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OF

BRIGHT'S DISEASE

WITH

CHANGES IN THE VASCULAR SYSTEM.

With Illustrations from the Sphygmograph.

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ON THE

CONNECTION OF BRIGHT'S DISEASE WITH CHANGES IN THE VASCULAR SYSTEM.

It has long been recognised that under the common name of Bright's disease are included conditions widely differing from each other both in pathology and causation. It may be regarded as generally agreed by all writers on this subject in England that under this name are comprised three principal forms of disease :—

1. Tubal, or Epithelial Nephritis, affecting mainly the epithelium of the tubes, and ending in the production of the large, smooth, white kidney. 2. Cirrhosis of the Kidney, or Interstitial Nephritis, affecting mainly the intertubular stroma, and leading to the granular, contracted kidney. And 3, Lardaceous disease, affecting mainly the arteries.

It is true that there are some who maintain that chronic Bright's disease with contracted kidney commences always by an alteration of the epithelium of the tubes. Thus Dr. Johnson includes it in his class of "Chronic Desquamative Nephritis;" but even he would allow that it differs in an important degree from the more acute disease associated with the large white kidney, and termed by him "Acute Desquamative Nephritis," by the fact that it may be chronic and insidious from its commencement. On the Continent, however, this distinction is not so generally admitted, and both

Frerichs and Niemeyer hold that the contracted kidney is the later stage of the same condition which exists in the large white kidney.

It was observed by Dr. Bright himself, and by all succeeding writers on the subject, that chronic Bright's disease is not a merely local affection, but is frequently associated with important changes throughout the body, of which the most marked are thickening of the arteries, and hypertrophy of the left ventricle of the heart. It has long been taught that these changes are especially characteristic of a granular kidney; but the question has remained undecided, whether they are the consequence of the renal disease, or whether both are the results of some common cause. Probably the majority have hitherto believed that the change in the kidney is the prime cause, though opinions have been various as to what is the mode of causation. Recent discussions, however, have raised the question whether this form of Bright's disease ought not to be removed altogether from the category of diseases of the kidney, and ranked as a general change in the vascular system, of which the renal affection is only a subordinate part, and which may work its most perilous effects in other parts of the body, whilst the kidney remains unchanged.

It has been further argued that this condition cannot fairly be brought within the definition of a disease, but is rather a senile change, and that thus the present conventional mode of treatment of it as a disease is worse than useless.

There are then two questions open to discussion:

1. Are the changes in the vascular system entirely confined to cases of granular kidney; or do they occur also in some degree in association with other forms of Bright's disease?

2. Are they the effects of the renal disease; or are both parts of some general condition?

I have endeavoured to obtain some evidence bearing on these questions—

1. From an analysis of all the cases of Bright's disease

contained in the *post mortem* records of Guy's Hospital during four years, 1869, 1870, 1871, 1872.

2. From microscopic examination of small arteries.

3. From observations on the pulses of some cases of Bright's disease, by means of the Sphygmograph, and on their eyes by means of the Ophthalmoscope.

At the outset I am met by a certain difficulty in classifying the cases. It is allowed that in any given kidney either any two of the three forms of disease, or even all three of them, may be combined in any proportion. But does tubal nephritis in its latest stage lead to wasting of the kidney, so that it comes to resemble in some degree one that has undergone cirrhotic change? I believe most authors hold that this does occur, and a distinction has been made as to the mode in which a granular surface is produced in the two cases; that in a cirrhosed kidney it is caused by contraction of fibrous tissue compressing the tubes, but that in the last stage of tubal nephritis it results from simple wasting of those tracts of tubules which have been rendered incompetent by inflammation. Now it is not uncommon to meet with such a case as the following. A patient's illness commences perhaps two years or more before his death, by symptoms of acute nephritis, with dropsy. It continues throughout to have mainly the character of tubal nephritis, but as it proceeds the urine becomes copious, and of lower specific gravity. After death the kidney is found to show chiefly changes of epithelium, but its surface is somewhat granular, and its cortex rather wasted with excess of fibrous tissue. I will here give very brief notes of a case which may have been of this nature. I have chosen that of a young person, because in such a case an antecedent insidious cirrhotic change is much less probable.

George G———, aged 17, was admitted October 12, 1869, with pleuro-pneumonia. Ten years before he was in hospital with dropsy, coming on after exposure to cold, and his urine was then scanty and dark. He left the hospital with his

dropsy cured, but his urine still contained albumen. He had afterwards occasionally swelling of the feet, and six years later he had pleurisy.

On admission his urine was of sp. gr. 1011, copious, containing much albumen. There was slight œdema of face and feet.

After death the kidneys were found very pale and opaque, their surface somewhat granular, the cortex rather thin in proportion; they weighed 10½ ounces. Microscopic examination showed some wasting of Malpighian bodies, and excess of fibrous stroma. The heart weighed 12½ ounces, the left ventricle was hypertrophied, and the arteries of the kidney were manifestly thick.

Now, if cases of this kind be regarded as tubal nephritis the question is settled that the changes in the vascular system do not belong solely to cirrhosis of the kidney, for in almost all of them the heart is hypertrophied. Thus in the cases enumerated by Dr. Grainger Stewart as fatal in the third stage of tubal nephritis there was in every one hypertrophy of the heart without other cause to account for it. But in all of these the surface was somewhat granular, and the cortex somewhat wasted. It may be argued, therefore, that in all an insidious cirrhotic change had preceded the acute attack; and there can be no means of settling this question, since such a change in its earlier stages is attended by no symptom that would attract notice. It is necessary, therefore, to exclude such cases in ascertaining the associations of tubal nephritis, and I have reckoned all of them among granular kidneys.

But the case is different with a kidney which presents to the eye the usual characters of tubal nephritis, but under the microscope shows some excess of fibrous stroma. There would here be no warrant for assuming that the disease had commenced as an interstitial cirrhosis. So far as my own observation has gone I have found the stroma always somewhat increased in cases of large white kidney in which the

disease had been of long standing. Moreover, it is our experience of chronic inflammation in other organs that it leads to an increase of fibrous tissue in its vicinity, and indeed the same thing results from merely mechanical congestion when long continued.

Referring now to Table I., relating to granular kidneys, I will first notice the very frequent association of atheromatous arteries. This relation agrees with common experience, and has an important bearing. It has been usual in England to speak of atheroma as a degenerative change, but on the Continent it is regarded rather as the result of inflammation, and observations have recently been published in this country, showing that in its early stage there is an active proliferation of cells, such as occurs in inflammation. We have thus associated with granular kidney not merely a thickening or fibroid degeneration of arteries, but an arteritis which may be comparable to the other inflammations common in the course of Bright's disease.

It will be seen that there were ten cases out of sixty-six of decidedly granular kidneys without any hypertrophy of heart. This is not a proof that a granular kidney is not a cause of hypertrophy; for there may have been some failure in nutrition, owing to which the heart did not respond to stimulus. But, on the other hand, we may infer that the absence of hypertrophy in any one given case does not absolutely prove that case to differ in pathology from other granular kidneys.

That condition of heart which is especially characteristic of Bright's disease, namely, simple hypertrophy of the left ventricle, though much the commonest, yet occurred in only half of all the cases in which hypertrophy was recorded. It is easy to understand that if increased pressure in the arteries be the cause of hypertrophy, it is liable also to cause dilatation.

The table of the ages at which granular kidneys were found may throw some light on the question whether they are the

result of disease or senile change. It is a difficult one to answer, since no one has ever yet given a satisfactory definition of the word disease. I should hesitate to give the name of senile to a change which in eight cases out of seventy-nine was found under the age of thirty; and which may occur, as in one of the cases published by Sir W. Gull and Dr. Sutton, in a child of nine. But the important fact remains that it is a change to which the liability rapidly increases with age.

It has been argued that if the disease occurs in the young the hypertrophy of the heart ought to be much greater, since in them the response to stimulus would be more rapid. This conclusion I do not find altogether verified. Hypertrophy occurred in six out of the eight cases under the age of thirty, thus showing that the causes producing hypertrophy were at work; but the average weight of the heart was only 12 ounces as compared with a general average of 15 ounces. I imagine the explanation to be that in the young the disease cannot have run so protracted a course, and that there has not been time for the development of that atheroma of arteries which must largely contribute towards the causation of hypertrophy.

In order to determine the effects of atheroma by itself, I have collected for comparison twenty-three cases in which the arteries were found very atheromatous. These were all cases of aneurism, senile gangrene, cerebral hœmorrhage, or cerebral softening. I have taken only those in which the kidneys were healthy, and also the valves of the heart, and in which there was no other apparent cause for heart hypertrophy. The heart was hypertrophied in thirteen out of the twenty-three, and its average weight was 12½ ounces. Comparing this with the average of 15 ounces for cases of granular kidney, it would seem that, although atheroma of large arteries tends to cause hypertrophy of heart, yet this cause would not be sufficient to account for the whole of the effect produced in Bright's disease, especially when it is remembered that in that case the heart may be hypertrophied and yet no athe-

roma be found. I have drawn the same conclusion from sphygmographic observations, to be referred to hereafter; for in cases of very rigid arteries where there is no evidence of kidney disease, although the arterial tension is greater than usual, it is yet never so great as in many cases of granular kidney.

Referring next to Table II. relating to cases of tubal nephritis (excluding those of scarlatinal dropsy), of which there were only twenty-five in the four years, it will be seen that there is a distinct association with hypertrophy of the left ventricle, although not so decided as in cases of granular kidney. Hypertrophy occurred in half the cases, and the average weight of the heart was 13 ounces. It will be seen, moreover, that in many of those cases in which no hypertrophy of the left ventricle was found, the disease had only lasted a few weeks.

