On tuberculosis : a practical examination of the action of local inflammation in cachectic subjects in the production of tubercular consumption : read before the Medical Society of London, January 23, 1865 / by James Jones.

ON

TUBERCULOSIS:

A PRACTICAL EXAMINATION OF THE ACTION OF LOCAL INFLAMMATION IN CACHECTIC SUBJECTS IN THE PRODUCTION OF

TUBERCULAR CONSUMPTION.

Read before the MEDICAL SOCIETY OF LONDON, *January* 23, 1865.

BY

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ON INFLAMMATION AS AN EXCITING CAUSE OF TUBERCULOSIS.

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Of the various theories that have been propounded by pathologists in explanation of the origin of tubercle, two only appear to have gained a firm position. The one, sometimes called the theory of spontaneous origin, accepts tuberculosis as an essential part in the progress of certain morbid conditions of constitution conveniently termed tubercular cachexia, and denies the necessity for a local exciting cause. The other maintains that tubercle is only the result of inflammation. Some years ago, the latter theory exercised so strong an influence over men's minds, as to lead to the free use of antiphlogistic treatment, including mercury, leeches, and even the use of the lancet. Then came a reaction in favour of the former theory, introducing as its natural consequence a treatment diametrically opposite. This, not unaptly called *the beef-steak and brandy system*, continues to exercise almost universal sway. The advocates of each system have laid claim to a certain amount of success; but, measured by the statistics of the Registrar-General, neither can honestly boast of much comparative superiority. It would appear, therefore, that the time has arrived for a re-investigation of the merits of each of those theories, and as a step in that direction I venture to lay before you a few observations on inflammation as an exciting cause or local factor in tuberculosis in cases which have come under my care as an hospital physician.

In taking a comprehensive view of the various cacoplastic formations found in cachectic subjects, we find a very marked analogy between them and the plasma exuded in ordinary sthenic inflammation, the chief difference consisting in the amount of *their vital capacity or power of organization.* In some, the resemblance is so great, as to amount, I think, to identity. Who, for instance, could draw a line of demarcation between tubercular infiltration and the low plasma found in the pneumonia of debilitated subjects? In others it is less marked, but still assuming a strongly analogous character; and, again, in others, morphology indicates, as their origin, a hyperactivity of the normal functions of the organ in which they are formed (corresponding to the exaltation of function attending the *first stage of inflammation,*

or, as it is generally called, "irritation"), coupled with a low vital capacity or imperfect power of organization.

1. If we commence at the bottom of the series by examining what is known as lardaceous exudation, we find a plasma, if, indeed, it merit that name, which is capable of advancing little or nothing in organization. Its vitality is so low, that as it exudes so it remains, with scarcely a trace of organization.

2. Advancing a little higher, we find the plasma known as tubercular infiltration. This, although but little superior to the lardaceous, asserts its claim to a higher place amongst the plasmata, by exhibiting a certain amount of capacity for organization in a scanty fibrillation and cellulation.

3. Next in the ascending scale comes the miliary, or grey semi-transparent tubercle, showing under the microscope a still greater development of fibre and cell.

4. Then comes the yellow, or common form of tubercle, to which, I suppose, we may give the next place in our ascending scale, inasmuch as it is found to consist of abortive cells, granules, &c., which indicates the presence of a degree of vital power sufficient to originate a cell-growth, but insufficient for its completion.

5. The next higher place may be assigned to cancer, inasmuch as it exhibits a greater degree of vital capacity for organization; for, instead of an abortive, we find a well-matured cell-growth. And although incapable of any higher organism, its cells are endowed with the power of reproduction, and consequently of unlimited increase; hence its malignancy.

6. Next, we have a plasma capable of an organization, nearly approaching normal growth, as found in fibrous tumours, &c.

The next point worthy of notice in our comparative examination of the cacoplasmata is their relation to the state of health under which they are formed. We find that their formation is invariably preceded and accompanied by a condition of general cachexia; but we also find that this condition of cachexia may exist for an indefinite time without being attended or followed by the production of any one of those substances. Therefore the condition of cachexia is not, *per se*, capable of giving origin to any one of them; it must be aided by some other factor.

