principle of the brace should be adopted. The idea was that the old form of accoutrement restricted the heart's action. The committee described the condition of irritable heart, which it seems to have recognized clearly as "an extreme excitability of the heart, combined with some, but not great, enlargement. During rest a heart of this kind beats easily, but on the least exertion its action becomes irregular, and the man becomes breathless." The recommendation of the committee regarding the change of accoutrement was later on adopted.

The American Civil War meanwhile was furnishing a large number of cases of "soldier's heart," and in 1864 Henry Harthorne published a paper in the *American Journal of Medical Science* which, I venture to say, furnishes an exact picture of the condition as Sir James Mackenzie has described it to-day. Dr. Harthorne laid stress on the rapidity of the pulse, on the acceleration of the heart's movements on the slightest exertion, on the shortness of breath, and on the cardiac muscular weakness. He suggested that in the soldier the heart was injuriously affected by long-continued over-exertion with deficiency of rest and often of nourishment, and he pointed out that several months of rest and treatment in hospital failed to do more than improve without really curing a large proportion of well-marked cases.

Two causes had thus been suggested so far back as 1864—tight accoutrements and over-exertion. The tight accoutrements were done away with, but the irritable heart remained. I pass over here the contribution to the subject made by Dr. Arthur Myers, of the Coldstream Guards, in 1870, because his description is similar to that already given, and because he also regarded accoutrements as the exciting cause of the trouble. But I must call attention specially to the paper of Da Costa published in 1871 in the *American Journal of Medical Science*. It is the best known contribution to the subject. Da Costa saw upwards of 300 cases in a hospital in Philadelphia, and he affords the following striking picture:

A man, three months or so on active service, was seized with diarrhoea, annoying yet not severe, soon rejoined, and then noticed that he could not bear exertion as well as formerly. Out of breath and unable to keep up with his comrades; dizziness and palpitation; accoutrements oppressed him, yet otherwise he seemed well and healthy. Sought advice; was sent to hospital, where his persistent quick heart confirmed his story. Digestive disturbances, if present, passed away, but the irritable condition of the heart remained, and only very slowly did this get normal or it failed to do so.

Da Costa then proceeded to paint the same picture as that which has been shown to-day by Sir James Mackenzie. He mentioned, however, that he had noted that intercurrent attacks of fever caused the pulse to become less and not more irritable, and to slow down while the attacks lasted.

In 1876 a new phase of the subject was presented when, the change in form of accoutrement having failed signally to effect any improvement, Surgeon Arthur Davy suggested that what he called "setting up" drill was the cause. His theory was ingenious. Setting-up drill, he said, by over-expanding the chest, caused dilatation of the heart and so induced irritability. This view found support in 1880 from Brigade-Surgeon Veale, and again in 1896 from Surgeon-Captain J. B. Wilson, but the discontinuance of setting-up drill does not seem to have put an end to the condition. On the contrary, I have met it in cases in which this cause could be absolutely excluded, the patients having undergone little or no drill.

The theorists, therefore, are bankrupt; the disease remains. More recently we have been asked to view it from the bacteriological standpoint, and more recently still as a condition depending upon faulty action or excessive action of the thyroid gland. I am not in a position to discuss these views, but it is interesting to note in this latter connexion that I have observed that in some eighteen of my cases the thyroid gland was definitely enlarged, and that administration of thyroid extract seemed to exacerbate the symptoms. The application of *x* rays to the thyroid gland is said to have effected improvement in some cases. Let me say, however, that I have seen cases presenting very much the same clinical picture in which the administration of *x* rays did not help matters at all.

In view of the dangers which have attended the making of theories to explain this disease in the past, I am

strongly of opinion that we must preserve an open mind. There would appear to be but one sound method in regard to the condition—the method of prolonged observation and continuous study. Many tempting views suggest themselves, but few stand the test of further investigation.

On the other hand, a few facts seem to have emerged. One of these is that a cheerful atmosphere confers benefit, and a depressing atmosphere exaggerates the condition. The patients are always better when their minds are enlisted in some more or less congenial occupation.

A NOTE ON CERTAIN OF THE VASCULAR PHENOMENA OF CARDIAC DISEASE.

BY

BASIL PARSONS-SMITH, M.D.

