2. Drugs.

In some cases severe symptoms are for the time greatly relieved by nitrites. But their effect is usually transient. Still, I have known men made much more comfortable by small doses of nitroglycerine given for a week or two. For a more permanent effect small doses of potassium iodide have been highly recommended.

But I do not think that any drug approaches calomel. Given in small doses of 1 gr. or $\frac{1}{2}$ gr. every night for a short time and repeated at intervals, it sometimes is of very great use. It is not its purgative effect that is needed; it is really what is called an alternative. Every one appreciates its effect on the spirits, and I believe this to be chiefly an effect on the blood pressure, for it is known that melancholy is a state in which the pressure is high.

Alkalies act chiefly by increasing the flow of urine, and this, especially in cases complicated—for this is my view of the position—by nephritis, is often of great importance.

3. Diet.

I think that diet is perhaps even more important than either habits or drugs. I have not any doubt that in some cases a very light diet acts like a charm. Let me give an example:

A man of over 50, very thin and spare, was admitted under me. He was complaining of severe dyspnoea and oppression, and had been in bed for some time at home without benefit. He could not lie down. There was no disease of his lungs, no oedema, no dilatation of the right side of his heart, and no albuminuria. I am not prepared to say his kidneys were healthy; probably they were fibrous. But the main thing about him was extreme degeneration of his radial artery and a very high blood pressure. He was put on our hospital milk diet, and from that moment improved rapidly. He went out, resumed his usual food, and in three weeks his symptoms returned. This time I made him go on milk diet at his own home and the result was as good as before.

This effect, however, does not always follow. Almost at the same time I tried this diet on a rather older man with the same symptoms without producing any effect on his blood pressure. He improved slowly, just as such cases usually improve when they lie up.

Whereas in the first case it seemed that the alteration in diet really removed to a great extent the cause of high pressure, which was, therefore, probably a floating impurity, in the second case it is reasonable to suppose that the pressure was due to some permanent changes in the vessels. Under such circumstances the high pressure is necessary for the blood flow, and all we can do is by rest and small doses of digitalis to strengthen the heart for its task.

I do not quite know how, when this treatment succeeds, the effect is produced. It is not the necessary result of milk, for if milk alone is given, which involves drinking 5 pints daily, it is said to increase the blood pressure rather than diminish it. Our milk diet, however, contains milk (2 pints), bread and butter, rice or custard pudding, and tea (1 pint). There are about 70 grams of proteid in it. The reduction of blood pressure, therefore, might be due either to the reduction of proteid or to the alteration of the kind of proteid taken.

In some cases I think the first is certainly the most important. In chronic nephritis, which always means diffuse nephritis—for interstitial nephritis is a latent disease—it used to be the custom to prohibit meat. Hale White and von Noorden have written against this treatment, and I fully agree with them. Such patients do much better with meat than without it, but the total of their food must be kept at a very moderate level, because their means of disposal of refuse are crippled.

But we must not expect to reduce pressure in them; in later stages, at any rate, it does not fall, even when, owing to uraemic vomiting, scarcely any food is taken. This obstinacy may perhaps be due to disease of the abdominal arteries. It is known that these vessels have a preponderant influence on blood pressure, and also that they are nearly always diseased in chronic nephritis of middle life. But it seems to me more probable that, as experiment suggests, the degenerating renal tissue pours a toxin into the blood which no change of diet could remove. In such cases the one important thing is to drain the poison off by increasing the flow of urine.

In other cases, however, I think it impossible to deny that the kind of proteid taken is of importance. I am not

at all a faddist about food. I do not share the belief that meat invariably floods our tissues with uric acid, and I think that many people are the better for a little wine. But my observation leads me to think that there is a great variety in the metabolism of different persons, and that the diet which is good for one is, quite apart from gastric indigestion, poison for another. There is exaggeration in this as in all things, and it is not often that a patient's observation of his own symptoms can be accepted without reserve. Still, it is my belief that chemical analysis has much further to go than the mere estimation of calories, or of the nitrogenous contents of the ingesta and excreta, and that our own observations if carefully made will often tell us more than chemistry can yet explain. Chronic rheumatism, of which you see plenty here, some forms of headache, and high blood pressure, appear to me to be clues of special importance in examining the effects of different forms of proteid, and I think there is a great deal to be done by following them up.