Thus, a person who inherits a cachectic constitution from cancerous parents may live to a good old age, and finally die of natural decay; but a brother or sister may, from the irritation of a tobacco-pipe, or a blow on the breast, die of cancer of the lip or breast.

Again, in a large family, the offspring of consumptive parents we often find the well-known characteristics of phthisical cachexia stamped on each one; yet some of them will survive to a good old age without a trace of tubercle, while the others

die in early or middle life of consumption. Now, it is evident that in the cases of the latter, some new and additional influence came into operation, taking the position of a complementary factor in the production of tubercle (cachexia + χ = tubercle).

The occasional occurrence of cases in which tubercle is formed in the offspring of consumptive parents without any *apparent* exciting cause, is considered by many well-known pathologists to be sufficient evidence to prove the doctrine of *spontaneous origin;* but it appears to me that such inference is not legitimate, and that it is much more probable that the immediate exciting cause or local factor escaped notice than that it had no existence. There is no caprice in Nature; there must be cause for effect.

We see tubercular cachexia continuing for years, or even a long life, without a particle of tubercle being formed, even under circumstances not the most favourable to the patient's escape. Now, it appears to me that one single instance of this kind is sufficient to upset the theory of the spontaneous origin of tubercle, for it shows that there is something more than a morbid constitutional diathesis necessary in order to bring tuberculosis into action.

Again, in all forms of cachexia, we find the general nutrition of the body to proceed *normally*, although languidly. The various parts and organs receive a supply of nourishment sufficient for the purposes of life, *but a period at length arrives in which, in some part or organ, an abnormal plasma or blastema is formed,* and this act of abnormal nutrition initiates the local disease. Now, I would ask, What is the cause of this sudden alteration in the nutrition of a part? Is it not the effect of some new power coming into operation, local in its action, and capable by its presence of perverting nutrition? What is this new power or factor?

I will now attempt to show by typical examples that in a large number, if not in all, of at least one form of cacoplasm, namely, tubercle, inflammation is the active factor or additional power sought for, not the sthenic inflammation of vigorous subjects, but inflammation of a low type, a type modified by the constitutional diathesis, whose products are low, imperfectly vitalised, and incapable of assuming any high degree of organization. (In the term "inflammation" I comprehend all those abnormal conditions of the circulation and innervation of a part which give rise to effusion of some form of coagulable lymph or plasma, or to an abnormal increase in functional activity of organs under their influence. I include in it, therefore, the condition commonly called "irritation" as one of its phases.)

I shall also point out the importance of watching for the earliest manifestations of inflammatory action, and the early use

of such remedies as science or empirical experience indicate for its removal, and thus, by anticipation, to oppose the onset of tuberculosis; also the necessity of contemporaneously pursuing a constitutional treatment suited to the peculiar nature of the cachexia.

Those who have an opportunity of seeing large numbers of cases of consumption must be struck with the fact that a majority of patients attribute the origin of their illness to "catching cold," or "an attack of bronchitis," or "inflammation of the chest," and that, if careful inquiry be made, their statements are found to be correct.

The following case is a type of a numerous class in example:—A patient, although showing symptoms of low health, is to all appearance free from actual disease until, through some indiscretion, a cold is caught. Cough comes on, with tightness of the chest, and chills, alternating with flushes of heat, are felt. After, a white expectoration appears, the cough continues, the urine is loaded with lithates, the tongue is coated, and the appetite fails; auscultation reveals nothing more than the ordinary signs of bronchitis, and *there is no dulness on percussion.* The patient hopes soon to get better of his cold, but he is disappointed; the cough continues, the expectoration is no less, and is not unfrequently tinged with blood. He loses flesh and strength, and is easily made to perspire. By-and-by, if the chest be examined, *dulness* under or behind one or both clavicles, or the supra-spinal ridge of the scapula, *is readily made out.* Tubercle has formed. This is not an ideal case, but one of a very large number, the history of one of which is the history of all. It is pulmonary phthisis, originating in catarrh. The patient was in pretty good health, or, at all events, free from tubercle until the continued disturbing influence of the catarrhal inflammation produced a condition of active tuberculosis.

