FACTS AND OPINIONS RELATING TO TUBERCULOSIS, WITH COMMENTARIES.

By HENRY ANCELL, Surgeon.

In my last paper, I explained the sense in which the term "tuberculous" is applied to a diseased condition of the pulmonary system, the origin alike of consumption and of scrofulous disease, in the view of some pathologists, is a morbid "action," as indicated by the radical meaning of the suffix "osis"; and tubercle is the special anatomico-pathological element of the morbid condition or morbid action. The paper embraced facts relating to the frequency of tubercle in 2,161 autopsies, made at St. George's Hospital, during a period of ten years, the relative number of males and females affected in this number of cases, and other interesting particulars, drawn from the Decennium Pathologicum of Dr. Chambers. The present paper will give some account of the pathological appearances in connexion with tubercular deposits in the lungs, observed in the same series of cases.

Of all the pathological appearances accompanying tubercle in the lungs, the results of inflammatory action are undoubtedly the most frequent and the most important. Various opinions were held at one time as to the relation that subsists between the special morbid deposit and these appearances; the deposit having been attributed by many, in the infancy of pathologic, to the inflammatory action, this latter being regarded as a necessary antecedent or concomitant of that action, a doctrine which was at times disputed. Of late years, in this country at least, the question appeared to be so far settled, that both our theory and practice have proceeded upon the principle that tubercles may be deposited in any organ independently of inflammation, and that, in a majority of instances, inflammatory action is a consequence, and not a cause, of the deposit. Two papers have, however, been lately published by Dr. Sieveking, in which the author considers that he has established by microscopic observation the inflammatory origin of tubercle. One of these papers, under the title of The Seat of Tuberele, appeared in the British and Foreign Review for April last, and the other, with the title of Illustrations of Tubercle, in the Association Journal of May 27. After stating the more important statistical facts, I propose to make a few observations suggested by them, and by a perusal of Dr. Sieveking's papers; advertising also to the views of Dr. J. H. Bennet, of Edinburgh, on the subject of inflammation in connexion with tubercles, contained in a valuable work, On the Pathology and Treatment of Pulmonary Tuberculosis, just published.

The anatomico-pathological appearances in the lungs, attributable to inflammatory action, which Dr. Chambers' papers contain, are those usually referred to pleuritis and pneumonia. The great frequency of pleuritic adhesions, and of the signs of inflammatory action in the pleura, is confirmed by the statistics of the Decennium Pathologicum; nevertheless, there is a great discrepancy between the results obtained by Dr. Chambers and those previously recorded by Louis. The eminent French pathologist found adhesions in every instance except one, in 112 subjects, where death had been produced by phthisis. The pleura is not mentioned in the records before us, in 93 of the 514 cases where tuberculosis was found in the lungs. Dr. Chambers conjectures that in a part of those it must have been overlooked; but we trust that such a circumstance could have happened in a very few cases only, for, if so obvious a pathological lesion were overlooked in from one-fifth to one-sixth of the cases, as indicated by the numbers, the value of the statistics is very much diminished.

Old adhesions were found without evidence of recent inflammatory action:

In 50.2 per cent. cases of hard tubercle.

\[
\begin{align*}
55.3 & \quad \text{vomicose.} \\
53.8 & \quad \text{tubercle in all stages.} \\
23.2 & \quad \text{diseases in general without tubercle.}
\end{align*}
\]

The evidences of recent pleurisy, consisting of purulent or soft fibrinous matter, were found:

In 21. per cent. cases of hard tubercle.

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\begin{align*}
23.2 & \quad \text{vomicose.} \\
22.5 & \quad \text{tubercle in all stages.} \\
20. & \quad \text{diseases in general without tubercle.}
\end{align*}
\]

Combining together the evidences of inflammatory action in the pleurs, old and recent, there were found:

In 71.3 per cent. cases of hard tubercle.