But turning to the cases of lardaceous disease, the result is very different. In only one case not complicated with granular change was the heart hypertrophied, and its average weight was only $9\frac{3}{4}$ ounces.

Now, if the hypertrophy of heart were generally due solely to the degeneration of the coats of arterioles, it would be strange that in lardaceous disease, in which such degeneration is much greater and more undoubted, and in which it may be diffused through at least all abdominal organs, no such hypertrophy is found. Indeed, it would seem that no perceptible obstruction is so caused to the blood current, for it is well known that a simply lardaceous liver gives rise in general to no ascites.

The general conclusion is that hypertrophy of the left ventricle belongs especially to a granular kidney, but in some considerable degree also to tubal nephritis of long standing, and this is a strong argument for the view that it results from chronic Bright's disease as a disease of the kidney, and is not a common result of some third condition.

Its absence in cases of lardaceous disease may be explained

on the ground that a lardaceous condition of the kidney does not notably interfere with its power of secretion until quite a late stage, when complicated with catarrhal nephritis.

The most crucial proof of heart hypertrophy being a consequence of renal disease would be to find it occur in cases of kidneys wasted from some cause other than Bright's disease. But in such cases the other kidney generally hypertrophies, and, as might be expected, no effect of renal disease is produced. I have collected, however, in Table III., several cases in which hypertrophy of heart appeared to me to be at least partially due to a condition of this kind.

I believe the old opinion to be the true one, that the heart hypertrophy is due to the altered quality of the blood. It is proved by the case of asphyxia, that the blood may be so changed that it will not pass through the capillaries. Some well-known experiments were made upon animals, by Dr. J. Reid, in which it was found that in the first stage of asphyxia the unaërated blood passed freely through the lungs, but was impeded in the systemic capillaries, so that the pressure in the arteries increased, and that in the veins diminished. In the later stage, after about two minutes, the blood was arrested in the pulmonary capillaries, and the pressure consequently rapidly diminished in the arteries, and increased in the veins.

Dr. Johnson has attributed to the small arteries an action like that of stop-cocks. He supposes that, the blood being noxious to the tissues, the small arteries resist its passage, and thus act in antagonism to the heart, so that both are equally hypertrophied. Of this contraction of the small arteries there is no proof, and the theory appears almost to attribute to heart and arteries an antagonism of intention, as if they were actuated by volition. It seems simpler to suppose that the impediment lies in the capillaries, and is due to a modification of capillary attraction between the blood and the walls of the vessels, and that, the arterial pressure being thus increased, the muscular walls of heart and arteries are both hyper-

trophied in concert, since both have to act against greater resistance.

Evidence is not wanting that the circulation in living bodies cannot be explained wholly by the mechanical conditions of heart and arteries, but depends also upon chemical or vital forces. In plants and in the lower animals, where there is no heart, the circulation certainly is due to these. Moreover, the phenomenon of stasis in inflammation cannot be explained by mechanical conditions, but must be supposed to depend on a vital relation between the blood and the walls of the vessels. Again, it is common to see persons, in whom there is no deficiency of heart-power, but who show all the signs of a sluggish circulation. Their hands and feet are always cold, and their skin purple, from blood lingering in the capillaries. In them there seems to be a passive congestion, due to a deficiency of tissue change. Many other proofs are collected by Dr. Carpenter to show the existence as a motive force of a "capillary power," that cannot be explained by mechanics. The most important of these are an instance of an acardiac fetus, in which the circulation went on up to the time of birth, and several cases in which the capillary circulation was proved to continue for some little time after death. Since, then, there is so much proof of the existence of this chemical or vital capillary force, it is more natural to suppose it modified by an altered state of blood, than to assume a continuously contracted state of the small arteries, which is entirely hypothetical.

The function of the muscular coats of arteries has been generally supposed to be to regulate the circulation by altering the calibre of the arteries at different times, and it is impossible to doubt that this effect does occur. Some, however, have supposed that they also act as a motive power by a kind of peristaltic action. Thus, a rythmical contraction has been observed by Wharton Jones in the wing of a bat, by Schiff in the ear of a rabbit, and the same has been seen in the retina by the ophthalmoscope. Now a peristaltic wave

in a tube would tend to produce a current in the liquid of its own velocity, and it would, therefore, accelerate a slower current, but retard a quicker one. Therefore no peristaltic wave could accelerate the arterial stream, unless it travelled with the velocity of the pulse wave. It is thus evident that no such slow rythmical motions as have been observed could assist the arterial flow. And it is inconsistent with the usual character of involuntary muscle to suppose it capable of transmitting a very rapid wave of contraction. The arteries themselves, indeed, when made to contract by artificial stimulus, do so slowly and gradually.

But whatever view be taken of the function of this arterial coat, in either case the resistance it has to overcome in contracting is the pressure of the blood in the artery, and if that pressure be increased, the muscular coat is likely to become hypertrophied.

I will notice only one other theory of the causation of hypertrophy, namely, that adopted by Traube. He considers it due to two causes: 1, The increased pressure in the vessels due to the retention of water; 2, The direct resistance caused by the impediment to the circulation through a contracted kidney. It is a fatal objection to the first that the heart is most hypertrophied in those cases in which the excretion of water is increased rather than diminished. His second cause would be a true one as far as it goes; but the quantity of blood passing at any moment through the kidney must bear such a small proportion to the rest of the circulation, that it could scarcely have a very important effect.

It remains next to consider the direct evidence afforded by microscopic examination as to the state of arterioles and capillaries. I have examined only those from the pia mater and arteries in the kidney itself. In the estimation of the hypertrophy of arteries there is a fallacy to be avoided. It is impossible to detect change in the absolute size of a small artery, since there can be no criterion what its natural size at that point ought to be: the only thing, therefore, open to

observation is the relation of its calibre to the thickness of its walls. Now, an artery is subject to *rigor mortis*, and, if placed in a hardening agent while in that state, its contracted condition is made permanent. In order to avoid this difficulty as far as possible, I have used no hardening agent for the pia mater, and in the case of the kidney I have placed it in chromic acid only after an interval of not less than forty-eight hours after death. Another consequence results from this impossibility of detecting by the microscope change in the absolute size of small vessels—namely, that it has not been, and cannot by this means be, proved that any thickening of the walls encroaches on the calibre of the vessel. Indeed we should suppose it is not so, if we judge by analogy from the case of the larger arteries, for as their walls are thickened they generally become dilated. But if the channel is not diminished, it is not clear why any obstruction or hypertrophy of heart should result. It is true that in the case of the larger arteries a mere loss of elasticity tends to cause hypertrophy of the heart, for if they cannot dilate to admit a larger quantity of blood during the time of the heart's contraction its action is made more laborious. But this effect occurs chiefly in the larger vessels, and especially in the aorta; and the minute vessels and capillaries must bear comparatively so small a share in it, that we should not expect that a mere loss of elasticity in them would produce any perceptible hypertrophy of the heart.

The small arteries of the pia mater I have examined first in a little salt and water of sp. gr. 1020, and have afterwards added glycerine at the side of the glass, in order to observe any change produced without disturbing them. I have found, as I believe, that in cases of granular kidney both muscular and external fibroid coats are thickened. On the addition of glycerine, the external fibrous coat and the tunica intima become somewhat swollen after a time, and the former assumes what has been described as the "hyalin-fibroid" appearance. I have never seen this without the use of

glycerine, and I do not think that it indicates the presence of any special material. If glycerine, very slightly acidulated, be used, the external coat becomes at once much swollen and very hyaline, and the tunica intima becomes wavy and its longitudinal fibres more distinct. This appearance is produced almost as much in a perfectly healthy artery.

In healthy arteries the thickness of the external fibrous coat varies much in different places, and some may be found here and there equalling in this respect those associated with granular kidneys. But in the latter the thickening of the external coat is more general. It is equally so, however, in some other cases in which kidneys and heart are both quite healthy—as in old men whose large arteries are atheromatous, and in drunkards and others in whom the membranes of the brain are thickened.

In a case of large white kidney of long duration, where the heart was hypertrophied, I found the arterioles corresponding in all respects to those in cases of granular kidney. In the case of a child aged five, who had had lardaceous disease probably for more than two years, the arteries were quite thin. This corresponds to the absence of heart hypertrophy in such cases.

In the kidney itself, both in cases of granular kidney and in that of large white kidney before referred to, the arteries appeared to be hypertrophied. It is somewhat difficult in a transverse section to distinguish between the circular muscular fibres and the fibrous tissue outside them; but it appeared that in the granular kidneys there was much more of fibrous tissue around the artery, and shading off into the kidney substance near.

As to the state of capillaries or minutest arterioles, I have found a granular, or apparently fatty, condition common in cases of granular kidney, but not unfrequent also in other cases, especially in old persons, where the heart is not hypertrophied.

My general conclusion is that microscopic examination

does not reveal such a change in arterioles or capillaries as to show that the prime cause both of kidney degeneration and heart hypertrophy must be sought for here. I believe that hypertrophy of the muscular coats of arteries, just as much as hypertrophy of the left ventricle, is the natural sequence of increased arterial pressure. On the other hand, a degenerated condition of capillaries or arterioles is comparable rather with the atheromatous condition of large arteries, since both occur in advancing life, under other conditions, but both are more marked in chronic Bright's disease. That a degenerated condition of small vessels does exist, and not merely a universal hypertrophy, is shown by the occurrence of epistaxis and of hœmorrhages in the retina, for these bear the same relation to the condition of minute vessels which cerebral hœmorrhage does to that of the arteries of the brain.

