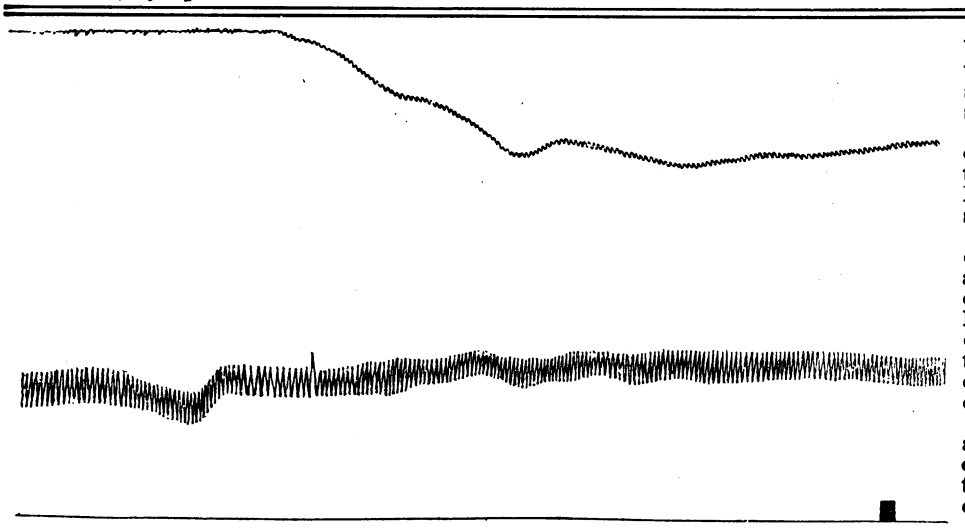

Fig. 4.—Dog. The lowest tracin is that of the intestinal plethysmograph; the upper one that of carotid blood pressure.

before any further injection of the drug, which then again causes some rise of general blood pressure.

4. The entire plant, therefore, contains two substances with distinct physiological actions; but I have not up to the present been successful in isolating them.

5. Watery extracts of the residue obtained by evaporating the alcoholic solution produce a fall of blood pressure and cardiac inhibition, due to the action of the drug on the nerve terminations in the heart, and not to direct action on the muscular fibres of that organ.

6. The substance which causes a rise of blood pressure is not contained in such watery extracts, or, if present, only in small quantities.

REFERENCE.

[1] *Pharm Journ.*, vol. i, 1895, p. 1195; vol. ii, 1895, p. 535; also *Compt. Rend.*, cxx, p. 1120.

ORAL SEPSIS AS A CAUSE OF DISEASE.

BY WILLIAM HUNTER, M.D., F.R.C.P.,

Senior Assistant Physician, London Fever Hospital; Joint Lecturer on Practical Medicine, Charing Cross Hospital.

I AM interested to see that the subject of oral sepsis in certain of its relations was brought under discussion at the meeting of the Royal Medical and Chirurgical Society on June 12th by a paper from Mr. Rickman Godlee.

My excuse for commenting on the discussion must be that the whole subject of oral sepsis as a cause of disease has been one of special interest to me for many years; that I have dealt with it at some length in published papers during the past year and a-half[1]; and that the more I study it the more impressed I am, at once with its importance, and with the extraordinary neglect with which it is treated alike by physicians and surgeons.

I have described cases identical with some of those referred to by Mr. Godlee; and shown—a point not even referred to by any, even the most recent, writers on diseases of the stomach—that not only is the constant swallowing of pus a most potent and prevalent cause of gastric trouble, but that the catarrh set up is not simply irritant but actually infective, and may lead in time to other more permanent effects—namely, atrophy of glands and chronic gastritis, and in certain cases even to suppurative gastritis.

This result is, however, by no means confined to and associated with any one mouth condition, such as pyorrhœa alveolaris. And I specially desire to draw attention to this point, since I note that several of the speakers desired information as to what degree of pyorrhœa alveolaris was necessary to produce the various ill-effects referred to.

I have to point out that for every case of gastric or other affection traceable to pyorrhœa alveolaris a hundred cases equally well marked are daily to be found associated with other dental and oral conditions of sepsis. In short, I deprecate this subject of oral sepsis and its effects being brought under discussion in connection with any one pathological condition of the mouth. The list of such conditions might be increased almost indefinitely. In my own experience they include not only pyorrhœa alveolaris, but stomatitis and gingivitis of every degree of severity—"erythematosa," "pustulosa," "ulcerosa," "gangrænosa," and indeed every other form of trouble, dental and oral, producible by septic infection for which an appropriate adjective can be found. The list, moreover, includes in my experience others for which a suitable qualifying adjective cannot so readily be found, and which I may describe as "foul septic toothplate" stomatitis, "bridge" stomatitis, and "gold cap" stomatitis; this latter group, I venture to think, considerably on the increase in this era of conservative dentistry and high professional mechanical skill.

