

trying to read it from a tracing, but attention to the following points usually enables one to form a fairly correct opinion from a tracing. In the first place the tracing must be taken by a person of experience. A poorly

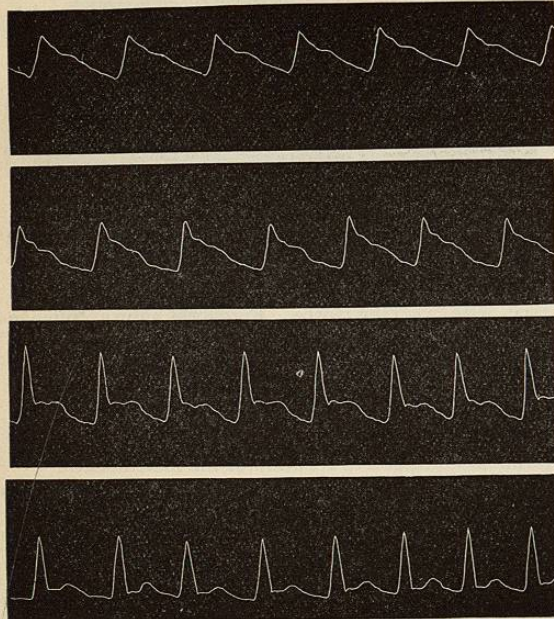


FIG. 3907.—Normal Pulse Tracings, Indicating Different Arterial Pressures.

taken tracing is very misleading. Where the blood pressure is high the rise and fall of the waves are more gradual, in low blood pressure quick or steep. In high blood pressure the dicrotic wave is small and high up on the descent. As the blood pressure becomes lower this wave becomes more distinct and approaches nearer to the base line, and may appear as a wave rivalling the primary wave in size and separated from it by a distinct interval. If the blood pressure becomes very low indeed, as in cardiac failure, then the dicrotic wave becomes smaller again and may quite disappear. A well-marked dicrotic wave is a sign of relaxed peripheral vessels and not of a weakly beating heart. It is seen best developed at the beginning of fevers when the skin is hot and the blood distributed to the surface, but the heart still beating strongly.

The predicrotic wave is most pronounced in cases in which there is most resistance to the outflow from the ventricle, and therefore usually indicates high blood pressure. It is also seen, however, in aortic stenosis (see Fig. 3899). A practical rule for judging of the predicrotic wave is given by Gibson: "Draw a line from the top of the primary wave to the lowest point of the dicrotic notch." If the predicrotic wave fails to reach this line the pulse is of low or medium pressure. If the predicrotic wave rises higher and crosses this line the pulse is of high pressure (better, the resistance to outflow of blood from the ventricle is great). Another peculiarity often

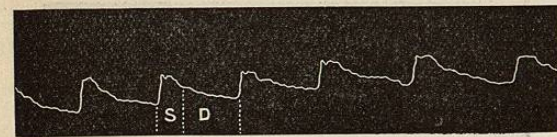


FIG. 3908.—High-Tension Pulse. (Hutchison and Rainey.)

seen in high-pressure pulse tracings is the presence of several oscillation waves on the descent such as those seen in parts of Fig. 3908.

When interpreting tracings it is well to bear in mind

that the part from the beginning of the primary wave to the bottom of the dicrotic notch corresponds to the time when the blood is being driven out of the ventricle into the arterial system. This almost corresponds with the ventricular systole, and may conveniently be called the *systolic portion* of the tracing. From the bottom of the dicrotic notch to the beginning of the next primary wave the aortic valves are closed and no blood is leaving the ventricle. This may be called the *diastolic portion* of the tracing.

Any peculiarities in the systolic portion may usually be traced to the ventricle; but the diastolic portion cannot depend directly upon the ventricle, as during this time the aortic valves shut off the arteries from the heart. Variation in the rate of the pulse is brought about principally by shortening or lengthening of the diastolic por-

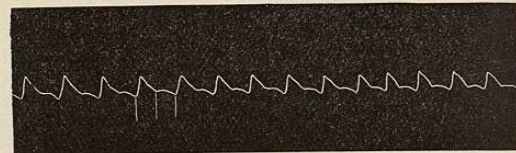


FIG. 3909.—Frequent Pulse.

tion. Thus in an infrequent pulse like that in Fig. 3908 the diastolic portion which I have marked *D* is about twice as long as the systolic portion *S*, whereas in Fig. 3909 where the pulse was 130 as the result of a fever, the two portions are of about equal duration.

In some cases the shortening of the diastolic portion takes place to such an extent that the next percussion wave commences before the dicrotic wave is complete and cuts into the descending limb of the latter. The result is a tracing like that shown in Fig. 3910. *S* is the percussion and *D* the dicrotic wave. It will be noticed that the dicrotic notch *N* is the lowest part of the tracing. Such a tracing is called *hyperdicrotic* or *superdicrotic*.