Before proceeding to consider the evidence derived from the sphygmograph, it is necessary to make a short digression as to the interpretation of the secondary waves seen in the pulse tracing. I shall merely state my own view, not having space to discuss the proofs of it, although it differs somewhat from that generally accepted.

In the healthy pulse (vide Plate I.), there is seen in the descent after the first high upstroke the first secondary wave (*b*), which should be but little marked; after this comes the principal secondary wave or dicrotic wave (*c*), and this is sometimes followed by yet another wave (*d*). It is generally said that the first upstroke or "percussion wave" is due to the shock communicated through the blood column from the first impulse of the heart, and some add that this is transmitted instantaneously; while the first secondary wave or "tidal wave" travels more slowly, with a velocity due to the elasticity of the arterial walls, and indicates the distension of the artery by the flow of blood. But experiments with an artificial heart readily show that the wave causing the first upstroke does not travel instantaneously, and that it is coincident with the flow of liquid. Moreover the most violent

impulse so communicated as not to cause any flow of liquid, produces no upstroke, but only a slight quivering of the lever.

The true explanation is that the "percussion" and "tidal" waves form in the artery but one wave, and are only separated by the sphygmograph. Owing to the inertia of the long lever it is carried up a little too high, and when in falling it meets the true arterial wave, it is again tossed up, and thus forms the tidal wave. I have endeavoured to represent diagrammatically the relations of the tracing to the true pulse wave in Plate I. I shall, however, use the terms "percussion" and "tidal" wave as being convenient. Probably the tracings are more useful as clinical indications than if they represented more accurately what occurs in the artery. If the sphygmograph used have a secondary spring to keep down the long lever, the tidal wave may be replaced by two or even by a jagged line. Such a spring is better omitted, because it is apt thus to introduce oscillations of its own.

The dicrotic wave is due to a reflection from the aortic valves at the moment of their closure. If the tension in the arteries is low, the valves close slowly, there is a considerable fall of pressure before their closure, and thus the dicrotic wave is more marked. Hence a dicrotic wave small in proportion, provided the aortic valves are healthy, is an indication of high pressure in the arteries. The wave which sometimes follows the dicrotic bears the same relation to it which the "tidal" does to the "percussion."

In tracings taken in Bright's disease perhaps the most important indication of any is the amount of pressure applied to the artery. In the sphygmograph I have used this is regulated by an invention of Mr. F. A. Mahomed, Resident Medical Officer to the London Fever Hospital—a spiral "eccentric," whereby it is registered much more accurately than by the use of a spring or weight.

Fortunately differences of opinion as to the physical cause of the "tidal" wave do not lead to any difference as to its

practical interpretation. The sphygmograph may give no characteristic sign of slight valvular disease of the heart, and in some cases it may fail to show any indication of thoracic aneurism, but it may be relied upon to reveal the existence of rigid arteries or hypertrophied heart. Indeed I believe that one of its most useful applications would be to estimate the probable duration of life, by showing how far the vascular system has undergone the changes to which it is subject with advancing years, or which may be the only indication of commencing Bright's disease.

Tracings 1, 2, 3, show healthy pulses at various ages. They prove that the mere increase of age does not produce the effects seen in those which follow. The pressure used for healthy pulses generally varies from $1\frac{1}{2}$ to 3 ounces.

Tracings 4 and 5 show the effect of rigid arteries without any Bright's disease. In such cases the pressure used is somewhat greater than normal, and ranges usually from 3 to 4 ounces. But their most prominent character is the occurrence of a large "tidal" wave. This is most manifest when the impulse of the heart is not very strong, as in 4. It indicates that the contraction of the heart is prolonged, and it may occur in even greater degree in cases of obstruction of the aortic orifice.

The tracings 6, 7 and 8, were taken from typical cases of granular kidney, with hypertrophied heart and rigid arteries. In these the high arterial tension is indicated by a pressure varying from 4 to 6 ounces, the latter of which is attained in no other condition, thus showing that in chronic Bright's disease there is something more than the effect of merely atheromatous arteries. The height of upstroke in 7 shows a very extreme degree of heart hypertrophy. In all the large tidal wave shows arterial rigidity, but this is especially marked in 6.

The tracings 9 and 10 were taken from cases in which dropsy had come on recently. They have all the characters common in cases of granular kidney ; and other evidence con-

curred with the sphygmograph in making it probable that in them a kidney, previously granular, had suffered a recent catarrhal attack.

I come now to some cases difficult of diagnosis, and in reference to them I have to consider the evidence to be derived from examination of the eyes. There are two conditions chiefly to be observed in Bright's disease—namely, hœmorrhages in the retina and albuminuric retinitis. In the latter the retina becomes dull and at length anæmic; lymph is deposited about the central spot, and the optic disc grows indistinct and its vessels irregular. These two conditions are often found together, but may occur separately. Hœmorrhages may be seen when no complaint is made of failing sight. Not so retinitis. The former indicates the same state of vascular system as does cerebral hœmorrhage—namely, increased pressure and diseased vessels. The retinitis may be regarded either as an interstitial scleriasis analogous to the cirrhotic change in the granular kidney, or as an inflammation, the liability to which arises from the altered state of blood. But I believe that most authorities testify that, though commoner with a granular kidney, it may occur in any form of Bright's disease, and even at an early stage, in which case the latter view would seem more probable.

I will here give brief notes of a case, which show that it may occur as the sequel of acute nephritis from scarlatina of not very long standing:—Joseph T., aged 35, admitted November 2nd, 1870. He had scarlatina three months before, and three weeks later his face and feet swelled, and his urine became scanty and high-coloured. His sight had been getting dim for some weeks. On admission there was much dropsy. Urine scanty (specific gravity 1015), containing fatty and epithelial casts. On ophthalmoscopic examination, the retina was seen to be anæmic, with white spots around the central spot, as they occur in Bright's disease. He remained in the hospital till February 3rd, 1871, when the dropsy was cured, but the urine still albuminous. His eyesight was improved.

Now, I have given the tracings of two cases of renal dropsy, in which the history and symptoms both pointed to a diagnosis of tubal nephritis, but in which the pulse-tracing has nearly all the characters usual in cases of granular kidney. These are 11 and 12. In one of them the occurrence of hœmorrhages in the retina bears the same testimony as to the state of the vascular system as does the pulse-tracing. In both of them the good specific gravity of the urine makes the existence of at least any considerable granular change in the kidney improbable. Having derived from post-mortem evidence the opinion that tubal nephritis of long-standing may lead to hypertrophy of heart and rigidity of arteries; I believe that these are probably cases of that nature.

Next come the tracings of two cases occurring in young persons in whom the condition of the eyes had led to the suspicion of the existence of granular kidney, although the extent of dropsy and some other circumstances pointed rather to tubal nephritis. These are 13 and 14. The tracings show high arterial tension, without any great hypertrophy of heart. That of 13 indicates also great rigidity of arteries. In the case of 14 the state of the kidney was disclosed by post-mortem examination. It corresponded to the ordinary characters of a granular kidney, except that its colour was unusually white, and the microscope showed greater irregularity than usual of epithelium. The history of the case, coupled with the age at which it occurred, appears to afford an instance rather in favour of those who believe that there may result, as a late stage of tubal nephritis, a state of the kidney, and of the body generally, closely resembling that which is generally the result of cirrhotic change.

Next come a series of cases (15, 16, 17) of recent acute nephritis, two of them apparently the result of scarlatina. In all of these the tracings show some characters approximating to those of chronic Bright's disease. The pressure is somewhat greater than is usual in healthy pulses, and the tidal wave is too large in proportion. It has been already

said that this increase in the tidal wave indicates a prolongation of the heart's contraction, such as results from atheromatous arteries or aortic obstruction. It is obvious that a similar effect would be produced if there were an impediment to the capillary circulation. Hence my interpretation of these tracings is, that even in the early stage of acute nephritis such an impediment to the circulation may occur from altered quality of the blood that the arterial pressure is increased, and the heart's contraction made more laborious. If this be true, it is easy to understand that this state of things, if continued long enough, will cause the muscular walls both of heart and arteries to hypertrophy.

The last two tracings, 18 and 19, are introduced to show that the characteristic pulse of Bright's disease may be entirely lost, when complications arise, especially such as are associated with a febrile condition. They show the effect of bronchitis in the first case on the pulse of a granular kidney; in the second on that of tubal nephritis. Bronchitis has a double action on the pulse. The obstruction in the lungs diminishes the pressure in front of the obstructed point, and thus makes the arterial tension less. Also the associated febrile condition has a similar effect from the relaxation of the vessels, and thus on both accounts the pulse tends to become more dicrotic, and its tidal wave to become less marked.

It is in the case of Bright's disease complicated by a febrile state that there is sometimes found a condition which does not otherwise occur, namely a dicrotic pulse which will bear a high pressure. Although the arterial tension in such cases has become low, as evidenced by the dicrotism, yet the heart, which had previously become hypertrophied, gives proof of its power in thus making a strong upstroke under pressure.

