A Lecture

ON

SOME OF THE RARER OCCURRENCES IN THE RHEUMATISM OF CHILDHOOD.

DELIVERED AT THE HOSPITAL FOR SICK CHILDREN,
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BY

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I HAVE for some years now occupied these lectures in demonstrating various points in the rheumatism of childhood with the particular intention of strengthening the view that the disease is the result of an infection. Sometimes I have illustrated the nature and processes of the infection, at others I have attempted to explain the clinical course of the illness by the facts forthcoming from the study of the pathology. It has so happened that these attempts have come at a very opportune time—at a time when in this country the State has increased its vigilance in the control of the health of its school children. I believe that in the future, when the details and difficulties of this supervision are surmounted—as they assuredly will be—this development promises to assist the ever-pressing problem of the treatment of rheumatism. Already I find in my out-patient department evidence of this in the increased number of children who are referred direct from the schools for opinions upon their hearts, or for the warnings of chorea. I look upon the medical inspection of school children, then, as the first concerted step to deal seriously with the problem of rheumatism, because I believe the first step to successful treatment is an early recognition of the symptoms.

I believe, too, that the teachings of many distinguished physicians upon the various manifestations of this disease will now begin to bear fruit more rapidly, and I am glad that I have assisted in some degree by adding to their observations some data which help, I think, to strengthen their position. It is, in my opinion, important that the view that rheumatism is the result of an infection should be thoroughly accepted, for otherwise some of the early manifestations, and, above all, the importance of unhealthy conditions of the throat, will be overlooked.

Now in my lecture to-day I am going to deal with some of the less frequent of the rheumatic manifestations in childhood, founding my remarks upon a series of 600 cases that have been under my personal observation, in some instances for seven or eight years.

RASHES IN RHEUMATISM.

My first subject will be the rashes in rheumatism.

These are of considerable clinical interest, and you can readily understand that their occurrence has always been a strong argument in the hands of those who have maintained that acute rheumatism is an infective disease. It is not, however, in the out-patient department that we can study them best, for it often happens that they appear before the child comes first under our observation, or that they develop later after its admission to the hospital. In 600 cases of rheumatism I have on record 40 examples of the occurrence of a rash associated with other definite signs of the disease at the first visit. The usual forms, as you know, are urticarias and erythemata, the latter often circinate in distribution, sometimes tuberculated in character, usually multiform; sometimes they are very extensive in distribution, at other times very partial. They are often fleeting, and are thus easily overlooked. If you follow the history of intractable cases of rheumatism, you will find that such children may sometimes develop arthritis, or pains, or chorea, or, again, a rheumatic rash—that is, the rash may be the most definite evidence of a recrudescence of the malady. Sometimes these rashes appear over the flush area of the face, and, spreading over the nose, have a butterfly shape. This is a puzzling arrangement the first time that one is

confronted with it, and we must not forget that this may be followed by desquamation.

Occasionally these erythemata and the associated symptoms are difficult to distinguish from scarlet fever, as in a case in which a mother reminded me that I had twice diagnosed scarlet fever, and, faithful to me though she was, she demurred at my third effort in this direction. Her attitude of mind was justified by the rapid appearance of arthritis and chorea for the third time. The desquamation is in these cases rapid in course and branny in character. The circinate variety is often called ringworm by the parents, but this mistake is not often made by the medical man who is acquainted with the rashes of rheumatism. You will find that some children seem to be more prone than others to these rheumatic rashes, and they may show not only at different times, but also simultaneously, rashes of different types. Thus erythema and purpura, and erythema, purpura and psoriasis, are combinations I have met with in my series.

This leads me to say a few words about psoriasis. The late Dr. Radcliffe Crocker called attention to its association with rheumatism, and I should like to give you a few illustrations from my own experience upon this point.

CASE I.

C. V., female, aged 11 years, who was subject to sore throats, came to the hospital in April, 1900, for arthritis. I found there were also early mitral endocarditis and fever. This child developed chorea in the following May and a guttate psoriasis of extensive distribution in June, from which there was slow recovery.

