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A CONTRIBUTION TO THE NATURAL HISTORY OF PULMONARY CONSUMPTION,

By HORACE DOBELL, M.D., &c., &c.,

Consulting Physician, late Senior Physician, to the Royal Hospital for Diseases of the Chest, &c., &c.

Showing the relation between Loss of Weight, Cough, Blood-spitting, and Lung Disease, in One Hundred Male Cases of Hæmoptysis.

(No case was taken unless both present and heaviest weight were known from actual weighing, and unless either the first or subsequent Hæmoptysis had been more than Streaks of Blood in the Sputa.)

TABLE I.

EACH HORIZONTAL LINE IS A COMPLETE CASE, FOR THE REMARKS ON WHICH SEE SEPARATE NOTES.

DESCRIPTION. — EXTENT OF LUNG DISEASE: Upper Lobe — LAST LUNG DISEASE: Upper Lobe — GENERAL PECULIARITIES — WEIGHT — FIRST COUGH — PRESENT COUGH — FIRST HÆMOPTYSIS — SUBSEQUENT HÆMOPTYSIS AND PARTICULARS OF LAST OCCURRENCE. — FAMILY HISTORY.

1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24	25	26	27	28	29	30	31	32	33	34	35	36	37	38	39	40	41	42	43	44	45	46	47	48	49	50	51	52	53	54	55	56	57	58	59	60	61	62	63	64	65	66	67	68	69	70	71	72	73	74	75
NUMBER OF CASE IN BOOK.	Mar.	Female.	Age.	Consolidation.	Softening.	Excavation.	Adhesions.	Consolidation.	Excavation.	Moderate.	Severe.	Extreme.	Present Weight.	Heaviest Weight.	Present W. Greater than Heaviest by	Present W. Less than Heaviest by	Heaviest W. Greater than Present by	Date of Heaviest W.	Cause or Age of Heaviest W. (Symptom)	How lost W. and how Gained. Oil.	Blood.	Natural.	How Cough first began.	Date.	Symptoms after Commencement.	Present Cough.	Paroxysms.	Expectoration.	Duration of Cough.	Date of Present Cough.	Cause assigned.	Present Weight less than Heaviest by	Present W. Greater than Lightest by	Lightest W. since Heaviest.	Age at Lightest W.	Duration of Present Cough.	Symptoms after Commencement.	Date of First Hæmoptysis.	Time elapsed before First Hæmoptysis.	Heaviest W. at First Hæmoptysis.	Present W. at First Hæmoptysis.	Lightest W. at First Hæmoptysis.	Loss of W. at First Hæmoptysis.	Duration of First Hæmoptysis.	Cause assigned.	Quantity lost at First Hæmoptysis.	Symptoms after First Hæmoptysis.	Recurrence of Hæmoptysis.	Interval between First and Second.	Age at Second Hæmoptysis.	Present W. at Second Hæmoptysis.	Lightest W. since Second Hæmoptysis.	Loss of W. at Second Hæmoptysis.	Duration of Second Hæmoptysis.	Cause assigned.	Quantity lost at Second Hæmoptysis.	Symptoms after Second Hæmoptysis.	Recurrence of Hæmoptysis.	Interval between Second and Third.	Age at Third Hæmoptysis.	Present W. at Third Hæmoptysis.	Lightest W. since Third Hæmoptysis.	Loss of W. at Third Hæmoptysis.	Duration of Third Hæmoptysis.	Cause assigned.	Quantity lost at Third Hæmoptysis.	Symptoms after Third Hæmoptysis.	Recurrence of Hæmoptysis.	Interval between Third and Fourth.	Age at Fourth Hæmoptysis.	Present W. at Fourth Hæmoptysis.	Lightest W. since Fourth Hæmoptysis.	Loss of W. at Fourth Hæmoptysis.	Duration of Fourth Hæmoptysis.	Cause assigned.	Quantity lost at Fourth Hæmoptysis.	Symptoms after Fourth Hæmoptysis.	Recurrence of Hæmoptysis.	Interval between Fourth and Fifth.	Age at Fifth Hæmoptysis.	Present W. at Fifth Hæmoptysis.	Lightest W. since Fifth Hæmoptysis.	Loss of W. at Fifth Hæmoptysis.	Duration of Fifth Hæmoptysis.	Cause assigned.	Quantity lost at Fifth Hæmoptysis.	Symptoms after Fifth Hæmoptysis.	Recurrence of Hæmoptysis.	Interval between Fifth and Sixth.	Age at Sixth Hæmoptysis.	Present W. at Sixth Hæmoptysis.	Lightest W. since Sixth Hæmoptysis.	Loss of W. at Sixth Hæmoptysis.	Duration of Sixth Hæmoptysis.	Cause assigned.	Quantity lost at Sixth Hæmoptysis.	Symptoms after Sixth Hæmoptysis.	Consumptive Tendency.	Bronchitis.	Asthma.	Pleurisy.	Pneumonia.	Malarial Fever.	Paternal Consumption.	Maternal Consumption.	NUMBER OF CASE.
Total, for Patients.	100	100	3,322										Total, in pounds. 12,191	Total, in pounds. 14,262					Total, in days. 93,057		Oil ... 313 Emulsion 31 Ointment 1 182 Blood 1 Iron Syr. 32		Total, in days. 3,613						Total, in days. 91,165										Total, in days. 3,613									

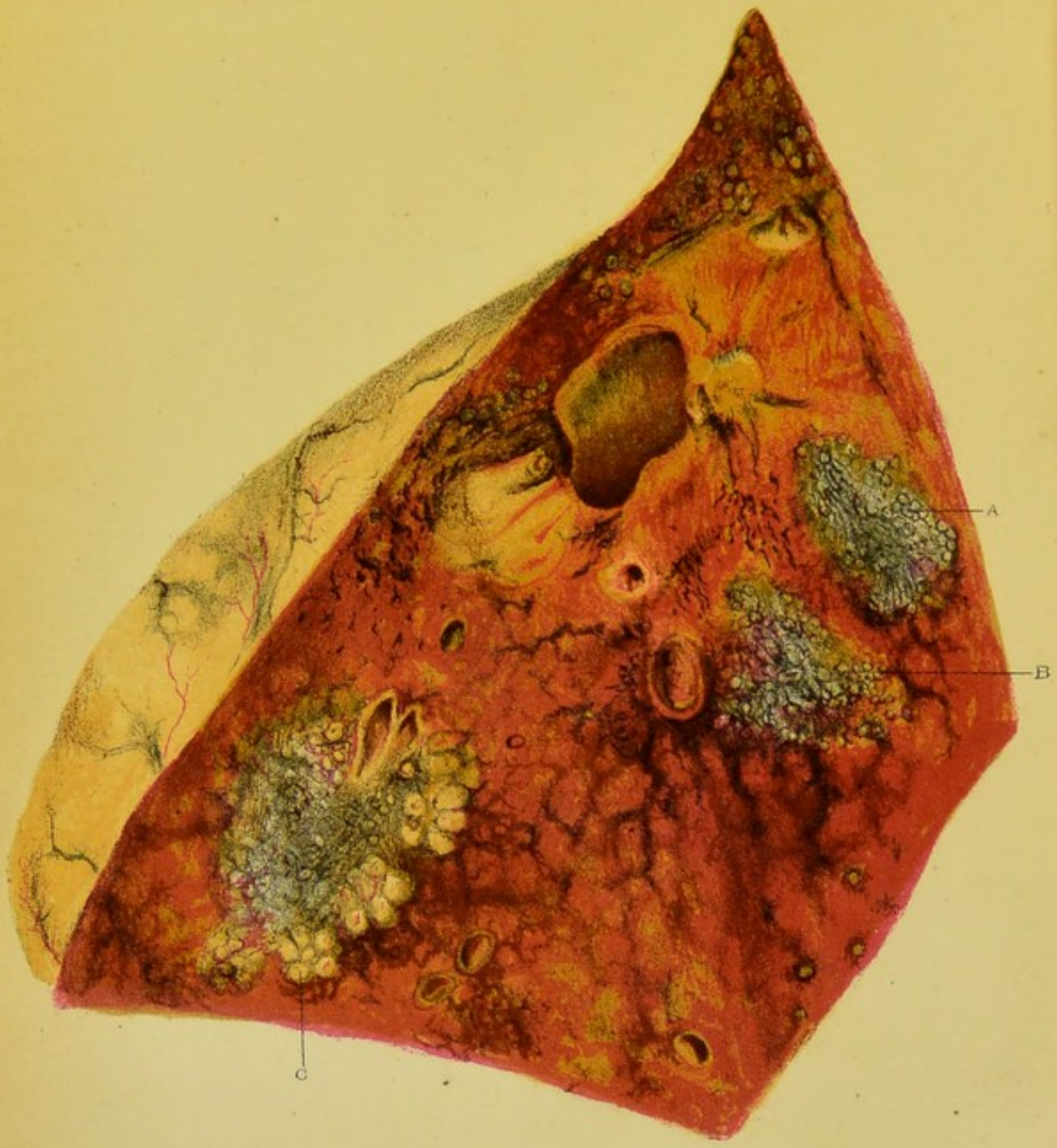
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15th Jan. 1896