I have a few words to say, finally, on the proximate cause of renal dropsy; for since the presence or absence of dropsy is a most marked difference between the various forms of renal disease, a correct interpretation of this is likely to throw

some light upon the difference of their pathology. The first requisite in a satisfactory explanation of renal dropsy is that it should account for that difference in its distribution from that of cardiac dropsy, whereby the one may generally be distinguished at a glance from the other. Now, the amount of transudation and absorption through the walls of the vessels must depend on the resultant of chemical or vital actions, and of the mechanical laws of osmosis, and any disturbance of either conditions may produce an altered result. There are only two mechanical conditions which would tend to increase exosmosis, namely, an increased pressure within, or a diminished specific gravity of the contained fluid. Now, pressure at a lower point of the body exceeds that at one which is higher, but in other respects exactly similarly situated, in proportion to the difference of vertical height. Hence any effect of excessive pressure would be seen most in dependent parts. This is exactly what occurs in cardiac dropsy, which is generally agreed to be the result of increased pressure, and does not occur in renal dropsy. The conclusion is clear that renal dropsy cannot result from increased pressure, and, therefore, if mechanical at all, it can only be the effect of diminished specific gravity. The effect of this would be equally diffused, and would, therefore, be most manifest, as in renal dropsy it is, where the tissues are loosest.

It is an old opinion that renal dropsy is caused by poorness of blood, resulting from the drain of albumen. Poorness of blood is a somewhat vague expression; for the blood is, in one sense, poor in cases of granular kidney, where there is no dropsy, for there is then a scarcity of red corpuscles. But it is only the specific gravity of the liquor sanguinis which can mechanically affect the result, since the corpuscles would behave only as foreign bodies floating in the fluid.

For direct evidence as to the state of the blood,—since bleeding is now never performed in Bright's disease,—I have to refer back, as all writers do, to the observations published by Dr. Christison. It is true that he regarded a granular

kidney as being the later stage of acute Bright's disease; but it is evident from the account of his cases that his instances of early stage were mostly cases of tubal nephritis, those of late stage cases of granular kidney; dropsy being generally present in the former, and not in the latter. His results were as follows:—In the early stages he found the serum to be in specific gravity from 1020 to 1022, instead of from 1029 to 1031 as it was in health. In late stages he found its specific gravity to be normal. Now, conclusions drawn from the state of the serum may be at once applied to that of the liquor sanguinis; since the variations which he found in the amount of fibrin were never sufficient to make a difference of one part in 1,000. We have thus, therefore, direct evidence that the diminished specific gravity, which would be a sufficient cause of the dropsy, does actually exist in acute Bright's disease.

But it is a fatal objection to the opinion which attributes the cause of this to the drain of albumen, that dropsy may appear coincidently with the albuminuria, or sometimes even before it, especially in cases which follow scarlatina. Moreover, Dr. Christison found the deficiency in solids to affect not only the albumen, but the salts equally. But the specific gravity would be equally diminished, and that immediately, by a failure in the power of the kidney to excrete water, and at the outset of acute nephritis the quantity of urine is constantly diminished. This retention of water has often been suggested as a cause of the dropsy, but only as leading to increased pressure in the vessels. For the reasons before mentioned I do not believe that a change of pressure can be the main cause, although it might in some degree contribute to the result.

It has been stated as an objection to the theory attributing renal dropsy to a defective excretion of water, that in cases of tubal nephritis which have reached a chronic stage the amount of urine may be even above normal, and yet dropsy continue. But this generally occurs under the use of diuretics, and, even

if it be otherwise, there is at any rate, so long as dropsy continues, an excess present of that most powerful of all diuretics, namely, water. It may well be, therefore, that the quantity of urine, even though absolutely above normal, is yet relatively too little for the requirements of the individual, and less than a healthy kidney would have excreted under the same conditions. It is also to be considered that in this chronic stage the effect of the drain of albumen may come into play, and contribute in an important degree to keep down the specific gravity of the liquor sanguinis.

I will briefly advert to some other theories of renal dropsy which have been suggested.

1. That it is the result of increased pressure, caused by resistance to the circulation of the altered blood. It is a fatal objection to this that in cases of granular kidney the arterial pressure is most of all increased, and then dropsy is generally absent.

2. That in consequence of the retention of the solids of the urine the specific gravity of the blood is increased, absorption thereby promoted, and, therefore, the pressure in the vessels increased.

If this were true, the amount of dropsy should always be proportional to that of uræmia; but it is found, on the contrary, that in cases of granular kidney uræmia may occur without any dropsy. This theory, as well as the last mentioned, are open to the objections before urged against attributing the result to altered pressure; and, lastly, it is contradicted by the observations of Dr. Christison, in which the specific gravity of the serum was found to be not increased, but diminished, in acute Bright's disease.

3. That renal dropsy is not due to any mechanical cause, but is the result of the irritation of the tissues caused by the altered blood, and thus is an universal inflammation of low degree throughout the areolar tissue. In this view it is compared to the dropsy occasioned by the presence of the trichina spiralis.

In answer to this it may be said that renal œdema may be greater than occurs from even a severe degree of ordinary cellulitis. Moreover, from so universal an inflammation, even if of very low degree, we should expect great constitutional disturbance. But it is not very uncommon to meet with patients in the first stage of renal dropsy, who come for advice simply on account of the dropsy, have not found their health otherwise affected, and have not even noticed anything wrong about their urine. Finally, this theory also is inconsistent with the occurrence of uræmia without any dropsy, for according to it the degree of dropsy ought to be proportional to that of uræmia.

I would draw the conclusion that in different forms of Bright's disease, the excretion of water and the excretion of solids being impaired in very different proportions, and the loss of albumen being again quite independent of either, many of their other characters result from these differences. In tubal nephritis water is retained, and therefore dropsy results; if solids are retained also, various sequelæ in the shape of inflammation may arise, and heart and arteries are often hypertrophied if the disease lasts long enough. In lardaceous disease there is no notable retention either of water or solids until a late stage, and, therefore, generally no hypertrophy of heart. In cases of granular kidney, solids alone are usually retained, and, the change being very gradual and slow, hypertrophy of heart and arteries are the marked result. In the arteries hypertrophy is not the sole change found, but atheroma of large arteries and fibroid or granular change in minute vessels are commonly seen, whether these are purely degenerative, or whether, as I rather believe, they are in part the sequel of inflammation. But such changes are frequent in advanced life, and though they are more common and more marked in chronic Bright's disease, yet the difference is, as I believe, not greater than may be accounted for by the increased strain and the impaired nutrition, which, in such cases, are both present.

TABLE I.

ANALYSIS OF ALL CASES OF GRANULAR KIDNEY

CONTAINED IN THE POST-MORTEM RECORDS OF GUY'S HOSPITAL DURING THE YEARS 1869, 1870, 1871, 1872.

Cases of granular kidney in men	55
„ „ women	24
Total	79
Out of these cases there was evidence of gout in	20
Cirrhosis of the liver in ...	10
The number of cases of cirrhosis of the liver in which kidneys were healthy were	23
Out of 36 cases occurring during the years 1870, 1871, it was specially recorded that the arteries were atheromatous in ...	19

ASSOCIATION OF GRANULAR KIDNEY WITH HYPERTROPHY OF HEART.

Out of the 79 cases the state of the heart was not noted in ...	8
There was hypertrophy of heart, with other causes to account for it, in	5
Cases remaining for comparison	66
Out of these there was no hypertrophy of heart, kidneys but little diseased, in	3
No hypertrophy of heart, kidneys much diseased, in	10
Hypertrophy of heart in	53
Total	66
In 34 of the cases of hypertrophy the state of the several cavities was specially recorded :	
There was simple hypertrophy of left ventricle in	17
Hypertrophy and dilatation of left ventricle	5
General hypertrophy	6
General hypertrophy and dilatation	6
Total	34

TABLE I.—(*continued.*)

	oz.
The average weight of the heart out of all the 66 cases was ...	15½
In 17 out of the 66 cases the kidneys were not smaller than natural. In 13 of these the heart was hypertrophied, and its average weight in the 17 cases was	14½
In 23 cases, in which the arteries were very atheromatous, but the kidneys and valves of the heart healthy, the average weight of the heart was...	12½

These were all cases of aneurism, senile gangrene, cerebral hœmorrhage, or cerebral softening. The heart was hypertrophied in 13 out of the 23.

TABLE OF THE AGES AT WHICH DEATH OCCURRED IN THE CASES OF GRANULAR KIDNEY.

Between 10 and 20	2
„ 20 and 30	6
„ 30 and 40	12
„ 40 and 50	19
„ 50 and 60	21
„ 60 and 70	16
Above 70	3
Total	79

The average weight of the heart in the 8 cases occurring under the age of 30 was 12 ounces. It was hypertrophied in 6 out of the 8.

TABLE II.—TABLE OF ALL CASES OF TUBAL NEPHRITIS, EXCLUDING SCARLATINAL DROPSY,

FROM THE POST-MORTEM RECORDS OF GUY'S HOSPITAL, DURING THE YEARS 1869, 1870, 1871, 1872.

