ENDOCARDITIS LENTA.

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The term "Endocarditis lenta" has been used to indicate a variety of active, progressive endocarditis in which the damage to the heart valves is often very extensive and in which a lethal result is the rule; yet the disease lasts for a much greater length of time than is usual in acute infective endocarditis.

At the outset it is essential to indicate exactly what clinical condition is being described and for this purpose to classify the forms of active progressive disease of the heart valves. (See Table 1.)

TABLE 1.

<i>Disease.</i>	<i>Duration.</i>	<i>Valve affected.</i>	<i>Pyrexia.</i>	<i>Organism.</i>	<i>Result.</i>
Acute Infective Endocarditis	up to six months	any or all of the heart valves	high and swinging	Hæmolyt. strep. Pneumococcus Gonococcus Staph. aureus.	Death
Chronic Infective Endocarditis	up to 15 months	previously damaged valves mitral or aortic	marked up to 102	Bacteræmia Non-hæmolyt. Strep. viridans	Death
Subacute Infective Endocarditis	up to 3½ years	usually aortic valve, often bicuspid	slight or none	Strep. viridans not in blood	Fatal usually
Active Rheumatic Endocarditis	recurs over years	valves of left heart	for several weeks	Rheumatic streptococci. ? ? ?	Recovers

For the purpose of this paper, the two groups Chronic Infective Endocarditis and Subacute Infective Endocarditis have been regarded as together constituting Endocarditis Lenta. It is suggested that it is now justifiable to replace all three of these names by one title upon an aetiological basis. The group of cases referred to in this paper would thus become **Viridans Endocarditis**.

It is for this reason proposed to recognize endocarditis lenta as a Streptococcus viridans infection which may be divided into two groups. In the first the infecting organism can be recovered from the blood stream and pyrexia is present, while in the second the blood is sterile and the temperature remains normal; but the ultimate result, though longer delayed in the second, is almost equally disastrous in the two groups.

In support of this view of the disease the papers of Libman⁽¹⁾ and Horder⁽²⁾ appear to describe cases which correspond to the first group, while those of Carey Coombs⁽³⁾ and Starling⁽⁴⁾ record cases representing the second.

Ætiology.

In the pyrexial cases a previous rheumatic history is extremely common and a superimposed infection upon a previously damaged valve is the rule rather than the exception.

In the non-febrile cases a rheumatic history is rare and previous clinical evidence of cardiac damage is unusual. The part played by congenital defects in the heart, particularly the bicuspid aortic valve, appears to be of importance in the latter group as was shown by Lewis⁽⁵⁾ in a very careful paper containing details for differentiating congenital from acquired bicuspid aortic valves.

The actual source of the infecting agent was long thought to be the digestive tract, since the *Streptococcus viridans* is reputed to be a normal inhabitant there, but the recent work of Okell and Elliott⁽⁶⁾ demonstrates the frequency with which the blood stream can be shown to carry such organisms after extensive tooth extraction. Thus a final link in a chain of evidence has been established.

Sex Incidence.

It is to be remarked that in the pyrexial group it has been found that twice as many males as females suffer from the disease, but in the apyrexial group the males are at least ten times as numerous as the females.

Morbid Anatomy.

In the pyrexial group fungating warty excrescences tend to form on valves which have been previously damaged, hence the mitral valve is more prone to show them than the aortic. The outgrowths tend to spread onto the heart wall and down the chordæ tendineæ, occasionally causing rupture. Organisms are often recovered from these lesions while in sections they are numerous and easily found.

In the afebrile group the valves tend to be more deformed, sclerosed and lime encrusted, the vegetations are less proliferative and show a marked tendency to sclerotic healing. The aortic valve is most often involved and the frequency with which a bicuspid valve is seen (congenital or acquired) is very striking. Recovery of the organism from these lesions has seldom been successful and even in sections it is by no means easy to find them.

In both groups the endocarditis is very marked and at the same time the involvement of the myocardium may be minimal, in sharp contrast to the usual findings in rheumatic endocarditis. Clawson⁽¹¹⁾, however, in his large study of cardiac disease, concludes that the changes in the heart muscle in this disease are at least equal to those found in acute rheumatism. It may be that the changes are more marked in the myocardium in the pyrexial group than in the afebrile one so carefully studied and recorded by Carey Coombs⁽³⁾.

