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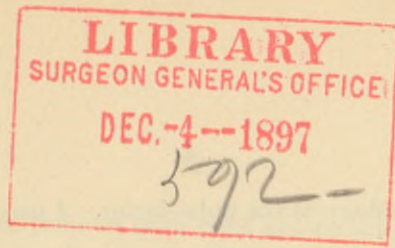


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A CLINICAL STUDY OF THE PULSE

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TO determine what treatment of the vascular system shall be adopted in any given case demands a careful review of all the possible signs that can be elicited by physical examination. The heart, its size and the relative size of its chambers, the large vessels at the base of the heart, the carotid, brachial and femoral arteries, the peripheral vessels, the undulations and pulsations in the large cervical veins, and even a study of the retinal arteries and veins may often be of service in making an eliminative diagnosis of vascular disease. After the clinician has satisfied himself as to the pathologic situation there is no field in medicine (barring, perhaps, syphilis and malaria) in which the therapist feels himself so sure of his ground, and there is no field in which he dare proceed with the administration of therapeutic agents to procure a definite effect with so much assurance as in diseases of the vascular system.

I wish this evening to call special attention to the tactile study of the pulse, and what that study may teach us regarding the pathology of the central and peripheral vascular organs. I fear this study has been much neglected by the modern physician. The medical men of the past century made very careful studies of the tactile pulse sensations, but their ignorance of the physiology of the circulation led them to apply terms of such mystic meaning to pulses of certain qualities, that in the beginning of the present century, when the physiology and pathology of cardiac diseases commenced to assume the form of a natural science, the ancient pulse-sensations were thrown aside with their misleading terminology. Clinicians since that time have been occupied chiefly with the study of the heart-signs, to such an extent that even in diseases of the heart's valves the pulse manifestations are very lightly treated. Think for a minute of what the pulse is a product, and how its various modifications may give us a clue that will lead to the

detection of the pathology of the pulse-factor. I use the term pulse-factor in its literal meaning.

The pulse of the radial artery, as we see and feel it, represents the correlative action of the myocardium, the four chambers, and valves at the auricular and arterial orifices; the elasticity of the aorta, and large and small vessels, and the muscular element of the arterial walls and their exquisite vasomotor equilibrium. Beyond and besides the vessels, the state of the capillaries and the specific gravity of the blood are all factors in modifying the regular movements seen in an artery accompanying each cardiac cycle. The qualities of a pulse, which it is my invariable rule to consider in examining a patient, are its rate, rhythm, regularity, volume, relative duration of anacrotus and katecrotus, any interruptions in the anacrotus or katecrotus, the compressibility of the pulse and nature of the arterial walls. These pulse-elements can be studied far better by the touch and sight than with the sphygmograph. What one cannot feel is often plainly visible at the bend of the elbow, in the *superficialis volae*, or a superficially placed radial or ulnar artery. A fortunate circumstance is, that in nearly all cases in which there is a departure from the normal, these pathologic elements make the pulse more plainly visible than in the normal state. A trained finger can palpate all of the pulse-phenomena that the sphygmograph can show, and generally we can see far more than either touch or instrument can detect. The excursion of the artery as a whole must be eliminated in purely instrumental studies of the pulse. The sphygmograph gives tracings from an artery in a fixed position, under a constant pressure, whereas the finger varies the pressure to accommodate the arterial excursion, thus enabling us to detect oscillations that a given pressure from the sphygmograph may mask completely. In feeling the pulse the most frequent error is in using too strong pressure, thus masking the fainter oscillations to the tactile sense.

I have made it a rule to make a graphic record of the pulse as it can be seen and palpated. These records I regard as better pulse-tracings than could be made with the sphygmograph. The recent work of William E. Ewart on pulse-sensations, or a study in tactile sphygmography is a beautiful demonstration of the superiority of the tactile method of studying the pulse. Merely a study of the radial pulse is not sufficient. There may be very marked changes in the carotid circulation and little to attract our attention in the brachial or femoral area. I have seen clinically marked arterial sclerosis of the brachial arteries and none in the carotids or femorals, and also advanced

sclerosis of the *dorsalis pedis* on both sides and no sclerosis elsewhere demonstrable. The following case shows very well the possibilities of regional arterial disease.