CASE II.

May, 1901. W. M., male, aged 9 years, had been subject to recurrent attacks of rheumatic arthritis occurring two or three times a year for the last few years. After the first rheumatic attack he developed his first attack of psoriasis. He came to me suffering from well-marked rheumatic arthritis and early endocarditis. From these he made a good recovery, but he later came to me again for a widespread guttate psoriasis.

CASE III.

November, 1902. A. S., female, aged 10 years, had an attack of rheumatism and chorea after scarlet fever when 5 years old. Simultaneously she developed psoriasis, and had had three attacks since. At 8 years she had a second attack of acute rheumatism. This child came to me with arthritic pains, an enlarged heart, and chorea. There was a well marked psoriasis of recent development localized over the lower extremities.

CASE IV.

In May, 1903, R. H., male, aged 11, came to the hospital suffering from chorea of moderate degree and of two days' duration, and a psoriasis, which had been coming out for two months, guttate in character. This boy was interesting, because he developed some enlargement of the thyroid and prominence of his eyes. His heart was dilated, and there was an occasional mitral systolic murmur.

In August, 1903, he had another attack of psoriasis without chorea until September, when once more chorea developed, this time with obvious heart disease.

You will find a strong family history of rheumatic fever in some cases of psoriasis which show no other more definite indications of rheumatism than sore throat, arthritic pains, and fever. These are, of course, not convincing, but they are suggestive, as are also the not infrequent examples of psoriasis appearing directly after an attack of scarlet fever. In one such case I saw erythema, purpura, and psoriasis all apparent in a child who was suffering from acute rheumatism associated with scarlet fever.

I have read in an authoritative article on *purpura* that its rheumatic origin is a myth. Now this is certainly an error. How many cases of purpura are rheumatic is a very different matter and a difficult problem, but that some of them are of this nature will be apparent from the following details.

I will commence with my most convincing and remarkable case. This was one that I published for Dr. D. B. Lees in the *Lancet*, October 28th, 1899.

A girl who at 12 years of age had suffered from chorea and at 15 from rheumatic fever came to St. Mary's Hospital at the age of 17 suffering from dyspnoea and purpura. Seven days before this she had developed a sore throat and arthritic pains, rapidly followed by purpura round the ankles. Then in sequence appeared severe carditis and arthritis, and meantime the

purpura became so extensive as to baffle description. Large sanious bullae developed over the back, and the chest, abdomen, and extremities were covered with large patches of hæmorrhage. Upon two occasions the micrococcus of rheumatism was isolated from the blood. This patient eventually died from cardiac failure after recovering from the purpura and active rheumatism. At the necropsy I found a severe rheumatic carditis, but there were no internal hæmorrhages.

I have seen severe cases since then, but none to approach this one, which has the advantage of providing a very fairly complete proof of the rheumatic nature of the purpura.

This tendency to purpura in rheumatism is to be expected from a study of the morbid lesions. In early rheumatic arthritis there is intense vascular dilatation and the exudation fluid may be hæmorrhagic. The capillary blood vessels in the regions of nodules are much congested, as are the intramuscular capillaries in carditis and the vessels in the deeper layers of the pericardium in pericarditis. Although hæmorrhagic pericarditis is not, to my knowledge, much dwelt upon in standard works upon rheumatism, it is not a very rare occurrence, as Dr. C. R. Box insisted in the recent discussion on pericarditis at the Royal Society of Medicine.

Other examples of purpura have come to my notice in acute rheumatism.

Thus, in a fatal case of carditis in a girl aged 9, a well-marked purpuric eruption developed a few days before death. In another milder case there was arthritis, endocarditis, and the well-known peliosis rheumatica. A fourth example, in which the purpura appeared in large patches associated with arthritis and endocarditis, occurred in a child whose sister was under me for rheumatism, and who simultaneously developed a recrudescence of rheumatism.

I have seen other cases in which it has been more difficult to assure oneself of the rheumatic origin of the purpura, but the circumstantial evidence has been very strongly in its favour, and in a long history of rheumatism purpura may be an incident pointing to activity of the process.