The great tendency to embolization is much the same in both groups and is frequently the immediate cause of death.

The renal changes have been repeatedly studied and are fully described both by Baehr⁽⁷⁾ and Gaskell⁽⁸⁾. They appear to be identical in the two groups, showing characteristic changes in the renal glomeruli . . . infarction of the tufts with secondary atrophy of the corresponding tubule, the whole showing but little inflammatory reaction.

Morbid Physiology.

In both groups the mechanical defect of the damaged valve will show itself in the usual way. At the same time ordinary cardiac failure is uncommon or only a terminal event, possibly in some cases because the myocardium suffers so little damage. A streptococcal toxæmia with marked pallor, embolism and uræmia are common events.

Clinical Course.

The onset in both groups is so insidious that a point of time for it cannot be fixed. Increasing fatigue, lassitude and pallor are the outstanding features, often accompanied by a peculiar bronzing of the skin, giving a *café-au-lait* or even deeper colour. The patients are prone to have a peculiarly optimistic outlook and to resent any suggestion that they are gravely ill. Most of them have to be pressed to submit to medical examination on account of their striking pallor.

The Heart is often enlarged and a well marked valvular lesion is usually present. Yet cases occasionally arise in both groups in which a valve lesion cannot at first be found. Still more difficult is the recognition of any progress in the lesion. In the pyrexial group the mitral valve is most frequently affected while in the afebrile cases the aortic valve is almost sure to be the source of the trouble. Cardiac failure is likely to be a late feature of the disease.

The Blood. In the pyrexial cases a positive blood culture yielding the *Streptococcus viridans* is most probable, but may require more than one attempt. In the apyrexial cases the blood will almost certainly be sterile. Very little change occurs in either group in the red cell count but the hæmoglobin is likely to be reduced to 50 per cent. The leucocyte count may vary from 5 to 20 thousand but a leucocytosis is neither common nor persistent in either group.

The Temperature has already been suggested as a means of separating the more active from the chronic cases, but even the afebrile patient is liable to suffer from profuse night sweats without any rise of temperature.

Embolism may declare itself in several forms, coronary occlusion and splenic infarction being relatively common. A large or middle sized systemic vessel may become plugged; and hemiplegia is common and may be fatal. Petechiæ are thought to have an embolic origin and are commonly found in crops more particularly around the clavicles, axillæ and groins.

The suddenly-appearing small rounded swellings, chiefly on the fingers, hyperæmic and exquisitely tender, disappearing after two or three days, which were so clearly described by Osler⁽⁹⁾, are much more common in the pyrexial than in the afebrile cases.

The Spleen. Enlargement of the spleen is an almost constant accompaniment of both groups of the disease. It is usually of very moderate degree and only recognized on careful examination. The grosser varieties are most frequently due to multiple infarction but the toxæmia alone would seem able to determine a palpable enlargement.

The Urine. In both groups albuminuria is usual, and in addition red blood cells can almost always be found in the centrifuged deposit. Gross hæmaturia occasionally occurs from renal infarction.

Retinal Changes. These may be slight or extensive and are usually embolic in origin but in some cases the changes are associated with the renal condition and may then be the immediate precursors of uræmia. The interpretation of these changes may be most difficult.

Clubbing of Fingers. This is one of the most striking characteristics of the disease, being almost constant in the apyrexial group and often serving to suggest a correct diagnosis. It may appear also in the pyrexial group and its development may be watched during the course of the illness.

Presumably its more constant occurrence in the afebrile group is associated with the longer duration of the disease. The condition is not dependent upon cyanosis but probably upon long continued toxæmia. The time necessary for its development may perhaps be three months at a minimum. It is said to appear first in the thumb, the toes are also sometimes affected. This aspect of the disease has been fully dealt with by Cotton⁽¹⁰⁾.

Termination. The vast majority of patients in both groups succumb, but there is reason to believe that this is not invariably the case, although in the present state of our knowledge clinical recovery must always throw grave doubt upon the diagnosis. Libman, however, has presented a great deal of cogent evidence in favour of occasional recovery, based partly upon long clinical observation and partly upon post-mortem evidence obtained in patients dying from other causes and showing what appeared to be healed lesions of the disease. The differentiation of healed lesions of viridans endocarditis from those of rheumatic disease must remain difficult but the renal changes are regarded as characteristic. The immediate cause of death is likely to be cardiac failure, uræmia, cerebral or coronary embolism in the majority of cases.

