THE CLINICAL USE OF THE SPHYGMOMANOMETER.*

BY

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ALTHOUGH Harvey announced the discovery of the circulation of the blood in 1628, our knowledge of the circulation did not advance materially until Carl Ludwig introduced the graphic method of studying the heart's action two centuries later. He fixed a float to a mercury manometer and arranged so that the movements of the float were recorded upon a revolving drum, and since this the evolution of instruments and knowledge has been rapid. Each instrument invented has added something to our knowledge —sphygmographs, polygraphs, electro-cardiographs, etc.— and it is an interesting paradox that the use of instruments of precision in medicine has a way (largely because of their educational value) of diminishing the necessity for their use. Thus the sphygmograph taught us many facts about the pulse which could be recognized subsequently without the aid of the instrument. The electro-cardiograph was necessary for the detection and explanation of auricular fibrillation, though we now recognize this condition without its use, and to some extent the same is true of the sphygmomanometer.

The pulse may be defined as a rhythmic variation of pressures in an artery. The highest pressure reached—the systolic—is the result of the output of blood from the left ventricle. The lowest pressure—the diastolic—occurs at the end of diastole and is the result of three factors: (1) the rate of blood flow through the arteries and arterioles into the capillaries; (2) the volume of blood in the arterial system; (3) the action of the muscular and elastic coats of the arteries on the contained blood.

The pulse pressure is the difference between the systolic and diastolic pressures. The most highly trained finger is incapable of estimating systolic blood pressure accurately, and cannot give any certain information about diastolic pressure; indeed, the routine use of the sphygmomanometer will quickly convince anyone that it is useless to expect any exact information about blood pressures from digital examination of the pulse, since the size of the vessel, the condition of its wall, and the force of the pulse may easily lead the finger to form quite an incorrect conclusion as to the pressures in the artery.

The best method of obtaining the blood pressures is by auscultation over the brachial artery, but palpation may also be used to determine the systolic pressure.

After reassuring the patient as to the painlessness of the procedure (this is important because fear is a frequent cause of a raised blood pressure) put the patient as flat as possible and apply the armlet to the upper arm well above the bend of the elbow. See that both the patient and the arm are comfortable and relaxed. Apply a stethoscope to the inner side of the bend of the elbow over the lower end of the brachial artery, and while listening pump up the armlet. At first there is no sound, but as the pressure rises a sound appears which is dull and indistinct at first, but later becomes loud and clear, and then as the pressure is raised further the sound gets softer and finally disappears entirely. Now let the pressure fall by just opening the valve on the apparatus; the point at which the sound first reappears is the systolic pressure; allow the pressure to fall further, and the point at which the loud clear sound becomes soft and dull is the diastolic pressure. The finger on the radial pulse first detects the pulse beat at a point just below systolic pressure as read by the ear, but the finger cannot estimate diastolic pressure. Often the patient can tell the diastolic pressure, for as the pressure in the bag is raised there is a point at which a sensation of throbbing in the arm becomes very evident, and this is at or very near diastolic pressure.

It is important to realize that an instrument only becomes really helpful when it has been used so often that its use is easy and its fallacies are obvious. In no individual does systolic or diastolic pressure remain constant: changes of position, exercise, food, or drugs produce changes of pressure which are more or less temporary. Excitement, with its accompanying cardiac frequency, always causes a considerable rise of pressure, so that it may be necessary to wait until the patient is composed before making an observation of blood pressure.

Calcification of arteries, even when considerable in amount, is of little importance as a source of fallacy in blood pressure estimations when more than two and a half inches of artery are compressed, for such a length of artery is never completely calcified. The normal artery offers but little if any resistance to compression, but when the artery wall is contracted to constrict the artery it may offer a considerable resistance. The best way to overcome this constriction is either to massage the limb for a short time or to repeatedly compress and decompress the limb with the armlet.

The first reading of a blood pressure is most likely to be fallacious, and if immediately subsequent readings show a lower pressure observations should be repeated until a constant reading is obtained. The lowest reading is most likely to be correct. In any case with a frequent heart action the readings are likely to be too high, and if possible blood pressure should be estimated during a normal heart rate.