A man 40 years of age complained of severe headaches, lasting for two weeks. He was unable to sleep and was forced to remain idle because of the persistent pain. The radial artery was of moderate rate, regular, rhythmic, moderate volume, monocrotic, easily compressed; the arterial wall showed fibrous thickening. The aortic second sound was loud, high-pitched, ringing in character and distinctly palpable. Looking for the source of this accentuation, I found the carotid pulse entirely different in character from the radial; at the same time I was struck with the inconsistency between the carotid blood-supply and the marked pallor of the man. The carotid pulse showed with every cardiac systole a marked excursion, a distinctly visible primary ictus followed immediately by an oscillation high in the descending arm, so as to give the pulse almost the bisferiens type. The descent of the pulse was slow, decidedly that of the tardus type. Here was the key of the situation. The accentuation of the aortic second, the high interruption in the katacrotus, the long duration of the descending arm of the pulse and the pallor of the patient were due to one thing, viz.: increased peripheral resistance in the carotid distribution. Trinitrin, 1-100 grain in tablet form, was given every four hours. In two days the patient returned, saying he had been directly relieved from his headache. After taking the tablets he would be seized with a sense of fullness in the head that lasted only a few minutes, followed directly by entire relief from the headaches. The aortic second sound was no longer accentuated. The carotid pulse was scarcely visible. The peripheral rebound was gone. The pulse was more nearly monocrotic in character. There was a little more color in the patient's face but the change was not at all marked. The trinitrin was stopped in four days. Two weeks after the administration of trinitrin was stopped there had been no return of the headache and the change in the carotid pulse persisted. The radial pulse did not change its character at all during the administration of the trinitrin.

By pulse-rate we mean the number of beats in a given time. Pulse-rhythm is the constancy of the pulse with reference to the lapse of time between the beats. Pulse-regularity is the constancy of the pulse with reference to size. The pulse-volume is difficult to determine; indeed, it can only be compared within wide limits. The excursion of the artery is not a faithful

index of the quantity of blood forced into the aortic system with each systole of the ventricle. We may have marked excursion of the artery under high tension, and at the same time have bloody pulmonary edema from stasis in the pulmonary circulation. The actual enlargement of the arterial caliber during the systole is so slight that it is only within wide limits that we can make any relative observations. Our sense of volume of the pulse is controlled by the tension of the arterial wall during the heart systole as it is perceived by the finger. It is true that it is not simply the acme of tension but the extent of the oscillations accompanying wall-tension that we appropriate for our estimations of pulse-volume. This, too, may be exceedingly misleading.

I have on several occasions seen men with arterial sclerosis and increased peripheral resistance, suffering from dyspnea on exertion, and showing the following signs; slight enlargement of the heart to the left, strong apex impulse, loud systolic and loud diastolic tones; the arteries empty between the beats; a long anacrotus and strong ictus, followed by a long katacrotus; a capillary pulse visible in the finger-nails; the excursion of the entire artery, from the inner aspect of the arm to the radial at the wrist being plainly visible; the pulse-rate 70-80. Here are no evidences of valvular lesion, but of myocardial insufficiency attending physical exertion (that is, dyspnea), and at the same time an apparently large amount of blood coursing through the aortic system with every heart's systole. I put these same men on trinitrin 1-100 of a grain. In a few days they returned to the clinic much improved, and with no more dyspnea. The systolic and diastolic tones were much diminished in their intensity, the apex pulse not so strong. The artery on the inner aspect of the arm was scarcely visible during the heart's systole, the capillary pulse much diminished; the anacrotus was shorter, the ictus much fainter, and the katacrotus of much shorter duration, and still the heart's rate exactly the same. If we were left to judge of the relative pumping power of the left ventricle in these cases with only the character of the pulse to guide us, we would have to estimate the pumping power before the administration of trinitrin as having been greater, but in reality the pumping power must be greater after the effect of trinitrin is apparent. The number of heart-beats is the same and there is no dyspnea on exertion, which is sufficient proof that the left heart is more efficient under the lowered than under the high tension. The prevalent habit of describing a pulse as of high or low tension is not sufficiently accurate for careful observation; we may

