On the origin of anaemic murmurs / by James Kingston Fowler.

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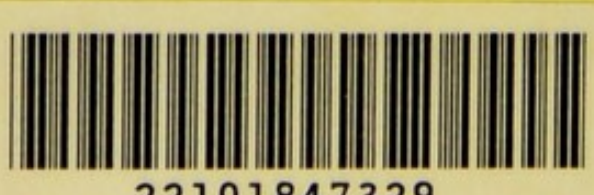
FOWLER



Lambard Owen Esq
with the author's kind regards

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BY

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RESPECTFULLY DEDICATED

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ON FUNCTIONAL DISORDERS OF THE HEART, AND FALSE MURMURS

A DESCRIPTIVE CATALOGUE OF THE PATHOLOGICAL MUSEUM OF THE MIDDLESEX HOSPITAL.

ON THE

ORIGIN OF ANÆMIC MURMURS

There is perhaps scarcely any point in Medicine on which more divergent opinions have been held by various authorities than as to the true interpretation of the murmurs audible in the heart and vessels of the subjects of anæmia.

I have ventured to re-open this discussion because the subject forms part of a larger one, to which I have for some time directed much attention, viz. the functional disorders of the heart and the conditions which may give rise to murmurs independent of disease of its valves.

I propose, after a brief reference to the altered composition of the blood in anæmia, to consider first the condition of the heart, and shall then, after discussing the origin of murmurs generally, pass on to the consideration of the mode of production and significance of the murmurs present in the anæmic state.

So far as regards the qualitative composition of

the blood is concerned I shall content myself with giving the two following tables quoted by Dr Coupland in his Gulstonian Lectures on "Anæmia" (1881). They refer, as will be seen, to chlorosis, which I agree with him in regarding not as a distinct disease, but as a variety of anæmia:

Table I

Mean chemical composition of blood

(Becquerel and Rodier.)

	In health.		In chlorosis.
	Adult male.	Adult female.	
Water . . .	780	791	828·2
Solids . . .	220	209	171·8
Fibrin . . .	2·2	2·2	3·4
Albumen . .	69·4	70·5	72·1
Corpuscles . .	140·0	127·2	86·0
Extractives and solids .	6·8	7·2	8·8
Fatty matters . .	1·6	1 9	1·5

Table II

Mean composition of blood (1000 *parts*)

	In health (Lecanu).	In chlorosis (Andral and Gavarret).	
		Incipient.	Fully developed.
Water . . .	790·0	801·0	852·2
Solids . . .	210·0	199·0	146·8
Fibrin . . .	3·0	3·5	2·9
Corpuscles . .	127·0	106·8	56·7
Solid residues of serum	80·0	88·0	88·0

It is impossible to speak with certainty as to the total volume of the blood in anæmia, but there can be little doubt that it is usually diminished. That it is so to a very considerable degree in pernicious

or idiopathic anæmia, and in anæmia following hæmorrhage, and in other varieties, is quite certain, and it is highly probable that a similar condition is present in the less advanced stages of anæmia.

Immermann* states that "the total volume of the blood is usually reduced in anæmia," and offers the following explanation of the deficiency:—"A certain portion of the water of the blood is fixed in and incorporated with the albuminates of the plasma in virtue of their colloid nature. It is thereby rendered in a certain sense permanent. The residual portion of the water is subject to great temporary variations in consequence of absorption from the stomach, &c. Hence the average volume of the total blood is primarily regulated by the proportion of albuminates in the plasma, and must therefore be reduced by the presence of absolute hypalbuminosis. Now, as most of the causes of anæmia induce hypalbuminosis as their primary and direct result, they must necessarily (apart from counteracting influences) tend to diminish the total volume of the blood. Moreover, the saline ingredients of the plasma stand in some ill-understood relation to the osmotic phenomena going on in the organism, and exert an influence upon the elimination of water through the skin and kidneys, and thus upon the volume of the blood." . . . "Now, it has been shown by C. Schmidt that the salts of

* 'Ziemssen's Encyclop.,' vol. xvi, Eng. trans., art. "Anæmia," p. 362.

the blood stand in a definite reciprocal relation to its albumen, one part of salts being taken up from the tissues for nine parts of albumen withdrawn from the blood. The connection between the volume of the blood and its centesimal composition thus assumes a more complex aspect, and cannot be explained by the degree of hypalbuminosis alone.

Finally, the elimination of water through the kidneys, lungs, or skin may be interfered with, or facilitated, in many cases of anæmia by local disease in these organs. Thus we see that no very simple answer can be given to the question concerning the way in which the total volume of the blood becomes diminished in the different forms of anæmia."

It appears, therefore, that the blood in anæmia is deficient, not only in its two most important elements, the albuminoid constituents of the plasma, and the hæmoglobin of the red corpuscles, but also in actual bulk, that is, the total volume of the fluid in the body is less than the normal amount. This impoverishment of the blood is felt in every tissue and organ of the body, and the sum total of the phenomena thus evoked constitutes the anæmic condition.

I shall here confine myself to a consideration of the symptoms connected with the circulatory organs.

Virchow,* as the result of the examination of cases of chlorosis, has put forward a theory which ascribes

* 'Sitzungsbericht der geburtshülflichen Gesellschaft,' Z. B., 1870, S. 17, ff.

that condition to an arrest of development of the vascular system. He has found, in these cases, an abnormal narrowness of the aorta and its branches, an irregularity in the mode of origin of the large trunks and also of the intercostal arteries. In association with these changes the heart may be either stunted in growth, dilated, or hypertrophied. The walls of the large vessels are described as much thinner than normal and the tunica intima as presenting areas of fatty degeneration.

Whilst acknowledging the importance of Professor Virchow's researches, we cannot, I think, at present admit that any constant malformation of the heart and vessels is present in all cases of chlorosis.