Sex.	Age.	State of Heart.	Weight of Heart in ounces.	Period since first attack of Dropsy.	Weight of Kidneys in ounces.	State of Kidneys.	State of Urine during life.
F.	49	Hypertrophy of left ventricle...	11	3 months ...	7½	Pale, smooth, cortex not thin in proportion	Sp. gr. 1017. Highly albuminous
M.	23	Healthy. Left ventricle firmly contracted	.....	4 months ...	15	Large white	
M.	23	Hypertrophy of left ventricle...	15	15 months...	23	Large white	
M.	36	Right side dilated. Embolism of pulmonary artery	18	.....	.....	Large white	
M.	43	.....	11	10 weeks ...	.....	Large, smooth, white	
M.	56	Hypertrophy of left ventricle...	13½	12 months...	.....	Small, smooth, and white. Cortex rather thin. Arteries thick	Sp. gr. 1012. Loaded with albumen
M.	38	Dilatation and hypertrophy. Adherent pericardium	17½	.....	.....	Smooth white; minutely mottled; natural size	
M.	26	.....	11	A few weeks	18	Pale and swollen	Scanty. Sp. gr. 1025
M.	54	Healthy.....	.....	3 weeks.....	.....	Large, pale, mottled on surface	
M.	26	Dilatation and hypertrophy. Adherent pericardium	20	18 months...	.....	Large white	
F.	64	.....	10	.....	15	Large white	
M.	34	Hypertrophy of left ventricle...	16	.....	11	Large, pale, very smooth	
F.	34	Healthy.....	.....	6 weeks.....	.....	Large, smooth, mottled	

TABLE II.—*continued.*

Sex.	Age.	State of Heart.	Weight of Heart in ounces.	Period since first attack of Dropsy.	Weight of Kidneys in ounces.	State of Kidneys.	State of Urine during life.
M.	48	Hypertrophy and dilatation, especially left ventricle	23	2 years	15	Large white. Arteries thick	Sp. gr. 1010. Loaded with albumen
M.	...	.....	18	3 months ...	12	White. Surface minutely granular	
F.	31	Slight hypertrophy of left ventricle	11	3 months ...	9	Pale, mottled, fatty	
M.	28	No decided hypertrophy.....	11	3 weeks.....	16	Large white	
M.	32	Hypertrophy and dilatation left ventricle	16	.....	20	Mottled, smooth	
M.	29	Healthy.....	.....	.....	....	Large, mottled, smooth	
M.	57	No decided hypertrophy.....	12	2 years	15	Large white	
F.	17	Hypertrophy, Disease of aortic valves	15½	2 months ...	18	Large white	
M.	52	Hypertrophy of left ventricle...	21	2 years	14	Pale and mottled. Surface very slightly uneven	Sp. gr. 1012. Very albuminous. Epithelial and fatty casts
F.	40	Hypertrophy and dilatation of left ventricle	12	18 months...	13½	Quite smooth. Mottled by opaque white	
M.	32	Healthy.....	.....	5 months ...	21½	Large white	
F.	55	Healthy.....	.....	.....	12	Large white	

TABLE II.—(*continued.*)

Number of cases of tubal nephritis, excluding scarlatinal dropsy, in 4 years	25
Of these there was hypertrophy of heart, with other causes to account for it, in	3
Of the rest, hypertrophy in	11
„ no hypertrophy in	11
Total	25
	Oz.
Assuming 10 ounces to have been the average weight of the heart in those cases in which it is only recorded to have been healthy, the average weight for the whole 22 cases is	13

Number of cases of lardaceous disease of kidneys during 4 years	15
Of these there was hypertrophy of heart, the kidneys being also granular, in	2
Of the rest, hypertrophy in	1
„ no hypertrophy in	12
Total	15
	Oz.
The average weight of the heart in 13 cases not complicated by granular change was...	$9\frac{3}{4}$

TABLE III.

CASE OF ATROPHY OF ONE KIDNEY—HEART HYPERTROPHIED.

Charles T., 43, admitted in January, 1870, with hemiplegia.

Post-mortem.—There was found cerebral hœmorrhage. The right kidney was healthy, and weighed 9 ounces. The left kidney was much wasted, and contained several calculi. The heart weighed 20 ounces—the left ventricle was hypertrophied. The arteries of the brain were atheromatous, the aorta not so.

CASE OF CONGENITALLY IMPERFECT KIDNEYS, VERY SLIGHTLY AFFECTED BY BRIGHT'S DISEASE—HEART HYPERTROPHIED.

George L., 26, admitted in May, 1869, with coma, following convulsions. He had had frequent micturition at night for two years. Urine albuminous.

Post-mortem. The kidneys weighed together 3¾ ounces. They were perfect in outline, and the cortex in due proportion. Surface smooth, but with very slight minute granulation. Under the microscope some of the tubes were found dilated; epithelium rather irregular, stroma fibrous in parts. The heart weighed 13½ ounces; the left ventricle was hypertrophied.

CASE OF ATROPHY OF ONE KIDNEY—HEART HYPERTROPHIED.

F. B., 19, admitted into Victoria Park Hospital, February 12th, 1872, with acute phthisis. On March 7th was attacked by right hemiplegia and aphasia.

Post-mortem.—Left ventricle of brain distended by clot from middle cerebral artery. Cerebral arteries and aorta atheromatous. The left kidney was wasted to a cyst, and contained several calculi. The right kidney was but little enlarged, and appeared slightly affected by tubal nephritis. The left ventricle of the heart was much hypertrophied.

APPENDIX.

PLATE I.

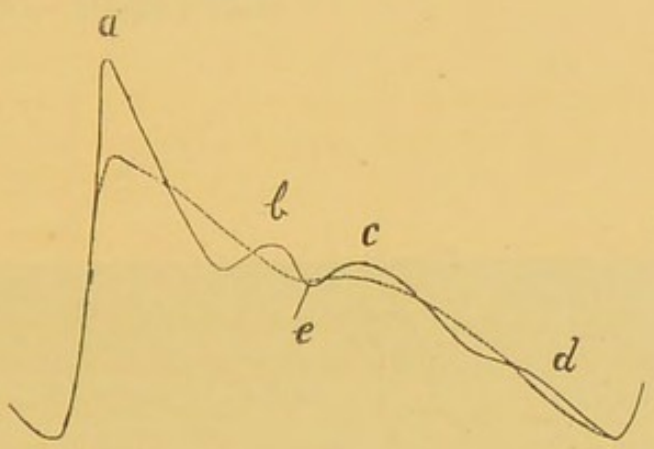


DIAGRAM TO ILLUSTRATE THE RELATION OF THE TRACING OF THE PULSE AS DRAWN BY THE SPHYGMOGRAPH TO THE TRUE PULSE WAVE AS IT OCCURS IN THE ARTERY.

a Primary or "percussion" wave.

b First secondary or "tidal" wave.

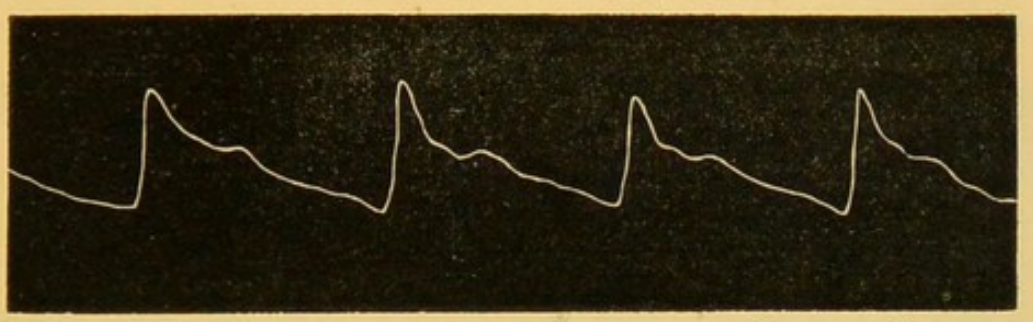
c Principal secondary or "dicrotic" wave.

d A nameless wave which, like the "tidal" wave, is the result of oscillation in the instrument.

e "Aortic notch."

The dotted line is intended to represent the tracing which would be drawn if the instrument followed the movements of the artery with perfect accuracy.

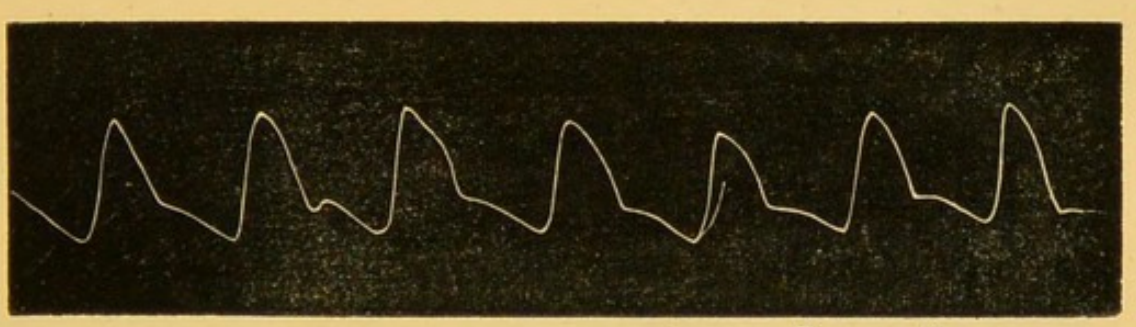
1



Pressure $2\frac{1}{2}$ *ounces.*

Man, aged 30. Healthy pulse.

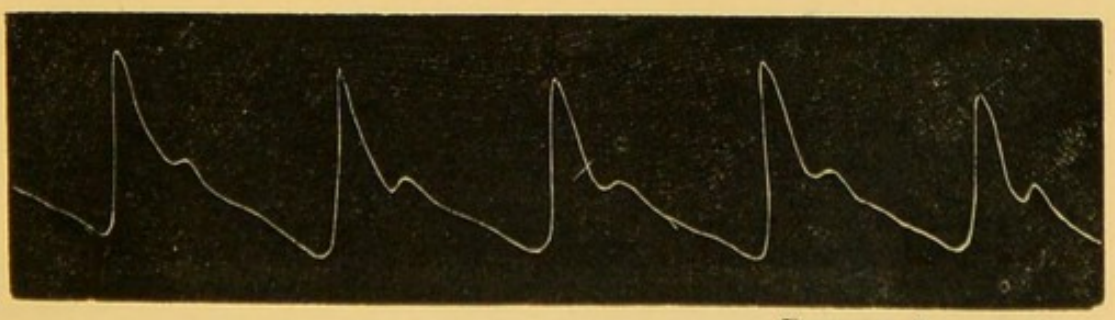
2



Pressure 2 *ounces.*

Woman, aged 46. Arterial elasticity somewhat impaired.

