

3. The after treatment is much easier, since the flank region can be better treated by washing than the region of the scrotum.

The artificial perforation of the abdominal wall by the Danish method is dangerous, and it would, in my opinion, be best to altogether give it up. In the cases where the testicle or the epididymis has its position in the inguinal canal Dégive's method is to be practised ; but in all other cases the method which I have sketched might be recommended as the least dangerous.

By numerous examinations I have been able to prove that the assertion in books that the non-descended testicle lies in most cases either in the internal abdominal ring or in its neighbourhood is erroneous. It might much rather be asserted that in most cases the testicle is fastened by a peritoneal fold in the lumbar region.

### TUBERCULAR MASTITIS IN THE COW.

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THE channel by which the udder becomes infected with tubercular disease is as yet a matter of speculation. In all the cases that I have had the opportunity to examine the lesion had already invaded a large part of the gland before the death of the animal. The microscopic examination of the gland in such cases, while enabling one to trace clearly enough the manner in which the disease extends when once it has obtained a foothold, throws no light on the starting-point of the lesion.

On theoretical grounds we may admit three possible channels by which the bacilli might gain access to the gland, viz. by the blood stream, by the lymph stream, or through the teat canal. As I shall presently show, the milk cistern, and even the teat canal itself, is not rarely the seat of a tubercular process, and this might be held to indicate direct infection from the exterior. But in general a comparison of the disease in the milk cistern with that in the higher parts of the udder shows clearly enough that the former has not been the initial lesion.

I am inclined to believe that in most, if not all, cases, the blood is the vehicle of infection. So far as I am aware there is not on record any case in which the udder lesion was the only evidence of tuberculosis in the body. But, at the same time, it must be admitted that the tubercular lesions elsewhere than in the gland may be of unimportant extent, and certainly the infection of the udder is not explainable as part of a general tuberculosis, using that term in the sense in which it is employed in human pathology. Instances of the sudden and simultaneously development of tubercles throughout the body (lung, liver, spleen, kidney, etc.) are very rare in cattle—much more rare than cases of mammary tuberculosis. And even although the udder were thus infected as the result of an abundant irruption of bacilli into the blood stream, the fact would not explain the ordinary udder lesion, for the progress of the disease in other organs would certainly entail the death of the animal long before the

mammary lesion had reached the advanced stage in which it is commonly encountered. If, as Bollinger's experiments appear to prove, the milk of tubercular cows, even when the visceral lesions are not very extensive, and where there is no discoverable disease in the udder itself, is in many cases infective, we are almost driven to admit that the circulation of bacilli with the blood is much more common than has generally been supposed.

Assuming for a moment that in chronic tuberculosis of some of the visceral organs there are occasional slight irruptions of bacilli into the blood stream, the udder, in common with the other organs of the body, would be exposed to the risk of infection ; and if, as there seems some reason to believe, the mammary tissue offers a very favourable situation for the settlement and multiplication of the bacilli, it might become the seat of disease, while organs which in the human subject appear much more susceptible might escape.

It must be admitted, however, that some strong objections suggest themselves to this hypothesis, the most forcible of which is that if bacilli are as commonly present in the milk of healthy udders as Bollinger's experiments would lead us to believe, we can hardly number the udder among the organs that offer a favourable soil for the settlement and multiplication of the bacilli, for in that case udder affection would be one of the most constant lesions in tubercular cows. Not only as bearing upon this point, but also, and in a greater degree, as bearing in a vital manner upon public health, it appears to me that a repetition of Bollinger's experiments is urgently demanded.

Whatever may be the circumstances that start the development of tubercular disease in the udder, when once it has obtained a foothold its subsequent course is one of steady progress. To Professor Bang of Copenhagen we are indebted for having brought into prominent light this marked clinical distinction between tubercular and other forms of mastitis in the cow. In the far more common non-tubercular forms of inflammation, the ultimate effect, after the subsidence of the more acute symptoms, is a decided diminution in the size of the diseased quarter. Tubercular disease, on the other hand, invariably adds to the volume and density of the quarter attacked. Whenever a chronic, comparatively painless, hypertrophic inflammation of the udder is met with, tuberculosis may almost with certainty be diagnosed.