I do not ascribe the same importance to carbohydrates. They have great influence on digestion. Some people cannot eat bread or potatoes, some can eat raw sugar but not cooked sugar, and so on; but I have not observed that they affect blood pressure.

I should say the same of minerals. Salt has a certain connexion with oedema, but this is not through blood pressure. Calcium I never saw have any effect at all.

This, however, may be from want of observation on my own part, for I see that others think differently.

Meanwhile there is one lesson that middle age has always to learn, and that is that it must be moderate, and that moderation means for it something very different from the ordinary meals of healthy and active youth.

REFERENCES.

¹Gutch, *Journ. of Pathol.*, 1901. ²Hirsch and Beck, *D. Arch. f. klin. Med.*, 1902, vol. lxxii, p. 560.

Remarks

ON

SOME POINTS IN THE PATHOLOGY AND TREATMENT OF ADHERENT PERICARDIUM.

By DR. K. F. WENCKEBACH,

PROFESSOR OF MEDICINE, GRONINGEN.

THE subject of this study is a boy, son of healthy parents. His brothers and sisters are also healthy. There are no cases of tuberculosis in the family. He is living in the country, under exceptionally favourable hygienic conditions. His father is a doctor.

There seemed to be nothing wrong with him until the summer of 1903; he then got an exudative pleurisy on the left side. There were pain and fever during some weeks, and a certain amount of fluid. The fluid was absorbed, but dullness remained, with marked retraction of the lung. He did not, however, regain his appetite, and there was some shortness of breath.

A year afterwards, in the summer of 1904, a pericarditis followed. There was again a considerable amount of fluid, there was fever, and the boy was very weak. Again the fluid and the fever disappeared, but at the moment the exudate seemed to be locally reabsorbed, alarming symptoms made their appearance. The pulse became very quick and weak; an extreme shortness of breath and a very painful and enormously swollen liver were developed.

I was asked to see the patient, and found the symptoms characteristic of adherent pericardium. The pulse was small, weak, and frequent, diminishing during inspiration (*pulsus paradoxus*); the veins were considerably over-filled, and during inspiration there was marked swelling of the veins in the neck. In the regio cordis there was a visible and palpable systolic retraction of the ribs and the intercostal spaces. The heart sounds were faint but pure. The liver was huge and hard, reaching nearly to the umbilicus, and very sensitive to touch. Its size seemed out of proportion to the stasis of blood in the venous system (see Fig. 8 a).

The history of this disease, or rather of its symptomatology, is connected with many a great name. Kussmaul, Friedrich, and many French and English clinicians, have

contributed to our knowledge of the disease, and of the genesis of its curious symptoms. Still there is much that is obscure, and there is a great deal of controversy concerning the explanation of some of its most striking features.

HAMPERING OF THE HEART.

The disturbance of the circulation in adherent pericardium is a purely mechanical one. We, therefore, have to study minutely what are the anatomical conditions that put such a strain on the heart and the circulatory system. The pernicious influence of the adhesion of heart and the pericardium to surrounding structures is described in a masterly way by Dr. Arthur Keith in his Hunterian Lectures.¹ The movements of the heart are very complicated. Keith points out that during systole the auricle first pulls up the ventricle, afterwards the ventricle pulls down and stretches the auricle, while even the contractions of the auricular appendages have a certain influence on the propulsion of the blood. For the perfect action of the heart it is necessary that all parts of the heart move freely within the pericardium and, to some extent, in the chest. An adhesion of heart and pericardium, therefore, has to be looked upon as an impediment to the propulsion of the blood through and out of the heart. There is a general opinion that this hindrance may be overcome by a healthy heart-muscle, so that an adherent pericardium may be found *post-mortem* in cases where there was no reason for suspecting the disease during life.

