THE
British Medical Journal.

THE JOURNAL OF THE BRITISH MEDICAL ASSOCIATION.

LONDON: SATURDAY, JULY 6TH, 1918.

Observations

ON

**THE NATURE AND SYMPTOMS OF CARDIAC
INFECTION IN CHILDHOOD.**

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III.—RHEUMATIC ENDOCARDITIS.*

I. OF THE MITRAL VALVE.

THE commencement of rheumatic endocarditis of the mitral valve is bound up with the early acute dilatation of the heart, and the symptoms are very few. There need be no pain, and fever is not essential. An increase in the rate of the pulse, some breathlessness and possibly palpitation, if the child is not confined to bed, are suspicious, but these are due to the myocardial weakness rather than to the valvular disease. We have only to picture the minute lesions that constitute an early endocarditis to realize this lack of symptoms, and it is amply borne out clinically by the ease with which a primary cardiac rheumatism can be overlooked. I am nevertheless opposed to any attempt to minimize the importance of valvular disease, for though, no doubt, in former years overmuch attention was paid to it as the agent in cardiac failure, we cannot neglect its extreme value as evidence of organic disease, or forget that, as in mitral stenosis, it may be the predominant cause of the failure of health.

In acute rheumatism mitral endocarditis is extremely frequent—how frequent no one can say, because, as I have already pointed out, it is impossible in some cases to be sure as to whether the condition is one of simple dilatation alone. In fatal cases, however, it is practically invariable, for in 149 out of 150 there was a lesion of this valve.

Two Types of Mitral Endocarditis.

A point of importance is the distinction between the classical mitral incompetence and stenosis. In order to understand this distinction some attention must be given to the morbid processes in the two lesions. Every one is acquainted with the *post-mortem* appearances of the dilated incompetent mitral valve, and with the cicatrized tissues of advanced stenosis; but it is nevertheless my experience that, among students, there is a vague idea that the latter is an advanced stage of mitral incompetence. This, however, is only true in a very restricted sense. The ordinary lesion of mitral incompetence will never give rise to a classical stenosis, for such a result as this is a pathological impossibility. In mitral stenosis, as will be described later in more detail, the entire valve flaps, the ring, and the chordae tendineae are infiltrated by a subacute rheumatic process. I do not deny that between the two extremes there are grades which bridge over these differences, but if we are to understand the history of mitral stenosis we

must first recognize this essential distinction in the nature of its development, that it is a subacute process involving the entire structure of the valve. This particular form of chronic inflammation is more frequent in the female. The explanation of this sex peculiarity must necessarily be hidden from us until we have some knowledge of the nature of the rheumatic poisons. The occurrence, however, of a process of this kind in infection is not in itself remarkable, for the nature of a fibroid phthisis has for many years been thoroughly appreciated and is as unlike an acute broncho-pneumonia as tuberculosis as mitral stenosis is unlike an acute carditis. The results produced by these two types of endocarditis are very different, for while mitral incompetence may be of little importance, mitral stenosis constitutes one of the most formidable of cardiac affections. It is so common and its early history is so unpretentious that I am doubtful whether the true gravity of the lesion is even yet sufficiently appreciated. In adult hospitals we have become so familiar with these cases that they are almost in danger of becoming either problems in diagnosis or occasions for the demonstration of auricular fibrillation, while in children's hospitals the symptoms are as a rule so slight that the future of the patients is liable to be forgotten. If, however, we merge these two aspects into one we cannot fail to realize the dangerous tendencies of this form of endocarditis.

A. Mitral Incompetence.

In childhood the course of a case of mitral incompetence depends almost entirely upon the myocardium. If this is severely damaged by an acute carditis the case will run a disastrous course, compensation will never be effective, and invalidism quickly follows. On the other hand, if the myocardium is sound the course is favourable, if the physician and parents have patience and exert much care. Complete recoveries are in my opinion frequent, and this even when the bruit is audible over a wide area, including the back of the chest. In order to realize this such cases must be followed for some years, during which time several interesting facts will be noted. Thus the systolic murmur, which at first was obvious and classical, will become in time indistinguishable from a so-called functional murmur in its behaviour to change of position and cardio-respiratory phenomena. I am very sceptical of the book distinctions between functional and organic bruits, and prefer to rely upon a study of the patient rather than the bruit as the best means in differential diagnosis. Another interesting point is that the time may come when the patient goes away to school with a warning note to advise that he has had rheumatic endocarditis, and we hear later that we have been mistaken, and that there is in reality nothing amiss. Remembering the pathological changes in a mitral endocarditis, there is no reason why the vegetations, just as the rheumatic nodules, should not gradually shrink in the course of cure, and then the slight thickening that is left can have no importance when we bear in mind the contractile nature of the valve ring and the great power of the cardiac muscle to adjust itself to slight deficiencies. The real importance of a mitral systolic murmur may eventually lie in the evidence it affords of