When a tubercular udder in the advanced stage of the disease is cut into after death, the most notable features of the affected portion of the gland are its increased solidity and firmness. I do not think, however, that, apart from the combination of enlargement and firmness, the macroscopic characters are so distinctive here as in tuberculosis of other organs. Discrete tubercles are very difficult to detect with the naked eye in the texture of the mammary gland, and, as will presently be shown, the formation of actual tubercles is quite exceptional in tuberculosis of the udder. The pultaceous yellow caseous material so commonly met with in the chronic tubercular disease of the lung and liver of cattle, I have never seen in the udder, nor had calcification taken place in any of the udders that I have examined.

The cut surface of the most solid parts of the gland are much smoother than normal, and greyish yellow in colour. The colour,

however, is not uniform over even small areas, and I cannot agree with Bang that there is always a sharp line of demarcation between the healthy and the diseased portions of the gland.

The mucous lining of the milk cistern, which is normally smooth, but slightly folded in the collapsed state, is sometimes uniformly studded with minute papillary or villous-like projections; and the lining membrane of the recesses of this cavity, the larger ducts which open into them, and the teat canal may show the same appearance.

When a well-stained section made from the margin of the more solid part of the udder is examined under a low power of the microscope, the first observable departure from the normal structure is a broadening of the interacinous septum or space. During the most active period of lactation the ultimate acini of the gland are, in the same

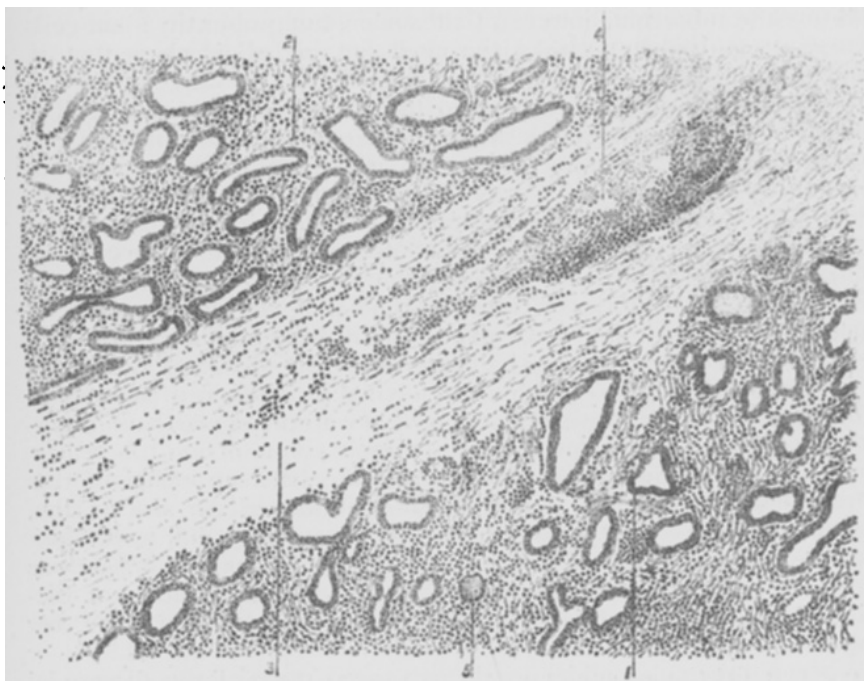


FIG. 1.—Section of tubercular udder, stained with micro-lithium carmine ( $\times 50$ ). 1 and 2. Portions of two adjoining lobules, showing interstitial new tissue surrounding acini, some of which are irregularly dilated, while others are compressed and atrophied; 3. An interlobular septum; 4. Tubercular infiltration of septum, with four giant cells; 5. A giant cell in the interacinous new tissue.

lobule, closely packed together, the breadth of the space between the epithelial lining of two contiguous acini being in most cases only a little greater than the height of the epithelial cells. The space which in a microscopic section of the healthy active mammary gland intervenes between the basement membrane of the one acinus and that of its immediate neighbours is occupied in general by a single blood capillary, running apparently in a lymphatic space. In general no nuclei are to be seen in this interacinous space, save those that belong to the capillary or an occasional leucocyte. In tracing the progress of the tubercular lesion within a lobule, one observes that the adjacent

acini become thrust apart ; and on closer examination the nuclei of the capillary wall are seen to be swollen and prominent, and external to the capillary there are found free-lying cells. The smallest of these cells have the characters of leucocytes, but when the cellular infiltration is more marked, larger and more irregularly shaped elements have made their appearance. Within the same lobule the process may be followed step by step, from a condition in which there are merely a few free-lying cells around the capillary, up to places at which the interacinous space has become increased to ten or twenty times its original breadth, by a dense cellular infiltration and proliferation.

