On defective closure of the tricuspid foramen as a frequent cause of dropsy / by Peyton Blakiston.

ON

DEFECTIVE CLOSURE OF THE TRICUSPID FORAMEN

AS A

FREQUENT CAUSE OF DROPSY.

BY PEYTON BLAKISTON, M.D. CANT. F.R.S.

Physician to the Magdalen Asylum, and to the General Dispensary, Birmingham.

Read at the Royal Medical and Chirurgical Society of London, January 12, 1841.

(From the London Medical Gazette.)

ON DEFECTIVE CLOSURE

OF

THE TRICUSPID FORAMEN

AS A

FREQUENT CAUSE OF DROPSY.

Numerous morbid appearances have been found in the hearts of persons, who, during lifetime, have had serous infiltration, and other symptoms indicative of obstruction existing at the centre of the circulation; and such morbid appearances have been usually considered as the causes of this obstruction. Those most commonly met with are diseases of the different valves, by which the respective openings to which they are attached have been contracted, or rendered permanently patulous—an increase or diminution of the substance of the walls of the heart, and of the capacity of its cavities.

Now, with the exception of those diseases which render the tricuspid valves incomplete, there is not one of these appearances which may not be met with in the bodies of persons who have never presented the slightest trace of dropsy.

As regards valvular disease, Bouillaud relates a case in which the mitral foramen was contracted to an opening of three lines diameter; and although this had in all probability existed three years, the ankles only became slightly œdematous a little before death. I have in my possession two preparations in which the same foramen is much contracted and rendered patulous. During lifetime there were several attacks of cerebral and of pulmonary apoplexy with hæmoptysis, but no dropsy.

Dr. Hope describes a case in which the aortic orifice was reduced to the size of a pea, without any serous infiltration; and in this paper two instances of extreme narrowing of the same orifice will be adduced, which had evidently been of long standing. In one of these, dropsy did not come on till a few months before death, and in the other the ankles became œdematous only a few weeks previous to that event.

The phenomena observed in these two cases may, in some measure, tend to explain why such extensive obstruction to the circulation may exist at the origin of the aorta without greatly affecting the general health. The heart's action was fluttering, and was accompanied by a very *prolonged* rasp sound, thus indicating the continued action of the ventricle, whereby the blood was at length squeezed through the contracted foramen, and explaining the cause of the thready vibratory motion which replaces the pulse in such cases. Andral relates a case* of extreme narrowing of the aortic orifice by ossific deposit, and states it as certain that many aged persons are similarly affected without being attacked by dyspnœa or dropsy. He attempts to account for the absence of such effects by the fact of the slowness of the circulation in aged persons; but it appears to me that the explanation given, based on the observation of Cases XI. and XIII., is more satisfactory.

Since, therefore, serous infiltration does not seem to depend on the degree of obstruction to the circulation existing at the left side of the heart, it is probable that there is some other obstacle more direct in its action and more constantly present. Dr. Hope, who takes this view, considers hypertrophy and dilatation of the heart to be the cause of obstruction. He remarks, "So long as the heart remains free from dilatation, hypertrophy, or softening, the valvular disease, according to my observation, is not in general productive of great inconvenience. This opinion is founded on the following grounds:—I have seen individuals, who were affected in an eminent degree with disease of the valves of the aorta, maintain for years a very tolerable state of health so long as there was no hypertrophy or dilatation of the heart: but, in proportion as these supervened, the symptoms of valvular obstruction became more and more developed, and eventually assumed their most aggravated form. I have reason to believe that in these cases the symptoms were attributable in a great measure to hypertrophy or dilatation, because I have seen a greater valvular contraction produce less severe symptoms when the hypertrophy or dilatation was less considerable. I have not only seen numerous cases of a mechanical obstacle unattended with passive congestions, dropsy, &c. but I have seen a still greater number of instances in which all the phenomena of an obstructed circulation were occasioned by dilatation alone, as no other obstacle capable of accounting for them could be detected in the course of circulation." And again, "I have repeatedly witnessed cases in which a well-marked, if not a considerable obstacle—as a contracted valve, a regurgitation, or a dilatation, or aneurism of the aorta—had subsisted for a long period, even for years, without producing any material symptom of an obstructed circulation; but the moment that dilatation of the heart supervened the symptoms made their appearance in an aggravated form."

Fully agreeing with Dr. Hope as to the frequency of hypertrophy and dilatation of the heart in cardiac dropsy, more especially dilatation, I do not think that they can be considered as the direct causes of it. "Every day (says Bouillaud) we find otherwise skilful physicians attributing to dilatation of the heart a number of evil effects, which in fact are only accidentally joined with it, such as passive congestion and the sense of suffocation which generally accompanies it." Active hypertrophy of the left ventricle may so increase its power that too much blood may possibly be thrown into the capillaries, thereby giving rise to congestion and serous infiltration; and concentric hypertrophy, whereby a diminution of the capacity of any one cavity of the heart takes place, must create a certain obstruction to the circulation; and yet numerous cases could be adduced in which each of these morbid states of the heart existed without having given rise to dropsy.

If the heart be generally dilated, and at the same time attenuated, it may be considered that it has not sufficient power to carry on the circulation properly so that congestion may supervene; but this is only one particular case.

