we do not find displacement, nor can we palpate any portion of the kidney, but still the symptoms definitely point to mobility. This is frequent in the early stages of descent.

SYMPTOMS.

Symptoms may be classified as direct or reflex. The former comprise intermittent pain in the small of the back of a dull aching kind, radiating from loin to thigh, described by some as dragging and tiring in character, increased by walking, long standing, or train journeys, inducing a feeling of over-fatigue. Reflex symptoms may arise in any organ supplied by the autonomic nerves.

Complaint may be made of violent headaches associated with giddiness and disturbance of vision, and sometimes ending in violent sickness and prostration. Or the patient describes palpitation, shortness of breath, increased upon exertion, and sometimes induced by the effort of undressing, but commencing after lying down. There is a small rapid pulse, with low blood pressure, and a mildly cyanotic appearance in face and lips, as if there were vasomotor disturbance. Again, the history may be one of chronic indigestion, sick-

ness, vomiting, in some cases haematemesis, discomfort after meals, giddy attacks, with sallow skin and tired appearance. Rarely is there any local tenderness in the region of the stomach. Or there is a feeling of sickness and giddiness on getting into the erect position after a night's rest. This symptom is very significant when it occurs in a young subject. It appears to be due to the sudden descent from the pouch of one or both organs. Similar attacks are common after any form of energetic physical exertion.

In another case the story may be one of intermittent diarrhoea with colicky pains, and evident tenderness in the caecal region, distension in the lower abdomen, a feeling of weight and heaviness, and great discomfort. Later on colitis may ensue. Or, again, the patient may complain of frequent micturition, day or night, often both, and sometimes haematuria, but there is no pus in the urine, no albumin, no sign of gravel, only an increase in mucus. The normal quantity of urine may not be increased in twenty-four hours, and generally the specific gravity is low. Many of these cases end with contracted bladder. In some cases the amount of urine secreted during the daytime is diminished, though there is no irritation, but, after retiring, there is frequent necessity to empty the bladder. This, I believe, is due to recession of the kidney, which, in the recumbent position, functions more actively when relieved from the inhibitory effect caused by descent.

We come next to that class of cases where the symptoms are referable to the female generative organs. The late Goelet of New York was one of the first to call attention to the reflex effect of nephroptosis upon the uterus and ovaries. The conditions he enumerated as provoked by movable kidney were: ovarian congestion due to interference with the ovarian vein; dysmenorrhoea, due to congestion of ovaries; profuse leucorrhoea; and endometritis. Menorrhagia and metrorrhagia are undoubtedly increased by this condition, and premenstrual headache is often intense. Amenorrhoea is sometimes present, and uterine displacement is a frequent concomitant. I am satisfied that in all cases of chronic pelvic symptoms, where there is no evidence of a local pathological condition, we should examine the renal region for mobility repeatedly if necessary.

Then we come to those cases with acute abdominal pain and vomiting, profuse sweating, pain in abdomen, and all the signs of shock—"Dietl's crisis"—sometimes diagnosed as due to acute appendix, acute gall stones, renal colic, or calculus. Lastly, I would draw attention to the fact that many of these cases of nephroptosis have chronic irritation of the appendix, and that it is not uncommon for the appendix to be removed, but without benefit. Conversely, the kidney may be fixed and a diseased appendix left. This coexisting condition is a sequel to the dropped right kidney producing irritation by its descent behind the caecum.

Many cases of nephroptosis are treated for neurasthenia, dyspepsia, and vague neuralgias, with little or no success. Closer investigation would reveal that the symptoms are secondary to and dependent upon renal displacement.

The Nervous System.

The autonomic nervous system comprises the sympathetic and parasympathetic; speaking generally of their functions, it may be said that when the sympathetic and parasympathetic are distributed to the same structure their actions are antagonistic. As Cannon graphically puts it, the parasympathetic serves for bodily conservation, as seen in its action on the pupil, which it contracts to save the retina from the excessive effects of light. By slowing the heart rate it gives the heart muscle longer periods of rest; by providing the flow of saliva and gastric juice, and by supplying the muscle tone necessary for contraction of the alimentary canal, it is essential to digestion. The sympathetic activates the body for struggle. It dilates the pupil to receive more light and stimulates the heart to beat more quickly to supply blood to the muscles. It contracts the blood vessels in the visceral area and inhibits the functions of the digestive organs.

Should the sympathetic be stimulated to overaction in any of its branches we should naturally expect disturbance of the ordered function of the gland or organ it governs. If this stimulation be more or less continuous there will be engendered in those nerve tracts a special susceptibility to such

stimulation, and consequently an increased liability to overaction. If we remember the distribution of the sympathetic chain with its numerous ganglia or exchanges acting as distributing centres, we can comprehend that stimulation received at one point may be passed along from the first receiving station to some other exchange centre, and produce disturbance of the function of another gland or organ remote from the point of stimulation. This action may be seen in reflex vomiting of early gestation, in unilateral renal calculus producing a pain in the other kidney, or in spasm of the pylorus in appendix disease. Sir James Mackenzie's investigations show that the majority of symptoms of disease are disturbances of normal reflexes, and give support to my view that abnormal stimulation of the renal nerves is transmitted to the proximal ganglia, and, passing through the synapses therein, sends out radiation impulses which affect other structures and organs. I believe that in this way a movable kidney, by tugging upon its pedicle, sets up irritation in the nerve, which is transmitted to the proximal ganglion, and that through the synapses therein the irritation is passed either upwards or downwards or both, through other ganglia, thus calling into action, or overaction, the nerve supply to the stomach, bladder, heart, or any structure controlled by the autonomic nerves, producing in one case dilatation, in another irritation, and in a third palpitation. It is this mechanism of transmission and radiation of stimuli which makes possible the comprehension of the numerous and varied symptoms arising from dropped kidney. It has been shown, moreover, that stimulation can be carried to the extent of causing pathological changes in nerve tissue. Langdon Brown says:

"The sympathetic response may be so dissociated, perverted, or prolonged as to produce through the thyroid, Graves's disease, through the pituitary, diabetes insipidus, and through the pancreas and other endocrine organs, excessive mobilization of sugar, which is the first stage of diabetes. It may disorganize digestion by exciting spasm, by producing atony in stomach and bowel, and inhibiting the secretion of gastric juices, etc."