PRESENTED BY

Dr de Havilland Hall

882



From a drawing by Dr Moxon of the lung of a man who died of Gangrene of the foot from exposure. A.B.C. are large clusters of tubercles with old pigmented centres and recent tubercles around. A and B. have the tubercles grey, C. has them yellow and softening. The yellow represent the grey in a changed state.

"Referred to at pages 68. 84. 85. 87. 171 & 172."

J. Godart Lith.

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With the author's kind regards

ON

LOSS OF WEIGHT, BLOOD-SPITTING AND LUNG DISEASE.

BY

HORACE DOBELL, M.D.,
ETC., ETC.,
CONSULTING PHYSICIAN TO THE ROYAL HOSPITAL FOR DISEASES OF THE CHEST,
LATE SENIOR PHYSICIAN TO THE HOSPITAL, ETC., ETC.

"It is recognised as an axiom among scientific speculators that the best tests of a scientific hypothesis are found in the simplicity of its conception and the universality of its application."

Times, April 15, 1874.



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IN REMEMBRANCE OF MANY YEARS OF VALUABLE CO-OPERATION,

AND AS A SLIGHT TOKEN

OF

GOOD FELLOWSHIP, PERSONAL REGARD AND THANKS,

THIS BOOK IS DEDICATED

TO

THE PRINCIPAL FOREIGN COADJUTORS

WHO HAVE ASSISTED THE AUTHOR IN HIS ANNUAL REPORTS

"ON DISEASES OF THE CHEST,"

AND

"ON THE PROGRESS OF PRACTICAL AND SCIENTIFIC MEDICINE

IN DIFFERENT PARTS OF THE WORLD."

TO

Professor Wm. Anderson (Yedo).
Dr. W. Anderson (Newfoundland).
Professor Beneke (Marburg).
Dr. Henry Bennet (Mentone).
Professor Biermer (Breslau).
Dr. G. Lindsay Bonnar (Natal).
Dr. Bowditch (Boston).
Dr. Brandt (Oporto).
Dr. Brinton (Philadelphia).
Dr. F. W. Campbell (Montreal).
Dr. A. S. Carroll (New York).
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PREFACE.

A PERSON who suffers from Loss of Weight, Blood-Spitting, and Lung Disease is generally thought to be "in a Consumption." But as either of these conditions may exist alone, may be due to a variety of causes, may be independent of the others, and may require special treatment, it is very important that each should be made the subject of a separate and careful consideration. Hence they have long been among the principal headings under which I have accumulated my observations. But it is obvious that, in writing a work like the present, much space would have been wasted and many needless repetitions involved, by keeping the consideration of Lung Disease separate from that of the Loss of Weight and Blood-Spitting, so often associated with it. It will be found, therefore, that my remarks on diseases of the lungs have been distributed throughout such parts of the work as seemed most appropriately to connect them with the other subjects under discussion. (See p. 267.)

My two books on Consumption—"On Tuberculosis," 1866; and "On the True First Stage," 1867, being just out of print, I have embodied in the present work such parts of each as I wished to reproduce, instead of re-editing them in a separate form. (See pp. 162-3, and 194-201.)

I have mentioned (at p. 11) that in order to obtain a fair basis for statistics, I have limited the Clinical records to an *average succession* of about eight hundred cases of Hæmoptysis, as they occurred at the Royal Hospital for Diseases of the Chest; cases of exceptional interest, therefore, are only given as they happened

to appear "in succession" with others. Thus, for example, only a few cases are given in which Lung Disease was complicated with Fistula, or had been treated with Setons, or in which Hæmoptysis appeared to be the Cause of Lung Disease, or in which there was suspicion that Tubercle had been communicated by Contagion; and no cases happen to be included in which Lung Disease was treated with Lung-splints. But under each of these headings I have long collected clinical records, and I hope, at some future time, to be able to add to the present work observations upon these and other groups of special cases.

With regard to the many commentaries upon the observations, theories and opinions of others which I have been obliged to introduce at various parts of this work, I particularly wish to emphasise the statement (see p. 166), "that these are not made in any spirit of dogmatism, but simply to lead the mind of the reader into my own course of ideas at apposite places for comparison and criticism, and thus to enable him to judge how far my own researches and hypotheses assimilate or otherwise with the researches and hypotheses of others."

In conclusion, I wish to express my best thanks to Dr. Moxon, for allowing me to copy his beautiful drawing; and to Professor E. Frankland, Professor S. Haughton, and Professor C. Meymott Tidy, for giving me permission to quote (at p. 148), from their unpublished communications.

NOTE.

In Table 1 it will be seen that Columns 26 and 34 are left blank. These columns are introduced into the printed table to make it complete as an example for future use. They were not provided in the manuscript table, otherwise Cases 17, 42, 58, 93 would have been entered in Column 26. (See Table 6, p. 57.)

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PART I.

HÆMOPTYSIS AND PULMONARY CONSUMPTION.

CLINICAL RESEARCH,

ETC., ETC.

ON LOSS OF WEIGHT, BLOOD-SPITTING AND LUNG DISEASE.

PART I.

The Interest, Difficulty and Importance of the Subject.—The proper Arenas for Science and for Practice.—Interdependence of Loss of Weight, Blood Spitting and Lung Disease.—Necessity for Caution and Consideration, Promptness and Energy.—The true position of Hæmoptysis in the Natural History of Consumption.—Morbid Anatomy subsidiary to Clinical Observation in discovering the Nature, Cause and Treatment of Diseases.—Statement of Problems to be solved, and of proper Methods of Clinical Research in Consumption.—Constitutional Decline. — Loss of Weight. — Cough.—Hæmoptysis.—100 Cases of Hæmoptysis, selected from a succession of 800 Cases.—Rigid Method of Selection, Tabulation, and Analysis.—Abridged Summary of Table 1 (Frontispiece).—Remarks on 100 Cases. — Arrangement into Clinical Groups. — Explanations of Tabulated Facts, and Deductions from them. — References to 17 Subsidiary Tables.—Principal Heads under which Hæmoptysis should be classified as a Symptom, and as a Cause of Disease.—Its place among other Causes.—Key to further Analysis of Cases in Special Groups.—Special Groups of Cases, with Commentaries upon them. Tables 2 to 18.