Another state of things arises when there is in addition a firm attachment of the pericardium to surrounding structures to the chest wall, in front and behind, and to the diaphragm. These adhesions may be formed by pleuritic adhesions (as probably in our case), or by inflammatory processes in the mediastinum, or by both affections. It is easily understood, and well described by Keith,¹ Harris,² and Brauer,³ what an enormous strain is thrown on the heart by these adhesions. Indeed, the heart is hindered in its lateral forward movements by tough and hard fibrinous masses, so that it can neither be properly filled nor properly emptied. By its shortening at every systole it pulls the chest wall in front, and, by means of the posterior mediastinum, at the vertebral column. Neither of these structures gives way. In youthful individuals the ribs may be so flexible that the heart is able to pull them inward; in older patients this will be impossible; and in both cases the heart has opposed to it an enormous amount of extra work. There cannot be a long struggle, the heart must be soon exhausted, and the well-known symptoms of heart failure supervene.

The mechanical impediments to the heart's action in the first instance are responsible for the following symptoms:

1. Insufficient circulation, frequent heart action, weak pulse, low arterial pressure, overfilling of the veins.
2. Systolic retraction of the chest wall in the regio cordis.

DISTURBANCE OF RESPIRATION.

There is another important function that is hampered by the adhesions in the chest, namely, the respiration. In literature we find but few and brief remarks on the respiratory disturbances in this disease (see Gibson⁴). Keith¹ says: "When the pericardium becomes fixed by adhesions to the lungs and chest wall a number of obscure symptoms follow, owing to the arrest of the normal respiratory movements of the heart." Indeed, I do not doubt that many of these "obscure symptoms" are much more the sequence of respiratory disturbance than of failing heart action.

In normal conditions the chest wall is lifted up during inspiration, by which movement the lungs expand in an upward and forward direction. At the same time the diaphragm descends, so that space is gained in a downward direction too. The diaphragm not only widens the chest, but compresses the abdominal contents; liver and bowels have to give way downward and forward, and the wall of the abdomen is thereby pushed forward. Thus the visible respiratory movements of the body wall are a moving of the chest wall upward and forward, of the abdomen in a forward direction only. These movements may be seen in Fig. 1, which shows the outlines by instantaneous photographs taken during inspiration (dotted line) and during expiration. The forward movement is easily recognized; the upward movement of

the chest is obvious from the changed position of the shoulder, the sternal end of the clavicle, and the nipple.

Fig. 1.—Normal respiration. The dotted line is the position in inspiration (from photographs of a healthy young man).

With the diaphragm the heart descends in inspiration, and not only the apex of the heart, but the whole heart with the roots of the lungs. Keith has worked out this problem in an excellent way, and showed how "the lungs carry their pump with them" in respiration. These complicated movements are very important for circulation and respiration. By the downward movement of the heart space for air is gained in the middle of the chest, as may be seen diagrammatically in Fig. 2. The upper parts of the lungs, taking the least advantage of the respiratory movements of the chest (Keith), profit most by this sinking of the heart. During expiration the heart, like a wedge between the lungs, contributes to the emptying of the lungs.

What will happen now, when adhesions fix the heart and the diaphragm to the chest wall?

The "crura" of the diaphragm will try to pull the diaphragm downward; the diaphragm is fixed to the heart; the heart is fastened to the chest wall in front and to the surrounding mediastinal parts behind. The heart is, therefore, unable to follow the diaphragm, and the diaphragm is hampered in its descent. As a result, note—

1. The descent of the diaphragm is insignificant.
2. During inspiration the crura pull at the heart, and, via the heart, at the chest wall, at the roots of the lungs, and the posterior mediastinum. The traction will be in the direction of the arrows in the diagram (Fig. 3).
3. The heart and large vessels being fixed to their surroundings, inspiration produces a worse condition than expiration.
4. Respiration becomes very defective in all its factors.

CLINICAL SYMPTOMS.

We have now to ask, What clinical symptoms result

from this disturbed mechanism of respiration?

Taking the last point first, we find the diaphragm greatly limited in its movements; thus the abdomen will not be pushed forward during inspiration, or very slightly. At that same moment the diaphragm pulls, by

Fig. 2.—Diagrammatical front view of the chest. Expansion of the lungs in down inward direction during inspiration (dotted line).

means of the heart, at the chest wall in the regio cordis, so there is no forward movement of the chest, but rather an inspiratory retraction of the chest (see later).

This failure of the respiratory movements will have a most pernicious influence on the flow of blood from the splanchnic area to the heart. It is well known that the alternating rise and fall in thoracic and abdominal pressures are the chief factors that force the blood from the splanchnic area into the thorax.

