

Cardiac form of American Trypanosomiasis

by

CARLOS CHAGAS and EURICO VILLELA

In the clinical picture of American Trypanosomiasis there are certain very constant and prominent symptoms which characterize the *cardiac form* of the disease. To give them firm foundation in fact and to avert the possibility of objections, our studies abound in decisive results derived from animal experimentation and human autopsies in which the constancy of the histopathologic processes and the presence of the parasites in the myocardium justify the interpretation of the principal features of the cardiac form.

In the clinical picture of this disease the lesions found in the cardiac muscle certainly represent the predominating factor; however, in a survey of the symptoms based on a large number of observed cases it is fitting to systematize the facts in two groups, one consisting of the cardiac changes of muscular origin and the other of changes associated with deficient nervous influences. These latter, which are attributable to the general pathologic processes of the disease, sometimes occur alone but usually are associated with changes of the former order. Here alterations of the cardiac rhythm occupy the chief place in the physical findings. Arrhythmia constitutes the predomi-

nant note in such cardiopathies, and in its various types are interpreted anomalies of the principal functions of the muscle. Furthermore, these types almost always succeed one another or complicate one another in the same individual. And this is not to be wondered at when one recognizes the progressive intensity and the diffusion of the pathologic process throughout the whole myocardium with its possible localizations in specialized functional zones of the organ.

For facility of description we must study the cardiac changes in the disease with the nature of the predominant arrhythmia. The functional changes of a general order, which give better ground for prognosis and so ought to form the criterion for a clinical classification, do not show appreciable variations but rather a relative uniformity in the different patients, and this prevents them serving for the basis of sharply delimited pathologic groups. Under these conditions we will study the following groups of alteration of rhythm:

1. Total tachycardias and bradycardias (of sinus origin)
2. Alterations of conductivity, manifested by

- a. delay in the conduction of the contractile stimulus (increase of the space *ac* or P. V.)
 - b. partial block
 - c. total block (dissociation of auriculo-ventricular rhythm with persistence of ventricular rhythm)
3. Premature contractions:
 - a. auricular extrasystoles
 - b. ventricular extrasystoles
 - c. nodal extrasystoles
 4. Auricular tachysystole (auricular flutter)
 5. Paroxysmic tachycardia (auricular, ventricular and nodal)
 6. Complete arrhythmia (auricular fibrillation)
 7. Cardiac alternations

These are the alterations of rhythm of which we have well-studied demonstrative examples. Some of them, of great frequency, indicate the affection of the organ in a very high percentage of the infected. This is true, in the first place, for the extrasystoles which are the most common of the changes of rhythm and constitute the best clinical symptom for the estimation of the endemic index of the disease. And the most frequent of the arrhythmias are those of conductivity, which among the many clinical features of trypanosomiasis represent that which is exclusively and peculiarly due to that disease. This is one of the great pathologic curiosities of the new disease entity, and in this chapter we find abundant valuable material for exemplifying and fundamenting the best theories of heart block, and for interpreting in the future, perhaps with greater certainty, the points that have remained open to discussion in this group of arrhythmias.

Other alterations of rhythm, associated like the two first with lesions of the muscle, are less common and we have only a few cases of them; in spite of this they are of value to characterize the sequence of the pathologic process and to better fundament the admitted pathogenesis. Among these are auricular tachycardia (auricular

flutter) and auricular fibrillation. Why are they less common in this immense accumulation of morbid statistics with the most varied forms of arrhythmia? Why is the auricular fibrillation especially less common when in the opinion of all the cardiopathologists it is the most frequent persistent irregularity of the human heart, represented perhaps by fifty per cent. of the clinical cases. We believe that in trypanosomiasis both are manifestations of the most advanced inflammatory processes of the myocardium and are the expression of a terminal condition rapidly followed by death. And, as a matter of fact, in the cases of this kind that we have observed up to the present time lethal termination occurred in a short time in the advanced conditions referred to.

Let us now see what are the appearances of the different forms of arrhythmia in the disease.

1. *Total tachycardias and bradycardias (sinus arrhythmia).*—The number of clinical cases of trypanosomiasis is high in which the cardiac rhythm is permanently accelerated or retarded, without the interference of accidental factors which could cause those anomalies. Usually these tachycardias and bradycardias are accompanied by extrasystoles of variable frequency; however, cases exist and are not rare in which protracted examinations do not show the presence of extrasystolic contractions, which are surely widely separated and even absent during long periods so that it is difficult to detect them. In this way the acceleration or slowing of the pulse with a regular succession of the pulsations predominates among the cardiac symptoms.

It must be emphasized that in such cases the auricles and ventricles pulsate at the same rate so that there is not here a sino-auriculo-ventricular dissociation.

How is the pathogenesis of these arrhythmias to be interpreted and how are their relations with the disease to be determined? Let us examine the clinical facts commencing with the tachycardias.

These tachycardias predominate in the female sex and there are a large number of patients in which the number of heart beats remains permanently between 100 and 200 or more per minute with regularity in the succession of the cycles or with the presence of extrasystoles. In these cases simultaneously with the acceleration of the heart beats there occur other symptoms which indicate the pathologic processes of the disease.

Furthermore, two large systems pertaining to the vegetative life, the thyroid and the genital, which play important parts in the physiologic equilibrium of the organism, show appreciable anomalies which are manifested by constant or very frequent syndromes in the patients of this group. The thyroid is almost always hypertrophied and the generative functions show marked changes which are expressed by intense dysmenorrhea with predominance of metrorrhagias. However, symptoms of hyperfunction of the ovaries constitute the principal genital syndrome, in which the considerable increase of the catamenias and the appearance of supernumerary menstrual periods characterize the functional derangement of the ovaries.

As regards the thyroid the facts are more complicated and the majority of the patients represent types of dysthyroidism with almost constant predominance of glandular hypofunction, but with isolated features of hyperthyroidism.

From this anomaly there must result physiopathologic consequences which are associated with either the function of one of the systems or with the disturbance of equilibrium in their interaction. Does the tachycardia observed constitute a part of such consequences? And does it indicate a hyperthyroidism? We must report that in men tachycardia is rarely observed although the anatomic alterations of the thyroid are of extreme frequency, and this indicates with all certainty the influence of the female genital apparatus in its mechanism. If the hypothesis of an endocrine influence on

the chronotropic function of the heart muscle be admitted, and its importance even exaggerated, it would still remain to determine the exact process by which that influence is exercised in the cases we are discussing. Is there an increase in the tonus of the sympathetic attributable to the functional disturbance of the thyroid, and in consequence the acceleration of rhythm? But we must observe that in the cases of this kind, at least in the majority of them, there is found ovarian hyperfunction, manifested by its classical symptoms. Now, in accordance with the modern doctrines the ovarian hormone exerts a depressive action on the sympathetic, and this fact may contradict the interpretation just formulated. However, in spite of this, in the tachycardias observed we are enabled to exclude extracardiac interferences of a nervous nature associated with the endocrine processes. In the first place the sinus region can be attacked by the pathologic changes involving all the myocardium, and from this would result a greater excitability of the node of Keith and Flack. This by itself is capable of either causing the increase of the contractile stimulus (and in this case the tachycardia would be of intramuscular origin) or of modifying too much the normal influence of the sympathetic, in this way occasioning the acceleration of the heart beats. Furthermore, and in the second place the possible interference of the suprarenal in these chronotropic changes must be admitted, since this is a frequent site of localizations of the parasite and of histologic changes, and its functional disturbance may act upon the cardiac rhythm.

And even this is not all of the difficulties in the interpretation that we are seeking, since those arrhythmias may be independent of extracardiac nervous factors and may indicate only the weakness of the muscle, thus representing the functional manifestation of the pathologic processes found there.

The complexity of the subject is evident and only protracted investigations can

later define the exact nature of the chronotropic disturbances in this disease, whose complicated pathology at times forms a decided obstacle to the interpretation of certain symptoms.

As regards the bradycardias of sinus origin we cannot escape the same lack of decision in attempting to interpret their mechanism. Do they indicate extracardiac influences in the form of deficient stimuli of the endocrine organs on the nervous apparatus that regulates the chronotropic function of the organ? Or do they rather indicate a disturbance of cardiac origin and manifest the weakness of the altered muscle? It should be emphasized that in these cases of bradycardia, as in the former case, the insufficiency of the organ becomes appreciable in all of its symptoms, which in a way may give a basis for the last hypothesis; far be it from us, however, to argue on this subject which should be reserved for later explanations. But we must refer to its predominance in males and this is an important factor to be considered in the necessary pathologic interpretation. Usually these bradycardias, as well as the tachycardias, are accompanied by extrasystolic contractions; cases exist, however, in which they constitute the only anomaly of rhythm.

The slowness of the pulse is very variable, oscillating in the vicinity of fifty beats per minute and is rarely seen below that figure.

Now let us pass to consider the arrhythmias that are indubitably related to the lesion of the myocardium. In the multiplicity of their varieties and in the frequency with which they are seen in nearly all of the clinical cases, these express a pathologic condition that belongs exclusively to this disease and they show the greatest curiosities in this new chapter of human pathology.

2. *Alterations of conductivity.*—The most curious and most characteristic aspect of the cardiac affection is that furnished by the alterations of conductivity, seen in all its phases of evolution from its slightest

grades, which are manifest by simple delay in the conduction of the contractile stimulus from the node of Keith to the ventricle, up to complete block with independence of the sino-auricular and ventricular rhythms.

The number of clinical cases with this symptom that has already been verified is considerable, and we may say with all certainty that the knowledge of American trypanosomiasis in this particular has opened a new field in cardiac physiopathology with valuable indications, in which to the abundant concrete facts there is added the appreciable advantage of a known etiologic unity. In other diseases these alterations have been found; in none, however, with the frequency here recorded or with the manifold aspects and stages of development that can be encountered in trypanosomiasis. To such an extent is this true that we might characterize this entity as the disease *par excellence* of alterations of rhythm and especially of slow pulse.

We have found these alterations in all epochs in life, including even children of eight years of age, and here they do not constitute an appanage of advanced years, as is the rule, but depend exclusively upon lesions of the primitive cardiac bundles. These regions are attacked by the diffuse pathologic processes characteristic of trypanosomiasis as much as the rest of the muscle; however, perhaps on account of their anatomic differentiation and greater functional importance there seems to be in these regions a preponderance of those processes which are manifest in the alterations of rhythm that we are discussing, and in others associated also with anomalies of the primitive bundles.

In the beginning the alteration is shown by the increase of the spaces *ac* and *PR*, which indicates the delay in the passage of the contractile wave through the normal paths of propagation. The increase of *ac* in many of our tracings reaches 0.6 and in rare cases has exceeded that time. The cardiogram and the radial tracing are sometimes regular; in the majority of the patient

however, the occurrence of extrasystoles comes in to complicate the arrhythmia and make the pulse irregular. In the jugular tracing the height of some or all waves constitutes a frequent anomaly and is attributable to the fusion of the ventricular wave of one cycle with the *a* wave of the following cycle. The number of cardiac pulsations depends on the sinus rate while the passage of the contractile waves is taking place and is not influenced by deficiency of conductivity.

In the next later phase of arrhythmia partial heart block appears, indicated at first by the periodic and spaced failure of transmission of some of the contractile waves and later by the greater frequency of the interrupted waves with establishment of a relation between the number of auricular and ventricular systoles (usually this relation varies between 3:1 and 2:1).

The immediate reason for the appearance of spaced failures of transmission of the systoles in the cases of increase of the space *ac* is variable and could only be appreciated by an analysis of the tracings. Often those failures indicate exclusively the exhaustion of the conductivity and no other factor is of influence; frequently, however, the gradual delay in the transmission of the systoles, causing the delay of the refractory phase of the ventricle, renders ineffective contractile waves which otherwise would be effective and could complete the cardiac cycle. And still other factors may intervene here. The same reasoning is applicable to the most frequent alternation of the relation of 3:1 to that of 2:1, or vice versa, and only concrete cases lend themselves to explanations in this way.

The last stage of these chronotropic alterations is that of complete block with a permanent idioventricular rhythm. We already have a large number of cases of this group, all more or less uniform in their clinical manifestation and in the course of their symptoms. When one considers the etiologic unity of the syndrome and takes

into account the short space of time that has sufficed for us to collect so large a number of observations in a scattered population, the high scientific interest of this chapter of cardiac pathology becomes abundantly evident.

The number of radial pulsations in cases of complete block oscillates in the vicinity of thirty and sometimes reaches, but rarely exceeds, forty. We have seen a rate below the first figure in some patients, almost always on taking more or less fatiguing exercise. And the progressive diminution of the pulsations to five per minute was observed in one case of death from exhaustion of conductivity.

In some patients it has been possible to accompany the progressive evolution of the arrhythmia from its commencement, with at first only a delay in the conduction, followed by the periodic and rare failures in the passage of the wave, and then the establishment of a fixed relation between the complete cardiac cycles and the blockades, and finally the complete block and consequent presence of the true ventricular rhythm. It must be noted that this last condition is the most persistent and the others represent in this disease only transitory passing phases of the ever advancing process. Some of our patients with complete block have survived the arrhythmia for a considerable period of time; in the majority of them, however, death has occurred as the result of the affection.

Among the most notable characteristics of the tracings in these cases is the frequent irregularity due to the presence of extrasystoles. And, indeed, the heterogenetic pulsations of ventricular origin form a symptom frequently preceding the disturbances of contractibility, which indicates previous changes of the whole muscle or of the primitive bundles scattered through it and localized later in the conducting regions. The simultaneous tracings of the jugular pulse, heart beat and radial pulse show here variations, all occurring more or less along the

general lines already known in similar cases of whatever etiology. Only the study of the concrete facts could furnish interesting pathognomonic data. Other subjective and objective symptoms of the cases of block are of great value; however, let us reserve them for later explanation and pass now to the study of the extrasystoles.

3. *Extrasystoles.*—The function of excitability is attacked in the majority or perhaps in all of the chronic cases of American trypanosomiasis. The patients with extrasystoles are almost counted by the number of infected, and such is the frequency of the symptom that we use it with profit as the best factor for estimating the endemic index of the disease.

Extrasystolic contractions are seen from the least advanced ages to old age and the number of children in whom we have found this alteration is quite high, and this is also a condition peculiar to trypanosomiasis. However, they are not found in the acute cases of the disease in spite of the intense lesions of the myocardium in the initial phases of the infection when the parasites are found in great abundance in the heart muscle.

It is clear from this that the extrasystole indicates rather a process of diffuse myocarditis and is more closely related to fibrous changes in the muscle than to the acute inflammation that is found in the early stage. As regards its origin these heterogeneous pulsations can be referred to the ventricle, to the auricle and to Tawara's node; here, however, as is general for this symptom, the ventricular extrasystoles predominate and are observed in a very high percentage relatively to the others. The auricular extrasystoles are somewhat rare, also those of nodal origin.

The inconstancy of the symptom in some cases should be emphasized, and this often makes its observation difficult and may lead to wrong conclusions from its absence. Some factors, whose intervention must be profited by in doubtful cases, especially in-

fluence its appearance. In the first place is the attitude; the extrasystoles are frequent principally in the dorsal decubitus, and many patients who show regularity of the pulse in the vertical position present extrasystolic contractions when lying down. Next come effort, fatigue and emotion. Often we only succeed in finding the symptom by causing forced movements of the patient or by producing sudden emotions. And in spite of the interference of the factors referred to the individuals are not rare in whom the extrasystoles are periodic, and only appear from time to time, with intervening phases of regular rhythm.

The frequency of the symptom in different patients or in different phases of the same case is very variable. Sometimes the extrasystoles are separated and only appear at long intervals in the tracings; others, however, present themselves with great frequency, being repeated in every cardiac cycle or two in the same cycle, giving the tracings the classic appearance of bigeminism and trigeminism.

It is of greater importance to note the prolonged sequence of extrasystoles with absence of compensating rest between the interposed beats, present in the beginning and end of the group, and this is characteristic of the crises of symptomatic paroxysmal tachycardia in this disease. We have various observations of this symptom which show the occurrence of the attack at the moment when we were taking the tracings; however, it is possible to judge of its frequency from the histories given by the patients with extrasystoles, who, as a rule, refer to transient crises of palpitation with rapid beating of the pulse and appreciable and very inconvenient contractions of the heart.

Taking into consideration the moment of the diastolic phase of the cycle in which the extrasystolic contraction appears, we must emphasize here the presence of interpolated extrasystoles in many cases. Relatively rare in the alterations of excitability

in general, these are here somewhat common, indicating the exaggerated irritability of the heart muscle. No common description of the tracings of extrasystoles would be possible, and in each one of them we find various factors which show the extreme variability of this symptom and its frequent association with other alterations of rhythm. We here show some very demonstrative analyses of tracings.

The subjective symptoms that occur in the course of this alteration of rhythm, or consequent upon it, are of decided interest and will be referred to in the following paragraphs.

4. *Auricular flutter.* The alterations of rhythm manifested by auricular flutter and auricular fibrillation are among the rarer observations, but are of the highest scientific interest. We have various cases with alterations of this nature and in them the essential characteristics of these forms of arrhythmia are well shown. Auricular flutter has been observed in various degrees of intensity with the number of auricular contractions varying between one hundred seventy and five hundred per minute. This last figure is one of the highest yet registered in the medical literature and was seen in a case that soon passed to the condition of complete arrhythmia. The number of auricular waves transmitted to the ventricle has always been variable in the different patients that we have observed, and no fixed relation has existed between the rhythm of the auricle and that of the ventricle within the limits of the tracings. This has given rise to irregularity of the radial pulse and this fact also shows the high grade of heart block of the cases of flutter in this disease.

Extrasystoles are also a somewhat frequent simultaneous occurrence in the cases that we are discussing, and their presence also gives rise to a greater irregularity of the cardiograms and of the radial pulse.

Some of our patients exemplify the transition from flutter to auricular fibrillation, and

other cases show intermediate phases between the two alterations of rhythm.

6. *Complete arrhythmia.* The observations collected on complete arrhythmia have been relatively small in number considering the high incidence of changes of the heart rhythm in this disease. The cases investigated occur in more or less the classic condition of this syndrome, as can be seen from a study of the tracings. We see ventricular venous pulse with waves of variable appearance, complete irregularity of the radial pulse and of the cardiogram, and it is sometimes possible to see in the jugular tracing small undulations which indicates the fibrillar state of the auricle.

The two last forms of alterations of rhythm without doubt express the greatest intensity of the pathologic processes of the myocardium in the disease, and also indicate localisations of those processes in the auricles, whose irritability becomes thus manifestly augmented even up to absolute inefficiency of the respective systolic movements.

From the beginning the affection of the myocardium is most intense in the ventricles as is well manifested by the extrasystoles originating in these regions. And when the alterations of rhythm indicate marked lesions of the auricles the prognosis becomes grave, as is shown by the termination in death within a short time of all our cases with auricular flutter and fibrillation. And, on the other hand, we must emphasize the relative scarcity of observations of these two arrhythmias, a fact which is evidently at variance with the constant intense lesions of the heart muscle and with the great number of other alterations of rhythm. On the best of grounds we believe that the rarity of these arrhythmias is only apparent as they are difficult to be detected in clinical investigations on account of the extreme gravity of their prognosis. The patients affected with them survive only a short time or remain in extremely serious condition; and as they are kept to their houses they escape the oppor-

tunity of medical observation, under the conditions under which our work was done. Another interesting thing with regard to the cases of auricular fibrillation is the absence of valvular lesions, since these lesions, especially of the mitral, are the most prominent features in the present medical literature on this chapter of cardiopathology.

