unexpectedly sterile condition of plates from some loaves—notably from tinned and small loaves (half-quarterns). This does not necessarily prove that a particular loaf is sterile, but only that in the parts examined no bacteria capable of growing were in contact with the gelatine. In the case of a tinned loaf the heat appears to penetrate more effectually, so far as the destruction of organisms is concerned, than in an ordinary loaf. In small loaves the temperature is higher on an average in the centre than in large loaves. 6. The organisms 8 and 9 (see List II.), or their spores, are killed with comparative ease—that is, their death point in dry heat is about the same as that of bacillus tuberculosus under certain conditions. We may therefore assume that if these organisms survive the baking the tubercle bacillus might also survive. We have other reasons for believing that pathogenic organisms often do survive the baking. 7. A further proof of the ubiquity of bacillus subtilis and of the bacilli resembling it is afforded by the frequent preponderance of those organisms in our cultures. On the whole, we are inclined to think that, notwithstanding the comparatively low temperatures reached in the centres of loaves, the conditions are such that the greater number of the bacteria present in the unbaked dough are destroyed.

Accepting the fact that certain non-pathogenic micro-organisms (or their spores) can survive the process of baking in the centre of an ordinary loaf, we argue by analogy that pathogenic forms of bacteria (and their spores) may also pass safely through a similar ordeal. Our experiments, however, have not been confined to mere cultures from bread. We have made many experiments with dough infected with pathogenic organisms, but, as the indispensable evidence, obtainable only by inoculation of living animals, is wanting, we are unable at this stage of the proceedings to publish our researches on this important point.

Enough has been said, we trust, to encourage other bacteriologists to repeat and extend our experiments. If it be proved, as we venture to think has been done, that certain non-pathogenic organisms are able to retain their vitality in the loaf as it leaves the baker's oven, the fact thus established will overthrow the last defence of insanitary bakehouses—namely, that bread is absolutely sterilised by baking, and is therefore harmless to consumers. Another important result may be deduced from the following data: (1) unwholesome bakeries are exposed to infection by pathogenic bacteria—e.g., typhoid fever in sewage; and (2) such bacteria may gain entrance to the dough and maintain their vitality after baking—by analogy with what has been shown to occur in the case of non-pathogenic bacteria. If these premisses be true, it follows that pathogenic organisms may under certain circumstances be distributed in loaves and so produce specific disease among consumers.

We see no particular reason why the origin of many mysterious septic invasions of the human body may not eventually be traced to the agency of bread. A generation ago milk was not suspected of being the means of spreading disease, and a similar observation applies to water. At any rate, the subject dealt with in this paper seems to us to be well worthy the attention of all who are interested in the scientific developments of preventive medicine, no less than in the protection of the public that consumes the bread.

THE PATHOLOGY OF ENLARGEMENT OF THE PROSTATE.

BY C. MANSELL MOULLIN, M.D. OXON., F.R.C.S. ENG.

THERE are three very different views current at the present day with regard to the pathology of enlargement of the prostate. One is based upon a supposed analogy between it and fibroid disease of the uterus. This was first suggested by Velpeau, and is supported by Sir H. Thompson and Professor White of Philadelphia. According to another, enlargement of the prostate is merely one of the occurrences in a constitutional disorder that begins as arterial sclerosis and ends in fibroid degeneration, affecting the genito-urinary organs in an especial manner. This has the support of Guyon, and, following him, of the whole French school. The third has been suggested by Mr. Reginald Harrison, who holds that the primary change is in the bladder, and that the enlargement is secondary to it and developed as a compensatory measure. None of these views can be considered satisfactory. The first is negatived entirely by the facts of development. The homologue of the uterus is not the prostate, but the prostatic utricle, an entirely independent structure, which is included in the prostate gland merely by an accident of growth, and which has never been shown to take the least active share either in tumour formation or in general enlargement of the prostate gland. The homologue of the prostate, if it exists at all in the opposite sex, is to be found in the tissues of the perineum or near the lower ends of the ducts of Gärtner—nowhere near the uterus. Nor is it supported by histology. Uterine growths originate as fibro-myomata. They have little or nothing to do with the mucous membrane or the glands that it contains. Enlargement of the prostate, on the other hand, is glandular from the first; it begins and spreads as an adenomatous growth, and even the rounded masses that it contains originate, with few exceptions, in the glandular tissue. Both organs, it is true, contain unstriped muscular fibre (although the proportion in which this is present in each and the arrangements and uses are entirely different), and the growths that originate in each of them resemble one another to a certain extent in shape (they are usually rounded) and in their anatomical relations (sometimes they are encapsuled, sometimes they assume a polypoid form); but these peculiarities are by no means confined to these organs or their growths. Unstriped muscular fibre occurs in abundance elsewhere. Rounded encapsuled masses that shell out when they are exposed by section are by no means rare in fibrosarcomata; and tumours, when they project into the cavities of other viscera, not infrequently become polypoid in shape. That these two organs, uterus and prostate, should both be liable to tumour formation and enlargement during the latter half of the reproductive period, when the first flush of full sexual activity is over, is a fact of great significance in many ways; but it is scarcely sufficient in itself to recommend the adoption of this theory even provisionally.

