which results from the combined action of a specific cell and its relation to innervation and blood supply. The effect of discord in this complex unity may be sudden or gradual. John Hunter, still young in mental power but elderly in years, and old as regards the heart itself, with its calcified coronaries and liability to angina under circumstances demanding sudden adaptation to effort or emotion, died suddenly of heart failure. The relation here between the neural demand for organic adaptation and the muscular failure is manifest. The cause of death was the failure of the specific cell to respond to the demand for increased action. It was, in all probability, in this case the pace that killed.

But, without such emotion, demand on the specific cell in excess of adaptability may be equally fatal and as suddenly so. Had Hunter been walking up a hill or lifting a weight in an effort exceeding the adaptability of the cardiac muscle cell to the demand made, the effect would have been the same. Had he been sitting still or lying in bed, and from some variation in blood distribution a momentary demand for adaptation to increased effort had been made upon that perpetual weight-lifter the heart, death might have been equally sudden.

But apparently healthy and younger muscle, as in the runner and swimmer, may fail in adapting itself to demand and the last straw, added by a momentary failure to distribute the weight in the circulation, may close the scene with tragic suddenness, such failure to distribute being due to a breakdown in vasomotor adaptability.

I shall not occupy your valuable space by dwelling upon the depressant influence on the nervous system and the specific cell of toxaemic conditions of blood. The pneumonic, the fevered, the exhausted, and the drugged die poisoned, under circumstances in which demand beyond adaptability is made on the specific cell. But increasing demand may be progressive and gradual, and failure likewise gradual and progressive, when the slowly manifested evidence of such failure in dilatation and stagnation become apparent. The failure of the specific cell—of the instrument—which is only one of the factors in the functional unit, must be due to defect in its nutriment or innervation. The uninnervated functioning of a specific cell, which is sometimes imagined and preached is as unthinkable as the existence of such a cell without blood supply, His's embryonic chick notwithstanding.

Whatever functions be assigned to a specific cell, whether it be the production of a digestive ferment, the excretion of waste, or the contraction of muscle, it is conditioned in the exercise of such function by the state of the other factors in the functional unit.

It is by the recognition of the complexity of this unit, of which the neural element is so important a factor, that I would suggest we are likely to attain to more satisfactory views of the nature and cause of heart failure, to a rational management of the condition when threatened, and to an explanation of some apparent contradictions in the pathological findings.—I am, etc.,

London, W., March 25th. ALEXANDER MORISON, M.D.

SIR,—In your last issue Sir Clifford Allbutt publishes an address on the Physician and Pathologist on Heart Failure. It is indeed a dark picture which he paints, steeped in melancholy tones. Let any reader peruse his article and admit the statements that it contains, and he will lay down this number of the JOURNAL with the sad conviction not only that prognosis in heart affections is impossible, but that any endeavour to disperse the gathered clouds is foredoomed, and that the source of heart failure will remain unknown.

The two chief schools have failed. To the questions of Sir Clifford morbid anatomists return him no answer, and those who employ the timeworn methods of percussion and auscultation are equally silent. "If, then," says Sir Clifford, "the delineations of the heart's dimensions which we obtain by percussion . . . give us no constant or trustworthy criterion, what of auscultation? Well, we are but little better off." But there is a third school of the clinicians who attempt a closer study of the morbid physiology of the *living* heart.

What is heart failure but perverted or submerged function? And how can we ever hope to solve the riddles of Sir Clifford till we are prepared to study, and study on a large scale, the action of the moving organ which one day is to cease beating? I earnestly declare my conviction that the chief problems of heart failure will be solved, nay, are being solved, by this method, and that the time is present, not to recount the failures, but the successes of those graphic investigations which form a part of it.

On the list of failures which Sir Clifford cites are examples which have no right there. Speaking of the morbid anatomist he says, "or if we show him another heart in which, by the poison of rheumatic fever, the tract of Tawara was eaten away, and yet, notwithstanding, there was during life no dissociation of auricle and ventricle. . . ." Did such a case ever occur? If so, I do not know of it. And again, "many cases are published (by Reinecke and others) in which, under what seemed to be complete demolition of the bundle, there was during life no dissociation." I challenge this statement without reserve, and affirm that in no case has discontinuity of the bundle been proved in published reports, where conduction was shown to exist within a reasonable time of death.

Further, Sir Clifford Allbutt speaks of arrhythmias "pertaining to the extrasystolic kind," and cites a group in which they may be harmless, but later he speaks as follows: "The symptom group of every such case must, of course, be sifted carefully, for, on the other hand, in pneumonia or diphtheria a dropped beat may be the first presage of heart failure." True, but the "dropped beat" of acute affections is usually due to a different phenomenon, namely, heart-block, and the recognition of this cannot be accomplished by sifting the symptoms, whereas it is at once accomplished, and with certainty, by graphic methods.