THAT considerable importance should invariably be attached to a complete investigation of the blood vessels in all cases of heart disease has long been maintained, and, particularly from the point of view of prognosis, with obvious advantage, for the circulatory mechanism, in its dual constitution of heart and blood vessels, needs a careful and complete study if anything approaching accuracy in the forecast is to be obtained.

Whilst, therefore, every cardiac case that comes before us must undergo a minute examination of the heart itself, its size, rhythm, sounds, etc., our responsibilities do not stop there: we must go further and undertake a rather more generalized study of each particular patient, inquire into the condition of the kidneys and their excretive powers, form some opinion as to the condition of the arterial walls, estimate the blood pressure and pulse tension, and glean additional information from various of the graphic tracings taken from the large superficial vessels, from electrocardiographic records, and by the method of direct vessel auscultation.

The strides which have been made in these latter methods during the past few years are very extensive and widespread in their application; they render possible a diagnostic accuracy hitherto unknown, at the same time facilitating treatment and firmly establishing it upon a sound scientific basis.

With regard more especially to the particular physical signs evinced by the vessels a mass of interesting data is forthcoming; for the purposes of the present communication it is proposed shortly to consider the great venous and arterial channels at the root of the neck and detail a selection of the causes which warrant their very careful study.

In the first place we may conveniently discuss the value of simple inspection, and by so doing formulate a working comparison between the healthy subject and the victim of disease. Reference to the former need but be brief, for the normal neck presents remarkably few noteworthy features; careful inspection discloses results of a singularly negative character; arterial pulsation cannot be made out, and it is only on looking carefully that one can appreciate some slight amount of venous movement, either produced by the external jugular vein, which pursues a superficial course for the majority of its length—in fact, to the root of the neck, where, immediately above the mid-clavicular point, it perforates the deep fascia to join the subclavian vein—or by the internal jugular vein, which, lying rather more deeply in the neck on the outer side of the common carotid artery, may be noticed to visibly pulsate, especially if the overlying sterno-mastoid muscle be relaxed. While, however, this slight amount of venous pulsation may be perfectly obvious in the healthy subject, we must clearly realize that it is by no means a *sine quâ non*, for one frequently notices that it is entirely absent, and unable to be appreciated by naked-eye observation, especially in those normal adults who are possessed of necks nicely proportioned and free from hollows. It seems, therefore, from the foregoing that one must accept both slight venous pulsation and its complete absence as conforming entirely to the healthy state of the normal adult, and, as a general rule, it may be taken that the amount of subcutaneous fatty tissue is the determining factor in every case.

Following inspection one must proceed a step further and endeavour to become conversant with any auscultatory

phenomena which may be normally recognizable at the root of the neck. This question is one which can readily be proved to offer little difficulty: the second aortic sound is clearly heard when one listens over the common carotid artery, also the first sound, which, though perfectly definite, is less well conducted than the former; both sounds, moreover, are better heard on the right side than on the left, as one would naturally expect from anatomical considerations, and both undoubtedly suffer some diminution in intensity by reason of the fact that they invariably mingle to some extent, not only with the respiratory murmur, but also with the venous hum which can so often be detected, especially if the venous channels be large. It is, however, possible, by using gentle pressure, to compress the veins sufficiently to abolish this bruit, but great care must be taken, for if the pressure be excessive the arterial lumen will also be diminished, and a bruit in the artery itself will thereby be produced.

These few remarks explain sufficiently what one must expect, and provide for us a clear mental picture of the characteristics peculiar to health, so that there can be no difficulty in recognizing the various and significant changes which manifest themselves coincidentally with the onset of cardiac disturbances.

That the comparison is striking and self-evident one needs but call to mind the clinical picture which valvular disorders so often present late in their history: the jugular veins, overcharged with blood, stand out in prominent fashion, and over them passes a swift wave-like rippling movement, the visible indication of the state of affairs in the auricular chamber.

This venous pulsation is very frequently of value from the point of view of diagnosis, for its types are many and varied. To mention but a selection will suffice:

(a) The extremely irregular rhythm of auricular fibrillation.

(b) The break of an otherwise regular rhythm by the incidence of premature beats; of the latter, possibly the most evident are those originating in the atrio-ventricular region.

(c) The flickering undulation of high velocity which characterizes the paroxysms of true tachycardia and auricular flutter.