The process which has just been sketched is simply that of an interstitial inflammation of a sub-acute or chronic character, and it so far is the exact parallel of what one frequently meets with in the cortex of the kidney. In its early stage there is absolutely nothing to indicate the tubercular nature of the lesion, but presently giant-cells appear at many places amid the round and epithelioid elements that constitute the bulk of the new material.

When attention is next turned to the glandular tissue proper, it is observed that, *pari passu* with the before-described interstitial changes, the acini themselves experience a marked alteration. The first evidence of the injury inflicted by the interstitial infiltration is a tendency of the epithelial cells to demit their milk-forming function. The cells shrink in breadth, and milk globules are no longer observable in their protoplasm or in the lumen of the acinus. As a result of the pressure exercised by the interstitial infiltration, the lumen of most of the acini becomes diminished, and eventually obliterated. Some of the acini and ducts, on the other hand, become distended, and assume an irregular outline. There is thus a tendency to the formation of cyst-like dilatations, but these appear never to attain more than microscopic dimensions.

A comparison has already been instituted between certain forms of interstitial nephritis and the lesion now under consideration, but I must here call attention to an interesting difference. In renal inflammation the interstitial change exerts a markedly injurious influence on the tubular epithelium, which speedily becomes denucleated and granular, and eventually destroyed. In this tubercular inflammation of the mamma the glandular epithelium seldom or never exhibits these appearances. As long as the outlines of the acini can be followed in the midst of the tubercular new tissue, the individual epithelial cells, save for alteration in shape and size, appear little affected, and their nuclei stains as deeply as in the healthy gland. Occasionally, however, where a marked thickening of the basement membrane occurs, the epithelium appears to suffer from the pressure, gradually disappearing by a process of atrophy.

In one case of advanced tubercular mastitis I found great numbers of the so-called amyloid bodies in the cirrhotic parts of the gland. They were in some cases connected obviously with the gland acini, but in numerous other cases they lay in parts where the gland texture had been entirely destroyed. In the latter situation also they had probably been formed in connection with the glandular epithelium, and had persisted after the latter had disappeared. The bodies seldom or never appeared to begin in the lumen of the duct or acinus,

but in the epithelium, where that had become stratified. They seemed to result from a metamorphosis of the epithelial cells, some of which, still giving a healthy reaction, could often be seen closely applied to the surface of the concretion (Fig. 2). Most of them showed distinct concentric markings, and they exhibited all the reactions of the amyloid substance, staining brown with iodine, faint blue with iodine and sulphuric acid, and pink with methyl-violet. Tubercle bacilli could never be detected in them.

In some instances the basement membrane of the acini becomes thicker and more distinct than normal, and not rarely their epithelium forms two or three strata. Sometimes, but only exceptionally, the glandular epithelium shows a catarrhal proliferation. When this occurs the lumen of the duct or acinus becomes distended with a mass of small cells, to which further reference will hereafter be made in speaking of the distribution of the bacilli.

As the interstitial lesion progresses the gland texture over large areas becomes entirely obliterated. The basement membrane of

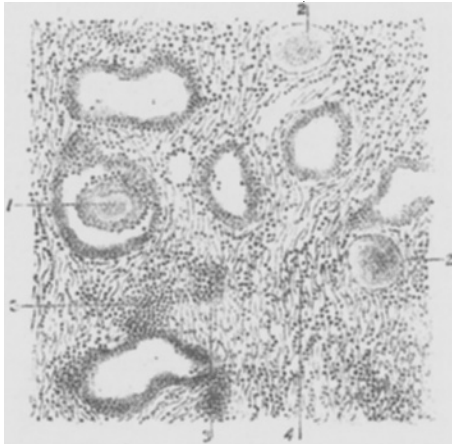


FIG. 2.—Section of tubercular udder, stained with hæmatorylin ( $\times 50$ ). 1. An amyloid body projecting into the lumen of an acinus and surrounded by epithelial cells; 2 2. Amyloid bodies in interstitial new tissue; 3 3. Epithelium of acini on the point of being destroyed; 4. Interstitial new tissue, faintly fibrous.

the compressed acini appears to become broken through, and the epithelium to be invaded from without by the interstitial infiltration. The outlines of the acini are thus gradually obliterated, but after this, at some places, groups of epithelial cells may still be recognised among the new elements of the part.