We need only continue the irritation for sufficient time and there must ultimately follow a complete breakdown in the normal responses. As Jelliffe puts it, "after years of maladjustment permanent changes result, and a vicious circle is established in which cause and effect are inextricably interwoven in their general result." Timme, by a series of experiments on the nerves of the stomach, has demonstrated the possibility of unbalanced action in them, and has also shown that such unbalanced actions are constantly brought into operation by mechanical factors, toxins, hormones, and by mental stimuli.

We thus realize the importance of the perfect balance of these two divisions of the autonomic nerves to the health of the individual. We are cognizant of many factors which disturb their normal harmony, and, though not yet generally recognized, I believe movable kidney is one of the most important of these factors, even in the early stages of descent. Chronic recurrent overstimulation of the nervous mechanism, whether from constant tugging as in movable kidney, or chemical as in retained products of excretion, produces permanent changes in the nerves and ganglia.

These factors have an important bearing upon treatment, for it will be obvious that the sooner disturbing causes are corrected the better for the patient, and, conversely, that in long-standing cases changes may have taken place which cannot be cured, either by palliative or operative treatment.

Suprarenal Glands.

In nephroptosis, as can be demonstrated at operation, the suprarenal glands are pulled down along with the kidney, and there must be some interference with the circulation, consequent upon stretching of their vessels. I wish to emphasize the importance of this point, for it has been shown that the blood supplied to these organs is proportionately greater than to any other organ in the body, and that they consume proportionately more oxygen. This consumption is increased threefold during a rise in blood pressure produced by the action of adrenaline. The chromaffin cells forming the medulla of the gland secrete adrenaline, and the action of adrenaline is to activate the sympathetic. It is also known that adrenaline has an effect upon the glycogenic functions of the liver, increasing the blood sugar content

which is necessary to supply the muscles of the body with energy. Excessive stimulation through temporary congestion caused by increased movement in the early stages of nephroptosis may produce an increased output of adrenaline, and as this is the hormone for activation of the sympathetic, this may be an explanation of the severe headache, rapid action of the heart, and increased blood pressure, which are common symptoms in renal ptosis.

On the other hand, any reduction of the amount of blood passing through the glands, as may arise in stretching of the vessels, thereby contracting the lumen, would reduce the output of adrenaline, or the same result may follow exhaustion after a period of overaction. This would diminish the production of blood sugar, which would affect the muscles by reducing their pabulum, and consequently engender a state of fatigue and exhaustion, which is a well known symptom of hypo-adrenalism—a very common sequel in chronic cases of nephroptosis. As the amount of adrenaline present in the blood under normal conditions is equal to a dilution of one in twenty millions, a slight increase or diminution in secretion will very seriously affect the action of the sympathetic and the musculature. The whole range of the chemical action of adrenaline has not yet been physiologically determined, but enough is known to justify our conclusion that variation in adrenaline output is an important factor in the symptomatology of nephroptosis.

Toxaemia.

Toxaemia, I believe, only becomes operative when the kidney has become so displaced as to interfere with the normal circulation and proper drainage through the ureter. The later effects of displacement are, first, obstruction to return of blood through the veins, increased size of gland, and hyperaemic congestion caused by descent and inward rotation of the lower pole. Secondly, obstruction of the ureter, with intermittent back pressure, resulting in dilatation of the pelvis of the kidney, widened calyces, and flattened pyramids. There is consequently a reduced excretion of urine, particularly during the daytime, with lowered specific gravity, causing retention of poisons. Continued mild auto-intoxication will in time reduce the vital powers of the strongest, and this is particularly the case if the primary cause lies in an organ of elimination such as the kidney. This is well shown in chronic Bright's disease, or in lethargic colon, or in chronic infection, as in antrum, nose, or throat. Suckling thought that most of the nervous symptoms of movable kidney (neurasthenia, melancholia, and insanity) were due to toxic products retained in the blood. In my opinion, however, the cardinal symptoms in the early stages of nephroptosis are due to nerve irritation and suprarenal instability. We need not, however, be surprised to find a moderate diminution of renal excretion producing many complex symptoms, among which not the least are changes in the functional integrity of the higher levels of cerebration, including, according to Suckling and Billington, acute mania and melancholy.

TREATMENT.

The opinions of physicians and surgeons have been variously expressed, some holding the view that a two months' rest in bed will do all that is required; others that a well made corset fitted with pads will effect a cure; and yet others that in most cases no treatment is necessary. This divergence of view may have arisen as a consequence of unsatisfactory methods of treatment. One cause of unsatisfactory results in surgical treatment has been imperfect method and technique. This has led conscientious surgeons to declare that the results of operation do not justify its general use. Failure to obtain good results is frequently due to a second movable kidney, which has been overlooked, being apparently less mobile, or considered unlikely to affect the symptoms. The left kidney often gives the impression that it is not freely movable, whereas I have been astonished at the extent of mobility disclosed when the left loin was opened. I would therefore lay great stress upon the omission to fix the left kidney as the explanation of operative failure in many cases. When surgeons recognize the frequency of double nephroptosis, and operate on both sides, I am confident that the value of nephropexy will be as well established as that of appendicectomy. If I find any mobility on the left

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,