It does happen, however, that the blood culture is persistently returned as negative in certain cases of indisputably infective endocarditis. The explanation for this is not easy; three suggestions may be made:

1. That organisms are present in the blood stream, but of a type which defies our present methods of culture.
2. That the bacteria, though massed on the cardiac vegetations, are not circulating in the blood stream.
3. That the vegetations, and the blood stream also, have become bacteria-free.

In support of the two latter suggestions proof of a definite character may be quoted, certain cases having been recorded in which a completely sterile blood stream has been associated with endocardial lesions demonstrated *post mortem* to be teeming with bacteria; in other cases with negative blood cultures examination of the crushed vegetations entirely fails to reveal the presence of bacteria. The whole subject of blood cultivation in subacute bacterial endocarditis is undoubtedly important, and needs further investigation; our present state of knowledge, however, enables us to state quite positively that, for purposes of diagnosis, a positive blood culture is certainly not essential.

With regard to prognosis, we have unfortunately grounds for extreme precision: the disease is invariably fatal; three to twelve months may be looked upon as an average period, though the patient may drag on slightly longer in some instances. The progress of the disease is relentless and rapid; infective emboli are responsible for widespread and lasting lesions, and incidentally cause a large part of the suffering which these patients undergo. The cause of death is cerebral embolism, or haemorrhage, uraemia, progressive anaemia, or intercurrent pneumonia.

The abbreviated notes of two patients recently admitted to this hospital will illustrate, from the clinical point of view, some of the features which characterize the progress of the disease.

CASE I.

G. W., aged 36, pensioner. Admitted September 14th, 1920; died November 7th, 1920.

Past History.—Negative except for trench fever in 1917. The serum test was negative. Onset of symptoms June, 1920: Dyspnoea, palpitation, pain under heart with exertion, slight cough; no haemoptysis; loss of weight.

On admission (September 14th) the physical signs were pallor, clubbing of fingers, enlargement of spleen and liver, oral sepsis; heart enlarged; free aortic reflux; lungs engorged.

Progress.—Patient was in hospital three weeks, and, during this period, the condition became steadily worse; fever of an intermittent character persisted, petechial haemorrhages were observed from time to time; sweats, rigors, and all the usual concomitants of septicaemia, complicated the final stages, and death took place on November 7th, barely five months after the onset of the initial symptoms.

CASE II.

S. B., aged 26, pensioner. Admitted September 1st, 1920; died September 25th, 1920.

Past History.—Negative. Discharged from the army March, 1917, for gunshot wound, face and neck. Serum test negative.

Onset of Symptoms.—May, 1920, palpitation after exertion, shortness of breath, vertigo, and fainting attacks.

Physical Signs on Admission (September 1st, 1920).—Pallor, anaemia, and poor nutrition; fingers clubbed; heart enlarged, free aortic regurgitation, vessels thickened, pulse collapsing in quality; liver and spleen enlarged and tender; petechial rash on neck and upper thoracic region; urine, heavy cloud of albumin and blood cells numerous.

Death from cerebral embolism on September 25th, five months after the first onset of the symptoms of the disease.

The *post-mortem* appearances in patients who have died of subacute bacterial endocarditis bear ample witness to the widespread nature of the disease. The heart is enlarged in most instances; the endocardium and valves are the seat of gross changes in the shape of extensive vegetations, which vary very considerably in appearance and consistence; the older lesions are usually firm, greyish, and calcareous, the most recent reddish-yellow and extremely friable.

The valves involved are the mitral and the aortic, the latter more frequently than the former; in the majority of instances, however, the infective process spreads from the primary seat of infection, both to the adjacent endocardial wall and to the neighbouring valve; under these circumstances one notices that the oldest infection is the most extensive. It is important to observe that the right side of the heart is never involved, furthermore that the endo-

cardiac process is of a purely vegetative character, ulcerative lesions playing no part in the condition.

A large number of conditions found *post mortem* elsewhere than in the heart might be mentioned; they depend in every instance upon embolic processes which may very obviously affect the arterial channels in any part of their course, and determine the production of visceral infarcts or haemorrhages, mycotic aneurysms, etc.

Treatment.

Treatment should follow the broad lines adopted for any form of septicaemia: rest in bed should be absolute, the diet should be light and easily digestible; the action of the bowels must be free; drugs are of little value, though some temporary benefit may follow the prescription of tonics such as iron, arsenic, and quinine. Strychnine, by reason of its effect on the vasomotor centre, and digitalis, may be employed for symptomatic reasons and will sometimes assist in tiding over critical periods.