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\begin{align*}
78.2 & \quad \text{vomicose.} \\
76.2 & \quad \text{tubercle in all stages.} \\
42.6 & \quad \text{diseases in general without tubercle.}
\end{align*}
\]

In 27 additional cases, fluid occurred in the pleural sac, without other evidences of inflammatory action.

These facts are in accordance with the observations of Louis, as to the absolute frequency of pleuritic adhesion and the effects of inflammation in tuberculous, and their relative frequency in this as compared with other diseases. But, as before remarked, the frequency was much less than in Louis' cases. In explanation of this circumstance we must bear in mind that M. Louis' were all fatal cases of tuberculosis pulmonalis, whereas these include fatal cases from a great variety of causes other than pulmonary affections, and many of which the disease of the lungs was a secondary affection; and we can readily conceive that in such cases congestion, irritation, and inflammation in the pleurs would be much less likely to occur than when tubercles are primarily developed in the lungs, the pulmonary form of the disease pursuing its usual insidious and progressive course to a fatal result.

Dr. Chambers concludes, from the evidences of pleurisy being nearly as frequent in association with hard tubercle as with vomicose, that pleuritis is a concomitant rather of the early than of the later period of pulmonary tuberculosis. He concludes also, that in the last and most fatal stage, tubercle is less liable to produce inflammatory action in the pleurs than many other diseases, and that severe cases are found old adhesions appear to operate as a check to pleurisy and also to the effusion of blood; this latter being an exceptional occurrence.

Condensation or hepatisation of the lungs was found in 45.4 per cent. of the males and 44.2 per cent. of the females affected with tubercle of the lung. In the 171 cases of crude tubercle it occurred in 29 per cent.; and in 343 cases of vomicose and tuberel cases it occurred in 45 per cent. The difference in the frequency increased with the advanced progress of the disease. At different periods of life, the ratios in which it occurred were as follows:

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\begin{align*}
\text{RATIO PER CENT.} & \quad \text{Vomicose.} \\
\text{Under 16 years of age} & \quad 40. & \quad 39.2 \\
15 to 30 & \quad 46.7 & \quad 47.5 \\
30 to 45 & \quad 38.5 & \quad 51.6 \\
45 to 60 & \quad 44.4 & \quad 9. \\
Above 60 & \quad 40. & \quad \text{—}
\end{align*}
\]

Age unknown

38.4

44.3

Total of all ages

43.4

44.2

Dr. Chambers is of opinion that the condensation or hepatisation, hitherto most frequently attributed to inflammation, is, in a great majority of cases, of a non-inflammatory nature; and, with a view to prove this point, an elaborate table is given of the frequency with which it occurred in males and females, at different periods of life, both with and without the evidences of pleurisy. The result is, that, in 11.7 per cent. of males and 16.6 per cent. of females, it coincided with the effects of pleurisy; and, in 30.4 per cent. of males and 25.6 per cent. of females, it occurred alone. This is taken as evidence that the frequency of inflammation in the chest. In the advanced cases also, where evidence of inflammation of the pleurs was least frequent, hepatisation of the lung was most frequent.

These circumstances lead Dr. Chambers to the conclusion, that the condensation is often asthenic and non-inflammatory. He considers that it depends upon a state of
atrophy skin to the condition of lungs that have never re-
spired, in the new born infant, or which have become de-
generate from chronic bronchitis impeding the entrance of
air. In 11.7 per cent. of the males, however, and 16.6 per-
cent. of the females; its probable inflammatory origin is
asserted. Referring to the coexistence of pleurisy. Dr. C. ad-
mits also that inflammatory condensation may be met with
after death without the results of active inflammation of
the pleura; that inflammation of the pleura may have been
subdued, leaving only adhesions of the sac; that the forma-
tion of adhesions in the early stage checks and prevents
pleurisy; and in the latter stages; that pneumonia may occur
without the total absence of pleurisy. So that I fear
the cases and the observations of this author will have very
little effect in settling this interesting and very important
point.

