their vital force and power. Cases of diffuse cellulitis can be recalled—the “diffuse phlegmon” of Dupuytren—where, in a previously debilitated system, severe constitutional disturbance supervenes, and a train of “typhoid” symptoms sets in. This is also the case in examples of acute spreading gangrene, a condition in some aspects allied to chronic septicæmia, in which the absorption of the chemical products of disintegration and putrefaction has from first to last a toxic and seriously injurious effect. The condition has also been noticed in wide-spread burns of the fourth and fifth degrees. Another instance of the “typhoid” phenomena occurs during an attack of “delirium tremens,” the result of hard drinking, when marked signs of exhaustion of the nervous system set in with failure of functional power, and again in those cases of “nervous traumatic delirium” which not infrequently arise in patients with an “irritable” and somewhat unstable nervous system, or in those who have suffered from prolonged mental anxiety and the enervating effect of worry and over-strain. If the patient be the subject of an almost impermeable organic stricture, and if unrelieved retention exist, and if, in consequence of the rupture of the dilated portion of the urethra which is immediately behind the seat of such a stricture, extravasation of urine takes place, the united effects of such a condition, both local and general, give rise, on the one hand, to the most deleterious consequences to the tissues themselves, and, on the other, severe constitutional disturbance; “typhoid” symptoms rapidly supervene, in which muttering, quasi-insensible delirium, a small and rapid pulse, and dry, brown, and cracked tongue, form the most prominent features. Clinically allied to the foregoing condition, and in natural sequence, is the disease described under the term “acute, diffuse, interstitial, suppurative nephritis,” associated with multiple scattered abscesses (“surgical kidney”) in which the patient sooner or later sinks into a heavy, drowsy state, “typhoid”-like in character, and succumbs within a comparatively short time from poisoning by absorption of septic matter and waste products, a condition of uræmia, and closely resembling chronic pyæmia or septicæmia. Lastly, one observes the development of “typhoid” symptoms in cases of acute traumatic meningitis and encephalitis, complications of some injury inflicted on the skull and the structures contained within it. In these cases, with an open wound of the head, infection readily and often insidiously takes place unless more than ordinary care is exercised to prevent the establishment of a condition of sepsis. If, however, septic meningitis has taken place there is a speedy exacerbation of the general symptoms, especially the temperature, which mounts up to 104° F., concurrently with restlessness and delirium, which, if symptoms of compression coexist, passes into that drowsy, stupefied type, the breathing becoming stertorous, the pulse feeble and failing, and the patient succumbs in an exhausted condition from the dual effect of failure of the nervous system and compression. In the intra-meningeal form of intra-cranial suppuration which is occasionally associated with pyæmia “typhoid” phenomena appear towards the close of the case. In the days before attention was definitely directed to hygienic conditions in relation to surgical procedures cases not infrequently occurred where anæmic, cachectic, and debilitated patients, crowded together in a small, ill-ventilated ward, suffered from languor, headache, “continued fever,” and similar symptoms indiscriminately passing into an “adynamic” or “typhoid” condition,⁷ which was in part due to the poisonous effects of an excess of carbon dioxide, gases of decomposition, and organic impurities in the air of the ward, and in part to the absence of sufficient air space per bed. Another important consideration in connexion with the development of the “typhoid state” during the course of any disease lies in the fact that in overcrowding and the continued inhalation of a vitiated atmosphere there is a marked prevalence of diseases of the respiratory tract, such as phthisis, pneumonia, and bronchitis, all of which, under the above-mentioned conditions, have a tendency to assume an “adynamic” type closely allied to the “typhoid state.” Of the twenty-three survivors of the Black Hole of Calcutta many suffered from a condition which is designated under the term “putrid fever,” probably allied to typhus fever,⁸ in which “typhoid” symptoms were