have high tension, as under the circumstances above described, or we may have a very small pulse with high tension, as seen in cases of mitral stenosis. Here the left ventricle is not suffering, but the left auricle and right ventricle are bearing the brunt of the lesion, and there is no necessity for relief of the left ventricle by relaxing the arteriole system as in the case of mitral insufficiency. The word tension, as generally used, comprises two elements—namely, size and compressibility. Extremely low tension, attended with relaxed peripheral vessels, may be misleading as to the heart's working capacity. Often in the course of typhoid-fever the pulse would indicate an extreme impairment of the myocardium. The artery offers very little resistance to pressure of the finger, but there may be no dilatation of either side of the heart and no stasis in the pulmonary circulation. The anacrotus is lengthened when there is some peripheral impediment to the work of the left ventricle, and in cases of obstruction at the aortic orifice. The *pulsus bisferiens* I have seen only in aortic stenosis. One case which occurred at the City Hospital last summer showed, besides evidences of mitral stenosis and insufficiency and aortic insufficiency, also evidences of aortic stenosis in the form of *pulsus bisferiens*, which could be felt and plainly seen along the carotids and brachials and radials. The autopsy showed a rigid mitral orifice from calcareous deposits following old endocarditis. The orifice was much stenosed and insufficient. Two of the segments of the aortic valves were rigid with united edges projecting as a calcareous plate into the aortic orifice. The third segment was much shrunken from old endocarditis. The *pulsus bisferiens* is very unique in its character; once seen it can never again be overlooked. With each systole of the heart the pulse in the artery shows two sharper elevations at its summit, with a sharp depression between them of very short duration. These two elevations are due to the obstructing plates at the aortic orifice intercepting the expulsion of blood from the ventricle. The anacrotic pulse occurs in the so-called button-hole mitral and aortic orifices. The hypertrophied left auricle during its contraction throws a wave into the aortic system which is under a minimum tension during the cardiac diastole. The auricular pulse being directly followed by the stronger ventricular pulse gives a further elevation in the anacrotus. The anacrotic pulse and *pulsus bisferiens* may be strongly simulated in the carotid in high arterial tension when the primary ictus from peripheral rebound is met by the secondary wave of rebound following the closure of the aortic valves. The summation of these waves will sometimes give a second elevation higher

than that of the original wave, but the difference lies in the fact that in the anacrotic pulse and *pulsus bisferiens* the double summit occurs during the systole of the left ventricle and before the aortic valves have closed, whereas the second elevation under discussion requires the reflected wave from the closure of the aortic valves to complete its form. This is in reality dealing with the katarotus. I shall not occupy any time in dealing with the pulse of high tension as seen in arterial sclerosis, but will mention only a few of the changes in the katarotus that are apparently inconsistent with the pathologic causes. There are two signs in the arteries associated with aortic insufficiency which are referred to the low arterial tension as an essential condition for their occurrence. Their presence is often indeed regarded as a safe indication for the administration of digitalis. They are a tone heard over the large arteries during the cardiac systole, and the capillary pulse. These two signs are of all the more interest to me in view of the fact that recently, from Schröder's clinic in Vienna, there were reported several cases described as pseudo-aortic insufficiency having these two signs as a basis for the new terminology. I have had two men during the last two years who illustrate very well the causes of those two signs. The first, a man with dyspnea and pulmonary edema with marked auscultatory signs of aortic insufficiency. The pulse was of short duration, but to my surprise there was no capillary pulse. The radial artery appeared to be contracted on its volume of blood, offering resistance to the sudden arterial excursion which occurs in this disease. Under trinitrin the man returned in a few days with the dyspnea and cough much improved and a distinct capillary pulse. Another man, a dock-laborer, while working in the hold of a vessel, was suddenly seized with dyspnea, nausea and extreme weakness; he struggled with difficulty to the deck and walked to his house, where he remained quiet for two weeks, but his strength did not return. He appeared at the clinic, a large, well-developed, muscular man. The heart's apex behind the sixth rib in the nipple line, very loud, clear systolic tone at the apex and a loud diastolic tone over the aortic area, the aortic closure being distinctly palpable. The pulsations along the radial and ulnar arteries were distinctly visible. The brachial showed a high interruption in the katarotus, with a long descending arm. The arteries were empty between the beats. Taking the finger-ends in one's hand a very distinct expansion with every heart-beat could be felt. There was a capillary pulse distinctly visible in the finger-nails, and a loud systolic tone was heard over the femoral and brachial arteries.