Guyon's hypothesis is still less tenable. It is true, as Sir Benjamin Brodie has pointed out, that enlargement of the prostate and atheroma frequently occur together; the time of life at which they are both most common is the same; but this does not show in the least degree that the one is the cause of the other. Enlargement of the prostate does not begin as fibroid degeneration but as glandular overgrowth. Arterial sclerosis and passive congestion may do a great deal, but they cannot cause this, and still less can they cause the growth of masses that sometimes almost fill the lower part of the bladder. Moreover, enlargement of the prostate is compatible with perfect health, continuing sometimes even to extreme old age without its existence being so much as suspected. Those who suffer from urinary troubles form but a small proportion of those in whom enlargement occurs. The vast majority are free, and, although some may be atheromatous, many live on with no evidence of vascular degeneration other than that which is the normal companion of healthy old age.

There is even less to be said in favour of the view that the primary change is in the bladder and that the enlargement is a compensatory contrivance. The prostate, both as regards its glandular substance and its stroma, is of sexual origin, and has to deal only with sexual functions. It develops with the testes and atrophies if they are removed. It has nothing to do with micturition, which, if the prostate remains undeveloped or undergoes atrophy, takes place just as well as it does if the prostate is normal. Nor is there any evidence that the sinking of the posterior part of the bladder is a primary change, or that when it does sink the prostate enlarges in compensation. This can only be imagined so long as the erect position is maintained and the prostate bears the same relation to the bladder that it does in man; whereas enlargement takes place in dogs and other quadrupeds, in which the anatomical relations are entirely different. Nor is it easy to understand why compensation for sinking of the posterior wall of the bladder is confined to one sex.

None of these theories, therefore, can be considered as offering a satisfactory explanation. There are, however, certain facts, some long since known and almost forgotten, and others of recent discovery, that suggest that the real cause is to be found in another direction. The enlargement is in some way dependent upon the testes. The evidence in favour of this is briefly as follows. The normal development

of the prostate is undoubtedly controlled by that of the testes. Up to puberty there is no prostate worth mentioning. If castration is performed before puberty the prostate never grows; if after puberty it wastes and disappears, and the same has now been shown to be true of the abnormal development known as enlargement. This rarely begins after an age at which it may be presumed that the testes are no longer functional (Sir H. Thompson and Sir G. Humphry), and it disappears if they are removed. There are now nine cases on record, in which castration has been followed by complete disappearance of the enlargement within a very few weeks, every single case in which any note as to the condition of the prostate has been taken.[1] There can, therefore, be no question that in some way or other the condition of the testes controls that of the prostate both in its normal and enlarged form.

In this relation between the prostate and the testes there is a very close parallel to that which exists between the ovaries and the uterus. Nor is this surprising. The testes in the one sex are the dominant organs in the sexual system, and the ovaries in the other. The influence that each exerts upon the other (secondary) sexual organs is closely similar, and, perhaps, identical; but this must not be taken as showing that any closer homology exists between the prostate and the uterus (or between prostatic enlargement and fibroid disease) than that which exists between the testes and the ovaries. In what way the testes exert this influence upon the prostate so as to cause it to enlarge must remain at present a matter for speculation. The mere induction of sterility—as, for example, by section of the vasa deferentia—although it may be followed by wasting of the testes, does not appear to be sufficient.[2] The influence, whatever it is, comes from the testes themselves, and it exists so long as the testes are present. (It is to be noted that although enlargement of the prostate rarely commences late in life, presumably because at that time the testes are no longer active, their loss of functional activity does not induce atrophy of an already enlarged gland.) It may be exerted through the agency of the nervous system, or—as there is little or no proof that the nervous system has any direct control over normal and much less over pathological growth—through that of the circulation. Evidence is slowly accumulating that the nutrition of distant parts of the body is in some instances dependent upon the integrity of organs with which at first sight they appear to have no connexion. Affections of the thyroid gland, for example, rendering it deficient in some unknown substance, can produce the most profound structural alterations throughout the body. It is believed, on experimental evidence, that the pancreas may have duties in connexion with general nutrition entirely independent of its secreting action. There is reason to think that the condition of the ovaries has some influence in connexion with mollities ossium; there are, at any rate, many cases on record in which the removal of these organs is stated to have been followed by recovery from this otherwise almost universally progressive disease. And though there is as yet no proof that morbid states of the testes (as distinguished from simple atrophy or removal) can induce changes in secondary sexual characters, this is undoubtedly true of the ovaries, as, for instance, the assumption of the male plumage by the females of various birds when the ovary is diseased.