So we may expect, and we find, a stasis of blood in the abdomen.

This inspiratory traction on the heart, which cannot give way, is bound to affect injuriously the action of this organ. The "debit" of the heart, too small in expiration,

Fig. 3.—Diagram of chest, heart, and diaphragm seen from the right. The arrows show the direction of the traction exercised by the crura diaphragmatis.

must become still smaller during inspiration. As a result of this embarrassing action during inspiration, the well-known symptom of pulsus paradoxus is produced—namely, a lessening of the blood pressure and of the pulse wave in the arterial system. Another symptom furnishes further proof that during inspiration the heart is working under worse conditions—namely, the inspiratory swelling of the veins at the neck. In normal conditions these veins are emptied by the increased suction of the thoracic cavity during inspiration. Here the contrary happens; the flow of blood to the heart is diminished. We may put aside the more subtle question whether there is a direct traction on the aorta or on the large veins, as some authors assume. The more embarrassed condition of the heart during inspiration is obvious, and both symptoms—the *pulsus paradoxus* and the swelling of the veins—are coupled together and have the same origin. Perhaps it is noteworthy that in the patient here described both symptoms were present; in another patient I saw both were absent.

I am quite aware that some clinicians give another explanation of the origin of the *pulsus paradoxus*. Sir James Barr⁵ says:

In the so-called *pulsus paradoxus* (there is no paradox, but merely an exaggeration of a perfectly normal phenomenon), and in Müller's experiment of expanding the chest with the glottis shut, the pulse may disappear at the wrist. This is due to the sudden emptying of the veins to fill up the vacuum of the chest, and with this removal of the obstruction to the capillary flow there is a simultaneous depletion of the arteries. It is not due, as has been supposed, to any kinking of the large vessels, nor to any sucking back of the blood into the aorta, as has been imagined by those who have forgotten that the pressure in the aorta is always high, and cannot be affected by a negative pressure in the thorax. It is due to a certain fall in pressure affecting the veins, capillaries, and arteries, and is most marked in cases of low arterial pressure.

A great deal might be said about this assertion. Certainly the explanation by an increased suction of the chest (be it working on the arteries or on the veins) holds good in a great many cases, distinctly where there are a

low arterial pressure, a weak heart action, and strong inspiratory movements. But it does not hold good in the cases of adherent pericardium. The particular consequence of this disease is the impediment to inspiration, not by narrowing of the air tract, but by the anchoring of the diaphragm. There is not an increased fall of pressure in the chest, but a very deficient inspiratory mechanism. And the proof that there is *not* an increased suction of the chest and *not* a sudden emptying of the veins is shown by the inspiratory *swelling* of the veins. So we have to adopt the explanation of an increased hindrance to circulation during inspiration; and I venture to think that the increase of the hindrance is due to the tugging of the diaphragm on the heart, that cannot give way on account of its being fixed to its immovable surroundings.

ANALYSIS OF HEART ACTION.

In the four or five months after my first visit to the patient he was growing much worse. The heart action became still more weak and frequent; there was dropsy of the whole body and the face. The liver remained enormously swollen and very sensitive to touch, so much, indeed, that the boy was not able to stoop, on account of the compression of the liver by this movement; nor could he lie flat on his back. The spleen was enlarged, and the veins were greatly overfilled, especially at the upper half of his body. The urine passed was not half a litre. The heart was irregular, and its size increased; the sounds faint, but pure. A minute analysis of the action of the heart was made by recording tracings of radial and venous pulses and apex beat.

Fig. 5 shows the radial pulse taken at the same time with the venous pulse, the apex beat, and the liver pulse. The venous pulse (phleb.) shows a large auricular wave *a*. The period of ventricular systole (*E*) in normal cases is represented by a negative phase in the venous pulse, lasting till the end of the systole and the closing of the semilunar valves. Here this depression is very shallow (*c* is the carotid wave), and at the end of the period *E* there is a rise in the tracing; this rise is due not to a regurgitation through the tricuspid valves, but to overfilling of the veins and the right auricle. The end of the systole is marked by a small wave (*v*) which appears in the "postsphygmie" period.