A normal child of under 12 years of age should not have a systolic pressure of over 100 nor a diastolic pressure of over 75 mm. Hg unless it be very obviously in advance of its years in size or weight. In adults the normal systolic pressure varies between 100 and 160 mm. Hg and the normal diastolic pressure between 65 and 90 mm. Hg; and while pressure tends to rise with age there are many old men with quite low pressures. Again, pressures vary with physique, big powerful men having higher normal pressures than their physical inferiors, while in women the pressures are five or ten points lower than in men, age for age, and type for type.

At each heart beat the left ventricle forces blood into the aorta and so maintains arterial blood pressure, but the height to which this pressure rises depends upon the resistance of the arteries, arterioles, and capillaries, and the response that the ventricle makes to overcome this resistance. With a normal heart the ventricular response varies directly with the resistance—that is, with the diastolic pressure—and this fact is the keynote of physiological and pathological variations in arterial pressures.

When (under experimental conditions) the inflow into the heart is kept constant the output of the left ventricle is unaltered by changes in the heart rate or arterial resistance; but when the inflow varies the heart automatically alters its output to equal the inflow, so that in a given time the amount of blood entering the aorta equals that entering the right auricle. With a heart rate of 60 per minute the duration of ventricular systole is about three-eighths of a second, so that the heart works three-eighths of the day (nine hours) and rests five-eighths of the day (fifteen hours). G. N. Stewart calculates the daily work of the heart (ventricles only) at about 150,000 foot-pounds, and this is the work of a heart maintaining a systolic blood pressure of 120 mm. Hg. Prolonged increase in arterial resistance acts as a stimulus to the heart, increases ventricular volume, and, by giving the heart more work, causes hypertrophy.

The pulse pressure and the systolic pressure are raised by:

1. Excitement, nervousness, exercise.
2. Tonic contraction of the arterial wall such as occurs in asthma, migraine, etc.
3. Nephritis, acute and chronic.
4. Aortic regurgitation (patent ductus arteriosus, and arteriovenous aneurysm).
5. Organic diseases of the artery, senile fibrosis, and arteriosclerosis, although a raised blood pressure does not necessarily accompany arterial disease.
6. Hyperpiesis—the condition described by Sir Clifford Allbutt in which there is a primary rise of blood pressure.

The pulse pressure and the systolic pressure are lowered by:

1. Loss of peripheral resistance from relaxation of arterioles, such as occurs in all atrophic and wasting diseases—phthisis, cancer, acute fevers, especially pneumonia and typhoid.
2. Anaemia from any cause, loss of blood (haemorrhage from lungs, etc.).
3. Cardiac asthenia in Addison's disease and starvation.
4. Vagus overaction in fainting attacks, collapse, and shock.

But apart from the detection of abnormally high or low pressures the sphygmomanometer gives us information about the regularity of pulse pressure, and is therefore of

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the greatest assistance in the diagnosis of cardiac irregularities, and indeed it often renders the electro-cardiographic or polygraphic examination of the patient needless, as it enables us to make an exact diagnosis.

In auricular fibrillation—important because it is so common, because it often implies grave myocardial disease, and because in certain cases the condition is now curable by means of quinidine—the pulse is irregular in force as well as in time, and in the cases with unusually slowly acting hearts the irregularity may not be easily felt by the finger. But when the sphygmomanometer is applied the irregularity is at once obvious by the variation in the sound heard over the artery—a variation which makes it difficult to determine exactly either the systolic or the diastolic pressure, for as the pressure in the bag is raised the more feeble beats are cut out and the heart appears to be less frequent, until at a certain pressure only an occasional beat can be heard. There are several facts which may enable us to diagnose the condition without the use of any instrument at all: a markedly irregular heart in which the heart rate as counted at the apex is greater than that at the wrist, because the weaker systoles fail to open the aortic valves; or the association of marked pulse irregularity with the disappearance of a presystolic murmur; or the marked slowing of the pulse which usually follows the use of digitalis, and the fact that neither exercise nor atropine as a rule produces either a greater regularity or a marked increase in heart rate.