7. *Alternation.* Alternating pulse figures in many of our observations accompanying various of the arrhythmias referred to. Its greatest frequency is observed in the cases of extrasystoles where it characterizes the post-extrasystolic alternation and where it almost always represents a transitory condition, disappearing after a few cycles of dominant rhythm.

The alternation is rarely observed as an isolated symptom, and from what we have observed we cannot establish a relation, in the cardiac form of the disease, between this symptom and any certain prognosis of great gravity.

General symptomatology of the cardiac form.

Although extremely variable in the different patients the clinical symptoms of the affection of the myocardium in trypanosomiasis can be considered in a joint description, leaving aside the details and using only the principal facts or, rather, those that are repeated with the greatest frequency in the manifestation of the cardiac form.

The symptoms of insufficiency of the organ occupy the first place and are indicated by their usual features: low arterial tension, visceral congestion, dyspnea, fatigue on exertion, edemas, etc., all expressing the progressive exhaustion of the heart's activity. Of these symptoms the edema deserves special mention as regards its appearance.

Even in the cases of most intense affection of the myocardium we rarely find here the extensive infiltrations seen in Bright's disease. The edemas in these cases are relatively slight, doubtless on account of the

absence of renal changes, and on account of this and the intense toxic conditions the origin of the infiltration in this disease is essentially limited to the weakness of the heart muscle. The great generalized infiltration is only observed in some relatively rare cases of cardiac asystole. And even at the time of the death agony with progressive weakening of the myocardium the edema is not present to the extent of that of renal origin.

As a physical sign of great frequency we must refer to the increase in volume of the heart. This sign is observed in any of the groups of the arrhythmias referred to, and sometimes indicates the hypertrophy of the muscle and at others the dilatation of its cavities or the two conditions simultaneously. We must also refer to other cardiac signs, such as the murmurs of relative valvular insufficiency, the obscuring of the heart sounds, especially of the first sound, the alterations in intensity and amplitude of the apex beat, etc..

Other cardiac and circulatory physical signs occur with frequency and ought to merit attention. But let us consider some of the more characteristic subjective symptoms, especially those directly related to the different forms of arrhythmia or, better, to the lesions of the myocardium.

Avexame (anxiety, agony, *angor animi*). This is the expression by which the patients characterize subjective phenomena, doubtless of circulatory origin, which figure in the history of numerous clinical cases whatever be the form of the arrhythmia. There is no uniformity in the facts included under this denomination, and it would therefore not be possible to refer them to a single origin. The patients indicate in various ways the subjective phenomena that they suffer which constitute the "agony." Some complain of precordial anxiety, of a sense of constriction originating in the epigastrium or the precordium and ascending to the throat, where it is most intense and causes phenomena of dyspnea and oppression frequently followed by fainting and

transitory dizziness. Others refer to a condition of general *malaise*, with unpleasant perception of the heart beats, rapid or slow, and painful sensations in the epigastrium and larynx accompanied by respiratory difficulty, etc. Finally, a large number of patients only complain of the "agony" without being able to define or localize the sensations that constitute it.

In any case, what is the exact pathognomonic value of this expression? Must we find a relation between it and some definite cardiac mechanism, and so find its true pathologic interpretation? Certainly not. "Agony" means nothing to the student of symptomatology but everything to the patient. This word includes the most varied sensations, all of circulatory origin but without the uniformity necessary for including them in a joint definition. No doubt can exist with regard to the relations between such phenomena and the processes of myocarditis and the various forms of arrhythmia of the disease. The «agony» is no nosologic novelty; it only reproduces here on a large scale subjective symptoms that are well known in cardiopathology and are associated with the different cardiac arrhythmias.

In this disease those sensations are often related to the extrasystoles and indicate the circulatory results of the compensatory pause, either alone or associated with an unpleasant perception of the extrasystolic beating. They may further be due to the crises of symptomatic tachycardia, to the alterations of conductivity, and to the other arrhythmias observed. And even this is not all of the pathologic conditions of "agony", that is, of the more or less painful sensations referred to by the patient. We believe in the interference of lesions of the intracardiac sensory nerves and are planning investigations to clear up this point. And, furthermore, we must add that "agony", or the sensations that correspond to it, is often complained of in the absence of appreciable arrhythmias.

Palpitations form another symptom of great frequency in the cardiac form of the disease. They occur almost always in crises of short duration, appearing and disappearing suddenly, and they subject the patient to the most painful sensations. The palpitations are not always related to the accelerated rhythm of the heart; often they appear with a normal or even diminished number of heart beats and radial pulsations. This symptom expresses the unpleasant perception of the heart beats, and for this very reason is not related to the various types of arrhythmia but to the anatomic processes that determine them. The palpitations in the patients that we have observed come on without any determining cause, when they are in repose or in motion, excited by slight exertion or by sudden emotions. They also disappear without any immediate cause.

The characterization of the symptomatic paroxysmic tachycardia is clear, and is so much the more admissible since it has been possible in some crises of tachycardia to detect in the tracings the beginning or the end by an extrasystole.

Faintness forms another symptom of great frequency, complained of in various degrees, sometimes limited to simple transitory obscurity of vision and at other times more intense and accompanied by vertigo with loss of consciousness. In any of the forms of arrhythmia this symptom is observed, but its greatest frequency is related to the circulatory changes caused by extrasystole. In addition to the faintness syncopal and convulsive attacks occur as nervous symptoms of the cardiac form. These may be observed in various of the arrhythmias, indicating the deficiency of irrigation of the nervous centers caused by the arrhythmia. However, such symptoms are better characterized in the alterations of conductivity and here complete the Stokes-Adams syndrome.

In the patients with heart block the nervous symptoms are very frequent in any

of the phases of the arrhythmia, in the earliest as well as in complete block. And here, with the great number of cases that we have, we cannot confirm the greater intensity of such symptoms in the phases that precede the establishment of the idio-ventricular rhythm. Vertigo, syncopal attacks and convulsions have been seen in patients with partial and total block, perhaps in a little larger number of the first but in all stages of the arrhythmia.

At this point it should be noted that the number of ventricular beats in cases of complete block undergoes frequent variations, which are doubtless related to the alterations in excitability of the muscle. From forty pulsations to the minute, and sometimes more, the rate of the idio-ventricular rhythm passes to thirty or even less, and this may explain the frequency of nervous symptoms in cases of this kind.

Other symptoms could be given here if we wished to glean them from the great number of observations of the cardiac form that we have. We prefer, however, to at once treat of other subjects.

Course.

As regards the clinical course of the cardiac form we must state that this is the type of the disease that occasions the greatest mortality.

The death rate in the cardiac form is relatively very high; in spite of this, a large number of cases remain in a state of tolerance for many years, capable of carrying on their work, although the insufficiency of the heart is more or less marked. According to our observations, this can be found in nearly all forms of arrhythmia except those of flutter and auricular fibrillation, the prognosis of which we consider very grave and we do not believe that they can long survive. In a general way we can state that the course of the cardiac affection in this disease is progressive. Here there is no possibility of regression and the patients proceed more or less rapidly to a lethal termination.

Death from the disease.

SUDDEN DEATH

Sudden death is extremely frequent in the regions of endemic trypanosomiasis. We have had the opportunity of seeing it in several cases of the cardiac form at the place where we were carrying on our work and its frequency is made more impressive by the unanimous reports of the inhabitants of such regions where a large number of the families mourn the sudden death of one or of various members. The individuals frequently die in the fulness of their youth and in an apparent healthy condition during the phase of tolerance of the cardiac affection. Many of them die while engaged in their usual work without any immediate cause to explain the occurrence; but others die at the moment of an extra exertion, or of fatigue, or of some other accident that is capable of exhausting the deficient energy of the myocardium. Facts of this kind are numerous and plainly show the intensity of the pathologic processes of the disease. We do not know of another condition in human pathology that causes sudden death in so high a proportion of cases as does American trypanosomiasis.

What is the exact mechanism of this death? Can we determine it with certainty in a way to cover all of the facts? And will it be the same for all of the occurrences of this order? In the explanation of this mechanism we must note in the first place that sudden death is observed in any of the forms of arrhythmia, which in a way renders its exact interpretation difficult. On the other hand we must remember that usually the patients show different forms of arrhythmia simultaneously and that cases are rare that have one form of rhythm exclusively. And if this is so, the unity of that mechanism must be admitted once there is recognized the occurrence of the fact in one of the most frequent arrhythmias. Now, of these, arrhythmia by extrasystole is doubtless that which is repeated in the greatest number of patients associated with other

forms of alterations of rhythm. Can sudden death in the disease depend upon the extrasystoles and upon these exclusively? We must remember that the compensatory pauses of the extrasystolic contractions and their inefficiency in the propulsion of the blood certainly cause profound circulatory embarrassment that frequently determines the occurrence of faintness, vertigo, loss of consciousness and syncopal attacks, etc. In this disease, as our accumulated mass of observations on the heart amply demonstrate, the alterations of excitability of the myocardium are profound, and as a result extrasystoles occur sometimes very frequently, repeated two or three times in the same cardiac cycle. Hence in many cases profound circulatory alterations intervene in the nervous centers of the myocardium itself, and these may explain the sudden death. This is an hypothesis.

There at once occurs to us another hypothesis, also based on fact and perhaps more in accordance with cardiac physiopathology. Auricular fibrillation has been found in this disease and we believe it is not a rare process although relatively less frequent. While limited to the auricle this alteration does not impede the circulatory efficiency; but propagated to the ventricle, as it may be, there soon occurs a failure of the circulation and as a direct consequence rapid death. Is this true in the cardiac form?

Let us discuss it briefly. The frequency of ventricular extrasystoles and their repetition two or three times in the same cycle express an irritability of the ventricular myocardium, a condition very near to that produced experimentally and indicated by a fibrillar state of the muscle. One degree further in the pathologic process and the excitability of the ventricle will perhaps reach a condition similar to that of the auricle, with the terminal failure of the rhythm. Moreover, we have the opportunity to exhibit tracings in which the ventricular extrasystoles, up to seven in number and all

abortive, are repeated in the same cardiac cycle. The extrasystolic contractions here are so weak that they look like simple tremors of the muscle and are completely useless in the circulatory mechanism.

We must at once refer to MacWilliam's hypothesis which has formed the basis for our argument and in which sudden death in intense disorders of rhythm would be explicable by fibrillation of the ventricle.

DEATH IN AGONY (MORTE AGONICA)

Here with greater frequency the essential fact is the progressive insufficiency of the myocardium from which asystole results. This insufficiency is principally of cardiac origin, and the renal apparatus, whose functions are little if any affected in this disease, does not participate in its occurrence. In this way the greater number of patients of the cardiac form die, in acute or chronic asystole, from rapid dilatation of the ventricles, and others die from progressive exhaustion of the muscle.

Another aspect of death in agony is observed by the diminution of the idio-ventricular beats, with long diastolic pauses. We have observed one case of this kind in which the ventricular rhythm fell slowly to the minimum of five pulsations per minute or with diastolic pauses of twelve seconds.

These are the commonest conditions of death in the cardiac form.

We have only considered the facts of greatest frequency and have left aside other occurrences such as fatal accidents in children with symptomatic tachycardia, deficiencies of conductivity with partial block, profound alterations of contractibility, etc. Death may here result from various conditions aside from those most frequent ones, as is not difficult to understand when we know the intense lesions that attack the muscle.

Influence of atropine on the alterations of cardiac rhythm in the disease.

The results of the experiments performed by one of us relative to the action of

atropine on the arrhythmias of the disease have been very interesting. As the result of a large number of cases we have concluded that the dromotropic action of that remedy is very marked and that its habitual chronotropic action is very small and even unappreciable in many cases. Here in some way there occurs the physiologic dissociation of the fibers of the vagus, and the atropine acts predominantly on those that interfere in the conductivity of the cardiac muscle.

Several cases of complete block, others of partial block and many of extrasystoles were the subject of the experiment.

In complete block the dromotropic action of the remedy was nil and the independence between the sino-auricular and ventricular rhythms remained unaltered. In the same cases the chronotropic action made itself felt very slightly, and often in a negative sense, that is, by causing the paradoxical effect of diminishing the number of beats of the auricle.

The experiments relative to partial block were of greater interest. In this case the elective action of the atropine on the conductivity was notable. In the majority of the experimented cases the remedy regulated the rhythm, that is, normalized the deficient conduction. The chronotropic action was unappreciable or at times acted in a negative sense, diminishing the number of auricular beats.

How is the restoration of conduction by atropine in the cases of heart block to be interpreted? Must the nervous origin of the alterations of conductivity be admitted here? No. The muscular nature of such alterations is clear and is demonstrated by the direct proof of the lesions of the myocardium. Furthermore, the cases of complete block, which represent only a more advanced degree of arrhythmia and whose pathogenesis is identical to theirs, was not modified by the action of the atropine, which excludes the entirely nervous nature of the disturbance of rhythm.

No doubt could exist about the only possible interpretation of these results. The lesions of His' bundle make it more excitable and give rise to the increase of physiologic tone of the vagus with the consequent alterations of conductivity that can be corrected by atropine. If these lesions were more intense, the atropine would not act in the same way, for there the block is already independent of the nervous action and is associated exclusively with the pathologic process of the muscle.

Moreover, the influence of anatomic changes of the conducting bundle on the inhibiting effects of the vagus is demonstrated in the experiments on the action of digitalis, although in a sense antagonistic to that referred to the action of atropine.

According to Mackenzie, digitalis exerts no action on the conductivity of normal beats, but with the presence of lesions of the muscle that remedy, even in a therapeutic dose, increases the time of conduction of the sinus stimulus to the ventricle, eventually producing partial block. We know of no case reported of total digitalis block in man, and therefore the importance of one of our cases increases in which the action of crystallized digitalin determined the appearance of a total block with the nervous symptoms of the syndrome. When the use of the remedy was suspended, the total block was transformed into a partial one, and the radial pulse rose from 35 to 50. Simultaneously the nervous symptoms disappeared and other symptoms associated with the block diminished. However, in this case the action of the digitalis caused a considerable increase of the vagus tonus on the injured His' bundle. When the use of the remedy was stopped, the altered dromotropic function returned to its former condition.

It is, without doubt, less easy to interpret the paradoxical effect of the atropine on the chronotropic function, especially appreciable in the cases of partial block. Similar facts are reported in the medical literature in cases of alteration of heart rhythm; but

in these cases there is a lack of acceptable interpretations, nor do we possess any facts that can serve as a basis for hypotheses that can explain that phenomenon.

Although our experiments were performed on a large number of cases, we can state nothing decisive in regard to the influence of atropine on the extrasystoles. We can state nothing from the occurrence of negative cases which indicate the absence of any action of the remedy beside others in which the rhythm was regulated. Furthermore, the inconstancy of extrasystolic beats is well known, as they often disappear for long periods even in clinical cases in which their frequency is greatest.

Allorhythmias.

In the tracings of the cardiac form of the disease we have found anomalies of rhythm that are repeated regularly and periodically, thus giving uniform appearances which can be grouped under the denomination of allorhythmias. Various factors enter into their origin and hence the principal interest is trying to recognize the conditions that determine them.

The most characteristic of such appearances are the bigeminism and trigeminism that may result in the same case or in different cases from differences of cardiac rhythm, isolated or combined.

The bigeminism usually results from extrasystoles which are repeated in all the cardiac cycles and the trigeminism from two extrasystolic contractions; further, both the appearances may depend upon alterations of conductivity or upon those associated with extrasystolic contractions. Only the analysis of each tracing in concrete cases can determine the relations between the allorhythmia and the facts that cause it.

Aside from these we have met other appearances of allorhythmia in which the irregularities are periodic and associated with different conditions, such as auricular fibrillation, and the interposition of extrasystoles which succeed one another regu-

larly. Such facts are exemplified in some of our tracings and their semiology can there easily be seen.

The cardiac form of the disease being thus described in its essential outlines, the interest of this new chapter of human pathology is evident and the great pathologic curiosity of the new trypanosomiasis in this clinical phase becomes apparent. It is clear that the subject is far from being exhausted in this first summary description of the cardiac form; there is much here for further investigation in the interpretation of the symptoms and for an exact knowledge of other cardiac conditions of the disease.

Aside from the present clinical observations the description here written is based on the works of pathologic anatomy and histology begun by the lamented Gaspar Vianna and completed by our eminent friend Professor Bowman C. Crowell and our companion Magarinos Torres.

The clinical observations that have been presented represent a small, selected, number of the large group of cases that have been studied. However, by the uniformity of the cardiac syndrome made manifest by them, they show the etiologic unity of the morbid process localized in the cardiac muscle.

This condition of the disease, in which cardiac symptoms predominate over all others, is generalized in the zones of endemic trypanosomiasis, and it is there observed with a maximum intensity and extension, thus forming the clinical characteristic *par excellence* of the American trypanosomiasis.

The certainty of the etiology of all the clinical observations here presented cannot be contested, although in many of the patients neither the parasitologic diagnosis nor the necroscopic proof has been made.

In the cases in which necropsy was possible, already a considerable number, the symptomatology was well studied and was in every point identical to those here reported. This assures with absolute certainty and irrefutable scientific precision the etio-

logy and pathogenesis of the numerous clinical cases of the cardiac form that we possess. Furthermore, the localization of the parasite in the heart muscle is a constant occurrence in the infections by the *Trypanosoma cruzi* not only in man but also in laboratory animals as well.

On the other hand the more recent histopathologic studies of Dr. Crowell show lesions that are considered by him as characteristic of the action of the parasite on the myocardium in the chronic process.

It may also be stated that the studies of Dr. Crowell have shown well localized lesions in the primitive cardiac bundle, which explains the anomalies of rhythm shown by the physical examination.

Observation no. 1.

Cardiac insufficiency. Total bradycardia. Sinus arrhythmia.

J. P.—White, 21 years, male, laborer, single, resident at Santo Antonio da Lagôa.

The patient has always been strong and mentions no former ailments. For the last few months he has a feeling of fatigue and gets tired after exertion. He came however to consult us on account of nocturnal delirium. In appearance the patient is strong, muscular and well built. There are no subjective signs to be noticed. Cardiac area enlarged, apex beat in the 5th intercostal space, outside the mamillary line. Heart sounds quite audible, no murmurs. Pulse arrhythmic, the beats now slow, now fast, at times simulating extrasystolic beats. Number of beats per minute: lying down 50; standing 82. Tmx.=140. Liver slightly enlarged. Spleen not enlarged. Cervical and inguinal glands slightly enlarged. Thyroid gland hypertrophied.

Record no. 1.

The radial and cardiac tracings show that between the first and fourth cycle the interval diminishes; the diastolic pause lengthens suddenly from the fourth to the fifth

cycle and diminishes again gradually until the seventh. At the seventh the same succession of sudden lengthening of the diastolic phase with gradual shortening every four beats begins anew. The long diastoles are however not strictly equal nor are the short ones so that the pulse is very arrhythmic. The amplitude of the pulse wave is in proportion to the length of the preceding interval. On analysis, the venous tracing shows the same irregularity in the succession of the auricular waves; it is the auricular rhythm which is altered in the first instance. The *a c* interval remains normal and there is no change in the conductivity. The regular succession of long and short diastolic phases every four beats shows that the arrhythmia is of respiratory origin, although the curve does not register the oscillations of the respiratory rhythm. The pulse is that found in total bradycardia, 50 beats per minute.

Observation no. 2.

Total bradycardia. Spasm of the esophagus.