On the other hand, when there is general hypertrophy and dilatation of the heart, so that the proportion between all the cavities is preserved, no cause of obstruction can be recognised; and yet such cases are very commonly accompanied by dropsy. Andral, al-

* Clinique Médicale, Brussels, 1834, vol. i. p. 50.

luding to such cases, observes, "What can produce dropsy in such a case? For all the cavities being in the same physical condition, ought they not to receive and forward the blood with equal proportion and freedom? If there is no obstacle to the circulation, no stagnation, no retardation of the course of blood in the heart, there ought to be no dropsy."

I think, however, that I shall be able to shew that there does exist an obstacle to the circulation in such cases, and if so it will be useless to search for the cause of dropsy, with Andral, "in the excess of the capacity of the heart relative to that which has been preserved in the blood-vessels."

As, indeed, dilatation of the heart, with or without hypertrophy, is, of all others, the most constant alteration of this organ, coexisting with cardiac dropsy, it is highly probable that some additional obstacle to the circulation is somehow connected with it, if not dependent upon it: and as the most direct obstruction to the venous circulation must exist on the right side of the heart, it is still further probable that it is connected with dilatation of that side.

Now the dilatation of the right ventricle necessarily gives rise to the enlargement of the tricuspid foramen*, unless in such cases where the fibrinous zone, from which its valves spring, shall have to a certain extent lost its natural elasticity. One such case I have seen. The tricuspid valves intended to effect the closure of this aperture during the contraction of the ventricle, are only just sufficient for this purpose. Hunter, and at a later period Dr. Adams and Mr. King, have observed that they do not very effectually close the orifice they are attached to in its healthy state. Consequently, if this orifice become dilated, the valves can no longer effect its closure, except they also increase in size. In some cases of hypertrophy such is the case, (Case V.) but I think not so generally as Dr. Hope supposes; at least, as far as my own observations, confirmed by those of Bouillaud, would lead me to suppose.

* Dr. Copland, in his Dictionary, Part V. article Heart, p. 210, remarks, "When both the ventricle and auricle of the same side are much dilated, the intermediate orifice is generally also widened, and the valves insufficient to close it."

Hence, regurgitation must not unfrequently take place through the tricuspid foramen during the systole of a dilated right ventricle; and thus a most powerful obstacle is opposed to the venous current, by a quantity of blood being constantly forced back upon it: and although this may in certain cases act as a safety-valve to the lungs, as supposed by Mr. King, yet if at all extensive and continuous, it can hardly fail to offer a most effective obstruction to the circulation returning from the system. This regurgitation through the tricuspid foramen I propose to shew, by the following cases, is a frequent and direct cause of cardiac dropsy*. The same obstruction would be produced by any other causes which prevent the tricuspid valves from closing the foramen to which they are attached, whether dilated or not. There are two causes of this sort which have hardly received the attention they deserve. The one is a shortening and thickening of the cordæ tendiniæ, which Mr. Hodgson tells me he has frequently seen; and the other, a partial or total adherence of the valves to the walls of the ventricle, unaccompanied by any other traces of disease. Instances of both kinds will be found in this paper. (Cases I. II. X. XI.)

A difference of opinion seems to

* Dr. Copland, in his Dictionary, Part V. article Heart, page 212, observes, "Dilatation gives rise to no indications of its existence, unless it is so considerable as to permit a reflux of the current of blood, and even then the signs are equivocal. This influx is one of the causes of the bellows sound and of the purring tremor. When it takes place through the right auriculo-ventricular opening, it causes venous pulsations." But there is not a word of dropsy; nor can we suppose from this that he considers regurgitation through the tricuspid foramen in any way intimately connected with dropsy, much less that it is a frequent and direct cause of cardiac dropsy. It will be shewn in this paper that such regurgitation does not necessarily give rise to venous pulsations. Dr. Adams writes, "Mr. Hunter, in his treatise on the Blood, has remarked that the valves of the right side of the heart do not so completely close the arterial and auricular openings as those of the left; but this circumstance, in my opinion, has not been sufficiently noticed, nor the influence that such a structure may have upon the circulation in its natural or morbid state considered." He then goes on to consider the safety-valve function, and the mechanism of venous regurgitation and pulsation; but there he stops, and does not insist upon the additional obstacle to the circulation produced by morbid regurgitation. Farther on he recommends accurately measuring the relative proportion between the valves and the opening, in all cases of regurgitation, looking especially to the presence or absence of pulmonary obstruction in these cases.—*Dublin Hospital Reports*, vol. iv. pp. 437-8.

exist between writers on diseases of the heart, as to the sufficiency of the proofs of regurgitation through the tricuspid foramen, both as drawn from observation during life and autopsy after death. Thus pulsations of the jugular veins, synchronous or nearly so with the systole of the ventricles, which have been considered by most writers as indicative of such regurgitation, are looked upon by Dr. Hope as produced by the force of the right ventricular systole, and altogether independent of the completeness or incompleteness of the tricuspid valves.

It must be borne in mind, however, that considerable hypertrophy of the right ventricle is not unfrequently found in the bodies of persons who during life-time presented no appearance of jugular pulsation. And were it possible that the ventricle could ever contract with such force as to communicate a shock to the valves and blood above them, which should run up the jugular veins, it could only be in cases of extreme hypertrophy of the right ventricle, and therefore would be of rare occurrence, and would be accompanied by signs of such hypertrophy.

When, therefore, pulsations or obscure fluctuations of the jugular veins are observed, unaccompanied by any very strong heaving impulse of the heart, it is, I think, highly probable that regurgitation takes place through the tricuspid foramen. A perusal of the cases brought forward in this paper will be found very much to favour this view, inasmuch as in all those where venous pulsations or undulations were observed during life-time, the tricuspid foramen was found incomplete, either from its dilatation or the imperfect action of its valves, or from both causes.