The important fact to be recognised is that one and all of these various conditions are septic in their nature, and produced by pus organisms; that these organisms are invariably associated with every case of dental caries, however slight; and that the question of effect in any one case is a matter of individual resistance.

The cause underlying them is oral sepsis of the most marked character. This sepsis, moreover, is of a particularly virulent character. For it is connected with disease of bone (that is, of teeth); and a somewhat extensive pathological experience has satisfied me that no pus organisms are so virulent as those grown in connection with necrosing bone.

No physician or surgeon would tolerate for a moment that a patient with a foul septic ulcer, say in his forearm, should from time to time apply his lips to the ulcer to clean it. Yet this is—pathologically—precisely what happens in the case of patients with necrosed teeth and stomatitis. Moreover, the swallowing is constant, and goes on for years, unheeded both by patient and doctor.

I recently saw a patient, a lady, who for twenty-five years had suffered at intervals of every three or four weeks from most inexplicable salivation and subsequent intestinal trouble, so severe in character as to confine her to bed. She had worn for the same period of time a toothplate, which she only removed irregularly, and only cleaned with a toothbrush. She displayed a condition of stomatitis connected with necrosed stumps that was quite remarkable, overlooked as it had been all that time.

I saw recently another patient, also a lady (it is among ladies that the best examples of conservative and artistic dentistry are to be found), who for several years suffered periodically from severe nervous attacks, complicated by gastritis and curious rashes, the whole symptom-complex being regarded as gouty manifestations. I was asked to see her in one of her rashes; and found it a typical blotchy septic rash. Only a month or two before, her dentist on the strength of the first of the papers below referred to, had insisted on removing a toothplate which had partially grown into her jaw, and which had been there for several years. In relation to gastritis and gastric catarrh, such cases could be multiplied indefinitely.

The matter is important, however, not only in relation to gastritis, but in relation to the whole group of infections caused by pus organisms—*local*, for example, as tonsillitis, glandular swellings, middle ear suppurations, maxillary abscesses; *general*, for example, ulcerative endocarditis, empyemata, meningitis, nephritis, osteomyelitis, and other septic conditions. Whence do they gain entrance into the system? They are not ubiquitous, as was formerly thought.

Nor are they necessarily disease-producing from their mere presence; for example, on skin, in the mouth, or in the intestinal canal.

But, given the suitable conditions, namely, diminished resistance on the part of the tissues, or increase of dose on the side of the organisms, they are disease-producing. These are precisely the conditions brought about in long-continued necrotic and septic conditions of the mouth.

It is probably impossible to keep pus organisms out of the mouth, just as it is impossible to prevent occasional access of tubercle, typhoid, and other infective organisms. But that fact does not deter us from taking the most exhaustive precautions to keep typhoid contamination out of our water and getting into our houses; or from initiating—as is at last happily the case—measures for preventing access of tubercle bacilli, whether through air or through milk.

I confess I think it urgent, in the interests of the many sufferers from gastritis as well as in the interests of those suffering from pyogenic conditions generally, that some similar steps be taken with regard to the mouth—the chief channel of access, in my judgment, of all pyogenic infections.

We may not be able to prevent their access into the mouth any more than we can prevent them adhering to the skin. But, knowing as we now do their potential qualities, there is not the slightest reason why the mouth, so easily accessible as it is to local measures, should be made into a perfect hotbed for their development and propagation.

In relation to the whole group of internal conditions caused by pyogenic organisms, I consider there is a wide field of preventive medicine open by the exercise of oral antisepsis, a field that can be worked in, with the most surprisingly satisfactory results, alike by the physician, surgeon, dental surgeon, and patient. And by oral antisepsis I mean no mere rinsing of the mouth with mildly astringent and antiseptic mouth washes, but (1) the direct application to the diseased tooth or inflamed gum of carbolic acid (1 in 20), repeated daily for just so long a period as the patient will persist in keeping his necrosed tooth or fang, still better (2) the removal of all diseased useless stumps (3) the most scrupulous daily sterilising by boiling of every tooth plate worn, and (4) on the part of dentists the avoidance of too much conservative dentistry and the use of contrivances like "bridges" which cannot possibly be kept aseptic.

REFERENCES.

[1] Dental Diseases in Relation to General Diseases, especially to Infective Gastritis, *Odont. Soc. Trans.*, January, 1899. [2] Oral and Gastric Infection in Anæmia, *Lancet*, February 3rd, 1900.

A CASE OF ANEURYSM OF THE ABDOMINAL AORTA POINTING POSTERIORLY IN WHICH THE INITIAL SYMPTOMS WERE THOSE OF CHRONIC COLITIS.

BY A. ERNEST MAYLARD, M.B., B.S.LOND.,

Surgeon to the Victoria Infirmary, Glasgow.