(d) The differing venous and arterial rates of auriculo-ventricular block, several auricular beats being recorded for every one of the ventricle, a physical sign which renders ordinary heart-block diagnosable by means of inspection alone.

One moment's consideration, therefore, will prove that venous pulsation at the root of the neck is by no means a rare phenomenon, being present in health to a slight degree, and in cardiac disorders frequently. Naturally, the extent of the pulsation and the particular amount of venous distension are variable factors and significant at times of widely differing conditions. More often than not a forcible deep inspiration will temporarily empty the distended veins, but there are conditions in which no such effect can possibly be produced, as, for instance, (a) pericardial effusion, if the amount of fluid be sufficient to effectively obstruct the onflow of venous blood to the heart; (b) adherent pericardium, the extreme fibrosis accompanying which spreads slowly and by degrees round the great vessels, tending in time partially to strangle these latter, an effect much exaggerated by deep inspiration; (c) aortic aneurysm, in which the venous overfilling assumes a more permanent quality, tends to be rather more prominent on one side than the other, and extends over a very wide area, possibly including the side of the face, the arm, and the chest wall in varying amounts; (d) new growth, probably a more frequent cause of permanent venous distension than any other.²

Such considerations, therefore, outline the importance attaching to over-distension of the veins. Equally important, however, is the other side of this picture, which, shortly expressed, may be taken to indicate a myocardial failure, the muscle being too weak to maintain even a small amount of venous plethora. This particular branch of the subject has been ably referred to by Stacey Wilson in certain of his recent lectures. He lays special stress upon the signs of heart weakness in later life, when the actual volume of circulating blood is cut down, the area of cardiac dullness is diminished, the loudness of sounds and

murmurs is lessened, the supraclavicular hollows are empty, and the cervical veins are maintained in a state of permanent collapse. Signs such as these, therefore, are the expressions of an enfeebled myocardium, whose mechanical powers have become curtailed to such an extent that a physiological rearrangement has become necessary—namely, an actual decrease in the amount of circulating blood, and a general vascular emptiness; memorable phenomena, in that they are so unlike the physical signs met with in cases of cardiac failure, where, in spite of a general venous distension, hepatic engorgement, oedema, etc., the cardiac muscle continues to contract in vigorous fashion, possessing, as often it does, abnormal powers of resistance.

THE ARTERIAL PHYSICAL SIGNS.

Under this heading it is proposed shortly to consider the diagnostic help which may be acquired by a careful inquiry into the conduct of the large arterial trunks at the root of the neck.

The carotid vessels, whose beat can be accurately located by palpation on a line drawn from the inner end of the clavicle to a point midway between the angle of the jaw and the mastoid process, are sufficiently deeply placed under fascial and muscular coverings that, under all ordinary circumstances, no pulsation is visible. With the presence, however, of certain diseases, notably aortic regurgitation, profound anaemia, exophthalmic goitre, etc., this obviously does not apply; one has only to call to mind an ordinary case of aortic disease to remember at once the intense pulsation visible in the neck in such a case, and, further, that the pulsation is of a peculiarly collapsing quality, an ill-sustained wave, especially in the later stages of the disease, so much so that one is persuaded to adopt as a general working hypothesis the fact that the greater the amount of collapse present the greater the amount of regurgitation, and necessarily the greater the progress of the malady. This, however, does not apply to those cases in which the aortic stem and large arteries have become rigid with disease and possibly there may be a progressive stenosis superadded to the original disease; such changes would obviously militate against an excessive pulsation.

This latter subject—namely, exaggerated arterial pulsation—should under no circumstances be dismissed without remembering a fact which seems very self-evident, but which is none the less important—that, when present in consequence of an aortic lesion, it constitutes a bilateral feature, in contradistinction with its one-sided appearance in aneurysm of the carotid artery and also when on one side the normal carotid artery is displaced forwards by some such agent as a neoplasm which causes the vessel to take up a more superficial position. The differential diagnosis between these conditions offers no difficulty when one remembers the generalized implication of the vessels in the one instance and the purely localized signs in the others.