I will next give a case of acute tuberculosis, consequent on severe bronchitis. It is taken from the work "On Diseases of the Chest" by Dr. William Stokes, p. 392:—"A patient previously in good health is suddenly attacked with the most violent symptoms. There is high fever, extreme dyspnœa, lividity, and tenacious expectoration. *In the early periods the chest sounds clear*, but the signs of the most intense bronchitis, affecting the tubes of all diameters, are universally audible. The symptoms and signs continue with unabated violence, and *after some time the whole chest presents a certain degree of dulness.* The patient dies from the violence of the pulmonary inflammation, and on dissection every bronchial tube is found inflamed, and the lung equably and closely studded with miliary and granular tubercles."

I have selected this highly acute case from the pen of an

eminent pathologist and careful observer, as showing most clearly, although casually, the fact that the dulness on percussion is not found until the inflammation has existed for some time, thus establishing the chronological sequence of the tuberculosis to the disturbing action of the inflammation. In the more common forms of miliary tuberculosis, the symptoms of antecedent bronchitis are less strongly marked, but to the careful inquirer they are unmistakably evident. The cooing, or musical râle, and the moist râles can always be found on full inspiration, but the *diminution in resonance on percussion* begins to manifest itself only *after the bronchitis has existed for some time.* We have now the same sequence of signs, the chest resonant in the early part of the attack becoming dull to the stroke after the inflammation has continued for some time. Now, if any faith may be placed in physical diagnosis, we have in this sequence of signs sufficient to prove the fallacy of the doctrine, that irritation arising from the presence of tubercle produces the bronchitis.

Again, if we watch closely the progress of ordinary phthisis, we find each fresh attack of tuberculosis to be preceded by the usual signs of bronchitis. A—— D——, æt. twenty-two, came under my care at the Infirmary for Consumption. On examination, I found dulness on and under the left clavicle and on the suprascapular ridge, with feeble respiration and prolonged expiration in the same parts. She one day got a severe wetting and took cold. On examination, there was *no alteration* in the extent of the *dulness on percussion,* but auscultation revealed a new set of signs, moist and cooing notes in the apex of the opposite lung, and also in the lung first affected, but extending far below the seat of the percussion dulness. No further change of signs or symptoms occurred until three weeks had elapsed, when dulness began to be detected in the apex of the right lung, and was also found to have extended lower than before in the left lung. In fact, the inflammation had given rise to a fresh crop of tubercle below the original deposit in the left lung, and a primary crop in the apex of the right. Thus, we find the same chronological sequence of tuberculosis to inflammation in the progress as in the origin of tubercle.

This sequence of signs is so constant, that we may predict the presence of tubercle above by a lurking bronchitis below, which is absent in the corresponding portion of the opposite lung. (Stokes.)

I will now examine ordinary chronic bronchitis as a cause of tubercle:—C—— B——, when to all appearance in good health, caught cold at the beginning of the winter; cough and spitting, with more or less dyspnœa, continued to torment him all through the winter, but passed off when the warm weather set in. The

next winter the same symptoms returned, and again passed off in the summer. My notes say, "Except for the cough and dyspnœa the patient appears in good health, the digestive organs are in good condition, and there is no loss of flesh or strength. *The chest sounds well on percussion.*" On the return of the fourth summer, the patient expected the cough to subside as usual, but it continued to trouble him all through the summer. On percussing chest, *some dulness was now found* to exist in the *apex of one lung*, and after a lapse of some weeks *the dulness was found to have increased*, and to engage the *apex of the other lung also.* He now lost flesh and strength, but not rapidly. His case proceeded slowly to a fatal termination. Now, in this case, so long as it remained an ordinary chronic bronchitis, the patient maintained flesh and strength, but the advent of dulness on percussion was contemporaneous with the loss of both, and clearly fixes the time at which tuberculosis set in. I have notes of a large number of cases of this kind, which clearly exemplify the same chronological sequence of dulness on percussion to the disturbing influence of chronic bronchitic inflammation.