After sixteen years of constant work at the Royal Hospital for Diseases of the Chest, and thirty years of private practice—during which it has been my custom to add the experience of others to my own, and to watch and criticise with anxious care all that has been done by my contemporaries, at home and abroad—it is but natural and inevitable that I should have formed many definite opinions on subjects of such stirring interest and vast practical importance as those which I have placed at the head of this publication.

So wide and so deep are the questions in Etiology, Physiology, Pathology, Histology, Chemistry, and Therapeutics, which each item of that heading raises in the mind, so absorbing is the interest with which speculation and investigation are led on from step to step, that it is fortunate the physician is so often summoned from such entrancing studies by the imperative calls of his medical brethren to come and help them in their difficulties, and to show, by practical results, what good has come of all his work.

I shall endeavour to divest the subject of theory and speculation, except so far as these are necessary to add point and meaning to such

practical remarks as I think will be most useful to my brother practitioners in medicine, when face to face with disease in a living patient, and whose object it is, above everything else in the world, to keep that patient alive, and, if possible, to restore him to health.

The science of our profession should be done in the hospital, the laboratory, and the study; and when we come into the arena of private practice we should keep all our wits about us, so that our learning may point out short cuts to safe conclusions by processes of ready reckoning.

"Confound your figures; I hate figures!" muttered a famous Chancellor of the Exchequer, thrusting back a proffered bundle of calculations to his astonished secretary, as he entered the House of Commons to make his most successful "Budget speech."

We cannot wonder if, rightly or wrongly, the practitioner is sometimes tempted to mutter, "Confound your learning; I hate learning," when a physician tries to air his pathology, while a patient is dying for want of a remedy. I well remember, some years ago, meeting a young physician in consultation on a case where the patient was *in extremis*, and when I called the doctor quickly into an adjoining room to suggest a last chance of succour, he commenced a learned and pathological discussion. "If we talk much longer, the poor man will be dead," said I, pulling him up as soon as I decently could. As we went back the patient expired.

The interdependence of loss of weight, blood-spitting, and lung disease is so intricate that, although either may occur without the others, neither can be properly treated without considering the rest. I select *Blood-spitting* as the first subject of my remarks, 1st, because it is at once so prominent, so urgent, and so alarming a symptom that it is the one above all others which leads patients and their friends to implore prompt and energetic help from their doctors; for help, too, in its treatment as a symptom, quite independently of its meaning. I select it, 2ndly, because, as medical practitioners, we cannot consider it or treat it without reference to its relation both to loss of weight and to lung disease, and because, in practice, immediately we have met the first anxious demand to stay the symptom, we are brought face to face with the no less anxious demand, that we shall explain its meaning and foretell its issue.

When called to a patient who is spitting blood, the medical man will speedily clear up such common sense and obvious questions as whether the bleeding arises from the anterior or posterior nares, the mouth, or pharynx. But it must be borne in mind that, although these points may be easily decided if the bleeding still continues, it requires a good deal of circumspection and careful investigation to avoid mistakes if, the bleeding having ceased, we have to depend upon the accounts and descriptions of the patient and his friends.

It is quite necessary, too, in passing, to caution the young practitioner against the possibility of intentional deception on the part of his patient, which, under some circumstances, may be of great importance to his own reputation if not to the life of his patient. I shall never forget the puzzle in which I was myself put, when a young beginner, by being called to see a fashionable "lady," whose extravagance had brought her into the old Whitecross Street Prison. She was found early one morning, with a good deal of blood on her pocket-handkerchief, and daubed about the sheets and the neck and front of her night-dress, about which she professed to be much alarmed, stating that she had been roused from her sleep by spitting blood. Nothing in her previous history and no present disease could be discovered to account for the bleeding, and it was supposed to have been brought on by the excitement of the few previous days, and by the anticipation of having on that day to appear in court. It was, therefore, thought safer to delay her appearance for a few days, lest the bleeding should recur. When, however, the appointed day for her appearance in court drew near, the same scene was re-enacted; and this time I was pressed to certify that her life was being endangered by the confinement of the prison, and that it was unsafe for her to appear in court. But, again being unable to discover any rational explanation of the hæmoptysis, my suspicion of foul play was aroused, and I carried home some of the blood, and examined it under the microscope, when it was found that the corpuscles were those of a bird. It was afterwards discovered that she had managed to get the heads of some fowls from the prison kitchen, with the blood of which she had bedaubed her clothes. I need not say that the "lady" did not get a certificate to the effect she desired.

If the hæmorrhage is profuse when the medical man arrives, he will probably have to form a rapid diagnosis on very scanty evidence, and to order speedy and active remedies for its arrest before he can safely proceed to a satisfactory investigation of the case. I have a vivid and painful recollection of the consternation of a young practitioner who called me to a case in which a profuse and sudden hæmoptysis had occurred about two hours before, but had nearly ceased when we arrived. The lightest possible tap under one clavicle was sufficient to suggest a thin-walled superficial cavity, and the consequent advisability of avoiding further meddling with the chest, lest hæmorrhage should be re-excited; but my young friend, in his zealous desire to make a more exact diagnosis, and failing to see the need for my extreme caution, gave a sharp thump over the affected region before I could stop him, when his first blow was answered by a perfect flood of blood rushing from the mouth and nostrils, by which the patient was nearly suffocated.

But the very fact that in hæmoptysis we may be called upon to act

under such pressing emergencies and to draw important deductions from such hastily gathered premises, makes it the more incumbent upon us to keep our minds fully stored with all the possible bearings of the case, so that we may make those "short cuts to safe conclusions by a sort of ready reckoning" to which I have just referred; and we must never forget that, having to the best of our power met the immediate emergency, we must *at once reconsider the position*, and bring to bear upon the case all the light that the most careful consideration can be made to shed. No excuse can then be found for hurried conclusions, or for merely palliative treatment.

The Tables and analysis of cases which we are now about to discuss will at least prove that in these remarks I am not preaching what I have failed to practise. They represent an amount of labour, first and last, which those will best appreciate who have attempted to carry out a similar investigation. Although only one hundred cases are selected for tabulation, it will be seen that these represent about 800 cases carefully investigated and recorded, for the purposes of a special enquiry into *the true position of hæmoptysis in the natural history of pulmonary consumption* which formed the substance of my paper read before the Royal Medical and Chirurgical Society, April 28th, 1874. Although the Society did me the honour to print my paper in Vol. 57 of its Transactions, want of space and the costliness of printing elaborate tables, necessitated the omission of the details of most of the cases, and of the eighteen tables in which the leading facts were analysed.

In now publishing the cases and tables *in extenso*, I hope it will be found that they are worth the trouble and expense I have devoted to them.

Highly important and essential as morbid anatomy is in its proper place—subsidiary to clinical observation—I am quite satisfied myself that it will never be the chief means by which we arrive at a knowledge of the causes, prevention, or cure of pulmonary consumption.

When the disease which has been the cause of death is one destructive of the affected structure, morbid anatomy comes all too late in the story to reveal the tale of the beginning. As well might we expect to discover the causes of a fire by examining the premises in which all traces of the cause have been consumed.

The most valuable information procurable from morbid anatomy with reference to *the cause and onset of disease*, is where death has occurred from accident or from some other disease than the one just beginning, and even then all the value of the investigation turns upon the fidelity of the clinical record relating to the disease which was *not* the cause of death. This conjunction of circumstances can only be rare and accidental, and, at best, it is accompanied by many damaging complications as a source of sound etiological conclusions. Intelligent, accurate, systematic clinical observation is, then, the source to which

we must principally look for the discovery of the nature, causes, sequence, prevention and cure of such affections as destructive lung disease.