In rare instances a pemphigoid or bullous eruption has occurred, of which the following are examples:

In June, 1904, a boy of 8 years, subject to night terrors and sleep-walking, developed rheumatic fever with endocarditis, and at that time "blisters" appeared on the body, and were attributed to "uric acid." When I saw the child there was an apparently characteristic pemphigus upon the extremities, which gradually disappeared.

A second case was that of a boy aged 6½ years, who came to the hospital in 1901 for rheumatic arthritis and endocarditis. I saw him again for a relapse in 1902, when he developed a double mitral lesion. At the end of 1904 he again had an attack of multiple arthritis with a pemphigoid eruption upon the arms and knees.

Lastly, herpes may also occur during active rheumatism, and this irrespective of the use of arsenic.

I have already mentioned combinations of these eruptions, and you will sometimes notice that if hot applications used to relieve pain are placed over an area of acute rheumatic erythema the added stimulus of heat will convert the erythema into a purpura. Again, you may occasionally see a rheumatic nodule in the middle of a small purpuric patch.

ERYTHEMA NODOSUM.

I have not mentioned erythema nodosum among these rashes, and I have omitted it because it is a condition about which, at present, there is no settled opinion. That it is a rash which tells of an infection very closely allied to the rheumatic, I believe to be undoubted. Sometimes it seems to me that erythema nodosum may be a result of various but allied infections, including the rheumatic. At other times I feel inclined to believe it is evidence of some special disease closely allied to, but not identical with, rheumatism.

There are many records, notably those of the late Sir Stephen MacKenzie, that make the intimate connexion of erythema nodosum with rheumatism apparent, and I will now cite a few cases that I have met with.

No. 1. A boy with fever, pallor, and erythema nodosum. This boy's father was subject to attacks of rheumatic fever.

No. 2. A boy with obvious erythema nodosum. His paternal uncle and aunt had both suffered from rheumatic fever.

No. 3. A girl who, in July, 1902, came for the characteristic rash, and at the end of September developed arthritis and

purpura, without heart disease. This child's father died of rheumatism.

No. 4. A girl who was suffering from a sore throat, fever, and erythema nodosum, had a sister under me at that time suffering from chorea.

No. 5. A girl who had sore throat, fever, and erythema nodosum. Her maternal aunt had suffered from rheumatic fever.

No. 6. A girl who gave no rheumatic history.

No. 7. A girl who gave no rheumatic history.

No. 8. A girl who gave no history of rheumatism.

No. 9. A boy who gave no history of rheumatism.

No. 10. A girl who gave no history of rheumatism.

No. 11. A girl had fever, arthritic pains and enlarged heart, and erythema nodosum. Her mother was the subject of chronic rheumatism.

No. 12. A girl who was subject to sore throat and stiff necks developed erythema nodosum. She had mitral disease and was short of breath.

No. 13. A boy who came to the hospital for erythema nodosum had suffered from rheumatic fever at 2 years and at 9 years.

No. 14. A girl who had been under me for two attacks of erythema nodosum came to me in March, 1909, for what appeared to be an ordinary rheumatic erythema associated with severe heart disease.

I have taken these 14 cases consecutively, and they show that there is some close connexion between erythema nodosum and rheumatism, but the impression that I have in my mind is that the condition is possibly not a manifestation of rheumatism in childhood, and for this reason I have not included it among the rheumatic rashes. I think it is certain that heart disease is unusual in an ordinary attack of erythema nodosum, while it is, as we know, very frequent in true rheumatism.

VENOUS THROMBOSIS.

The next complication is one which I have only met with in severe examples of acute rheumatic carditis, and at the present time it is not decided whether the lesion is rheumatic in origin or is a mechanical result of the gross heart disease. This complication is thrombosis of large veins during acute rheumatism. In all the cases I have met with the vessels affected have been the large veins in the neck and upper extremities. This complication is not, I believe, very well known. Some twelve years ago I published for the late Dr. W. B. Cheadle and Dr. D. B. Lees three examples in the *Lancet*, and I have notes on several other cases since then. One of the three original cases may be looked upon as a classical example, for I was able to investigate it thoroughly, although, unfortunately, at that time I could not identify the micrococcus.