Dr. Sieveking’s papers, to which I now beg the reader’s
attention, relate to the morbid appearances of tubercular
lungs observed microscopically, and especially to the evi-
dences of inflammatory action which they present. This
author unhesitatingly affirms, that, “on close examination
of incipient tubercular deposit, we may always note that
there is congestion of the tissues surrounding it; and
again, that “the deposit is never effected without those
local inflammatory changes of the vascular system charac-
terised by inflammatory action.” Thus, the tendency of
these communications is retrogressive. If the statements
they contain be true, and admit of universal application,
as assumed, and even boldly asserted, by the author, we
must revert to the Broussanian or a more ancient doctrine.
One of these papers was read before a learned society pre-
vious to its publication; and I am totally ignorant of the
manner in which the subject was treated by the assem-
bled members; but of this I am certain, that the profession
will require cumulative or very conclusive evidence before
the principles sought to be established can be accepted.
The remark is scarcely necessary, that it is one of the
principles in relation to the theory of tuberculosis which
must, to a greater or less extent, modify our practice in
every form of the disease.

Dr. Sieveking founds his opinions on his own micro-
scopical examinations. Admitting a pre-existing “tuber-
cular cachexia” or disease of the blood, and paying due
compliment to quote my work on this part of the sub-
ject, he states, that tubercle takes its origin from blood in
this diseased state; but it is never deposited in the more
solid structures without congestion and inflammatory action.
In illustration, the author states that, in connexion with
the deposit of tubercle in the pia mater of the Sylvian
fissure, we see an increased redness, in which a few
more prominent than usual in the pulmonary par-
enchyma, we may, especially by the use of the microscope,
discover the engorgement of the capillaries, investing the
air vesicles in which the tubercle is being secreted; in
the mucous membrane of the intestines, we see “the exquisite
arborescent arrangement of the congested vessels, tending
from the mesenteric attachment to the point where we ob-
serve the deposit shining through the mucous surface from
the submucous tissue in which it is collected.” These are
the instances given, the amount of vascular action accom-
panying the elimination differing in different individuals.

There cannot be a momentary doubt that the whole of
these appearances have presented themselves again and
again to Dr. Sieveking. So they have to myself, and to
every microscopist who has looked for them; and there can
be little doubt that they occur in a very large number of
cases, from a very early period of the disease. But this
by no means establishes the coincident occurrence of tu-
bercle and inflammation as a constant fact, nor the doctrine
of a necessary relation between the occurrence of inflam-
mation and the deposit of tubercle.

In the first place, as to facts. I will abstain from
quoting observations the direct converse of those described
by Dr. Sieveking, to be found in the works of the most
eminent pathologists of former years, by which both the
theory and practice of Broussais and his followers were
overturned; but, from more recent authors, I will give
illustrations which, if to be depended upon, completely in-
habit the generalisation sought to be established by the
author. Turning to Dr. Hennis Grenn’s paper on Tubercle
in the Brain in Children, which comprises a most careful
analysis of 30 cases, and was published in 1842, long sub-
sequent to the agitation and apparent question of the
necessity of tuberculous origin of tubercle, we find the follow-

ging statement:—“In many cases, we can

not discover the slightest change in the surrounding
nervous substance, or in the neighbouring membranes.

Looking, in the next place, to the lungs, Mr. Rainey,1
whose microscopic observations are too apt to be ignored
over, thus describes the state of the vessels when tubercle
is deposited: “The perfectly natural appearance of the
vessels close to a tubercle, and even of the cells containing
a small quantity of tubercular matter, not sufficient to
have impeded their circulation in the capillaries during
life, when compared with the tortuous and unevenly

dilated constant of vessels going to air cells filled with

lumen, in consequence of inflammation, are pathological con-
sideations in favour of the non-inflammatory nature of
the deposit.” In reference to the serous membranes, Carwell2
and various authors state that, where minute tubercle is
scattered over the pleura or peritoneum, the membrane in
most cases remains transparent up to the tubercular point,
and only becomes reddened or opaque when the tubercle
begins to act as an irritant, or passes into some new phase
of development. In Lebert’s description of the micro-
scopical appearances presented by the mucous membrane
lining the intestines, it is stated, that where tubercle has
been deposited, the membrane is found at first to be per-
fectly sound, then slightly raised by the aggregation of
tubercular matter; and it is not until subsequently that it
becomes injected and softened.