That the force of pulsation of the jugular veins is a measure of the power of the right ventricle, and not a measure of the degree of regurgitation and obstruction to the circulation, I readily admit; for in all cases the stronger be the force of the right ventricle which throws the blood back upon the veins, the stronger will be the pulsation propagated up them. Not so as regards the obstruction to the circulation, which depends on the relative proportion between the power of the two ventricles. If both be hypertrophied, the pulsation or shock will be great, and if both be attenuated the pulsation will be weak, while the obstruction to the circulation is the same in each case; for the venous current and that of the regurgitating fluid, being both mainly derived from ventricular contraction, when the one is strong it is opposed by the other equally strong; and when one is weak, by the other equally weak: the obstruction is therefore the same in each case, and it is perfectly independent of the shock or pulsation. This is strikingly illustrated in Case XIII., where the pulsation of the jugular veins was most vehement, but the effect on the circulation slight.

If the proportion between the power of the two ventricles be altered, the effect on the circulation is altered; thus, if the right ventricle be hypertrophied and the left attenuated, a weak venous current is opposed by a powerful current of regurgitation, and the circulation is powerfully obstructed; if, on the other hand, the left ventricle be hypertrophied and the right attenuated, a strong venous current is opposed by a weak current of regurgitation, and a feeble resistance is offered to the circulation.

While, therefore, pulsations of the jugular veins may, in certain cases, be considered as indicative of regurgitation through the tricuspid foramen, their absence affords no proof of the non-existence of such regurgitation.

It has been proposed to test the completeness of the tricuspid valves after death by the manner in which they retain fluid in the right ventricle, which has either been forced or injected into it through the pulmonary artery. Mr. King has seldom found the valves to retain fluid when the experiment has been made with a healthy heart; and I have several times repeated the experiment with no better success, unless I pinched in the base of the right ventricle surrounding the foramen with my hand. This therefore would be an unfair test, as it would go to prove that almost all tricuspid valves were incomplete. Bouillaud has compared the measure of the circumference of the foramen with the height of the valves from their apex to the middle of their base. This latter plan gives a very insufficient idea of the area the valves can cover; because unless the circumference of the foramen be retained in its proper position, which is

very difficult to do, the valves will be stretched, and will measure more than they can do in action.

He gives three inches eleven lines as the mean circumference of an undilated tricuspid foramen.

I have been in the habit of removing the apex of the heart and laying open the right auricle, and then raising up the tricuspid valves into their plane of closure, by means of my fingers introduced into the right ventricle. By looking at them from the auricle when in this position, a tolerably accurate idea can be formed of the area they cover, and whether any and how much space is left uncovered by them, through which the blood can regurgitate. I have then usually gauged the tricuspid foramen by the introduction of the fingers; its ordinary size not quite admitting the three first fingers up to their first joint, and then measured the circumference after it has been laid open. I regret that the size of the heart should have been noted in such loose terms. I have lately measured its bulk and capacity, by ascertaining the quantity of fluid it displaces both when full and empty, and hope by this means to arrive at a more accurate standard of measurement.

In the cases which follow, and which I have condensed as much as possible, the heart presented a great variety of morbid appearances; but one peculiar state was common to them all, and that was the incompleteness of the closure of the tricuspid foramen. In two cases this arose from adhesion of one or more valves to the ventricle; in eleven from dilatation of the foramen to such an extent that the valves could not effect its closure. In most of them the dropsical effusion was preceded by turgescence of the jugular veins: in those of them where the heart was not softened or attenuated, it was preceded by pulsations in these vessels. I shall not in this place enter upon the causes which tended to produce dilatation, which will in most of the cases appear tolerably clear.

Being anxious to know how far the views in this paper would be borne out by cases recorded by others, I have consulted the work of Bouillaud, who is the only writer on diseases of the heart that has carefully measured its orifices. He gives thirty-four cases of cardiac dropsy, of which the following table gives the analysis:—

Analysis of 34 Cases of Cardiac Dropsy, detailed by Bouillaud.

Tricuspid foramen dilated and its closure incomplete, with the valves adherent in some	16
Tricuspid valve diseased so as to interfere with its action	4
Their chordæ tendiniæ shortened, producing the same effect	2
Concentric hypertrophy of right ventricle, with great diminution of cavity	2
Foramen dilated, and valves enlarged, so as to close it	1
Valves and foramen healthy	2
No mention of foramen, but both right auricle and ventricle dilated	7
	34

In these last seven cases there appears to have been no measurement of the orifices of the heart, but as there was dilatation of both ventricle and auricle on the right side, we may conclude that the foramen between them was also dilated.

If this be allowed, it would follow that there were only three cases out of the thirty-four in which there was complete closure of the tricuspid foramen.

From this it might be supposed that Bouillaud was fully aware of the frequency of this pathological state of the heart, and of its intimate relation to dropsy as cause and effect. He simply remarks, however, "The dilatation of the orifices of the heart is not less common than that of the cavities themselves," and he recommends "diligent observers not to neglect examining the orifices of the heart in all its organic diseases, for if the dimensions are such that the valves cannot close the orifices, the circulation of the blood through the heart must be more or less seriously disturbed*."