**THE PULSE IN VARIOUS PATHOLOGICAL CONDITIONS.**  
*Affections of the Aortic Valve.*—The pulse of aortic regurgitation is one of the most typical, and is known as *Corrigan's pulse*, from Sir Dominic Corrigan, who was one of the first to describe it, or as the *water-hammer pulse* from a toy of that name that gives a sudden shock to the

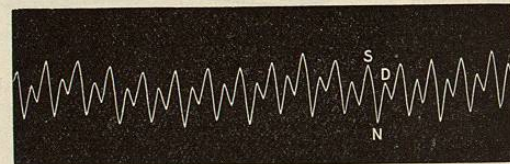


FIG. 3910.—Hyperdicrotic Pulse. (Mackenzie.)

fingers. Corrigan's pulse may be described as large and quick, usually infrequent and regular. It is sometimes described as collapsing because the vessel seems to become empty between the pulse beats. The impression given to the finger is that of a momentary tap.

The peculiarities of Corrigan's pulse are best made out by raising the patient's arm high above his head while feeling his radial.

The statement made by Henderson, Balfour, and others that the rate of propagation is slower in aortic regurgitation than where the heart and vessels are normal has been shown by François Frank, Key, and Mackenzie to be without foundation. A prominent feature of aortic regurgitation is the violent throbbing of the vessels of the neck. A capillary pulse may also usually be seen. In some cases the pulse wave passes right through the capillaries into the veins and may be observed in the veins of the back of the hand, and, sometimes, a tracing may be obtained of this rare form of venous pulse. Such a tracing is figured in Gibson's book on the heart.

Tracings from the radial in aortic regurgitation are characterized by a steep rise and fall of the percussion wave and by the fact that the predicrotic wave is usually more pronounced than the dicrotic. The tracing shown



FIG. 3911.—Aortic Regurgitation.

in Fig. 3911 is from a case of moderate severity and illustrates these points. In a more pronounced case the dicrotic wave would be still less marked or even absent altogether, and the percussion wave might be even steeper in its rise and fall. The presence of a fairly pronounced dicrotic wave does not, be it noted, exclude a moderate degree of aortic regurgitation.

The pulse of *aortic stenosis* is not so characteristic as that of regurgitation. It usually feels slow and sustained to the examining finger, is of normal or diminished frequency, and, like that of aortic regurgitation, regular in rhythm and volume. The tracing is usually anacrotic (see Fig. 3899). The predicrotic wave rises higher than the primary, indicating the difficulty the ventricle has in emptying itself. The details vary in different cases. Sometimes the primary and predicrotic waves form two well-marked crests separated by a distinct depression, forming the so-called *pulsus bisferiens*. In other cases instead of the tracing being anacrotic we may simply have a primary wave with a gradual rise and a rounded crest, followed by a poorly marked dicrotic wave.

In *mitral regurgitation* and *mitral stenosis* the pulse may be quite indistinguishable from the normal. As one or other of these diseases progresses, however, dilatation of the ventricle, and especially of the auricle, takes place; then the pulse tends to become weak, rapid, and markedly irregular in both rhythm and volume, constituting the so-called "*mitral pulse*." This pulse varies so much in its details in different cases that it is useless to figure any one form as typical.

*Affections of the Heart Muscle.*—There is nothing uniform or typical about the pulse in these conditions. It may be abnormally slow or fast or irregular. The cardiac field of response is diminished. Any departure of the pulse from the normal rate or rhythm should lead one among other things to consider the probability of the heart muscle being diseased, but other considerations besides the pulse will have to be depended on for the solution of the question.

The presence of the *pulsus paradoxus* in certain cases of *adhesive pericarditis* has already been referred to.

In *aneurism* of the transverse part of the aorta the left radial pulse is often smaller and slower than the right. It sometimes feels delayed too, but this is questioned by some writers. The exact character of the radial pulse varies greatly in cases of aneurism according to the situation of the disease, the degree of degeneration of the vessels generally, and the condition of the aortic valves. Fig. 3912 shows simultaneous tracings from the aneurism and the right radial in a case in which a large pulsating aneurism extended from the chest up the right side of the neck. The exaggeration of the predicrotic wave seen in this tracing seems to be very common in aneurism, as it is in both forms of aortic valvular disease.

In *fever* we have to distinguish between the effect of the pyrexia as such upon the pulse and the effect of the disease causing the pyrexia. Moderate pyrexia tends to modify the pulse through both the heart and the peripheral resistance. The heart is made to beat more rapidly, partly by a direct action of the increased temperature on the heart tissue itself, and partly indirectly through the cardiac centres in the medulla. The superficial arteries are dilated and an increased amount of blood is carried to the skin. The effect of these changes is to cause a pulse

of increased frequency and diminished pressure with exaggeration of the dicrotic wave. To the finger such a pulse feels frequent, large, and soft. It is often referred to as bounding. A tracing would more or less approach the type shown in Fig. 3913.