The liver pulse is more interesting. In cases of regurgitation from the right ventricle the well-known "positive" or "ventricular" venous pulse may be found, not only in the veins but even in the liver. MacKenzie showed that in some rare cases, such as tricuspid stenosis, a venous pulse of the normal "auricular" type may be found in the liver. Here we find the same phenomenon: by taking the radial pulse as a standard we easily recognize that the waves of the liver pulse are the same as those in the jugular pulse. The only difference is that they come just a little later, which may be explained by the longer way the waves have to travel before arriving in the periphery of the liver.

In the cardiogram (card.) the details of the systolic retraction of the chest wall may be studied. Comparing this tracing with the venous pulse we see that not only the ventricular contractions (card., 2—3), but even the auricular systole may be recognized (card., 1—2). Of course the ventricular contraction gives a deeper fall in the tracing. As soon as the contraction stops the ribs fly back; this moment (3) is exactly marked in the venous

Fig. 4.—Paradoxical respiration. The patient in the expiratory (a) and the inspiratory (b) position.

pulse by the beginning of the wave *v*. There is still more to be seen in this tracing—namely, the inspiratory tugging of the diaphragm at the heart, which is apparent from the deeper depression of the tracing during inspiration, at the same time the pulse wave becomes smaller. The whole tracing is sinking to a lower level, proving that there is not a heaving of the chest (and therefore a deeper systolic retraction), but a retraction of the whole chest wall. From this analysis it is evident that the heart symptoms in our patient are exactly what they might be expected from deduction.

A closer inquiry into the changes of the respiratory mechanism brought about by the adhesions in the chest is not less interesting. When the patient is breathing quietly scarcely any respiratory movement of the chest is visible. Only on deep and forced inspiration is there visible an upward movement of the chest. A lateral expansion of the chest, as was expected by Keith, was absent in this case. The increase of circumference of the chest was, in forced inspiration, not quite 1 cm.

Instantaneous photographs of the inspiratory and expiratory positions show clearly that there is only an upward movement of the chest, and that very limited. (Compare Fig. 6, I, with Fig. 2). There is no forward movement of the chest wall whatever. Looking at the patient, we see an inspiratory retraction of the lower part of the sternum and the ribs.

Looking at the photographs of Fig. 4, one might think *b* to be the rest position—*a*, with the vaulted chest, to be the inspiratory position, but really it is just the contrary; *b* is the chest in deep inspiration. That there is a real inspiratory retraction and not only a semblance of it is proved by Fig. 6, I, which gives the outlines of the photographs superimposed. So we find in this disease a paradoxical respiratory movement of the chest, a pendant to the so-called paradoxical pulse. The explanation of the phenomenon lies in the fact that by the adhesions the diaphragm not only pulls at the heart, but pulls at the chest wall in front. In youthful individuals, the lower part of the sternum and

the cartilages of the ribs may give way a little. The absence of a marked descent of the diaphragm in inspiration is proved by the absence of the vaulting of the abdominal wall. Even

we see (Fig. 6, I) an inward movement, probably due to the fact that the chest with the adherent diaphragm is strongly heaved off from the abdominal contents.

I found this paradoxical respiration in another patient (Fig. 7). Here we see not a true retraction of the sternum, but an absolute standstill of this part. (The little knob on the sternum was due to a cold abscess.) Looking at this patient there seems to occur a real retraction, but this semblance is caused by the heaving of the chest and a little vaulting of the epigastrium, the lower part of the sternum remaining on the same level. But one may gather from this fact that this loss of forward movement of the chest is easily recognized in patients. It is not at all necessary to take photographs to detect this phenomenon, and I insist on this point because I think it is a very valuable sign for the diagnosis of adherent pericardium, and reliable symptoms are greatly wanted here. Indeed, all other symptoms mentioned may be

found in other morbid affections or may be absent in adherent pericardium. Recent writers are unanimous, and I join them, in stating that the pulsus paradoxus is a fallacious symptom; one may find it in perfectly healthy people or in other diseases. It may be absent in cases of adherent pericardium (in the case of Fig. 7 for instance). Inspiratory swelling of the veins I found in other conditions, for example, in cases of very big hearts (cor bovinum). Every one who has studied Mackenzie's book on the pulse knows that a negative apex beat may be found in many people with healthy or enlarged hearts. A strong systolic retraction of the ribs may be conclusive, but it is very often absent. Some authors regard a diastolic collapse of the neck-veins as pathognomonic for this affection. As may be seen from Fig. 5 there was no such collapse in this case and from analysis of many hundreds of tracings I can say that this

Fig. 5.—Tracings of radial pulse (rad.), taken together with venous pulse (phleb.), apex beat (card.), and liver pulse (hepar.).

