the bright ones, it will be a valuable confirmation of my hypothesis as to the causes which produce a new star, namely, the collision of two meteor swarms. On this supposition, the spectrum of Nova Aurigæ would suggest that a dense swarm is moving towards the earth with a great velocity, and passing through a sparser swarm, which is receding. The great agitation set up in the dense swarm would produce the dark line spectrum, while the sparser swarm would give the bright lines.

In taking the first photograph, I was assisted by Mr. Fowler; the second was taken by Messrs. Fowler and Shackleton. Mr. Baxandall is responsible for the determination of the wave-lengths of the lines, and Mr. Shackleton for the determination of relative velocity.

II. "Contributions to the Physiology and Pathology of the Mammalian Heart." (From the Cambridge Pathological Laboratory.) By C. S. Roy, M.D., F.R.S., Professor of Pathology, and J. G. Adami, M.A., M.B., Fellow of Jesus College, Cambridge. Received December 31, 1891.

(Abstract.)

Our communication begins by stating that we have sought to study the action of the Mammalian heart in conditions (unexcised and intact) as nearly approaching the normal as we were able to make compatible with the employment of exact methods of research. This is followed by a general consideration of the difficulties attendant upon such a study, and of the means by which these difficulties may be overcome.

Under the heading of Methods we describe a *cardiometer* which we employed to measure the contraction volume and the "output," as well as the changes in the volume of the heart other than those due to its rhythmic contractions and expansions. A description is also given of the method of employing it, together with a statement as to the degree of the accuracy with which, according to our experience, the instrument supplies information regarding the changes in the volume of the heart. We then describe an automatic counter, which we employed for measuring out and recording the output of the heart, as obtained by the cardiometer.

This is followed by a description of our *myocardiograph*, which we made use of to record the contractions and expansions of any part or parts of the ventricular and auricular walls without interfering with the movements of the heart. In most cases we employed this instrument to obtain simultaneous records of the contractions of one auricle and one ventricle. We state also our doubts as to the

value of observations made on the heart by "button" cardiographs.

Section III begins by a consideration of the relationship between the circumference of a hollow spherical muscle and its cubic contents, this being illustrated by a diagram, and by one or two concrete examples with regard to the bearing of this subject upon the physiology of the ventricles.

We then state the relation between the internal circumference of a hollow spherical muscle and the resistance to contraction of its walls. Reference is also made to the elastic resistance which the heart wall itself offers to contraction, and the bearing of this upon the production of negative pressure within its cavity under certain conditions.

We then consider briefly the effect on the ventricular contractions of changes in the blood pressure within the systemic and pulmonary arteries, pointing out how much the heart has in common with the voluntary muscles of the body, and explaining why the amount of residual blood is liable to changes, concluding with a few remarks upon "failure of the heart."

In Section IV we enter upon a study of the effects of the vagus nerve upon the heart. We begin with the changes in the contraction volume, and point out that, at first sight, our curves seem to show that, other things being equal, the volume of blood expelled at each systole varies in inverse ratio to the rapidity of heart beat. We show, however, that this general law does not hold good for vagus slowing (if, indeed, it be exact for slowing of any kind), which is found to be accompanied by a lowering of the output; that, with moderate slowing, this diminution of the output may be as much as 30 or 35 per cent.

We then speak of the increase in the amount of residual blood in the heart which is produced by vagus excitation, showing that this does not necessarily indicate any weakening of the ventricular contractions.

We next analyse myocardiographic records of the action of the vagus upon the heart, showing that the auricular contractions are weakened or arrested, and noting that the influence of the vagus upon the force of the auricular contractions bears no constant proportion to the vagus slowing. By strong vagus excitation or by muscarin the auricles may be completely arrested, it may be, for hours. This complete arrest is, in some cases, led up to by progressive weakening, but sometimes arrest occurs immediately after fairly strong beats, or with fairly strong beats presenting themselves at times during the arrest. These latter cases may be explained by weakening of the excitations which reach the auricles from the sinus, although they are possibly due to diminished excitability of the auricles.

On coming to the effect of the vagi upon the ventricles we find that the distension of the heart during vagus actions is due to the ventricles being more expanded, both in diastole and in systole. We point out that the increased volume of the heart at the end of systole is a necessary result of the increased contraction volume, and combat the conclusions of those who ascribe it to weakening of the ventricular contractions, pointing out that the greatly increased contraction volume increases to a corresponding extent the work done at each contraction. We give detailed reasons for concluding that this suffices to explain the apparent diminution of the ventricular contractions.