These cases require careful and frequent examination in order to guard against error of diagnosis. "Dilated bronchi will sometimes so closely mimic advanced phthisis, that a post-mortem inspection can alone remove our uncertainty; but the progressing dulness on percussion, the regular progress of the symptoms, and the absence of the bronchial breathing and resonance of the bronchial voice, which exist in dilated bronchial tubes," are generally sufficient to enable us to differentiate between them.

I will now give a case or two of tuberculosis from parenchymatous inflammation of the lung. Jane S——, æt. twenty, four years ago, when apparently in very good health, was seized with inflammation of the left lung, for which she was treated by an eminent physician, to whom her father was coachman. She kept her bed for six weeks, and has never since recovered her strength nor felt as she did before. About one year ago she had a sharp attack of hæmoptysis. On examination, the right lung was found to be resonant throughout, and the respiration highly puerile. The left lung was dull throughout, the dulness being most intense in the apex and diminishing downwards to the base, but there was no ægophony. In the infra-clavicular region there was well-pronounced gurgling, posteriorly and inferiorly coarse moist râles, cough very troublesome, some nummular sputa, loses flesh, sweats at night. The father of this patient is suffering from some chest affection, supposed to be phthisis. This case is a type of a large class, in which a constitutional tubercular diathesis is roused into a condition of active tuberculosis by local parenchymatous inflammation.

In the following case inflammation and debility, induced by the mode in which it was treated, appear to be the sole factors of tuberculosis :—George E——, admitted under my care at the Metropolitan Free Hospital. Tall, thin, sallow complexion, face marked with small-pox. Chest flat, left side somewhat collapsed, nails curved; mother living, and healthy; father killed by accident. Was in perfect health up to three years ago, when, being then a soldier stationed at Shorncliffe, he was "told off" with a party of men to bathe in the sea, at the end of October. Chills came on the same evening, followed by fever and pain in the left side, with dyspnœa and expectoration of red-stained glutinous sputa. He was bled largely from a vein in the arm, and cupped four times. This was followed by extreme weakness. He remained in the Military Hospital thirty-two days, and was then discharged as unfit for service. He has never since regained health and strength. About two years ago hæmoptysis came on, and it has recurred from time to time ever since. The day prior to his admission it was very copious. He expectorates half a pint of thick yellow phlegm every day, and loses flesh rapidly. The left pleura now contains no fluid, but there are well-pronounced signs of a large cavity in the superior part of the left lung. There are also signs of condensation in the apex of the right. Had this poor fellow been treated less *heroically*, he would probably have recovered from the effect of the inflammation; but the vital powers being reduced by the treatment, the plasma exuded was probably of a very low type, which, if not tubercular *ab initio*, rapidly degenerated into tubercle.

I will now relate a case in which tuberculosis was excited by an attack of pneumonia, in a subject debilitated by the action of other disease. Charles D——, a sailor, was attacked by fever on the West Coast of Africa. During convalescence, between two and three weeks before his arrival in the port of London, he caught cold from exposure to the weather. Chills and flushes, cough, and dyspnœa came on. On his admission to the Metropolitan Free Hospital, the right lung was found to be somewhat dull on percussion in its lower and posterior part, and auscultation revealed a fine crepitation. The patient had lost flesh, and was very weak. He continued in this state, without any improvement, for some weeks. There was occasional hæmoptysis. The apex of the same lung then became dull on percussion, with coarse moist sounds. The expectoration was a copious muco-pus. He became extremely attenuated, had night sweats, and frequently a sharp diarrhœa. He was removed into the country by his friends, and I lost all trace of him, although he promised to write.