Here he stops short, and does not seem to be aware of the extent of obstruction which is thus so frequently offered to the circulation, and that in a majority of cases dropsy does not come on till the tricuspid valves have become incompetent to close the dilated orifice to which they are attached. Overlooking the fact that contractions of the orifice of the left side are not uncommonly found unaccompanied either by extensive dilatation or by dropsy, he considers, throughout the whole of his work, that these contractions are the main cause of those disturbances to

* Paris, 1835, vol. ii. p. 526.

the circulation, of which dropsy is the most frequent and the most dangerous consequence. Nor does he seem to suspect that incomplete closure of the tricuspid orifice and consequent regurgitation through it, is the real and only impediment to the circulation in those cases where the heart is dilated and hypertrophied in an equable manner through its whole extent.

Case I.—A married lady, æt. 29, had not enjoyed good health since an attack of rheumatic fever in early youth. She had for four months suffered from dyspnœa, during which time she constantly expectorated clear mucus, sometimes mixed with dark blood, and had occasional attacks of severe pain in both sides, with fever. She could not lie flat; her urine was not coagulable by heat. In about a month after this her legs began to swell, and a slight bellows sound was heard over the præcordial region, with the first sound of the heart, when she was hurried or excited. The veins of the neck, which had always been rather full, now became much swollen, and pulsated strongly. The œdema of the legs increased, ascites appeared, and she died, the subject of extensive anasarca, three months after my first visit.

Autopsy.—There was considerable serous effusion in both sides of the chest and in the abdomen. Some patches of circumscribed pulmonary apoplexy were seen in the lungs.

The heart was more than twice the size of the fist, being hypertrophied and dilated throughout. Two flaps of the tricuspid valve acted imperfectly, one being partially, and the other entirely adherent to the right ventricle. The tricuspid foramen was greatly dilated, and its circumference measured five inches and a half. An immense transparent coagulum, of the consistence of jelly, was lying in it, extending into both the auricle and the ventricle. The other organs were healthy.

Case II.—Sarah Richards, æt. 14, pigeon-breasted; had rheumatic fever twelve months since, with very severe pain under the left breast, and distressing dyspnœa. When examined, complained of great dyspnœa, and occasional severe pain in the præcordial region; the urine was not coagulable by heat.

On the left side, the chest sounded dull from the second rib downwards. A purring thrill was felt under the left mamma. At the same spot bellows sound was constantly heard, and for some distance around it.

In two months' time the veins of the neck were observed to fluctuate obscurely. In another fortnight the ankles and legs became œdematous, ascites soon followed, and in three months from my first visit she died extensively anasarcous.

Autopsy.—A large quantity of clear lemon-coloured fluid was found in the abdomen, about a pint in each pleural sac, and three ounces in the pericardium. The lower part of each lung was slightly carnified; they were otherwise healthy.

The heart was dilated to nearly double its natural size; the walls retaining their usual thickness. The valves were not thickened, but a part of the tricuspid valves was glued down to the side of the right ventricle, thereby preventing the closure of the foramen, the circumference of which measured four inches and a quarter. The other organs were healthy.

Remarks on Cases I. and II.—The hypertrophy and dilatation of the heart in Case I., in all probability resulted from an attack of pericarditis and endocarditis during rheumatic fever—a result which Andral and others have shewn to be by no means uncommon. The intimate adhesion of one of the tricuspid valves to the right ventricle may also have taken place at the same time; but when we recollect that this fever took place in early youth, it is hardly possible to believe that incompleteness of the tricuspid valves could have existed for so long a period without giving rise to more serious consequences. The hypertrophy of both ventricles accounts for the strong pulsations observed in the jugular veins. The manner in which their appearance and increase was followed by dropsical effusion cannot but lead to the belief that it depended on the dilatation and consequent incompleteness and regurgitation through the tricuspid foramen.

In Case II. nearly the same phenomena were observable, with this difference, that there was only obscure fluctuation and not pulsation observable in the veins of the neck, and this because the ventricle was not hypertrophied.

I may here remark that Mr. Baynham has given me the particulars of a case

of general hypertrophy and dilatation of the heart, with incompleteness of the tricuspid valves from the enlargement of their orifice, unaccompanied by dropsy. The foramen ovale, however, was unclosed and spacious, so that the shock of regurgitation was divided between the veins of the general circulation and those of the lungs. As might be expected from this, there was much dyspnœa in this case, the foramen ovale acting as a kind of safety valve to the general circulation at the expense of that of the lungs.

Case III.—Mary Newey, æt. 60, some years ago had an attack of acute rheumatism, since which time she has had a cough, with copious clear expectoration. During the last seven months she had suffered much from dyspnœa and palpitations; her pulse was feeble and irregular; the veins of the neck were much swollen, and presented a knotted appearance; they pulsated faintly. There was dulness for a large space around the præcordial region. The sounds of respiration and of the voice were natural. Bellows sound was heard over the heart. The heart's action was irregular; her urine was not coagulable by heat. The ankles were œdematous, and serous infiltration was becoming apparent in the thighs, when she was suddenly seized with apoplexy, and died in twelve hours.

Autopsy.—A great quantity of black coagulum was spread over the surface of each hemisphere of the brain, between the dura mater and the arachnoid membrane. There was a clot and laceration in the interior of each hemisphere. The heart was dilated to an enormous size, the walls being of the usual thickness, soft and flabby, of a yellow mottled colour, crackling under the fingers like a piece of lung. No valves were diseased.

The circumference of the tricuspid foramen measured nearly six inches, and its valves could not effect its closure. The other organs were healthy.