* Articles I and II were published in the JOURNALS of March 2nd and April 13th.

the rheumatic attack, and this is of value, for rheumatism in the child is a disorder very likely to recur.

When mitral regurgitation runs an unsatisfactory course in childhood the symptoms of breathlessness and palpitation are soon apparent. The pulse is rapid and later irregular. The impulse, even if it becomes more forcible than normal, is diffuse and the transverse diameter of the heart enlarged. The systolic murmur is loud and long, conducted to the axilla and audible at the back, and often a bruit develops which is to-and-fro in rhythm. There is a tendency to bronchitis, the liver enlarges, and oedema appears. Eventually a condition strictly comparable to an adult case of severe mitral incompetence results.

It is remarkable how such cases resist remedial treatment, a result due to several factors, particularly weakness of the myocardium, adherent pericardium, and destruction of the tone of the valve ring.

B. Mitral Stenosis.

For a thorough understanding of mitral stenosis the early developments should be carefully studied in childhood. This is the more needful because the medical treatment is most unsatisfactory, and I am convinced that no single step in its history should be left unexplored, even if, as it seems to me at present, this only deepens the conviction of our powerlessness.

We are faced by a most insidious form of subacute inflammation the duration of whose progress is often measured not by weeks but by years. In its earliest stages the absence of symptoms may easily lull our fears; in the latest stages, when the systemic blood system has been throttled at its source, we are powerless to do more than ease the suffering. It is a lesion in which the valvular change is the essential factor, for we often find the myocardium remarkably healthy, and feel that if we could only free the ligature round the blood stream the heart would recover.

There is no doubt that this process of contraction may stop, and if this happens before much stenosis has occurred then life will be prolonged, but the lamentable feature is our inability to influence the process by medical treatment. However carefully we treat these cases by rest and medicine the stenosis often develops under our eyes, and we are driven to the second line of defence—that of minimizing by palliative measures the results that will follow.

It has been pointed out that there are cases of mitral endocarditis in which some scarring of the free edges of the flap occurs, thus causing a certain degree of narrowing; but in these cases the stenosis is of pathological rather than clinical interest. I have had one opportunity of observing not only the development of the condition from week to week, but of ascertaining the pathological changes in the valve after death in a case of true mitral stenosis of unusually rapid course.

This was the case of a little girl of 6 years who was under my care for five months in hospital suffering from severe myocarditis. In the first month there was apparently no valvular lesion; then there steadily developed the signs of a stenosis, with the usual thrill and presystolic murmur. The necropsy showed general thickening of the segments of the valve from subacute inflammation, with small areas of necrosis such as are seen in the rheumatic nodule. Had life been prolonged a general and severe contraction must have followed.

In lectures and papers I have several times drawn attention to the association between repeated attacks of chorea and the development of mitral stenosis in childhood. This association is one which I feel certain is of clinical interest, and I illustrate it by three examples out of a number of similar observations.

Example 1.

A. K. A., female, aged 6 years, came under my observation in August, 1907, for an attack of chorea, with multiple arthritis. The attack of arthritis was interesting because the metacarpophalangeal joint of the right great toe resembled a gouty arthritis. An occasional mitral systolic murmur was noticed at that date.

In October, 1907, there was another attack of chorea paralytic in type. In December, 1909, a third attack occurred, and now the first sound at the apex was noted to be short. In May, 1910, there was a fourth attack, and a systolic mitral murmur was audible. In November, 1912, there was a fifth attack, and now a presystolic murmur had developed. In January, 1914, there was a sixth attack, and the condition of the heart was a classical example of mitral stenosis.

Example 2.

C. C. A., female, aged 6 years. She was first under observation in March, 1907, suffering from chorea of seven days' duration; the only other rheumatic manifestation was a mitral systolic murmur.