It is therefore of the utmost importance to decide—(1.) What are the heads under which such clinical observations are most essential. (2.) How they can be recorded in the simplest and easiest way in practice. (3.) How this record can be so arranged that the observations of each individual may be readily summed up and put into statistical form. (4.) How the system of record can be so arranged that the observations of any number of independent observers can be added together on exactly equal terms, and thus contribute the materials for immensely numerous statistics, and for vast and safe generalizations. With regard to these statistics and generalizations, it must be especially remembered that the greatest interest may, and always does, attach to the exceptions to the general rule. Each of these exceptional cases requires special investigation, and they should be accumulated in sufficient numbers to allow of extensive comparative analysis, so that, if possible, the reasons for their exceptional character may be brought under a special set of general rules. Such an accumulation of exceptional cases can only be eliminated by a most extensive system of comparable observation.

All these points were deeply considered by me in arranging the headings under which the following cases were recorded in Table 1, and in the analysis and tabulation of the exceptions. (See Subsidiary Tables.)

The opinion held by some, that hæmoptysis causes pulmonary consumption, is only part of a wider opinion to the same effect, viz., that its causes are not general but local—that they begin in the diseased organ itself, not in a constitutional state. (See Part II.) No question in practical medicine can be more vital than this. It strikes at the root of all ideas of prevention as well as of treatment, and the only way to clear it up is to push a searching investigation into the early clinical history of destructive diseases of the lungs:—

1. To ascertain in what proportion and at what intervals the earliest signs or symptoms of local disease are preceded by signs or symptoms of constitutional disease.

2. To ascertain the typical character of the signs and symptoms of such local and constitutional diseases.

3. To ascertain whether the earliest stages of local disease can exist without indicative signs and symptoms. It is here that the aid of morbid anatomy may be called in with signal value in the class of accidental cases before referred to.

4. To accumulate any cases which appear to be exceptions to general rules. To scrutinise these with the greatest care, and to endeavour in every possible way to explain their exceptional character.

If it is found that in a vast majority of cases of pulmonary consump-

tion (destructive lung disease with constitutional decline) local disease is preceded by constitutional disease by an unequivocal interval; and yet that in a minority of cases local disease precedes constitutional disease by an unequivocal interval; the conclusion is almost inevitable that there are at least two modes in which pulmonary consumption may commence. It then becomes a point of the greatest interest and importance to ascertain whether these two classes of cases are to be considered as absolutely distinct throughout; or whether there is a period in their causative history at which they meet on common ground. That is to say, is there a stage at which the constitutional disease sets up the local disease? And is this first stage of the local disease, in these cases, the same as the first stage of the local disease in the cases not preceded by constitutional disease? And again, starting from this point, are the subsequent symptoms of constitutional decline in the two sets of cases due alike to the effects of the progress of the local disease? Or are they, in the first case, due to the progress of the original constitutional disease plus the effects of the local disease? And in the second case, are they due to the effects of the local disease alone? Again, does this local disease set up a state of constitutional disease of *the same nature*, and leading to the same local effects as that which precedes the local disease in the first set of cases? Or is a *new constitutional disease of special character* set up by the local disease in the second set of cases? And, if so, is the local disease of the first set of cases also competent to set up *a special constitutional* disease the same as that of the second set? Finally, are the symptoms of constitutional decline, *which follow the establishment of local disease in each set of cases*, devoid of all special character, and only such as may accompany the progress of any local disease proceeding to a fatal termination? (See Part IV. for answers to these questions.)

Much light may be thrown upon these apparently complicated questions by the clinical record of the following simple facts:—

1. The date of the earliest symptoms of local disease.
2. The condition of health previous to the earliest symptoms of local disease.
3. The rate of decline after the onset of symptoms of local disease in the two sets of cases.
4. The rate of decline in the two sets of cases after the local disease has arrived, in each set, at a comparable stage sufficiently advanced to have allowed time for the local disease to have set up a second special constitutional disease.

It is necessary, then, to fix upon some typical and prominent signs or symptoms of local disease and some similarly unmistakable signs or symptoms of constitutional disease for the record.

In the case of pulmonary consumption, *Loss of weight* is the best typical sign, we yet know, of *Constitutional Decline*; and *Cough* is the best

typical sign, we yet know, of *local disease*, confirmed or otherwise by physical chest signs.

With regard to the *onset of cough* it is necessary to register both the *first cough* and the *present cough*, *i.e.*, the cough present when the case first comes under observation. When some reasonable connection can be traced between the *first cough* and the commencement of subsequent symptoms of either the constitutional or the local illness, I consider the question of the onset of "first cough" of great importance in the clinical history, as indicating the earliest palpable symptoms of something wrong in the respiratory tract; *e.g.*, if a patient had first hæmoptysis twelve months ago, and had "*first cough*" thirteen months ago, it carries back the chest symptoms a month beyond the hæmoptysis. Yet he may have lost that cough with or after the hæmoptysis, and "*Present cough*" may have come on since. If, therefore, present cough were noted, and not first cough also, the most significant clinical fact would be lost from the history, and the whole case falsified.

In *Hæmoptysis*, where it occurs, we have another typical and prominent sign of local disease, confirmed or otherwise by physical chest signs. (For further remarks on this symptom, see Index.)

It is by the accurate and systematic record of the sequence of these signs and symptoms, and of their attendant circumstances, that we shall be able most powerfully to assist in reading the answers to the before-stated questions.

Let us, then, proceed to examine the contribution which I have made to this subject in my paper to the "Royal Medical and Chirurgical Society."

The primary object of this investigation was to assist, by clinical observation, in settling the important question of the *True Position of Hæmoptysis in the natural history of Pulmonary Consumption.*

With this intention the cases discussed were recorded upon a plan of enquiry so designed that it should, as far as possible, avoid the chance of prejudice from preconceived opinions and impressions, and include all the facts essential to the investigation in *a tabular form.*

With so ample a field for observation as is afforded by the Royal Hospital for Diseases of the Chest, it would have been easy to accumulate any number of cases, if I could have entrusted the work to an assistant. But feeling that the whole value of the investigation depended upon my being able to *guarantee* that the histories of the cases had been sifted and verified in the most rigorous manner, there was no alternative but to do all the work myself.

I regret that this necessity and the pressure of other professional work obliged me to limit my analysis to 100 cases of hæmoptysis, a number too small, statistically, to form more than a contribution to the subject. I trust, however, that if the plan which I have adopted should be thought worthy of imitation, other observers, with more

time at their disposal, will follow it up, and thus accumulate a sufficiently large mass of comparable facts arranged on a uniform plan to form a wider basis for reliable conclusions.

Having decided upon the plan for the tabulated records, it was next of the highest importance that the cases should be representative of the ordinary average varieties of hæmoptysis, absolutely free from any *selection* dictated by special views, and yet that *they should be selected* in the sense of each containing the elements essential to the leading points of the enquiry. To obtain these ends the following precautions were rigorously carried out:—

All cases occurring in my practice at the Royal Hospital were asked if they had ever expectorated any blood. If the answer was "Yes," the case was not recorded unless it was certain that the blood had been seen in some other form than "*streaks in the phlegm.*" (See Part II. and Index.) After this point had been made clear, the case was carefully recorded, and *afterwards* sifted in the following manner before being selected for statistical analysis.

1. All cases were rejected unless the heaviest weight before the occurrence of hæmoptysis could be stated from *actual weighing*, and reasonable evidence given as to whether this had been the average weight up to the time of first hæmoptysis.

2. All cases were rejected who could not stand a searching cross-examination as to the time at which the *first loss of weight*, if any, had begun.

It is in this enquiry that the greatest patience and caution are required, for the first answers given by patients to direct questions on the subject of loss of flesh and loss of weight are *almost invariably wrong* and of the most misleading description. Seeing the great difficulty there is in getting at the truth in this matter, one cannot help the conviction that many delusive conclusions may have been arrived at, based upon statements elicited without a sufficient knowledge of the necessity for caution on this point. (See "Loss of Weight," Part IV.)