Who cares for the authority of Leyden, Bäumler, or Ebstein, to whom the differentiation of the simplest irregularities was unknown, when they state "that in the pulse we have no guide to myocardial damage"? Indeed, their very statement is now false. Who cares whether in any instance a lesion can be found to account for an irregularity? The irregularity itself is evidence that the cardiac muscle has suffered functional change. It must be fairly analysed, and this analysis can now be made. Heart-block, extrasystoles, auricular fibrillation, pulsus alternans, etc., can be identified with certainty, and their pathological significance will soon be fully known if the analysis is made in all cases of irregularity. It will not be known if Sir Clifford's views prevail and deter such investigation.

I come to a crucial question, one for which Sir Clifford seeks a reply: To what can we ascribe sudden death in a heart patient, in whom recovery has been generally expected? He has no answer which satisfies. The methods of the new school are young, but I venture to predict that if that question is again asked in a few years only, the new school will be ready with its answer. And that answer is contained in Sir Clifford's own writing: "Fibrillation of the ventricle may be one of the modes of sudden death, but it is hardly consistent with survival." Graphic methods have isolated three stages of perverted auricular action—the production of extrasystoles, of paroxysmal tachycardia, and fibrillation; they have isolated the two first stages of the same process in the ventricle, and the third, being incompatible with life, has not been caught; yet precisely those patients in whom the third stage is expected fall dead, when to the old school such death is least anticipated. We know that ventricular fibrillation terminates many experiments in animals; death has so far hidden its demonstration in man, but the danger signs which give warning of its imminence may be seen; closer study, wider study by more workers, is needed to ascertain their exact value.

Sir Clifford Allbutt has done great service to English medicine in the long course of his distinguished career. Disparagement of new methods at this stage of their development may cripple them. The new school requires additional and active members; it asks the sympathy and support, not only of Sir Clifford, but of all leaders of English medicine.—I am, etc.,

Cardiographic Department, University College Hospital, March 24th. THOMAS LEWIS.

LEPROSY IN THE UNITED KINGDOM.

SIR,—In the letter signed by Dr. H. Bayon there are inadvertencies, to which I must ask you to allow me to direct attention. Dr. Bayon is advocating the desirability

of a compulsory registration of lepers in England, and in doing this he attempts to confute the statement which has been made that no instances have been verified during a long period of any cases as originating in England. In order to prove his point he quotes cases, and apparently takes for granted that the expression "England" includes the whole United Kingdom. Now, it is no mere quibble to remind him that this is far from being the case. It is well known—and I have myself mentioned it many a time—that one or more cases of quasi-indigenous origin have occurred in Ireland and in the North of Scotland. It is a fact that it was in these districts that leprosy, as regards the United Kingdom, lingered latest, and it is one of the arguments by which my belief that leprosy is merely a dietetic modification of tuberculosis is supported.

Dr. Bayon mentions cases (exceedingly few) as originating in Ireland and in the Shetland Islands, whilst he gives but one as originating in England itself. The subject of the last case had resided all his life in England, but both his parents, who were also lepers, had lived in Poland, and were supposed to have acquired the disease there. Their son's malady was therefore quite as likely to have been the result of inheritance as of contagion, and there is no hint that the disease spread further. On the whole, then, I think I am entitled to say that Dr. Bayon's cited facts support the conclusion that no proven cases of leprosy have originated in England within recent years.

My assertion as regards sporadic cases of leprosy is that they may originate and may be followed by others of apparent contagion wherever the conditions render likely the consumption of ill-cured fish. These conditions occur wherever fish is remarkably abundant whilst other forms of animal food are scarce or dear, good salt deficient, or the religious usages of the district more or less discourage the employment of other forms of animal food. It is under one or other of these heads that I seek to explain the very rare cases which yet occur in Ireland, in Switzerland, and other places where we still occasionally hear of it.

The fact that in none of these places do these sporadic cases—although, as a rule, for long undiagnosed and unsuspected from the beginning—form the centre of any leprosy epidemic is, I think, an emphatic disproof of the theory of contagion.

We have always plenty of cases in England as well as in the rest of the United Kingdom which might easily prove centres for an outbreak of leprosy, and the fact that they never do so is, I think, an emphatic refutation of the contagion hypothesis and the asserted desirability either of compulsory registration or of the institution of asylums for segregation.

If, as I contend, neither isolation nor registration are necessary, it is not difficult to see that they are unjust, and, in the case of leprosy, cruel. The word "leprosy" carries with it, in the popular imagination, such a frightful amount of exaggeration that it is much to be desired that it should be entirely disused; and the facts, if viewed in a common-sense way, would, I am sure, thoroughly justify this being done. It is unwise to attempt to base important practical conclusions upon facts which are, at best, very few in number and most of them somewhat doubtful.
—I am, etc.,

Haslemere, March 23rd. JONATHAN HUTCHINSON.