In September, 1908, after a sore throat and a rheumatic erythema she developed headache and chorea and there was also mitral incompetence. In February, 1910, there was a third attack with active carditis but no evidence of mitral stenosis. In February, 1911, she had an attack of arthritis following a sore throat, but there was no obvious mitral stenosis. In May, 1912, a fourth attack of chorea occurred with fainting attacks, and the usual evidence of early mitral stenosis was obtained.

Example 3.

J. B., a boy, aged 3½ years. He was first under observation in May, 1908, for chorea of five weeks' duration.

In 1909 there was a second attack. In June, 1910, there was a third attack, when mitral regurgitation was first noticed. In January, 1911, there was a fourth attack, and in April a fifth with arthritis. In October a sixth attack commenced and mitral stenosis was first detected. In 1912 and 1913 further attacks developed, and by September, 1914, he had suffered from ten attacks of chorea and there was an obvious mitral stenosis.

These three cases and many others I have notes upon enable us to trace the development of mitral stenosis. It is obviously a very gradual process, taking many months and sometimes some years to become unequivocal, and during its development the child may never complain of cardiac symptoms. The chorea reminds us, however, that the rheumatic infection is active on frequent occasions, and we are led to realize how stealthy may be the progress of the rheumatic infection in the human tissues. Further, these cases lead us to understand clearly how in an adult we may meet with well-marked mitral stenosis and yet obtain no history of rheumatism, for if the heart is primarily affected and there is no chorea an advancing stenosis may occur without any symptom that calls attention to its presence.

Mitral stenosis is, I think, not by any means an easy lesion to diagnose in the early stages, and a knowledge of the history of the child's health in recent years often gives us valuable information. The presystolic thrill is at first very local and accurately limited to the region of the heart beat. A systolic murmur is often audible, but there are cases in which it is never heard although all the cardinal signs of stenosis appear. In others, again, the systolic murmur gradually fades away, but the first sound becomes short and sharp and a presystolic murmur appears. It is easy enough, if the heart is examined just at the time when the systolic murmur is disappearing, to overlook the character of the first sound and pass the heart as sound. The persistent third sound, commonly expressed by Zubb-tut-tut, is well recognized, as also the presystolic, mid-diastolic, and diastolic types of the rumbling murmur.

Attention must be drawn to a to-and-fro murmur, blowing in character, which is not infrequently met with in the mitral disease of childhood. It is very like the to-and-fro murmur heard in adult aortic disease, and does not point to mitral stenosis. Both these murmurs are soft and blowing, and a necropsy will show a widely dilated mitral opening with a large dilated heart and very probably also an adherent pericardium. It is in my opinion a sign of value in mitral incompetence pointing to a severe lesion, and is one often met with in the disastrous cases alluded to under that section.

In the early stages of mitral stenosis symptoms are usually absent, and it is a sound rule in prognosis to look upon the development of definite symptoms in young children as a serious matter. There is, however, one which is of great interest and difficult of explanation. This is tachycardia. Physicians are well acquainted with tachycardia and its danger in the advanced disease of adults, but the same may occur in childhood from the earliest appearance of the stenosis. It is not, however, dependent upon the stenosis, for it may even pre-date it. The following example will illustrate this occurrence.

Example 4.

L. G., a girl aged 5 years, whose father had suffered from rheumatic heart disease, was brought to hospital in March, 1902, suffering from chorea. She lived in a damp house near the Thames, and for four months had become increasingly nervous, until at last chorea developed. The feature of the case was a persistent tachycardia, the pulse varying between 120 and 148 in the minute. The heart was dilated.

She was kept in hospital for four months, and though treated very strictly, the tachycardia persisted until June, when, after

removal of decayed and septic teeth with subsequent administration of the bromides, it disappeared. A systolic murmur at the apex was occasionally noticed when the tachycardia was unusually severe.

Six weeks after leaving the hospital she returned, very excitable and with a recurrence of the tachycardia, but there was no cardiac murmur. A month later some irregularity of action developed, and the impulse of the enlarged heart was very forcible. The thyroid gland was palpable, but there were no other signs of exophthalmic goitre.

In 1906 the condition remained much the same, and there was one attack of chorea. Nervousness and tachycardia were very constant, and the pulse-rate sometimes reached 156 to the minute. The first sound was becoming more abrupt, but no murmur of any kind could be detected. I saw the child again seven years later in 1913, at the age of 16, and found then severe mitral stenosis with tachycardia, cyanosis, and the same remarkable nervousness.