Proceeding next to direct vascular auscultation, and remembering that the heart sounds, both first and second, may be heard quite clearly in the arteries of the neck under normal conditions of health, it does not seem in the least surprising that murmurs may also, at times, become evident. First, the true systolic murmur, which, possibly, of all murmurs is the most readily conducted towards the periphery. This murmur may be produced in one of two ways—either by a true aortic stenosis, or by an aortic dilatation causing a condition of virtual valvular stenosis, with possibly the presence of rigid, rough valves; and with the first of these conditions, the seldom seen true stenosis of the aortic orifice, one hears a rough systolic murmur on auscultating the carotid or subclavian artery in the neck, a marked diminution in the loudness of the second sound, and palpation reveals the conduction of the systolic thrill into the same vessels. The second condition—namely, aortic dilatation with valvular roughening—also yields a loud systolic murmur with upward conduction; but in this case there is no thrill and the aortic sound is accentuated in loudness.

Deserving, moreover, of some attention in the present context are the upwardly conducted murmurs which result from some one of the large variety of congenital lesions—such, for instance, as (1) the murmur produced by pulmonary stenosis, which, resembling the pulmonary

dilatation murmur of anaemia, suffers conduction into the cervical vessels on the left side but not on the right; (2) the murmur of congenital aortic stenosis, and (3) the murmur of pulmonary stenosis, which, complicated as often it is by the patency of a ductus arteriosus, is conducted by the agency of the latter into the systemic vessels, and therefore can be heard clearly in the carotid arteries on both sides.

These considerations concerning the upwardly conducted systolic murmur, with brief reference to the arterial murmurs in exophthalmic goitre and anaemia, lead to an examination of the murmurs which are found in the diastolic interval. It will suffice for this present purpose if we make short mention of the murmur and some of the physical signs associated with incompetence at the aortic orifice.¹ In the writer's opinion, apart from a very natural study of the amount of dilatation present, and also the progress of the sclerotic changes in the heart and vessels in each particular case, one may form a very reasonable estimate of the progress of the malady by a careful investigation of the murmur itself and its routes of conduction. In the early stage one hears the typical murmur following the second sound, and seemingly preventing the valves closing with the usual snap; this murmur is conducted down the sternum apexwards, and auscultation in the neck reveals only the normal heart sounds, possibly the second being somewhat weakened. Stage II arrives with a conduction of the murmur into the vessels—the well known murmur of Duroziez, the first and second sounds being, however, still faintly heard. Stage III differs only from Stage II in that the murmur is more distinct in carotids and the second sound can no longer be detected. This departure of the second sound from the carotid, by preference the right carotid, where one is out of reach of the pulmonary sound, always marks a serious advance of the disease, as does the advent of Stage IV, when one is only able to hear a loud murmur in the vessels, possibly even the double aortic (bellsows murmur), by reason of some certain amount of added sclerosis producing a narrowing of the aortic orifice, and the heart sounds are quite inaudible.

It seems valuable, therefore, to listen especially for the normal second aortic sound in the neck in patients with aortic incompetence; also to ascertain if the murmur is conducted into the vessels, its relative loudness in this situation and at the aortic cartilage, and to base one's prognosis upon the results of such an investigation, combined with the ordinarily careful physical examination.

THE VENOUS PHYSICAL SIGNS.

Under this heading we may range a varied and interesting group, the several members of which not only afford valuable comparison with the state of affairs normally obtaining at the root of the neck, but assume an importance equal in every respect to that attaching to the arterial physical signs just enumerated. Visible venous pulsation, so insignificant in the healthy, becomes with disease a striking feature, and may assume, broadly speaking, one of two great varieties—either the positive or the negative type. By these terms one distinguishes the pulsation observed in a vein which is filled during systole—as, for instance, when, with an incompetent tricuspid valve, every contraction of the ventricle reflects, quite unopposed, a wave into the proximal veins—from the opposite type, the negative pulsation, so called because, with the systolic phase, there is a definite venous emptying. As a rule it is quite impossible to differentiate these pulse variations by inspection alone, and, as we shall see later, one can only form an accurate diagnosis if aided by some instrumental method—either a graphic record of the excursions of the vein wall, such as can readily be furnished by any ordinary polygraphic machine, or by the more recently devised method of Parkinson,³ who photographs directly the venous pulsation by seating the patient so that the neck is placed directly in the beam of light from an electrocardiograph. By this proceeding one overcomes the disadvantage which sometimes attaches to records obtained by the tambour process—namely, the instrumental delay and possible overswing—and, in addition, one can simultaneously record the electrocardiogram, thus, for timing purposes, producing a combined tracing of the greatest possible value.