Intravenous injections of collargol and other metallic and antiseptic substances have been tried without success, no useful purpose therefore can be served by advising treatment of this nature.

Vaccines should be given a trial in all cases, but, in the experience of most observers, their remedial effect on the disease, in its fully developed stage, must be considered to be small.

The whole subject of treatment in this disease forces upon us the conclusion that subacute bacterial endocarditis, in the stage which permits diagnosis by gross physical signs, is an incurable malady. This being so, our duty becomes clearly defined: we must endeavour to recognize the initial stage of the disease, the early evidence that fresh infection has developed upon some old and quiescent lesion, the origin of the infective process, the factors which permit of its spread, the influence of a lowered tissue resistance, etc., for we know full well that, until we possess reliable data concerning the preliminary phases, our chances of lessening the mortality of this dread disease are wholly insignificant.

AN OPERATION FOR THE RADICAL CURE OF PRE-MASSETERIC FISTULAE OF STENSON'S DUCT.

BY

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THE ideal treatment for this condition is the restoration of the continuity of the duct (Nicoladoni). This, however, must always be difficult, and in any but recent cases must be impossible. An alternative method is to dissect out the duct, sever it from the fistula, and turn it into the mouth (Languebeck, also Riberi).^{*} As the original cause of the fistula usually produces considerable local scarring, this also is an operation that can only occasionally be successful. A large group of operations aim at the formation of an internal fistula, either by means of a ligature causing sloughing of the tissues between the duct and the mouth cavity (Deguise and P. Gould), or by means of a small tube passed through from the fistula into the mouth (Kauffmann's modification of Descoult's operation). All these methods are tedious and at the best uncertain. If these fail it is recommended either to ligature the duct, admittedly entailing the risk of abscess formation in the gland,† or to dissect and remove as much of the gland as possible without damage to the facial nerve. This method admittedly produces disfigurement, but is regarded as preferable to a persistent salivary fistula.

The number and varied character of the methods described for the treatment of this condition, and the drastic

^{*} As an alternative to this method, when a considerable portion of the duct is destroyed, or much fibrous tissue is present, Dujardin and Gulikers (*Ann. de la Soc. Belge de Chir.*, October, 1900) incise in the direction of the duct and dissect up both this and the masseteric promulgation of the gland, which is then passed between the masseter muscle and the mandible, and stitched into the opening in the mucous membrane of the oral cavity.

† Morestin (*Bull. Aut. Men. de Chir.*, Paris, 1917; Abstract S.G.C., 1917) makes a rule to dissect out and ligature the duct, the gland subsequently degenerating. This author has done 32 cases since 1913 by this method.

nature of some of them, is a sufficient indication of their uncertain value, and has induced me to publish the operation now described. Its advantages are great simplicity and, as far as I have practised it, reliability; while it permits of the complete removal of the fistula and surrounding scar tissue from the cheek with primary union of the remaining linear incision at one operation.

I have performed this operation in three consecutive cases.¹ In two the fistula was due to a gunshot wound in the malar region, and in one it followed the external rupture of an alveolar abscess. In each case the fistula was situated immediately in front of the anterior border of the masseter, and had been in existence for over a year. In one case a course of several months' treatment with the hard x-ray tube had failed to effect any improvement, and in another the fistula had previously been dissected out and the cheek closed by a plastic operation, without alleviating the condition.

The Operation.

Two horizontal incisions, about a quarter of an inch apart, are made, the one above and the other below the fistula.

Fig. 1.—Showing the incisions. A probe has been inserted into the fistula. The triangular areas of skin on either side of the area bearing the opening are discarded.

once until it enters the mouth immediately below the reflection of the mucous membrane opposite the upper molar teeth. In one case this area of the cheek was adherent to the alveolar border owing to scarring following the injury, and was mobilized by means of an epithelial inlay some weeks prior to the operation. The probe is now withdrawn, and small cat-gut mattress sutures inserted in the anterior and posterior lips of the skin button, and drawn through the wound into the mouth. Similar mattress sutures are inserted in the lips of the opening of the buccal mucous membrane, and these are now

Two short vertical incisions serve to isolate a small button of skin bearing the fistulous opening. The posterior of these incisions is cautiously deepened, a probe being meanwhile retained in the duct lest the latter should be too closely encroached upon (Figs. 1 and 2). The anterior vertical incision is deepened at

Fig. 2.—The island of skin bearing the fistulous opening in course of mobilization. Deepening the posterior incision, with probe in position in the duct.