A. S., negro, 23 years, male, single, resident at Santo Hipolyto, near Lassance. Examined June 23, 1916. The patient came to the hospital on account of an *entalo* (spasm of the esophagus) which he has had for four years and which started after a fever which lasted about fifteen days. The patient has difficulty in swallowing both liquid and solid food; it "goes down with difficulty and stops in the throat"; the patient finds deglutition painful and often has spasms of pain after it. Sometimes while swallowing the food is regurgitated. The difficulty is not continuous, but rather periodic and irregular. At times the patient swallows both liquid and solid food easily, at others, he has to take a draught of water after each particle so as to be able to swallow it, and, at others he cannot swallow even water. Certain foodstuffs, such as beans and peppered food, seem to bring on the spasm whilst farinaceous ones do not. The pa-

tient retains his appetite, but feels burning in the stomach, which is allayed by draughts of cold water. For about a year now, he gets easily tired and has palpitation after making any effort. The patient also mentions a feeling of weakness and pains in the legs, and slight vague pains in the body. At times, when obliged to drink much water so as to swallow, he vomits his food.

The only former ailments the patient mentions are attacks of fever, the first of which appeared six years ago and the last about two months ago.

The patient is well built and strong looking. On both sides of the parotid region, there are soft masses about the size of almonds, which seem to be due to a hypertrophy of the parotid glands. The patient states that their size varies, increasing at times and diminishing at others. Heart not enlarged; apex beat 7.5 cm. from the mid-sternal line, the right margin 3 cm. from the same line. Heart sounds clear. The second sound increased and reduplicated at the pulmonary area. Pulse slow, 44 lying down and 52 standing, accompanying the heart beats. After slight exercise, 70. Katzstein test positive; Tmn.=75. Tmx.=115.

Liver not enlarged, not painful on pressure. Thyroid gland enlarged, with a cyst about the size of a walnut in the middle, and smaller local ones. Inguinal glands slightly enlarged. Nervous system negative. Intelligence of a low order.

An X-ray examination of the process of swallowing was made. A bismuth emulsion was easily swallowed, but corn mush containing bismuth was swallowed very slowly and stopped several times owing to a spasm of the esophagus, provoking a feeling of discomfort and pain. The spasm was stronger at the cardia; as the patient put it "the stomach does not want to let food in".

Record no. 2 and 2-A

1) Pulse as in total bradycardia, as may be seen by the jugular and venous tracings.

The *a c* interval not lengthened; the dominant rhythm regulated by the auricular waves. The venous tracing shows the *b* waves of Hirschfelder well. Sudden pressure on the eye-balls produces a stoppage of the pulse for 4:5 seconds.

II) It is also a total bradycardia tracing like the former one and shows the effect of slow ocular pressure. The pulse has become much slower and the retarding influence seems to make itself felt after the pressure has stopped. It is interesting to note that after the compression has ceased the ventricle goes on beating with its own idio-ventricular rhythm, independently of the auricular rhythm, which is slower and the waves of which make themselves felt at the systolic ventricular phase by very acute rises.

The auricular beats gain in speed gradually until they dominate the rhythm. The last beat in the tracing belongs to the auricular rhythm. The last but one shows a fusion of the *a* and *c* waves.

This shows that though the diminution of excitability produced by ocular compression acts on all the points of origin of the cardiac contractions, it has a much stronger influence on the sino-auricular ones than on the starting-points of the idio-ventricular rhythm.

Observation no. 3.

Simple tachycardia.

G. do N., negress, 30 years resident at Lassance.

Examined at

Previous history of no special importance; the patient states that she has always been well, and has had no disturbances other than those which go with gestation. She has three children one of which was born prematurely at seven months. She has been ill for three months with loss of appetite and a feeling of fulness in the stomach after even moderate eating; poor digestion. Is easily fatigued and has "ave-xame", agony in the heart and a feeling of

anguish. Palpitation with rapid beats, which condition sometimes lasts an hour and is accompanied by a feeling of agony ("avexame").

Record no. 3 and 3-A.

Taken at a crisis of palpitation. The tracing is perfectly regular only the rhythm is accelerated, 120 beats per minute. We were unable to follow the transition between this and the slower rhythm of 3 which accompanied the disappearance of the subjective sensation of palpitation and agony. Nor can we affirm whether the greater rate was brought on by auricular extrasystoles or by the simple acceleration of the normal rhythm.

Observation no. 4.

Tachycardia. Palpitation.

F. P., negro, male, 25 years, resident at Lassance.

The patient came to be treated on account of strong and uncomfortable heart beats and agony ("avexame"), and states that he often gets frightened without any reason. This condition is accompanied by palpitation and agony ("avexame") and causes him much discomfort; it has lasted for over a year.

Signs of slight cardiac insufficiency. The heart is not enlarged. Heart sounds clear, without murmurs, Thyroid enlarged, with moderate sized goiter. During palpitation the heart beats are frequent and violent so as to shake the thoracic wall; the respiratory movements are more frequent and the breathing anguished. There are no lapses of the pulse either at the time of the palpitation or in the intervals.

The tracing shows an extrasystole at the end of an attack of palpitation.

Record no. 4.

Taken at the final phase of an attack of palpitation. A ventricular extrasystole is shown, but it was not possible to take trac-

ings showing the real nature of the acme beats during palpitation.

Observation no. 5.

Asystole.

R. M., white, male, 17 years, resident near Lassance.

The patient suffers from palpitation, is easily fatigued and has attacks of dizziness almost always followed by fainting. He has become thin. No edema. Signs of cardiac insufficiency. Pulse arrhythmic, at times with heart beats in bigeminal and trigeminal groups. Heart beats 84, which is more than that of the radial pulse, 73, because not all the extrasystoles are shown in the pulse. Tmx.=105. Thyroid gland hypertrophied, specially the middle lobe. Many cervical glands.

This patient was put in hospital during a crisis of cardiac asystole. At the time there was generalised edema with intense dyspnea and visceral congestion. The patient died of cardiac collapse. The histopathologic examination made by Prof. CROWELL showed parasitic foci and intense lesions of the myocardium.

Observation no. 6.

Ventricular extrasystole. Cardiac insufficiency. Palpitation. Agony.

M. A., mulatto, 23 years, female, married, resident at Pirapora.

Previous history of attacks of intermittent fever, and a little while ago arthritis of the wrist after a gonococcal infection which was cured. For some time the patient has been feeling nervous excitability, palpitation and fatigue on exertion. Nocturnal dyspnea with palpitations. These symptoms have become more pronounced in the last fifteen days; she has also gastric pains, a feeling of sickness and abundant salivation. Palpitation with agony ("avexame") almost constant.

Precordial beats very violent, with very marked apex shock. Cardiac erectism. Apex

beat in the 4th intercostal space, 7.5 cm. from the midsternal line; right margin 3.5 cm. from the midsternal line; no increase of the cardiac area. First sound muffled; second reduplicated, with pulmonary accentuation. No murmurs. Pulse very visible in the veins of the neck. Pulse full and unstable, accelerated on any emotion. Extrasystolic arrhythmia. Number of pulsations varying from 96 to 85 when lying down, the extrasystoles being more numerous when the pulse is more rapid. Standing, the number of pulsations rises to 100 and after slight exercise to 108. Tmx=140.

Liver not enlarged. Spleen slightly enlarged. Thyroid enlarged, with slight exophthalmus. Peripheral glands not enlarged. Menstruation regular, generally accompanied by slight pains. There are signs of double ovaritis.

July 15, 1913. General condition without noticeable change; but the indefinite malaise, agony and following palpitation continue. 88 beats per minute. Tmx=130.

Record no. 5.

The radial tracing shows that extrasystoles interrupt the dominant rhythm frequently and that the latter is also variable; the *a* waves not being perfectly rhythmic. On the right side of the tracing the arrhythmia of the *a* waves and of the pulse are accentuated by the movements of deglutition; acceleration at the moment of swallowing and subsequent retardation.

The extrasystoles are ventricular with complete compensating period. This tracing was taken at a time when the patient complained of palpitation with strong feeling of agony ("avexame".)

Observation no. 7.

Ventricular extrasystole.

E. A., Syrian, 20 years, male, single, resident at Lassance for the last two years.

Examined July 20, 1910.

The patient is a native of Syria and has been in Brasil for two years. About three

weeks before being examined he noticed that his thyroid had become enlarged and felt uncomfortable. There were no other morbid phenomena, or fever.

Pulse regular, 64; the rhythm interrupted by extrasystoles. Slight signs of cardiac insufficiency, with positive Katzenstein test.

Record no 6.

The rhythm of the radial tracing interrupted by a feeble extrasystolic beat which according to the analyses of the jugular tracing seemed to be an interpolated extrasystole (marked *c*).

Observation no. 8.

Interpolated ventricular extrasystoles.

E. A., resident at Santa-Maria.

April 2, 1911.

Large goiter. Liver enlarged. Signs of cardiac insufficiency. 57 pulse-beats, when lying down. Tmx=100. Frequent extrasystoles.

Record no. 7.

Radial tracing rhythmic and regular, with extrasystolic interruptions. Slow pulse, 53 beats per minute. The jugular tracing shows that the extrasystoles are of ventricular origin. There are two extrasystoles to be seen in this tracing; the first rather anticipated the *c* rise coinciding with the *a* wave: the second still more anticipated the *c* rise coinciding with the ascending phase of the *a* wave. Auricular rhythm unchanged. The *a c* interval lengthened after the extrasystoles.

Summary: ventricular extrasystole, slow heart. At the extreme right of the tracing an interpolated extrasystole.

Observation no. 9.

Ventricular extrasystoles. Cardiac insufficiency.

M. R., negress, 30 years, resident at Santa Rita.

Signs of marked cardiac insufficiency; rate 82 per minute, with frequent extrasystoles. Tmx=110. Large goiter. Thyroid much increased in size.

This woman is the mother of the patient Geraldo, who was in the hospital with diplegia.

Record no. 8

The only remarkable thing about this tracing is the extrasystoles of ventricular origin.

Observation no. 10.

Ventricular extrasystole. No signs of cardiac insufficiency. Atropine test.

B. C., white, male, 44 years, married, farm laborer, resident at Trahyras.

Examined March 23, 1913.

Previous history as in the preceding case; only intermittent fever. This patient came to consult us on account of a strong facial neuralgia which had lasted for 5 days. He is moderately tall and well built. No subjective symptoms. Heart not enlarged. Heart sounds without noticeable alterations, with the exception of tympanism of the second sound in the aortic area. Pulse with numerous extrasystoles. Pulse 64 when lying down, standing 75. The atropine test accelerates the pulse and makes it more regular. Liver and spleen not noticeably enlarged. Thyroid enlarged, with a cystic nodule.

Records 9 and 9-A.

I) The radial tracing shows frequent extrasystolic beats, which interrupt the rhythm. The extrasystoles are ventricular, as may be seen in the venous tracing.

II) An hour after injection of 0,001 of atropine the pulse is perfectly regular and slightly accelerated.

Observation no. 11.

Ventricular extrasystole. Dilatation of the heart. Atropine not modifying the extrasystoles.

C., white, male, 13 years, resident at Muquem.

Examined October 24, 1913.

Previous history. Malaria with recent attacks of fever. This patient complains of gastric pains, a feeling of gastric fulness and sometimes has attacks of giddiness. He does not complain of dyspnea, palpitation or any other symptom. Heart enlarged, apex beneath the fifth rib in the mammillary line, 8,5 cm. from the midsternal line. First sound muffled. Second reduplicated. Mesosystolic murmurs in pulmonary area. Pulse 84 per minute, with frequent extrasystoles. Tmx.=110. The atropine test accelerates the heart beats (105 per minute). Liver not enlarged. Spleen enlarged and painful on pressure. Thyroid increased in volume. Inguinal glands enlarged.

November 24, 1913. Pulse rate 85. No change in the physical signs of the heart. Numerous extrasystoles.

January 15, 1913. No modifications in the physical signs of the heart. Numerous extrasystoles. Number of beats 90 when lying down and 104 standing; the extrasystoles are as numerous in one position as in the other.

Record no. 10.

The rhythm of the pulse is interrupted by extrasystolic beats of ventricular origin. There is also a slight sinus arrhythmia.

Observation no. 12.

Ventricular extrasystole; the atropine test makes the rhythm regular.

V. D., mulatto, 35 years, widow, resident at Lassance.

Examined April 18, 1913.

Previous history, only attacks of intermittent fever.

Complains of occasional attacks of palpitation. No other subjective symptoms.

Heart increased in volume, apex beat in the fourth intercostal space, in the mammillary line, 8, 5 cm. from the midsternal line.

No noticeable change in the heart sounds. Pulse 76, with frequent extrasystoles.

Katzenstein	{	before—76 ps. ? Tmx. 130.	
		during—82	» 120.
		after—80	« 125.

Pulse 92 standing. Liver not enlarged. Spleen enlarged, with a thin, hard margin. Glands not hypertrophied. Thyroid with moderate goiter. Her husband died suddenly.

Records no. 11 and 11-A.

1) Radial pulse full, rather slow, rhythmic, the dominant rhythm interrupted by extrasystoles with ample compensating period. Rate little less than average, 68 per minute. Extrasystoles frequent, and scattered irregularly in the tracing. The venous pulse with strongly accentuated carotid waves; *a* waves well marked. *v* waves slightly marked.

Corresponding to the extrasystoles of the radial pulse there are exaggerated rises of the jugular pulse due to the simultaneous contraction of auricle and ventricle. The auricles contract rhythmically; the contraction of the ventricles is anticipated. Ventricular extrasystoles.

II) Taken after injection of 0g,001 of sulphate of atropine. Rate increased; from 68 to 92. Disappearance of the extrasystole. Radial pulse rhythmic and regular. Venous pulse with carotid appearance. Ventricular extrasystoles in the heart, rather slow; disappearance of the same half an hour after atropine, with moderate acceleration, 68; 92.

Observation no. 13.

Ventricular and nodal extrasystoles. Palpitation.

A. C., mulatto, 40 years old, male, single, laborer, resident at Beltrão.

Examined March 3, 1913. In his history there is only a reference to intermittent fever that he had some months previously, from which there remains pain in the right hypochondrium. At present, aside from this

pain, he complains of attacks of palpitation which are transient and infrequent. No other subjective symptoms elicited. Heart not enlarged. Heart-beats interrupted in their rhythm by extrasystoles in bigeminal series. Sounds normal. Pulse 78, one beat being strong and one extrasystolic. Tmx. 110. After the injection of 0,001 g. atropine the pulse becomes regular, 92 lying down and 115 standing. Liver and spleen are a little enlarged. Thyroid enlarged with a voluminous goiter.

Records no. 12, 12-A and 12-B.

(I This tracing shows a type of bigeminal pulse caused by ventricular extrasystoles which succeed each other in alternating cycles. The legend explains itself.

II) Tracing taken fifteen minutes after injection of 0g,001 of atropine. The succession of bigeminal beats is interrupted by a trigeminal group. This group seems to be formed by the addition of an auriculo-ventricular extrasystole to the ventricular extrasystole. The *a* wave which is fused with the *c'* wave of the first extrasystole belongs to the dominant rhythm; the second *a c* is due to the fusion of two synchronous extrasystolic waves. The auricular rhythm regains its rate from the extrasystolic auricular wave.

III) Taken one hour and twenty minutes after atropine. The pulse has become regular and the rate somewhat increased.

Observation no. 14.

Ventricular extrasystoles. Cardiac insufficiency. Palpitation.

J. L., mulatto, 34 years, male, married, farm laborer, resident at Bagre.

Examined April 14, 1913.

The patient had convulsive attacks up to the age of 18 when they ceased. He also had intermittent fever about twelve years ago. There is nothing worth mentioning in the previous history of this man who was strong and hardworking until the beginning of his present illness, about a year ago. After an attack of "Dysentery" has suffered

from pains in the legs and vague pains in the body. These were followed by palpitation, precordial pulsation which extended to the veins of the neck and were felt in the ears: the beats are strong and provoked by emotion or exertion, or come on even while the patient is quiet or during sleep, so that he wakes with a start. He is fatigued by the slightest exertion and scarcely able to walk 20 or 30 meters without fatigue accompanied by palpitations and cold sweat. Abundant sweating even when quiet. Gastric disturbances; the ingestion of food causes much agony ("avexame").

Cardiac percussion area not increased. Apex beat at the fifth intercostal space, 7.5 cm. outside the midsternal line; right margin 2.5 cm. from the midsternal line. Heart sounds without noticeable change. No murmurs; 76 beats with numerous extrasystoles in bigeminal series.

Katzenstein	{	before - 76 ps. Tmx. 130
		during - 82 « « 130
		after - 72 « « 130

Atropine test. Pulse 108 standing.

Liver not enlarged nor painful on pressure. Spleen not enlarged. Thyroid not noticeably enlarged. Inguinal glands slightly enlarged. Further examination on July 13, 1913. For some time the patient showed marked improvement, but a fortnight ago he became worse again. The old disturbances appeared anew with increased edema of the face. Cardiac area not modified. First sound reduplicated; the second accentuated in the pulmonary focus. 70 pulsations with extrasystoles. Tmx. = 135. Liver slightly enlarged and painful.

July 24, 1913.

Pulse in vertical position, 100. Occasional extrasystoles.

Katzenstein	{	before - 72 ps. Tmx. 145.
		during - 72 « « 140.
		after - 78 « « 140.

Record no. 13.

The radial pulse is irregular on account of the interpolation of numerous ventric-

ular extrasystoles, which are easily seen by the legend.

13 a) After the injection of atropine the number of extrasystoles diminished considerably, only one being recorded in a number of tracings. It was of ventricular origin.

Observation no. 15.

Ventricular extrasystoles. Fainting attacks.

S. A., 36 years, female, resident at Santa Rita.

Examined April 2, 1911.

Marked melanoderma. Absence of arteriosclerosis. Large goiter. Cervical glands enlarged. In the last few days the patient has had fainting fits. Liver not enlarged.

Katzenstein	{	before - 91 ps. Tmx. 105
		during - 78 « « 105
		after - 91 « « 105

Numerous extrasystoles.

Record no. 14.

The radial and cardiac tracings show strong arrhythmia, but the dominant rhythm is to be seen in the group of pulsations to the right of the central vertical line. This rhythm is interrupted by numerous extrasystolic beats. The combined analysis of the 3 tracings elucidates the nature of these extrasystoles. In group I, II, III, IV. The beat IV is of ventricular extrasystole as the legend shows. The group a, b, c, d, f, is more complex. To 5 auricular beats there are 6 ventricular ones. This is because the extrasystolic beats c and d succeed each other rapidly. The a III wave falls within the refractory period of the cycle and is blocked. The premature beat c is anterior to a IV being very much anticipated and does not prevent the excitation of the latter being transmitted to the ventricle, increasing only the time of conduction. The beat of cycle (d) might be considered dependent on a III, with very much delayed rate of conduction of the stimulus. The polygraphic tracing is not sufficient to elucidate the case. The

beats marked on the left of the tracing are susceptible of the same interpretation.

Observation no. 16.

Ventricular extrasystole. Cardiac insufficiency. Spasm of the esophagus.

J. G., white, male, 39 years old, resident in Beltrão.

April 7, 1911.

The patient complains of palpitation, fatigue on exertion and nocturnal attacks of suffocation and giddiness. He also has difficulty in swallowing solid food, spasms of the esophagus, which oblige him to drink water after every mouthful. Well-marked bronze coloring. Large goiter. Spleen slightly enlarged. 110 beats when lying down, 130 standing. Tmx. 115. Numerous extrasystoles.

Record no. 15.

April 7, 1911.

Radial pulse with regular dominant rhythm, with slightly increased rate, 83 per minute; interrupted by extrasystolic beats which occur irregularly. The extrasystolic beats have a complete compensating period of rest. Jugular pulse; the *a c v* waves occur in the normal succession; wave *c* in some points hardly noticeable. Wave *a* not raised. In the cycles interrupted by extrasystoles, the latter only appear synchronously with the auricular contraction, whence the fusion of waves *a* and *c* indicated by the legend. Auricular rhythm not modified except slightly by the respiratory movements. The extrasystoles are of ventricular origin and more or less premature.

