lose their seat of action. However, it is a common observation that arterial pressure, which drops by 25 or 30 mm. Hg at the moment of section, soon regains its former level, and vagal excitation is ineffective to lower it. Nevertheless, it may be that splanchnic tone is the point of vagal purchase, the secondary or subsidiary tone, which replaces it, being immune to vagal influence.

A more serious objection to accepting the view of transference of elements to the splanchnic trunk is that the vagal hypotensive reaction, abolished by splanchnic section, occasionally shows a tendency to restoration after a time, a fact which suggests fatigue as the cause of the phenomenon. But fatigue was almost certainly excluded in some cases where the phenomenon occurred. Another consideration is that trauma must be great when the splanchnics are cut. Slight traction or pressure on the intact nerves produces convulsive twitches, which indicate a very sensitive structure, and the shock of rupture may cause a depression of peripheral as well as central excitability. Whatever the explanation, splanchnic section, which gives rise to increase of the intestinal and gastric muscle contraction following pneumogastric excitation, causes, in a large proportion of experiments, a depression or even abolition of the vascular hypotensive reaction to this excitation. There are good reasons for the view that the pneumogastric nerve contains sympathetic fibres, and it must seem strange that it should receive these only to restore them to the sympathetic system for final distribution. While we cannot affirm that this is the case, we cannot assert that it is not so; further work is necessary to determine the point. The splanchnics were divided in the first instance with a view to demonstrating the possible transference to them of intestinal motor fibres from the vagus, since it had been found in previous work that excitation of certain bundles from the splanchnics caused increased intestinal activity. Splanchnic section, however, far from diminishing this activity, has an augmentor influence on it, eliciting spontaneous movements and increasing the effect of vagal stimulation. This observation points to the absence of motor communication.

It is important to ascertain the seat of the vaso-dilatation resulting from pneumogastric excitation, and efforts have been made in this direction by taking plethysmographic records of the kidney, intestine, spleen, and the limbs. Since the demonstration by Kuré, Hasama, and others of vaso-dilator nerves coming from different spinal segments it was thought well to test the limbs for enlargement, although it was unlikely that a vagal supply to them existed. The splenic and intestinal volume did not show any significant alterations; the variations in these parts were mostly passive in relation to blood pressure changes. The small portion of gut, however, which is introduced into the oncometer does not necessarily reveal what may be occurring in other parts. It is quite possible that a vagal control of mesenteric vessels may exist without a uniform distribution of vascular elements. The renal reaction showed some differences in individual animals. A marked vaso-constriction was observed after the vagal stimulation in two experiments. This was soon followed by enlargement of the viscus, but the renal spasm was synchronous with the fall in blood pressure, and still occurred when this was compensated. In two other experiments some degree of expansion of the kidney coincided with the depressor phase of tension, but a general feature of the renal change was alternation of expansion and contraction for some time after stimulation, more or less independently of the blood pressure. Tournade described a renal shrinkage as a result of pneumogastric excitation, which he ascribed to increased production of adrenaline. In the present experiments the latent period

of the early constrictor effect was too rapid to be accounted for in this way. The liver and stomach, of which the changes of volume were not recorded, remain as possible regions where the vascular change could occur.

It will suffice to state that the records obtained in the work and held over for publication provide clear evidence of the chief point in the contribution—namely, that there exist hypertensive and hypotensive fibres in the vagus trunk, the latter particularly being paralysed by ergot and yohimbine and unaffected by atropine. They are consequently to be considered of sympathetic type.

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THE SPHYGMOMANOMETER IN THE DIAGNOSIS OF CARDIAC IRREGULARITIES

BY

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The clinical differentiation of cardiac arrhythmias has been much simplified as the result of electrocardiographic studies of the mechanisms involved, but there are always certain cases in which a definite diagnosis is impossible without the aid of some means of graphic registration. It is probable that no method except the graphic will ever be devised which will serve to distinguish with certainty between auricular fibrillation and those cases of frequent extrasystoles (more particularly those in which premature beats of both auricular and ventricular origin are present) which give rise to a complete disorganization of ventricular rhythm.

The fact that pulsus alternans may often be recognized during the routine taking of the blood pressure is fairly well known. It deserves, however, to be far more widely appreciated. The sphygmomanometer is an instrument which is now as much part of the everyday equipment of the medical man as the stethoscope; and pulsus alternans is a portent of such gravity that no method by which it can easily be recognized should be overlooked. It is not common for the alternating beats to be so marked that they can be distinguished by even the most sensitive finger. The sense of hearing is far more delicate than the sense of touch, and the auscultatory method of estimating blood pressure provides a means of detecting quite small differences in the strength of the ventricular contractions.

It is common knowledge that the systolic pressure is remarkably inconstant, even in the same individual. Not only does it vary from day to day but from minute to minute and even from beat to beat. Other factors remaining constant, the maximum arterial pressure achieved by any beat will depend upon the strength of the ventricular contraction. Where such contractions alternate in strength there will be alternation of maximum (systolic) pressure. This is, as a rule, quite easily recognized with the manometer. A variation of 5 mm. of mercury

between the beats can certainly be recognized with ease and smaller variations might be distinguished if the possibility of alternation were borne in mind when the blood pressure was being taken. If alternate beats reach a different systolic pressure the audible number of beats will be halved as soon as the pressure in the cuff exceeds the lower of the two maximum pressures. When all the beats have been obliterated by inflating the cuff to a pressure higher than the higher of the two systolic readings the halved rate will again be heard on deflation until the pressure in the cuff falls below the lower of the two systolic readings.