The parts at which the gland texture has been entirely destroyed are found in one of two conditions. In the first of these there has been an attempt at the formation of fibrous connective tissue. This is delicate and scarcely ever distinctly fibrillated, and giant-cells are generally present in it. The second condition, which in point of time follows very closely upon the preceding, is that of necrosis and caseation. These processes do not present any very notable features compared with those seen in other organs, save that the caseation shows much less tendency to softening than in most other situations in cattle,

and it is at the same time much less prone to be followed by calcification.

It has already been stated that the occurrence of actual discrete tubercles similar to those met with in a general miliary tuberculosis is very rare in the udder. Occasionally within the coarser interlobular connective tissue discrete islands of small cells may be found, and the same may be seen within a lobule. But it seems to me that these always tend to grow peripherally, and never show the concentric grouping of their elements described in connection with the typical tubercle of human pathology. Sometimes, within an area in which the gland texture has been completely destroyed by the steps already described, small more or less well-defined foci may be seen which differ from their surroundings in having experienced a more advanced fibroid transformation. But the common lesion in tubercular mastitis

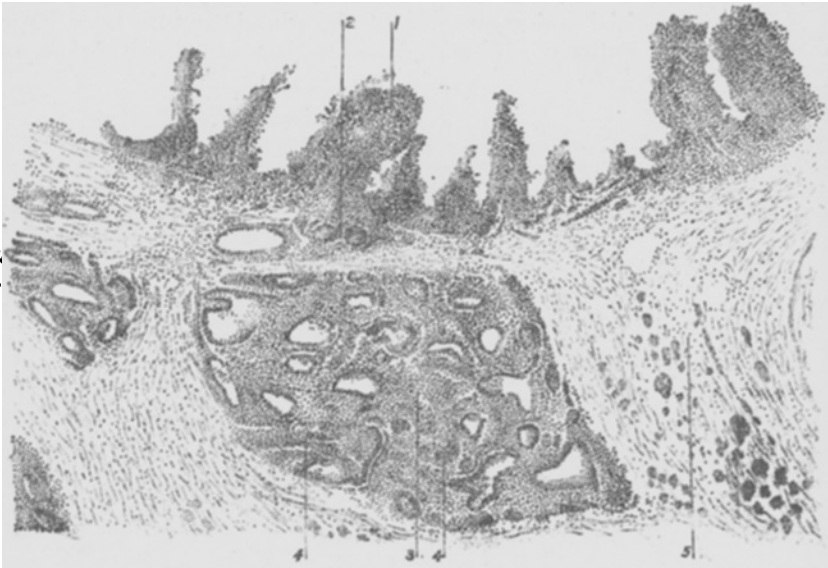


FIG. 3.—Section to show tubercular lesion of milk cistern, stained with picro-lithium carmine ( $\times 40$ ).  
 1. Villus-like granulation projecting from lining membrane of milk cistern (compare Pl. II, fig. D); 2. Two small acini on point of being destroyed by small-celled infiltration; 3. A lobule showing acute tubercular small-celled infiltration destroying the gland acini; 4. Remains of acini with epithelium still secreting fat droplets; 5. Interlobular connective tissue.

is a chronic diffuse interstitial inflammation, and it is neither tubercular nor nodular, using these words in their anatomical sense.