This is an extreme example; far more frequently we meet with cases in which, after a rheumatic attack, the condition has seemed satisfactory, except that the action of the heart has been persistently more rapid than normal, and, except for some shortness of the first sound, no other evidence of actual disease has been apparent. In such cases I have been struck with the frequency with which mitral stenosis eventually develops. This tachycardia is much influenced by emotion; the child does not, as a rule, complain much, but there is a general weakness and sense of exhaustion.

In the adult all physicians are acquainted with severe tachycardia in mitral stenosis, which is sometimes persistent and sometimes paroxysmal, and recognize it as one of the gravest symptoms in the final stages. Of particular interest to me is the tendency for some cases to develop this symptom even before any stenosis is apparent, and often in the early stages when the mechanical difficulties must be small; and I would dwell upon the gravity of this phenomenon as one which tends to complicate the history of the illness with increasing weight as the lesion progresses and, owing to the nervous disturbances so constantly associated, to aggravate the discomfort of the patient. This symptom must not be confused with the rapid action produced in a nervous child by arrival in hospital among strange faces and sounds. These factors will naturally add to the symptom, but the obstinate swiftness of the heart beat in these cases is easily distinguished when the child has been under close observation for some time.

When symptoms develop in childhood, they do so along different lines in different cases. Some show embolic phenomena early, and these are likely to recur, and may end fatally in a cerebral attack. In others bronchitis is the first serious event. In others an acute carditis may entirely alter the outlook, or a malignant endocarditis may supervene. Lastly, in some cases the stenosis proceeds apace, and we have the picture of the disease in its last stages, as it occurs in adult life, and may even see the child lingering on with gangrene of fingers or toes.

II. AORTIC AND MITRAL DISEASE.

The importance of this combined lesion in childhood has not, I think, attracted the attention it deserves. It is not that I imagine any peculiar mechanical disability results from this combination; on the contrary, it is apparent that the general trend of such cases will be mitral if that lesion is predominant, and aortic if the aortic valve is chiefly damaged. The great feature of interest lies with the infective process rather than with the mechanical disability, for in many cases where both valves are affected we are confronted with a severe type of cardiac rheumatism, and one which may terminate in a malignant or progressive endocarditis in early adult life. Later some figures will be given to illustrate this point. Meantime I would point out that this terminal malignancy is not by any means always the cause of the involvement of the second valve, for the necropsies show, and the former history has proved during life, the existence of these two lesions in the so-called simple phase. The aortic lesion usually develops later than the mitral, and the valve may be damaged during the same illness, or in a recrudescence, or in a later attack. The essential feature in this condition is, in my opinion, the spread of the infection. This places it in position between a simple endocarditis of one valve and a malignant endocarditis involving two valves or spreading to the cardiac chamber.

Several practical points about this combined lesion deserve attention. The commencement of an aortic lesion in a child is very easily missed, for in itself it gives rise to no definite symptoms and requires for its detection a routine and careful examination of the heart. In some cases I have noticed its development coincides with a rise of temperature after the first bout of fever has subsided, which, though not severe in degree, remains persistent for some days or longer. The steps in the development are that a systolic murmur appears in the aortic area and the second sound becomes faint, and then later the diastolic murmur develops. The aortic lesion is the more easily missed because the diastolic murmur in childhood is heard most clearly to the left of the sternum in the third space or behind the sternum itself, or again at its lower end. Students, in my experience, repeatedly miss this physical sign from ignorance of this fact, and also because they are misled by the systolic murmur over the mitral area. It may seem a gross mistake, but it is one easily made, to confuse the diastolic aortic murmur to the left of the sternum with the systolic mitral murmur conducted upwards. The involvement of the two valves by the infection is well explained by their close anatomical relations.

It is my belief that the aortic lesion may be sometimes recovered from, and it is based upon clinical observation and the study of necropsies. I have observed the diastolic murmur disappear and have also seen at necropsies slight damage to the aortic valve which has, during life, given rise to no physical signs. Putting these two observations together, I believe it possible for an aortic valve to heal as effectively as the mitral, though less frequently.

These combined lesions represent by far the majority of cases of aortic regurgitation in early life, and it is not, in my opinion, correct to attribute the systolic mitral murmur to a relative incompetence of the valve secondary to the aortic disease. As I have already stated, the mitral lesion is the earlier, and the result of endocarditis.