Those writers who regard chlorosis as a disease distinct from anæmia are compelled to admit that one of the conditions which most frequently precedes the development of either affection is the removal of a previously healthy young woman from the country districts to the confinement and strain of domestic work in a large town. We can hardly suppose that in all such cases there is a congenital narrowing of the aorta.

The general condition of the heart in anæmia of a mild type is one of functional weakness and dilatation, possibly unaccompanied by organic changes in its muscular tissue, although on this latter point we cannot speak with certainty, for it may well be that, even with only a moderate degree of anæmia, a beginning is made of those molecular changes which

are seen in their full development in the striated fatty degeneration of the heart's walls in the subjects of idiopathic anæmia; with the weakness and dilatation there is often found some degree of hypertrophy, possibly induced by excessive action, but never present to any marked extent owing to the deficient formative power of an enfeebled system.

Such a condition as that above described fully explains the prominent cardiac symptoms of anæmia, the breathlessness on exertion, attacks of palpitation, vertigo, syncope, and sense of oppression and readily-induced fatigue, all of which clearly point to a dilated and enfeebled heart.

The nervous centres and cardiac ganglia also suffer from the defective quantity and quality of the blood, and thus there is induced a condition of irritable weakness of the heart as shown by the attacks of palpitation, which often occur in anæmic subjects from emotional causes only and quite independent of exertion.

During these attacks, however produced, precordial pain and a sense of oppression are often present, symptoms which may be partly subjective, and due to a hyperæsthesia of the heart.

The physical signs to which these changes give rise are usually somewhat as follows: the apex beat is usually displaced, somewhat outwards, often as far as the mammary line or beyond it. The impulse may be feeble and diffused, but is sometimes strong and thrusting, evidence that hypertrophy is present.

The area of dulness may be slightly enlarged; this is usually noticeable in the region of the apex, where also a systolic thrill is very commonly present. On auscultation various murmurs may be heard, invariably systolic in time, differing much in loudness, but usually of low pitch, soft and blowing. In the veins at the root of the neck, usually most distinct on the right side and accompanied by a continuous thrill, the venous hum and bruit de diable are audible.

Before considering the significance of these sounds it will be necessary to discuss briefly the causes of murmurs in general.

Of the various theories which have been put forward in explanation of cardiac murmurs two require consideration.

The first and older theory attributes them to the friction of the blood against the rough surfaces of the valves, walls, or orifices of the heart, thereby throwing these parts into sonorous vibration.

A later theory ascribes them to vibrations which take place in the blood itself owing to friction of the particles of the blood against each other. These vibrations are produced by eddies or oscillations which originate during the transition of the stream from a narrower into a broader part of the vessel.

This latter theory eliminates the influence of tension and lateral pressure whilst it assigns great importance to the rapidity of the blood stream.

The friction theory, though still supported by some authorities, must, I think, be held to have been dis-

proved by the experiments of Corrigan, Neumann, and others.

Corrigan* in his well-known experiments demonstrated that when a stream of water is flowing through a piece of intestine, no sound, or a murmur exceedingly indistinct, is audible whilst the intestine is tense, but if any part be constricted so as to produce an alteration in the motion of the fluid, a very loud bruit is at once produced. This bruit is audible only on the distal side of the constriction.

F. Neumann in experiments on the motion of fluids in closed vessels has shown that the extreme peripheral layer merely moistens the walls and cannot therefore give rise to sound by its friction against the surface. It has also been shown by Weber, that if a stream of water be only sufficiently rapid, murmurs may even be produced in a glass tube of uniform diameter.

That the rapidity of the stream is an important factor in the production of murmurs is quite in accord with clinical experience, as we often find a sound which, when the heart is acting quietly, is only "murmurish" or prolonged, become a distinct bruit when the pulse-rate is accelerated by exertion or excitement; and conversely; it explains the absence of murmur in some cases of mitral stenosis when the left auricle is feeble, and its disappearance in the later stages of valvular disease when the power of the heart is failing.

* 'On the Mechanism of Bruit de Soufflet,' "Dub. Journal of Med. Sc.," 1836 and 1839.

The clue to the origin of murmurs lies in the fact brought out by Corrigan's experiments, that the sound is audible only on the distal side of the constriction; that is, where the blood is passing through a narrow into a wider part of the vessel.

Two conditions are necessary, then, for the production of a murmur—1st, a sufficiently rapid stream; 2nd, a narrow orifice, leading to a wider space beyond.

Dr Hayden refuses to accept the latter view on two grounds—first, that it necessarily implies a murmur of diastole at the auriculo-ventricular orifices in the normal state of the heart; and second, that it fails to explain the remarkable differences in the quality of murmurs.

As regards the first objection, Dr Hayden, I think, correctly rejects the statement of Corrigan that normally such a murmur is audible over these points, as no such sound can be heard.

It is absent, I believe, because the flow of blood from the auricles is at first not sufficiently rapid to produce one, even if the difference in size between the auriculo-ventricular orifice and the cavity of the ventricle at the beginning of diastole be sufficient to constitute the former a "narrow orifice." There is in addition the fact that the systole of the auricles, which is a graduated contraction, does not take place until after a part of their contents have passed into the ventricles. With regard to the second objection, that this theory is incapable of explaining

the different quality of murmurs, it is, I think, scarcely more tenable than the first.

There can be no doubt that the quality of these sounds is affected by certain conditions of the valves or orifices, although they are not the primary factors in their production.

Such conditions are a ruptured valve or tendinous cord, or a pendulous mass of fibrin, as such bodies, by projecting into the stream during or after its passage through the orifice, may, by interrupting the regularity of its oscillations, vary the quality of the murmur produced.

All murmurs, then, are hæmic or blood murmurs, and the restriction of that term to murmurs independent of organic disease, although hallowed by custom, is incorrect; they should be termed mitral or tricuspid direct, or regurgitant; or aortic or pulmonary systolic, with, if necessary, the prefix anæmic.