Summary: ventricular extrasystoles occurring irregularly.

Observation no. 17.

Ventricular extrasystole. Bigeminism.

M.M.; 38 years old, female, resident at Porto-Faria.

April 19, 1911.

Complains of lumbar pains and pains

in the stomach. Frequent attacks of palpitation. Large goiter. Melanic pigmentation well-marked. Liver increased in size; 90 heart beats with extrasystoles in bigeminal series. Tmx.=125.

Record no. 16.

This is a tracing of bigeminism of heart and pulse with an intercalated trigeminal group. The bigeminism is produced by the regular succession of ventricular extrasystoles with compensating period of rest. The trigeminal group is formed by the intercalation of an interpolated extrasystole in the cycle. The *a c* interval which corresponds to the beat of normal cycle which succeeds the extrasystole is very much increased and the next *a* wave is blocked.

Observation no. 18.

Ventricular extrasystole. Bigeminism. Spasm of the esophagus (Mal de engasgo—Difficulty in swallowing.)

Examined May 23, 1913.

Previous history; all that is worthy of notice are pains in the joints which appeared when the patient was 18 years old, and which recur on cold and damp days. For about three years the patient has had palpitation. Feeling of limpness in the body and pains in the legs. Dyspnea on exertion and inability to work as well as his fellow-laborers, being easily tired. Nocturnal dyspnea. Giddiness. Sensation of distension of the stomach with acid eructations. Choking. Sometimes the patient cannot swallow his food without the help of water. Heart slightly enlarged. Apex beat in the fifth intercostal space a little inside of the nipple, 9,5 cm. from the midsternal line. Apex shock large and strong. No noticeable change in the heart sounds. Pulse 65 per minute, ample, full, and unstable, the number of beats changing from one moment to another. Acceleration of the pulse after swallowing. Frequent extrasystoles, at times isolated, at others in bigeminal series.

Record no. 17.

The waves of the radial and of the venous pulses are found in this tracing. The radial pulse shows a very clear bigeminism. At the right side of the tracing there are regular waves of the dominant rhythm to be seen. The bigeminism arises from the succession of ventricular extrasystoles in alternating cycles. The venous tracing confirms this interpretation, the auricular waves fall rhythmically; at one time the ventricular wave preceding the normal beat, at another succeeding that of the premature one, falling in the final phase of the systole of the latter.

Observation no. 19.

Bigeminism from nodal extrasystoles. Interpolated extrasystole. Atropine test with change of conductivity.

J. Q. R.; white, 25 years, old, male, tradesman, resident at Sant'Anna dos Alegres. Previous history, merely different attacks of colds and bronchitis *a frigore*. Denies having had venereal or malarial history. About five years ago a beginning of *ankylostomiasis* with pains in the legs and fatigue, which was cured. He has felt ill for about a year, and has had attacks of precordial strong and rapid beats. At first short and far between, these attacks have become more intense, more frequent and of longer duration. They are brought on by the slightest emotion or effort or even come on without any provocation. They are independent of meals. Sometimes they come on at night and the patient wakes up with a start. They are very uncomfortable and bring on uneasiness, spots before the eyes, sweating, trembling, labored breathing and strong agony (avexame). The patient does not mention giddiness. For about two months he has had a feeling of fatigue after any effort, even after the slight exercise of walking, which brings on tiredness, palpitation and pains in the legs. He does not mention edema, nor has he any. Heart enlarged, apex beat in the fifth

intercostal space outside the mammillary line. Right margin 2,5 cm. from the midsternal line. Sounds without noticeable change. No murmurs. Extrasystoles with bigeminal appearance. 72 to 80 pulsations. Tmx.—135. Atropine test. Liver not enlarged. Thyroid enlarged. Inguinal glands not enlarged. Nervous system negative.

Record no. 18.

Tracing I was taken before the injection of atropine. The tracing of heart and pulse which are analogous show waves in bigeminal groups interpolated with regular waves of the dominant rhythm between. The second wave of the bigeminal group is of extrasystolic appearance; the jugular tracing shows that they are "nodal extrasystoles"; the auricular and ventricular contractions are premature and synchronous. The dominant rhythm is slow; the jugular curve tracing is peculiar as it shows a bifid V wave and very clear Hirschfeld C waves. The *a* wave of the dominant rhythm which follows the extrasystole is slightly anticipated.

Summary: bigeminism caused by nodal extrasystole.

Record no. 18-A

Taken fifteen minutes after the injection of atropine.

The pulse has become regular, but not entirely so. The *a* waves do not succeed each other in perfectly regular rhythm; an extrasystole corresponding to *c' v'* of the venous curve is to be seen.

Record no. 18-B

Taken one hour after atropine. The beats are slightly accelerated and are becoming rhythmic.

Record no. 18-C

Taken four hours after atropine. The pulse has again become irregular though it still is accelerated. The *a* waves succeed each other irregularly. Some are blocked,

which explains the lapses of the radial pulse. The change in conductivity which appears many hours after the action of the atropine is interesting; this is not seen in other tracings. Here also the variations of the dominant rhythm are much greater.

Some beats (*a' c'*) are probably auricular extrasystoles.

Observation no. 20

Interpolated extraventricular systoles. Palpitations. Cardiac insufficiency.

Examined December 29, 1912.

M. F., mulatto, 40 years, female, married, resident at Santa Rita. For some months the patient has suffered from "agony" with great fatigue, anxiety and uncomfortable heart beats. Dyspnea when lying down. Palpitation; attacks of strong and rapid beats, sometimes provoked by exercise, by standing or even coming on when the patient is quiet without any special reason. Sometimes the beats are slow and strong. Gastric uneasiness after swallowing, the ingestion of food provokes agony. She feels better when she is fasting. Frequent and abundant eructations. A feeling of depression and general weakness. Lack of appetite. Heart but slightly enlarged, measuring 10,5 cm. at the base; apex beat 8 cm. from the midsternal line, a slight downward dislocation of the heart; Sounds clear, the second somewhat tympanic at the aortic area. Beats rhythmic, the rhythm interrupted by extrasystoles, either in series or singly. Pulse 60 per minute. Tmx. 140. Pulse small and hard. Liver much enlarged. Spleen without noticeable enlargement. Thyroid gland with cystic goiter.

Further examination January 1, 1913.

Frequent attacks of giddiness. Buzzing in the head, vague pains in the body. Frequent palpitation, sometimes attacks of strong rapid beats, at others strong and slow beats. At the time of examination, the patient complained of agony and had strong slow beats but the objective examination revealed nothing abnormal. Pulse 50 beats per minute: no dyspnea. Sometimes the beats were

so rapid that the heart seemed to tremble. Condition of the heart same as before. Generalised edema. Liver still much enlarged.

January 19, 1913.

No more edema. Liver a little smaller. Improvement of the subjective symptoms. Physical state of the heart unaltered.

June 4, 1913.

After a period of improvement the patient, being worse, came to consult us again. There was nothing, however, which called for special interest. After a few days treatment she got better.

Record no- 19.

This tracing is interesting because it shows trigeminal rhythm caused by the interpolation of ventricular extrasystoles every four cycles, with a quartan rhythm.

The indication of the waves shows that the extrasystoles are interpolated,

Observation no. 21.

Interpolated ventricular extrasystoles.

M. I. do N., white, female, 27 years old, married, field-laborer.

Examined October, 1914.

The patient has been married for thirteen years and has had three miscarriages at three months and five children born at term; she lost a child of three from sore throat. The last child is 18 months. First menstruation at the age of 12, regular and painless. Has had intermittent fever. Heart beats, fatigue and agony, she feels worse at rest and improves with exercise. The heart-beats accompanied by giddiness and tremblings. Vague headaches. Trembling.

Heart. Apex beat in the fourth intercostal space, 7,5 cm. from the midsternal line. Tmx=150. 74 pulsations per minute. Extrasystoles frequent after exercise and exertion. Liver extending 19 cm. from mammillary line. Spleen not palpable. Goiter with cysts of different sizes.

Record no. 20.

An interesting formation of trigeminal rhythm by the regular intercalation of ventricular extrasystoles. They appear every 3 cycles, in tertian rhythm. Compare with record 19.

Observation no. 22.

Ventricular extrasystoles in series.

P. F. S., white, male, 45 years, resident at Coração de Jesus. Signs of cardiac insufficiency, Katzenstein's test positive. Heart sounds muffled. Frequent extrasystoles. Liver enlarged and painful on pressure. Thyroid much enlarged. Cervical glands enlarged.

Record no. 21.

The dominant rhythm is interrupted by a series of six consecutive extrasystolic beats. These beats are due to extrasystoles of ventricular origin; in another part of the tracing is seen an isolated extrasystole which is also ventricular.

Observation no. 23.

Ventricular extrasystole. Post-extrasystolic alternation. Palpitation. Marked dilatation of the heart.

C. C. da S. mulatto, male, 27 years, married, farm laborer, resident at Porto Faria.

Examined September 12, 1912.

No subjective signs, except occasional passing attacks of palpitation. The patient is tall, above middle height, well-built and of robust appearance. Heart not enlarged, apex beat in the fifth intercostal space outside and below the nipple, 14 cm. from the midsternal line. Right margin 6 cm. from the midsternal line. Heart sounds without any marked alteration. No murmurs. Pulse lying down 82, standing 107.

Katzenstein { before - 81
 { during - 92
 { after - 92

$T_{mx}=125.$

Pulse with numerous extrasystoles. Liver slightly enlarged. Spleen not noticeably enlarged. Goiter, thyroid gland moderately enlarged.

Record no. 22.

Besides the falling out of the radial pulse corresponding to the slight beats of the cardiac tracing, there is a very marked alternation in the beats which follow the lapses, which are extrasystoles and are not registered in the pulse. These are ventricular.

Record no. 22.

The sphygmogram registers the interruption of the rhythm by extrasystolic beats and lapses. The alternation of the pulse is also noticeable and more apparent in the pulsations that follow an extrasystole. The extrasystoles are ventricular.

Observation no. 24.

Ventricular extrasystoles. Alternation of the pulse.

T. X., male, 31 years, resident at Beltrão.

The patient came to consult us on account of fatigue, palpitation and gastric disturbances.

Heart enlarged, apex beat in the sixth intercostal space outside the mammillary line, the base measuring 16,3 cm. Pulse arrhythmic, with lapses which correspond to the extrasystolic beats in bigeminal series. Pulse 76 lying down, 74 standing up, $T_{mx}=110.$ Thyroid enlarged with cystic goiter of the middle lobe; cervical and axillary glands enlarged. The patient came back to consult us three months later and was put in hospital as he showed intense signs of cardiac insufficiency, which brought on asystole.

He was observed by a commission of professors who were there at the time. This case was considered very interesting from a clinical point of view and one of the best examples of the cardiac form. Furthermore

an autopsy of this case was performed and showed for the first time the presence of parasites in the chronic form of the disease.

Record no. 23.

In the radial tracing were seen extrasystoles with complete compensating repose, possibly ventricular, and marked alternation.

Observation no. 25.

Extrasystole. Palpitation. Post-extrasystolic alternation.

C. L. A., mulatto, female, 30 years, resident at Contendas.

Examined January 1, 1913.

Has been ill for a year until which time she was strong; there is no history of previous disease. For about a year she has had frequent headaches, lack of appetite, difficult digestion, gastric pains, to which were added later on limpness, pain in the legs, weariness and heart palpitation, provoked by the slightest effort. At present it is the palpitation that disturbs most, as it comes on the least exertion or even while at rest. Neither nocturnal dyspnea nor edema. Heart not enlarged. First sound unchanged, second with a pulmonary accentuation. Mesosystolic murmur at the pulmonary area. Cardiac erectism, with very visible pulsation of the veins of the neck. Pulse unstable, the number of beats varying from 98 to 112. Tmx. = 125. Very numerous extrasystoles at the beginning of the examination afterwards diminishing. Liver and spleen not enlarged. Thyroid enlarged.

Record no. 24.

Radial pulse with dominant rhythm interrupted by ventricular extrasystolic beats; in the venous tracing the *a* wave of the auricular rhythm and the *c'* wave of the premature ventricular beat are fused. The interval *a c* unchanged. In the radial tracing a post-extrasystolic alternation is indicated.

Observation no. 26.

Ventricular extrasystole, Post-extrasystolic alternation. No subjective symptoms.

F. R. A., white, male, 26 years, widower, farm laborer, resident at Bebedouro.

Examined December 7, 1912.

Previous history: about 10 years ago was jaundiced, edematous, with easily brought on fatigue and palpitation; he was cured. The patient has also had several attacks of ill defined fever.

At the present date he is strong, inclined to work, without dyspnea on exertion. No edema. He came to consult us on account of a traumatism of the thorax which left a local pain, about 20 days ago. No enlargement of the cardiac area, apex 7, 5 cm. from midsternal line, below and within the nipple; the right margin 3,5 cm. outside the midsternal line. Tachycardia: Pulse 102, with numerous extrasystoles. During the examination he complained of uncomfortable heart-beats (emotion). Liver not enlarged. Thyroid much enlarged.

Record no. 25.

The radial tracing shows very marked alternation of the pulse principally after the extrasystoles, which are ventricular as shown by the venous pulse. The cardiac tracing shows, besides the respiratory oscillations, very marked auricular waves besides the ventricular waves.

The jugular tracing is not disturbed; the *a* waves succeed each other in even rhythm. The high points which are seen on the tracing arise from the fusion of *a* and *c'*. Ventricular extrasystoles coinciding with the auricular systole.

Summary: Ventricular extrasystole with marked alternation.

Observation no. 27.

Ventricular extrasystole. Post-extrasystolic alternation.

F. X., white, male, 55 years old, resident at Beltrão.

Examined May 4, 1911.

The patient complains of vague pains in the extremities and palpitation. Very marked bronze coloring. General emaciation. Pulsation; 76, with occasional extrasystoles. Tmx.=100. Pulse standing, 108.

Record no. 26.

The appearance of the radial tracing is that of a series of rhythmic beats, interrupted by lapses and slight extrasystolic beats. The lapses and the extrasystolic accidents take in the space of 2 or 3 normal beats, according as to whether there are 1 or 2 extrasystolic beats; full compensating period of rest. The post-extrasystolic alternation is well marked and lasts during many beats after the extrasystole. It is well marked on the whole of the right side of the tracing; 20 beats after the extrasystolic one.

Observation no. 28.

Ventricular extrasystoles.

M. L., de M. mulatto, male, 28 years resident at Santa Maria.

Examined April 29, 1911.

He complains of giddiness, palpitation and precordial pains. Pulse arrhythmic, with frequent extrasystoles, sometimes in bigeminal series. Pulse 62: Tmx.=110. The extrasystoles are of ventricular origin; the a-c intervals lengthened, as may be seen in the tracing. Thyroid very much enlarged, with goiter.

Record no. 27.

The radial pulse is interrupted in its rhythm by premature beats of ventricular origin, shown in the venous tracing by high waves; fusion of a' and c'. A slight lengthening of a c interval is also noticed; this is a sign of a slight alteration of conductivity.

Observation no. 29.

Extrasystoles. Lengthening of a-c. Sudden death.

L. J. V., mulatto, male, 35 years, laborer, single, resident at Urucuia.

Previous history: patient only mentions having had intermittent fever several times. Denies venereal disease. Robust constitution, apparently strong. Skin pale, slight generalised edema, more marked in the face. Has been feeling ill for about eight years. His legs are weak and he has cramp at night. Palpitation, coming on sometimes when at rest, at other times on exertion; precordial pulsation, also in the veins of the neck; these beats are strong and uncomfortable; dyspnea on exertion and nocturnal dyspnea. Frequent cough. Constant sensation of gastric fulness, with great discomfort and respiratory difficulties, increased by taking food, however small the quantity. These symptoms have gradually increased so that patient can no longer work. Heart considerably enlarged. Apex beat in the sixth space, 15 cm. from the midsternal line, reaching to the axillary line; right margin 5 cm. from the midsternal line. Precordial shock diffuse, apex shock ample and intense. Heart sounds muffled, especially at the base. No murmur. Bigeminal beats; the second beat has at times two tones, at others only one, simulating the auricular systolic beats which are not transmitted, being very muffled. Pulse slow; sometimes the extrasystolic beats of the bigeminal group are felt in the pulse, at other times this is not the case. Pulse 46; Tmx.=135. The number of beats varies but slightly with the difference of position or on exertion. Lying down 55, standing 58, after exercise 56. Atropine test: bigeminism not modified—beats accelerated: 80 per minute (v. tracing), Left lobe of liver slightly enlarged and painful on pressure. Spleen enlarged, not painful. Thyroid enlarged with cystic nodules.

August 17, 1913. Edema disappeared and all the symptoms improved without any change in the physical condition of the organs. August 20, 1913. Gastric uneasiness more intense. Strong palpitation. Increase of dyspnea on exertion. 58 beats when standing, lying down 56. Bigeminism. Tmx.=120. Physical condition of organs unchanged. On going out from the hospital for a short walk this patient died suddenly.

The histopathologic examination of the heart was made by Professor B. C. Crowell, who found lesions peculiar to trypanosomiasis, this case being one of those which enabled him to establish the histopathologic characteristics of the cardiac lesions in the chronic form of Chagas' disease.

Record no. 28 and 28-A

I) The radial tracing is that of a slow arrhythmic pulse, with cycles varying very much in length; in some there are extrasystolic beats.

The cardiac tracing is of bigeminism; the groups of bigeminal beats vary in their succession. Some of the auricular waves are well marked in some points of the cardiogram.

The first beat of the extrasystolic group is of the dominant rhythm. The jugular pulse shows that it is always preceded by an auricular wave and that the *a-c* interval is lengthened. The second beat of the bigeminal group is extrasystolic. The origin of the extrasystoles is in some points frankly auricular. Others seem to be auricular, like that which accompanies the radial beat II and that which is marked in the right half of the tracing. Others are probably nodal, like those which are registered by acute and high waves in tracing II.

II) The rate of the auricular waves is arrhythmic and some are blocked. The causes of the arrhythmia of the pulse are consequently multiple and very difficult to analyse. Atropine had no appreciable action to be seen on the tracing, except acceleration.

Observation no. 30.

Extrasystole. Lengthening of the a-c interval.

D. M. da C.; negress, 60 years old, widow, resident in the neighborhood of Lassance.

Examined March 9, 1913.

Previous history obscure, intellect of very low order, so that it is very difficult to question the patient. She complains of

palpitation, dyspnea on exertion, dyspnea when lying down and at night, so that she cannot sleep. No edema. Heart much enlarged, apex beat outside the mammillary line at 11 cm. from the midsternal line. Right margin 4 cm. outside the midsternal line. Irregular beats with numerous extrasystoles sometimes in bigeminal series. Sounds muffled, no murmurs. Pulse 56, irregular and arrhythmic. Tmx.=145 Left lobe of liver slightly enlarged. Spleen not palpable. Nervous system negative. Thyroid enlarged.

Reexamined April 19, 1913. Dyspnea on exertion, when lying down and at night. Sleeplessness. Generalised though slight edema. Bases of the lung congested. Frequent cough. Heart enlarged; apex beat in the fifth space on a level with the anterior axillary line. Heart sounds muffled, principally the first. Marked swelling of the neck veins, with scarcely noticeable pulsations. Pulse 55. Liver rather enlarged.

Record no. 29.

The radial and cardiac tracing show bigeminism, with some beats of the dominant rhythm at the right side of tracing. The second beat of the bigeminal group is extrasystolic; the extrasystoles are nearly all ventricular. The marking of the tracing indicates our interpretation of it; *a c* interval lengthened so that the conduction of the contractile stimuli is delayed.