Fig. 6.—Respiratory movement (I) before operation, (II) after operation.

diastolic collapse may be seen in most cases of marked stasis in the veins with an enlarged heart acting not too quickly. The second diastolic heart sound, a nearly forgotten symptom recently called to memory by Brauer, is not always present; I missed it in 2 cases.

So every new symptom has to be welcomed, and I venture to think that the arrest or even the retraction of the lower part of the sternum with its cartilages in inspiration *must* be found in every case where there are adhesions between diaphragm, heart, and chest wall. At least I should be greatly pleased if some of my readers would seek for this symptom (shortly mentioned by some others, vide Gibson⁴) when they have the opportunity of examining such patients.

CAUSE, PROGNOSIS, TREATMENT.

In our patient there can be little doubt about the cause of the disease. The pleurisy, followed a year later by pericarditis, and the long-continuing rise of temperature, point to tuberculosis, which so often affects the serous membranes. There is no tuberculosis of the lungs, but probably the mediastinum is affected; there is a broad area of dullness on the sternum and a corresponding broad central shadow on the Roentgen screen. This mediastinitis, with the adhesions of the lung and the pleura on the left side, may form the "external" adhesions for the heart and the pericardium.

Prognosis is extremely sad in these cases, and therapy has been regarded as utterly powerless. In his admirable monograph on this subject, Harris,² after giving sixty-five pages of valuable information, deals, in a few lines, with therapy. He says: "There is little to be said on the question of treatment of this affection. . . . Our chief attention has to be directed to support and maintain the strength of the heart and to postpone, as long as possible, the onset of heart muscle failure." That was all, indeed, that might be said about treatment!

We have to thank L. Brauer for an improvement of this sad prognosis. Some years ago attempts had been made to bring relief to these patients by surgical treatment.

Beck and Delorme proposed to loosen the adhesions of the heart and the pericardium by operation. This operation was generally considered as too radical, and, moreover, the adhesions might be expected to be formed again in a short time. So no more was heard of this operation. Brauer, on the other hand, argued thus: The heart is fixed to the chest wall in front and behind. At every systole it has to pull on these immovable parts, and is hampered in its contraction; the bony construction of the thorax gives way neither behind nor in front. Would it not be a great saving of work for the heart if at the apex of the heart the chest wall could be changed into a weak and supple membrane by resecting the ribs? The idea was as simple as that of Columbus and the egg. Brauer realized it, and at the German Congress of Surgeons in 1903 had the pleasure of showing three patients greatly improved by his operation. Brauer called it "cardiolysis"; it loosens the chains of the heart. The resection of some ribs and cartilages in the regio cordis was generally regarded as not very dan-

gerous. Moreover, the improvement of the patients was so remarkable that Brauer's proposition to try this operation in cases of adherent pericardium was received favourably by the surgeons present.

In our case prognosis seemed as bad as in every other case. Dyspnoea and dropsy went on increasing; digitalis and other tonics and stimulants had only a temporary effect; diuretics and a salt-free diet were useless. An attempt to help the patient by operation therefore seemed justified. Brauer thinks that the best cases for operation are those where a strong systolic retraction of the ribs may be seen as a sign of a still vigorous heart muscle.

In our case this retraction was not so very strong; but, seeing no other issue, operation was decided upon with general consent. I am indebted to Professor Koch of this University for his interest in this case, and for the skilful performance of the operation. On January 11th, 1905, a horseshoe-shaped incision was made in the regio cordis. (See Fig. 8b.) The cartilages and part of the fourth to seventh ribs were removed and the left border of the sternum was nibbled off. The first phase of the operation gave some trouble; the recumbent position plus the chloroform gave the patient an attack of extreme oppression with severe dyspnoea and a very small pulse. Happily this attack soon ceased, and when narcosis was well established all went well. The operation requires a well-trained hand, the periosteum of the ribs having to be removed carefully, which is made somewhat difficult on account of the stormy movements of the heart. These wild movements give one the impression that the operation has really been a releasing of the heart. Another observation made during operation was the apparent relief to the respiration. As soon as the arcus costarum was removed respiration became more effective, and the patient became much quieter. Strong adhesions in front of the heart were not found, so here the fixation of the heart must have been produced chiefly by the pleuritic adhesions on the left side. Perhaps this may be the reason that the result of the operation has not been so successful as in Brauer's cases.