If the pyrexia runs high or lasts long the heart suffers and becomes feeble and often irregular. In such cases the pulse is very frequent and is small, soft, and compressible. Tracings taken at intervals, when the heart is failing, show gradual disappearance of the dicrotic wave, the absence of which in a case of fever is usually a very bad sign. Irregularity of the pulse in the early stages of a fever or increase of rate in an adult beyond 140 per minute are symptoms which usually indicate great danger. During convalescence slight irregularity is common and of little significance. The character of the pulse in fever may be much modified by other influences, such as specific poisons or mechanical interference.

**SUMMARY OF THE DIAGNOSTIC VALUE OF THE PULSE.**  
—The pulse furnishes the best single indication of the state of efficiency of the circulation. In it we find indications both of the condition of the vessel walls and of the strength of the heart beat.

Very important information may be obtained by observing the changes in the pulse during bodily activity.

The discovery of high tension may direct our attention to the presence of nephritis or lithæmia.

In aortic valvular disease a quick collapsing pulse or a

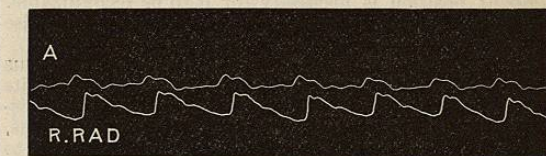


FIG. 3912.—Simultaneous Tracings from a Thoracic Aneurism and from the Right Radial Artery.

slow, small pulse will indicate the predominance of regurgitation or stenosis.

In mitral valvular disease a rapid irregular pulse is suggestive of loss of compensation and dilated or paralyzed auricles.

In pericarditis with effusion, where the heart sounds are faint, the pulse is of especial value in indicating the degree of cardiac failure.

In fever the rate, the tension, and the presence or absence of irregularity are of great prognostic value. A sudden change in the pulse may be the first indication of a crisis or a fresh complication. In many diseases the pulse is of value in furnishing indications for treatment (stimulants) and in exhibiting the effect of the remedies used.

In the search for all these indications the finger is the best means to employ. In special cases some of the more elaborate instruments may furnish additional or confirmatory information.

**THE VENOUS PULSE.**—*Introductory.*—The term venous pulse is applied to changes in size and tension occurring in the veins as the result of the action of the heart. The

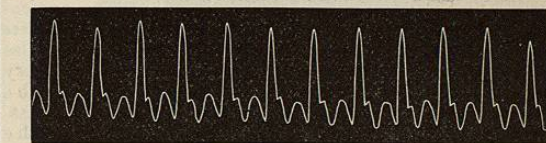


FIG. 3913.—Pulse in Sthenic Fever. (Mackenzie.)

distention and collapse of the veins caused by the alternate phases of respiration are not included. A true venous pulse in a healthy animal seems to have been first described by Wedemeyer of Hamburg, in 1828. He made his observations on a horse. Since that time numerous physiologists have seen and recorded the venous

pulse in healthy dogs, cats, rabbits, and other animals. It has been noted in the veins of the thorax, neck, abdomen, and limbs. Mosso obtained the first venous pulse tracing from a human subject in 1879. Since then those

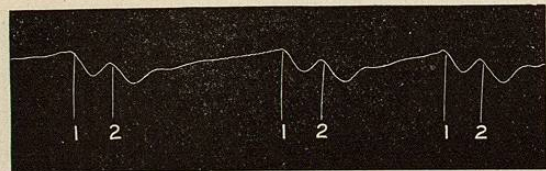


FIG. 3914.—Negative Venous Pulse (dog).

who have looked for it in man have found it to be very common. In the writer's experience it may be seen and recorded in the majority of people by those who make the examination under suitable conditions. It is most frequently seen in the jugular veins, external and internal. In the case of the external jugular one can usually see the blue vein through the skin; in the case of the internal jugular one can see the movements imparted to the skin over it. Sometimes one vein can be seen best, sometimes the other. For the observation of the venous pulse two conditions are usually necessary, viz., that the veins be reasonably distended with blood, and that the neck be not too fat. Probably in the majority of people the recumbent position is necessary for it to be recognizable. For the method of taking tracings of the venous pulse see *Sphygmography*.