This is a picture of what may be termed pseudo-aortic insufficiency, were there any ground for the introduction of such a term into medical literature. After taking trinitrin gr. 1/100 every three hours for several days the patient returned with an entirely different picture. The heart's impulse and tones were greatly diminished in intensity, the pulse was of much shorter duration, the ictus was moderate, the brachial pulsation was only slightly visible, systolic tone in the large arteries no longer audible and the capillary pulse only slightly apparent.

Here are two cases in which both were benefited by trinitrin; in the one a capillary pulse is made to appear when absent, in the other it is made to diminish when present, and both by dilating arterioles. The first case is perfectly clear. In the second case we have an increased muscular resistance from the arterioles, a moderate sclerosis of the large arteries and loss of elasticity of the aorta. A strongly contracting left ventricle forces its contents into an aorta that cannot expand to accommodate the new accession of fluid, nor do the smaller arteries, (because of their rigidity) absorb as much of the tidal blood-wave as they should, so that the strong myocardium forces its wave directly into the capillaries. Relax the arterioles, there is a larger reservoir offered for the reception of blood from the heart, and the force of the heart's contraction is so much diminished that though the large arteries may be as empty between the beats as before the use of trinitrin there is no systolic tone audible over them and the capillary pulse is much diminished. If the elasticity of the aortic wall be lost, the pulse in the radial artery can have only the duration of the heart's systole, thus making the katacrotus very short.

On the other hand hyper-elasticity of the aorta as it occurs in congenital small aorta is attended with a pulse of short duration in the large arteries and a loud systolic tone over the femorals, because of the rapid and complete emptying of the arteries during the heart's systole and diastole. In severe anemia following hemorrhage, (with marked hydremia) there is marked shortening of the katacrotus because of the vasomotor relaxation and the lowered specific gravity of the blood. There is an increased peripheral leakage which gives the same pulse modifications that a central leakage would give, for instance, in aortic insufficiency.

In septicopyemia attended with marked dilation of the peripheral vessels, the capillary pulse was so marked that it could be plainly seen by one standing at the bedside as the patient's hands were lying outstretched on the coverlet. In this same instance, too, the tone in the carotids, brachials and

femorals, accompanying the heart's systole, was as loud as ever I heard it in aortic insufficiency. The tone was like the so-called "pistol-shot" tone of aortic insufficiency. The sudden spanning of the arterial wall was distinctly palpable. In listening over the heart's apex and base there was a distinct reduplication of the first sound, the second element of which was arterial in origin. The autopsy revealed no disease of the heart or arteries.

In administering drugs to affect the vascular system, this question always arises, are we to relieve the heart's work by diminishing its resistance? Are we to stimulate the myocardium to renewed efforts? or is there some faulty metabolism, the products of which have a toxic effect on the myocardium, and should our attempts be directed in the line of inhibition of this faulty metabolism?

Nitroglycerin in tablet form is the most convenient method of diminishing the peripheral resistance. In the earlier stages of arterial sclerosis when the vasomotor reflexes are exaggerated and the clinical signs are those of increased myotatic excitability of the vascular system the results are often brilliant and not of short duration. One often sees arterial tension remain lowered and the heart's work much relieved for months without any treatment whatever. 1/100 grain four times a day is a safe dosage at the start, gradually increasing the number of doses until the desired result is obtained, or the throbbing headache forbids further use of the drug. If no effect is procured from the use of the tablets use the 1% solution in drop doses before abandoning the trinitrin. One patient I had under observation used five drops of the 1% solution every half hour for an entire week without any good or evil results. Nitrite of amyl however, inhaled in drop doses three times daily gave great relief.