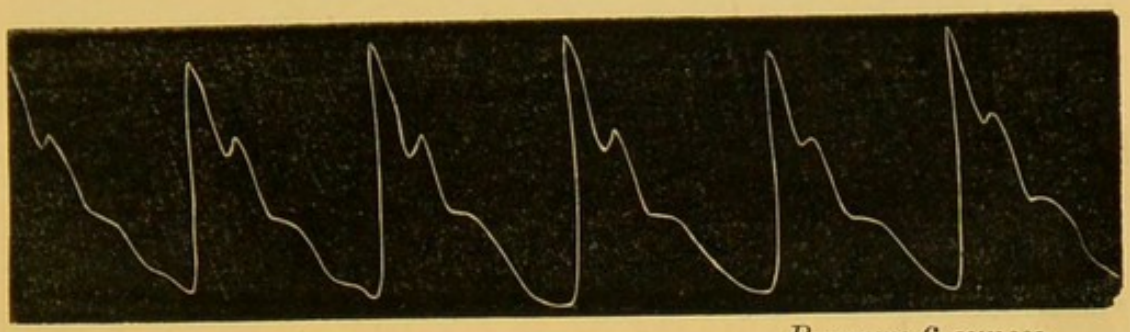
3



Pressure 3 *ounces.*

Healthy man, aged 50. Amplitude greater than normal, probably from dilatation of the radial artery.

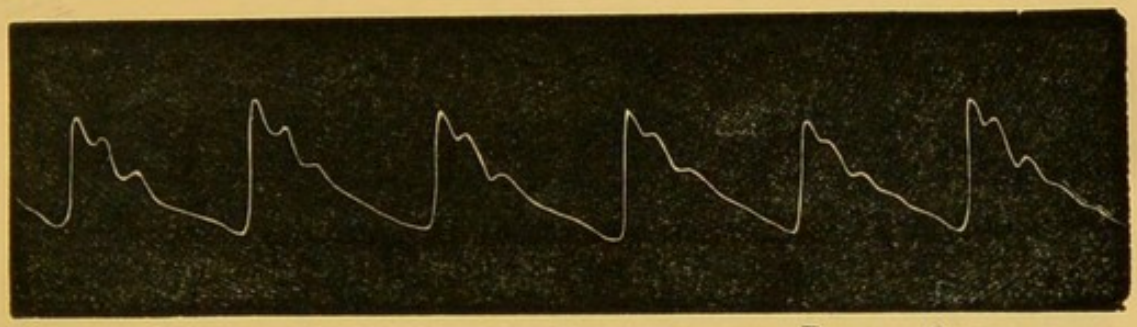
7



Pressure 6 *ounces.*

Man, aged 38. Albuminuria. Diagnosis: granular kidney. Heart much hypertrophied.

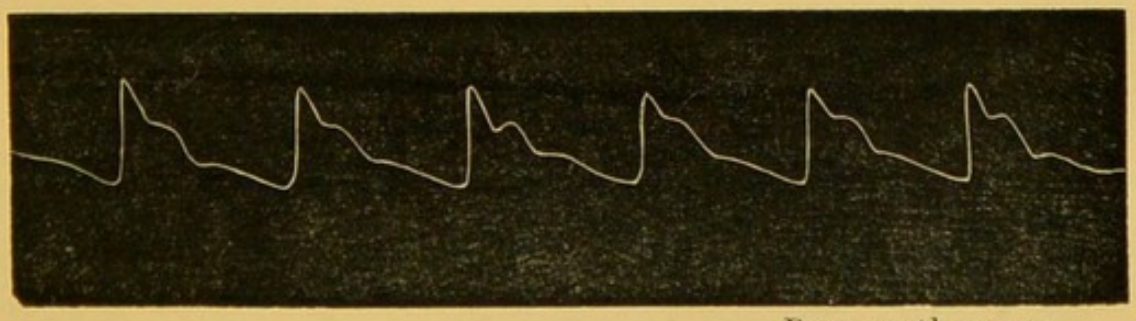
8



Pressure 6 *ounces.*

Woman, aged 59. Albuminuria. Diagnosis: granular kidney. Arteries rigid.

9



Pressure 4½ *ounces.*

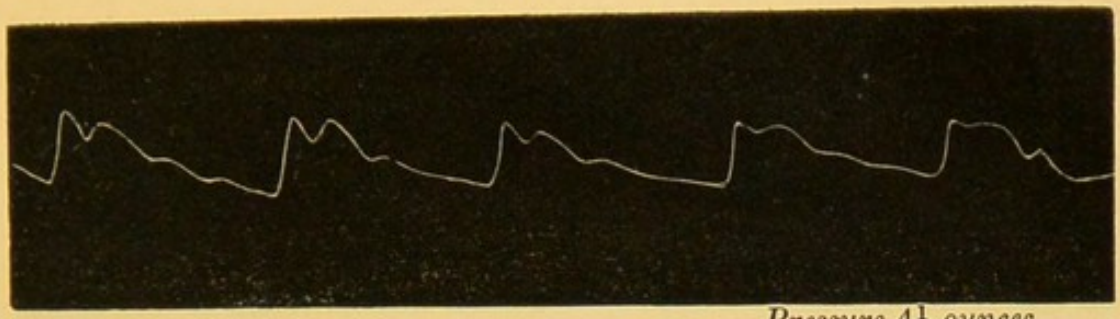
Woman, aged 47. Ten years ago she had dropsy, from which she recovered. Her present illness began with sickness five months ago. For six weeks she has had a cough, with attacks of dyspnœa, and during the last three weeks dropsy has come on.

Urine contains a good deal of albumen: specific gravity 1013. It contains granular and epithelial casts.

Spots of old hœmorrhage are to be seen in both retinæ, but she makes no complaint of her sight.

Diagnosis: granular kidney with catarrhal attack.

10



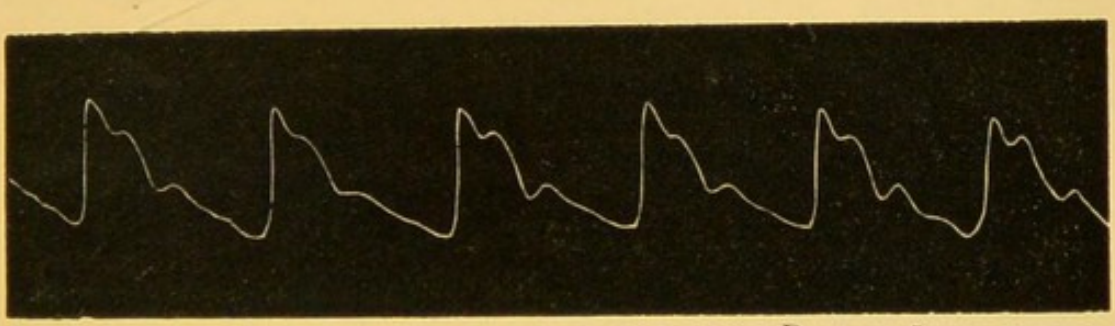
Pressure 4½ ounces.

Woman, aged 50. Seven weeks ago she had epistaxis which lasted almost continuously for a week. At the end of that time dropsy commenced in her hands and face, and is now considerable. For some years she has been subject to cough.

Urine loaded with albumen: specific gravity 1011; 46 ounces were passed in twenty-four hours. Granular, epithelial, and hyaline casts were found.

Diagnosis: Granular kidney, with catarrhal attack.

11



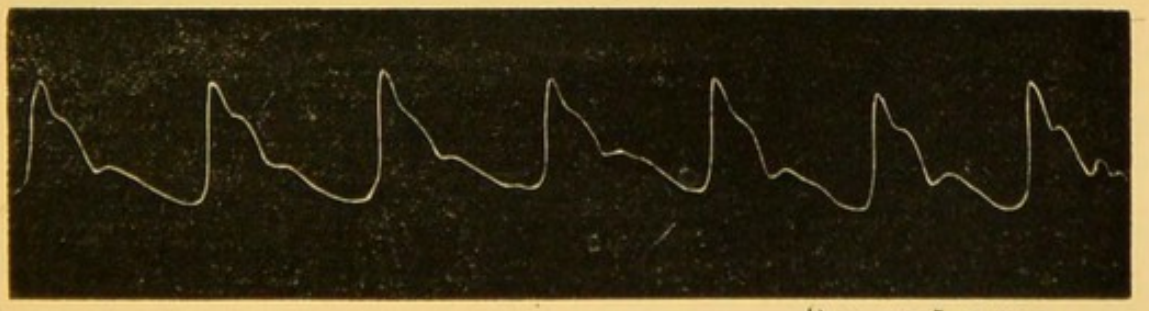
Pressure 5 ounces.

Woman, aged 29. Thirteen years ago she had dropsy, which occurred five weeks after a sore throat. She recovered in three months. She has now extensive general dropsy, which commenced seven weeks ago.

Urine contains much albumen, and epithelial casts are found in it. Its specific gravity varies from 1016 to 1022.

Diagnosis: Chronic tubular nephritis.