It will at once be recognised how very large a number of cases of hæmoptysis must have been rejected on these two counts.

3. I soon found that it was necessary to reject all females from the tabulated analysis for the following reasons:—(*a.*) Their weights before admission could not be relied upon; the majority had never been weighed till they came to the hospital, and, even if they had been weighed, the differences of clothing were found to present more unavoidable sources of fallacy than in men. (*b.*) Pregnancy, childbearing, and lactation, were constant elements of fallacy as to weight. (*c.*) Hæmoptysis was found to be complicated with climacteric and other derangements of the menstrual functions, to an extent seriously damaging its clinical meaning with relation to consumption. (*d.*) In pushing a close inquiry into the history of a case, it was much more difficult to obtain a con-

nected and reliable account of facts from the *hospital class* of women than from men of the same class. (It is evident that for these reasons cases of hæmoptysis in females have an interest and importance special to themselves, but that they must be analysed as a completely separate class from males.) The rejection of all females from my tables was a serious loss, diminishing the number of otherwise eligible cases by about one-half.

4. All cases were rejected in which there was a reasonable suspicion of cardiac complications.

5. All cases were rejected who had not been able to give a fairly succinct account of the *onset of cough*.

6. All cases were rejected who had not been able to give an approximative estimate of the quantity and character of the expectorated blood in the first and subsequent hæmoptysis.

7. And finally, after the inquiry had been completed, all cases were rejected if it was found, on comparing the principal statements, that they were inconsistent with one another.

It will be seen that the elements of these rigorous rejections are such that they do not give the cases a *selected character*, in the sense of invalidating their claim to represent an *unprejudiced average* of cases of hæmoptysis occurring at a public hospital. They may, therefore, be considered to form a fairer basis for statistics than if no selection had been made, having the great advantage that all incomplete and unreliable reports are excluded.

It is evident that the labour involved in the analysis of one hundred cases was immensely enhanced by the above plan, but I think it will be felt that the value of the facts obtained has been proportionately increased. If we allow for each case accepted for tabulation one rejection on the 3rd count (sex) : two on the 2nd count (history of first loss of weight) ; two on the 1st count (history of actual heaviest weight); one between counts 5, 6, and 7 ; and one for count 4 (cardiac complications), and these numbers are below the truth, the one hundred cases will be representative of an average succession of not less than eight hundred recorded cases in which *hæmoptysis occurred in other forms than streaks in the sputa.*

On examining Table 1 (Frontispiece) it will be seen that each line in the horizontal direction represents a complete case, which may be read off by reading the statements made in the columns, and by reading the headings of each column in which is placed an affirmative figure 1. In the manuscript table a final column was added, in which a brief connected note on each case was given under the head of "Remarks;" but in printing the table this was found to encumber it so much that these remarks have been printed separately. (See p. 4.)

In order to simplify the references to Table 1, I have here pre-

pared a concise summary of some of the most important facts which it contains, to which I have added references to special sets of cases, and to the seventeen supplementary tables, in which I have analysed and compared some of the principal groups of facts.

Many other tables may yet be constructed out of Table 1, to elucidate the meaning of other groups of facts therein contained; but these I do not propose to introduce in this work.

ABRIDGED SUMMARY OF TABLE 1.

Columns 1 *to* 20.

The table consists of an analysis of 100 cases of hæmoptysis in males, in all of which blood had been expectorated *in some other form than streaks in the sputa*, and some disease existed in the upper lobes of the lungs at the time the note of the case was taken.

The average age was 33·37 years.

The condition of the lungs is classified in Table 2.

The general symptoms were moderate in 43 per cent., severe in 41 per cent., extreme in 11 per cent.

The average present weight of each patient was 122·97 lbs.

The average heaviest weight ever attained by each patient was 142·62 lbs.

The average present weight was, therefore, less than average heaviest weight by 19·65 lbs.

In not one case was the present weight greater than the heaviest previous weight.

The present weight was less than the former weight in every case.

The heaviest weight had been entirely regained in 2 per cent., but in each of these cases present weight was less than previous heaviest weight. (See these cases, Cases 43 and 97, with commentaries.)

The average time elapsed since loss of weight began was 950·17 days.

A cause for the loss of weight was assigned by the patients in 70 per cent.

Columns 21 *to* 37.

Previous to admission of patient, loss of weight had been treated with cod oil in 31 per cent., with pancreatic emulsion in 1 per cent., with oil and emulsion in 15 per cent., with a voyage in 1 per cent.

No treatment had been adopted in 52 per cent.

The average time elapsed since first cough began was 1464·4 days.

A cause of first cough was assigned in 50 per cent.

First cough began *before first loss of weight* in 69 per cent.
after first loss of weight in 31 per cent.
before first hæmoptysis in 87 per cent.
after first hæmoptysis in 2 per cent. (These two cases G. H., 26, R. W., 62, are further analysed hereafter).
at the same time as first hæmoptysis, *i.e.*, was accompanied by it, in 12 per cent. (These twelve cases are further analysed in Table 3.)

The average time elapsed since present cough (cough present at time of admission) began, was 915·45 days.

The average time elapsed between the beginning of first cough and the beginning of present cough was 548·95 days.

A cause for present cough was assigned in 50 per cent.

Present cough began *before* loss of weight in 63 per cent.
after loss of weight in 37 per cent.
before first hæmoptysis in 77 per cent.
after first hæmoptysis in 10 per cent.
at the same time as first hæmoptysis in 13 per cent.

Columns 38 *to* 52.

The average time elapsed since the occurrence of *first* hæmoptysis in each case was 633·05 days. Comparing this with Column 19, which shows that the average time elapsed since first loss of weight began was 950·17 days, it is seen that *first loss of weight began*, on an average, 317·12 days *before first hæmoptysis.*

But this general average is corrected in Tables 4, 5, 6.

Table 4 shows that in eight cases loss of weight began on an average 853 days *after first hæmoptysis.*

Table 5 shows that in eighty-two cases loss of weight began on an average 469·78 days *before first hæmoptysis.*

Table 6 shows that in ten cases first loss of weight and first hæmoptysis were coetaneous.

The blood expectorated in *first hæmoptysis* was *mixed in the sputa* (flesh-coloured) in 12 per cent. *Black or dark blood* in 1 per cent. (In this case quantity was over ℥x., followed by smaller quantities on several occasions—attributed to lifting.) *Florid blood* in 76 per cent. *In small clots* in 15 per cent. *In streaks* in 9 per cent. (In each of these cases blood had been subsequently expectorated in some other form.)

The *quantity* of blood expectorated in first hæmoptysis was—

Under ℥ss.	in twenty-four hours,	in	11	per cent.
Over ℥ss.	„	„	34	„
Over ℥ss.	„	„	27	„
Over ℥x.	„	„	26	„

A cause for first hæmoptysis was assigned in 89 per cent.

First hæmoptysis occurred *before the period of heaviest weight* in 2 per cent. (See analysis of these cases, T. P., 7, and A. G., 17, hereafter.)

After the period of heaviest weight in 87 per cent.

First hæmoptysis occurred *before first loss of weight* in 8 per cent. (Analysed in Table 4.)
after first loss of weight in 82 per cent. (Analysed in Table 5.)
at the same time as first loss of weight in 10 per cent. (Analysed in Table 6.)

Columns 53 *to* 67.

Hæmoptysis had recurred in 78 per cent.

First loss of weight occurred at the time of *Recurrence* of hæmoptysis in 2 per cent. (See these two cases—J. P., 7, and H. F., 104, hereafter).

For further details of recurrent hæmoptysis see columns 54 to 66.

Columns 68 *and* 75.