Aortic regurgitation is not frequent as a solitary lesion, but it does occur, as experiment proves, for an intravenous injection may produce an endocarditis of this valve alone. In children also we occasionally find aortic regurgitation with no evidence of a mitral lesion.

The practical outcome is that if in the combined lesions the aortic predominates, the case is to all intents and purposes one of aortic regurgitation, unless the mitral lesion has developed into a stenosis. In that event we have another important valvular defect in the combination of aortic regurgitation and mitral stenosis.

Signs and Symptoms.

The physical signs in aortic regurgitation, once established, rapidly develop their characteristics in the young. The pallor, the strongly beating vessels, the large heaving heart, and the collapsing pulse, all appear in due course, and may, as is well known, reach in young adult life an extreme degree.

Breathlessness, pain over the heart, and epistaxis, are frequent, but angina not so frequent as in the adult, for the aorta is seldom severely damaged. Nervousness is a symptom upon which I would lay much emphasis. These children suffer much from it, and may be quite unable to attend school for this reason. No symptom, in my experience, is more often mentioned by the parents.

Compensation is often remarkably effective, and these children may be remarkably active. I have in my notes a case in which such a patient became a first-class cricketer, though, let me add, not with my sanction, for I think it is a very extravagant use of the cardiac reserve to employ it in strenuous games, and even if there is no disaster, nevertheless it must shorten a useful life; that man, however, led a happier life, in my opinion, than one who has been frightened into invalidism because he has some degree of aortic regurgitation. The happy mean must be the obvious line to take, and with the young heart the aim should invariably be to make the life as full and not as empty as possible.

As a rule these children do not die suddenly but succumb to a renewed attack of carditis. Later in life they may die in the same way, or may strain the heart in an entirely improper occupation in life, or develop malignant endocarditis, or, again, die suddenly or suffer from the mitral lesion and its consequences. When the heart fails from a carditis or from general breakdown of compensation,

the signs and symptoms of the aortic lesion become much less obvious and may be entirely overlooked until recovery sets in and the bruit reappears.

Table illustrating the Relation of Aortic and Mitral Disease to Malignant Endocarditis.

No.	Sex.	Rheumatic Attack, Mitral and Aortic Disease.	Death from Malignant Endocarditis.
1	M.	In 9th year	In 10th year.
2	M.	In 10th year	In 13th ..
3	M.	In 6th, 8th, 10th, and 12th years	In 16th ..
4	F.	In 12th and 13th years	In 14th ..
5	F.	In 7th and 20th years	In 21st ..
6	M.	In 11th, 14th, 17th, and 18th years	In 19th ..
7	F.	In 12th year	In 16th ..
8	F.	In 9th and 16th years	In 11th ..
9	F.	In 12th year	In 16th ..
10	M.	In 7th year	In 11th ..
11	F.	In 12th and 15th years	In 17th ..
12	F.	In 8th, 13th, and 15th years	In 24th ..
13	F.	In 13th and 17th years	In 27th ..
14	F.	In 7th year	In 12th ..

This group strengthens, I think, the view that I hold that aortic and mitral disease is not of importance only because two valves are affected, but because the lesions point to a tendency to spreading activity and the possibility of malignant development.

As already stated, aortic regurgitation as a solitary valvular lesion is exceptional in childhood, and for the purposes of this article sufficient attention has been given to it under the combined lesion just described. Aortic stenosis and tricuspid endocarditis occur in childhood, but they illustrate no special principles and need not detain us.

MALIGNANT ENDOCARDITIS.

Space will not permit any detailed description of this deadly disease in childhood, which resembles in its behaviour the condition in adult life; but I would repeat here my belief that the rheumatic infection is a frequent and important cause, and will very briefly state the reasons.

There is frequently an antecedent history of rheumatism; it may occur together with such active symptoms as arthritis and pericarditis, and these may be overcome although the valvular lesion may run a fatal course. Both simple and malignant endocarditis may occur in the cardiac valves in the same case, and connecting links between simple and malignant endocarditis may be met with in childhood. Experimental inoculation with the rheumatic infection may produce both forms of endocarditis.