Remarks on Case III.—Here again the abnormal changes in the heart had probably their origin in the inflammatory action which was set up during rheumatic fever. While the symptoms of venous obstruction and dropsy were developing themselves, the patient was suddenly cut off by apoplexy. There can be little doubt that this was produced by the regurgitation of the venous blood through the dilated tricuspid opening, a similar instance of which came under my notice a short time since. A man had been under my care for twelve months, presenting all the symptoms of an hypertrophied heart succeeding rheumatic fever. Having one day stooped down much in the course of his employment, he was taken suddenly ill, and expired in ten minutes. The heart was found to be large and firm, being both hypertrophied and dilated. The tricuspid foramen measured five inches in circumference. The brain was gorged with dark blood, of which an immense quantity issued from the jugular veins.

It may be said that the soft and flabby state of the heart, by diminishing its muscular energy, and thus rendering it incompetent to propel the blood with sufficient force, would retard the circulation and give rise to the commencing anasarca, and would dispose to the apoplectic attack. Such a state of the heart might have this tendency; and yet I have several times found the heart in a similar state when no apoplexy and no trace of dropsy had been present.

Case IV.—John Higginson, æt. 46, boot-maker, an old soldier, who had lost the right leg above the knee: had been accustomed to drink spirits to excess. Nine months since he felt a pain under the sternum, followed by dyspnœa, both of which have continued ever since. When I visited him the dyspnœa was most distressing; he was unable to lie flat, and had a strong sensation of constriction across his chest. There was œdema of the leg and stump, and some ascites. The jugular veins pulsated strongly. There was dulness on percussion for some inches around the præcordial region. The respiratory sound was natural. The action of the heart was heaving, and its sounds distant. The pulse was very full and hard, and its stroke remarkably quick and sharp, which character it retained till death took place, three weeks after my first visit, having been preceded by expectoration of dark clotted blood for two days.

Autopsy.—There was serous effusion in the chest and abdomen. A patch of pulmonary apoplexy as large as an orange existed about the middle of the concave portion of the right lung, near

the pericardium, and was continued gradually into the healthy portion of the lungs. The heart was enormously hypertrophied and dilated. On its surface was a patch of thick yellow lymph. The dilatation of the right cavities was very great; the circumference of the tricuspid foramen was five inches and three quarters; one of the valves had short thickened chordæ tendiniæ; they could not close the foramen.

The aortic valves were thickened, but acted well. There were many patches of atheroma under the lining membrane of the left auricle. The aorta was a mass of disease, being thickened and puckered, and, in many places, denuded of its lining membrane, and so throughout all the large arteries.

The liver was of a nutmeg colour. The kidneys and other organs healthy.

With this last case I would contrast the following, wherein, in addition to the disease of the arteries, and the hypertrophy and dilatation of the heart, with enlargement of the tricuspid foramen, the tricuspid valves were so increased in size as to effect its closure, and hence there was no dropsy:—

Case V.—Priscilla Sturck, æt. 50: had a severe illness, with pain in left side, eight years ago, since which time she has occasionally felt beatings extending from her heart up to her head and down her arms, with giddiness. She has had latterly a constant cough, and when examined the pulse was small. Pulsations were felt above each clavicle. The action of the heart was heaving and tumultuous. The carotid arteries pulsated very strongly. Double saw sound was heard, its maximum being under the bottom of the sternum. Single saw sound under each clavicle, loudest under the right, was heard, without any second sound. Two years after this, being excited, she suddenly placed her hand over her heart, drooped her head, and died in half an hour.

Autopsy.—The heart was very much increased in size, being generally hypertrophied and dilated, more especially the left ventricle. The tricuspid foramen measured five inches in circumference; but the valves were hypertrophied in substance and extent, so that they effected the closure of the foramen perfectly. The aortic valves were slightly cartilaginous, but acted well. The lining membrane of all the arteries, particularly the aorta, was much diseased, being puckered and having much atheroma under it, and being deficient in some places. The arteries of the arms were diminished in calibre by the deposition of atheroma in their middle coats. The other organs were healthy.

Remarks on Cases IV. and V.—In Case IV. the hypertrophy and dilatation of the heart, and the diseased state of the arteries, would seem to have been induced by that sub-acute inflammation of the surrounding and lining membrane of the heart and large arteries, to which spirit-drinkers are subject. The tricuspid valves retaining their usual size, while the orifice to which they were attached was enormously dilated, strong regurgitation would take place, and, as the heart was firm and hypertrophied, would be marked by strong venous pulsations.

In Case V. a similar state of the heart and arteries was probably the result of an acute attack of inflammation; but as in this case the valves had increased in size in the same proportion as the orifice to which they were attached, they were capable of closing it, and no dropsy or venous pulsation ensued.

Now if in Case IV., exactly parallel to Case V. in all but the state of the tricuspid valves, the dropsy depended on any other cause than regurgitation through the incompletely closed tricuspid foramen, why did it not also supervene in Case V., where the complaint had been protracted through a period of eight years?

A comparison of these two cases goes strongly to prove that venous pulsations are not produced without regurgitation through the tricuspid foramen, because in Case V. the heart was quite as firm and as much hypertrophied as in Case IV., and yet there were no venous pulsations.

Case VI.—William Matthews, æt. 32, porter: was attacked with acute rheumatism twelve years ago, and again eight years since, when he had very severe pains in the left breast, troublesome cough, and dyspnœa. Eighteen weeks previous to my visiting him, his ankles began to swell; then his legs and abdomen. There was much dyspnœa. The urine was not coagulable by heat. Pulse hard, sharp, and vibratory. The jugular veins were swollen, and pulsated. There was dulness on percussion at the bottom of

each side of the chest. The first sound of the heart was accompanied by marked bellows sound. In a few days time intense pain at the præcordial region suddenly made its appearance, with most distressing dyspnœa. Pulse 120, very sharp and hard, with some fulness. Venesection being freely employed, with the administration of digitalis and mercurial frictions, the acute symptoms were removed; the anasarca and the pulsation of the jugular veins disappeared. The pulse remained very hard and vibratory. In a few weeks venous pulsation of the neck, rapidly followed by anasarca, reappeared, and he gradually sank.