The case was that of a girl aged 9, admitted to St. Mary's Hospital for advanced morbus cordis, with active pericarditis and pleurisy, oedema and ascites. For a while there was improvement, and the oedema and ascites disappeared. Suddenly, while the temperature was subnormal, there supervened oedema of the right side of the neck and swelling of the eyelids and lips. Then the entire neck became swollen and tender, and first the right arm and next the left swelled. Lastly, the chest became oedematous, and the child somnolent and apathetic. Meantime general pericarditis had lighted up again, and the internal jugular veins could be felt as hard cords. Death rapidly followed. The necropsy showed active pericarditis and multiple endocarditis. The following veins were occluded by thrombi: Both subclavians and innominates, both internal and external jugulars, the inferior thyroid, both axillary veins, and the upper two-thirds of the superior vena cava.

Another case in a girl of 19, the subject of previous rheumatic endocarditis and pericarditis, showed thrombosis of both internal jugular veins and innominates, and partial occlusion of the superior vena cava.

The third case, in a young woman of 21, showed sudden oedema of the left side of the face. In this instance there was no pericarditis, but active endocarditis. The left internal jugular vein was closed by a thrombus at the lower end.

A fourth case was that of a boy, aged 8 years, who seven months before had suffered from chorea. He came under observation with active rheumatism, anasarca, and advanced heart disease. Sudden swelling of the right side of the face and right arm developed, with fever and some pericarditis; later the right internal jugular, subclavian, and innominate veins were apparently blocked, and the jugular and brachial could be felt as firm cords. This patient recovered, and left the hospital.

A fifth case, which was fatal, was that of a very fat and pale little girl, of 4½ years. She had developed an insidious chorea, followed by morbus cordis. When she was seen first, there was general pericarditis, with considerable fever. Death ensued rapidly, and the necropsy showed thrombosis of the left internal jugular veins, recent pericarditis, and multiple endocarditis.

I need not burden you with further examples, but wish to call your attention in these cases to the sudden develop-

ment of localized oedema, and when both internal jugulars are occluded you may mistake the swelling for a parotitis. There is considerable danger of cardiac embolism, symptoms of which were present in the first case I mentioned. In all the cases that I have seen there has been severe heart disease, and only two in my experience have survived. Oedema of the face in the heart disease of childhood is curiously frequent, and it is not at all uncommon for one to feel convinced that there must be renal disease. In my experience the urine does not bear out this suspicion, and we are still in the dark as to the real meaning of this symptom. I do not intend to suggest that venous thrombosis is the usual explanation, but would remind you that it is the explanation in some few examples. The most convincing proof of the occurrence of this thrombosis is the discovery of the tender, hard, cord-like veins, but the palpation should be cautious in case a clot should be inadvertently detached.

In a lecture such as this I will not enter upon the debated question as to whether a rheumatic phlebitis or thrombosis occurs in those cases, for these are questions which are not yet finally settled.

TACHYCARDIA.

The next condition I allude to is tachycardia. This is an occurrence of considerable interest. When we concern ourselves with rheumatic heart disease in adults, we recognize clearly that tachycardia is not an infrequent and is sometimes an extremely dangerous symptom in mitral stenosis. It appears to occur more frequently in mitral stenosis than in other valvular lesions, and may be paroxysmal in onset and character or more or less persistent. Further, it would seem that some cases are much more prone to tachycardia than others. With these points in my mind I have always watched for this symptom in children, and have assured myself that in some instances tachycardia may appear in the first illness, and may be followed later by mitral stenosis, and that when the stenosis appears you may then have a condition resembling the mitral stenosis and tachycardia of the adult. In such a case as this it is clear that the rapid action of the heart, although doubtless encouraged by the mitral lesion, is not in reality dependent upon it; and this in turn raises the question as to the explanation of tachycardia in mitral stenosis. I may at once say that I have never, up to the present, had a chance of examining a heart *post mortem* in a case of early rheumatic tachycardia, so that any explanation would be purely hypothetical.