We then examine the influence of the vagus upon the tonus of the relaxed ventricles, and point out that the great distension during vagus action is due entirely to increased intra-ventricular pressure during diastole, and not, as has been asserted by some, to any change in the elasticity of the relaxed ventricular wall.

Next, we consider the cause of the rise of venous (systemic and pulmonary) pressure, and find that this is due not to any increase in the amount of blood entering the veins in a given time or to contraction of their walls, but that it is to be ascribed to the diminished inflow into the ventricles.

The cause of this diminished inflow into the ventricles leading to corresponding diminution of the output is twofold, namely, weakening or arrest of the auricles, and, secondly, the elastic resistance of the ventricular wall to distension. We show that this explanation must apply to both sides of the heart, and that observed facts correspond with it.

We then consider the after-effects of vagus excitation, and show that the temporary increase in the output which is sometimes present may be explained by a temporary increase in the force of the auricular contractions, and by the venous pressure taking some little time to fall after the vagus excitation has ceased.

After this, we examine the influence of the vagus upon the heart rhythm, and show that, when the vagus excitation reaches a certain degree (varying in different animals), the ventricles begin to beat independently of the sinus and auricles; that this rhythm, which is at first slow and irregular, gradually becomes fairly rapid and almost completely regular.

This rhythm, we show, must be looked upon as the same as that which, as Wooldridge and Tigerstedt observed, makes its appearance when the ventricles are severed from the auricles. We point out, however, that the independent ventricular rhythm of vagus action is characterised by the slowness with which it establishes itself.

This characteristic is due to the lowering of the excitability of the ventricles produced by vagus action, and we adduce a considerable

number of facts showing that the vagus *does* lower the excitability of the ventricles, and that, by means of muscarin and by discontinuous stimulation of the vagus, it is possible to isolate the influence of the vagus on the rhythm and force of the auricles from its influence upon the excitability of the ventricles. The power of the vagus to stop the ventricles temporarily can only be explained by this diminution of their excitability.

We show that, with a certain degree of vagus excitation, irregularity of the ventricles necessarily results, in consequence of the sinus and the ideo-ventricular rhythms interfering with one another; that this is the common cause of irregularity; and that irregularity may also be caused by the auricles not responding to all the impulses which reach them from the sinus.

We explain that, in rare instances, direct excitation of the vagus may so lower the excitability of the ventricle that the contractions may not extend over the whole of their walls, and may in this way produce the apparent weakening which is sometimes met with.

In Section V we pass on to study the effect of direct excitation of the *nervi augmentores* (*accelerantes*) upon the heart, and show that the acceleration of the rhythm may be extremely slight if the heart be beating fast, and that the acceleration and augmentation of force of the heart bear no constant proportion to one another. The augmentor nerves increase the diastolic expansion of the auricles and also increase their systolic contraction; but these two effects do not go hand in hand.

Excitation of the augmentors increases the output of the heart, owing to the increased force and frequence of the auricular contractions, the result of this being that the pressures in the systemic and pulmonary arteries rise, while the systemic and pulmonary venous pressures fall. If there be but little quickening, the contraction volume of the ventricles is increased.

The augmentors, on direct stimulation, cause a slight increase in the diastolic expansion of the ventricles, which is passive in nature and due to the increased force of the auricular contraction. The force of the ventricular contractions is increased; they contract more completely, diminishing the amount of residual blood, in spite of the fact that the arterial pressure is usually somewhat raised.

There are certain nerve fibres other than the *nervi augmentores* proper which pass from the stellate ganglion to the heart, sometimes by the annulus of Vieussens to the inferior cervical ganglion, but sometimes as separate branches passing directly to the heart from the ganglion stellatum, or the annulus. On peripheral excitation of the cut nerves there is marked weakening of the contractions both of the auricles and of the ventricles, usually with some degree of slowing, this being sometimes followed on cessation of the excita-

tion by a very well-marked increase in the force and frequence of the auricular and ventricular contractions. They may be vaso-constrictors for the coronary vessels, although we give no proof of this.