12

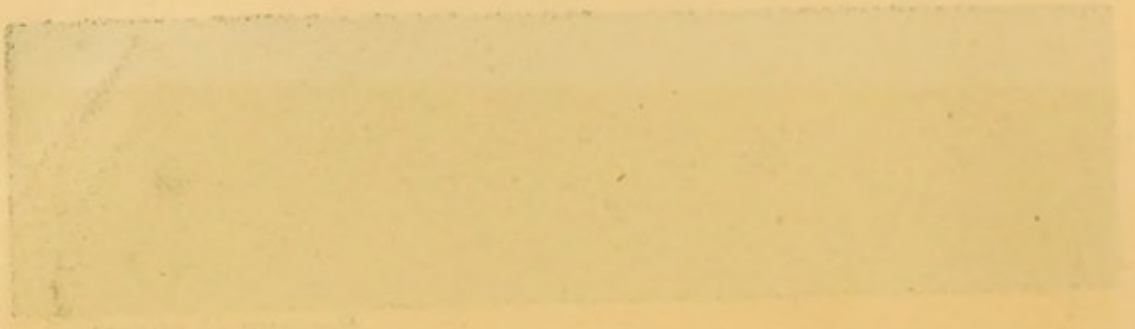


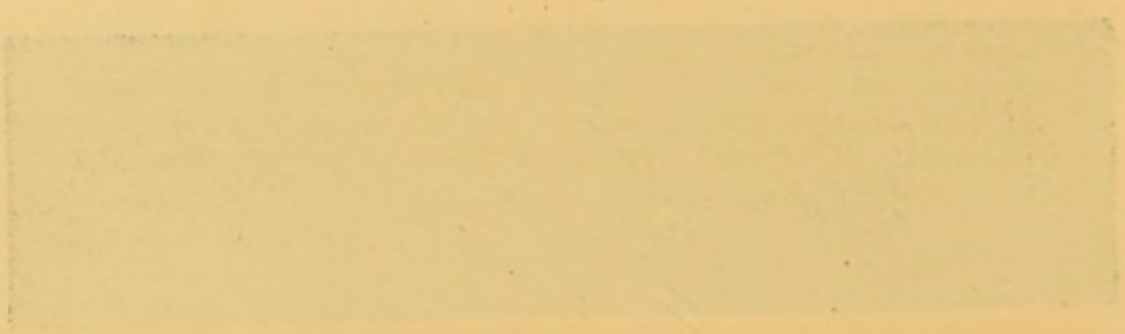
Pressure 5 ounces.

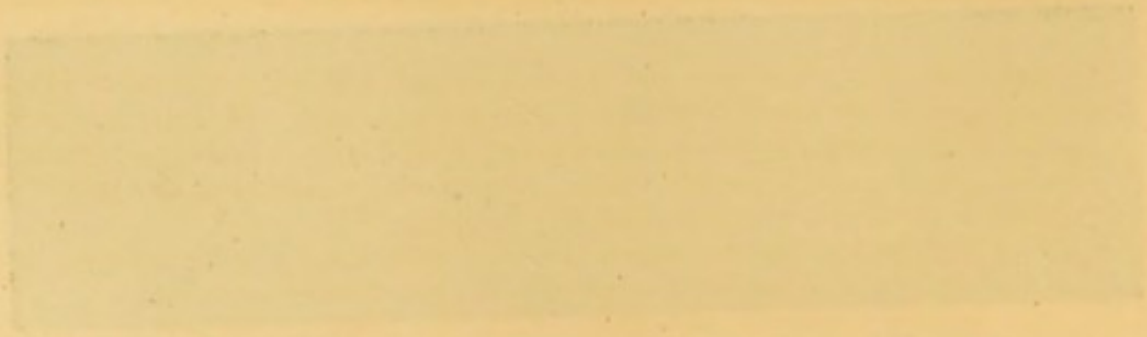
Woman, aged 44. She is now seven months pregnant, and dropsy came on suddenly for the first time three months ago. Her urine was then scanty and high-coloured. Her dropsy is now very great.

Urine nearly solid with albumen: its specific gravity is 1020; 30 ounces are passed in twenty-four hours.

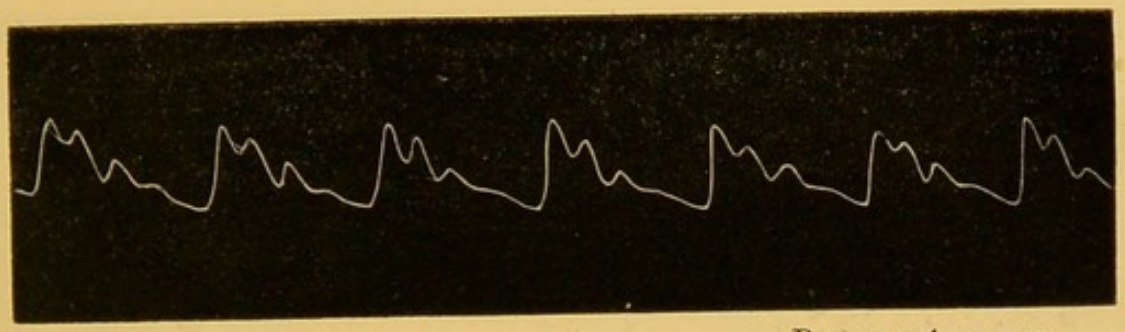
Her sight has failed during the last month. By the ophthalmoscope are seen patches of old and recent hæmorrhage in both eyes, and lymph in radiating yellow lines around the central spot.







13



Pressure 4 *ounces.*

Boy, aged 18. He had scarlatina at eight years old, not followed by dropsy.

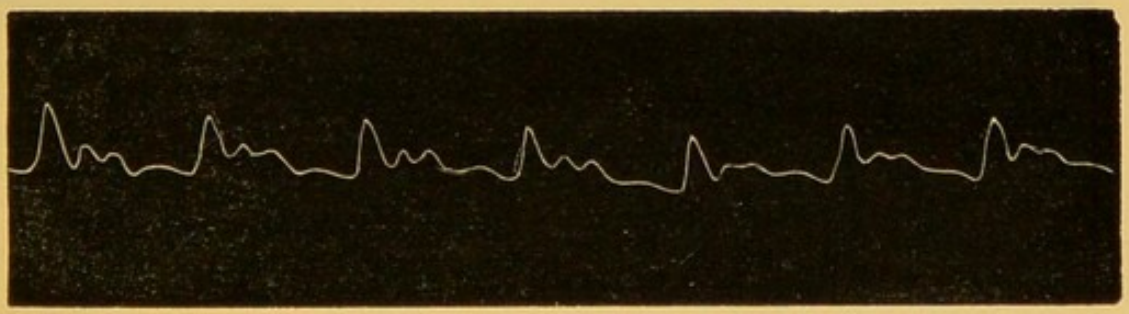
He has been ill ten months with dropsy, moderate in degree. He has been subject to vomiting for six months, to occipital headache for four months, and for two or three months his sight and hearing have been failing. He is subject to fits, apparently uræmic, and to epistaxis.

Urine loaded with albumen: specific gravity 1012. About 80 ounces are passed in twenty-four hours.

Both retinæ are mottled all over with old and recent hœmorrhages.

P.M. The kidneys were granular: the left ventricle of the heart was hypertrophied: the arteries were much thickened but not atheromatous.

14



Pressure 5 *ounces.*

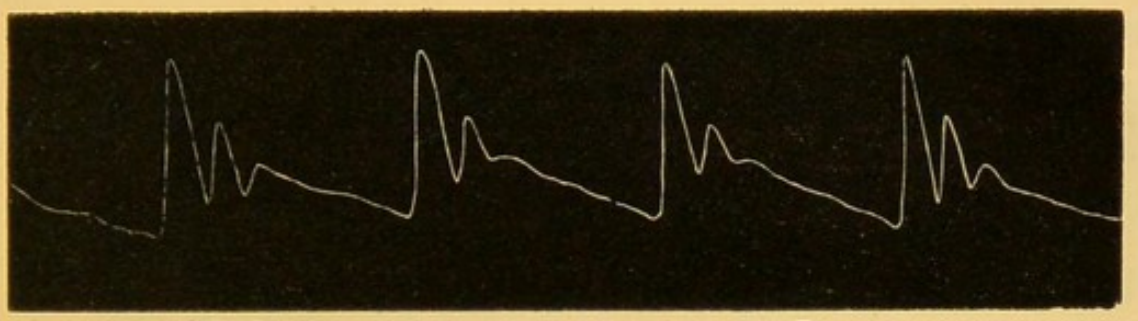
Girl, aged 18. One year ago she was in hospital with extensive general dropsy, and acute tubal nephritis was then diagnosed. Her urine was then loaded with albumen: its specific gravity 1012; and it contained granular, hyaline, and epithelial casts. Her dropsy was cured, but her urine still contained albumen when she left the hospital.

Two months ago she was again attacked with dropsy after catching cold, but her dropsy was not then so great as on the former occasion.

She became comatose and died. P.M. her kidneys were found smaller than natural, granular on the surface, and differing from the usual appearance of granular kidneys only in the fact that they were very white. On microscopic examination they showed the general characters of a granular kidney, except that the tubes were more than usually choked with epithelium.

There was hypertrophy of the left ventricle of the heart, and its weight was 17 ounces. The arteries were not atheromatous, nor those of the kidney much thickened.