Still, the result of the operation was very remarkable, as may be seen by comparing Fig. 8a and Fig. 8b. The most visible improvements were a better respiration and the decrease of the enormous liver. Having to deal extensively with these two phenomena at the end of this paper, we may first consider the other symptoms.

The heart's action did not improve much, the pulse frequency fell from 125 per minute to 110 and 100 per minute, the pulse remained small and weak, the arterial pressure (diastolic after Oliver) remained 73 to 75 mm. The pulsus paradoxus and the inspiratory swelling of the veins persisted. The flow of urine did not increase very much, seldom reaching 1 litre; the general anasarca diminished slowly.

Still, after four or five weeks the patient was much better, there being only some oedema of the legs, and the general anasarca had greatly diminished. He felt much better, and was now able to lie on his back and to stoop.

In the eighteen months now passed away the heart's action has not improved much, which makes it probable that there is still a hindrance. But we have to consider that the morbid affection itself did not come to a standstill after operation. Until the three last months there was a daily rise of temperature, probably due to tuberculous processes in the mediastinum. Another impediment to the training and re-education of his heart was the appearance of ascites. It is well known that ascites (often without general anasarca) is a very common occurrence in adherent pericardium. There is a great deal to be said about the genesis of this ascites, but it would take me too far to discuss this point here. So I will merely state that puncture and drainage have been performed twice, after which the ascites, without any

Fig. 7.—Inspiration (dotted line) and expiration in a second case.

Fig. 8.—Aspect of the patient before (a) and after (b) cardiolysis.

special treatment, disappeared. Small doses of digitalis were given occasionally to stimulate the heart, and to increase the flow of urine. Only at present, after the disappearance of the fever and the ascites, we may make our first attempt to improve the circulation by carefully graduated gymnastics, by massage, and other methods of physical therapeutics. Still, looking back at the time before operation, we may surely say that cardiolysis, though not so successful here as in some other cases, has saved the life of our patient, at least made life bearable to him.

THE SWELLING OF THE LIVER.

Before taking leave of the subject, we have to deal with one of the most striking symptoms of adherent pericardium—namely, the enormous liver. In nearly all cases of adherent pericardium there is a history of swelling of the liver, and many authors mention the fact that the swelling of the liver is beyond all proportion to the general venous stasis. As a matter of fact, we may find, as in our case, the liver a huge hard and painful mass in the abdomen before there is any sign of back pressure in the other veins. Some authors have mentioned the fact without giving an explanation, simply putting the problem before their readers; others have given explanations which cannot be called wholly satisfactory. One may find complete information about this

subject in an excellent little book of Otto Hess.⁶

The following explanation I deduce from the very instructive figures given by Keith in his papers, and from the result of the operation of cardiolysis in our case.

After operation, it struck me that, as I pointed out before, the mechanism of respiration had profited from the cardiolysis much more than the heart

Fig. 9.—Diagram of heart, liver, and diaphragm, and the relation of the crus to these organs.

itself. An objective proof of this improved respiration is given in Fig. 6, II, where the inspiratory heaving of the chest is seen to be much higher than before operation. A second change, not visible in the figure but very conspicuous on inspection of the patient, is brought about by the deep retraction of the operated area during inspiration. Coincident with the improved respiration, the liver became much smaller.

Taking into account the insignificant improvement of the heart's action, it seems probable that a relation exists between the improvement of the respiratory mechanism and the decrease in size of the liver. Keith has shown how the diaphragm in its descent not only compresses the abdominal contents and widens the chest, but actually compresses the liver, so that this particular function of the diaphragm is lost. In adherent pericardium, the wide and flaccid inferior vena cava passes to the heart behind the liver, practically in a groove. Immediately below the diaphragm it receives the contents of the liver veins; immediately above the diaphragm it pours its contents into the right auricle. Behind the vein the crus dextrum, the chief motor of the diaphragm, extends from the lumbar vertebrae to the aponeurosis of the diaphragm, fixing itself to the pericardium under and behind the right auricle (see Keith⁷ and the diagram Fig. 9).