Observation no. 31.

Ventricular extrasystoles. Lengthening of the a-c interval.

M. P. dos S.; mulatto, female, 33 years, married, resident at Lassance.

Examined April 18, 1913.

Has been ill for about twelve years, and thinks her illness began after child birth, when she had fever for fifteen days. Her subsequent confinements aggravated her condition and after the last, a year ago, she became much worse. Present condition: Slight icterus. Cyanosis. Edema of the lower extremities and trunk, however not very

marked. Headache and vague pains in the body. Cardiac palpitation, dyspnea on exertion and even attacks of dyspnea when at rest and at night so that she cannot sleep. Refractory cough, the last few days bloody sputum. She has had edema of the legs. Patient feels that the stomach is swollen. For the last few days has had attacks of vomiting accompanied by great anxiety. Nearly incessant cough and dyspnea. Lack of appetite. Constipation. Heart much enlarged, apex beat in the sixth space, slightly within the axillary line, 15 cm. from the midsternal line. Right margin 5 cm. from the midsternal line. Precordial region with a rounded prominence. Strong precordial shock, shaking the thoracic wall, its violence being especially noticeable in the fourth, fifth and sixth spaces. First heart sound muffled with slight murmur at the apex, without propagation. Second sound accentuated in the pulmonary area. Pulse weak, soft and empty. Extrasystolic arrhythmia. Number of pulsations varying from 88 to 92. Tmx=110. Liver much increased in size, painful on pressure. Spleen without noticeable enlargement. Thyroid enlarged. Congestion of the bases of both lungs, the right more marked.

The patient was reexamined on June 20, 27 and 28, and on July 3 and 5. She was only slightly better and did not come back for consultation after the fifth. On that day her condition was as follows: Dyspnea as well as general condition improved. Pulse 82, sometimes in bigeminal series. No more vomiting.

Record no. 30

The radial pulse is entirely arrhythmic but not that of complete arrhythmia. Analysis of the venous and pulse tracings shows that the arrhythmia is produced by different causes. The first is a change in the auricular rate, the *a* waves appearing irregularly spaced. The conductivity is changed, the *a c* interval lengthened; this induces a delay in the conduction of the contractile stimulus.

Extrasystolic beats of ventricular origin. The legend of the tracings shows these alternations, as well as their interpretation clearly.

Observation no. 32.

Ventricular extrasystole. Lengthening of the a—c interval. Marked cardiac insufficiency.

O. N. C.; mulatto, male, 38 years, farm laborer, resident at S. João da Ponte.

Examined September 12, 1912.

He has been ill for about two months; fatigue on exertion gradually increased. Pale, slight edema of face and lower extremities. Dyspnea on exertion and at night. Cardiac area much enlarged. Apex beat in the fifth intercostal space, 12 cm. from the midsternal line. Right margin 4,5 cm. from the midsternal line. Systolic murmur at apex, replacing the first heart sound with propagation towards the axillary line. The first sound can be heard in the tricuspid area. Second sound reduplicated with pulmonary accentuation. Pulse 88; seated 99. Numerous extrasystoles. Tmx.=105. Liver enlarged painful on pressure. Spleen with noticeable enlargement. Thyroid enlarged, with voluminous goiter.

Record no. 31.

The radial pulse shows very clear alternation, extrasystolic lapses and beat with compensating complete repose. The jugular pulse shows the premature beats *c'* of ventricular origin, coinciding with the auricular beats *a* of the dominant rhythm.

It can be seen that the conductivity is altered, there being delaying of the conduction which is indicated by the lengthening of the *a—c* interval.

Observation no. 33.

Ventricular and nodal extrasystole. Tachycardial crises. Lengthening of the a—c interval. Ventricular escape.

P. A. S.; mulatto, male, 39 years, married, resident at Contria.

Examined February 17, 1913.

Previous history of only blenorrhagia and intermittent fevers. Until about two and a half years ago the patient was strong and hard working. About that time he began to feel fatigue on exertion, dislike for work gastric fulness almost always present, increased by the ingestion of food. Dyspnea on exertion and when lying down. Attacks of dyspnea even when at rest. Nocturnal dyspnea which interrupts the sleep. Frequent palpitation; attacks of strong, rapid, inconvenient, uncomfortable, and at times painful, heart beats coming on without noticeable cause even during sleep, causing him to wake with a start. These attacks are quickly over, not lasting more than half an hour. Frequent giddiness, but not enough to make him fall. As to the feeling of gastric fulness, the patient states that sometimes he feels "as if he had a ball in his stomach which came up to his throat and choked him". The patient is below average height, of cretinised appearance, his skin of a dirty yellow color; large goiter. Generalised edema. Heart much dilated. Apex beat in the sixth space, a little within the anterior axillary line. Right margin 4.5 cm. from the midsternal line. Precordial area with a rounded prominence. First sound silent at the apex, replaced in the tricuspid area by a systolic murmur. Second sound reduplicated, with pulmonary accentuation. Arrhythmic pulse, with frequent extrasystoles. Pulse 88 to 90. Tmx.=120.

Arteries slightly hardened. Liver enlarged and painful on pressure. Spleen slightly enlarged. Examination of lungs negative. Examination of nervous system negative.

Feb. 21. All the subjective phenomena are better. Diminished edema. Physical signs not changed. 76 radial pulsations with numerous extrasystoles.

Feb 25. The patient is better and the edema has largely disappeared. Size of heart very little changed. No more tricuspid murmur. Pulse arrhythmic, with attacks of tachycardia. Liver not noticeably enlarged. He came to consult us again on March 9, 1913. Palpitation diminished. There had been no

more dyspnea. There is no change in the area of cardiac dulness. Pulse 72 with numerous extrasystoles. Tmx.=115. Liver not noticeably changed. Heart sounds muffled, with very clearly heard tricuspid murmur.

March 11, 1913. General condition better. No more tricuspid murmurs. Arrhythmic beats in bigeminal series, alternating with series of rapid beats. Liver slightly diminished.

March 14, 1913.

Slight aggravation of the subjective symptoms. The tricuspid murmur had reappeared.

March 15, 1913.

Improved again, Diminished palpitation. The improvement is more evident, and the patient left three days later.

We heard that he died some months afterwards in asystole.

Records nos. 32, 32-A, 32-B, 32-C.

1) The radial tracing is very arrhythmic, also the cardiac one.

The arrhythmia is of very complex origin and only the joint examination of the three curves can explain it.

At the left in the center, and at the right side of the radial tracing are seen groups of beats of the dominant rhythm, with a rate of 83 beats per minute, but the cycles are not perfectly equal. The examination of the waves of these cycles on the venous tracing shows the *ac* interval to be much lengthened, which indicates an alteration in conductivity. In the cardiogram the auricular wave is clearly marked in the diastolic phase of the ventricles. The first extrasystolic beat which appears after the fifth beat of the normal cycle is an extrasystole, intervening between the fifth and sixth beats of the dominant rhythm. The *ac* interval of the sixth beat is lengthened; the ventricular systole is so retarded that it falls in a refractory phase and is blocked, leaving a long pause between the sixth and seventh beats. The interval between the fifth and seventh beats in the dominant rhythm is perceptibly equal to three times

the normal cycle. The following extrasystole is ventricular. The beat which follows the extrasystole begins a series of beats of the dominant rhythm; its cycle is lengthened by the retardation of the following ventricular systole, on account of the delay in the conduction of the contractile stimulus. The *a c* interval is lengthened.

Thus the arrhythmia comes from ventricular extrasystoles, interpolated or otherwise, and from the alteration of the conductivity.

II) The tracing shows a tachycardial crisis with a rate of 130 beats per minute. The radial pulse shows a very noticeable alternation. Jugular pulse with high rhythmic waves at the same rate as those of the radial pulse, which are the auricular waves coinciding with the ventricular waves of the anterior cycle that are marked at the origin of the *a* waves. The tachycardial crisis is therefore probably due to a succession of auricular extrasystolic waves at a rapid rate transmitting themselves to the ventricle with delay. The *a c* interval much lengthened. At the right side of the tracing are seen some beats of the dominant rhythm and many extrasystolic beats.

III) This shows bi and trigeminal rhythm from ventricular extra-systoles.

IV) Tracing 32-c shows the effect of digital.

The effect on the conductivity is manifest; the *a c* interval is much lengthened, having apparently at some points "ventricular escape". Numerous extrasystoles in bi- and trigeminal groups.

Observation no. 34.

Ventricular extrasystole. Lengthening of the a—c interval. Cardiac insufficiency.

J. D., mulatto, male, laborer, married, resident at Contria. Previous history only mentions intermittent fevers. For a long time he has been subject to attacks of palpitation, without having his general condition affected. About a year ago the patient began to feel weakness in the legs and dyspnea on exer-

tion which increased gradually, preventing him from working. Lately he has had dyspnea from lying down, also at night, a cough, and edema of the lower extremities and face. The palpitation has considerably increased in frequency and intensity; it may be even brought about by drinking water. Heart much enlarged. Apex beat in the sixth space, near the anterior axillary line, 13 cm. from the midsternal line. Right margin 4, 5 cm. from the midsternal line. Precordial shock intense and wide. Epigastric beats intense. First sound very muffled, with slight murmur at the apex. Second sound muffled. Pulse very irregular, with frequent extrasystolic lapses. The number of pulsations is more or less 82 per minute. Tmx.=120. Pulse 95, standing. Liver considerably enlarged. Spleen not enlarged. Thyroid enlarged, with voluminous cysts of the lateral lobes. Edema. Bronchitis. Glands enlarged.

After a few days in hospital the patient was discharged as he was much better. He came back on June 5, 1913. Edema. Intense dyspnea after the slightest effort. Nocturnal dyspnea. Frequent palpitation. Heart continues much enlarged. Apex 12,5 cm. from the midsternal line, within the anterior axillary line. Right margin 4,5 cm, from the midsternal line. Precordial shock ample and violent, shaking all the precordium. First sound muffled, second reduplicated, with strong pulmonary accentuation. Cardiac beats very irregular, with slight repeated crises of tachycardia; number of beats varying between 75 and 82 per minute. Tmx.=110. Turgescence of the veins of the neck. (See tracings). Liver much enlarged. Spleen not palpable. Slight improvements only; the treatment was interrupted. We heard that he died in asystole a month [after leaving the hospital.

Records nos. 33 nda 33-A.

May 11, 1913.

I) The radial pulse shows beats of the dominant rhythm, their sequence interrupted by extrasystolic cycles. The beats proper of

the dominant rhythm are not perfectly rhythmic, there being variations; the examination of the jugular pulse shows them to arise from different causes.

First of all, the rate of the *a* waves is not regular, sometimes more rapid, sometimes slower. Some waves are so much anticipated that they seem to be rather auricular extrasystoles than beats of the dominant rhythm.

The conduction of the stimulus retarded, and the delay of the conduction also contributes to the arrhythmia. The extrasystoles are generally ventricular. II) Tracing 33-*a* was taken 15 days after the anterior one, and does not differ from it substantially. The rate is more rapid and the conduction slower; the *a* waves often fall in the systolic phase of the previous cycle.

The extrasystoles are numerous and of ventricular origin, and in the first part of the tracing an interpolated extrasystole is seen.

Observation no. 35.

Partial block. Rhythm of 2:1 and of 1:1.

J. C. de A., mulatto, male, 28 years old, laborer, married, resident at N. S. da Gloria. Previous history of attacks of malaria nearly every year. Some attacks of bronchitis with chills. He does not mention venereal disease or rheumatism. This patient smokes and takes coffee; formerly he drank spirits, but he has stopped doing so for the last two years. About two years ago he had palpitation, strong rapid and uncomfortable beats and fluttering of the heart. The palpitation came on in attacks sometimes after an emotion and sometimes without any apparent reason. Great irritability and nervous excitability; he cannot stand being with many people; any loud talking, or rapid movements or effort provoke an attack of palpitation. This condition is accompanied by trembling and a sensation of cold; the patient says he feels a ball in his stomach which rises to the chest and throat, stopping his breath and causing agony (*avexame*). Frequent giddiness without causing him to

fall. General weakness. Inability to work. The slightest exertion fatigues him and brings on palpitation and agony (*avexame*). He has good appetite, but the ingestion of food causes uneasiness and abundant eructations. No edema.

Heart enlarged. Apex beat in the fifth space on a level with the mammillary line, 9,5 cm. from the midsternal line. Right edge 4 cm. from the midsternal line. Shock diffuse. Pulse arrhythmic, with frequent pauses which are not extrasystolic. The jugular tracing shows the presence of a wave during the long pauses of the ventricle. Pulse ample, empty, soft, from 43 to 55. Tmx.=125. The number of beats rises to 92 when standing and 100 after exercise. The neck veins are not turgid; the jugular waves are very visible. We proceeded to the atropine test. The analysis of the tracings may be studied. Liver enlarged, painful on pressure. Spleen not palpable. Thyroid without noticeable enlargement. Inguinal glands slightly increased. Nervous system negative. Intellect normal.

Record no. 34.

I) The radial pulse is arrhythmic, not only on account of the lapses of the pulse after three or four beats of the dominant cycle, but also because the cycles of the dominant rhythm are not exactly equal. In the venous pulse, *a* waves are seen in regular succession. There is also no ventricular systolic wave in the venous pulse at the points which correspond to the lapses of the radial pulse. The lapses come from the block of auricular waves. Immediately after a blocked wave, the *a c* interval of the following cycles is normal, but it lengthens gradually in the following cycles until a new block occurs; this goes on in succession. The gradual retardation of the ventricular systoles by the changed conductivity explains the variation in the rhythm of the radial pulse.

Record no. 34-A.

II) In the tracing, which was taken half an hour after the injection of a milligram of

atropine, the pulse has become regular, the conductivity being reestablished, The *a c* interval is normal. The pulse was a little accelerated.

Observation no. 36.

Ventricular extrasystoles. Partial block.

T. M. P., 58 years resident at Pilar.

Examined June 2, 1911. Complains of palpitation with continual giddiness. Pulse 59 with frequent lapses. Hypertrophy of the thyroid.

Record no. 35

The pulse is arrhythmic, the arrhythmia being caused by complete lapses and premature beats which interrupt the dominant rhythm irregularly. The cardiac tracing does not furnish any indications that can be used, as the respiratory movements affect it a great deal. The collective analysis of the tracings shows that the lapses of the pulse are caused by the irregular block of auricular waves. Thus between the beat III and IV an *a* wave is seen in the jugular pulse; this wave is seen in the cardiac tracing as well is in the catacrotic wave of beat III, but there is no corresponding ventricular systolic wave. The same is repeated in beat VIII—IX and in other parts of the tracing.

Beat X is premature, as a result of a ventricular extrasystole which falls at the same time as the auricular systole of the dominant rhythm. Other extrasystoles are seen in the tracing, all of them ventricular. The *a* waves appear rhythmically in the jugular pulse, the *a c* interval lengthening slightly and gradually until the block, as may be seen in beats IV to VIII.

Observation no. 37

Partial block. Rhythm 2:1 and 1:1, alternating irregularly. Giddiness.

A. D., negro, male, 27 years old, laborer, married, resident at Piedade.

Has had intermittent fevers for many years. A year ago he had milkpox. Has gon-

orrhoea and venereal chancres. He has been ill for a long time, with an undefined uneasiness, which however did not prevent him from working. Has frequent attacks of giddiness, sometimes vertigo and falls. Rarely has palpitation. Dyspnea on exertion. These symptoms have become worse lately; the last few days he has had edema of face and abdomen. Heart not enlarged. Apex 7 cm. from the midsternal line and right margin 3 cm. Apex beat below and within the nipple. Heart sounds muffled. Pulse ample, strong, arrhythmic and irregular, accompanying the heart beats. Series of frequent beats interrupted by slow series. In the long ventricular diastoles is heard a sound which is probably auricular. Jugular pulse hardly visible. Pulse 56 per minute, varying, however, according to the number of the slow beats. Tmx.=140. Atropine test. (See tracings). Liver, left lobe enlarged. Spleen not palpable. Thyroid enlarged. Inguinal glands not enlarged.

Record no. 36.

The radial tracing shows beats sometimes more rapid; the interval between the systoles of the slow beats is perceptible, but not strictly equal to twice the rapid beats. The cardiac tracing shows, besides the identical rhythm, very clear *a* waves which are followed or not by ventricular systolic waves. The *a* waves which are not accompanied by ventricular systoles correspond to the catacrotic phase of the pulse waves of the slow rhythm. In the jugular tracing are seen *a* waves which succeed each other in regular intervals. They are not all, however, followed by a ventricular systole, some waves being blocked. In the beats 1 to IV the block occurs in the rhythm of 2:1. The beats V and VI follow in the rhythm 1:1, with gradual increase of the *a c* interval and the rhythm that of partial block; 2:1 reoccurs following the VI beat. The gradual lengthening of the *a c* is marked,

Record no. 36-A

A quarter of an hour after the injection of 0.001 g of atropine the pulse has become regular, there remaining a perceptible lengthening of *a c* interval. The acceleration of the rhythm is as follows 77:83.

Observation no. 38.

Partial block. Bi- and trigeminal rhythm.

E. S., male, about 30 years old, Previous history obscure. Cretinised appearance. Mouth half open, with saliva running down at the corners. Defective speech with marked general trembling. Bilateral dysbasia, the patient admitting previous paralytic condition.

Patellar reflex on both sides very much exaggerated. Intellect much below the normal. Liver and spleen much enlarged. Numerous glands in the neck, in the axilla and in the inguino-crural region. Thyroid not noticeably enlarged. Pulse arrhythmic, with bigeminal appearance, sometimes trigeminal, sometimes with beats in regular succession; there appear to be extrasystoles. Pulse lying down 52: standing 57. Tmx.=135. Signs of cardiac insufficiency; partial fatigue on exertion; giddiness and uncomfortable palpitation which prevent him from working.

Record no. 37.

The tracing of the heart and pulse have bi- and trigeminal appearance, interrupting a succession of slow beats. This is caused by the partial and irregular block of auricular waves. The auriculo-ventricular rhythm is sometimes 1:1, sometimes 2:1. The slow beats show the rhythm of the block to be 2:1 and 3:1. The origin of some beats, as for instance X, is doubtful; it is perhaps heterogenetic, not depending on *a* 13 as might also be interpreted. The polygraph is not enough to decide this question.

Observation no. 39.

Partial block; irregular rhythm 2:1 and 1:1; Cardiac insufficiency; Sudden death.

G. N., mulatto, male, 40 years old, laborer, married, resident of Burytys. November 11, 1913. Previous history includes attacks of malaria, gonorrhoea and probably syphilis. Does not mention rheumatism. For some time past has suffered from palpitation, rapid, strong and annoying beats, with respiratory difficulty, localized in the precordium and the vessels of the neck, coming on in attacks provoked by emotion or without perceptible cause. Dyspnea on exertion and at night. He is always short of breath. Gastric pains and headaches. This patient has had attacks of intermittent fever and has frequent giddiness.

He is above middle height and of strong constitution.

Intense jaundice; at present he has no edema, nor does he mention any in the past. General trembling, when examined very intense from emotion, afterwards diminishing. Heart much enlarged, apex beat in the fourth space, 13 cm. from the mid-sternal line. Right margin 6 cm. from the mid-sternal line. Precordial shock ample, outside the nipple, the third and fourth intercostal spaces retracted in the precordium, First heart sound very muffled, followed by murmur with an area inside apex. Second sound reinforced in the pulmonary area. Rhythm of three beats at apex, the additional tone clearly separated from the first murmur. Pulse slow, irregular, arrhythmic with frequent lapses; the number of beats varies from 52 to 56. In the long pauses of ventricular systoles is heard a shock, and also a sound synchronous with the venous wave of the jugular; auricular systole not transmitted. The beating of the neck veins is clearly seen as also that they are more numerous than those of the pulse; clearly perceived are also the venous waves which coincide with the cardiac sound as well as a slight shock, without ventricular contraction. Tmx.=110. Liver and spleen much enlarged. Thyroid enlarged; has cystic nodules.