These recorded observations might be received as con-
clusive both as to the matter of fact, and the inference that
might be drawn from such a fact, were they not liable to
be met with the arguments that microscopists may be de-
ceived, and that their views were often erroneous in the ear-
erlier periods of microscopic observation; that our attention
is more completely fixed upon the point; our instruments im-
proved; that we know better the extent of power to employ;
and that our observations are more correct. Dr. Sieveking
is also a gentleman of undoubted ability and reputation,
and founds his opinions on his own examinations; it is,
therefore, due to him to examine a little further into the
details of the subject.

In the next place, then, as to the doctrine sought to be
established, “that the deposit of tubercle is never effected
without those local and molecular changes in the vascular
system characterised by inflammatory action”; it is but
just to state, that Dr. Sieveking does not employ the terms
“cause” and “effect”; nor does he state, in so many words,
that the deposit of tubercle is necessarily preceded by in-
flammatory action; but I think the enunciation just given, and
the whole context, tend to establish a necessary relation
of cause and effect. The remarks which suggest themselves
under this head apply also to the former; for, in truth, the
question turns in a great measure upon the question of the
fact. The proof given by Dr. Sieveking of the constant co-
existence of inflammation and tubercle is the microscopic
appearances presented by tubercular lungs, consisting in an
obvious enlargement and congestion of the vessels lining the
air-cells, and the formation of exudation matter. Passing
over the descriptions of the vascular appearances in question,
previously recorded by some of the most eminent of the old
microscopists, and the interpretations put upon them, which
are sufficiently familiar to the profession,—in the author’s
instances of this increased vascularity we are not informed
as to the part of the lung upon which his observations were
made, whether on a portion taken from the surface, or from
the interior. We find, however, that the cases from which
the diseased structure was obtained had been cases of active

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1 Medico-Chirurgical Transactions, vol. xxv. p. 192.
pneumonic disease, by which the death of the patient was wholly or in part produced. Every pathologist knows that the air vesicles of the lungs, in their condition of normal, are lined by a dense network of capillary vessels; and that this vascularity is greater in the interior of the lung than at its extreme parts; so that what might present the appearance of morbid enlargement and congestion towards the surface would be deemed normal in the more deeply seated parts. But the greatest importance is to be attributed to this appearance of increased vascularity, even admitting its constant existence, is, that the cases selected are not suitable ones. The investigation ought to be made in cases of tubercle deposited in the lung where the patient's death has been wholly caused by some accident, or some disease not in the slightest degree implicating the lung, and where none of the ulcerative changes which accompany tubercle have taken place. In such cases, if those selected by the author, the existence of increased vascularity cannot assist us in determining the main question at issue, viz., whether the tubercle produces, or is produced by, the inflammatory action.