Autopsy.—Much fluid was found in the abdomen and thorax, and four ounces in the pericardium. This membrane was thickened and opaque in patches. The heart was generally hypertrophied, and dilated to more than twice its natural size. The tricuspid valves were a little thicker and larger than usual, but could not nearly close their foramen, which would admit four fingers and the thumb nearly up to the knuckles, and the circumference of which measured nearly seven inches. The mitral valves were thickened, but free to act. The lining membrane of the left auricle was thickened and opaque, studded with several horn-like patches. On one of the aortic valves was a patch of soft, reddish, recently organized false membrane, of the size of a fourpenny-piece. The lining membrane of the aorta seemed thickened, and puckered unevenly. The other organs were healthy.

Case VII.—John Wilson, æt. 62, wood-turner: had an inflammatory attack of the chest eighteen months ago, since which time he has felt a tightness of the chest; for the last nine months dyspnœa, and lately palpitations. Six weeks ago his legs swelled for a few days; this went off, and returned three weeks since. Dyspnœa was most urgent. The jugular veins were seen to pulsate, chiefly on the right side; but were not turgid. The urine was not coagulable by heat.

There was dulness, more extensive than usual, around the region of the heart. The respiratory sound was coarse in places, with slight cooing. The action of the heart was tumultuous, heaving, and irregular. Pulse irregular, but moderate. The sounds of the heart were feeble and obscure, but on the right side of the sternum the first sound was louder, being slightly coarse and prolonged.

In two or three days time he began to expectorate dark blood, sometimes in clots. This, with increasing dyspnœa and anarcarsa, continued up to his death, which took place three weeks after my first visit.

Autopsy, forty hours after death.—The legs and thighs were anasarcous; a little fluid was found in the abdomen. At the inner edge of the back and lower portion of the upper lobe of the right lung (both of which were otherwise healthy) was a mass of pulmonary apoplexy, of the size of an orange, with abrupt edges. The heart was immense, and measured fifteen inches around the outside of the base of the ventricles. It was generally hypertrophied and dilated, firm and red. Within the folds of one of the aortic valves was a patch of semi-osseous matter, which did not prevent the free action of the valves. The circumference of the tricuspid foramen measured full six inches. One valve was large, the other two small. They were roughed by small hard substances, rather less than split mustard-seeds, particularly on their free edges. The coronary vessels were large, but healthy; and the aorta was studded with numerous soft elevations, produced by atheromatous deposit. The other organs of the body were healthy.

Remarks on Cases VI. and VII.—In these cases hypertrophy and dilatation of the heart seem also to have resulted from old attacks of inflammation; but in Case VI. an acute attack of endocarditis supervened; and this being met with active treatment, the dropsy disappeared for a time. The pulsations of the veins of the neck also ceased; but whether this arose from contraction of the tricuspid foramen when the heart was relieved from the pressure of blood by venesection, whereby regurgitation was prevented; or, which is more likely, by the diminished energy of the heart's action, produced by digitalis, cannot be positively determined. All the valves were slightly increased in size, yet the tricuspid orifice was so enormously dilated that they could not nearly close it; hence, in a short time, the old symptoms reappeared in an aggravated form.

I now pass on to cases wherein the dilatation of the heart may in whole,

or in part, be attributed to other causes than inflammatory action.

Case VIII.—A lady, æt. 43, was married at the age of thirty. Previous to her marriage she was very pale, and was subject to palpitations, dyspnœa, and swelling of the ankles. Menstruation was, at that period, very scanty and irregular. Since her marriage a slight improvement had taken place in this respect, but at times she suffers in an exactly similar manner. Has no family. For the last four months she had menstruated but very slightly, and had suffered much from flatulence and other dyspeptic symptoms. In this state she applied for advice. The sounds of the heart were feeble, but clear, and were heard over every part of the chest. The veins of the neck were slightly swollen. She derived much benefit from tonic alterative medicines, which, in a great measure, removed her dyspepsia. In a few months time she returned, with œdema of the legs and thighs, and a trace of ascites. Dyspnœa and palpitation were oppressive. The most active diuretic medicines had been employed without the least benefit. The veins of the neck were now seen to be very much more swollen, and a fluctuation in them was manifest. The urine was not coagulable by heat.

A small quantity of blood was taken from the arm, and tonics were combined with diuretics. In less than three weeks the swelling and fluctuation of the veins subsided, and every trace of dropsy disappeared.

Remarks on Case VIII. — There appears to be no reason why the heart should not partake of the general flaccidity which is found in all the other muscles of persons affected with chlorotic anemia. Hence, in Case VIII. such may be supposed to have been the case; and, as clear evidence of dilatation existed, we may conclude that it arose from the diminished tone of the parietes of the heart. Hence when depletion or diuretic medicines were employed, no good effect was produced; but the moment a tonic treatment was adopted, the heart seemed gradually to regain its power and usual dimensions; and the cause departing, its effect, dropsical effusion, departed also.