November 13, 1912. After resting and treatment this patient got much better. All his symptoms diminished. Cardiac area per

ceptibly reduced. Apex at 10,5 cm. from the midsternal line and right margin at 4,5 cm. The pulse remained irregular. Sometimes longer and sometimes shorter pauses. Number of beats varying from 50 to 52. The atropine test regulated the pulse, as is seen in the tracings, though there appeared rare occasional extrasystoles. Liver and spleen still enlarged. We did not see the patient any more, but heard that he died suddenly in July, 1913.

Records no. 38 and 38A.

These tracings were taken on successive days and show the same characteristics.

The radial pulse shows occasional, perceptibly rhythmic, beats, whose sequence is interrupted by shorter cycles, which appear irregularly. The cycles of long pauses are twice less than those of short pauses. All the heart-beats are amply represented in the radial pulse.

In the venous pulse the auricular waves are rhythmic and more frequent than the ventricular ones; not all the auricular waves are followed by the corresponding ventricular wave. Thus *a* I and *a* III are blocked, not being accompanied by ventricular systolic wave. *a* II corresponds to radial beat I with the *a c* interval lengthened with retarded conduction of the stimulus.

In the radial beats 2 to 3 and 4 to 5 the *a* waves are successively accompanied by ventricular *c* waves, with the *ac* interval gradually lengthening from one cycle to another.

Collectively the tracing registers a partial block in rhythm 2:1, with irregular return to rhythm 1:1.

The tracings 38 b, 38 c, and 38 d show the action of 0g 00075 of atropine.

In tracing III may be seen extrasystolic beats with complete compensating period of rest. They are ventricular extrasystolic beats. The stimulus is conducted through all the auricular waves but the *a c* interval is lengthened.

In tracings 38 c and 38 d the pulse is perfectly rhythmic, but the lengthening of *a c* continues.

The elective action of atropine on the conductivity must be noticed, and there is no perceptible acceleration of the auricular beats.

Observation no. 40.

Partial block.

J. S., male, 11 years, white, resident at Lassance.

First consultation March 3, 1913. Born at term; normal development. Family history obscure. Former illness: repeated attacks of malaria. This patient complained of fatigue and general weakness. Skin discolored and slight general edema. His face was swollen with marked subpalpebral infiltration; base of the thorax enlarged; abdomen flaccid and voluminous. Liver enlarged, passing the costal margin by two fingers and measuring 11 cm. in the mammillary line; the edge sharp and hard and not painful on pressure. Spleen enlarged and hardened. Epigastrium prominent with marked gastric tympanism. Thyroid enlarged. Cervical and inguinal glands enlarged. Bronchitis of the larger bronchial tubes diminishing. Nothing noteworthy in nervous system. Circulatory system: Heart enlarged, apex beat in the fifth space outside the mammillary line, 9 cm. from the midsternal line; right margin 4 cm. from the midsternal line; upper margin reaching the second rib. Arrhythmic beats, with frequent lapses; no sounds to be heard during the lapses. The first sound not perceptibly changed except that it is slightly muffled. Mesosystolic murmur at apex, musical, not propagated. Second sound reduplicated, with pulmonary reinforcement. Number of beats lying down 82; standing 118. Tmx.=105. Katzenstein: before compression of the femorals 82 beats; arterial tension 105; during compression 86 beats; tension 105; after 82 beats. An injection of 3/4 of a milligram of neutral sulphate of atropine produced the following modifications; 104 beats per min

ute; the pulse became regular one hour after the injection; arterial tension unchanged. The analysis of the tracing shows the details of the arrhythmia, and the influence of atropine on the cardiac rhythm. Progress of disease: on the day following the first examination gave 70 beats lying down, and 60 standing, as the lapses were more frequent in the latter position. Pills of quinine and belladonna were prescribed.

5, 3, 1913.

Infiltration considerably diminished, abdomen less voluminous. Arrhythmia persisting and mesosystolic murmur at apex is audible when the patient is lying down, and not when standing.

6, 3, 1913.

Standing 87 beats; lying on his back 76. General condition much improved.

7, 3, 1913.

Size of heart diminished; apex a little below and outside the mammillary line, 7,5 cm. from the midsternal line. Right margin 3,5 cm. from the midsternal line. Beats 85 lying down, the pulse ample, regular and rhythmic. Liver measuring 10,5 cm. at the mammillary line. Spleen unchanged. Stomach dilated, prominent in the epigastric region. The patient took the medicine referred to on the third, fourth, and sixth.

23, 3, 1913.

General condition of the patient much improved so that he can run about 200 meters without fatigue. Size of heart diminished; transverse diameter 10,5 cm. Number of beats lying down 96; standing 124; pulse arrhythmic, with frequent lapses. Tmx. = 110. The patient was not seen for a month and a half and only returned on the fifth of June, 1913. His general condition was good, and he no longer had fatigue or edema. Heart-Apex in fourth intercostal space on a level with the mammillary line. 7,5 cm. from the midsternal line. Right margin 3,5 cm. First sound slightly muffled; mesosystolic murmur at apex. Pulse regular and arrhythmic. Beats lying down 70; standing 82; after exercise 96; decreasing rapidly to 82. The

patient was re-examined in January 1919, about six years later. He had signs of slight cardiac insufficiency, dyspnea on exertion, acceleration of the pulse after slight exercise and change of position. Heart enlarged, apex beat outside the mammillary line. Precordial shock wide and somewhat violent. First heart sound slightly muffled and lengthened. No murmur. Pulse rhythmic, with no discordancy between the cardiac and radial beats. The enlargement of the heart was confirmed by an X-ray examination made by Dr. R. Duque-Estrada.

Record no. 39.

The radial pulse and heart tracing show frequent lapses which correspond to each other; they appear irregularly, sometimes alternately, sometimes after two or three beats, followed by the dominant rhythm. These also do not occur in perfectly equal cycles. In the venous pulse the *a* waves are at a rapid rate, 115 per minute and rhythmic. The *a c* interval is variable, augmenting gradually after every lapse of the pulse, as may be seen more clearly from *a XXI c* onwards. The lapses of the pulse are due to the irregular block of the auricular waves.

Record no. 39-A, 39-B, 39-C, 39-D, 39-E, 39-F. II, III, IV, V, IV

Show the effect of half a milligram of atropine. In tracing II there is nothing noteworthy, besides what has already been said of I, except that the rate of the auricular wave beats is less rapid.

Record no. 39-B (III).

Taken 20 minutes after the injection of atropine shows that the rhythm of the radial beats is already reestablished, and the *a c* interval is normal. Acceleration of the beats is minimal, 103 to 109, and the action of the atropine on the conductivity lasted an hour, although it is to be noticed that the rate is slower, 103 to 100.

Record no. 39-F (VI).

Shows that the effect of the atropine on the conductivity is diminishing; the *a c*

interval is already much lengthened, being equal to four tenths of a second. On the other hand, the rate is much less than before the injection of atropine, 92 to 103. The conductivity is kept in good condition by the use of belladonna with the rate comparatively slow: 85 beats per minute.

Record no. 39-H (VIII).

Shows a return to the former state of partial block, with varying lapses in the conduction. The patient had left off taking belladonna some days before this tracing was taken.

Record no. 39-I (IX).

Taken about six weeks after the last; this tracing is quite normal: rhythmic beats; conductivity reestablished as indicated by an *a c* interval of normal length.

Record 39-J (X).

This tracing was taken six years after the preceding one. The pulse is perfectly rhythmic, the restored conductivity continues. Compression of the eye-balls retards the cardiac beats very much and remains after the compression has ceased. The *a c* interval is normal even after ocular compression.

Observation no. 41.

Partial block. Rhythm 2:1.

M. P. F., mulatto, male, 30 years old, laborer, single, resident at Andrade Quicé.

Examined May 4, 1913. The patient had always been well until about two years ago when his present illness began. Fatigue and palpitation with heavy heart thumps. He had edema which disappeared. Some two years ago the patient had malaria. A few days before he came to consult us he had caught cold and had catarrh in the nose and bronchi at the time of examination. Liver and spleen much enlarged and painful on pressure. Thyroid very much enlarged. Glands generally enlarged. Heart much increased in size. Apex 12 cm. outside the

midsternal line; right margin 4 cm. Precordial shock large being perceptible in the third, fourth, fifth and sixth. Apex in the sixth space, and passes the mammillary line by 2,5 cm. First sound lengthened and muffled. Mesosystolic murmur, not propagated. Second sound tympanitic and accentuated in the pulmonary area. Pulse ample, full and slow. Beats of the neck veins very easily visible, twice more rapid than the radial pulse. Atropine test. (See records).

May 5, 1913.

Yesterday's examination was made after a long journey and an attack of malaria. After a rest and quinine.

Heart perceptibly diminished in size. Apex in the fifth space, a little outside and below the nipple, 11,5 cm. from the mid-sternal line. First sound clearer, with inconstant murmur when lying down, which disappears when the patient is standing. Number of beats lying down 70; standing 40;. When standing, the beats are regular and slower; during the long pauses there are auricular contractions to be heard. After the patient has been standing for some time they become irregular; there appear rapid and slower series in succession. Number of beats variable, ranging from 66 to 82.

This takes away the value of the Katzenstein test, during which the following interesting fact was noticed; at the beginning of the compression of the femorals the heart became perceptibly dilated; apex beat in the sixth space. When the compression was continued for five minutes, it returned to the fifth space as before compression.

Record no. 40.

Partial block. Ventricular and nodal extrasystole. The venous tracing shows rhythmic *a* waves at a rate of 107 per minute. The *c* waves follow each other alternately after each *a* wave, the *a c* interval lengthened. The retarding of the ventricular systole thus caused, and the frequency of the auricular rhythm, make the *a* waves fall alternately in the refractory phase of the former systole, so that they are blocked.

The block is consequently in 2:1 rhythm. In some cycles, for example in F, the rhythm returns to 1:1 with the *a c* interval much lengthened.

The beats of cycle P are probably extrasystolic and of ventricular origin.

Record no. 40-A (II).

The injection of 1 milligram of atropine did not improve the conductivity; the block persists in 2:1 rhythm.

Record no. 40-B (III).

The auricular rate has become slower—84 instead of 107 beats; conductivity improved, cycles of 1;1 rhythm with *a c* interval much lengthened. At some points the block reappears in 2:1 rhythm. Some beats, such as those of cycle XIII, are frankly extrasystolic and of nodal origin, both the auricular and ventricular beats being premature. On the heart tracing the auricular waves are seen very clearly.

Observation no. 42.

Partial block 2:1. Cardiac insufficiency. Sudden death.

M. D. M., white, male, 52 years, married, resident at Porto Manga.

Examined October 14, 1912. Previous history. The patient mentioned attacks of malaria and venereal chancres, but not rheumatism. He has been ill for about 12 years with fatigue, edema and sensation of gastric distension. He has had no attacks, or palpitation, either now or before, and is a well-grown man of robust constitution. Very marked paleness; general edema, more marked in face and lower limbs. Coughs and has dyspnea on exertion. Feels no discomfort when lying down. Frequent giddiness. Heart enlarged, apex beat in the fifth space 11,5 cm. from the midsternal line. Right margin 3,5 cm. from the midsternal line. Base: 15 cm.; height 8 cm. First sound preceded by a sound and substituted by a prolonged murmur during the whole systole, propagated towards the axillary line and not

audible at the back. Second sound clear. In the middle of the ventricular diastole is heard a tone accompanied by a slight shock with venous wave in the veins of the neck. These and those of the arms and thorax are turgid. Pulse 36, not varying or only very slightly in different positions. 37. Tmx.=160. The atropine test produced a slight modification of pulse, as can be seen in tracings. Liver and spleen very much enlarged with moderate goiter.

This patient stayed three days in Laspance and returned afterwards to his home, three leagues away. On arriving he died suddenly.

Record no. 41.

October 14, 1912.

This is a tracing of partial block. There are three very clear waves to be seen in the jugular tracing; 2 of them form an *a c* group which is repeated in regular periods of 17 tenths; the *a c* interval is variable; sometimes there are 2 tenths, sometimes slightly more or even 3. In the midst of each interval between 2 *a c* groups appears an *a'* wave which is not transmitted to the ventricle so that the ventricle beats just twice less often than the auricles. The V wave is slightly marked and in some places does not even show. At some points the wave V is however clear so that it can be marked precisely. The seventh and eighth beats of the tracing show rather anomalous intervals. The seventh is shorter and the ninth longer; one might suggest an extrasystole followed by a compensating period of rest. The analysis of the jugular pulse shows, however, that it is really a variation in the time of production of the stimulus. The auricular waves appear clearly in regular succession; they are slightly accelerated in group 7 and retarded in group 8. Tracing 3a taken 20' after injection of 1,001 of atropine shows nothing noteworthy except that the atropine has had no influence either on the auricular or ventricular beats. This has also been noticed by some authors without a satisfactory explanation. In tracing IV taken 80

minutes after the atropine injection there is some improvement in the conductivity; there appear 2 beats in 1:1 rhythm. It is remarkable that far from the auricular rhythm being accelerated, it is on the contrary slackened, 70: 65.

Observation no. 43.

Partial block, 1, 2, 3. Agony. (avexame).

I. N. C., mulatto, male, 20 years old, laborer, single, resident at Jatobá.

About ten years ago he had intermittent fevers and mentions nothing else. This patient has had fatigue, pains in the legs, pain in hypochondrium, attacks of palpitation with very rapid precordial beat accompanied by agony. Giddiness, especially when getting up. He does not mention attacks. Has had edema but not at the time of examination.

Under-sized individual. Marked paleness. Heart enlarged. Apex in fourth space in the mammillary line. Right margin 4 cm. from the midsternal line. Precordial shock diffuse and strong, shock double as if reduplicated in the pulmonary area. First sound prolonged, weak, without murmur. Second sound reduplicated and accentuated in the pulmonary area. Mesosystolic murmur in the pulmonary area. Pulse irregular, arrhythmic and very variable. Sometimes slow, about 50 beats, sometimes rapid, with extrasystoles chiefly brought on by emotion. Number of beats lying down 52; standing 62; after exercise 66. Beat of neck-veins very clear with more waves than the radial pulse. In the ventricular pauses is heard a muffled tone of auricular contraction which is not transmitted.

Katzenstein	{	Before - 65 Tmx. 135.
		During - 68 Tmx. 145.
		After - 54

When standing the number of pulsations rises to 76, and after exercise to 84.

Liver not enlarged. Spleen enlarged. Marked gastric dilatation. Thyroid much en-

larged. Inguinal and epitrochlear glands slightly enlarged.

Records nos. 42 and 42-A.

The tracings are identical, varying only as to the velocity of the record.

The tracings of heart and pulse are analogous and show irregular and arrhythmic beats, the origin of which can be traced by the analysis of the jugular pulse. Here the *a* waves are seen succeeding each other very frequently; some of them are blocked. The block is irregular, sometimes in the proportion of 2:1, sometimes 3:1. At some points, as at beat XI, it is difficult to decide whether the rhythm has returned to 1;1 with a greater lengthening of the *c* interval or whether the beat is extrasystolic.

Observation no. 44.

Partial block. Stokes-Adams' syndrome. Harmful action of digitalis.

A. A. C., male, 39 years, resident at Curvelo.

Examined December 12, 1914.

The patient does not mention lues nor are there any signs of it. He has had palpitations now for four years, and for the last two has had passing giddiness. About one month ago he had giddiness causing him to fall and lose consciousness; the giddiness lasts for some seconds but occurs rarely. After the use of digitalis had giddiness more often.

Pulse slow, 34 radial pulsations to the minute. Sometimes cardiac bigeminism, the second beat not being perceptible in the pulse. Cardiac area frankly increased.

Liver enlarged.

This patient came back to consult us six weeks after the first examination. He has had no more giddiness. Pulse more rapid, 60 beats. There are lapses in the radial pulse which correspond to long cardiac diastoles in which a tone, probably of blocked auricular systole, is heard. In the in-

terval between the two examinations the patient stopped taking digitalis.

Record no. 43.

The radial pulse is irregular and arrhythmic. In the jugular tracing the *a* waves follow one another regularly but are irregularly blocked, sometimes with rhythm 2:1, and sometimes with 3:1.

Observation no. 45.

Total block. Stokes-Adams' syndrome.

M. A. L., male, 54 years of age, resident at Curvelo.

Examined October 22, 1914. Previous history unclear; no sure indication of syphilitic infection, of which there are no traces. The patient complains of epigastric anxiety, sensation of oppression and vague pains in the precordial region. For seven months he has had attacks of giddiness for a minute or more, without convulsions, brought on by a short run or any other effort, sometimes coming on at short intervals for some hours on stretch. Copulation always brings on these attacks. Abundant eructation with aerophagy. Pulse slow, 32 beats, lying down or standing. Cardiac auscultation during the long pause reveals a short tone of blocked auricular contraction at varying intervals from the cardiac systoles.

Record no. 44.

The radial pulse accompanies the cardiac tracing in slow beat—32 a minute. The waves are regular and rhythmic. The venous tracing shows more frequent auricular waves, 65 per minute, but not in constant relationship to the ventricular systolic waves. The block is total, the ventricle following its own rhythm.

Observation no. 46.

Total block. Stokes-Adams' syndrome.

J. O., male, white, 12 years, resident at Gustavo de Silveira.

Examined July 9, 1912.

Family history: Mother strong, 45 years old; father died suddenly; he suffered from heart trouble. Eleven brothers and sisters alive and strong; one brother died at the age of three and one was still-born. Personal history; the patient was strong until seven months ago and does not mention having had any illness until then. About seven months ago he began to have dyspnea on exertion, at first only when he had to walk far and fast; now he has it even when he walks slowly on flat ground. Weakness in the legs. Giddiness. No edema. Has had fainting fits and loss of memory, sometimes with convulsions.

The boy is well developed, pale and has myxedema in a slight degree. No edema. Heart enlarged; apex beat in fifth space, 9 cm. from the midsternal line. Sounds quite audible. First sound is accompanied by musical systolic murmur within the apex not propagated. Second sound reduplicated and reinforced in the aortic area; at times an isolated tone of auricular systole, which is not transmitted, is to be heard at the ventricular diastole, 37-38 a minute. Liver enlarged. Spleen not enlarged. Thyroid enlarged. Nervous system negative. Respiratory system negative.

July 19, 1912.

Between the two examinations the patient had three attacks. Slight edema of face and limbs. Pulse 37; physical condition of the heart unchanged.

Record no. 45.

A classic total-block tracing. The radial waves and the apex beats succeed each other rhythmically; pulse slow; 37. In the jugular tracing the *c* waves show no stable relation to the *a* waves; they are more numerous, 100 per minute. The *a* waves succeed each other rhythmically; sometimes they are elevated, at others less marked, according to whether they do or do not fall within the ventricular systolic period. C. V. (E interval). No atropine test.

Observation no. 47.*Total block. Stokes-Adams' syndrome.*

P. C. G., male, white, 18 years old, resident at Morro da Garça.

The patient says he had ill-defined attacks when a child; he has frequent attacks of giddiness and about six months ago convulsions. Six months ago he had milk-pox. Complains of gastric pains with sensation of weight in the stomach. Intestinal functions normal. Liver enlarged and painless. Spleen slightly painful and enlarged. Heart much enlarged, measuring 16 cm. at base. No murmur, but after each normal systole is heard a muscular sound, apparently due to the contraction of the auricle, which is irregularly heard. Pulse arrhythmic; slow; sometimes there seem to be extrasystolic waves. 36 pulsations a minute. $T_{mx.} = 135$. Pulse lying down 36; standing 44.