The conclusions, therefore, at which I arrive as to the causes of murmurs are—

1. That all murmurs are due to sonorous vibrations occurring in the blood-stream, the result of friction of the particles of the blood against each other.

2. That this friction is induced by eddies or oscillations, which originate during the passage of the stream through a narrower into a wider part of the vessel.

3. That murmurs are primarily independent of

the condition of tension of the structures forming the orifice, and are chiefly affected by the rapidity of the flow.

4. That the primary vibrations in the stream may be varied by certain conditions of the orifice and valves.

5. That these primary vibrations may produce secondary vibrations in the containing vessel and neighbouring parts.

From the time of Laennec to the present day, the site and mode of origin of the murmurs audible in the heart and large vessels in the subjects of anæmia has been a matter of dispute, and very probably will remain so to the end of the chapter. In order to show at a glance the great diversity of opinion which has prevailed, I have drawn up the following tables, giving a list of the principal writers on the subject and the views they have severally advocated.

Author.	Reference.	Site of origin.	Mode of production.	Remarks.
Laennec	Traité de l'Ausculte. Médiate, Paris, 1818	—	Spasmodic contraction of the muscular fibres of the heart or arteries	All murmurs referred to a common origin, *i. e.* spasm.
Andral	Notes au Jr. de l'Auscult. Méd. de Laennec, t. iii, p. 103, Paris, 1837	Aortic	Doubtful if due to spasm of orifices. Coincides with a diminution in the quantity of blood or of some of its principles	—
Hope	Treat. on Dis. of Heart, Lond., 1839	,,	Bruit and thrill produced by increased velocity of blood current and consequent increase of friction	Dilatation, with hypertrophy of left ventricle, caused by impoverishment of blood producing relaxation of all tissues, and especially of the heart.
Bouillaud	Journ. Clin. des Malad. du Cœur, Paris, 1841	,,	Increase in friction of the blood during its passage through orifices or cavities of the heart	—
Gendrin	Leçons sur les Malad. du Cœur, p. 115, Paris, 1841-42	,,	Change in condition of blood causes changes in its sonorous vibrations	—
Corrigan	Dublin Journ. of Med., vols. x and xiv	,,	A current-like motion of the blood, tending to produce corresponding vibrations in the sides of the cavities or arteries through which it is moving	A diminished tension of the parieties of the vessels facilitating vibration is an important element.
Leared	Dub. Quart. Journ., May, 1852	,,	Reciprocal pressure of particles of the blood resulting in secondary and irregular waves	—
Latham	Dis. of Heart, Dublin, 1853	,,	Abnormal vibrations of particles of the blood	—
Bellingham	Dis. of Heart, Dublin, 1853	,,	Vibration produced by diminished viscosity and increase in the watery element	—

Skoda	Journ. de Per. et Auscult. (trad. d'Aran, Paris, 1854, p. 270)	„	Friction between the blood and the walls of the organ; condition of the blood not a factor	Possibly by the vibrations of the walls excited by their distension. The connection between these factors doubtful.
Hughes	Guy's Hosp. Rep., ser. ii, vol. vii, 1854	Pulmonary and aortic	Two causes of basic bruit:—*α*. Sudden contraction of ventricles, and consequently increased rapidity of blood stream. *β*. Watery condition of blood producing easier movement in molecules and increased friction of particles *inter se*	—
Beau	Journ. d' Auscult., Paris, 1856	Aortic	Increased friction against walls by large blood wave of dilated ventricle	Dilatation, with hypertrophy of left ventricle, caused by impoverishment of blood, producing relaxation of all tissues, and especially of the heart.
Monneret	Journ. de Path. Gén., t. i, Paris, 1857; also Union Méd., 1849, p. 499	„	Diminution in number of red blood corpuscles	—
Bamberger	Lehrbuch.des Krank. des Herzens, Wien., 1857, p. 89 and 246	Pulmonary and aortic	In chlorosis a relaxation of the fibres of the ventricular walls follows on malnutrition	—
Austin Flint	Dis. of Heart, Phil., 1859	Aortic, pulmonary, and mitral	In palpitation of functional disease mitral regurgitation and murmur may be produced by spasm of the papillary muscles	—
Sterk	Arch. der Heilkunde, 1863, p. 47; Gaz. Hebdom., Paris, 1863, p. 262	Aortic and pulmonary, ? mitral	Relaxation of fibres of ventricular walls following malnutrition and causing a passive dilatation of cardiac cavities	Possibly when the bruit has its maximum intensity on a level with the mitral there is a relative insufficiency of that valve.
Marey	Phys. Méd. de la Circul. du Sang, Paris, 1863, ch. 24	Aortic	Diminished arterial tension and increased rapidity of the ventricular systole.	—

Author.	Reference.	Site of origin.	Mode of production.	Remarks.
Parrot	Arch. Gén. de Méd., ser. vi, t. viii, p. 129, 1866	Tricuspid	Dilatation of right ventricle and enlargement of tricuspid orifice	Bruit never audible to left of nipple. Importance of venous pulsations in neck.
Potain	Dict. Encyclop. des Sciences Médicales, 1866, t. iv, p. 392	Pulmonary and aortic, especially the latter	1. The condition of the ventricular walls 2. The condition of the blood 3. The condition of the circulation	—
Hayden	Brit. Med. Journ., 1867, Nov. 16 & 23, "Of Dynamic Murmur"	Mitral	Independent of dilatation, due to yielding of muscular walls during systole from atony causing musculi papillars to drag open the mitral valve	—
Hayden	Dis. of Heart and Aorta, Dublin, 1875, "Of Anæmic Murmur"	Aortic and pulmonary	1. Friction of blood corpuscles against one another and against edges of opening and walls of vessels 2. Vibration of heart and walls of vessels	Friction mainly due to atony of vascular walls and low tension from malnutrition of vaso-motor centres, vibration due to same cause.
Walshe	Dis. of Heart, 4th ed., 1873, p. 86	Anæmic murmurs: aortic and pulmonary. Dynamic murmurs: mitral and tricuspid	Altered composition of the blood *a.* Violent action, dilatation with hypertrophy *b.* Irregular contraction of musculi papillares	Audible at second left cartilage, nearly below nipple, never at apex.
Balfour	Clin. Lect. on Dis. of Heart, 2nd ed., 1882	Mitral, propogated to auricular appendix	Mitral regurgitation	—
Russell	Trans. of Med.-Chi. Soc. of Edin., vol. i, 1882	Pressure on pulmonary artery by distended auricle	Dilatation of left auricle from imperfect emptying and pressure of blood into it from surcharged pulmonary veins	—

It is unnecessary and would be wearisome to discuss in detail these various theories. I shall therefore proceed to enunciate some propositions which are at the present day admitted by all authorities, and shall then consider some points on which a divergence of opinion still exists.