I look upon simple endocarditis as evidence of destruction of the micrococci by the tissue resistance, and malignant endocarditis as proof of the failure of that resistance. I cannot accept the view that septic micrococci have a predilection for damaged and sclerotic valvular tissues, but I think it highly probable that in the necrotic areas of vegetations micrococci may remain latent and spring into virulence. How is it that in written answers we find examinees insisting upon the multiple abscesses found in the viscera in malignant endocarditis? The great feature is the usual absence of such abscesses, and the term arterial pyaemia is as a rule quite incorrect.

It need hardly be added that I do not suppose rheumatism to be the only cause of malignant endocarditis, which we all admit may be produced by various infections. Lastly, I venture to think that more attention directed to the supervision of patients with organic heart disease would throw light upon the all-important factors tending to cause this most dangerous condition.

It follows from the above considerations that I recognize three cardinal processes in endocarditis: (1) The simple; (2) the sclerosing; and (3) the malignant.

NEPHROTOMY COMBINED WITH CAESAREAN SECTION IN THE TREATMENT OF ECLAMPSIA WITH SUPPRESSION OF URINE.

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THE number of patients suffering from eclampsia appears to have increased recently, this complication having occurred at Queen Charlotte's Hospital one and a half times more often during 1917 than in the year before the war. Hence the question of its treatment, important at any time, is now the greater.

Before discussing whether transperitoneal incision of the capsule of the kidney may be a useful addition to Caesarean section, it is necessary to consider in what type of eclamptic patient Caesarean section should be performed. Stated briefly, the indications for Caesarean section are the occurrence of fits or the onset of severe toxæmic symptoms in a primigravida with an undilated cervix, especially if little urine is being secreted and if generalized oedema and cyanosis are present. The advantage of Caesarean section in such cases is that no rapid method of delivery *per vaginam* is possible except at the cost of local trauma and shock that exceeds that of laparotomy. If the view be taken that such cases are better left undelivered and treated on general lines, we are faced immediately with the undesirability of infusing fluid into the veins or subcutaneous tissues of an oedematous patient who is frequently already bronchitic, and the equally undesirable practice of giving large doses of morphine to a cyanosed patient. One of the few drugs that is still available in such cases is veratrine, and this, unfortunately, frequently fails. After Caesarean section the eclamptic seizures rapidly improve in the great majority of cases, but in some there is difficulty in eliminating the toxin, owing to more or less marked suppression of urine.

It is usual for eclamptic patients to pass only a diminished quantity of urine for some days after delivery, and this diminution is considerable in quite a large number. The occurrence of acute suppression of urine in toxæmic patients immediately after delivery, although uncommon, is well known, and is frequently fatal. The pathological explanation generally accepted is that the suppression of urine is due to the thrombosis of the intertubular vessels, with consequent necrosis of the renal cortex. This theory seems to rest on the publication of very full and careful *post-mortem* records of certain classical cases.

Bradford and Lawrence (*Journal of Pathology and Bacteriology*, 1898, vol. v, p. 195) recorded the case of a multipara who had practically complete suppression of urine after delivery till her death on the seventh day. There were none of the usual symptoms of acute uraemia—vomiting, delirium, coma, dyspnoea, and amaurosis being absent. *Post-mortem* the convoluted tubules were found to be necrotic and the intertubular arteries thrombosed.

Herringham and Griffith (*Ibid.*, 1906, vol. xi, p. 237) gave details of a patient who, from the time of delivery till her death nine days later, passed only ten ounces of urine. The ordinary symptoms of uraemia were absent till the end. *Post-mortem* the kidneys showed cortical necrosis and thrombosis of the intertubular vessels.

Lloyd (*Lancet*, 1909, vol. i, p. 156) recorded the case of an eclamptic patient from whom only an ounce and a half of urine was collected from the time of delivery till her death on the eleventh day. There were slight twitchings from the seventh day onwards, but no fits or other symptoms of uraemia. The *post-mortem* description of the kidneys is that together they weighed seventeen ounces. Microscopic examination showed coagulation necrosis of the cortex; the large arterioles were not thrombosed, although the intertubular vessels were, and the intertubular spaces infiltrated with leucocytes.

It is of paramount importance to consider whether this explanation is correct in the majority of cases, as if primary renal thrombosis occurs and is the cause of the suppression, it is in the highest degree unlikely that any surgical interference will save the patient's life once the symptoms are established. There are, however, reasons for doubting if this explanation is correct in all—or any—cases. In 1909 Weber¹ criticized this pathology, and pointed out that "there is nothing in the description of the thrombi to indicate that they were formed several