One more case, and I have done. J. B., æt. thirty-five, of irregular habits, obtaining a living by "jobbing" about the

docks and river, had been out of work and badly fed, when, on going to a "job," he fell into the water and then allowed his clothes to dry on him. The next night he had shivering and flushes of heat. On admission to the Metropolitan Free Hospital, crepitation, rather fine, was audible in the posterior inferior part of the right lung. His symptoms were not severe, but remained unchanged for a *month*, and then slight dulness began to appear. The crepitation became coarser, and in three months from the commencement of his illness the signs of a cavity were obvious. The expectoration became copious and nummular. He lost flesh rapidly, and died with all the symptoms of pulmonary consumption.

We can differentiate between these cases and mere pneumonia, by the long continuance of the crepitation, by the absence of bronchial or tubular breathing, by the slight degree of dulness, and the lateness of the period at which it manifests itself. Also between them and capillary bronchitis by one lung only being engaged (a rare thing in bronchitis), and by the slow progress of the symptoms as well as by the general history of the case.

I will merely allude to the inhalation of stone-grit, &c., as a cause of tuberculosis (a subject already ably handled by Dr. Peacock), although its bearing on the question under discussion is most important, inasmuch as by it we can trace tubercle back to inflammatory irritation caused by the presence of a mechanical irritant in previously healthy subjects. In all those cases *dulness is a late symptom*, appearing only after the health has been impaired by the continued bronchitis. I have seen cases of this form of tuberculosis in granary men, who inhale the silicious dust from the tunic of the grain while shovelling it. The grinders' rot of Sheffield and Birmingham is a form of tuberculosis produced in a similar manner by inhaling particles of steel cast off in the process of dry-grinding cutlery, &c.

The above cases are sufficient to exemplify the important position which inflammation holds as an exciting cause of tuberculosis; but we must, at the same time, avoid the grave error of ignoring a predisposing cause.

Inflammation does not, I believe, in any case give rise to the formation of tubercle in a healthy subject. There must be also a condition of low health or cachexia present. This cachexia may be either inherited or acquired. In some of the cases I have related it was of the latter character. In the soldier it was probably caused by the depletion, in the sailor it arose from the fever, in the dockyard man from irregular living and insufficient food. I would argue, therefore, that neither the cachexia nor the local inflammation is capable *per se* of producing tubercle, but that tuberculosis is the result of their combined action.

It may be objected by some that the inflammation in these

cases was the consequence of irritation produced by tubercle already present, but the difficulties attending this theory are much greater than the other; for, in the first place, the physical signs of tubercle are at first wanting, and show themselves only after the inflammation has continued for some time, and, in the next place, there is the difficulty of comprehending how a few soft or semi-fluid tubercles in a yielding and easily-compressed structure like the lungs could possess such irritative qualities, or why they should, after remaining dormant for an indefinite time, suddenly assume the character of irritants capable of exciting inflammation. Viewing tubercle as a cacoplasm, it must be subject to the same general laws as all other plastic exudations. Consequently, it must be the result of a disturbing action. As well might we attribute pneumonia to the presence of the plasma which we all admit to be a product of preceding inflammation. If the presence of tubercle in the lungs were capable of causing bronchitis or pneumonia, why are those consequences not always found? Why is the caprice? Why is the effect not constant?

I shall now give a brief recapitulation of the points exemplified in the foregoing cases.

1st. That a condition of low health or cachexia, although a necessary factor in the production of tubercle, may exist for an indefinite time without giving rise to any local lesion.

2nd. That, for the production of tubercle, some additional factor, in the form of a local exciting cause, is necessary.

3rd. That inflammation, in the form which it assumes in cachectic subjects, is capable of acting this part, and is probably the only exciting cause of tuberculosis.

4th. That a close analogy exists between the cacoplastic or lowly-vitalized formation found in cachectic subjects and the more highly-vitalized products of sthenic inflammation in healthy persons, and that this analogy is especially close in the substance called tubercle.