The family history shows that—

Father only was consumptive in 9 per cent.
Mother only was consumptive in 3 per cent.
Both father and mother were consumptive in 5 per cent.
Either one or both parents were consumptive in 17 per cent.
One brother (and no sister) was consumptive in 14 per cent.
One sister (and no brother) was consumptive in 8 per cent.
Three sisters (and no brother) were consumptive in 1 per cent.
One brother and one sister were consumptive in 4 per cent.
Two brothers and one sister were consumptive in 1 per cent.
Either brothers and sisters or brothers or sisters were consumptive in 26 per cent.
One paternal uncle or aunt (no maternal) consumptive in 3 per cent.
One maternal uncle or aunt (no paternal) consumptive in 0 per cent.
One paternal and one maternal uncle or aunt consumptive in 1 per cent.
Either paternal or maternal or both paternal and maternal uncles and aunts were consumptive in 4 per cent.
In no case more than one uncle or one aunt on the same side consumptive.
One paternal (and no maternal) first cousin consumptive in 1 per cent. (In no case more than one.)
One maternal (and no paternal) first cousin consumptive in 1 per cent. (In no case more than one.)
Both paternal and maternal first cousins consumptive, 0 per cent. (In not one case.)

Only one relative was consumptive in 19 per cent.
Only two relatives were consumptive in 7 per cent.
Only three ,, ,, 7 ,,
Only four ,, ,, 1 ,,

In only one case were more than four relations consumptive. This one case was Case 5, in which mother, one brother, three sisters, and one first cousin (paternal) were consumptive.

It is seen, therefore, that in the 100 cases, there were 65 consumptive relatives, or, including the patients themselves, 165 consumptive individuals.

REMARKS ON CASES IN TABLE 1 (Frontispiece).

(The tendency of all patients is to date loss of weight from hæmoptysis. They take fright, and notice loss of weight. This is a constant source of fallacy.)

Case 1.—A very large man, and although weighing much more before than after his cough began, he was uncertain whether he had not lost weight when he weighed 13 stone 11½ lb. three months before hæmoptysis, as he was losing when he was weighed.

Case 3.—Case of old catarrh dating from infancy, now disintegrating lung disease.

Case 4.—Only one hæmoptysis 4 months ago, profuse expectoration.

Case 5.—Spat blood several times after first note; catarrhal case with consolidation

of right upper, back and front; hæmoptysis first in streaks, afterwards fleshy sanguinolent sputa; three sisters, one brother, mother, and paternal first cousin consumptive.

Case 6.—Father bronchitic; loss of weight and first hæmoptysis said to be coetaneous. But he had cough summer and winter 4 years, and had not weighed for a long while till hæmoptysis occurred.

Case 7.—Loss of weight said to commence with second hæmoptysis 4 months ago; no hæmoptysis between 4 years and 4 months; under treatment with oil, emulsion, &c., the heaviest weight known was regained within 1 pound.

Case 8.—Very badly off, short diet. Losing flesh over 13 months; tried oil and could not take it; cough 12 months; about a month after cough expectorated 2 ounces of congealed blood mixed with phlegm; none since (very tall and thin).

Case 9.—Weight maintained at 8 stone by emulsion for 12 weeks, then lost. Never weighed till 8 years ago, and then had lost some flesh; weighed 10 stone 4 lb.; never so heavy since; often had gnawing pains in chest; first hæmoptysis slight, 6 years ago; second hæmoptysis ℥ ss. a-day 5 years ago; third hæmoptysis ℥ j. a-day 3 years ago, and again this week.

Case 10.—Very chronic case, lost and gained flesh 10 years ago 5 years ago and 4 years ago under me, got well; 2 years ago lost flesh and had hæmoptysis, but regained. For 12 months has lost flesh again, but under oil and emulsion has gained up to 9 stone 5 lb. in 12 weeks.

Case 11.—First hæmoptysis 1 week after first cold and cough; normal weight 10 stone just before cold; lost 2 lbs. more during 6 weeks, in spite of treatment.

Case 13.—Severe catarrhal congestion now, cough, summer and winter 2 years.

Case 14.—Gained 9 lb. in 12 weeks' treatment; legs were swollen on admission.

Case 15.—Lost profuse hæmoptysis 18 months ago, but frequent bloody sputa since; states that usual weight, 10 stone 6lb., was kept till 2 years ago; 5 healthy brothers and sisters, 1 consumptive. Although stated by patient that loss of flesh was due to hæmoptysis, there is no evidence of the fact, as 365 days elapsed between first hæmoptysis and first loss of weight.

Case 16.—Growing; weighed 9 stone 5 lb. 6 months ago; only 8 stone 9 lb. on admission; gained 6 lb. in 12 weeks' treatment.

Case 17.—Had profuse hæmoptysis 9 months ago; weighed at hospital same as now; no oil taken since; all weight lost since 6 years; under treatment gained 4 lb.; no moist sounds. He had not weighed for 2 years before first hæmoptysis, but declares he had not lost flesh till hæmorrhage occurred; the cough may have begun a little before hæmoptysis.

Case 18.—Sister died of rapid phthisis 2 months; patient continued to lose flesh during treatment; never had free bleeding, but frothy sanguinous sputa.

Case 19.—Five years ago had cough and loss of flesh; regained some flesh, but not original weight; first hæmoptysis 6 months ago lasted 1 week; second hæmoptysis 2 weeks ago, slight; was much thinner at time of hæmoptysis than he had been 6 months before; died after 6 weeks' treatment.

Case 20.—Patient at hospital 16 months ago; had lost 5 lb., although growing; took oil and emulsion 30 weeks, gained 4 lb.; since then has grown 3 inches and lost 2 lb. He lost cough under treatment; soon got bad again; had spat blood 18 months ago after loss of flesh.

Case 21.—Coughed and spat 1 oz. of blood 2 years ago; none since; cough off and on; never quite well since; weighed, as usual, 9 stone 10 lb. 6 months ago, has lost quickly since; under treatment 9 weeks, lost 3 lb.; cavity cleared out; no advance occurred; seton put in right upper; health much improved.

Case 22.—Formerly very intemperate, not lately; sputa flesh-coloured, now and then, 1 year.

Case 23.—Fistula cut, but not cured, 9 years ago; 4 years ago treated again (St. Mark's); lately stopped of itself. Took oil off and on 9 years. 6 years ago first hæmoptysis, 4 years ago second hæmoptysis, cough and spitting all the time. Weighed 13 st. at 19 years, 12 st. when hæmoptysis occurred, after that 11 st. 10 lb., and later 11 st.; began to waste rapidly 12 months ago. Under treatment 12 weeks, improved in health and strength, but lost 17 lb.

Case 24.—Wife died of consumption after 3 or 4 years' illness. He broke up during this time. Since that, son died of phthisis, and father got worse nursing him. 12 months ago gained 6 lb. in 12 weeks, on oil and emulsion, reaching 8 st. 6 lb., the heaviest weight before illness; he mended again, under oil and emulsion, after son's death.

Case 25.—Improved greatly under treatment, and maintained weight, 8 st. 6 lb. Oil produced sickness, emulsion agreed.

Case 26.—Took oil and emulsion 12 weeks, and maintained weight. No history of cough till 1 month after hæmoptysis. Hæmoptysis (not accounted for) lasted 1 week, and did not return. He was sure he had lost weight before hæmoptysis.

Case 27.—He had pleurisy in October, 8 months ago; spat blood in November, and cough began; in November weighed 7 st. 7 lb., very light weight, but he thought he never weighed much more; he had, however, in all probability, lost some weight during illness with pleurisy. Hæmoptysis returned twice. Scars of excavated glands above both clavicles. Bruit de pot fêlé near left of sternum under clavicle, dull above both scapulæ (Query disease of bronchial glands?). After 12 weeks oil and emulsion weighed 7 st. 10 lb.