In the form of irregularity known as pulsus alternans the sphygmomanometer may enable us to detect the irregularity before it is obvious to the finger. Here the pulse is regular in time but the beats are alternately strong and weak. Occasionally there is a slight alteration in time also, and where this is present the longer pause follows the stronger beat. In some cases extra-systoles follow every normal beat, so that the pulse waves are felt as alternately strong and weak, but there is always a well marked irregularity in time, and the longer pause always follows the weaker beat; or, to put it in another way, the pulse is felt as two beats, the second of which is the weaker, and a pause. Except in the alarm that they occasion in the patient these extra-systoles are of no importance. The true alternating pulse is always indicative of a very serious condition; it is found in advanced cases of myocardial disease, in renal disease, and in advanced arterio-sclerosis; it is sometimes discovered in acute diseases such as pneumonia, typhoid, or influenza; and may be the only discoverable physical sign in patients with angina. In my experience this form of pulse irregularity always implies an early fatal termination of the condition in which it appears.

The pulsus paradoxus is the pulse irregularity which is commonly associated with pericardial effusions. In this irregularity there is a failure of pulse volume during inspiration, so that at each inspiration the pulse either disappears entirely or becomes reduced in size. A slight degree of this irregularity is produced by quite small effusions into the pericardium, but such slight irregularity may be quite imperceptible unless it is sought for by means of the sphygmomanometer. Although the pulse pressure appears to be quite regular to the finger in such a condition, when the armlet is applied and the pressure in it is raised to near the systolic pressure the sound will be heard to vary markedly in intensity, being louder in expiration and softer or absent entirely during inspiration. Pericardial effusion may be particularly difficult to detect, and this sign is often of assistance in doubtful cases. Conditions which interfere with the inspiratory negative pressure in the thorax can also produce this pulse irregularity. A large pleural effusion, whether of gas or fluid, may lead to the paradoxical pulse, and in the war this irregularity was fairly commonly found in men with wounds of the chest, especially those in whom there was a large wound which allowed free ingress and egress of air to the pleural cavity at each respiration. Some children also show a slight degree of this irregularity of pulse volume in association with the juvenile type of irregularity in which the heart rate quickens during inspiration and slows

In heart disease severe heart failure may take place without any noticeable change in blood pressure, for the heart will maintain the blood pressures at or near the normal even when its output per beat is insufficient to ensure an efficient circulation. As Sir T. Clifford Allbutt says, "the heart dies sword in hand." But sometimes we can recognize impending heart failure in changes in blood pressure. In cases of hyperpiesis, renal disease, or arterio-sclerosis in which there has been a high pressure for years, a fall in the systolic pressure without any change in the diastolic pressure is significant, and whether it be associated with an increase in distress, or with dyspnoea, or appear alone, such a change in pressures is to be regarded with apprehension. It has recently been shown that the higher the diastolic pressure the greater must be the pulse pressure for the same diastolic output per beat; so when a patient with a high diastolic pressure shows a fall in systolic pressure without any corresponding fall in diastolic pressure the output of his heart per beat is less, and, except there be a corresponding increase in pulse rate, his circulation is already beginning to fail. This fact shows the importance of reading the diastolic pressure as well as the systolic.

One of the well known signs of aortic regurgitation is the collapsing pulse, which was described by Corrigan ninety-two years ago—its peculiar character being due to the combination of a raised systolic pressure and a lowered diastolic pressure. In adults with aortic incompetence the systolic pressure is always above 120 mm. Hg, and the diastolic pressure is usually below 65 mm. Hg. Often it is very difficult to ascertain the exact diastolic pressure, as the pulse sound is loud even when the pressure in the armlet is at zero. Sometimes the sound heard over the artery is so loud that it resembles a pistol shot.