1. A systolic murmur is usually heard in the second intercostal space on the left side. This may be the only one present.

2. The second sound of the heart may be accentuated in the pulmonary area.

3. A systolic murmur is occasionally audible in the aortic area, and is conducted upwards in the course of the aortic stream.

4. A systolic murmur may be present at the apex in the mitral area.

5. A systolic murmur may sometimes be heard internal to the apex in the tricuspid area.

6. Murmurs are often audible in the veins at the root of the neck, usually most distinctly on the right side; these are either continuous or intermitting.

Let us consider first the murmur in the second left interspace, around which the great controversy as to the genesis of the anæmic murmurs has raged.

This sound varies much in intensity, it may be soft and blowing or harsh and almost grating in character; it coincides almost exactly with the commencement of ventricular systole and gives the idea that it is produced in a quickly flowing stream,

just beneath the end of the stethoscope. It is not distinctly conducted in any direction.

According to my experience it is by far the most common of the anæmic murmurs, an opinion which is supported by Dr Balfour,* who states that "the primary or typical murmur is basic in position."

The point of maximum intensity of this bruit is a question of some interest. Dr Balfour,† following Naunyn, is of opinion that it is most distinctly heard "about one inch and a half, or more to the left edge of the sternum in the second interspace."

I have made some observations on the exact position of the pulmonary artery in subjects who have not died from any disease of the respiratory organs or heart, and find that not uncommonly the vessel is only just missed by needles inserted one and a quarter inch from the sternum in the second interspace.

Dr Russell ‡ has described the results of three observations upon cases of pernicious anæmia, in which great care was taken to ascertain the exact position of the pulmonary artery and conus arteriosus.

As these observations are interesting and to the point I append brief abstracts of them.

Case 1.—The conus arteriosus occupied the second

* Clin. Lect. 'Dis. of Heart,' 2nd edit., p. 172.

† Op., p. 171.

‡ 'British Medical Journal' of June 2nd, 1883.

left interspace, and the origin of the pulmonary artery was under the second rib. The left auricular appendix was invisible, and its tip was only revealed by drawing aside the conus arteriosus and adjoining artery.

Case 2.—The conus arteriosus occupied the second left interspace for about an inch and a half and extended upwards under the second rib, under which rib also lay the origin of the pulmonary artery. The left appendix was invisible and could only be seen by turning the heart round and removing about one ounce of fluid from the pericardium.

Case 3.—The origin of the pulmonary artery was under the second rib. The conus arteriosus occupied the second left interspace for fully two inches. The left appendix was not visible, and the heart had to be turned up to bring it into view.

The importance of these observations as regards the auricular appendix will appear presently.

It seems, therefore, that in the most extreme degree of anæmia the pulmonary artery and conus arteriosus may occupy the second left interspace for a distance of from one and a half to two inches.

The point of maximum intensity of the basic bruit when it alone exists as the cardiac sign of anæmia, is, according to my experience, over the area corresponding to the first inch of the second left interspace and occasionally the first inch and a half;

when, however, a distinct bruit is present at the apex, in the mitral area, a systolic murmur is at times audible in the second left interspace as far as two or even two and a half inches from the sternum, but is of an entirely different character to the primary bruit and is due to the conduction upwards of the murmur of mitral regurgitation. It is never, so far as I have observed, most distinct at that spot when there is no evidence of mitral reflux.

Where then is this primary murmur produced? Dr Balfour* says "in the appendix of the left auricle where it pops up from behind just to the left of the pulmonary artery."

According to Naunyn's† theory, which is supported by Dr Balfour, reflux through the mitral orifice secondary to dilatation of the left ventricle present is in all cases of anæmia presenting the primary basic bruit, and the pulmonary or rather auricular murmur is "often the only sign of mitral regurgitation."

"Naunyn explains this phenomenon by attributing it to the better conduction of the murmur along the course of the regurgitating blood, the fluid veins producing sonorous vibrations louder at the point of impingement than at the point of origin, to the circumstance that in all such cases the auricle is closer than usual to the anterior surface of the heart and therefore nearer to the ear."

* Op. cit., p. 171.

† 'Berliner Klinische Wochenschrift,' 1868, No. 17, S. 189.

Again Dr Balfour says,* "the fluid veins formed in the early stage of chlorotic regurgitation are of low tension and but little force, hence the vibrations they originate are but slightly propagated to the left ventricle and only with difficulty from it to the chest wall in the mitral area, where they are heard as an impure first sound. But, on the other hand, these vibrations are readily communicated to the wall of the auricle on which they impinge and are easily transmitted to the chest wall with which the auricular appendix is in contact, becoming audible in the auricular area as a distinct murmur." I am unable to see why there should be any difference between the mode of propagation of the mitral regurgitant murmur of anæmia and of organic disease.

The so-called fluid veins in the latter must be often "of low tension and but little force," yet it cannot be contended that a systolic murmur in the second interspace near the sternum is a common physical sign of mitral reflux of organic origin, and one could without difficulty bring forward almost any number of cases of organic mitral disease where no murmur at all is present in any part of the second interspace.