well marked. An example of the supervention of such symptoms is also met with in animals in severe cases of “foot-and-mouth” disease, in which there is much fever, prostration, and thirst, these symptoms being the more pronounced in sucking animals.⁹ Writing on the “typhoid condition,” Tweedie states that “it is announced by the decline of the previous more acute symptoms: by the pulse becoming more rapid and soft; the tongue dry and brown, tremulous, and protruded with difficulty; by the incrustation of the teeth with sordes; by the increasing intellectual disorder, indicated by the more constant low, muttering delirium, and the greater insensibility and deafness; and by the condition of the muscular system evinced by muscular tremor and sub-sultus tendinum, and in some cases irregularity or intermission of the pulse; by the patient lying sunk on his back or sliding to the foot of the bed, the muscles being unable to support the body even in the horizontal posture.” Milner Fothergill reviewed this subject under the following considerations:—(1) the association of high temperature with its effect on the entire muscular system—this point has also been emphasised by C. B. Radcliffe, Wunderlich, and Bennett Dowler of New Orleans; (2) the general wasting of the muscles in fever; (3) the marked sense of prostration (*debilitas febrilis*); (4) the excess of urea excreted in the urine, there being a definite relation between temperature and amount of urea (in ‘typhoid fever’ about 1065 grains of urea are excreted in twenty-four hours, the normal being 400 grains); and (5) the pathological condition of the muscular structures before and after death. He also considers that this condition is due to an increased metamorphosis of blood and tissues with subsequent rapid wasting of the latter (“histolysis”), and that it is a common and necessary result of fever. He insists on the importance of “muscular prostration”—the patient being often found lying on his back in the middle of the bed, having gradually slipped down—as a characteristic clinical sign, and notes that “muscular weariness” is one of the earliest symptoms. The remark of Da Costa that “no blunder is more common than to misconstrue into typhoid fever a typhoid condition of the system” is not without its weight and value in reference to diagnosis.

III.—CONCLUSION.

In concluding these incomplete remarks on the “typhoid state” I regret that I am unable at the present to furnish further details as to its pathology, and as to the temperature or the condition of the circulation as shown by sphygmographic tracings. I have not yet been able to find an adequate reply to the query, Does the “typhoid” condition occur more frequently among persons of a highly nervous temperament or of a hereditarily unstable nervous system? I have not been able to discover any examples of this condition in cases of malignant disease, whether sarcoma or carcinoma.

In asking the indulgence of the readers of THE LANCET I am reminded of Boileau’s lines:—

“Hâtez-vous lentement; et, sans perdre courage,
Vingt fois sur le métier remettez votre ouvrage:
Poli-sez-le sans cesse et le repolissez;
Ajoutez quelquefois, et souvent effacez.”

Tunbridge Wells.

ON THE TREATMENT OF ACUTE RHEUMATIC ENDOCARDITIS.

By RICHARD CATON, M.D. EDIN., F.R.C.P. LOND.,
HONORARY PHYSICIAN, LIVERPOOL ROYAL INFIRMARY.

IN the year 1881 I commenced a definite inquiry, which I have pursued steadily for fourteen years so far as cases suited for the purpose came under my observation, on the course and treatment of the acute endocarditis of rheumatism, and I now propose briefly to state its results.

Gravity of cardiac rheumatism.—Numerous cases of acute rheumatism necessarily come under the care of every hospital physician—I have treated about 320 cases of acute and sub-acute rheumatism during the fourteen years in question. While speedy cure of the rheumatism itself usually follows, a proportion of these cases—perhaps 1 in 3 or 4—suffer

⁷ Whitelegge: p. 21 &c.; Plates of Air Samples, Parkes’ Hygiene, sixth edition, p. 112; and Dupuytren and Pirry, *Traité de Chirurgie*, Paris.

⁸ Cf. “Om status putridus och nervosus.” Naumann. Stockholm, 1834

⁹ Wilson, “Hygiene,” p. 43.

cardiac complication, and the physician has the pain of seeing the patient leave the hospital bearing the seeds of a malady which is certain to cause suffering and shorten life. Perhaps the physician follows the whole career of such a case extending over some years. A vigorous man comes to him suffering from acute rheumatism, but with a sound heart; endocarditis affecting the mitral valve occurs and persists; the rheumatism subsides, the patient returns home thinking himself well, but on exertion suffers from an unaccustomed shortness of breath; bronchitis, increased difficulty of breathing, inability to follow his occupation—if it be a laborious one—and dropsy follow; dilatation of the right ventricle, and tricuspid disease may set in and death closes the scene. Many of us have seen cases like this, and have felt their extreme sadness.