Naturally, however, one cannot invariably obtain these

venous pulse records. There are at times difficulties, as, for instance, with the excessive arterial pulsation of aortic disease, when the venous pulse is quite obscured; also at times of respiratory distress, when, all the accessory muscles of respiration being brought into play, it becomes quite out of the question to attempt any form of tracing at the root of the neck, and in the extreme venous emptiness which is known not only to result from severe heart weakness but also to accompany certain types of adherent pericardium, though in this latter condition one more usually finds a universal venous engorgement, somewhat approaching the condition observed in cases of aneurysm and pericarditis with effusion. These two diseases, however, are somewhat distinct, in that with them venous distension is quite continuous, not disappearing even when a deep inspiration is taken. Moreover, with the presence of pericardial adhesions, the fullness or emptiness respectively of the great veins being determined by the actual position and consistence of the adhesions themselves, one usually finds that the veins are also permanently distended and cannot be emptied, even by a forced inspiration. There is, however, another frequently occurring lesion which participates in the production of venous distension; it is tricuspid incompetence, and the progress of this particular valvular mischief may be very fairly judged by carefully examining the great venous channels connected with the right auricle. For our present purpose we may take the jugular vein and investigate, by the graphic method, the different stages which characterize the condition; its early course, as portrayed by means of ordinary phlebographic tracings, presents no features of note; in fact, the record appears normal. Fig. 1 is an example, and, taken from a

FIG. 1.

patient whose tricuspid valve had for some months been continuously leaking, amply supports the contention; the wave of auricular contraction is well marked, and occupies a normal situation in respect of *c*, the wave caused by the ventricular systole, and this latter is followed at a perfectly normal interval by the *v* wave, which results from the blood stagnating in the auricular chamber prior to the opening of the auriculo-ventricular valves.

When, however, the valvular incompetence progresses we soon begin to notice changes in the venous tracing; the auricle, which at ordinary times was filled solely by the onflow from the veins, is now, in addition, supplied by the regurgitant stream from the ventricle; hence not only is there an overfilling and distension of the upper chamber, but clearly also an auricular filling which is earlier accomplished than formerly it was, and, this being so, one is not surprised to notice that the *v* wave appears prematurely if the tricuspid valve has suffered much damage. To make quite apparent the foregoing remarks one has only to compare the relative positions of the *v* waves in Figs. 1 and 2,

FIG. 2.

which are reproduced to illustrate the first two stages of the condition as recognized by polygraphy.

As time progresses the prominence of the *v* wave becomes more marked, and it merges gradually into *c* wave so that a composite wave is produced, itself ample evidence of the auricular engorgement; but later, and

possibly not until after the lapse of some years, the venous chambers become overtaxed and the auricular muscle, following the protracted period of stress and overstrain, capitulates completely, its wall ceases to contract co-ordinately, and frequently passes into a condition of fibrillation, which, though implying intrinsic activity, is to all intents and purposes useless for the onward propulsion of the blood stream.

FIG. 3.

Fig. 3. is a tracing typical of this advanced stage in the history of tricuspid incompetence; we notice in it—

1. A complete absence of the auricular waves.
2. A fusion of the *c* and *v* waves.
3. A similarity between the arterial and venous tracings in that in both one observes a systolic filling and a diastolic emptying, a state of affairs entirely different from that observed at all ordinary times.

Bearing these observations in mind, and admitting that they are by no means comprehensive, but, rather, isolated facts which tend towards and amply warrant a careful study of the varied and widely differing phases which the great veins may assume, one would recommend their careful examination as a matter of routine, especially in cardio-vascular disorders, and, in conclusion, suggest that venous auscultation be not forgotten, for by its aid one gains additional advantages, abnormal sounds become audible—for instance, the well-known *bruit de diable*, characteristic of anaemic states, which, caused by loss of tone in the vein walls, is heard best when the patient is in the standing position, also the upwardly conducted venacaval murmur, the sounds resulting from the closure of the venous valves, and, lastly, the clicking sounds to which the rapidly-contracting auricle of auricular flutter gives rise and which are aptly referred to by Morrison as jugular embryocardia.