Another point which I have not seen noticed is that when from disease of the mitral curtains slight reflux has been permitted, the course of this stream, which can often be detected by the roughening of

* Op. cit., p. 176.

the endocardium of the auricle, is seen to be directed backwards and to the left toward the superior pulmonary vein—that is in a direction exactly opposite to that of the auricular appendix, and on testing the valve with water the reflux is seen to follow the same course.

There is, so far as I can find, absolutely no positive evidence in favour of the view that "in these cases the auricle is closer than usual to the anterior surface of the chest," or that "the auricular appendix is in contact with the chest wall." The latter view is disproved by Dr. Russell's observations, and my own experience of the condition of the heart in cases of pernicious anæmia is entirely in accord with his. In such cases on opening the pericardial sac the appendix of the left auricle is rarely, it might be said, never visible, but is found lying deeply placed behind the conus arteriosus. If Naunyn's theory be true of the mild cases it should *à fortiori* hold good in the most extreme examples of anæmia.

The only condition which I have found to produce a degree of distension of the auricular appendix sufficient to bring it into contact with the chest wall in the second interspace is extreme stenosis of the mitral orifice, and under these circumstances it is usually filled by a thrombus. I doubt also whether in chlorotic regurgitation the volume of the stream is sufficient to produce distension of the appendix; if it were so, we should probably have more distinct evidence of the increased tension in the pulmonary

vessels than is afforded by the accentuated second sound, in spite of the diminished volume of the blood.

The only sign suggesting that the appendix is in contact with the chest wall is that a systolic impulse is occasionally seen in the second interspace. This is, I believe, due, as Dr Russell has shown, to the pulsation of the dilated conus arteriosus.

Lastly, the character of the murmur prevents our believing that it is due to vibrations suddenly arrested by impingement against a surface, it suggests so unmistakably that it is produced in a rapidly flowing stream.

When discussing the order of disappearance of the bruits in cases undergoing cure, I shall offer some further objections to the acceptance of the view that this murmur is produced in the auricular appendix.

Dr Russell * has advanced another explanation of this murmur : that, owing to the dilatation of the left auricle and the instantaneousness of its diastolic fulness, due to its imperfect emptying into the ventricle on the one hand, and, on the other hand, from the volume of blood pressing into it from the surcharged pulmonary veins, the pulmonary artery is, during ventricular systole, compressed by it ; and that this is the cause of the murmur heard in the vessel.

It appears to me that all the conditions necessary for the production of the murmur in the manner

* 'Ed. Med. Journal,' Aug., 1882.

here described are present in a far greater degree in cases of mitral stenosis with regurgitation, when it is, in my experience, quite unusual for any bruit to be present in the pulmonary artery.

It is unnecessary to discuss Parrot's * view that this murmur is invariably due to tricuspid regurgitation; that condition is no doubt present in advanced degrees of anæmia, but it is not the primary change.

The theory which to my mind most satisfactorily explains the mode of production of this murmur, and also that heard in the veins of the neck, is the one first advocated by Chauveau,† and may be stated thus :—"In anæmia there is a general reduction in the volume of the blood ; the blood-vessels generally with two exceptions adapt their diameter to the reduced volume. The exceptions are (1) the aorta and pulmonary artery, which, owing to the absence of the contractile and the preponderance of the elastic element in their walls, cannot reduce their diameter proportionately to that of the current passing through them ; (2) the roots of the innominate veins are fixed and kept permanently dilated by the cervical fascia, which not only ensheaths them but is connected with the sternum, clavicle, and first rib. Hence, whilst the jugular and subclavian veins above accommodate themselves to the reduced dia-

* 'Archiv Gén. de Médécine,' 6me serie, tom. viii, 1866, vol. ii, p. 158.

† 'Gaz. méd. de Paris,' 1855.

meter of their respective currents, the commencing portion of the innominate vein is incapable of a reduction of calibre and becomes relatively dilated." The blood-stream, therefore, at these points passes through a narrow orifice into a portion of the vessel having a wider calibre, which, as we have seen, is one of the conditions necessary for the production of a murmur. There is an interesting confirmation of the truth of this theory in the fact that a similar bruit to that audible in the innominate veins may occasionally be heard over the cerebral sinuses at the Torcular Herophili where the same conditions as to non-contractility are present, and also in the fact which I have observed, that in anæmic subjects who suffer from deafness not dependent on disease of the auditory nerve and in whom the conduction of the skull vibrations is normal, a similar sound becomes audible on the affected side. This is, no doubt, the bruit produced in the lateral sinus conveyed to the ear through the medium of the temporal bone.

There is one point for which this theory does not account, and of which no explanation has, so far as I am aware, been offered, viz. the appearance of the bruit in the pulmonary artery earlier than in the aorta. Marey claims to have demonstrated that the right ventricle contracts in a manner somewhat different to the left, its systole being more sudden and having relatively a greater initial force. If this be so the blood stream in the pulmonary artery must

have a greater initial velocity than in the aorta; and as the rapidity of the current is a most important factor in the production of a murmur, it may be that this difference in the initial velocity determines the appearance of the bruit first in the pulmonary artery.

I now pass on to consider the murmurs audible at the apex in the mitral and tricuspid areas. These are systolic in time, of medium pitch, usually soft and blowing in character, and dependent on regurgitation through the auriculo-ventricular orifices.

The exact mode in which this reflux is brought about has been a matter of much speculation. Hayden* refers it to a perverted action of the musculi papillares, which are normally "so placed that, in a state of complete ventricular systole, they interlock or pass to opposite sides of the chamber. In virtue of this interchange of position they so act upon the segments of the mitral valve that the lines or axes of their contraction decussate at an acute angle and the valves are drawn each towards the opposite side of the ventricle.

"If from any cause the musculi papillares are prevented from performing the movement of transposition already indicated they will act upon the valves not in the direction of the opposite wall of the ventricle, but towards that from which they take their origin, and will therefore divaricate the valves and permit regurgitation."