I shall again illustrate these remarks by taking first a classical example of early rheumatic tachycardia.

This was the case of a girl aged 9 years who lived in a damp house near the Thames. For four months she had been noticed to be very nervous, and to have headaches, which were followed by chorea. When I saw her she had well-marked chorea, some dilatation of the heart, and a pulse varying between 140 and 200 to the minute. The thyroid seemed a little full, but I could not persuade myself it was pathologically enlarged.

In the hospital the tachycardia persisted. She was admitted March 2nd, 1902, and left in June, her rapid pulse persisting for some two months, the rate being usually between 120 and 180 to the minute. Throughout all this time there was not the slightest evidence of pericarditis and the child did not seem ill in herself, although an occasional faint systolic mitral murmur made its appearance toward the end of the period of tachycardia. Her teeth being in a bad state and the tachycardia persisting, many of them were extracted, after which she recovered, this being aided, possibly, by small doses of potassium bromide. The natural suggestion at the time was to attribute the tachycardia to a toxæmia from the unhealthy condition of the mouth, and she left the hospital with that explanation, I believe, generally accepted. The later history was, however, opposed to this, for her recovery was only temporary. In July, 1902, she saw me again, when her pulse could scarcely be counted, and in October she was very excitable, and there was a rapid, irregular cardiac action with heaving impulses, but there was no murmur or renal disease.

In January, 1903, there was improvement, but in June, after the house had been flooded during great rains, she developed rheumatic pains and tachycardia again. In the following August chorea once more developed, but there was still no obvious cardiac murmur. In 1904 chorea developed again, and now it was apparent that a presystolic murmur was appearing. Once more I saw this child in 1905 with chorea, an obvious double mitral murmur, and the same curious tachycardia. My last acquaintance with her was in June, 1910, when she came to a demonstration here. She was then 17, and in domestic service, with an enlarged heart and mitral stenosis. Upon any excitement the pulse rose to 150 per minute. This is

really a very interesting case when you review the history over these eight years, and it is, I think, quite obvious that throughout that period there had been a remarkable tendency to a severe tachycardia, which dated from the commencement of her rheumatism.

I have no other example as complete as this, but a good many cases in which a remarkably excitable heart was a feature of the rheumatism.

In 1903 a child of 7, a girl, came to the hospital with a history of chorea of three months' duration, a very rapid heart and early endocarditis. After recovery from the active rheumatism, her heart remained rapid (pulse 110), although there was no murmur. A year later she had signs of early mitral stenosis, and four years after this I saw her again with chorea and well-marked mitral stenosis.

There is a very important group of rheumatic cases in which the myocardium suffers out of proportion to the valves and in these cases you will generally find tachycardia and irregularity are prominent. Such cases closely resemble the conditions that sometimes arise after influenza or diphtheria. In these there is generally considerable enlargement of the heart. Some of these cases of tachycardia make complete recoveries, as I have observed, after many months or even some years, but those in which mitral stenosis developed have, in my limited experience, always preserved a tendency to the rapid cardiac action. It is only natural to suspect hyperthyroidism in such, for we recognize a connexion in adult life between rheumatism and some cases of exophthalmic goitre. A good many French and of late some American writers have called particular attention to enlargement of the thyroid in acute rheumatism. I have also devoted much attention to this point, because it is one of the many interesting problems in rheumatism. We must most of us recall cases of exophthalmic goitre in adults who have developed the disease, as Dr. S. West has emphasized, after rheumatic fever, and who have in addition to the symptomatic tachycardia clear evidence of this rheumatism in well-marked valvular disease. Now, I have never seen the direct development of this disease after rheumatism. All the cases I have come across with a rheumatic history have shown a considerable latent period between the two illnesses. In childhood, again, I have never met with a case of exophthalmic goitre associated with rheumatism, and, although I have many times looked for a decisive enlargement of the thyroid gland during attacks of rheumatism, I cannot say that I have been able to convince myself that it is often affected. In a few instances, some five or six, I have noted a possible slight swelling of the gland, and in two cases children have complained of pain referred to the thyroid, but, as a rule, I have failed to obtain any evidence at all of damage.