The pulse tracing is the only other method which can be relied upon to detect alternation of the pulse, and this is not likely to be used unless the condition is suspected. Small differences in systolic pressure cannot be recognized by palpation, and the electrocardiogram may be normal in spite of alternation. Every cardiac case is (or should be) investigated with the sphygmomanometer, and if the possibility of alternation is not forgotten few cases, where this type of arrhythmia is persistent, should be missed. It must be remembered that alternated strong and weak beats may be due to extrasystoles alternating with normal beats. In such cases there will be a disturbance of rhythm which will not, as a rule, be difficult to recognize, although the halving of the pulse rate will be observed as soon as the pressure in the cuff of the sphygmomanometer is sufficient to obliterate the beats due to the extrasystoles.

A study of the pressure relations of individual pulse beats is sometimes helpful in determining the cause of other forms of arrhythmia. Auricular fibrillation can often be diagnosed with confidence by palpation of the pulse and apex beat. If the blood pressure is taken in such cases it is found that the systolic pressure is impossible to estimate in the ordinary way; the first beat that is heard on deflating the cuff is usually an isolated one, and it may be necessary to reduce the pressure in the cuff by 20 or 30 mm. before the number of beats heard bears any relation to the charted pulse rate. If the cuff is held inflated at the pressure at which the first beat is heard only two or three beats a minute may be heard. As the cuff is *slowly* deflated the number gradually increases, and it is possible to get some idea of the variations in systolic pressure between the biggest and smallest beats. The complete disorganization of ventricular rhythm is suggested by the fact that the *sound* of each beat varies, even though the pressure in the cuff is kept constant. And the number of beats which get through at a given pressure of the cuff bears no sort of relation to normal cardiac rhythm.

In some cases where frequent extrasystoles closely simulate the arrhythmia of fibrillation careful attention to the rhythm heard with the cuff of the manometer partially inflated may give a clue to the true source of the ventricular irregularity. Ectopic beats of ventricular origin do not as a rule disturb the dominant rhythm of the heart. These beats can be excluded by raising the pressure in the cuff to a point just below the pressure generated by the normal beats. The equal spacing of the remaining (normal) beats can then be recognized with a fair degree of certainty. The method cannot be as accurate as measurement on a pulse tracing, but I have been able, on several occasions, to diagnose correctly the presence of extrasystoles in patients who have previously been thought to be suffering from fibrillation, and in whom palpation of the pulse and apex beat had certainly suggested the latter condition. Where the ectopic beats are of auricular origin there is usually interference with the dominant rhythm, and the distinction is less easy if the abnormal beats are very frequent. But in many cases the extrasystoles seem to generate a fairly constant pressure. For example, a patient may have a systolic

pressure of 160 mm., but the abnormal beats may sometimes be shown to appear in the brachial artery only when the pressure in the cuff is between 120 and 130. If one series of beats remains fairly constantly at one pressure and another series appears to lie between pressures not more than 15 mm. apart the second series are likely to be extrasystoles rather than irregular responses to a fibrillating auricle. In the latter condition the systolic pressures resulting from individual ventricular contractions are much more variable, and in this way the two conditions may sometimes be distinguished. It must not be expected that all cases can be distinguished by the method described. Ectopic beats are sometimes very variable in the pressures which they generate; in the course of half a minute there may be ventricular contractions which fail to exceed the aortic diastolic pressure and so fail to give rise to a pulse beat at all, while others may exceed the diastolic pressure sufficiently to give rise to a pulse beat whose pressure is not very far short of the maximum pressure of the normal beats. But if careful attention is paid both to the rhythm and to the actual pressures of abnormal beats I believe that valuable information may be obtained.

This method of studying the pulse is possibly well known to cardiologists, but it does not seem to be mentioned in their works, except in so far as it applies to the detection of *pulsus alternans*. Anything which simplifies clinical investigation and eliminates the necessity for employing elaborate apparatus is clearly useful, and I have therefore ventured to draw attention to a method which has proved of value to me in the routine investigation of blood pressure in patients suffering from cardiac irregularities.

CHOICE OF OPERATION FOR CATARACT ON A PREVIOUSLY TREPHINED EYE

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It is well known that cataract is frequently met with in association with glaucoma. Sometimes the patient shows evidence of the former condition when he first presents himself to the surgeon. At others, the opacification of the lens follows an operation for the relief of tension. In certain cases it is obvious that the glaucoma operation has been, to some extent at least, responsible for the occurrence of the cataract, for the opacification may spread from the points where the iris has adhered to the lens capsule, thus suggesting that these synechiae are in some way responsible for the commencement of the trouble. In others the first signs of opacity are seen in the depth of the lens and are obviously independent of any interference with the capsule, though the suggestion that the lenticular nutrition has been affected cannot unhesitatingly be set aside. Whatever may be the cause of the cataract, and whether it is primary or secondary, the consequent failure of vision may often demand the removal of the lens.

Such an operation is bound to be fraught with anxiety, for added to the many risks that are always associated with cataract extraction, the surgeon is confronted with others peculiar to the condition under discussion. The cataract is rarely fully mature, the optic nerve has probably been damaged to some extent, and the interference with the circulation which was associated with the glaucomatous attack has left the eye in a condition less able to stand the strain to which a cataract operation necessarily subjects it.