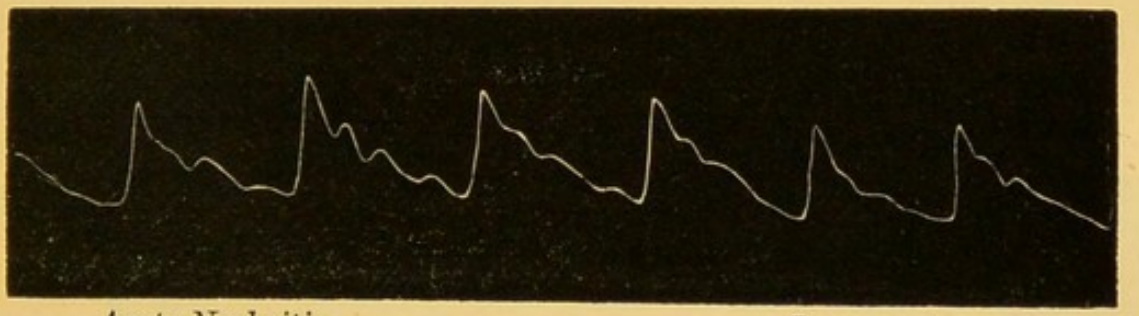
15



Acute Nephritis. *Pressure 4 ounces.*

Man, aged 24. Five weeks ago he had sore throat. His skin is now desquamating, and his urine is albuminous. For one week he has had slight dropsy.

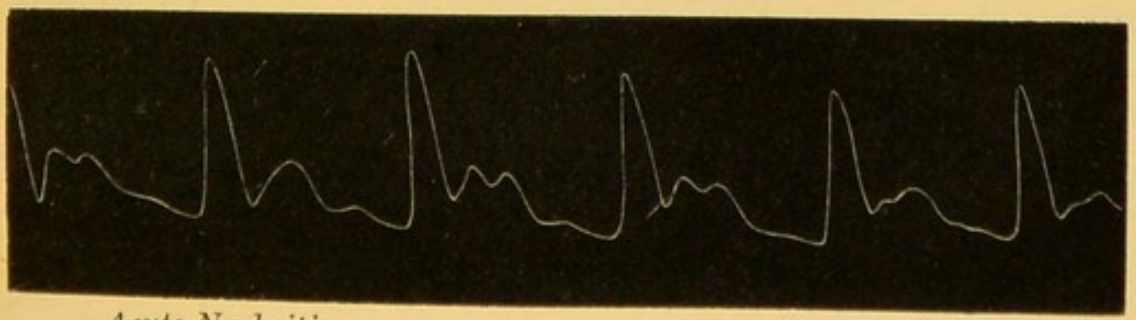
16



Acute Nephritis. *Pressure 4 ounces.*

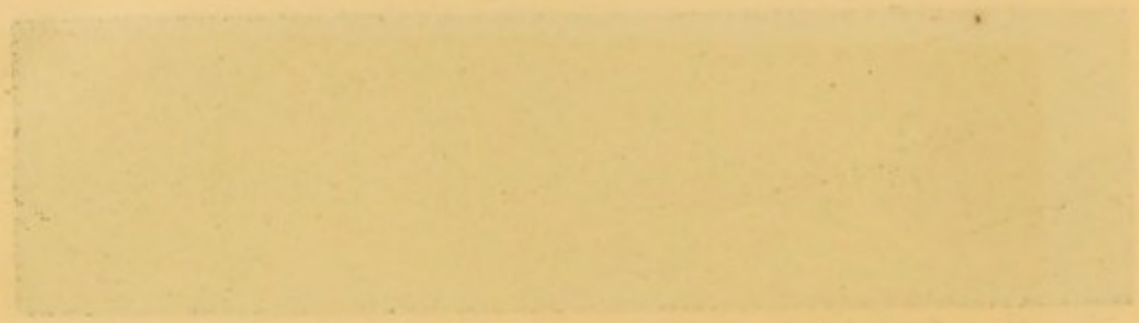
Boy, aged 17. He was admitted with albuminuria, following a sore throat. His face and hands were desquamating. He had no dropsy, and the albumen disappeared after a few days.

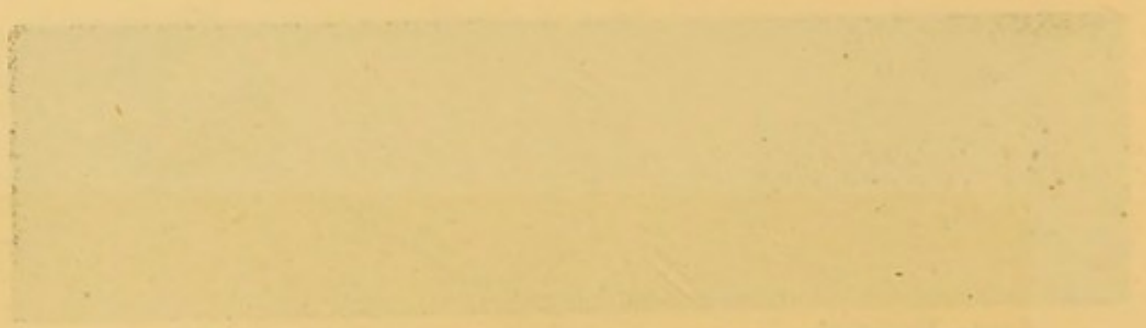
17

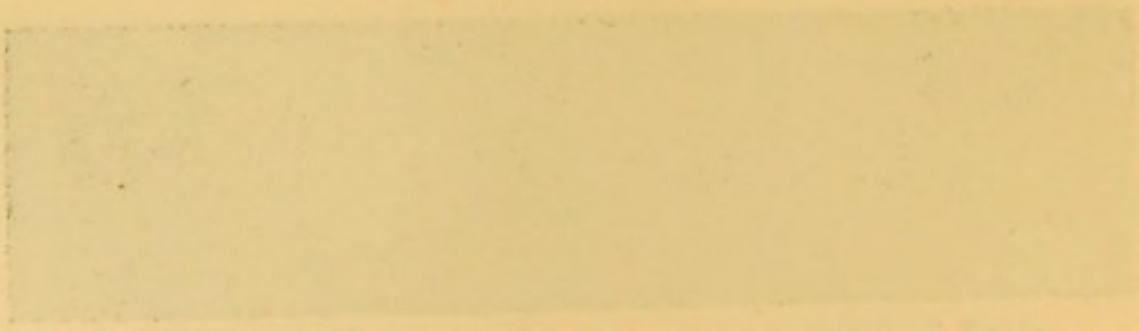


Acute Nephritis. *Pressure 3½ ounces.*

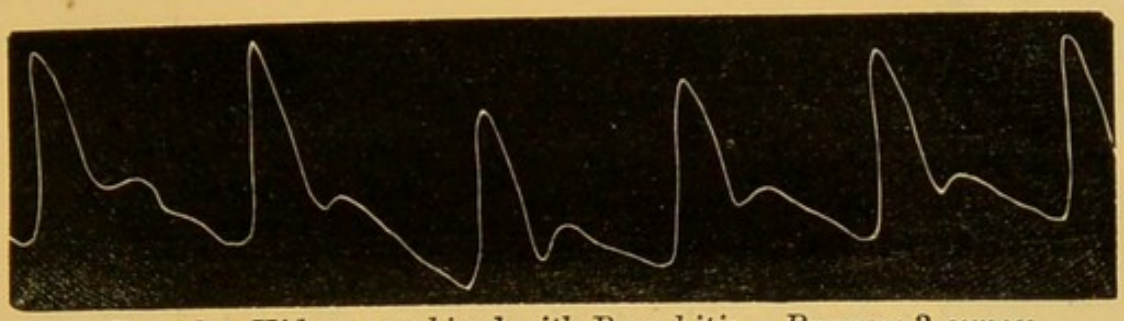
Man, aged 24. Three weeks ago he was attacked with dropsy, after taking cold. His urine became scanty and dark, and micturition frequent. Urine loaded with albumen: specific gravity 1020.







18



Granular Kidney, combined with Bronchitis. Pressure 3 ounces.

Man, aged 44. For several years he has suffered from bronchitis. For the last five weeks he has been ill with gout. He has now slight œdema of the face.

Urine contains a little albumen: specific gravity 1010.

This tracing shows the variation in the line of arterial tension due to laborious respiration.

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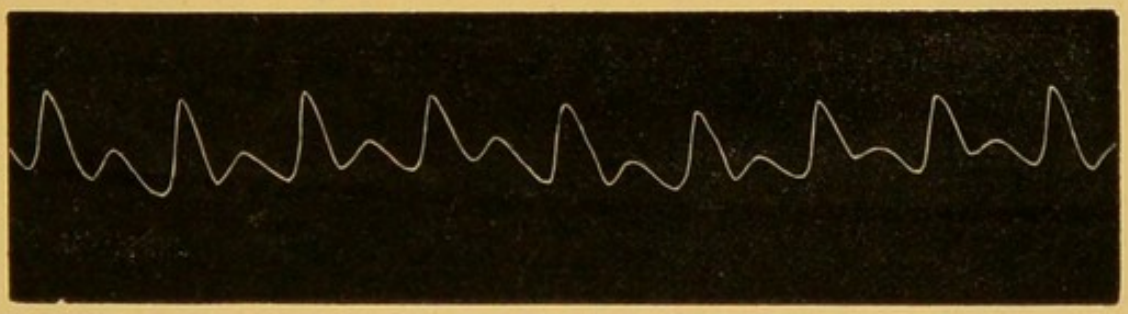

Tubal Nephritis, combined with Bronchitis. Pressure 2 ounces.

Man, aged 32. For five years he has had bronchitis, with fits of dyspnœa. Five weeks ago general dropsy came on.

Urine scanty, containing blood, and much albumen: specific gravity 1018.