It does not necessarily follow that the thyroid secretion may not be altered by an attack of rheumatism, but then again I am confronted with the fact that not one of the many cases I have seen has developed exophthalmic goitre in childhood. I must confess that my personal observations upon the behaviour of the thyroid gland in the rheumatism of childhood have been singularly barren in result, and for this account I have hesitated to ascribe early persistent tachycardia to any disturbance of this organ. The most definite associated symptom that I have found in these cases has been nervous instability. This instability is a well-known feature in the rheumatic, but I think it is more persistent and reaches a higher degree in these cases of tachycardia.

MASTITIS.

A symptom that I have met with in girls who have suffered from rheumatism has been tenderness and slight inflammation of the breasts. When mentioning this to my friend and colleague, Professor G. F. Still, he made the same remark from his independent observations. I mention it with reserve, for an individual experience is sometimes quite misleading, and those in wide general practice may be acquainted with such a phenomenon as not infrequent in many girls about 10 or 12 years of age quite apart from rheumatism. It is, however, a fact that I have met with it in hospital practice under these circumstances, and will give you some examples.

CASE I.

Hilda S., aged 11, subject to chorea and sleep-walking, whose father's family was strongly rheumatic, one uncle having died

of rheumatic fever, came to the hospital for pain and swelling of the left breast. The swelling had lasted a week, and there was tenderness and some hardness on examination. This symptom rapidly disappeared with the aid of mild aperients and warm applications.

CASE II.

Anne M., aged 10 years, was under me at 7 suffering from rheumatism, with arthritis, chorea, and endocarditis. She made a good recovery, and had kept apparently free from any relapse. Her mother now brought her for pain, tenderness, swelling, and hardness of the left breast. There was definite evidence of her previous attack of rheumatism in an enlarged heart and a double mitral murmur. Her temperature was not raised, and the condition rapidly subsided.

CASE III.

Florence M., aged 13 years, had been under my observation through two attacks of chorea, one at the age of 9 and another at the age of 10. There had been dilatation of the heart and arthritis, but a long rest in the hospital followed by a convalescent home had brought about complete recovery. Although over age, she was brought to me in 1909 with pain, tenderness, and swelling of the left breast. She had been free of chorea since her second attack, and had no symptoms of rheumatism.

CASE IV.

Alice P., aged 10 years, had been under me at intervals for some years suffering from chorea, endocarditis, and arthritis. In October, 1910, she again came back suffering from rheumatic pains, with a well-compensated mitral lesion, and complaining of pain and tenderness of both breasts, which were harder than normal.

Three other cases gave almost precisely the same history. I am not, perhaps, justified in quoting these cases under the title of my lecture; but they have interested me because later in life I think dysmenorrhoea of a painful kind is not infrequent in the rheumatic.

EPISTAXIS.

Lastly, I should like to direct your attention to epistaxis in rheumatism. My attention was first called to this by a paper of Dr. Sydney Phillips upon the subject, and since that date I have learnt to appreciate its reality. Epistaxis may occur at the commencement of an attack, during the illness, or as incident in the more quiescent phases of a long rheumatic history. Exceptionally it may be very severe, but as a rule it is moderate in degree and not of any moment in itself. I would add that it is not by any means necessarily associated with obvious nasal disease, although I hardly think we lay sufficient emphasis upon diseased conditions of the nose in rheumatic children as a possible source of infection, in spite of the writings of Dr. C. R. Stratton.

In this lecture I have dwelt a good deal upon haemorrhages in rheumatism, and I would add further to them the occasional occurrence of haematemesis. This I have only seen at present in grave heart disease, and it is possibly only a mechanical event, although in some examples it has also been associated with profound toxæmia and extreme anaemia.