5th. That the condition of constitution known as tubercular cachexia, from whatever cause it may arise, whether an inherited vice of constitution, or the result of depressing agents, is capable of modifying the products of inflammation so as to give them the characters of tubercle.

6th. That the varieties of tubercle depend on the acuteness or chronicity of the inflammation, the degree of the cachexia, and the nature of the structure on or in which they are formed.

7th. That the presence of tubercle in its first stage is insufficient for the production of inflammation.

8th. That the doctrine of spontaneous generation of tubercle is fraught with danger to the patient by diverting the attention from watching for and guarding against the immediate exciting

cause, and is also calculated to make our treatment one-sided by being directed solely to the constitutional malady.

9th. That, in the treatment, our remedies must be directed both against the constitutional vice and the local malady, and that both indications may be fulfilled contemporaneously.

When the subject of tubercular diathesis is seized with catarrh, bronchitis, or other pulmonary inflammation, or with pain in the clavicular or humeral region, with fever and sonorous or moist râles in the corresponding part of the lung, his state is highly critical and entails a fearful amount of responsibility on the physician who undertakes the treatment; for if the local inflammation or irritation be allowed to proceed, tubercle will form; but if, in his zeal for its removal, he adopt measures productive of permanent debility, such as leeching, cupping, purging, and very low diet, he aids powerfully the constitutional vice, and lessens, if he do not destroy, the patient's chance of recovery. If, on the other hand, he looks only to the constitutional malady, leaving the local mischief to the *vis medicatrix naturæ* and doses his patient with beef-steak and brandy, &c., a crop of tubercle is the sure result. The most successful practitioner is he who, keeping in mind the ever-present cachexia, deals with the local inflammation as the immediate cause of tubercle. The local inflammation being of a low type and slow in progress, it is easy to combine the treatment necessary for both indications. I have found excellent results from a combination of ammonia salines with tartarised antimony and morphia. When the inflammatory action is severe, this treatment should be continued for some days, aided by blisters of acetum cantharidis or other counter-stimulants over the affected parts, while the strength is supported by beef-tea and other light nutriments. The tartarised antimony appears to control inflammation without producing any permanent debility. As soon as the inflammation is somewhat subdued, a combination of perchloride of iron and nitrate of potass, given contemporaneously with the antimonial saline, is eminently useful. When the inflammation is less severe, the two modes may be combined from the very commencement. The dose of tartarised antimony should be much smaller than is usually given in sthenic inflammation. I find from ten to fifteen minims of the antimonial wine of the London Pharmacopœia every four hours to be sufficient. I know of no drug which possesses so powerful a sedative action in pulmonary inflammation as tartarised antimony. In small doses it calms irritation, while at the same time it promotes secretion. Coupled with morphia or cannabis Indica, and given with ammonia salines, it rarely fails as a remedy for low pulmonary inflammation.

The topical application of hot steam to the bronchial mucous membranes by inhalation is a valuable adjunct to the other treatment. When the inflammation is dry, not attended with secretion of mucus, the vapour of hot water alone is very useful. It should be inhaled at frequent intervals, and at as high a temperature as the patient can comfortably bear. Its effects are so soothing that few patients fail to repeat its use. When the sputum is viscid and hard to expectorate, vinegar may be added to the hot water. The French white wine vinegar, owing to the presence of a peculiar aroma, is usually most grateful to the patient. It can be got at Crosse and Blackwell's, Soho Square. When the cough is troublesome, the addition of acetum opii (Pharm. Dubl.) is useful, as are also chloroform or spirit of chloroform, Hoffman's anodyne ether, and Battley's sedative solution of opium, when inhaled conjointly with hot water vapour. When a more perfect inhaler is not at hand, the ordinary small circular stoneware teapot is a very good substitute. The vapour is inhaled through the spout, removing it for expiration and depressing the handle to keep the liquid contents from entering the spout.

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