* 'Brit. Med. Journ.,' 1867, Nov. 16, p. 23.

Dr Walshe, who divides murmurs independent of organic disease into two classes, hæmic and dynamic, scarcely admits the existence of this class of anæmic murmur. Of the former he says: "This murmur is, as far as I have observed, invariably basic in seat and systolic in time, produced at the orifices of the aorta and pulmonary artery . . . only in exceptional cases audible below the nipple, and never within my experience perceptible as far as the left apex."* In a note, however,† he says he has been misunderstood as saying that a systolic murmur is never audible at the apex as a result of anæmia, as he believes that a dynamic murmur may be produced under such conditions. We shall, however, not be doing injustice to his opinions by representing him as regarding it as a rare condition, for he says:‡ "I have never yet heard in a purely chlorotic woman a murmur having all the characters of a mitral regurgitant one." To this I shall recur later. Dr Walshe, in speaking of the dynamic systolic murmur, says: "Irregular and occasional reflux takes place at the mitral orifice through disordered action of the muscular apparatus connected with the valve."

The very important researches of Hesse,§ of Leipzig, made in the laboratory of Professor Ludwig, of which an able account has been given by

* 'Dis. of Heart,' 4th edit., 1873, p. 86.

† Op. cit., p. 89, 4th edit. ‡ Loc. cit.

§ "Beitrage zur Mechanik. der Herzbewegung," 'Archiv für Anatomie und Physiologie,' 1880, p. 328.

Dr MacAlister, of Cambridge,* have thrown much light on this as on other questions connected with the mechanism of the heart's contractions.

Dr Hesse, by very ingenious means, has contrived to obtain casts of the heart and its cavities, representing as nearly as possible its exact condition in systole and diastole. From an examination of these it appears that during systole the apices of the papillary muscles approach the aortic orifice, not, however, by any upward motion, but by a movement in a plane parallel with that of the ostia venosa. The result of this movement is to allow the anterior curtain of the mitral valve, which is during diastole stretched obliquely across the cavity, to be floated up so that it may assist in closing the mitral orifice, the papillary muscles at the same time contracting in order to prevent the valve from being driven too far into the auricle.

Hesse has also shown what a very important part the contraction of the muscular tissue of the base plays in producing the closure of the auriculo-ventricular orifices.

The average of three observations on different hearts showed that the circumference of the base during diastole was 65 cm., and during systole 36 cm., or a mean difference of 25 cm.; the ratio of the circumference of the base during diastole to that during systole being therefore as 13 : 7·2, or a reduction of nearly one half.

* 'Brit. Med. Journal,' Oct. 28, 1882.

Now, in anæmia the muscular tissue of the ventricles is feeble if it be not in a state of commencing fatty degeneration, the latter hypothesis having been rendered much more probable by the discovery that retinal hæmorrhage, the consequence of disease of the vessels, is a common occurrence when the quantity of hæmoglobin present is less than 50 per cent. of the normal. The ventricles are dilated, and the cordæ tendineæ consequently stretched, and the auriculo-ventricular orifices probably enlarged; when systole begins instead of a reduction in circumference of the base of nearly one half, there is probably only a feeble contraction, and this, added to the increased initial tension of the tendinous cords, which prevents the valves from being immediately floated upward, allows of reflux into the auricle, the result being a systolic murmur at the mitral or tricuspid apex, or at both points if regurgitation occurs on both sides. It is interesting to note that, as in pernicious anæmia the left ventricle is first affected with fatty degeneration, so in ordinary anæmia the bruit at the mitral orifice precedes that at the tricuspid, a further argument in favour of the presence of that change in cases of chlorotic anæmia. The line of conduction of the systolic apex murmur of anæmia is a matter of great importance, and to which I wish to draw particular attention.

I have already referred to the statement of Dr Walshe that he has "never yet heard in a purely

chlorotic woman, a murmur having all the characters of a mitral regurgitant one." Dr Walshe thus defines those characters : "A systolic murmur of maximum force at, and immediately above, or to the outside of the left apex, but faintly audible, or wholly inaudible, at the right apex (say the ensiform cartilage), the mid-sternal base, and the pulmonary or aortic cartilages—more or less clearly audible about and within the inferior angle of the left scapula, and in the left vertebral groove from the 6th to the 9th dorsal vertebra ; audible or not round the lateral base of the chest from the cardiac to the scapular region—is essentially characteristic of regurgitation through the mitral orifice at the moment of ventricular systole."

"A murmur possessing all the characters just enumerated may lay claim to indicating, with well-nigh absolute surety, the existence of *mitral reflux of organic mechanism*." "To have such significance must it actually possess the entire group ? This is still to a certain degree a moot question in regard to one of the most important of the whole series, *audibleness within the inferior angle of the scapula*." . . .

"If the murmur be imperceptible posteriorly, I believe it *very seldom depends on regurgitation of structural mechanism*, possibly it may never do so, but I am not prepared to adopt so sweeping a formula. Still, I think we may say it *never* depends on *well-pronounced organic regurgitation*. On the

other hand systolic apex murmur *inaudible posteriorly* is the exponent of dynamic mitral reflux."

Dr Walshe classifies the systolic apex murmur thus:

Audible in left ventricular groove.	Mitral orifice.	Structural insufficiency of valve.	Reflux through orifice, often with roughened edges.
Inaudible in left ventricular groove.	Mitral orifice.	Dynamic interference with closure of valve.	Reflux through orifice in itself smooth and natural.

Hayden is opposed to Walshe as to the absolute diagnostic value of the conduction of the murmur to the angle of the scapula, but agrees with him that this sign is absent in cases of functional mitral reflux; he states*: "I have repeatedly found indubitable mitral regurgitant murmur of organic origin totally inaudible at the left scapula and unaccompanied by intensified second sound." . . . "As above shown these negative signs are characteristic of functional mitral murmur."