Record no. 46.

July 5, 1911. Pulse slow and rhythmic, 34. Cardiogram more frequent than the pulse, not full, with the auricular waves well marked in the diastolic phase and falling at varying distances from the ventricular systoles. The jugular tracing shows the *c* waves placed in varying relations to *a*, there being no dependence upon the latter. The *a a* waves are frequent and rhythmic, 93 per minute. The *v* waves show nothing noteworthy. It is a record of easily interpreted complete block.

Observation no. 48.

Ventricular extrasystole evolving towards complete block.

J. C. F. R., male, white, 17 years old, native of Lassance. This patient, who has a goiter with a generalized enlarged thyroid, came to the Hospital for the first time in May, 1911. He had slight indications of cardiac insufficiency and extrasystolic arrhythmia. In January, 1915, about four years later, he came back to consult in an asystolic crisis, with slow and arrhythmic pulse

The asystolic symptoms were improved, the edema disappeared, but the arrhythmia remained, with slow pulse. Heart much enlarged. No murmur, or valvular lesions.

Records no. 47 and 47-A.

The records show the evolution of the arrhythmia which is wholly extrasystolic in the first record, and three years and a half afterwards has become a complete block with extrasystoles.

I) The collective record rhythmic, the dominant rhythm interrupted by premature beats of ventricular origin, with complete compensating period. *a c* interval not lengthened. Nothing else worth mentioning.

II) Taken three years and a half after the former.

The radial pulse is slow and irregular; there are a few extrasystolic beats. The cardiac record is defective, and furnishes few data. In the jugular record *a* waves are seen succeeding each other regularly and much more numerous than the ventricular beats *c* waves. The relation between *a* and *c* varies every moment; at times they fall together, at others *a* comes before *c* in varying time, sometimes it comes after *a*. The *c* waves of the ventricular extrasystole interrupt the succession of *a* and *c* which are regular, without any accidental connection which might be attributed to the auricular waves. The record is of complete block, with ventricular extrasystoles.

Observation no. 49.

Alteration of conductivity with extrasystole. Evolution of morbid process till complete block.

M. F., male, 23 years old, white, resident at Lassance.

He came to consult us stating that periodically he has vertiginous crises with loss of consciousness. Previous history unknown. Lives in a house infested by *Triatoma*. Family history: His parents had extrasystolic arrhythmia and hypertrophy of thyroid gland. Three brothers with goiter and al-

ternation in the functioning of the heart. One brother with goiter, extrasystolic arrhythmia and cardiac insufficiency. Four daughters with signs of *trypanosomiasis*. Wife of healthy appearance, has had no miscarriages.

At time of examination: Robust appearance, below medium height, well-proportioned, muscular. Hypertrophy of thyroid gland. Liver and spleen enlarged. No edema. Extrasystolic arrhythmia. In this phase the extrasystoles were frequent after groups of five, six or seven normal systoles.

April 9, 1910.

Cardiac pulsations 62; radial 45. The patient only came back to consult us on the 22nd of November; he had rare extrasystoles, evident alterations of conductivity; pulse lying on his back 48; standing 45.

At present no crises of giddiness.

February 7, 1911.

The alterations of conductivity are very distinct. Pulse 44. The patient was not seen, again until 1913, when he came back on the fifth of March with the following symptoms: Dyspnea on exertion and when lying down so that he can only rest propped up. Nocturnal dyspnea marked. General weakness, no giddiness, nor vertiginous crises. Appetite good. Liver enlarged and painful on pressure in the epigastric region. Spleen slightly enlarged. Heart: apex in sixth space, below and outside the nipple, 11 cm. outside the midsternal line. Right margin 5 cm. outside the midsternal line. Precordial shock ample, with perceptible beats in fifth and sixth intercostal spaces. First sound very muffled, accompanied by inconstant murmur in the strong beats, with slight propagation towards the axilla. In the long intervals after the second sound are heard short slight tones without repercussion on the radial pulse, coinciding with the rises of the neck veins. The second sound reduplicated with pulmonary accentuation, muffled in the aortic area. Pulse ample, slow and regular. Extrasystoles at long intervals. Forty-two beats. Venous pulse very clear.

Like the cardiac beats, it shows that there is complete disconnection between the auriculo-ventricular beats.

May of the same year.

Condition of the heart the same. Liver diminished. Subjective signs much better.

July of same year.

Fatigue on exertion and walking. The patient sleeps well and has no dyspnea at night or when lying down; some attacks of giddiness. Examination of heart: Apex in the sixth space a little outside the mammillary line and 10 cm. from the midsternal line. Shock but slightly perceptible. Right margin 4 cm. from the midsternal line. First sound muffled but with irregular tone, sometimes being much higher and more intense. Auricular tone audible in different phases of diastole, sometimes nearer and sometimes further from the next ventricular systole. The coincidence of the auricular and ventricular beats seems to explain the greater intensity of the sound at times. Pulse: lying down 42; standing 46. Tmx.=135. Liver slightly enlarged, painless. Thyroid perceptibly enlarged.

September 9, 1913.

Dyspnea on exertion, even when walking normally at ordinary pace and on even ground, much worse when he goes uphill. No nocturnal dyspnea. Heart much enlarged, first sound lengthened and muffled. Second sound also muffled, sometimes reduplicated. The auricular beats are also heard well and are not followed by ventricular contractions. 40 beats with a few extrasystoles. Terminal phase: The patient was not seen for some months. In May, 1914, he came back in asystole with general edema, dyspnea, congestion of the bases of the lungs, turgid neck veins, liver enlarged, etc. Heart greatly enlarged with dilatation of all the cavities. He died at home in this state, and a post-mortem was not made.

Records no. 48, 48-A, 48-B, 49-C, 48-D.

These tracings show the evolution of alteration of the rhythm from extrasystole to complete block.

I) Radial record, on the right side, in which is seen the dominant rhythm is rapid, 100 a minute.

In this record are seen slow beats occupying the space of two cycles of the dominant rhythm; they are probably due to extrasystoles which are not represented in the radial pulse. At some points they are shown by slight beats. The third beat is succeeded by two consecutive extrasystoles.

II) Taken about six months after the first; radial record similar to the cardiac one, slow beats interrupted in their succession by cycles of shorter diastolic phases. The long diastolic phases are perceptibly twice as long as the short ones.

In the cardiac record small waves of auricular contraction are to be seen in the long diastolic phase, but none of ventricular systole.

In the jugular pulse the *a* waves are marked rhythmically. They do not however all have a corresponding ventricular wave as many are blocked. The block is in 2:1 rhythm corresponding to the slower ventricular beats and returns to 1:1 in the rapid ventricular beats. The *a c* intervals, where the rhythm is 1:1, as in cycle XIX, perceptibly lengthened.

III) About two years later; the tracing is already that of complete block. The ventricles beat regularly at the rate of 40 per minute; nothing noteworthy in the radial and cardiac tracings.

In the phlebogram are to be seen auricular waves at an accelerated rate of 100 per minute; there is no constant relation between the auricular and ventricular rhythms.

IV) After an injection of 0,001 g. atropine there was no change in the relation between the auricles and ventricles, nor in the cadence. There appeared however a ventricular extrasystole at the left of the tracing.

V) Tracing taken little more than a year later, during an asystolic crisis. Auricular waves in the venous pulse show increased rate, 120 per minute. *A* and *c* waves completely independent. On the left side of the

tracing the ventricular beats marked in the radial pulse and in the cardiogram; they are rhythmic and at a rate of 40 per minute. On the right side they are irregular, with numerous extrasystolic beats. The patient complained of uncomfortable heart-beatings at the time.

Observation no. 50.

Total block.

J. M. S., mulatto, male, 38 years old, widower, laborer, resident at Areias. (near Curvello).

Examined June 26, 1913. Previous history only intermittent fevers. He has been ill for about three months. Until then he worked regularly without fatigue. At the time he had violent giddiness, with darkened field of vision, but without loss of consciousness or fall. This giddiness did not return but there appeared dyspnea on exertion, and agony with weight in the stomach. Lack of appetite; the taking of food in even small quantities often provoked a feeling of fulness, anxiety and fatigue. These symptoms became gradually worse, and lately he has had dyspnea when lying down and at night, preventing him from sleeping, and also edema of lower extremities. Does not complain of palpitation, and does not feel the heart beats. The patient is of medium height, well built and robust.

At present edema of the lower extremities, neck veins turgid. Abdomen distended. Dyspnea on lying down so that the patient cannot occupy a horizontal position. Very emotional, much impressed by the examination. Heart much enlarged. Apex beat in the sixth intercostal space, between the mammillary and axillary lines. Right margin 4 cm. from the midsternal line. Transverse diameter 15,5 cm. Precordial area very prominent. Precordial shock slow, diffuse and undulated. First sound muffled and lengthened. Second sound reduplicate, with pulmonary accentuation. No murmur. Beats arrhythmic with extrasystoles and long pauses. During the long pauses

es sometimes a muffled sound to be heard; it coincides with wave in the veins of the neck and not to a ventricular contraction; an auricular contraction sound. During the examination numerous extrasystoles occurred, almost all of them not shown in the radial pulse. Heart beats 59. Pulse 36.

Katzenstein { before - 59 Tmx. 160
during - 66 Tmx. 160
after - 69

The extrasystoles were probably due to the strong emotion brought on by the examination. Liver enlarged and painful on pressure. Spleen also (Previous malaria). Thyroid enlarged, with large goiter.

Examined June 27, 1913. Patient calmer. Occasional extrasystoles. Auricular sound very audible. Pulse varying between 32 to 35. Standing: 40.

Katzenstein { Before - 36 Tmx. 168.
During - 44.
After - 44.

The increasing of the beats after compression of the femorals is due to the numerous extrasystoles. Atropine test: While the effect lasted the pulse became regular. Iliac compression hardly alters the number of beats.

Before compression: 36

During " : 36

It is interesting to note that emotion and compression of the femorals brings on extrasystoles.

Record no. 49.

February 27, 1913.

Pulse interrupted by a beat of a lesser cycle followed by the dominant rhythm. The cardiac record shows the same succession of beats with the same rhythmic cycle of 9, interrupted by a lesser one of 6. In the diastolic phase are seen diastolic rises corresponding to the rises of the jugular record. Jugular record with a series of rises of equal cycles not in constant relation to the *c* waves; some are not accompanied by

ventricular contraction. They are *p* waves which are not transmitted to the ventricle. Total block.

Taken 35 minutes after 0,001g atropine.

The number of ventricular beats has not changed much; 34:35. The number of auricular beats diminished 100:76. The independence between auricular and ventricular contractions continues.

Observation no. 51.

Complete block. Sudden death.

A. F. C., male, white, 29 years, old, laborer, married, resident at Maquiné.

Previous history: attacks of intermittent fever and gonorrhoea some years before. Has been feeling ill for about a year, fatigue on exertion, cannot walk fast, nor go up-hill, as he is so easily tired. He could however do ordinary farm work. About five months ago he became considerably worse, fatigue increased, his stomach seemed to have swelled and has pains like a belt round his stomach and gastro-intestinal pneumatosis. The dyspnea has become gradually worse and comes on after the slightest effort or even when at rest, specially when lying down and at night, so that he can only sleep propped up. At times cannot sleep at all on account of nocturnal dyspnea and cough. Lately, he has had edema of lower limbs, which afterwards extended. Palpitation with uncomfortable heart beats. Has an uncomfortable feeling on account of the extrasystoles which he says 'makes his heart beat twice in one beat' and is accompanied by uncomfortable feeling of fatigue. At times he feels rapid, strong and very uncomfortable beats.

Present condition, general edema. Dyspnea which gets worse after the slightest effort or when lying down.

Turgidity of neck veins with well-marked pulsation. Prominent swelling of the precordial region. The area of cardiac dullness very much increased. Apex beat in sixth space, beyond the anterior axillary line, 18 cm. from the midsternal line. Pre-

cordial shock ample and slow. First sound lengthened and muffled, substituted in the tricuspid area by a systolic murmur which is hardly audible at the apex. Second sound muffled in the pulmonary and aortic areas. Cardiac rate slow, 48. At the beginning of the examination there were numerous extrasystoles which became rarer in the course of examination. Beats of the neck veins more frequent than the ventricular systoles. Venous pulse positive with systolic thrill in the veins at the right side of the neck. In the intervals of the ventricular systoles, at times an isolated tone is heard; it is synchronous with the venous wave of the jugular, without ventricular contraction. at times producing a slight diastolic impulse; isolated systoles of the auricles. Pulse irregular and arrhythmic on account of the extrasystoles which are sometimes perceptible in the jugular pulse and at others not. Liver enlarged. Slight peritoneal effusion. Bronchopulmonary edema. Thyroid enlarged, principally the right lobe.

October 31, 1913.

Improvement of all the symptoms, both subjective and objective. Dyspnea decreased, as also palpitation and edema. The patient still feels his heart "*beat twice in one stroke*". No more tricuspid murmur. Area of dulness much diminished: apex 12,5 cm. from the midsternal line; right margin 5,5 cm. Cardiac beats irregular and arrhythmic with frequent extrasystoles; 47 beats per minute, but the number varies according as to whether there are more or fewer extrasystoles.

November 3, 1913.

Improvement continues. Ascites diminished, though not entirely gone. Slight edema of body. Cardiac dulness not modified in relation to last examination.

Tricuspid murmur again to be heard. Heart beats 33, 44, or 46 according to the number of extrasystoles. At beginning of examination extrasystoles in bigeminal series.

November 5, 1913.

Improvement more marked. No more tricuspid murmur. Heart area reduced, principally at the right side. Cardiac beats slow and regular.

November 6, 1913.

Improvement. Cardiac area reduced; apex 11.5 cm. from the midsternal line; right margin 4 cm. from the same. Cardiac beats regular and slow. Sounds more distinct. No murmur. Neck veins no longer turgid. Extrasystoles, sometimes in bigeminal series. 50 beats. The reduplication of the first and second sounds clearly heard, the latter with pulmonary reinforcement. Palpitation rare.

November 10, 1913.

After a relapse brought on by neglect of treatment, the patient improved again. He was not observed for about two months.

Examined January 12, 1913.

Condition like at the last examination with symptoms of asystole. Number of beats varies around 50, according to the greater or lesser frequency of extrasystoles.

January 16, 1916.

General condition improved without reduction of the area of dulness. Number of beats: 46 per minute. On the 20th when going to the Hospital, he died suddenly. A histopathologic examination of the heart was made by Professor B. C. Crowell who confirmed the existence of the lesions characteristic of Chagas' disease, which he has described in his paper on the subject.

Record no. 50.

The beats of the cardiac and pulse tracing show a slow rate with some extrasystolic beats, which are not all marked on the latter, producing long pauses of the pulse. The extrasystoles are ventricular as is shown by the venous record. In this the *a* waves are seen completely dissociated from the *c* waves. It is a case of complete block with frequent extrasystoles which the patient felt as uncomfortable beats.

Records no. 50-A, 50-B.

They show that atropine had no influence on the conductivity, accelerating the auricular beats slightly. The ventricular rate slower owing to the lesser number of extrasystoles.

Observation no. 52.

Complete block. Stokes-Adams' syndrome.

I. F., mulatto, female, 20 years, single, resident at Bananal, near Lassance.

Less than average height. Earthen paleness. Cretin-like appearance with slight exophthalmia. No noticeable asymmetry of face. Complains of fatigue, giving way of legs, general trembling which prevents her from working. Strong, measured heart beats, bringing on agony, a feeling of oppression, agony in the chest and the throat. Almost constant feeling of depression and discouragement with weeping fits without any real cause. She has had giddiness and vertigo, sometimes with loss of consciousness. Constant uncalled-for sadness, always agony; general limpness. During the examination, the emotion brought on an attack of strong and uncomfortable beats. The rate and rhythm of the beats, however, did not differ from those observed at other times. Dyspnea on exertion and when lying down. She has been ill for a long time, having grown worse in the last three months.

Does not mention edema before, nor has she any at present.

Heart much enlarged, apex beat in the sixth space at the level of the anterior axillary line. Right margin 4,5 cm. from the midsternal line. Transverse diameter 16,5 cm. Precordial shock strong and diffuse. First sound lengthened and accompanied by murmur audible in the whole of the cardiac area, with slight propagation towards the axilla. Second sound reduplicated. Pulse slow and ample, the number of beats varies from one moment to another, oscillating between 27 and 32. Different polygraphic records were taken. The atropine test

was also made and did not increase the number of pulse beats. Liver enlarged, painful on pressure. Spleen slightly enlarged (previous history includes malaria). No marked digestive trouble. Appetite good; digestion good; does not complain of indigestion. Thyroid enlarged, with large goiter. Regular menstruation at right time, scanty, accompanied by abdominal pains and colics, general condition being much worse at such times. Feeble intellect. Apathetic. Uneasy and uncertain movements. Patellar reflexes slightly exaggerated. During the two and half months in which the patient was under observation there was little change in her condition. The "agony" continued and also the inability to work, frequent fits of weeping, undefined and constant sadness. Sleeplessness. Profuse perspiration especially of hands and feet. Cardiac area unchanged. Pulse varying between 27 and 36.

Examined March 7, 1911.

Complains of general indisposition, headache, profuse perspiration, pains in her legs, giddiness even when lying down; all these symptoms have become worse in the last few days. On the day of examination she had bilious vomiting with marked epigastric feeling of anxiety. Icterus. Furry tongue, with constant bitter taste. The patient complains of heat although the temperature does not rise above 36,8. Rapid emaciation in the last few days. No perceptible change in heart as compared to the result of former examinations. Cardiac area not modified. Pulse, 32 beats. Liver enlarged, but not very much. Urine: no albumen. Abundant bile pigments. This state with a slight change for the worse until ninth. From the ninth to the tenth frequent convulsions came on with loss of consciousness. Pulse feeble, oscillating between 25 and 28. Superficial respiration interrupted by deep sighs. The attack came on at ever shorter intervals, the patient comatose, the coma lasting for about twelve hours. During the coma the pulse was irregular, sometimes slower, sometimes more rapid, at times very slow with pauses of from 10 to 15 seconds.

Convulsions, fits of agitation, with intervals of rest. Death followed from heart-failure. A histopathologic examination of the heart was made by Professor Crowell, this being one of the cases which he has used for his description of the specific lesions of the heart in the chronic form of Chagas' disease.

Record no. 51

Pulse and cardiac tracing slow, 32 beats. The phlebogram shows that the *a* waves appear more frequently than the *c* waves, without keeping up constant relations. Total auriculo-ventricular disconnection.

Record no. 51 A.

The disconnection persistent. The injection of atropine half an hour previously did not improve the conductivity. The number of auricular beats diminished a little, 61 beats, instead of 66, as in the former tracing.

Record no. 51-B.

An hour after the atropine injection the auricular rate has returned to 66 per minute without changing the auriculo-ventricular dissociation.

Observation no. 53.

Total block. Stokes' syndrome.

M. R. L., white, female, 38 years old, resident at Curvello.

The patient states that she has had undefined attacks of fever for about five years. About 2 year ago she had fever again, with edema. Traces of hypoovarism, deficient menstruation. Very marked bronze coloring. Thyroid slightly hypertrophied. Convulsive crises, which come on at irregular intervals of a week or more. During the crises the convulsions sometimes recur every five or ten minutes. Painful sensations. A feeling of pain and prickling over her heart. Pulse slow, 33 beats, accompanied by cardiac beats. Sometimes extrasystoles. The analysis of the tracing shows that it is evidently a case of total block.