It is to be remarked that the pyrexial type has been observed over many years amongst the civil population, especially in those whose health has already been debilitated by a chronic heart lesion. The apyrexial type, however, has occurred very largely amongst men of military age who have done heavy service and who were not affected by any antecedent heart lesion. The number of afebrile cases since 1923-4 does appear to have been very small and most observers are agreed as to the present rarity of this form.

Treatment.

Treatment may be considered under three heads—specific—general—and preventive.

(a) **Specific.** Various sera and vaccines have been used quite extensively by a number of observers, without any effect upon the almost uniformly fatal results.

Intravenous antiseptics of various sorts have likewise been tried but without success, many of them, indeed, would seem to have been definitely harmful. Blood transfusion and immunotransfusion have failed to give much help.

(b) **General.** Symptomatic treatment for the cardiac failure or the uræmia which are likely to arise in the later stages of the disease will call for the usual measures.

The various embolic phenomena which may take place early or late will also require adequate care and morphia if painful. Apart from this, there seems little which can be profitably recommended—rest in bed for the pyrexia—iron for the anæmia—a bitter tonic for the lost appetite; these appear poor efforts in the face of so serious a disease.

It would seem justifiable to attempt to build up the resistance of the patient along the lines used for pulmonary tuberculosis, by organized rest in the open air, night and day; this in some cases has appeared to prolong life. It is possible that such treatment might just turn the scale in favour of slow recovery in the most highly resistant cases.

(c) Prevention. It is in this field that the profession carries so large a load of responsibility, because supervision and advice are alike so difficult.

Because the *Streptococcus viridans* is already present in the digestive tract, either as *S. salivarius* or *S. faecalis*, it is possible that the portal of entry, and therefore the source of infection of the damaged heart valves, is to be found either in the mouth or the bowel. Dental, tonsillar or intestinal sepsis should therefore be avoided in all patients suffering from any form of valvular heart lesion. Yet it is obvious from the paper of Okell and Elliott, already quoted ⁽⁶⁾, that radical treatment is in itself a grave source of danger. It is within the experience of the majority that the breakdown of a chronic heart patient may date from some operative procedure in or about the buccal cavity. From the observations already made it would seem wise to undertake radical treatment with very great circumspection.

If tonsils require removal or sinuses require drainage, or teeth require extraction, these measures should be carried out by some means which reduce to a minimum the amount of tissue damaged at any one time. Teeth, for example, should be extracted singly and at long intervals and tonsils cauterized or removed singly. The size of the area submitted to trauma at any one moment appears to bear a definite relation to the probability of blood infection. And all this must enter into our careful consideration in every case of chronic valvular disease of the heart.

It is possible that the risk of infection might be reduced very considerably by a process of prophylactic vaccination against *S. viridans*, although there is little evidence to show that this procedure is of material help once the disease is established. It would appear highly desirable to attempt to raise the resistance in valvular heart cases by an intensive course of vaccines immediately prior to any operative procedure, however small, if it involve the digestive tract.

It is by means such as these that we must attempt to prevent a grave infection which in the present state of our knowledge we are regrettably unable to cure.

REFERENCES.

- (1) Libman, E., Brit. Med. Jour., 1920, ii, 304.
- (2) Horder, T., Quar. Jour. Med., 1909, ii, 289.
- (3) Carey Coombs, F., Quar. Jour. Med., 1922-3, xvi, 309.
- (4) Starling, H. J., Quar. Jour. Med., 1922-3, xvi, 263.
- (5) Lewis, Sir T., and Grant, R. T., Heart, 1923, x, 22.
- (6) Okell, C. C. and Elliott, S. D., Lancet, 1935, ii, 869.
- (7) Beahr, G., Jour. Exper. Med., 1912, xv, 330.
- (8) Gaskell, J. F., Jour. Path. & Bact., 1911-2, xvi, 287.
- (9) Osler, Sir W., Quar. Jour. Med. 1908-9, ii, 219.
- (10) Cotton, T. F., Heart, 1921, ix, 347.
- (11) Clawson, B. J., Am. Heart Jour. 1928-9, iv, 1.