I have quoted these authorities at length because I wished to make their views quite clear on a matter which is of great practical importance. Is every patient presenting the signs of mitral regurgitation, a systolic apex murmur conducted to the angle of

* 'Diseases of Heart and Aorta,' p. 289.

the scapula and audible in the vertebral groove between the sixth and ninth dorsal vertebræ, to be considered the subject of organic disease of the mitral valve? According to Dr Hayden and Dr Walshe this question must be answered in the affirmative. My own experience points to an exactly opposite conclusion. I have within the last three months seen at least fifteen cases of advanced chlorotic anæmia among my out-patients at the Middlesex and Brompton Hospitals, of whose cases I have careful notes, and in whom I have detected a systolic apex murmur, which has been distinctly audible not at the angle of the left scapula only but in many at the right also, and in most of which cases the bruits have already disappeared under appropriate treatment. I have frequently demonstrated the presence of these bruits to the students, who have rarely failed to distinguish them. I have long taught that the anæmic murmurs obey the same laws as to conduction as those of organic origin, and particularly that the conduction of the systolic apex murmur to the angle of the scapula is no sign of organic disease of the mitral orifice.

To establish this latter point I may quote very brief abstracts of the five following cases of chlorotic anæmia which have been recently under my care, premising that if necessary their number might be largely increased.

Case 1.—Victoria B—, aged 16, dressmaker,

attended as out-patient at Brompton Hospital, November 21st, extremely anæmic; health has been failing for two months, complains of breathlessness on exertion and palpitation. Thrill and bruit de diable at root of neck on both sides; apex beat in fifth interspace just internal to mammary line. No pulsation in second interspace. No pulsation of vessels of neck. Loud systolic bruit in second left interspace most distinct just to left of sternum and audible also in aortic area, where it is slightly conducted upwards. Soft blowing systolic murmur at apex conducted through axilla to angle of left scapula and of right also. Ordered—

℞ Ferri Sulph. exsicc., ʒss;
Potassæ Carb., ʒss;
Tragacanth, q. s.
Ft. pil. 96. Q. s. pil. ij ter in die.

December 12th.—Colour remarkably improved; no bruit anywhere; first sound at apex very slightly prolonged. Repeat—

Pil. Ferri Carb., gr. v.
Pil. iij ter in die.

January 2nd.—No bruit; first sound normal.

Case 2.—Annie B—, aged 18, no occupation; was an in-patient of the Middlesex Hospital for anæmia from September to January, 1881; attended again as an out-patient November 30th, 1883. Extreme anæmia, dyspnœa on exertion; palpitation on exertion, on lying down at night, during the night, and at

other times, often quite independent of movement. Cardiac apex in mammary line, area of dulness slightly increased; impulse thrusting; no pulsation in second interspace; pulse 114, small, short, and sharp. Bruit de diable and thrill on both sides of the neck. In the second interspace on the left side there is a blowing systolic murmur, which is also audible in the aortic area and conducted upwards. At the apex a soft blowing murmur is audible, this can be traced round the axilla to the angle of the left scapula and also to that of the right. There is also a faint bruit audible at the base of the ensiform cartilage. Ordered—

℞ Ferri Sulph. exsicc., ℥ss;
Potassæ Carb., ℥ss;
Tragacanth, q. s.
Ft. pil. 96. Q. s. pil. ij ter in die.

December 7th.—Colour slightly improved; no bruit at angle of right scapula; faint systolic murmur audible at angle of left scapula.

January 4th.—Bruit at base only; no bruit at apex or at angle of scapula.

18th.—Colour improved; soft bruit still remains in second interspace.

Case 3.—Clara T—, aged 18, dressmaker, attended as out-patient at the Brompton Hospital, October 27th, 1883. No history of rheumatism. An extremely anæmic girl, complaining of palpitation, headache, and dyspnœa on exertion, and amen-

orrhœa. Cardiac dulness normal, apex in mammary line; venous hum in neck; soft systolic pulmonary bruit. Distinct systolic murmur at apex which can be traced through the axilla to the angle of the left scapula. Not audible at angle of right scapula. Ordered—

℞ Tr. Digit., ♏v;
Liq. Ferri Perch., ♏xx;
Æth. Chlor., ♏x;
Infus. Quass., ℥j ter.
Pil. Aloes et Myrrhæ, gr. v.

November 10th.—Colour much improved, bruit disappeared from angle of scapula, first sound at apex prolonged, no bruit in pulmonary area, venous hum has disappeared. Ceased to attend.

Case 4.—Eliza T—, aged 17, domestic, attended as out-patient at Brompton Hospital, November 21st, 1883. No rheumatic history. Complains of headache, palpitation, and dyspnœa. Patient is extremely anæmic; the thyroid is rather larger than normal. Pulse 96, small, short, and sharp. The cardiac dulness is slightly increased, the apex beats in the fifth interspace in the mammary line. No pulsation in the second left interspace, where a loud systolic murmur is audible; the bruit is also distinct in the aortic area, and is from that point conducted slightly upwards. At the apex there is a blowing systolic murmur which can be heard plainly at the angle of each scapula. Ordered—

℞ Ferri Sulph., ℥ss;
Pot. Carb., ℥ss;
Tragacanth, q. s.
Ft. pil. 96. Q. s. pil. ij ter in die.

November 28th.—Colour improving, very faint bruit at the angle of the left scapula, none at the right.

December 12th.—No bruit de diable, no bruit in the pulmonary area. A very faint murmurish first sound at the apex. No bruit at angle of either scapula.

Case 5.—Hannah B—, aged 19, upholstress, attended as out-patient at the Middlesex Hospital, December 28th, 1883. No previous illness. No history of rheumatism; catamenia irregular; palpitation and dyspnœa on exertion. An extremely anæmic girl. The cardiac apex is in the fifth interspace one inch inside the nipple line. Venous hum and thrill in neck. Distinct systolic murmur in second left interspace and also in aortic area. Soft blowing systolic murmur at apex distinctly audible at the angle of the left scapula. Ordered—

℞ Pil. Ferri Carb., gr. v.
Pil. ij ter in die.