Objects of the inquiry.—Feeling the importance of doing what little was in my power to further any inquiry which might mitigate the frequency of heart disease, I set myself during the last fourteen years to try to obtain information on two points—viz., (1) as to how the occurrence of endocarditis may be prevented during acute rheumatism; and (2) whether or not, when it does occur, any treatment during the incipient stage would favour recovery. When the disease is fully established of course there can be little hope of benefit. I had first to acquaint myself with the experience of former writers in our own country, in Europe, and in America. That of the earlier authors I found of less value on account of their imperfect diagnosis; and also their treatment, on account of its severity, did not commend itself to me. The opinion of more modern writers was highly conflicting. Some considered all measures having in view either prevention or cure practically useless. The weighty names of Niemeyer and of Osler were among these. It was encouraging to find Sibson and others pointing out the value of absolute and protracted rest, both as a prophylactic and as an aid in the cure of incipient endocarditis. Various writers described the occasional spontaneous disappearance of bruits in acute rheumatism. This, I think, there can be no doubt of: I have myself seen it happen twice. The general opinion was that a bruit when once established remains in the large majority of cases, and is followed by permanent heart disease. Many forms of treatment were suggested. The older writers advise bleeding and mercurial salivation, the local employment of dry and wet cupping, leeches, blisters, mustard plasters, iodine, ice-bags, poultices, the actual cautery, the internal administration of potassium or sodium iodide, digitalis, aconite, mercury, &c.

Bad results of expectant treatment.—Meantime I proceeded to observe a number of cases, merely treating the rheumatism by salicylates and keeping the patient in bed under generally favourable conditions, but doing nothing of a special kind for the endocarditis. In other cases I experimented with some of the milder among the agents I have referred to. Up to the end of December, 1885, the results obtained from merely treating the rheumatism and doing nothing for the endocarditis, also from partial blistering or the use of leeches or poultices, were not favourable; of fourteen cases one lost the bruit spontaneously, although no treatment was employed; a second lost it after repeated blistering and the use of potassium iodide; and the remaining twelve went out with a bruit which I fear has been permanent in each case, and has probably terminated in death in many instances before now. The experience of these four years disposed me to adopt some definite method. Adopting no treatment had led to unfavourable results alike during this period and during previous years.

Method of treatment selected.—Guided by the physiological facts to be stated later in this paper, it seemed to me that the use of a succession of small blisters along the course of the fourth, fifth, and sixth intercostal nerves, together with the internal use of potassium or sodium iodide not only gave as much promise as any other method, but tended but little to disturb or to weaken the patient. I had, moreover, had one successful case already, Case 12 (see Abstract), in which an aortic bruit disappeared; so I proceeded to treat almost all subsequent cases in this way, enjoining also a prolonged rest in bed.

Before detailing the results I ought to say something about (1) the general treatment of these cases of acute rheumatism; (2) about the efforts made to prevent cardiac disease; and (3) about the evidence I relied upon as indicating the presence of genuine endocarditis in the cases on the list which I subjoin.

1. *General treatment of rheumatic cases.*—The patients

are kept in bed; I prohibit as far as possible all exertion or exposure. No getting out of bed is allowed, and to prevent chills a long flannel nightshirt is worn, a stock of these garments being kept on purpose for rheumatic cases in my wards. Thus the profuse perspiration incident to the disease is not likely to be checked. A light milk diet is given. Gentle cholagogues are administered and salicylates are given in such measure as the disease seems to demand. If the pain fails to yield in any joint I immediately apply small blisters followed by poultices, a method which rarely fails to give great relief, as I can testify from the experience of hundreds of patients. Hyperpyrexia or any special symptom receives appropriate treatment. The patient is kept in bed and on a light diet for a considerable time after the disappearance of pain.