Case IX.—James Brittain, æt. 55, had, during several winters, cough, with expectoration of pearly mucus, wheezing, dyspnœa, and, last winter, œdema of the legs. In the spring all these symptoms, except the expectoration, disappeared. When visited at the commencement of another winter, he was found to have been ill for some weeks. There was great œdema of the legs, thighs, and scrotum. Urine was scanty and high coloured; not coagulable by heat. Respiration was hurried, and he had frequent palpitations. There was dulness on percussion around the heart. Sub-crepitant râle was heard all over the chest. The sounds of the heart were sharp and clear, and were heard over the whole thorax; but seemed distant when listened to over the præcordial region. He died in fourteen days.

Autopsy.—The lungs were gorged with serum. Large patches of emphysema existed at the summit of each lung; some cells were of the size of a horse-bean, with tough inelastic walls. The heart was dilated to three times the size of the fist, its parietes being of the usual thickness. The right ventricle and auricle were particularly large, and the tricuspid foramen measured six inches in circumference. Its valves were small and thin, and could not close the foramen. The other organs were in a healthy state, except that the lining membrane of the stomach was of an uniform bright scarlet colour, and rather mammelated at its great curvature.

Remarks on Case IX.—There is no cause of dilatation of the heart to be detected in Case IX. arising out of any affection of the organ itself. The emphysematous state of the lungs was, however, probably of long standing. This must have greatly affected the passage of blood through them, and therefore it would have a tendency to stagnate and accumulate in the right side of the heart, and thus promote its dilatation. Somewhat similar to this was the case of a lady whom I attended with Mr. Wickenden. The dilatation and attenuation of the heart was evidenced by its sounds, sharp and clear, being distinctly heard over every portion of the chest. This had been perceived, more or less, ever since a protracted attack of bronchitis. She was much relieved by the administration of expectorant and tonic medicines.

Case X.—Eliza Babington, æt. 14, had articular rheumatism, with some pain under the left breast, two years and a half ago. Six months after that she began to perceive palpitations, which have occasionally reappeared ever since, with depression, nausea, and great dyspnœa on exertion.

When visited she complained of constant nausea, long and violent attacks of palpitation, occasional headaches, and a dry cough. The pulse was feeble and small at times. The left side of the chest sounded dull from the second rib downwards; and over this space the respiratory sound was inaudible. A thrill or pulsation was felt between the fifth and sixth ribs, alternating with a similar one between the second and third ribs. A grating sound was heard with the first sound of the heart.

Six months after this, turgescence and strong pulsations of the jugular veins were seen. The feet, legs, and abdomen, became gradually and successively infiltrated. Urine became scanty; not coagulable by heat. A double grating sound was heard, which continued till her death, which took place nine months after she was first seen.

Autopsy.—There was much fluid in the thorax, pericardium, and abdomen. On either fold of the pericardium were several rough patches, feeling like a calf's tongue. The heart was hypertrophied, and dilated to more than double the size of the fist. The chordæ tendiniæ of one of the tricuspid valves were shortened and thickened. The tricuspid foramen measured nearly five and a half inches in circumference. The valves could not close it, a large space being left uncovered in the middle, not only from the inability of one of the valves to rise up to the plane of closure, but from the disproportion existing between the size of the valves and that of the foramen. The lining membrane of the left auricle was thickened and opaque, and near the mitral valves was granulated. These valves were thickened, and adhered to each other at their base, so that two fingers could not pass between them. The other organs were healthy.

Case XI.—James Bull, æt. 13, had a severe attack of rheumatic fever when five years old. Four weeks since felt palpitations, and three days back first felt pain under his left breast. When first seen by me he could not lie down, and dyspnœa was distressing. The pulse was small and vibratory; there were strong palpitations under the xyphoid cartilage. From the second down to the seventh rib of the left side the chest was bombed out an inch beyond the level of the other side. This space gave out a dull sound on percussion, and over it existed purring thrill. A loud coarse bellows sound was heard all over the chest.

In a month's time the projection of the left side had much decreased under the employment of iodine and mercurial frictions. A pulsation and thrill was then felt between the fifth and sixth rib, alternating in time with another between the second and third rib. In another month the veins of the neck became very turgid, the ankles œdematous, then the legs, and ascites followed. The urine was not coagulable by heat. In four months from my first visit he died, extensively anasarcous; the whole of the left side of the chest sounding dull, andthe respiratory sound being only heard on that side up the spine.

Autopsy.—The heart was enormous, extending up to the second rib, and upwards of two inches to the right of the sternum. The lining membrane of the left auricle was thickened and opaque, and was covered with patches of horn-like substance. This appearance was continued into the mitral valves, which were much thickened, and would not allow the passage of two fingers. The base of the aortic valves was thickened. The right ventricle and auricle were immensely dilated and attenuated. The tricuspid foramen measured five inches and a half in circumference. One of the tricuspid valves had its chordæ tendiniæ much thickened and shortened, so that it could not rise into the plane of closure; consequently the valve was very incomplete. Other organs healthy.

Remarks on Cases X. and XI.—The causes of dilatation in Cases X. and XI. may have been double, because, although the obstruction at the mitral valves may have caused stagnation of the blood in the cavities of the heart behind it, and thereby have given rise to dilatation, yet it must be borne in mind that the original attack of endocarditis, in which the alteration of the mitral valves originated, may, at the

same time, as in other cases, have predisposed the organ to hypertrophy and dilatation.