A microscopic examination of those cases in which the mucous membrane of the milk cistern presents the papillary or villous character already mentioned, brings to light some points of great interest and importance. The minute structure of this lesion is shown in Fig. 3. The surface of the lining membrane is seen to be studded with minute conical projections, each of which is composed of granulation tissue. The early stage in the formation of these elevations is a catarrhal desquamation of the epithelium, accompanied by a small-celled infiltration of the subjacent mucosa. The larger projections have entirely lost their epithelium; they possess a structure

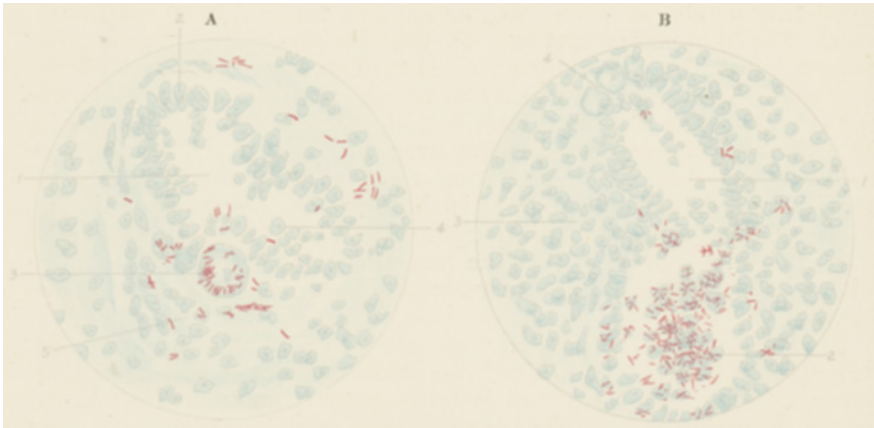


FIG. A.—1. Lumen of an acinus; 2. Epithelium of the same; 3. Giant-cell containing bacilli; 4. Free-lying cells in lumen; 5. Tubercular new tissue with bacilli scattered through it.

FIG. B.—1. Lumen of an acinus; 2. Group of cells with numerous bacilli within the lumen; 3. Interacineous new tissue, with a few bacilli at some places; 4. Large vacuolated epithelial cells.

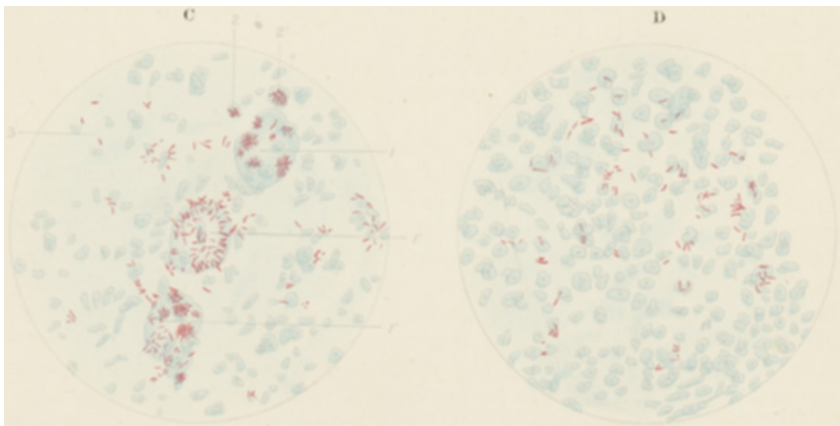


FIG. C.—1, 1'. Giant-cells containing numerous bacilli; 2, 2'. Nuclei containing clumps of bacilli; 3. Tubercular new tissue on the point of caseating, containing a few scattered bacilli.

FIG. D.—Tubercle bacilli in tip of villus-like projection from lining of milk cistern (compare with Fig. 3, p. 124).

### TUBERCULAR MASTITIS IN THE COW.

The whole of the figures are drawn under Reichert's  $\frac{1}{2}$  oil-imm., oc. 3, and the sections were stained by the method of Ziehl-Neelsen.

identical with that of a wound granulation, their deeper parts showing distinct capillary loops. At the apices of the projections many degenerating or necrosed elements are on the point of being detached to mingle with the contents of the milk cistern. The large ducts which open into the latter are in a condition of catarrh, and the mammary lobules adjoining the cistern show an early stage of tubercular interstitial inflammation. In the case from which the accompanying illustration was taken the lesion of the cistern was not due to a simple extension of the tubercular process from the adjacent lobules, for these were for the most part but slightly affected, and formed, in fact, the least altered part of the quarter.