**VARIOUS FORMS OF VENOUS PULSE.**—The venous pulse is seen in many different forms. This renders its study more difficult than that of the arterial and has discouraged many from undertaking it. Its modifications, however, may be traced with considerable confidence to their respective causes, and are replete with indications of the condition of the heart for those who will take the trouble to familiarize themselves with them. James Mackenzie, who has written more exhaustively on the venous pulse than any other English writer, claims that it "gives us far more information of what is actually going on within the chambers of the heart" than the

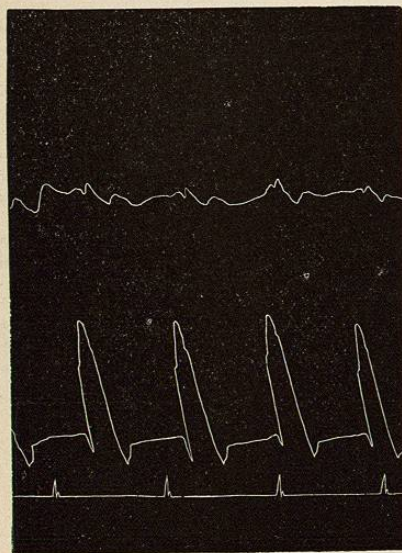


FIG. 3915.—Curves of Auricular (above) and Ventricular Pressures, from a Dog. Time in seconds.

is not too frequent. Fig. 3914 is taken from the internal jugular of a dog under the influence of morphine with a pulse rate of 45 per minute.

It will be observed that the down strokes in the trac-

ing which denote the collapse of the vein (negative pulse) are far steeper than the rises which indicate refilling. The fall beginning at 1 is called the *systolic collapse*, and is due to the diastole of the auricle drawing in blood from the veins during the ventricular systole. The fall beginning at 2 is the *diastolic collapse* due to the diastole of the ventricle. The ascending portions of the tracing, which indicate filling of the veins, are caused principally by the blood flowing in from the capillaries faster than the heart can receive it. Just before the systolic collapse (1), however, we may in some cases observe two small elevations, the *presystolic* and the *systolic rise*. These may be traced back to their origin in the systole of the auricle and of the ventricle. We speak of the long ascent leading up to these waves as the *diastolic rise*. The irregularities at the beginning and in the middle of this diastolic rise are unexplained. The interpretation given above of the systolic rise as due to an impact propagated backward through the auricle and veins by the ventricular systole is disputed by some writers (Mackenzie), who

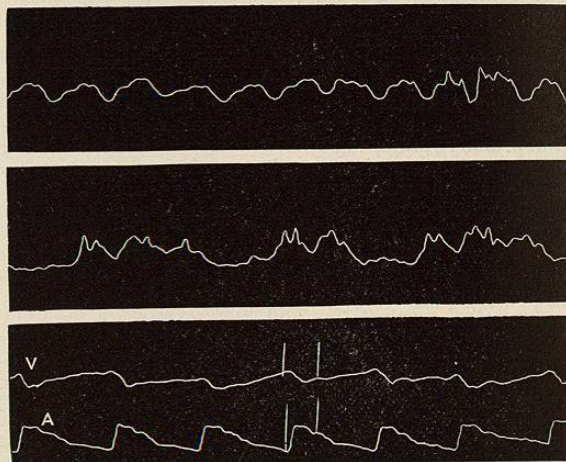


FIG. 3916.—Three Types of Negative or Auricular Venous Pulse (human).

ascribe it entirely to a shock imparted to the vein by the pulse in the carotid. This view is doubtless partly true, as there is often a wave in the venous pulse due to the carotid impact; but it is equally true that there is quite frequently a wave at the top of the diastolic rise that can be found both in the venous pulse and in the curve of auricular pressure, and that may be shown to be synchronous with the ventricular systole. In Fig. 3915 simultaneous tracings are shown of the pressures in the right auricle and ventricle of a dog taken with Hürthle's catheter. Corresponding points of time are marked by the vertical lines. It will be noted that there is a very distinct wave in the auricle synchronous with the ventricular systole. This is no doubt due to the rise of pressure in the ventricle pushing the tricuspid valves back and thus imparting a shock to the auricle.

For the rise in the tracing (Fig. 3914), leading up to 2 there is, so far as I know, no satisfactory name. Some writers call it the first diastolic rise, but it is systolic in time. Others call it the ventricular rise, but it is not caused by the ventricle but by the blood flowing in from the periphery. A rational term to apply to it would be the *prediastolic rise*, for it leads up to and is interrupted by the ventricular diastole.

When the pulse is frequent the waves are crowded together, and some of those described are not seen at all or cannot be identified. Take, for instance, Fig. 3916 where three examples are given of venous pulse tracings from human subjects. It is very difficult to distinguish the different waves described above in these three tracings. In the last of the three a method is shown by which the difficulty may be partially solved. In this, simultaneous

records are taken from the external jugular vein (above) and the radial artery (below). Corresponding points of time are marked on the two tracings, and by these it can be seen that synchronous with the rise of the radial pulse tracing there is a fall in the venous, the systolic collapse.

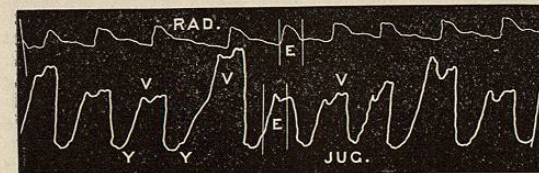


FIG. 3917.—Ventricular Venous Pulse (below); Radial Pulse (above). (Mackenzie.)

