THE EARLY SIGNS OF RHEUMATISM OF THE HEART.

To the Editor of THE LANCET.

SIR,—Dr. A. M. Gossage's article in your issue of August 21st, under the above title, gave me great pleasure. It was very gratifying to find my work on rheumatic myocarditis so thoroughly discussed by a critic of Dr. Gossage's ability, still more to find that he is in nearly all points agreed with me and that the points of difference are apparent rather than real. They are, I fear, due to the lack of explicitness in the article which I wrote and which Dr. Gossage has criticised. Two of these, at any rate, can be cleared up by an explanation from me.

Dr. Gossage says that I exaggerate the importance of "myocarditis." This is because I use the term in its widest sense, while he restricts it to the truly inflammatory interstitial nodules, my description of which he quotes at some length. I include in the term not only these nodules but also the degenerative changes in the muscle cells which accompany them, and which are described fairly fully in the paper under discussion. In that paper he will find the following sentences (p. 38) in a paragraph summarising the evolution of rheumatic myocarditis. "Poisonous substances are disseminated from these foci (i.e., the nodules) throughout the cardiac muscle, upon which they exert a deleterious influence. This reveals itself in ventricular dilatation ..... while microscopical proof of the same poison is yielded by the presence of fat within the cells." If Dr. Gossage will include these among the features of myocarditis he will, I think, find that we are in agreement as to its importance. A purist may object to the inclusion of degenerative changes under a term which primarily stands for an inflammation, but it is a common and convenient usage (e.g., in nephritis, myelitis). It may be asked why the nodules are so important if their immediate effect upon the cardiac functions is so small. Because they afford strong evidence of an actual invasion of the muscular wall of the heart by micro-organisms in every case of rheumatic carditis, a fact of great importance, as I hope to show in the next paragraph.

Dr. Gossage suggests that the changes in the muscle cells (which we agree in holding responsible for ventricular dilatation as well as for most of the physical signs and many of the deaths in rheumatic carditis in childhood) may be produced entirely by a rheumatic toxin manufactured by bacteria at a distance from the heart, as in the superficially analogous case of diphtheria. I say "superficially" because while diphtheria is an intense toxæmia rheumatism is in all probability a not very toxic bacteriæmia. His theory is perhaps true for the transient cardiac dilatation seen in adults with rheumatic fever, but I do not think it can be considered equally important with actual invasion of the cardiac muscle by micro-organisms, as evidenced by nodules, for the following reasons. First, there are not more than one or two fatal cases of myocardial poisoning in rheumatism, without cardiac inflammation, on record in spite of the fact that rheumatic carditis is not an uncommon cause of death; and even in these one or two I suspect that a careful examination of serial sections might have discovered nodules. Second, if the micro-organisms of rheumatism are (as the nodules suggest) actually encamped in the myocardial tissues, the muscle cells will bear the brunt not only of toxins carried to them in a dilute condition by the general blood stream, but also of relatively concentrated toxins carried to them from neighbouring myocardial nodules by the lymph stream. I do not wish to underrate the possible importance of blood-borne toxins in the rheumatic infection; but it is, I believe, a general infection rather than a localised infection diffusing toxins.

As to the way in which rheumatic myocarditis leads to mitral incompetence, I must thank Dr. Gossage for his valuable and explicit addendum to my article. I am afraid, however, that he over-estimates the degree of concentration of the nodules upon the neighbourhood of the mitral ring. It is difficult to speak of this with confidence as I have only examined 15 hearts carefully (seven since my article was published), but my impression is that whereas the nodules are, as a rule, but few in the right ventricle, the septum, and the papillary muscles, and absent from the auricles, they are scattered fairly evenly through the wall of the left ventricle with some predilection for its basal rings and the apex. This I stated in the paper alluded to. One remaining point of difference, as to the origin of apical mid-diastolic murmurs in rheumatic carditis, vanishes in the light of the explanations given in the first and third paragraphs of this reply.

In conclusion, I must thank Dr. Gossage for his careful consideration of my paper and the courteous criticism to which I have in this letter replied briefly. His explicit remarks on the production of mitral regurgitation by depression of tonicity in the mitral sphincter are an interesting expansion of the ideas which in my paper in the *Quarterly Journal of Medicine* for October, 1908, I sought to express.

I am, Sir, yours faithfully,

Clifton, Bristol, August 23rd, 1909. CAREY COOMBS.

A DISCLAIMER.

To the Editor of THE LANCET.

SIR,—The medical board of this hospital at their first meeting since August 30th have desired me to state that the reported interview with the director of the serum-therapy department of the hospital, published under the heading "New Consumption Treatment," in the *Daily Mail* of August 30th, was unauthorised by the medical staff and does not accurately represent the proposed work of the serum-therapy department of the hospital.

I am, Sir, yours faithfully,

W. J. MORTON, Secretary.

The Mount Vernon Hospital for Consumption and Diseases of the Chest, Hampstead and Northwood, Sept. 7th, 1909.

DERMATITIS DUE TO THE USE OF METOL.

To the Editor of THE LANCET.

SIR,—The use of the generally accepted formulæ of the metol and hydroquinone developer in combination with sulphite of sodium is held in high place amongst all photographers as a rapid means to an end with clear, definite results. And my reason for approaching the subject is not to criticise the advantage of the developer over many others for producing fine resulting negatives, but rather to utter a word of warning to those who use it without due precautions.

In the early summer I had occasion to develop a batch of negatives of scenes taken during an exceptional spell of hoar frost, and as most of the latter were under-exposed owing to climatic conditions I proceeded to intensify them in the usual way with perchloride of mercury and liquor ammoniæ. About two days after I had finished the latter process I found that all the ends of the fingers of my right hand were intensely inflamed, tender, and swollen, and with an amount of local anæsthesia corresponding to the areas of immersion. This at the time I attributed to the action of the strong ammonia solution. On the following day a certain amount of onychia of the third finger manifested itself with an exudation of serum at the base of the nail which resolved into crusts. Ultimately during the next few days all the skin came off these affected fingers, shrinkage of the tissues occurred, and sensation returned. A few weeks now passed before I had any occasion to require the use of any developer, but about a month ago on resorting to the same combination and not having the opportunity of washing freely after its use I was attacked next day by urticarial eruptions which appeared wherever the solution had become applied. The irritation was even more severe than in the first attack, showing probably a cumulative action in the system. The skin of the thumb, first two fingers, and fourth finger was intensely tender and swollen for two or three days, tingling and burning as in a case of chilblains. Since this has happened I have heard of three other cases similarly affected; two are connected with a firm of photographic printers and another who has been ill through the same cause. In the two above mentioned the skin came off all the fingers and left open sores.

Even in mild cases it takes about a fortnight before the affected parts recover from the tumefaction and sensation is fully re-established. In conclusion, I do not mean to imply that all persons are affected in the same way. But to those who have experienced any of the symptoms described after having used metol developer or metol in combination with other substances, I think it necessary to give this advice—