The minute anatomy of tubercular mastitis affords an explanation of the recognised clinical characters of the disease. The reaction of the tissues is of a sub-acute or chronic character, and the violent vascular disturbance so common in acute non-tubercular forms of mastitis is not seen. Hence the lesion in general runs a comparatively painless course.

The lesion is essentially an interstitial one, and catarrh of the secretory epithelium is never a prominent feature of the morbid process. Hence, the milk in general is not notably altered in its physical characters during the earlier stages of the disease.

There is, of course, a steady diminution of the milk secretion, proportional to the extension of the disease and the consequent destruction of gland tissue. But even after the epithelium of a compressed acinus has ceased to form milk globules, some watery fluid is probably secreted by it. This supposition would at once explain the tendency to cyst formation, and the watery character of the otherwise apparently normal milk secreted during the early stage of tubercular mastitis.

The tubercular process when once started steadily spreads by peripheral extension, destroying the gland texture, and substituting for it a firm new tissue. In this respect the lesion resembles much more closely the growth of a neoplasm than it does the other forms of mastitis.

It yet remains to describe the distribution of the specific bacilli in the tubercular lesions. In the first stage of the process, such as that depicted in Fig. 1, bacilli are very sparingly demonstrable in the interstitial infiltration, and in the glandular epithelium or the lumen of the acini they are in my experience not to be found at all. But when the interstitial growth has advanced to that condition in which giant-cells are numerously present in it, and when the acini are becoming destroyed, bacilli are always present in large numbers. The giant-cells generally contain great numbers of them, sometimes radially disposed around the margin, sometimes irregularly grouped throughout the cell substance. Bacilli are also found lying free, or singly or in packets in the round and epithelioid cells of the new tissue.

When a section suitably stained is examined with a low power, the bacilli are always seen to be plentiful in those parts where the destruction of the gland tissue is most complete. Here they are frequently so numerous that after decolorising in acid a degree of colour obvious to the naked eye still remains in the section. They are most numerous in the parts that are in the incipient stage of necrosis, and as caseation sets in their numbers diminish. In the actually caseous portions some



of the still visible rods stain faintly, and both here and in the still living texture many of the bacilli are seen to be sporulating. Wherever the tubercular new growth has broken through the basement membrane of the acini or ducts, bacilli may be found between the epithelial cells or in the lumen; and even where the basement membrane still appears intact, if the bacilli are numerous in the immediately adjoining texture, groups of rods may be seen internal to the basement membrane. Here they are contained within round cells which appear to be leucocytes that have penetrated the basement membrane.

It has previously been mentioned that although catarrhal proliferation of the glandular epithelium is always a subsidiary phenomenon, it does occur in some of the tubes or acini, and in these cases the bacilli are often extremely abundant in the catarrhal products. Lastly, the bacilli are numerous in the before-described villous-like outgrowths of the milk cistern, and in the lumen and walls of the large milk ducts when these become the seat of catarrh. In the villous projections the rods are most abundant towards the apex, where the vitality of the cells appears to be feeblest. Their great importance here is obvious. The presence of bacilli in the interstitial tissue, or even in a catarrhal plug of the ultimate acini or smaller ducts, is of far less consequence, for when so situated they may never leave the udder. But the lesion of the milk cistern illustrated in Fig. 3, and in Plate II. Fig. D must, as long as any part of the affected quarter is active, seriously contaminate the milk with bacilli.

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### SOME OF THE MORE IMPORTANT ANTISEPTICS AND THEIR USE IN VETERINARY SURGERY.

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THE modern researches on the nature of traumatic infective fevers, septicæmia, pyæmia, and the allied conditions, it need scarcely be remarked, at this stage of the controversy, all go to prove that the *fons et origo mali* may be summed up in the single word Bacteria. These bacteria having found suitable nidus and pabulum in the damaged tissues and exuded fluids in or near a wound, be the wound designedly or accidentally produced, are enabled to multiply locally, and then in some cases to invade surrounding tissues or distant organs or tissues, where by their action they may set up further changes. Even when their localisation is complete, they may form special, readily absorbable ferments, xymines, or ptomaines, which have most marked actions on the nerve centres or upon some special set of functions.

Having become disciples of Lister to the extent that we accept this doctrine as true in all essential features, to what conclusions does he lead us as to the prevention of these distressing conditions?

Listerism may be interpreted to mean asepticism, not antisepti-