Case 28.—Cold and cough in April, got better; May, lifting a press (to which he was accustomed) gave sudden pulls, ½-pint blood rushed up, and 3 days after, another ½-pint, and a little now and then since. Extensive catarrhal signs plus right, and loss of resonance above. Cough very bad; felt blood come from right side. In six weeks' treatment gained to 8 st. 1 lb. on oil and emulsion.

Case 29.—Fingers clubbed, nails incurved. Had pleurisy at 17; at 19 weighed 10 st. 8 lb. Ill 4 years and 2 months. Severe hæmoptysis 4 years ago, 1½ pints; repeated returns for 12 months, not now for 2 years. Loss of flesh began with cough, but was more rapid after loss of blood. At Brompton, in-doors, 1 year ago. With oil and rest got to 10 st. 4 lb.

Case 30.—Patient here four times, "always took emulsion, always got well." Full weight never regained. Hæmoptysis 4 years ago, 2 years, 2 months, 2 weeks ago. Deep softening signs right apex; respiration feeble left.

Case 32.—Cough and loss of weight 7 years; heaviest weight just about time cough began. (Pleurisy 4 years ago *treated here*, it was at lowest part of both sides.) After six weeks patient was too weak to attend. Cough brought up food and medicine till he sank.

Case 33.—Left police 1868, on account of difficulty of breathing on foggy nights; cough and expectoration began 1869 winter; July, 1870, overworked, spat up a lump of chalky deposit. He weighed then 12 st. 6 lb.; never weighed more. Eleven days afterwards broke blood-vessel; spat "half-gallon" fluid blood, and fell down fainting; continued to spit blood one month; none since. Treated here and took oil, and has taken it for two years. Has lost weight since July, 1870. Patient here again April, 1871. Took emulsion, and gained weight (not former weight). Dulness of left upper extending over mesial line of manubrium, as though the disease were in the glands. Disease appears to have been old and cured (probably bronchial glands diseased in childhood), re-excited by overwork and exposure. Hæmoptysis from dislodgment of cretaceous deposit and the extension of disease. He had diarrhœa June, 1872, after 1st note, and after a seton put in by me over cavity kept open 12 weeks, and then healed, he had lost weight, viz. 7 lbs. (weight 10 st.); but was otherwise much better, and started on a sea voyage.

Case 34.—At time of hæmoptysis no loss of flesh had occurred since heaviest weight; loss of weight began with disintegration of lung, following or coincident with hæmoptysis. Seton put by me over diseased part.

Case 35.—Patient here four years ago with cold in right lung. Had already lost weight; took oil and emulsion, and partly regained weight. Two years ago first hæmoptysis, free for a week, no more till now. Always subject to cough with colds.

Case 36.—Patient improved under oil, emulsion, and arsenic, and gained 2 lb.

Case 37.—Subject all life to winter cough; had pleurisy six years ago; one year after sputa streaked with blood; none since till two weeks ago. Suffered from post-nasal-catarrh and cough, and the streaks of blood probably were from back of nose. Health kept up till six months ago, when weight gradually failed, and cough became worse and breath short. On admission, patient had congestion of right upper lobe, relieved by leeches, also severe post-nasal-catarrh and large disintegration signs in left upper, which became cavernous. He afterwards improved and went to Eastbourne, where he had hæmoptysis ʒ v., and signs of disease in right upper.

Case 38.—Has a fistula still running, date of commencement not known, supposed to be over two years.

Case 39.—He thought his cough, of three years' duration, did no damage till he got severe cold two years ago, after which he lost weight, and one year ago first brought up blood. On admission, catarrhal signs marked throughout; food came up with cough on first admission. Had never taken oil or emulsion; after taking them six weeks had gained 5 lb., and chest sounds were dryer and food kept down.

Case 40.—A printer, intemperate. First hæmoptysis "one quart" in three days. Could not touch oil.

Case 41.—Although taking ʒ ij. oil daily, was losing flesh rapidly; took emulsion, and aften ten weeks went back to work, weighing 3 lb. more than six weeks before.

Case 42.—Certain he was in usual health 18 months ago, when he overlifted, and broke blood-vessel; never well since. Right upper half dull and deficient expansion, back and front creaking crepitation up to apex; pleuritic pains; no friction. Blisters and oil, etc., produced great improvement, and in six weeks increase of 4 lb.

Case 43.—Had debility three or four years, cough two years, but kept average weight of 9 st. 3 lb. up to 12 months ago, then spat streaks of blood, and went to Australia; on voyage gained weight, and on arrival weighed 9 st. 11 lbs., 8 lbs. over average weight, but soon lost, and eight months after weighed 15 lbs. less. Had several spittings of blood in the eight months. Right chest was depressed, and had old cavity. Under oil and emulsion much improved, and gained up to 9 st., only 3 lbs. less than average weight.

Case 44.—Mother and father died of chronic chest complaint, probably consumption. Had had cough summer and winter 14 years. Heaviest weight 15 years ago, 11 st.; 8 years ago found he weighed 10 st. 2 lb.

Case 45.—Gained 3 lb. in 6 weeks; treatment, oil and emulsion.

Case 46.—Only 1 hæmoptysis "3 or 4 pints," 1 week ago, lasted 3 days. Gained 4 lb. in 6 weeks; treatment, oil and emulsion. Larynx was affected, and left upper yielded Bruit de pot fêlé.

Case 47.—The heaviest weight 11 st. 9 lb., 7 months ago, was reached under oil and emulsion, as treatment for cough. Before that he had lost weight, and weighed 10 st. 10 lb. when he began treatment. He improved again under treatment, but did not reach heaviest weight.

Case 48.—He had been sleeping 3 months with a son dying in consumption. Had suffered much grief. Had rheumatic fever 3 years ago, pleurisy lower left many years ago, and has a fistula in ano discharging. Took emulsion and oil 10 months off and on, and left upper cleared into cavity. Weight increased 7 lb.

Case 49.—Stone-mason. Weight 12 st. in 1856, 17 years ago; average weight since then, 11 st. 4 lb., taken as most reliable heaviest weight; kept that up to 3

years ago. General naso-pulmonary catarrh, especially marked in trachea. Disintegration left upper. Hæmoptysis described as "blood and corruption."

Case 50.—Mother died of pleurisy and dropsy. Father asthmatic. 1 brother throat consumption. When weighed 17 months ago, found he had lost 1 stone. Had then been hoarse about 7 months. Since then has taken lots of oil, and weight has fluctuated a few pounds up and down, but gradually lost.

Case 51.—February, weight 11 st. 9 lb. March, 11 st. 7 lb. May, weight 11 st. 4 lb. Previous March, 12 st. 9 lb. Loss of 16 lb. from March to March. Subject to coughs 3 years. Special cough and distress of breathing 5 months, accompanied by hæmoptysis; at first frothy blood 3 days, afterwards streaks, and finally over a pint in 2 days, attributed to fright.

Case 52.—Only weighed first time when cough began 3 months ago; does not know previous weight. Voice had long been hoarse before cough.

Case 53.—Twenty-five years ago weighed 9 st. 10 lb. Had cough as long as he could recollect, and 9 years ago spat a pint of blood in 48 hours. Average weight since then 8 st. 13 lb. Did not notice special loss of weight till 1 year ago. Since then loss of 8 lb. Has spat bloody sputa ever since. Hæmoptysis till 6 months ago. Left upper lobe dull, with squeezing cough-sounds at sternal end (Query diseased bronchial glands?) After 4 weeks emulsion, gained 3 lb.

Case 56.—Had been most anxiously treated by Dr. Lockhart Clark, who sent him to hospital, getting worse and worse. Under emulsion he gradually mended, and after change of air, got well. Lung remaining dull, but no moist sounds. Chronic lung disintegration after pneumonia.