January 4th.—Rep. pil. Colour improved, bruits same.

17th.—No bruit at angle of scapula or at base. At the apex there is still a very faint bruit; the venous hum is also just audible.

The order in which these murmurs make their appearance varies somewhat in different cases, but, so far as I have observed, is usually as follows:— The bruit in the second left interspace appears first. To this, as the anæmia progresses, succeeds the venous hum in the veins of the neck. In a more advanced stage of the disease the apex murmur becomes audible, and is at first confined to that spot, but subsequently may be detected in the axilla, and is heard at the angle of the left scapula. Should the anæmic condition become still more pronounced the murmur of tricuspid regurgitation becomes audible at the base of the ensiform cartilage, accompanied by a venous pulse in the veins of the neck; the systolic basic murmur is now usually audible in the aortic area, and is conducted upwards in the course of that vessel. When the apex murmur is very loud it may be heard not only at the angle of the left scapula, but at that of the right also. I have also heard the pulmonary bruit distinctly in the interscapular region on a level with the fourth dorsal spine.

The order of extinction of the murmurs as the anæmia gives way to treatment is also somewhat variable, but is usually as follows:—The mitral murmur ceases to be audible at the angles of the scapulæ, leaving the right side before the left. The basic bruit can now perhaps be detected only in the pulmonary area, and the apex murmur, though present, is less distinct, and is confined to the

mitral area, where in some cases it may be heard after the pulmonary bruit has disappeared. In other cases, however, I have found the pulmonary murmur persist longer than that at the apex. The venous hum I have also noted in many cases as absent when the apex murmur is still perceptible. It is difficult to understand how, on the hypothesis that the basic murmur is the result of mitral reflux, that bruit can disappear whilst the cause of its production is still operative.

The fact that mitral regurgitation to a slight degree may continue after the other cardiac signs of anæmia have disappeared is interesting, and seems to show that by the simple process of osmosis the volume of the blood is sooner restored to the normal point than the heart can recover from its dilatation, and possibly fatty degeneration. This is quite in accord with clinical experience, for after these murmurs have entirely disappeared the patient is for some time incapable of any considerable exertion, and is very liable to a return of the symptoms unless the treatment be continued for some time longer.

I have not been able to find a record of any attempts, though doubtless such have been made, to trace a direct connection between the degree of anæmia and the murmurs, assuming as I do that they represent in the order of their appearance progressive stages of the disease.

The few observations which I here record, and

which I hope to extend very considerably, have already yielded some result in a hypothesis which, though it may be ultimately disproved, yet has some working value. They have been made upon patients in the Middlesex Hospital under the care of my colleague Dr Sidney Coupland, to whom I am indebted for permission to use the results; and I would also express my thanks to Mr Stace, Dr Coupland's house physician, for his kindness in undertaking the numerations.

Date of observation.	Sex.	Age.	Degree of anæmia.	Numeration of corpuscles per c.mm.	Percentage of corpuscles.	Percentage of hæmoglobin.	Murmurs present at time of observation.
1884.							
Case 1.							
Jan. 2	F.	23	Extreme	Red 294,000 White 20,000	58·8	28	Venous hum; systolic pulmonary; systolic mitral.
„ 16	...	...	Unchanged	Red 3,930,000 White 40,000	78·6	42	All murmurs present, but much less distinct.
Case 2.							
Dec. 27	F.	17	Extreme	Red 3,440,000 White 9,000	68·8	50	Venous hum; systolic pulmonary; first sound at apex prolonged.
Jan. 7	...	...	Less marked	Red 4,320,000 White 10,000	86·4	60	Same.
„ 16	...	...	Less	Red 4,550,000 White 20,000	91·0	82	Pulmonary bruit present; other murmurs gone.
„ 18	...	...	...	...	...	...	Murmur gone.
Case 3.	F.	22	Extreme	Red 3,840,000 White 40,000	76·8	34	Systolic pulmonary; faint venous hum.
Case 4.							
Dec. 5	F.	17	Slight	Red 3,860,000 White 18,200	77·2	...	Venous hum; systolic pulmonary; ? systolic aortic; systolic mitral.
„ 12	...	...	Same	Red 2,850,000 White 17,800	57·0	45	Same.
Case 5.							
Jan. 5	F.	20	Extreme	Red 4,970,000 White 50,000	99·4	36	Systolic pulmonary.
„ 17	...	...	Less	Red 3,710,000 White 30,000	74·2	30	Same.
„ 22	...	...	...	...	...	...	Murmurs gone.

The foregoing observations are somewhat contradictory, but appear to show that a murmur at the pulmonary orifice may signify a loss in hæmoglobin of 20 per cent. and in corpuscular richness of 9 per cent.; whilst a bruit indicating mitral regurgitation implies a loss of at least 55 per cent. of hæmoglobin and 43 per cent. of corpuscles.

It will be seen, however, that in Case 5 a loss of 70 per cent. of hæmoglobin failed to produce an apex murmur. It is clear, therefore, that other factors are at work besides the loss of hæmoglobin.

If, now, we consider the corpuscular richness a loss of ·6 per cent. appears to produce a systolic pulmonary murmur; and for a murmur at the mitral orifice a loss of 22·8 appears necessary.

If, however, the two sources of blood change be considered together it seems that with a loss of 64 per cent. of hæmoglobin, but of ·6 per cent. of corpuscles only a pulmonary murmur is heard, yet whilst the loss of hæmoglobin is reduced to 58 per cent. a mitral bruit may appear if at the same time there be a diminution in corpuscular richness to the extent of 21·4 per cent.

From this it would appear that the loss in corpuscular richness has more effect in producing the changes in the heart's sounds than that of the hæmoglobin. But further observations are needed before any definite statement is possible.

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