2. *Efforts to prevent cardiac complication.*—There seems to be strong ground for believing that perfect quiet and rest and avoidance of chills during acute rheumatism lessens the liability to cardiac complication. Cold to the surface undoubtedly increases the inflammation of synovial membranes; rheumatic inflammation of the endocardium is similar in kind and is likely to be affected similarly. My own experience certainly is in favour of this opinion. In what proportion of cases is the heart affected? There is much divergence of opinion as to the proportion of cases in which the heart is attacked. Codet, the French writer, puts it down at 81 per cent., Picot at 78 per cent., Mansir and Shadwell's list from Guy's Hospital Reports gives over 50 per cent. The lowest average I read of is 25 per cent. During the period my list of cases covers I have had about 320 cases of febrile rheumatism, but in many of them the fever was slight and temporary. My proportion is under 20 per cent. Probably the great discrepancy in percentages is due to different conceptions of the meaning of the term "acute rheumatism." If it be restricted to the highly febrile type the high estimates (60, 70, 80 per cent.) I have quoted will probably be correct. Though, according to my own experience, endocarditis is not confined to pyrexial cases, it is certainly more frequent in them, and I think I may consider my proportion of cases of rheumatism with cardiac complication to have been comparatively small.

3. *Were my cases examples of genuine endocarditis?*—I should like to say that of course cardiac murmurs are of very different values. The bruit of anæmia, for instance, heard chiefly about the pulmonary area, and the bruit of muscular weakness which is sometimes met with in fevers and other conditions of exhaustion, are not to be mistaken for signs of endocarditis. The former occurs sometimes in acute rheumatism, but only in the later stages. The murmur at the pulmonary cartilage in Case 41 (see Abstract) was probably of this order. Also the bruit due to muscular impairment, if it occurs at all in rheumatism, happens at a late period, when the patient is enfeebled by a lengthy illness. I have always been on my guard against mistaking either of these forms of bruit for that occasioned by endocarditis. The great majority of the cases in the list are those of commencing mitral disease. The aortic valves were affected in five cases, and in seven pericarditis occurred. I make it a rule to examine the heart of every rheumatic patient daily, so as to be on the lookout for any commencing mischief.

Period at which the heart is affected.—In a few cases endocarditis seems to occur coincidently with the first rise of temperature and the joint affection. More often it happens during the first ten days; sometimes it is later when the temperature has fallen and pain gone and the patient thinks himself getting well. Sometimes it occurs during a relapse of the rheumatism, as in Case 18. The patient had been going on well for a week with normal temperature, when suddenly, on March 9th, the temperature rose and endocarditis became apparent. If a bruit has disappeared it is apt to recur during a relapse of the rheumatism, as in Cases 44 and 48. As to symptoms, there are sometimes præcordial discomfort and further rise of temperature, but generally, in my opinion, no special symptom has attracted the patient's attention when an endocarditis has first become perceptible to the physician. In pericarditis symptoms are more common. The following has been my experience of the first signs of endocarditis. Suppose a case of mitral regurgitation. The patient has been admitted with a heart the sounds of which were normal excepting as regards rapidity of beat. One notices a change in the character of the first sound—what the French writers call "assourdissement"—a loss of clearness and sharpness in the tone. The note is dull

ABSTRACT OF FIFTY-TWO CASES OF ACUTE RHEUMATIC ENDO- AND PERI-CARDITIS, SHOWING TREATMENT AND RESULT.
(When the date of first appearance of bruit is not given it was present on admission, but believed to be recent.)