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THE HEART'S ACTION IN JAUNDICE.

BY

J. DAVENPORT WINDLE, M.D.,

SOUTHALL.

THE belief is prevalent that the pulse becomes unusually infrequent and at times irregular in rhythm in jaundiced patients. These changes in the pulse are said to occur commonly in catarrhal jaundice, although met with occasionally in jaundice from other causes. It is curious why there should be this difference if, as is stated, the slowing of the heart is due to the action of bile salts on the heart muscle and intrinsic inhibitory mechanism of the heart.

I have investigated the heart's action in a number of jaundiced patients; in some I have found the pulse infrequent, in some irregular, in others both infrequent and irregular, but I have not been satisfied in any case that the jaundice was responsible for the infrequency or the irregularity. It is said the pulse may fall as low as 40 a minute; if by "pulse" is meant rate of ventricle, this rate, then, must be rare. I have not met with an instance in over 100 cases of jaundice. Slowing of the ventricle to 40 or below, whether occasioned by disease or poison, is, with rare exception, due to partial or complete heart-block, and so far as I know heart-block in the course of jaundice has not yet been illustrated.

A pulse-rate about 50 and 55 a minute is not uncommon in patients with jaundice when free from fever and the patient is kept in bed, but this is not proof that the jaundice is the cause of the slow pulse; the patient's normal pulse-rate must be known; and how it behaves in

other conditions, because the pulse has a tendency to become infrequent in many non-febrile illnesses when patients are confined to bed. The slowest pulse-rate I have met with due to total bradycardia, in an adult with jaundice, was 50 a minute. The patient was a man aged 65, with severe jaundice from gall stone disease; his normal pulse-rate was about 72 a minute. A year after this attack of jaundice he had severe bronchitis with fever and a fast pulse. When he was getting better the pulse fell to 50 to 52 and kept at this rate until he got about again. I could relate many similar examples; on the other hand, in the vast majority of cases of jaundice in adults I have found the frequency of the heart about or above the normal.

In a number of cases I have found a pulse-rate of 40 and below; but the heart-rate has been double the pulse-rate, the infrequent pulse being due to an alternate premature beat of the ventricle too feeble to send a wave to the wrist. This is shown in Fig. 1.

FIG. 1.—Showing two beats of the left ventricle to one of the pulse. A. Apex.

Cases of jaundice with this form of irregularity of the heart and pulse slowing have been published to illustrate the action of the bile salts on the heart, the assumption being that they increase the excitability of the heart muscle, and thus provoke the premature contractions; but there is no other proof advanced, and information is wanting as to the rhythm of the heart before the jaundice occurred and after it had passed off. My own experience indicates no proof that patients with jaundice are more liable to develop extra-systoles than are patients suffering from other toxic conditions. All the cases of jaundice with premature beats I have observed have occurred in persons past middle age; I have not met with any example in a child or young adult, whereas poisons—notably the digitalis group—which in very large doses may produce extra-systoles, do not show this selective action as regards age. In most of my cases of jaundice showing premature beats I know extra systoles were present in health. In some, in which premature beats were noted for the first time during jaundice, I found the extra-systoles persisted long after the patients were free of jaundice, and although this fact does not disprove the provocative action of the bile salts, nevertheless experience has shown that extra-systolic irregularity due to poisons ceases within a few days after the cause is removed. I do not contend that irregularity due to extra-systoles may not be favoured during jaundice—it may be; but I submit it is not because of any action of the bile salts on the cardiac muscle, but because the illness keeps the patient resting. This slows the heart, and premature beats, perhaps only occasional in health, tend to become more frequent with the slower heart action.

As already stated, the pulse is said to be affected more particularly in catarrhal jaundice. This is not a common ailment in adults, and I have notes of only four adult cases; in three of these there was nothing noteworthy in the pulse, in one case sinus irregularity was present. This patient was a woman aged 35; jaundice was marked. In the tracing (Fig. 2) is shown a waxing and waning in rate

FIG. 2.—R, Respiration; I, inspiration; E, expiration.

of pulse, which is 65 per minute on the average. The variations in length of diastoles are independent of the respiratory movements. I have frequently noticed this relation in sinus arrhythmia induced by digitalis, and the irregularity in this case may be due to the jaundice.