Record no. 52.

Radial pulse ample, slow, beating at unequal intervals and with unequal amplitude. A perfect analysis of tracing cannot be made as the marking of the time was defective. Heart tracing ample, the auricular waves with exceptional rises; it is only by their location in different points with relation to the rises of the ventricular systoles that the diagnosis of cardiac block can be made. The jugular pulse confirms this diagnosis. The *a a* waves are very frequently repeated, and rhythmically, not in relation to the *c c* waves. For each group of 10 *c c* waves there are more or less 26 *a a* waves. The block is total.

Observation no. 54.

Total block. Stokes-Adams' syndrome.

G. S., white, male, 50 years old, married, resident of a place near Lassance.

First examined April 9, 1910. Previous history obscure; he only mentions vague attacks of fever. For about ten years he has had giddiness and fainting fits which last from fifteen to twenty minutes. Hypertrophy of the lateral lobes of thyroid. Pulse slow, 37 beats; number of heart beats the same. On auscultation there seems to be a murmur added to the second sound, probably auricular.

April 13, 1910.

Pulse beats equal to heart beats; that is 29. General condition middling. Dyspnea on exertion, after walking quickly, or heavy work. When walking at ordinary pace the patient can however do four leagues and more without much fatigue. No dyspnea when lying down and sleeps well in horizontal position. No edema and no more giddiness. Heart enlarged, apex beat in fifth intercostal space at the level of mammillary line. Transverse diameter 14 cm. First sound lengthened and muffled. Systolic murmur at apex with small area of propagation. Second sound reduplicated with pulmonary accentuation. During the diastole a

muffled isolated tone is heard corresponding to auricular systole. Pulse rate: standing 25; lying down 24.

Katzenstein { before - 24 ps. Tmx. 105.
during - 25 ps. Tmx. 100
after - 25 ps.

Liver and spleen not enlarged.

Examination August 17, 1913.

General condition middling. Subjective symptoms almost nil. No edema. Heart enlarged. Transverse diameter: 15,5. Pulse rate 29, not changing in different positions. Tmx. = 130. Sounds muffled. Auricular tone with non-transmitted auricular systoles, distinctly audible during the ventricular diastole. Beats of neck veins very visible. Liver slightly augmented, not painful on pressure. The patient died suddenly in 1916 whilst at work.

Records nos. 53, 53-A and 53-B.

I) The radial and cardiac records similar; beats slow and rhythmic, rate 30 per minute.

In the venous record there are more frequent auricular waves, one ventricular wave to every three auricular waves, of which two are blocked; there is regular block in the rhythm of 3:1. Is it block or simply coincidence that the auricular is three times more accelerated than the ventricular one? It is impossible to decide this point by the record of the tracing.

II) and III) These records taken at intervals of one or two years from the preceding clearly show the complete block. The ventricles beat at their own idioventricular rhythm. In record II the beats are not rhythmic, there being slight variations between one cycle and the other.

Observation no. 55.

Total block.

J. C. B., white, male, 30 years old, resident at Paraopebeba.

Previous history obscure. The patient complains of dyspnea on exertion and gid-

diness. No vertigo. Lateral lobes of thyroid hypertrophied. Liver enlarged. Pulse slow and regular, accompanying the heart beats. Rare extrasystoles. Pulse rate 34 beats. The simple inspection of neck veins already shows distinct signs of alterations of conductivity.

Examined August 24, 1912.

General condition middling, with relative compensation. Good appetite, normal digestion. No dyspnea either at night or when lying down. Dyspnea on exertion. No giddiness or seeming epileptic fits. No edema. The patient states that his feet and hands go to sleep and feel cold. Heart enlarged; apex beat 11.5 cm. from the mid-sternal line. Right margin 4 cm. from the same. Sounds muffled. In the ventricular diastoles is heard a short tone of isolated contraction of the auricles, without fixed relation to the ventricular systoles. 38-40 beats to the minute. Tmx. = 110. Liver enlarged, hard, painful on pressure.

Records nos. 54 and 54-A.

February 12, 1911.

Cardiogram and sphygmogram of slow regular rhythm, exactly corresponding to one another. In the diastolic phase on the cardiogram are seen auricular waves which are not in relation to the ventricular systole. These waves are also marked in the catacrotic phase of the pulse. It is these characteristics that allowed us to affirm that there is a total block which is confirmed by the analysis of the venous record. The *a* waves more frequent than the ventricular waves which do not depend on them. The ventricles beat according to their own idioventricular rhythm. Record 54 a is not to be distinguished from the preceding; it shows the persistence of the block at an examination made a year and a half later.

Observation no. 56.

Auricular tachysystole (auricular flutter)

F. N., male, 25 years old, resident at Lassance.

May 15, 1911.

Sign of cardiac insufficiency. Palpitation. Rate standing 32, lying down 64. Arrhythmic pulse, with numerous lapses. Liver enlarged. Peripheral glands enlarged. Considerable hypertrophy of thyroid.

June 22, 1911.

General condition without perceptible change. Cardiac beats more regular, 44 when standing, 40 lying down. Tmx. = 125.

In July, 1912, he came back to consult us, complaining of strong dyspnea and palpitations.

Heart greatly enlarged, apex beat in fifth space outside the nipple. Tachycardia with arrhythmic pulse, number of pulsations varying between 120 and 130. Cardiac excitability. A rapid examination was made, and we decided to move the patient to the Hospital. On the following day we heard that he died suddenly at his own house in syncope.

Records nos. 55-A and 55-B.

I) The cardiac beats and those of the radial pulse at slow rate, 55 beats, very arrhythmic, the diastolic pause varying from one cycle to the other, although within narrow limits. In the radial record the *a* waves accelerated, 230 a minute, not all accompanied by ventricular systole. A great number blocked very irregularly, sometimes in 3:1, sometimes 4:1 rhythm. The change in the rhythm of the block explains the irregular pulse.

II) Taken 1 month after the former.

Pulse slower, 42 beats; some beats of extrasystolic aspect. The duration of the diastolic phase is very variable. The auricles beat with the same frequency, 230 to the minute. The block is more intense, the rhythm being 4:1 or even 7:1. The most frequent is 4:1.

III) About a month later another record was made; it is not worthy of special mention. The frequency of the auricles is increased to 250. This is a case of auricular tachystole with block of varying rhythm.

Observation no. 57.

Auricular tachysystole (auricular flutter)
Cardiac insufficiency.

M. A., mulatto, female, 45 years old married, resident at Curralinho.

Examined Sept. 16, 1912.

Until about two months ago she had only waves of heat with abundant perspiration. Five months ago menstruation, which until then was quite regular, ceased. For the last two months this patient has had nervous excitement and sleeplessness. Dyspnea at night and when lying down so that she can only sleep propped up; sleep is agitated and broken by starts. Dyspnea on exertion. General edema more marked and in lower extremities and lower part of body. Constant dyspnea and cough.

Heart greatly enlarged. Apex beat 15 cm. from the midsternal line on a level with the axillary line and much lowered. Right margin 5 cm. from the midsternal line. Systolic murmur at apex, audible at the back. First sound audible in the tricuspid area. Second sound reduplicated. No murmur in basal area. Pulse entirely arrhythmic. Tmx. = 115. Liver much enlarged, painful on pressure. Thyroid enlarged, with cystic goiter.

April 18, 1912.

Improvement of all the symptoms. Edema nearly disappeared. Dyspnea improved, so that the patient lies down. The cough has almost disappeared. Little change in the physical state of heart. Complete arrhythmia. Incipient bigeminism. 64 radial pulsations. Tmx. = 120.

September 22, 1912.

After she was discharged and getting ready to leave the Hospital, she died suddenly. The post mortem showed that death was due to a rupture of the anterior wall of the right ventricle with intrapericardial hemorrhage. The histopathologic examination was made by Professor Crowell.

Records no. 56 and 56-A.

I) The heart and pulse records show marked ventricular arrhythmia, so that the dominant rhythm could not be fixed. The beats are frequent and completely irregular; in the jugular record are seen auricular waves in rhythmic succession and with great frequency: 290 to the minute. The ventricle does not answer to all excitations, many being blocked in a variable way from one to another cycle. Some beats apparently extrasystolic though it is impossible to say if they really are.

II) Taken three days after the latter; the patient had taken digital. The number of auricular beats diminished a little, from 290 to 260. The auricular waves well marked; in the venous pulse they are irregularly blocked and the digital has intensified the block; in some points there are 7 auricular beats to 1 ventricular one. The ventricles beat more slowly on account of the more intense block, but still very irregularly.

Observation no. 58.

Auricular tachycardia with transition to fibrillation. Cardiac insufficiency.

A. M., mulatto, female, 35 years old, living in Pirapora.

Signs of cardiac insufficiency. Heart enlarged; 62 beats with extrasystoles and alterations of conductivity. Tmx.=100. Liver enlarged. Large goiter. Strong bronze coloring.

Examined April 19, 1912.

She complains of general uneasiness with great sadness. Dyspnea on exertion which has increased gradually to such a point that it appears even when resting; it is accompanied by a cough. Lately edema appeared. She has vertigo sometimes with falls. Heart greatly enlarged, the base line measuring 16 cm. The heart beats shake the thoracic wall; and the rate is sometimes 107. Many systoles are not perceptible in the pulse, where 56 beats are to be count-

ed. Systolic murmur at apex, propagating itself to axilla. Tmx.=120. Tmn.=95. Liver much enlarged. Spleen enlarged and hardened. Thyroid with some hard nodules. Congestion of pulmonary bases especially the left. The condition varies; the patient is sometimes better, sometimes worse.

July 23, 1913.

General condition worse. Stitch in left side. Intense dyspnea. Cyanosis. Bloody sputum. Slight edema of lower extremities. Cardiac beats very irregular, now slow, now fast, sometimes very frequent, with trembling that cannot be counted. Pulse varying, on an average 56 beats. Heart much enlarged. Apex seventh space, 14 cm. from the mid-sternal line. Right margin 2 cm. from middle line. Liver enlarged. Signs of pulmonary engorgement at the right base.

July 28, 1912.

Pleurisy with effusion on the right. Bloody sputum. Cardiac bigeminism (digitalic) 78 heart beats; pulse 39. No perceptible change in the size of the heart. No murmurs. Sleepiness. Vomits. Cough with bloody sputum. Pain at the right side of thorax. Cyanosis. Slight edema of lower limbs. Pleural effusion increased. Bigeminism continues. 68 cardiac beats; radial pulse 39. The pleural fluid was extracted, and was serohemorrhagic. Patient got alternatively better and worse, and died on August 22, 1912 in syncope.

Records nos. 57, 57-A, 57-B and 57-C.

I) Examination of the three records shows first the great irregularity of the radial pulse and of the cardiac beats which succeed each other arrhythmically, at a rate of more or less 93 beats. The auricles beat perceptibly twice as often as the ventricles, 170; auricular *a* waves are seen in the jugular pulse. The irregularity of the pulse comes from the block of the auricular waves, in varying numbers, from one cycle to another, and perhaps from extrasystoles.

II) This record was made about a year after the other and shows the radial pulse with the appearance of a completely ar-

rhythmic pulse. In fact there is no method whatever in the order of beats which succeed each other in a completely arrhythmic and irregular way; the cycles vary in duration from one to the other and the amplitude is not in direct proportion to the duration of the preceding pause. In the jugular pulse are seen little waves which recur with great frequency, about 500 a minute. These represent auricular contractions—almost fibrillation, which appear in the following record.

III) Was made about one month after the last one. In the jugular pulse it is seen that the ventricular waves appear as acute rises in some points and that the signs of movement of the auricles are marked by small undulations. The pulse is completely arrhythmic.

IV) Two days afterwards, under the influence of digitol, the venous pulse has the appearance of a ventricular venous pulse; there are series of bigeminal pulsations, one of which is seen in the record.

Observation no. 60.

Auricular fibrillation. Cardiac insufficiency.

E. J. M., mulatto, 50 years old, married, laborer, resident at Lassance.

Examined December 8, 1912. Previous history, malaria. For about five months he has been feeling ill with fatigue on exertion, which he had never felt before. No dyspnea on lying down or at night, sleeps well. He has had slight and transitory edema. His legs feel heavy. Giddiness at times very marked. Attacks of palpitation which come several times a day; strong and rapid heart-beats, coming on without apparent cause and causing agony. Sometimes the beats are strong and slow, "measured"; his heart seems to be "pushing". At present edema of legs and abdomen. Heart much enlarged; apex in the seventh space on a level with the midaxillary line, 16 cm. from the midsternal line. Inside apex there is systolic retraction of the fifth, sixth and

seventh spaces. Right margin 4 cm. from the midsternal line. First sound lengthened, dull, without murmur. Second sound reduplicated. Beats arrhythmic and slow. Precordial shock strong, shaking the whole precordium.

Katzenstein { before—51 Tmx. 125.
during—52 Tmx.
after—48

Liver enlarged and painful on pressure. Spleen same. Thyroid much enlarged. Dec. 9, 1911.

Palpitation during the night and early in the morning; 47-48 beats. Complete arrhythmia. Tmx.=125. Atropine did not perceptibly modify the pulse.

December 10, 1912.

Slight improvement.

December 12, 1912.

More marked improvement; was discharged. He died months later in asystole.

Record no. 59.

Pulse absolutely irregular. The beats succeed each other in varying spaces, so that it cannot be foreseen where any given beat will fall. There is also no direct and constant relation between the preceding pause and the amplitude of the beat. At first sight it seems to be a bi- and trigeminal pulse, but there are many variations. In some points there are pauses which seem to be compensating pauses and do not appear in other points. The jugular pulse has the characteristic appearance of a ventricular venous pulse. The only really distinct waves are *c* and *v*. The *a* wave does not appear on the record. Rate of beats slow. At some points of the long diastolic phases (before and after the beat) are seen small undulations, which might be considered as the auricular fibrillations which exist in these cases. The same aspect of fibrillation is seen in the heart record. It is a case of complete arrhythmia, with ventricular venous pulse and auricular fibrillation. The records I, II, and III show slow rate with complete arrhythmia and ventricular

venous pulse, which I compared with II and III shows that atropine does not appear to have modified the arrhythmia.

Observation no. 61.

Complete arrhythmia. Cardiac insufficiency. Spasm of esophagus.

P. N. A., black, female, 41 years old, single, living in Vargem da Palma.

Examined August 24, 1913.

General slight edema. Turgidity of neck veins. Gastric disturbances. Lack of appetite. Spasm of esophagus, great difficulty in swallowing solid food so that she is obliged to drink water after each mouthful. Dyspnea on exertion and when lying down.

Heart much enlarged, apex beat beyond the anterior mammillary line. Right margin 5 cm. outside the midsternal line. First sound accompanied by systolic murmur, audible at the back. Number of beats lying down 66; seated 72; standing 82. It is difficult to count as there is complete arrhythmia. Tmx. = 135. Liver much enlarged. Spleen not enlarged. Thyroid with large goiter.

August 28, 1912.

Improvement of all the symptoms by treatment; no change in cardiac area.

Record no. 60.

The radial, cardiac and jugular tracings are similar; the latter has the aspect of ventricular venous pulse. The waves are bi- and trigeminal but the intervals between them are most irregular. It is a tracing of complete arrhythmia of auricular flutter; the *a* waves do not appear on the jugular tracing.

Observation no. 62.

A. S., male, white, 33 years old, widower, laborer, resident in the neighborhood of Lassance.

Examined April 13, 1913.

Very short but robust. Marked paleness of skin with special coloring. Mucous membranes rather discolored. Slight not con-

stant edema of face. Previous history; nothing worth mentioning. Had grippe during the great epidemic. For about three years he has had a feeling of fatigue on the slightest exertion; he gets tired after quick walking or climbing up hills, or even when at his usual work. Sometimes has palpitation and feels as if his heart were stopping. He is subject to giddiness, which comes on after exertion or even without any perceptible cause. Heart not enlarged. First sound slightly muffled; second not altered; there is mesosystolic murmur in the pulmonary area. 76 beats, with frequent extrasystoles, which appear either isolated or in long bigeminal series. Arteries not hardened. Thyroid enlarged, with cystic lobules. Examination of other organs negative.

In her house were found numerous infected specimens of *Triatoma*.

Several electrocardiographic records were taken of this patient. We give n. 1 and 2 Complement fixation reaction positive (with heart of a dog infected by *Tryp. Cruzi* as antigen).

Record no. 61.

Was made with derivation 1 (right arm—left arm). The time marked is 1/25 of a second. The derivation of the galvanometer is 2 cent. to 0.001 volts. The left side of the record shows a succession of rhythmic regular contractions, in which note the deviations P. Q. R. S. and T. The PP. waves have nothing interesting about them. The space PQ is within the normal limits, less than one fifth of a second. S phase does not appear. T is inverted in all the cycles. At the right side of record are seen 4 extrasystolic beats in bigeminal series. The extrasystoles are of ventricular origin and of the type which springs from the area situated in the right ventricle. The variation of the auricular systole appears in the diphasic variation of extrasystole in normal time.

Record 2 of the same patient. Derivation III (left arm-left leg). Time 1/50 of a se

cond. The tracing shows a succession of extrasystolic beats recurring alternatively with normal cycle. The derivation *R* is bifid. *S* appears in this deviation and *T* leaves of being inverted. The extrasystoles are of ventricular origin, probably from the right ventricle. The deviations *P* keep their rhythm marked in the diphasic variation of the extrasystoles.

Observation no. 63.

F. Mor., mulatto, male, 30 years old, married, laborer, resident in the neighborhood of Lassance.

Examined April 13, 1921.

Is of medium height, well-built and of robust appearance. He did not come to consult a doctor and was only examined as he had a daughter under our care, who had an acute form of trypanosomiasis, with numerous parasites in the peripheral blood. He lives in a house infested with infected triatomas. Previous history: nothing worthy of notice. There are no subjective symptoms except rather easily brought on fatigue, and sometimes lapse of the pulse, though he did not pay much attention to this. The objective examination reveals a slight increase of the cardiac area, and numerous extrasystoles, without other perceptible alterations of circulatory system. Thyroid enlarged, with hard cysts of different sizes not being bigger than a walnut. The examination of other systems proved negative.

Complement fixation reaction positive (with heart of a dog infected by *Tryp. Cruzi* as antigen).

Several electrocardiographic tracings were taken; we give Nos. 3, 4, 5 and 5a.

Electrogram no. 3 was made with derivation I. $T = \frac{1}{50}$; 1 cm. = 0.001 volt. Two extrasystoles are seen on the right side of record, which interrupt the regular succession of the heart beats. In the beats of the normal cycles are marked waves *P*, *Q*, *R*, and *T*. The space *PQ* is normal. *T* is inverted in all the cycles; *S* does not show. The waves *P* succeed each other regularly; they are seen in the extrasystolic variation where they are marked. The extrasystoles are ventricular and come from the right ventricle. Record no. 4 came from patient taken in derivation II (right arm—left leg). Two extrasystoles are seen on the left of the record, both probably originating in the right ventricle. In the beats of the normal cycles it may be seen that *P* is frankly bifid. In the cycle which follows the second extrasystole *P* is inverted, without there being premature auricular contraction. *T* is inverted in all the cycles. It is clearly more marked in the cycles which follow the extrasystolic beats which is a common fact. *S* is deeper in this derivation, and that corresponds to the predominance of V E. Records 5 and 5-A were taken at the same time as the former ones but in D III. *P* is much more elevated; it is still bifid. The PP waves are arrhythmic. *S* is less deep. *T* still appears inverted. In both the records are seen extrasystoles of ventricular origin. The last of both the records comes from the left ventricle, the others come from the right ventricle.