This systolic collapse alone is sufficient to prove the venous pulse to be of the auricular or negative variety. Using the radial pulse as a guide, it is possible to find traces of the other waves described as typical, although they are not very distinct. Be it noted here that the systolic and diastolic portions of the jugular pulse correspond very closely in time with the systolic and diastolic portions of the radial pulse, because, as has been pointed out by the writer, the greater distance of the radial from the heart is compensated for by the fact that the rate of propagation of the venous pulse is only about one-third that of the arterial.

As a rule, the auricular venous pulse can be readily recognized without taking a tracing. It is characterized by a sudden collapse of the veins of the neck followed by a more gradual filling. Where the pulse is infrequent the collapse is double, corresponding to the systolic and diastolic collapse seen in a tracing (Fig. 3914). The proper organ for the examination of the venous pulse is the eye, and it is best seen with the patient reclining with the head on a level with the body (no pillow). The finger is of little use as the changes in tension are too slight to be appreciated by it. It is a good plan to have the finger on the radial as a guide to the time relations. In cases of doubt, as when the pulse is frequent, simultaneous tracings must be taken of the jugular pulse, and either the apex beat, or the carotid, or the radial.

The presence of the auricular venous pulse has little significance. I have seen it at all ages from infancy to old age, and have recognized and recorded it in healthy and athletic young men as well as in a variety of diseased conditions. It is practically never absent from healthy dogs, and I believe that when it cannot be observed in a human subject, who is in a proper position for observation, it is because the tissues of the neck are too thick for it to show through rather than because it is not there. It is more marked than usual when the tissues of the neck are specially thin or when the veins of the neck are distended. For it to be seen at its best the heart must still be beating with fair vigor. Among the conditions in which the auricular venous pulse is pronounced are nearly all forms of emaciation and general debility, diseases in which the entrance of blood into the chest is interfered with, such as rickets and chronic coughs, conditions in which there is slight dilatation of the heart as at the end of long continued fevers or in the various forms of anemia.

The *ventricular or positive venous pulse* is seen where the high pressure existing in the ventricle during systole is transmitted to the veins so as to prevent the usual systolic collapse. In a typical ventricular venous pulse the only collapse we have in the veins is the diastolic collapse caused by the blood being sucked in by the diastole of the ventricle. In these cases the veins of the neck can usually be seen to be distended, and the pulse can be seen in them even with the patient standing or sitting up. Moreover, the filling or distention of the veins is seen to take place, or, at least, to be completed suddenly, and the finger can detect a positive impact corresponding in time with the carotid pulse. Simultaneous tracings from the jugular vein and the radial artery show absence of the usual systolic collapse. Instead, we

see a continued rise or a sustained elevation of the tracing during the ventricular systole, succeeded by a sudden diastolic fall. These points may be very well seen in Fig. 3917. In this figure simultaneous points of time in the radial and jugular pulses are marked by vertical lines including between them the systolic period *E*. It will be noted that the venous pulse consists of a single large wave with a divided crest, and that the only pronounced collapse is after the systole is over. A somewhat less typical case observed by the writer is shown in Fig. 3918. This was taken from a case of tricuspid regurgitation in which compensation had been partly restored by digitalis.

The ventricular venous pulse is found in three conditions. By far the most usual cause of it is *tricuspid regurgitation*, but it cannot quite be called pathognomonic of this lesion, as there are two other rare conditions in which it is found. One of these is mitral regurgitation with patent foramen ovale, of which a case was recently reported in "The Johns Hopkins' Hospital Bulletin" by W. S. MacCallum. The other is adhesive pericarditis, in which the contraction of the ventricle draws in the thoracic wall and causes compression of the thoracic viscera; sufficient pressure is thus exerted on the great veins to initiate a positive wave which is propagated into the veins of the neck.

The ventricular pulse may in some cases be confounded with the pulse of auricular paralysis which will be described shortly.

The *arterial venous pulse* includes four different forms of pulsation in the veins, of which none call for more than a mention.

1. A pulse may be transmitted from the arteries through the capillaries in aortic regurgitation or where there is great dilatation of the peripheral vessels.

2. Cases are on record in which there has been an anastomosis between a peripheral artery and vein with consequent transmission of a pulse.

3. Pulsations which are arterial in origin are sometimes seen in the veins of closed cavities like the eyeball.

4. Veins may have a pulsation transmitted to them from arteries as a result of mere juxtaposition. This last fact must be remembered in interpreting the tracings obtained from the veins of the neck, especially the internal jugular, as frequently one of the waves seen is due to the impact of the carotid upon the vein. Such waves are best identified by comparing the tracing with a simultaneous one from an artery.