There is another curiosity in the arterial pressures in aortic disease which was first described by Professor Leonard Hill. In a normal individual standing up the arterial pressure in the leg at the knee is higher than the pressure in the arm at the elbow by the weight of a column of blood equal in height to the distance from the knee to the elbow; but when the normal man lies down, the pressures in arm and leg soon become approximately equal. On the other hand, in the great majority of cases of aortic regurgitation, when the patient is lying flat the pressure in the leg is considerably higher than that in the arm. The suggested explanation is that, in order to maintain a good supply of blood to the brain when the patient is erect, those arteries in which blood flow is retarded by gravity (those above the level of the heart) are completely relaxed, while those arteries in which the blood flow is assisted by gravity are in a state of tonic constriction, and that this spasm persists even when the patient is lying flat. In observing the leg pressures the armlet (or leglet) should be applied just above or just below the knee, and the stethoscope is applied either over the popliteal or posterior tibial artery. It is important to have a bag of sufficient width, as too narrow a bag gives high readings. The width of the bag should be about a quarter the circumference of the limb.

While this peculiarity is not invariably present in aortic regurgitation there is usually a difference of 30 mm. Hg between the systolic pressure in the arm and leg. The severity of the valvular lesion may be gauged by the pulse pressure—the higher the pulse pressure the worse the lesion; but if the diastolic pressure is 70 or more it is certain that the amount of the aortic regurgitation cannot be serious.

In hemiplegia from a cerebral haemorrhage the pulse pressure is always high, while in a thrombosis the pulse pressure is less than the normal for the diastolic pressure. If the hemiplegic is found to have a high and rising pulse pressure he is sure to die of his haemorrhage, or if the pulse pressure is low and falling he will die of his thrombosis. As an example of this statement I will quote a case, a man of 57, who had extensive arterial disease and a large heart. His blood pressure was 200/140. (Note that the normal pulse pressure for a diastolic pressure of 140 should be about 120 mm. Hg, while here it is only 60 mm. Hg, so that the systolic output of the heart must have been

accidents, and finally succumbed. The *post-mortem* examination showed a very extensive cerebral thrombosis.

In enteric fever there are two accidents which it is often difficult to differentiate—perforation and haemorrhage. In the former a rise of blood pressure occurs after the primary shock, but in the latter the fall of blood pressure is always very marked and prolonged.

In pneumonia a rise of blood pressure before the crisis is often the first sign of some complication, while a fall may indicate impending heart failure.

Finally, blood pressure observation may be of considerable assistance in cases of albuminuria, a high blood pressure indicating renal disease—either a nephritis or that type of renal damage which results from hyperpiesis or arterio-sclerosis. In pregnancy the sphygmomanometer may be especially helpful, for it is often difficult to decide how much importance should be attached to a small amount of albumin in the urine of a pregnant woman. The normal systolic pressure of a woman of about 30 of average physique is about 112 mm. Hg. A pressure of over 130 would suggest the need for careful and frequent examination, and a pressure of over 150 would probably indicate the onset of eclampsia.

I do not think that a blood pressure of over 160 systolic or 90 diastolic is ever normal, although there are cases in which the diastolic pressure remains about 100 for a time without any ascertainable cause, and then returns to normal limits. Excitement may cause such considerable rises of pressure that it is well to bear in mind the rule that a high pressure is of greater significance when accompanied by a slow pulse.

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CHRONIC INTESTINAL STASIS AND EPILEPSY.*

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IN the study of epilepsy it seems clear that whatever the actual exciting element the vascular system is the main source of the epileptic phenomena, either alone or as a carrier of toxic agents, and the work that has been done experimentally by the advocates of both views has been very considerable in the last twenty years. Cells cut off from their circulation, even momentarily, show a reaction towards acidity, and this acidity factor must not be lost sight of in the interpretation of the experimental results recorded by the mechanical and vascular theorists. Faradization, and epinephrin will also blanch pial vessels and produce epileptiform convulsions. The proteoses of Cuneo, to be presently described, have the same effect when painted on the cortex. Lastly, lactic acid given intravenously will also produce convulsive seizures. Acidosis, therefore, whether produced locally or remotely, would appear to play a considerable role in the production of the epileptic convulsion, and the work of Cuneo[1] during the last nine years at Genoa goes perhaps further than that of any other investigator to establish its importance.