The occurrence of pus and corpuscles, which is in fact the fatal point in Dr. Sieveking's observations, and the one upon which, I presume, he places his greatest reliance, tends to prove the converse of that for which the author contends. It is to be especially noted, that these exudative corpuscles are described as forming a ring around, or lining the air vesicles, and in another place, as coating the tubercular membrane of the lung. Why shall we assume, that the deposit of pus, or tubercle, or both, in the sheath-like basement membrane of the vesicle, as a well clipped bullet fills its mould. So that there is a clear demarcation between tubercular matter on the one hand, and exudative matter on the other, and the latter is always on the outside of the former. Is it this a positive proof that the tubercle produces the inflammatory action, in consequence of which exudative corpuscles are produced, and that the inflammation takes place subsequent to the deposition of tubercle? If tubercular and exudative corpuscles were both the effects of those local and molecular changes in the vascular system characterised by inflammatory action, we should expect to find the two products uniformly commingled, or at all events, shading off gradually from circumference to centre, as ulcerative changes take place in the matter first deposited; but, in Dr. Sieveking's own description of tubercle, we find it to be a substance differing totally from exudative matter; and also that exudative corpuscles are found only rarely and incidentally mixed with tubercle, while the tubercular corpuscle is totally distinct from the exudative corpuscle when taken together, the occurrence of this product of inflammation in connection with tubercle, as described, I am inclined to believe, that the author has furnished the best proof hitherto given of the non-inflammatory origin of tubercle, and of the intercurrent and consecutive nature of the inflammation.

Independently of all that relates to direct observation, let us for a moment admit the truth of the doctrine of congestion, irritation, increased vascular action, inflammation, or a process allied to inflammation, and taking promiscuously a few of the anomalies that present themselves, look them fairly in the face. Not to advert to the observations of the eminent pathologists, Carrell, Andral, and Cruveilhier, who led them to reject the doctrine in question, but limiting ourselves even to facts contained in the present series of papers, how could it happen in the cases in St. George's hospital, included in the statistics above recited, that the evidences of condensation of the lung should present themselves at the utmost in 29 per cent., only of crude tubercle, and in 49.2 per cent., only of inflammatory tubercle; that inflammation predominate above all, in a majority of cases the inflammatory action which necessarily precedes, or constantly attends tubercular deposit, at some very early period of the deposit ceases to produce that cumulative effect which becomes obvious and palpable in the state of condensation. How does it happen that pleuritis should be so frequent an occurrence in tuberculous subjects, even more frequent than pneumonia, and again still more frequent in the earliest stages of the disease, and the deposit of tubercle in the pleura so rare an occurrence, as shown also in the Decennium pathologicum? The serous membranes are by no means insusceptible of tubercular deposit. How again are we to explain the facts contained in the second of this series of papers, from which it appears that no constant relation subsists between the frequency of inflammatory affections of the lungs and of phthisial, or that which might happen that the lungs, the mucous membrane of the alimentary canal, and the membranes of the brain, the parts which are most liable to inflammatory action, are least liable to the deposit of tubercle? These and many other anomalies would have to be explained, before we could rest satisfied, upon microscopic evidence, that tubercle is essentially an inflammatory deposit.

Lest I should produce in the slightest degree an erroneous impression as to Dr. Sieveking's views, it is right to mention that in one place he states that the appearance of increased vascularity "almost always" accompanies the deposit; and in another place, he designates the process upon which the deposit depends as one "allied to inflammation"; although the general tenor of those parts of both communications which relate to the subject, is to prove that the deposit is never effected without inflammatory action. These expressions seem to imply that the author's mind wavers as to the tenor of his doctrine.

Dr. J. R. Bennett has given a much more rational view than some pathologists seem to entertain of the relation of inflammatory exudation to tubercle, in his chapter on "The Nature of Tubercle" (Lib. cit. p. 23.) He regards tubercle as an exudation of the liquor sanguinis, presenting marked differences from inflammatory exudation on the one hand, and cancerous exudation on the other; and he points out very truly, that inflammatory and tubercular exudation more frequently alternate than accompany each other. I must defer what I have to assert respecting inflammatory action, and inflammatory products in a tubercular state of the blood, but I think all must agree with this author, that if by inflammation we understand pain, heat, redness, and swelling, or the presence of pus and phlegm, this is not the case; but if we consider inflammation to be an exudation of the blood plasma, then tubercle as well as lymph and cancer are inflammatory products. Dr. Sieveking evidently does not adopt the latter view, since he applies the term to those appearances of ordinary inflammation, which all observers have found so frequently accompanying tubercle, and respecting which the question is, whether the tubercle is the consequence or the cause of the action which produces them; and in describing the exudative corpuscles surrounding tubercle, he evidently means the exudation of ordinary inflammatory action, in contradistinction to the tubercle corpuscles.