The *Venous Pulse of Auricular Paralysis*.—When the auricle is paralyzed the presystolic (auricular) wave is absent and so is the systolic collapse (auricular diastole). The tracings obtained are very similar in form to those of tricuspid regurgitation, as the tracing continues to rise until the ventricular systole is complete, and then a fall due to the ventricular diastole occurs. The venous pulse of auricular paralysis with competent tricuspid valves can be distinguished from the systolic pulse of tricuspid regurgitation better by the finger than from a tracing. In a tracing, it is true, the rise is more uniform and gradual in auricular paralysis than in the ventricular pulse where a systolic elevation may be made out; but in some

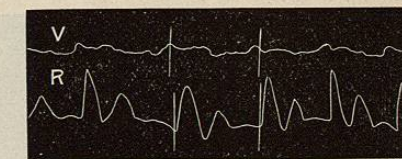


FIG. 3918.—Venous Pulse from External Jugular (above); Arterial Pulse from Radial (below).

cases it is difficult to decide from a tracing which we have to deal with. The finger, on the contrary, can recognize a distinct positive impact in the veins in the case of a ventricular venous pulse, whereas in the pulse of auricular paralysis no such positive impact is felt. Fig. 3919

shows a tracing such as we sometimes meet with. This is from an old woman with a dilated and irregular heart, but no murmurs. The venous tracing shows, as a rule,

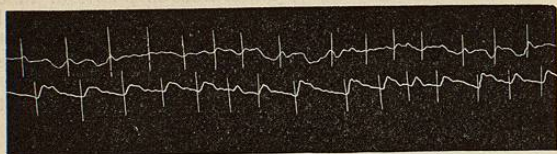


Fig. 3919.—Jugular Pulse (above), Radial (below). Corresponding points are marked.

the most pronounced collapse during diastole and resembles somewhat the ventricular pulse shown in Fig. 3918. There was no positive beat in the veins of the neck, however, and there were no heart murmurs, so I ascribed the condition to dilatation and threatening paralysis of the auricles without any serious amount of regurgitation. In this tracing a presystolic rise and systolic collapse are occasionally seen, so that the paralysis of the auricles was not absolute.

**THE VENOUS PULSE IN IRREGULAR HEART ACTION.**—A number of observers have recently been making use of the venous pulse as a means of deciding the primary seat of irregularity in the rhythm of the heart. One example will have to suffice. Fig. 3920 is a tracing taken from one of the cases referred to, in discussing the arterial pulse, of a father and son, both in good health and both with irregular pulses. This tracing is taken from the son. The venous pulse is small, as is usually the case

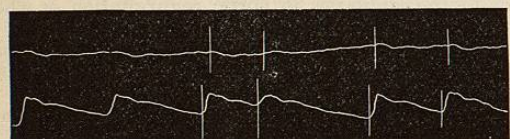


Fig. 3920.—Jugular Pulse (above); Radial (below). Corresponding points are marked.

in a healthy adult, but suffices for the purpose. An irregularity may be seen in both the arterial and the venous pulse of the nature of a premature beat. In the ordinary beats preceding and following the premature one, a faint wave may be detected in the venous pulse synchronous with the primary wave in the radial. This is the systolic wave. Just preceding the systolic wave a fainter one which is presystolic and due to the auricular systole may be seen. In the venous beat corresponding to the premature wave in the radial a systolic wave may also be seen, but the auricular wave follows it instead of preceding it, showing that the auricle in this case contracts after the ventricle, and therefore the anomalous stimulus causing the premature beat must have acted on the ventricle. If measurements be made it will be found that the pulse intervals on either side of the premature beat are together equal to the preceding and succeeding ones, or to two average pulses. This, according to Hering, Cushman, and

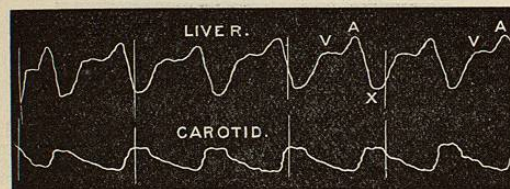


Fig. 3921.—Auricular Liver Pulse.

others, points to the auricle not being implicated in the irregularity. When the auricle is the primary seat of the disturbed rhythm, such a correspondence is not usually found. This rule is said by Gerhardt, however, to

be not without exceptions. The full importance of thus differentiating the seat of the irregularity is not fully worked out, but on the whole those cases in which the irregularity is confined to the ventricle are less serious than those in which the auricle is also irregular in its rhythm.

**THE LIVER PULSE.**—A pulsation can be felt and recorded in the liver in certain cases in which the right side of the heart and the veins are much distended. In some cases the tracing has the form corresponding to the auricular venous pulse. In these cases, according to Mackenzie, there are usually tricuspid stenosis and auricular hypertrophy, as the normally weak auricular waves have not force enough to make themselves felt in the liver.