THE following case, which I will first give the history of before passing any comments upon it, was a peculiarly interesting and puzzling one. The patient was under the care of Dr. Shiels, of Glasgow, to whom he had been sent for special treatment, more particularly of a dietary kind, it being supposed that he was suffering from some irritative or inflammatory disorder of the alimentary canal. Dr. Shiels, however, finding that no permanent improvement seemed to follow purely medical treatment and careful attention to diet, asked me to see him with a view to some possible surgical treatment. Our attention was drawn one morning to the appearance of a marked pulsating swelling in the back close to the spine. It was apparently quite sudden in its appearance, and a very cursory examination on our part left no doubt as to its true nature. Sudden death a few hours later, with immediate collapse of the tumour, equally left no doubt in our minds as to what had taken place.

History of Illness.—Mr. D., aged 35, an engineer by profession, had for the last eighteen months suffered from a troublesome bowel complaint. He was unable to ascribe the commencement of his illness to any particular cause. He believed he once suffered from dysentery when in Egypt. His trouble had consisted mostly in obstinate constipation, with the occasional passage of mucus in the motions. It was stated that on one occasion a mucous cast of the bowel had been passed. The small flakes of lymph that passed were on more than one occasion submitted to microscopical examination. They were found to consist of merely mucous shreds in which were entangled *débris* of granular material; neither blood nor pus cells were ever observed. Pain became in the later stages of his illness a very trying symptom. He stated that he suffered most pain in his bowels prior to the passage of accumulated masses of lymph. The administration of an enema always produced a sense of considerable soreness in the left lumbar region, if the amount introduced exceeded two pints. He further complained of more or less constant "nipping, burning pain" about the eleventh and twelfth ribs in the left axillary line, with tenderness when digital pressure was exercised over this area. His most severe pain, however, was in the region of the epigastrium, which at night so distressed him that morphine had to be given. He always felt pain if he attempted to move or sit up, and selected the almost complete prone position as that which afforded him most ease and comfort.

About twelve days after he had been under my observation, my attention was directed by Dr. Shiels to a pulsating swelling which had more or less suddenly appeared in the left lowest dorsal region close to the spine. It was about 4 inches in length and 3 inches in breadth, occupying a position in the region of the vertebral ends of the ninth, tenth, and eleventh ribs. The pulsation was as marked as if the aorta itself were exposed. The patient complained of a throbbing sensation in the swelling, and also of much discomfort from flatulence. The "wind," he said, collected on the left side and bulged his abdomen there. This was quite evident on inspection; palpation elicited an unduly flaccid condition of the abdominal parietes, and percussion a highly tympanitic state of the bowel beneath. He now complained of pains radiating from the region of the swelling round to the front of the abdomen, and of a loss of tactile sensation. He remained in much the same condition for about twenty-four hours, when, on the nurse returning to his room after an absence for about ten minutes, she found him dead, in the same position as she left him.

The condition of the bowel supposed originally to be present was one of chronic ulceration or membranous colitis, and the surgical treatment under consideration the performance of right inguinal colostomy in order to give temporary rest to the bowel. I had, however, advised against operation on the ground that for some inexplicable reason in the contents of the bowel as removed by the daily use of water enemata no signs of bowel casts, or, indeed, of any inflammatory exudates were present.

A flood of light seemed at once to fall upon the whole history of the case when the aneurysmal swelling appeared in the back. To read the symptoms in the light of this new revelation was to make perfectly clear what had been previously most puzzling. His varied pains and complaints so mystifying to us, and sufficiently delusive to cause some of his previous medical attendants to believe he was unduly complaining, became now all perfectly explicable, as the probable results of the pressure of the aneurysm upon the various nerves, sympathetic and spinal. His obscure bowel condition, also, now seemed to receive its explanation. Pressure had been exercised by the aneurysm upon the mesenteric vessels, and so led to a chronically congested condition of the mucous membrane. When at a later stage, that is to say about the time he was under my observation, this internal pressure was beginning to be lessened by the gradual extension of the aneurysm backwards, some of the more prominent symptoms, such as the passage of flakes of lymph and mucous casts, disappeared.

The case doubtless has an interest from the point of view of an aneurysm of the abdominal aorta presenting posteriorly. Sir William Gairdner, a recognised authority on the subject of aneurysm, in reply to a question of mine on the subject, was kind enough to inform me that he could only recall one case, and that very many years ago, at all presenting features similar to the above. Let alone, therefore, the very puzzling and almost misleading character of the early symptoms, the case seems well worthy of record. It is to be regretted that there was no *post-mortem* examination, but neither Dr. Shiels nor I could persuade the friends into giving their consent. An investigation might have settled more definitely the relation of the aneurysm to the bowel and mesenteric vessels, and also the actual seat and nature of it. There was but one explanation of the sudden death, and that the internal rupture of the aneurysm. Whether this occurred into the bowel or into the general peritoneal cavity it is not possible to say.

DR. PIETRO GROCCO, Professor of Clinical Medicine in the Reale Istituto di Studi Superiori of Florence, has given 5,000 lire (£200) towards the establishment of a provincial sanatorium for poor consumptive patients belonging to Florence.