SIR,—Dr. Bayon has done me the honour to quote from an article of mine on "Leprosy," and to mention a case in which I am interested, which is now under the care of Dr. Woodyatt at the Whitechapel Infirmary. He does not, however, inform you that the two cases alluded to are practically the only ones undoubtedly known during the last forty years in which there is authentic evidence of the disease having manifested itself in a subject who has not spent some time in a country where leprosy is endemic. Any one who has made himself acquainted with the facts concerning the disease in Great Britain and Ireland must be aware that although we have always with us a certain number of lepers—probably, at any one period, not less than fifty—we can find no evidence of any actual spread of the disease. As far as I have been able to discover, all the patients have either been colonists or have spent some time in a leprous country—that is, they have contracted leprosy abroad; and in the large majority of the cases—since their arrival in this country—no precautions whatever have been taken as regards contagion to others. If leprosy were the actively infectious malady that it is in the popular mind—and apparently in the minds of some of our medical officers of health—we should surely meet, occasionally at any rate, with some "indigenous" cases—some instances, in fact, where persons who have never been out of the country, but who may have been associated with an imported leper, have contracted the disease; but as a matter of fact, if we exclude the above two exceptional cases, we can, so far, find no valid evidence of anything of the kind.

I cannot, indeed, agree with Dr. Bayon that "the St. Pancras Borough Council were justified in passing the resolution they did." However advisable and necessary stringent measures, notification, etc., may be in places where leprosy is endemic or where it shows signs of spreading, such measures, in the interest of the public health, are not at present called for in this country. It might certainly be useful to a keen pathologist to know where the cases are, and where material could be obtained for further study, but with reference to the idea that the stray leper in this country is a serious source of danger to others, notification is quite unnecessary. As I have remarked elsewhere:

> When the carriers of such easily communicable diseases as tuberculosis and syphilis are allowed to go freely about and to engage in any or every occupation, it would appear, to say the least, somewhat redundant to unnecessarily penalize the poor sufferers who are afflicted with leprosy. The official stamp of danger which notification would imply would doubtless further enhance the popular fear of its contagion, and render the burden of the unfortunate leper still more hard to bear.

—I am, etc.,

London, W., March 25th. PHIN. S. ABRAHAM.

KALA-AZAR.

SIR,—I see in the JOURNAL of February 17th, on page 386, a small paragraph that Surgeon-General Bannermann, of Madras, has cabled to Sir R. Ross, of Liverpool, of Captain Patton's discovery of the complete development of the parasite of kala-azar in Indian and European bed-bugs. This discovery seems to harmonize with the observations of Wenyon[1] and of Franchini,[2] and is practically the same thing as obtaining developmental forms of the parasite in a haemoglobin-containing medium found in the digestive tract of blood-sucking insects who retain the blood (sucked) for several days. In other words, the culture takes place in a living tube, just as one can show this taking place in a test tube containing haemoglobin solution, even extraneous contamination within certain limits being no bar. The developmental forms of both *Leishmania tropica* and *Leishmania donovani*, as they take place *in vitro*, were demonstrated by me[3] on January 4th at a meeting of the Bombay Branch of the British Medical Association, in their complete cycle—namely, from ⊙ body to flagellate and from the flagellate back to ⊙ body. However interesting the parallelism of development of these parasites, whether in the bug, mosquito, or in glass test tube, may be, the important point in the study of the etiology of the diseases produced by these parasites is whether it is possible transmit the disease in susceptible animals with the aid of the ⊙ bodies obtained from the flagellates. Let us hope this will soon be demonstrated, and the real culprit caught and condemned.—I am, etc.,

Bombay, March 2nd. R. ROW.

PROGNOSIS IN CASES OF CHRONIC ARTHRITIS

SIR,—Many of us will have read with much interest and pleasure the excellent address by Dr. Fred. Gardner on prognosis, published in the JOURNAL of February 10th, and probably most of us would endorse his opinion that "optimism in prognosis is not only our pleasant privilege, but our bounden duty." Will you allow me to emphasize this in one large group of cases, and to put in a word of warning against a wrong form of optimism under certain conditions that I have become familiar with?

As regards the first, is there not in the profession generally too much pessimism in the prognosis given in

[1] Wenyon, *Parasitology*, October, 1911, vol. iv, No. 3.
[2] G. Franchini, *Lancet*, November, 1911; and *Malaria e Mal. di paesi Caldi*, Roma, 1911, ii, 235.
[3] R. Row, paper read before the Bombay Branch of the British Medical Association, January, 1912, and published in this issue of the JOURNAL (p. 717).