In other cases the liver pulse has the characters of the ventricular venous pulse, and then we may be reasonably

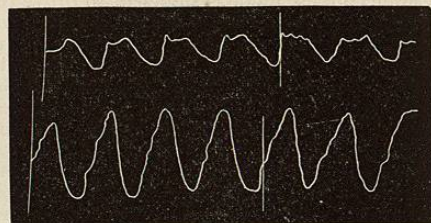


Fig. 3922.—Ventricular Liver Pulse. (Carotid above; liver below).

certain of the existence of tricuspid regurgitation. Tracings of these two forms of liver pulse taken from Mackenzie's book are shown in Fig. 3921 and Fig. 3922.

**CAPILLARY PULSE.**—This consists in alternate reddening and paling of an area of the skin with each heart beat. It is most frequently looked for in the bed of the finger nails, and may be brought out most distinctly by raising the arm. Quincke, who first described the capillary pulse, recommends rubbing gently a spot upon the forehead and looking for it there. The capillary pulse may be taken as an indication of aortic regurgitation with a strongly acting ventricle (hypertrophied).

William S. Morrow.

REFERENCES.

I desire to acknowledge my special indebtedness to the book on the pulse, by James Mackenzie, and warmly to recommend it to those desiring to read something more exhaustive than this article. I have also received help from the following: The Physiologies of Schaefer, Halliburton, Howell and Hall; Vierordt's "Medical Diagnosis"; Hutchison and Rainy's "Clinical Methods"; Gibson's "Diseases of the Heart"; Balfour's "Diseases of the Heart"; Fagge's "Practice of Medicine"; Green's "Examination for Life Insurance"; Ewart's "Heart Studies"; Hürthle's "Beiträge zur Häemodynamik" in Pfüger's Archiv, vol. xlix.; D. Gerhardt's "Klinische Untersuchungen über Venenpulsationen" and "Einige Beobachtungen an Venenpulsen" in Archiv für experimental Path. u. Phar., vols. xxxiv. and xlvii.; Karl Schmidt, Jr., "Herz-Kammer Systole und Pulsecurve," Pfüger's Archiv, 1902, Heft 5 u. 6; W. S. MacCallum, Johns Hopkins Hospital Bulletin, March, 1900; Cushman, "On Intermittent Pulse," British Med. Journ., September 29th, 1900. A good bibliography will be found in Gibson's "Diseases of Heart and Aorta."

**PUMPKIN SEEDS.**—*Pepo*, U. S. P. *Semen Peponis*. The dried ripe seed of *Cucurbita pepo* L. (fam. *Cucurbitaceae*).

The nativity of the pumpkin is not certainly known, though it was probably North American. It presents numerous varieties, and the squashes, at least some of them, have been regarded by some botanists as pertaining to the same species. Although squash seeds appear to possess similar properties, they are not included, as a drug, under the above title.

Pumpkin seeds are about 2 cm. (¾ in.) long, broadly ovate, flat, white, or whitish, nearly smooth, having a shallow groove near to and parallel with the edge; containing a short conical radicle and two flat cotyledons; inodorous; taste, bland and oily.

The active constituent is supposed to be a small amount of a soft, green, acrid, and bitter resin, which possesses the same properties as the entire drug. With this there

exists a yellow or somewhat reddish-yellow, bland fixed oil, to the extent of about thirty-five per cent., a little sugar, crystallizable albumin, and other unimportant constituents. The oil, which consists of glycerides of palmitic, myristic, and oleic acid, portions of which acids also exist in a free state, has been credited with the properties of the drug, but possibly, if pure, does not possess them.

Pumpkin seeds are markedly diuretic, but their medicinal use is as a pleasant and moderately certain tenicidic. Only the kernel should be used, and it is commonly given in the form of an electuary or emulsion, the dose amounting to from 25 to 50 gm. (¾ to 1 iss.). Fifteen grains of the resin is an equally efficient dose, though not so pleasant.

Throughout the West Indies, Mexico, Central America, and many other countries, pumpkin seeds, as well as squash seeds, are largely consumed as food.

Henry H. Rusby.

**PURGATIN.**—Purgatol, anthrapurpurin diacetyl ester, is an odorless, tasteless, yellowish-brown powder recommended by von Hösslin as an agreeable laxative. It acts slowly, requiring thirteen to twenty-four hours, and produces a copious, non-liquid stool. The urine is colored red. Dose, 0.5-2 gm. (gr. viij.-xxx.).

W. A. Bastedo.

**PURGATIVES, OR CATHARTICS,** are medicines which are used to produce alvine evacuations. According to their activity and power, they are divided into laxatives and mild and drastic purgatives.

Purgatives which act very gently, producing soft, feculent stools without notable irritation, are called *laxatives*. This term is also applied to more powerful purgatives when they are given in small doses, so as to act mildly. (See *Laxatives* in Vol. V.)

Purgatives which operate briskly, usually producing more or less fluid evacuations, sometimes with griping and tenesmus, but without serious irritation, are called *mild or simple purgatives*. To this group belong some of the salts of magnesium, sodium, and potassium, which, from their resemblance in chemical and physical properties, and in physiological action, are termed *saline purgatives*.