It is instructive to remark that, although the dilatation of the left auricle had in each case existed for a long time, as evidenced by the dulness on percussion, and the pulsations alternating with those of the ventricle, which were perceived much higher in the chest than usual—in a position, indeed, where the dilated auricles were found after death—yet that dropsy did not supervene until there were evidences of incomplete closure of the tricuspid foramen. This additional obstacle to the venous circulation was necessary to produce serous infiltration.

Case XII.—William Cashmore, æt. 61, iron-caster: had oppression of the chest and indigestion for twelve months. He complained of troublesome cough and distressing dyspnœa. The pulse was very small and thready. An obscure thrill was felt to the right of the upper part of the sternum. A single prolonged rasp sound was heard, its maximum being over the same spot where the thrill was felt.

Four months after this the pulse in each wrist was reduced to a mere flutter: nine months after this the intensity of the rasp sound was much diminished. The whole of the right side of the chest sounded dull, and the respiratory murmur of that side could only be heard up the spine. He could not lie on the left side. There was extensive dulness around the heart. In six months the veins of the neck became almost varicose; the ankles became œdematous, and in another month general anarsaca appeared. The urine was not coagulable by heat. A little before his death, which took place nearly two years after my first visit, a slight fluctuation could be occasionally detected in the jugular veins.

Autopsy.—The right side of the chest contained a gallon, the left a pint of fluid; there was a large quantity in the abdomen. The size of the heart was truly astonishing; it projected for some distance under the sternum into the right side of the chest. All the cavities were uncommonly dilated, and the walls of the right ventricle were attenuated. There were only two aortic valves, which were converted into bone, and stretched tightly across the orifice of the vessel. The free edge of the smaller valve was cartilaginous, so that it could slightly recede and allow a slit for the passage of the blood from the heart. These valves had long bony bases extending some distance into the ventricle. The tricuspid foramen measured six inches in circumference; its valves were small, and totally incompetent to close the foramen. There was atheroma in all the great arteries. The other organs were healthy.

Case XIII.—Henry Wilson, æt. 66, gun-stocker: felt palpitations of the heart when in bed a year ago, which occasionally reappeared for six or seven months, and then ceased; great dyspnœa then came on. Had lost flesh for three weeks previous to my seeing him; and for three days had pain below the left breast. The veins of the neck pulsated most violently, and felt as firm and hard as arteries. The pulse was converted into a tremulous vibration. There was cough, with expectoration of much clear mucus, sometimes streaked with blood. The chest sounded dull about three inches around the heart. One coarse, hollow, and amazingly prolonged sound, was heard synchronous with the ventricular systole, and its maximum was to the right of the sternum. Cooing râles were heard over the chest. There was œdema of the ankles, and slight œdema of the legs. Five weeks after this he was carried off by an attack of acute bronchitis.

Autopsy.—The veins of the neck were very much distended with dark blood. The pericardium was opaque and thickened, and had on its cardiac portion a patch of soft yellow lymph as large as a crown piece. The heart was nearly three times the size of the fist, and very firm, from general hypertrophy and dilatation. The aortic valves were unfolded, grown together, and turned into bone, so that the passage for the blood was contracted into a small triangular opening which could barely admit a quill.* The aorta was studded with osseous scales. The arch was slightly dilated, and its middle coat easily torn. One of the tricuspid valves adhered to the right ventricle. The tricuspid foramen measured five inches in circumference. Other organs healthy.

Remarks on Cases XII. and XIII.—The extreme narrowing of the orifice of

* In the museum of King's College, London.

the aorta in Cases XII. and XIII. must have greatly tended to produce dilatation of the cavities of the heart, and it might be supposed hypertrophy of the left ventricle. While both hearts, however, were dilated, the walls of the left ventricle were hypertrophied in Case XIII. and attenuated in Case XII. Their history would lead us to conclude that the dilatation in Case XII. had been purely passive from obstructed circulation, while that in Case XIII. had been partly induced by pericarditis; traces of both ancient and recent attacks having been found on the pericardium.

From the nature of the morbid alterations of the aortic valves they must have been a long time in progress, and yet the health was not seriously affected until a comparatively short time before death. This is, in fact, explained by the prolonged sound heard during the systole of the ventricles, which proved that their contraction was continued much beyond the usual time, and that by this means the blood was gradually squeezed through the contracted orifice of the aorta, and was thus prevented from stagnating in the heart. But the circulation could not be forced on in this manner without injurious effects, and consequently in course of time dilatation of the heart situated behind the obstructed orifice of the aorta ensued, which was followed by congestion of the lungs and dyspnœa. The latter symptoms were doubtless much relieved by a copious secretion of mucus from the lining membrane of the bronchial tubes. Dilatation at length commenced on the right side of the heart, and the tricuspid foramen having been dilated to such a size that its valves could no longer close it, regurgitation, evidenced by jugular pulsations, ensued, and anasarca and death closed the scene. On this supposition the dilatation of the right side of the heart in Case XIII. must have been very rapid, as anasarca only appeared a little before death. It was probably hastened by the attack of bronchitis. A comparison of these cases offers a remarkable confirmation of the connection which exists between regurgitation and venous pulsations. In Case XIII. the heart was firm and hypertrophied, consequently the shock or pulsation was most vehement, but the dilatation of the tricuspid foramen being carried only to a moderate degree, the obstruction to the circulation was slight, and the dropsy inconsiderable.

In Case XII. the venous pulsations were slight, because the heart was attenuated, but the obstruction to the circulation was great, and the dropsy considerable, because a vast quantity of blood must have regurgitated through a widely dilated triscuspid foramen.

WILSON AND OGILVY, 57, SKINNER STREET, SNOWHILL, LONDON.