It will need another lecture at least to complete the remarks that I have to make upon other of the less common occurrences in the rheumatism of childhood, and I shall hope to continue with them when the next opportunity arises.

DR. ROBERT WILLIAM COE, of Clifton, consulting surgeon to the Bristol General Hospital, who died on August 11th, 1910, aged 89, left estate valued at £28,612 gross, and £27,485 net.

THE Secretary of the Medical Missions Department of the Society for the Propagation of the Gospel in Foreign Parts asks medical men and women of Great Britain, who may not wish to preserve medical journals after reading, to consider the needs of the medical missionaries in the foreign field. He states that any journals sent to Miss B. Gambier Parry, Bacton, Wokingham, Berks, will be gratefully received, and forwarded without delay to a missionary doctor.

Clinical Lectures ON KIDNEY PAIN.

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[With Special Plates.]

LECTURE I.

GENTLEMEN,—Although expert radiography has conclusively proved that calculous disease of the kidney is comparatively rare, the profession cling to the belief that any severe pain experienced in the region of that organ is due to stone. I suggest, therefore, that you consider with me this session the varieties of kidney pain and their approximate causes, and for the first step we will discuss that form most frequently encountered—the pain of a distended renal pelvis. It is probable, on physiological and on clinical grounds, that the kidney is only able to feel in two areas—the pelvis and the neighbourhood of the true capsule. Parenthetically, I may remind you that patients are very occasionally met with who do not have sensation in either area, and then crippling and progressive disease of the kidney may be painless from start to finish. As examples you may call to mind the hydronephrosis the size of a small football I operated upon a short time ago, the patient having no inkling that he was a sick man until

a week before he was admitted here, when he noticed the increase in the size of his belly. Then, again, the largest stone I have removed from the kidney was fifty years in the organ without causing any pain.¹ Generally, however, there is pain in surgical kidney disease characteristic both in degree and place.

I start with two aphorisms which I have taught here in the operating theatre since 1900. The

commonest structural change accompanying kidney pain is a distended pelvis; the commonest cause of kidney pain is dilatation of the renal pelvis.

To prove my contention, I must refer to certain salient symptoms which afford a valuable clue to the existence of surgical disease of the kidney pelvis or cortex.

1. EXCESSIVE THIRST.

If you eliminate the thirst produced by certain poisons, such as sugar, alcohol, opium, chloroform, you will find that a consuming thirst is a danger signal in urology, because it is the first decided and noticeable symptom of uraemia, in that it appears before any alteration in the urine. Sir A. Clark,² one of our most revered teachers here, read a paper in 1884 on "Catheter Fever." He noticed that death occasionally occurred after using the catheter in cases of enlarged prostate, and he contended that this untoward result generally occurred in persons who appeared in perfect health, but whose urine was low in density. He considered such patients to have "renal inadequacy." Of course we had known for long that "renal inadequacy" was merely another term for a low form of interstitial nephritis, induced by prolonged back pressure exerted by an unrelieved bladder, and marked by filtration of water rather than the excretion of urinary salts; but we did not then know bacterial invasion was the fire which fell on the prepared "tow"—the weakened kidney. Even now the profession at large does not realize that this renal inadequacy is marked by *excessive thirst as an onset symptom*. This thirst is mostly due to diuresis—noticed or unnoticed. The symptom commences when the specific gravity of the urine is 1010, and it becomes very pronounced when it has sunk to 1004. It is then accompanied by a graver symptom of depreciated kidney

DESCRIPTION OF SPECIAL PLATES.

Fig. 1.—To show upper and lower polar aberrant arteries, and the area they affect.

Fig. 2.—Aberrant vessels to the lower pole underrunning the ureter and dilating the pelvis (Newton Pitt).

Fig. 3.—A one finger-tip pelvis (normal).

Fig. 4.—Kidney in section. A one finger-tip pelvis in which one or even more papillae are touched, showing the commencement of the pelvic dilatation.

Fig. 5.—A two finger-tip pelvis, showing elongation of kidney and some dimpling of cortex.