The term *drastic* is applied to those purgatives which operate energetically, producing numerous evacuations, and, in excessive doses, more or less gastro-intestinal irritation.

Purgatives which produce watery stools, especially the salines and some of the drastics, are called *hydragogues*, and those which cause the evacuation of large quantities of bile, *cholagogues*.

**MODE OF ACTION.**—All purgatives accelerate the peristaltic movements of the intestines. Radziejewsky carefully observed the rapidity of peristalsis in dogs, both before and after the administration of purgatives. In the normal state the movements of the small intestine were rapid, those of the large intestine very slow. After the administration of purgatives, the movements of both became much accelerated, but most markedly those of the large intestine.

It was assumed that purgatives, especially the hydragogues, also induce a discharge of fluid from the intestinal mucous membrane. Experiments on animals at first seemed to show that this was an error. Thiry completely separated a portion of the small intestine from the rest of the bowel, without dividing its vessels and nerves, sewed up one end, which was returned into the abdominal cavity, and attached the open end to the wound in the abdominal wall. Into the cul-de-sac thus formed he introduced croton oil, senna, and Epsom salt. No accumulation of fluid took place. Schiff experimented in a similar manner with aloes, jalap, and sulphate of sodium, and Radziejewsky with croton oil and sulphate of magnesium, both with the same negative result. Radziejewsky also analyzed the feces before and after the administration of purgatives. The evacu-

ations produced by purgatives contained more water and sodium salts than normal feces, and sometimes products of pancreatic digestion, but never as much albumin as should have been present if transudation of fluid from the intestinal blood-vessels had taken place. It was therefore concluded by these investigators, and is still maintained by some recent authors, that purgatives do not induce either transudation or increased secretion, and that the watery character of the stools results only from the greatly accelerated peristalsis, which interferes with the absorption of the fluid normally secreted.

But subsequent investigations yielded different results. Moreau introduced sulphate of magnesium into a portion of intestine isolated by means of two ligatures, and after some hours found a decided accumulation of fluid. Brunton, experimenting in a similar manner, found that croton oil, gamboge, elaterin, and Epsom salt caused a decided accumulation of fluid. That the accumulated fluid was not a transudation was evident from the fact that it contained very little albumin. Brieger injected into an isolated portion of intestine very small quantities of colocyth. No accumulation of fluid took place, but the bowel was contracted and slightly reddened. Larger quantities of colocyth, as well as croton oil, caused an accumulation of bloody fluid, with decided inflammation of the mucous membrane. After injecting calomel, senna, rhubarb, aloes, and castor oil, Brieger found the bowel empty and firmly contracted. Sulphate of magnesium in very dilute solution caused no accumulation of fluid, but concentrated solutions of this salt, so also Glauber salt, caused very decided accumulation. That the fluid was a secretion, and not a transudation, was evident from the fact that it readily converted starch into sugar and dissolved raw fibrin.

Thus it has been found in experiments that sulphate of magnesium, sulphate of sodium, croton oil, gamboge, colocyth, and elaterin, not only accelerate the peristaltic movements of the intestines, but also induce a secretion of watery fluid from the intestinal mucous membrane; and that castor oil, rhubarb, aloes, senna, calomel, and minute quantities of colocyth accelerate peristalsis, but do not notably increase secretion.

Hess, in experiments on dogs, endeavored to determine the manner in which purgatives increase the peristaltic contractions. He made gastric fistulae a short distance from the pylorus, so that he could easily introduce purgatives into the duodenum. After paving determined the quantity of the purgative (sulphate of sodium, castor oil, croton oil, senna, colocyth, gamboge, and calomel) which would act briskly, he introduced into the duodenum a small, empty india-rubber ball, to which was attached a long, fine india-rubber tube. After this had been carried by the normal peristaltic contractions a certain distance, which varied in the different experiments, he filled it with water to such a degree as to obstruct the bowel. The purgatives which previously had acted briskly then completely failed. Hess therefore concluded that the peristaltic movements excited by purgatives are probably not propagated through long distances by means of nervous apparatus, or, according to Engelmann, from muscle to muscle, but that they are reflexly excited in each part of the intestine by direct stimulation of its mucous membrane.

**MILD PURGATIVES.**—Of the purgatives which act vigorously, without causing severe irritation of the intestines, the following are commonly employed: aloes, rhubarb, senna, castor oil, salines, and mercurials.

**Aloes.**—In large doses, from five to twenty grains, aloes produces semi-liquid or liquid stools. The first evacuation rarely occurs before six hours, and often not before ten or twelve hours. Some griping usually precedes the evacuations, and they are often attended by a feeling of heat in the anus, and by straining, especially if the medicine be repeatedly taken. From the slow action and the tenesmus, it is supposed that aloes influences the rectum more than other parts of the intestines.

In experiments on rabbits, Kohn found that aloes caused moderate hyperæmia of the stomach, intestines,