An employee of the City Hospital whom I had occasion to examine over a year ago showed no signs of heart or vascular disease at the time, and had no illness whatever during the past year. Last fall the patient complained of severe precordial pain, palpitation and dyspnea. Examination revealed a rapid arrhythmic pulse of short duration, moderate volume and easily compressed, with no arterial thickening. The left border of the heart was nearly two fingers external to the nipple line, and the right border at the midsternum, the pulmonic closure distinctly palpable at the second interspace to the left of the sternum. There was a loud blowing systolic murmur over the apex and over the base of the heart to the left of the sternum. The pulmonic second was much accentuated and reduplicated. Digitalis and strychn-

nin had no effect. Trinitrin in doses of 1/200 grain caused violent headache without affecting the heart. Inhalation of a half a drop of nitrite of amyl gave immediate relief. The doses were repeated three times daily. Within a few days the pulse became perfectly rhythmic, the murmur disappeared. The heart's area of dullness extended from the nipple line to the left border of the sternum. The dyspnea disappeared and with it all evidence of increased tension in the pulmonary circulation. Since that time the patient has had several returns of her difficulty, each time being relieved by the nitrite of amyl. In the intervals the patient does her work as a cook, which makes no small demand upon her physical powers.

Another man, 23 years of age, a sailor, suffered for three years from severe pain under the left shoulder-blade, which always became more severe after exercise. He says his pulse three years ago had a rate of 48-50 a minute and was very strong. He has noticed that it has gradually been growing weaker and more rapid, and recently he noticed arrhythmia. The pain under the shoulder-blade is now constant. There is also severe pain in the precordial region and left shoulder and he has been much disturbed by growing nervousness.

The pulse-rate on December 5, 1896, was 100, of fair volume, short duration and easily compressed. There was moderate arrhythmia, slight irregularity and marked thickening of the arterial wall. There was no edema, dyspnea nor distention of the cervical veins. The hearts' apex is in the fifth interspace in the nipple line. There was no palpable thrill or valve-closure over the heart. Over the aortic area, at the second interspace to the right of the sternum, there was a low pitched murmur of moderate intensity during the systole. The aortic closure was high-pitched, ringing in character. There was no retinitis or papillitis, no albumin or sugar in the urine. A careful search for all the signs of aortic aneurism other than those above mentioned gave negative results. Trinitrin with strychnin caused considerable headache without altering the heart's action. Nitrite of amyl inhaled in drop doses three times a day caused the pulse to become rhythmic and regular, the pain ceased and the nervousness no longer disturbed the patient; the pulse-rate remained the same. Another similar experience with nitrite of amyl after trinitrin had been abandoned, occurred in an old lady with inelastic dilatation of the aorta, probably of syphilitic origin, as there were other marked symptoms of syphilis. These three cases point to some essential effect of nitrite of amyl on the coronary circulation. Although it is the routine practice

to give potassium iodid in arterial sclerosis, I have never been convinced in a single case that it modified the arterial circulation in any degree whatever.

The value of saline cathartics to relieve an embarrassed heart is so well-known that it will not bear a discussion at the present time. The use of a direct cardiac depressant never seemed indicated in any case I have ever seen. If nothing can be gained by diminishing arterial resistance or stimulating the myocardium or by the use of alcohol or opiates, I can see little prospect of benefiting a patient by depressing the heart's action.

Digitalis is credited with having the property of contracting the arterioles. Clinically I never ~~see~~^{see} any evidence of such action. I strongly suspect that this idea has originated in insufficient study of the pulse elements. Before digitalis is used one should consider the condition of the myocardium, the elasticity of the aorta and large vessels and the peripheral arteries. When there is no hope of favorably affecting the arterial circulation digitalis should be used to strengthen the action of the heart's muscle. Camphor subcutaneously in the form of camphorated oil is much used in Germany whenever there is an indication for a direct cardiac stimulant with a quick and temporary affect, but with us strchnin takes its place. That alcohol in infectious diseases is of great service in supporting the heart's action every clinician will concede. But my own clinical experience leads me to agree with deductions of the pharmacologists, that alcohol does not stimulate the heart directly but conserves its energy by inhibiting the metabolism of the body, thus saving the heart from the metabolic products of the tissues of the body in diseases which act as intoxicants to the heart's muscle.