And it respects the deposit of tubercle in the various tissues, though the whole of the observations more recently recorded, instead of shaking, tend to establish still more firmly than ever the received doctrine, that it may and frequently does occur without infection; that inflammation may and frequently does occur in tuberculous subjects, and even in organs containing tubercle, without causing a deposit of tubercle; that inflammation and tubercle may alternate, and tubercle frequently alternate with each other; that the former is more frequently a consequence than a cause of the latter; and that no necessary relation of cause and effect can be established between them.

Reverting to the morbid appearances in connexion with tubercle in the lungs, I have referred in the last chapter to Dr. George's hospital, in addition to the more frequent direct results of pleuritis and pneumonia, the Decennium em-
SPONTANEOUS AMPUTATION IN UTERO OF THE RIGHT FOREARM.

By GEORGE GREENWOOD, Esq.

The monographs by Dr. Montgomery of Dublin, and Dr. Simpson of Edinburgh, on Spontaneous Amputation of the Limbs of the Fetus, contain a collection of cases more or less similar to that which I am now about to record. These valuable papers appeared in the first, second, and tenth volumes of The Dublin Journal of Medicine. In the Medical Times of December 1855, Mr. Macalpine reports a case of a child born without arms, which evidently belongs to the same class. The nature of the disjunctive agency in such cases is well explained by the writers to whom I have referred.

Case. On the 13th of last month, I was called upon to attend A. F., aged 23, in her first confinement. Labour was natural. The child, a female, was born without the right forearm; the stump having the appearance of a very neat amputation. The humerus can be distinctly felt at its extremity, diverging into two equal and well rounded condyles. Wishing to ascertain if any, or what influences, maternal solicitude or mental impression, could be traced in connexion with such a defect. It was thus inferred that, in the earlier months of utero gestation, the mother had received a severe shock by the sudden intelligence of her husband having had his arm nearly cut off by machinery, at the mill where he is accustomed to work. The report, however, was exaggerated, as he had merely sustained a slight injury to one of the fingers. The grandfather of my patient, residing in the same house, lost the case of his right hand, from an injury to the wrist, terminating in exfoliation of some of the carpal bones, and permanent immobility of the fingers.

Having stated the facts of this case, and some collateral circumstances in which an interest may be felt, I leave my readers to draw their own conclusions regarding a curious subject in intra-uterine pathology.

Ossett, near Wakefield, January 2nd, 1854.

BIBLIOGRAPHICAL NOTICES.


These Lectures were originally delivered at the Brompton Hospital for Consumption, and published in a cotemporary Journal. They are now collected into a volume, and contain such modifications as the author's further reflections have suggested. The lectures are thirteen in number, in which the wide subject of consumption is treated discursively, the particular points in each lecture having been most part determined by the facts that presented themselves in the wards of the hospital. The author's views throughout are illustrated by cases, for the most part treated under his own eye, and there are two well executed plates, in one of which the peculiar condition of the gums indicative of the consumption disease is represented. An index is very properly appended, containing a reference to the more prominent topics of discussion.

The introduction contains a simplified nomenclature of the best marked auscultatary signs, with a definition of each, a description of the morbid condition it is intended to indicate, and the synonyms or corresponding terms in general use. Dr. Thompson objects greatly to the term "subcrepitant" applied to the term "crepitus", and considers that no medical expression has tended to produce more danger in practice by leading to injudicious depletion. The term "smaller bubbling rhonchus" is substituted; and instead of designating the stethoscopic sign of pneumonia as a "crepitant rhonchus", he calls it simply "crepitation".

The lectures do not profess to comprise the whole sub-