Fig. 3.—Semi-diagrammatic horizontal sections showing: a, The condition of duct and fistula prior to operation; A and B, position of incisions. b, The mobilized area of skin bearing the fistulous opening, and containing the salivary duct in its pedicle; A and B, anterior and posterior lips of mobilized skin area; c and d, lips of the opening through the mucous membrane. c, The mobilized skin area seen in b has been inverted, and its edges sewn to those of the opening in the mucous membrane. The letters A, B, C, D have the same significance as in b.

tied together within the mouth. The original anterior edge of the skin button is stitched to the posterior lip of the opening in the mucous membrane, while the posterior edge of the latter is tied to the anterior lip.

(Fig. 3, a, b, c, shows diagrammatically the successive stages in this process.) No attempt is made to mobilize the skin button sufficiently to allow of its complete reversion. If it is turned over to face forwards and slightly inwards, the elasticity and loose attachment of the oral mucous membrane will allow of the latter being drawn up to the button, the post-operative appearance in the mouth being that of a conical recess of mucous membrane, passing backwards and outwards to the fistulous opening. The lips of the original incisions are now very slightly undercut and brought together, thus covering the reversed surface of the skin button bearing the fistulous opening.

In one of the three cases the tip of a defective probe remained in the duct, and was subsequently discharged through the operation wound. This has resulted in a slight external leakage, which, however, does not amount to a complete drop during a meal. In the other two cases healing was by first intention, and the wound has remained dry since the operation, now more than two years ago. In all three cases there is now a free and normal discharge of the parotid secretion into the mouth.

ADRENALIN IN RESUSCITATION FROM APPARENT DEATH.

BY

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ANAESTHESIA, asphyxia, the passage of a powerful electric current, violent emotion, and violent sensory stimulation may place the body in a state which resembles that of a clock which has been stopped, but not permanently damaged; if the machinery can be restarted it will continue to run automatically. Relatively simple means will often suffice to set things in motion again, as a mere shake will serve to start a clock. Sometimes it appears as though a more accurately fitting stimulus were required, like a swing in time with that of the pendulum.

Mr. Welby Fisher, in a very interesting paper,¹ advocated cardiac massage applied through an epigastric incision. He makes no reference to the use of adrenalin. The following notes of a limited experience may be of interest.

Adrenalin applied to the heart muscle acts on the sympathetic neuro-muscular junction, and very greatly increases both the excitability of the tissue and the energy output of the muscular contractions. In some twenty cases of death from various causes, when all signs of life had ceased for several minutes, I have injected a minim or two of 1 in 1,000 adrenalin chloride directly into the heart muscle. A fine needle was passed through the fifth left intercostal space, just mesial to the lung resonance; the point was directed slightly inwards and upwards, and could be felt to enter the apex of the heart; when firmly imbedded in the muscle the injection was made. Several of these needle tracks were examined *post mortem*, and in no case did it appear that any drawback would have ensued had the patient survived.

In most of the cases death had occurred as the result of long established disease, and adrenalin was neither expected nor observed to have any effect (a fact not without interest). In the following three cases circumstances were more favourable.

CASE I.

A splendidly built young man had received a powerful electric shock while fitting an electric lift, and all appearance of life had instantly ceased. When seen by me it is estimated that about twenty minutes had elapsed; there were no signs of circulation, the upper parts of the body were pale, the lower were dusky, showing that the blood distribution was determined by gravity; the skin was already cool. Artificial respiration was applied, adrenalin injected into the heart muscle, the needle shaken *in situ*, the heart was massaged, but without abdominal incision, and was stimulated faradically. There was no response.

CASE II.

A child of 11 months during a circumcision under chloroform had stopped breathing, and became pale and flaccid; the pupils were dilated, no pulse could be felt, and no heart sounds could be heard. Artificial respiration was started, and the heart was massaged by pressing the fingers under the costal margin; in this case the heart could be felt, and almost gripped between the fingers. There was no response, and the body was cooling perceptibly. Adrenalin was injected into the heart muscle, and immediately the heart started beating; shortly after respiration recommenced, and in a few minutes the child appeared normal. The time was not accurately observed, but it is estimated that at least four minutes had elapsed between the stoppage of the

¹September and December, 1918, and May, 1919.