There are nerve fibres which descend to the heart by the vago-sympathetics, which, on excitation under certain conditions, increase the force and frequence of beat of the auricles and ventricles, and which may be vaso-dilators for the coronary vessels.

Reflex excitation of the vagus produces results which are the same as those of direct excitation of the nerve, and the curves are more typical and satisfactory than those obtained on direct excitation of the nerve.

Excitation of a mixed nerve like the sciatic usually produces effects on the heart similar in kind to those due to direct excitation of the augmentors, but the phenomena are complicated by the greater rise of the pressure in the systemic arteries. Sometimes the increase in force of the ventricle more than counterbalances this increased resistance to contraction, and the amount of residual blood in the left ventricle is reduced; in other cases the increase in force of the ventricular contractions is not sufficient to counterbalance the increased resistance, and the residual blood in the left ventricle is increased.

In Section IX we show that excitation of the central end of a mixed nerve like the sciatic or splanchnic usually affects both the augmentor and vagus centres in the medulla, and that, in nearly all cases, the augmentor centre is the more strongly excited of the two, so that augmentor effects show themselves during the excitation, but are succeeded by vagus action on ceasing to excite the nerve. In many cases augmentor effects alone show themselves. When excited reflexly the augmentor centre ceases to act earlier than the vagus; the opposite, therefore, to what takes place with direct excitation. In rare cases the excitation of the vagus centre may be stronger than that of the augmentor from the first. Although, in the absence of any augmentor action, the vagus does not reduce the force of the ventricular systole, it does unmistakably have the power of inhibiting the strengthening influence which the augmentors exert upon the ventricular contractions.

In Section X, upon the part played by the vagus in the economy, we show that vagus excitation relieves the heart of work and therefore of waste to as great an extent as is compatible with a continuation of the circulation, and conclude that the vagus acts as a protective nerve to the heart, reducing the work thrown upon that organ when from fatigue or other cause such relief is required by it. The presence of fibres in the sciatic and other mixed nerves which cause reflex excitation of the vagus would seem to indicate that this nerve may be used by other parts of the body to diminish the out-

put of the heart and lower the blood pressure, thereby reducing the activity of the circulation as a whole. The influence of the blood pressure in the systemic arteries on the degree of vagus activity and the readiness with which the vagus centre is called into play by raising the intercranial pressure indicate that the vagus mechanism is specially employed in lowering the circulation so as to limit cerebral congestion. The vagus acts chiefly in the interests of the heart and central nervous system.

The power of the vagus over the heart is limited, and the ideo-ventricular mechanism, which comes into play when the vagus action exceeds a certain limit, must be looked upon as the means by which arrest of the circulation and death is prevented, whenever from any cause the nerve exerts a maximum influence. The power of the vagus to lower the excitability of the ventricles makes their temporary arrest possible, but this reduction of the excitability of the ventricles cannot be kept up, no matter how strong the stimuli applied to the nerve, for a period long enough to endanger the economy.

In Section XI we show that the function of the augmentor in the economy is to increase the work and tissue waste of the heart as part of the mechanism by which the nervous system governs the circulation, and that the augmentor mechanism sacrifices the heart in order to increase the output of the organ and enable the ventricles to pump out their contents against a heightened arterial pressure. Such excessive action of the heart is limited by the vagus, which, as we have seen, readily steps in so soon as the call for an increased supply of blood has ceased. It may do so earlier, presumably because the increased blood pressure or the fatigue of the heart calls for vagus intervention.

In Section XII we consider the mode of interaction of the vagi and augmentores; we point out that when the vagi are paralysed by section or atropin the augmentores have no control over the cardiac rhythm, and that therefore they can only act by inhibiting the influence of the vagi on the rhythmic centre of the heart. When neither nerve is acting on the auricles they contract with a certain force, which is increased by the augmentores and diminished or inhibited by the vagi. The force of the ventricular contractions is increased by augmentor action: this increase can be inhibited by vagus excitation, which latter has otherwise no power to reduce the strength of ventricular contractions.

The force of the heart's contractions is influenced by other factors than the vagi, augmentores, and other nerves. The pressure of the blood in the coronary arteries is one of the most important of these factors. If this be lowered, the contractions of both auricles and ventricles diminish in strength, while a rise of pressure in the systemic arteries causes an increase in the force of the heart's con-

tractions, so that the force of the heart's contractions is to a certain extent regulated automatically by changes in the blood pressure in the aorta, which is one of the variable quantities affecting the work of the left ventricle.