If this suggestion that enlargement of the prostate is dependent upon some change that takes place in the testes during the latter part of their active life should be accepted—and certain facts point strongly in its favour—it is a curious instance of the way in which modern views are sometimes foreshadowed by the speculative theories of a long past date.

Note.—Since the above was written cases have been published by Watson of Boston and Lannois in which unilateral atrophy of the prostate has followed removal of one of the testes. If this can be established by post-mortem examination it may be taken as proof that the agency through which the influence of the testes is exerted upon the prostate is the nervous system and not the circulation.

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[1] Ramm of Christiania, Centralblatt für Chirurgie, No. 35, Sept. 2nd, 1893, and No. 17, April 28th, 1894; Arthur Powell, Brit. Med. Jour., Nov. 18th, 1893; Professor White of Philadelphia, Brit. Med. Jour., June 23rd, 1894; Mansell Moullin, Brit. Med. Ass., Aug. 1894; and the following quoted by Professor White: Haynes of Los Angelos, California, Buffalo Med. and Surg. Jour., March, 1894, and Fremont Smith of St. Augustine, Florida, New York Academy of Medicine, 1894.

[2] Griffiths, Brit. Med. Jour., Sept. 30th, 1893, and Haynes, loc. cit.

A SUCCESSFUL CASE OF ENTERECTOMY WITH THE USE OF MURPHY'S BUTTON.

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ATTENTION has recently been drawn in THE LANCET[1] to the use of Murphy's button in intestinal resections. The following case I venture to report at some length, as the use of the button by English surgeons has not hitherto, so far as I am aware, been extensive. In October, 1889, I was asked by Messrs. Chubb and Vinter of Torpoint to see a lad who had received a severe injury by falling under the prow of the steam ferry. The principal lesion was abdominal, and resulted in acute peritonitis, from which the boy recovered after a prolonged illness. I saw him some little time afterwards, when he was suffering from fæcal impaction, and it was only after many days' use of injections and other remedies that his bowels were "cleared out." In the middle of September of this year I was again asked to see him, and the following history was obtained. His age was fifteen. Ever since the accident he had suffered from periodical attacks of violent abdominal pain, accompanied with great distension and violent vomiting, which on occasions had become fæcal. The attacks commenced after some constipation and terminated in violent diarrhœa. Their frequency had of late increased, recurring sometimes twice a week. When I saw him he had just passed through one of these attacks. His abdomen was much distended but not tender. There was dulness on percussion over the cæcum and ascending colon. The countenance was pinched and anxious. My diagnosis was that, as the result of the peritonitis, he had some constricting band, and my advice was that an operation should be performed for his relief. He was consequently admitted into the Private Home for Patients at Plymouth, and on Sept. 27th I opened his abdomen. On passing my hand down into the right iliac fossa I found collapsed bowel and a broad band descending into the pelvis. The small intestine above this was enormously dilated, the circumference of the bowel being in some parts from twelve to fifteen inches. The constricting band was divided, but the intestines were glued together to such an extent that I had very considerable difficulty in separating the adhesions, as many as twenty pressure forceps being on at once. When I was able to draw out the part it was found that a dense stricture existed, through which it was nearly impossible to pass on the contents of the upper bowel. During the attempt to do so a small leak was detected at the point of stricture. It was at once decided to remove the constricted portion of the bowel. An elastic ligature was placed above and below this point and about five inches of gut were removed. The contents of the upper bowel were emptied as far as possible; it contained a large number of cherry and plum stones. The intestine was reunited with Murphy's button. A description of the method is fully given in the article alluded to above. The actual excision of the stricture and the reunion of the gut did not occupy ten minutes. During the night the patient was sick several times, and about midnight he became rather collapsed. He was fed per rectum every four hours with beef-tea and brandy. On Sept. 28th he was very quiet and was not in pain. The temperature was 98·4° F. in the morning and 99° in the evening. He was fed by the mouth every hour with small quantities of peptonised milk-and-water (equal parts), with an occasional nutrient enema. On the 29th he was comfortable. He passed flatus freely by the rectum. The temperature was 99·6° in the morning and 99° in the evening. The milk-and-water was continued in increased quantity. On the 30th he passed a dark liquid motion on three occasions. On Oct. 1st the bowels were opened; the resulting evacuation contained three orange pips. On the 2nd he passed an offensive motion containing milk curds and orange pips. On the 4th the silkworm gut sutures were removed; the abdominal wound had healed. On the 6th there had been no motion for three days. A large quantity of fæces was felt in the rectum. A simple enema produced a hard, lumpy evacuation. On the 7th he passed a loose motion containing a cherry stone, and another containing a plum stone on the following day after an injection. On the 10th, after an enema, he passed a large motion containing the button, nine

[1] THE LANCET, Sept. 15th, 1894.