No.	Age.	Dates of		Physical signs of cardiac disease.	Special treatment of cardiac complications.	Cardiac physical signs on discharge.	Remarks.
		Admission.	Discharge.				
1	21	Dec. 21st, 1881	Jan. 11th	Mitral bruit	None	Mitral bruit	—
2	19	Dec. 29th, "	Jan. 7th	"	"	"	—
3	25	March 9th, 1882	May 12th	Mitral bruit on March 21st	"	"	—
4	33	May 22nd, "	May 31st	Mitral bruit	"	"	—
5	28	March 19th, 1883	May 1st	Aortic systolic bruit	"	Aortic bruit	—
6	23	Dec. 27th, "	Jan. 17th	Aortic, diastolic, mitral, and pericarditis	Poultices	Mitral bruit	—
7	15	Dec. 29th, "	Jan. 11th	Mitral bruit	None	"	—
8	23	April 17th, 1884	May 22nd	Mitral bruit on April 19th	One blister	"	—
9	26	July 8th, "	July 29th	Mitral bruit	None	"	—
10	26	Nov. 17th, "	Jan. 30th	Mitral bruit on Dec. 9th	Blisters, pot. iod.	"	Rheumatism long continued.
11	21	March 9th, 1885	March 26th	Mitral bruit	One blister	"	—
12	12	June 15th, "	July 13th	Aortic systolic bruit	Blisters, pot. iod.	No bruit	—
13	17	Sept. 18th, "	Nov. 9th	Mitral bruit, pericarditis	One blister, leeches	Mitral bruit	Adherent pericardium.
14	21	Dec. 7th, "	Dec. 21st	Mitral bruit	None	No bruit	—
15	16	Jan. 5th, 1886	March 14th	Pericarditis	Blisters, pot. iod.	Normal	—
16	18	Jan. 6th, "	Feb. 23rd	Mitral bruit on Jan. 26th	"	No bruit	—
17	19	Jan. 22nd, "	Feb. 19th	" on Jan. 25th	"	"	—
18	21	Feb. 23rd, "	April 14th	" on March 9th	"	"	Relapse on March 9th.
19	16	March 3rd, "	March 29th	"	"	"	—
20	49	March 13th, "	April 30th	"	"	"	—
21	19	March 27th, "	May 10th	"	"	"	—
22	18	May 10th, 1887	May 23rd	" on May 18th	None	Mitral bruit	—
23	34	April 23rd, 1888	May 24th	Aortic systolic bruit on April 30th	Blisters, iodides	No bruit	—
24	19	May 10th, "	June 14th	Mitral bruit	"	"	—
25	17	Jan. 10th, 1889	May 6th	"	"	Mitral bruit	—
26	21	May 9th, "	May 22nd	"	"	Faint mitral bruit	Bruit lessened greatly under treatment.
27	66	June 7th, "	July 12th	"	"	No bruit	—
28	28	Feb. 6th, 1890	March 22nd	Mitral bruit on Feb. 11th	"	?	Result not stated.
29	16	June 4th, "	July 16th	" on June 27th	"	No bruit	—
30	21	June 24th, "	August 18th	" and pericarditis	"	"	Pneumonia.
31	19	Dec. 27th, "	Feb. 26th	" on Jan. 1st; aortic on Jan. 21st	"	Bruit on Feb. 12th	No later report.
32	29	Feb. 7th, 1891	March 14th	Mitral bruit on Feb. 10th	"	No bruit	Much delirium.
33	14	May 13th, "	June 11th	Mitral bruit	"	"	—
34	18	June 13th, "	July 13th	Mitral bruit on June 17th	"	"	—
35	18	Sept. 23rd, "	Nov. 5th	" on Oct. 7th	"	"	Result not stated.
36	13	Nov. 27th, "	Dec. 31st	" (loud)	"	Faint bruit	—
37	18	Dec. 20th, "	"	"	"	No bruit	—
38	28	Oct. 16th, 1892	Nov. 28th	" pericarditis	"	"	—
39	26	Nov. 18th, "	Dec. 7th	"	"	"	—
40	28	Jan. 18th, 1893	Feb. 10th	" on Jan. 22nd	"	"	—
41	21	" 25th, "	" 19th	" on Jan. 26th	"	No mitral bruit	But faint bruit at third left cartilage.
42	35	March 11th, "	April 20th	" on Jan. 14th pericarditis	"	No bruit	—
43	30	May 17th, "	June 23rd	" on May 23rd	"	"	—
44	19	July 19th, "	Sept. 17th	" on July 21st	"	Mitral bruit	Bruit disappeared under treatment, but returned during relapse of rheumatism, Aug. 21st, and persisted.
45	42	Jan. 11th, 1894	March 3rd	"	"	No bruit	—
46	19	May 2nd, "	May 30th	"	"	"	—
47	18	June 14th, "	Aug. 9th	" on June 16th	"	?	Result not stated.
48	17	July 4th, "	Aug. 13th	" on July 16th	"	1st sound slightly prolonged	Bruit disappeared under treatment, but returned during relapse on July 27th, and again disappeared.
49	15	" "	July 28th	" and pericarditis	"	No bruit	—
50	24	July 25th, "	Oct. 3rd	"	"	Mitral bruit	—
51	22	Dec. 8th, "	Jan. 18th	"	"	No bruit	—
52	18	March 4th, 1895	May 15th	" on March 5th	"	"	—

Summary.