Cuneo established the presence, in the blood of persons who had just had an epileptic seizure, of a substance which he identified with the proteoses, and this substance introduced intravenously into animals faithfully reproduced the epileptic phenomena. At the same time he showed that the urine of these persons contained an extraordinarily high percentage of organic acids. Other features were a retention of total nitrogen, an increase in the nitrogen content, and continuous acidity of the faeces. Comparative tests in epileptic groups with a protein-rich and with a carbohydrate-pure diet next gave unexpected results, the acidosis in the former group being reduced, with a corresponding improvement in the severity and number of the fits, whilst there was a percental increase in acidity in the second group, and the fits increased in number and severity. It was next established that the fault in the starch metabolism occurred in the first stage of conversion to the disaccharides, maltose, lactose, cane sugar, etc. Normally this takes place through the action of the amylolytic ferments, the subsequent conversion of maltose being effected by maltase ferment through hydration into glucose. The failure in the first stage resulted, in the absence of sufficient amylolytic ferment, in the production of a more or less intense concentration of organic acids—acetic, butyric, lactic, tartaric, etc.—and these, which should normally have been united with alkaline bases in the small intestine, passed in the epileptic into the blood stream and urine unchanged. The alkaline salts in normal individuals are oxidized—if ammoniacal through ammonium carbonate into urea, if sodium into sodium carbonate, and this is the vitally necessary salt for maintaining the alkalinity of the blood and tissues. Cuneo concludes from his work that this transformation of organic salts into sodium carbonate begins in the mucosa of the small intestine and is completed in the liver, whence the sodium carbonate is returned to the gut with the bile. Lack of bases favours the production of acidosis, while a sufficiency of sodium tartrate favours the activity of the liver ferments and the production of sodium carbonate. If sodium acetate or tartrate is introduced directly into the circulation it causes typical epileptic convulsions, and Cuneo was able to remove from the carotid artery of the animals used a substance which proved to be a proteose.

The source of these proteoses Cuneo found in the reduction of that fundamental constituent of all cellular nuclei, nucleo-histone, and in the case of the serum and leucocytes of the blood also this reduction takes place under conditions of acidosis due to the concentration of organic acids. In an acid medium he was able to demonstrate the reduction of nucleo-histone from both these sources into an innocuous acid and a toxic base having the chemical and biological properties of nucleinic acid and of proteose. Introduced intravenously in dogs the latter not only produced typical epileptic convulsions, but the *post-mortem* appearances in dogs dying in status epilepticus so induced were identical with those found in the viscera and the brain and its coverings in persons dying of proteosaemia (albumosaemia), and as described by Tanzi and Lugaro in their findings in patients who died in typical status epilepticus.

He believes that albumosaemia exists in epilepsy and that this is caused by an insufficiency of the alkali-forming function in the liver and the small intestine, so that the acid organic salts, which are transformable into sodium carbonate, do not undergo this metabolic change, but enter the circulation and cause a division under certain conditions of the nucleo-histone element and the nucleo-proteins into nucleinic acid and proteoses. These latter remain free and exhibit their convulsive action.

Localized acidosis in derangements of the circulation may also set free albumoses, or it may be other toxins, as in Jacksonian seizures; and in the convulsions associated with cranio-cerebral injury with scar adhesions, brain abscesses, circumscribed areas of encephalitis, brain tumour, cysts, general paralysis, cerebral arterio-sclerosis, the acidosis produced by interference with the vascular supply may, as suggested by Professor Osnato,[2] determine the release of proteoses locally in cells cut off from their circulation, and so excite the convulsions associated with these conditions. In general paralysis and arterio-sclerosis, and probably also in encephalitis, the vascular disturbances and the local cellular destruction processes in the brain elements are ideally combined to produce generalized convulsive seizures.

A little over two years ago it was my privilege to address you on the possibility of proving a close connexion between epilepsy and chronic intestinal stasis. More recently I have published a preliminary report[3] on a small group of epileptics treated on lines directed exclusively to the relief of the gastro-intestinal conditions found associated in these patients with the epileptic phenomena. To-day, thanks to Cuneo's researches, I find myself in a stronger position to