In normal conditions the arrangement of the crura and the border muscles of the diaphragm causes a downward

and forward movement of the central tendon, pericardium heart, and roots of the lungs (Keith), and the liver is pushed in the same direction. All these organs coming down in perfect harmony, the vena cava inferior is gently compressed between the crus and the liver, and by this aid is emptied into the heart. In adherent pericardium, however, the crus and the border muscles of the diaphragm are pulling, but the whole complex of organs is immovably fixed in the chest. The contraction of the crus has not now a gentle action on the vena cava, but compresses it more strongly, the liver remaining in its expiratory position; at the same time the crus pulls on the heart and the pericardium at the spot marked X in Fig. 9. A narrowing or a closing of the vena cava must be the consequence of this movement, the heart not giving way to the traction. So I think in inspiration the issue of the vena cava is narrowed by the traction of the crus, the part of the vena cava behind the liver is compressed by the same movement, and the contents of the vein, not being allowed to flow back, are pumped into the liver veins.

REFERENCES.

- ¹ *Lancet*, 1904, i, pp. 556, 629, and 703. ² Harris, *Indurative Mediastino-Pericarditis*, London, 1895. ³ Brauer, *Arch. f. klin. Chir.*, Bd. 71, Heft 1; and *Verh. des XXI Congresses f. inn. Med.*, 1904. ⁴ G. A. Gibson, *Diseases of the Heart and Aorta*, ed. 1898. ⁵ J. Barr, *BRITISH MEDICAL JOURNAL*, 1906, ii, p. 401. ⁶ *Ueber Stauung und chron. Entzündung in der Leber und den serösen Höhlen*, Marburg, 1902. ⁷ Keith, *Journ. of Anat. and Physiol.*, vol. xxxix, p. 243.

An Address

ON

ANOMALOUS CASES OF PERNICIOUS ANAEMIA.

DELIVERED BEFORE THE PERTHSHIRE BRANCH OF THE BRITISH MEDICAL ASSOCIATION ON NOVEMBER 9TH, 1906.

BY G. LOVELL GULLAND, M.A., B.Sc., M.D., F.R.C.P.E.,

PHYSICIAN TO THE CHALMERS AND ROYAL VICTORIA HOSPITALS ASSISTANT PHYSICIAN TO THE ROYAL INFIRMARY, EDINBURGH.

GENTLEMEN,—I have been much honoured by your kindness in asking me to come here to-night, and I had some difficulty in thinking of a subject upon which to address you which would be of interest. My experience with the post-graduate classes which have recently been held in Edinburgh has shown me that diseases of the blood are now awakening a very wide interest among men in general practice, and I should like to take the opportunity of talking over with you what I have seen in a large number of cases of pernicious anaemia which have specially impressed me in the last year or two.

I do not propose to discuss the ordinary typical cases, but desire to take up rather those which showed unusual features, either as regards diagnosis, clinical appearances, or the results of treatment.

I have a distinct impression that pernicious anaemia is becoming a more common disease than it was fifteen or twenty years ago. During the last year I have seen 37 cases, 21 in private, 16 in Chalmers Hospital, and in addition a number of cases which were under the care of the physicians in the Royal Infirmary, or were seen in the medical waiting-room, and of which I have not kept full records. A large number of these were, of course, sent to me as being specially interested in the disease, but an almost equal number were met with, so to speak, by chance. A dozen years ago a case of pernicious anaemia was regarded as something of a rarity, even in the Edinburgh Infirmary. Now one would hardly ever see a ward on the medical side without one, and I have on one occasion had as many as six cases at one time in Chalmers Hospital. If this increase in number is a real one, it behoves us to be very much on the look-out for the condition, and to be ready to diagnose it; and this all the more, because it may present itself under the most diverse aspects, and the anaemia may apparently be quite a secondary consideration.

In thinking of the disease, one must keep clearly before one's mind the fact that it is not essentially a blood disease at all, but is a toxæmia of largely unknown, and