Change of the volume of blood in the body affects greatly the contraction volume and output of the heart. Injections into the veins of a volume of defibrinated blood equal to one-tenth of the total blood in the body may double the output. It is important to note here that there is no increase in the strength of the ventricular contractions; increase in the work, therefore, of the ventricles due to increase in the output has no tendency to automatically increase the force of the ventricular contractions, as is the case with rise of pressure in the systemic arteries. We refer to the bearing of this in cases of plethora.

Increase of the watery constituents of the blood increases the contraction volume and output to the same extent (though only temporarily) as does transfusion of blood, but acts more unfavourably on the heart, seeing that the work done by the ventricles is increased, while the nutritive value of the blood supplied to the coronaries is diminished.

The increased output of the heart both in plethora and in hydræmia is due to rise of pressure in the systemic veins increasing the volume of blood which enters the right ventricle during diastole. We refer to the bearing of these facts upon the treatment of chlorosis and heart disease.

In Section XIV we consider the limits of the power of the heart to perform the work thrown upon it, and show that in strictly physiological conditions, and in spite of the beautiful mechanism by which the force of the ventricular contraction is regulated, the heart, like the voluntary muscles of the body, is liable to fatigue when the work thrown upon it greatly exceeds that required to maintain the circulation under ordinary circumstances. We take as example the increased work thrown upon the organ during active muscular exertion, and show that exertion and endurance of fatigue are limited mainly by the limited power of the heart to continue supplying the increased amount of blood which is required by the acting voluntary muscles. We show that those luxuries which are forbidden or limited in "training," and which are known to hinder prolonged exertion, such as water, alcohol, tobacco, caffein, all directly weaken the force of the heart's contractions, and, in the case of water, place the organ under a disadvantage; also that fatigue of the heart leads to dilatation of the organ.

On comparing the power of fatigued ventricles to carry on increased work, as compared with well-nourished unfatigued ventricles, it is found that not only is the strengthening effect of the augmentor

nerves upon the individual contractions less in the former case, but also that the fatigued and therefore dilated heart is *per se* unfavourably placed for meeting increase in the work thrown upon it. An explanation is given of the reason why in heart disease failure takes place during exertion.

The part played by the vagus in protecting the diseased heart from harmful over-work is referred to, and it is shown that irregularity of the heart in disease may be explained by the mode in which this nerve, when acting powerfully, releases the ventricles from the control of the rhythmic centre in the sinus. The chief forms of rhythmic and arrhythmic irregularity are considered, and it is shown that these correspond with the forms of irregularity which can be produced by vagus action. The irregular heart expends more energy, and its tissues therefore are more wasted, for a given amount of work than the heart which is beating regularly.

The effect upon the heart of imperfect aëration of the blood is, first of all, to produce powerful vagus action from the medullary centre; this is usually, though not always, accompanied in curarised animals by diminution of the output of the heart. But reasons are given for assuming that the output would be increased in uncurarised animals, owing to the high venous pressure which results from struggling. Besides the vagus action, it can be shown that asphyxia causes progressive weakening both of the auricles and of the ventricles, and attention is drawn to the fact that the considerable rise of pressure in the systemic arteries in asphyxia is accompanied by vagus effects upon the heart, and not by augmentor action, as is the case, so far as we know, in all other instances in which the vaso-constrictor centre is excited in the normal individual.

It is noted that the change in the heart and circulation which takes place during asphyxia points to the conclusion that, when the total amount of oxygen in the blood is lowered, it is for the benefit of the economy that those organs, such as the central nervous system, whose continuous blood supply is a vital necessity, should be richly furnished with blood by constriction of the vessels of the spleen, kidney, and digestive system, whose blood supply can be cut off temporarily without danger to life, and also that the heart should carry on the circulation in a manner involving as little as possible waste of its own substance. This, as we have seen, it is the function of the vagus nerve to bring about.

III. "The Rôle played by Sugar in the Animal Economy. Preliminary Note on the Behaviour of Sugar in Blood." By VAUGHAN HARLEY, M.D. Communicated by GEORGE HARLEY, M.D., F.R.S. Received January 4, 1892.