Of 39 cases treated by repeated blistering over heart and administration of iodides (average stay in hospital, 41 days), 29 left hospital without a bruit; 7 left with a bruit; and 3 were uncertain—they probably had a bruit. Of 13 cases not treated, or treated otherwise (average stay in hospital, 25 days), 1 left hospital without a bruit, and 12 with a bruit.

and woolly, so to speak; it becomes prolonged, and this dull, prolonged sound by degrees changes into an undoubted bruit. Many and many a time have I noted these changes, and foretold them from the time when the first dullness of tone was observed. The bruit is best heard about the apex, but is audible in the axilla; it is usually systolic, occasionally pre-systolic, and sometimes it is both. Frequently it is accompanied by accentuation of the second sound in the pulmonary area.

Details of treatment of incipient endocarditis.—As soon as the bruit is perceptible, or even prior to that, during the preliminary stage of "woolliness" I at once give potassium or sodium iodide in ten-grain doses thrice daily in addition to the salicylates, and apply near the apex a blister about the size of half-a-crown; as soon as the irritation of this blister is subsiding I apply a second close to it, then a third and so on, keeping the patient meantime as quiet as possible, strictly forbidding exertion, exposure, or too much nitrogenous food. I have scarcely ever known the patient to complain of the blisters, which practically occasion little or no discomfort. The heart should be examined daily. Frequently in a week or in ten days' time I have had the satisfaction of noting a gradual subsidence of the bruit; day by day it becomes less clear and distinct, until at length it is found to be only occasional, and at last to be entirely absent. Sometimes several weeks elapse before this desirable change is accomplished. I think the prolonged rest in bed has an important share in this good result. It will be noted that the thirty-nine cases which I treated on the above principle were, on the average, forty days in hospital, and were confined to bed for thirty-five or thirty-six days. When the bruit has gone, I have, when possible, kept the patient under observation for awhile. Sometimes this is not practicable, but I have often for many weeks noted the condition of the heart and found it perfectly normal. In some instances I have done this for two or more years.

Excellent results obtained.—It will be observed from my list that in the thirteen cases which were either not treated at all or imperfectly treated by blistering, poulticing, leeching, &c., with an average residence in hospital of twenty-eight days, the result was that twelve left hospital with a bruit which I fear they retained throughout life. One case, however, lost his bruit spontaneously. But of the 39 cases treated by prolonged rest, repeated blistering, and the administration of iodides, 29 left hospital with normal heart sounds, in 3 the result was doubtful, and 7 left with a bruit. I must confess that this series of twenty-nine patients in whom distinctly marked bruits had been heard, but who left the hospital with normal heart sounds, constituted a better result than I had ever ventured to hope for. It is, in fact, so good a result that I do not expect that the readers of this paper will altogether accept it. Many will believe that mistake or error has crept in somehow. To the best of my knowledge and belief, what I have stated is accurate. The cases were not examined and recorded by myself alone, but by registrars, house physicians, and clinical clerks.

Theory of the treatment.—I claim for this method that it is essentially a reasonable one on the following grounds. So-called "counter-irritation" has fallen much out of vogue in the profession, partly because the old theories on which it was formerly supported are felt to be untenable. We cannot believe that a poultice or blister draws blood from, or acts directly in any manner upon, an organ in the thorax or abdomen, but it by no means follows that the practice is bad because the former theory was unsound. Such treatment has been partially abandoned also because our predecessors carried it out to excess so as to cause pain and to enfeeble the patient. If we apply a small blister or a poultice to the skin of the thorax or abdomen I believe its effect on the organs situated at some distance beneath is solely an effect transmitted through the nervous system and reflected from the nerve centres in the spinal cord and sympathetic system. The nervous system may be summarised as a series of segments arranged in a line corresponding to the various somites of the body; each nerve segment governs to some extent the nutrition of its own somite, alike as regards epi-, meso-, and hypo-blastic structures. If a cutaneous nerve is stimulated the nerve cells in the spinal and sympathetic centres are influenced, and the impression is reflected to the visceral branches. It may be said that this is mere theory, but it is a theory which satisfactorily explains many familiar facts—e.g., it explains the fact that a slight chilling of the skin of the thorax may cause pneumonia or bronchitis, and that a splash of cold water

will excite respiration in the infant or set a-going the action of the heart in a faint. I may add that I think I proved this theory in a paper read before the Physiological Society at Oxford a few years ago by demonstrating that stimulation of the surface skin altered the diameter of the arterioles in the lung and caused variation in the electrical condition (as manifested by the galvanometer) of thoracic and abdominal organs. It is quite clear that stimulation of the skin of the abdomen by heat, by chemical irritation, by faradism, or by mechanical irritation produces instantly a variation in the electrical potential. This, however, is so extensive and complex a subject and occupied so many months of hard work in the laboratory that I cannot hope to make it clear in the space of a few lines. Turning to more familiar evidence, few persons, I think, will deny the soothing effect of a poultice in a case of bronchitis, nor, I think, can any explain that effect satisfactorily in any other manner than that I have just stated. When that form of impaired functional activity is occurring in the tissues of the heart which is called "rheumatic endocarditis," a stimulus is applied to the nerve centres, and the trophic nerves of the heart help nature to overcome the defective action. That is the theory on which this treatment by blistering is based. I believe that the assisted natural powers succeed where, if unstimulated, they would fail.

I should like to say here that after twenty-five years' experience of counter-irritation in many forms, carried out in view of the theory I have stated, I think it is one of our most valuable therapeutical expedients for the relief of pain, for lessening inflammatory action, and for the stimulation of restorative processes. A very limited irritation of cutaneous nerves will suffice; I never use large blisters—generally they are smaller than a florin; small vesications give great relief and cause practically no suffering and no loss of strength. As to the sodium or potassium iodide, it is given on account of its value in absorbing inflammatory products of low vitality. I am anxious to know what the experience of others has been in regard to the very grave disease in question, and to hear of alternative or superior plans of treatment. In conclusion, the one thing I am specially anxious about is that members of the profession who have the opportunity of observing cases of acute endocarditis should give this method a fair trial during the early stage of the malady. If they do, I think they will not be disappointed.

Liverpool.

INFECTIOUS SORE THROATS.¹

By J. C. THRESH, M.B. AND CH.B. VICT.,
MEDICAL OFFICER OF HEALTH FOR CHELMSFORD, ETC.

THE importance of obtaining early information of outbreaks of infectious disease led me some three years ago to issue a circular letter to all the school managers in my districts asking them to instruct their head master or mistress to give me the earliest possible intimation of the presence amongst the children attending school of any affection which appeared to be infectious, and I also drew attention to the fact that sore throat, especially if associated with headache or sickness, was one of the earliest symptoms of certain infectious diseases. The result of the above procedure has been that many epidemics in which the most constantly marked symptom was sore throat have been brought under my notice which could neither be referred to diphtheria nor to scarlet fever. The following are examples.

In February, 1893, I received a note from the schoolmaster of an isolated parish saying that he was afraid that diphtheria had broken out in the school. I found several children were then in school who had been absent during the previous week for three or four days. They had suffered from headache, more or less marked, a little feverishness, and sore throat. In a very few days they were sufficiently well to return to school. Examination of the tonsils simply showed that they were enlarged, pale, and somewhat flabby. I visited several of the absentees and found in most cases they had suffered from a little headache and chilliness. The tonsils were enlarged, not abnormally red, but the mucous membrane round the apertures of the follicles was distinctly infiltrated, slightly thickened, greyish, and opaque. Exactly the same

¹ Paper read at a meeting of the Home Counties Branch of the Medical Officers of Health Society, Feb. 7th, 1895.