Case 57.—A plumber. Very weak. Compression of bronchi right side, by enlarged bronchial glands?

Case 58.—It is doubtful whether he had not lost weight before hæmoptysis. He weighed 8 st. 6 lb. just before it occurred, but had not weighed before, and thought he had not lost.

Case 59.—Extensive excavation left, softening right. Father intemperate, consumptive; mother cancer. After 16 weeks oil and emulsion, weight 9 st. 9 lb.

Case 60.—Although he had cough 197 days before hæmoptysis, had not weighed till just before, might therefore have lost weight before hæmoptysis, but he does not think so. Lost rapidly after it; first hæmoptysis profuse 5 days, more or less almost daily since. Took oil at first.

Case 61.—He was so weak as hardly to be able to get to hospital, thinks his chief loss of weight was before hæmoptysis; attributes loss of weight to marriage, and hæmoptysis to working in gas. Could not take oil, but on emulsion gained 1 lb. in 6 weeks. Sounds dried and he improved.

Case 62.—Never weighed more than at time of hæmoptysis, and does not think he lost weight till a month afterwards. Had no cough till 3 months after hæmoptysis.

Case 63.—Eight months ago he had lost weight as at present, weighed 8 st. 4 lb., but took cod oil for 1 month and gained 2 lb. Since then he has lost the 2 lb. His first wife died 8 years ago in consumption; 7 years ago he married the widow of a man who died in consumption. She is now dying of consumption, and he attributes his disease to nursing her.

Case 64.—Weighed 12 st. 13 years ago, lost weight and was ill but not laid up, and 9 years ago weighed 11 st.; he then over exerted himself and broke a blood vessel, spat for 4 or 5 days profusely, coughed up ℥j. occasionally for several years; his weight averaging 10 st. 7lb.; kept at work as coachman; has lost rapidly lately.

Case 65.—Was under this hospital 10 months ago with cough, had then lost weight, took emulsion and oil, lost cough, and considered himself well, till cough returned 4 months ago.

Case 66.—Right upper half consolidated; distant large crepitation; 6 months

ago had lost from 8 st. 12 lb. to 7 st. in two months, with cold; picked up since to 8 st. 6 lb., without oil, and notwithstanding cough and hæmoptysis.

Case 67.—Four years ago, weight as usual, spat a few small clots of blood, with cough, and repeated this once or twice in three months after. Does not think he lost weight till twelve months after. Two months ago profuse hæmoptysis occurred, and he found he was then, at time of hæmoptysis, much thinner. Seven attacks have occurred since.

Case 68.—First hæmoptysis six months ago, ℥ iij. per day for six days, black and clotted. Has spat again in same way twenty times since within six months. Heaviest weight three years ago. Has a syphilitic-looking rash. Had syphilis thirteen years ago, but has since had five healthy children.

Case 70.—When cough began three years ago, he was treated at this hospital, and got better, but did not permanently lose cough; much worse of late; brought up 1½ pints of blood first time, and a less quantity three times in four days. After six weeks oil and emulsion, weight 9 st. 13 lb.=loss 3 lbs.

Case 71.—Cough winter and summer five years; loss of flesh four years; first hæmoptysis streaks, off and on one year; last and largest quantity, a small clot, two weeks ago. General catarrh both lungs; complete disintegration and excavation left upper; right softening. Has taken oil; it disagrees.

Case 72.—Losing health and flesh two years, with cough, due to dusty trade and colds. First hæmoptysis ten months ago, not profuse; second, yesterday, profuse; resonance of lungs equal but defective; crepitation severe, left, middle, and upper.

Case 74.—Although appetite continued good, loss of weight went on, first hæmoptysis occurred on renewing his cold two months after first attack; second hæmoptysis ℥ ij. in a day: occurred after severe coughing one week ago; upper part of left lung consolidated, bronchitis general.

Case 75.—He improved in general condition under oil, emulsion, and arsenic, and weighed 8 st. 13 lb. after six weeks; a seton was then put in over cavity, and on the tenth week weighed 9 st. 5 lb.

Case 76.—On admission he had gained 2 lb. from oil and improved appetite; before that weighed 7 st. 5 lb.; after six weeks oil and emulsion weighed 7 st. 12 lb., then lost appetite, and went down to 7 st. 4 lb. after six weeks.

Case 77.—Heaviest weight known 10 st. 5 lbs. two years ago; 9st. 10 lb. one year ago; 8 st. 10 lb. now. He was sure the loss of weight began before hæmoptysis. Dr. Williams, of York, said he brought up four to five quarts in twenty-four hours eighteen months ago. Only streaks since. Four brothers and father died of consumption.

Case 78.—Had cough about ten years; began losing weight two years ago; profuse hæmoptysis, two quarts in five days, first in bulk, then oozing, five weeks ago. Diarrhœa immediately after. Had taken lots of oil, but continued to lose flesh. Symptoms of general catarrh. Deep cavity right upper, softening left. Under treatment with oil and emulsion seventeen weeks; first five weeks maintained 7 st. 4 lb., next six weeks lost 5 lb., next six weeks lost 3 lb. Final weight, 6 st. 10 lb.

Case 79.—Traced all illness to severe cold two years ago, renewed one year ago; since then pains over chest. Bruit de pot fêlé right lung, softening left; much catarrhal tube obstruction.

Case 80.—Cough and short breath 10 months; subject to cough off and on before; fifteen years ago took cod oil for consumption; cough much worse four or five months; ten weeks hæmoptysis ¼ pint in four days; then went on slightly; loss of weight since cough got worse four months ago.

Case 81.—Larynx much implicated. Bruit de pot fêlé left upper. Loss of voice, two months.

Case 82.—Slightly losing flesh 13 years. Under late hours became thin and weak, and without over-exertion or other cause, brought up a little blood, and a few

days after ℥ iv. in a day; under treatment and rest got well, but never regained weight, and has slowly lost since. No more hæmoptysis or cough till three weeks ago, when, after losing flesh more rapidly for five months, got cough and expectorated ℥ ss. blood, with phlegm every morning; upper right front dull, upper left back dull; left front super-resonant. Under oil and emulsion gained 3 lbs. in six weeks, and improved generally. No more hæmoptysis.

Case 83.—Laid up eleven years ago with cold from wetting, but had no cough, and remained well till he began to lose flesh six years ago. Has spat blood mixed in sputa eight months constantly. Extensive consolidation and softening of right upper. Larynx implicated.

Case 84.—Oil, emulsion, and blistering six weeks; slight hæmoptysis had continued pretty steadily for six months; discharged doing well.

Case 85.—Hæmoptysis eight months ago; small clots three or four times a day for a week. Had taken oil four weeks at Bartholomew's.

Case 86.—Gastric fever twenty months ago, followed by cough. Weighed 11 st. 8 lb. before fever, 10 st. ten months ago; one week ago brought up ½ pint of florid blood mixed with clots, followed by oozing; four days ago brought up a quart, and a tinge continues. Right upper disintegrating.

Case 87.—Has been taking ℥ ij. oil a day, and growing, and yet has lost weight steadily.

Case 88.—First hæmoptysis occurred at 17, a few little clots with cough; after that completed growth, and had no chest symptoms till slight cough began at 20; at 21 reached heaviest weight, and although he has lost and gained often the balance has always been to the bad; heaviest weight never regained. At 23, second hæmoptysis, small clots as before; at 25 cough got much worse, and so continues at 26. General catarrh; consolidation of both upper; softening of left; after six weeks oil and emulsion had lost 5 lb.

Case 89.—Four years ago weighed 9 st. 4 lb.; then had accident and shock; lost health from that time; one month after accident coughed up a pint of florid blood; twelve months ago coughed up over a pint, and four months ago a pint. Left lung extensively excavated. After six weeks' treatment, oil, emulsion, etc., had gained 6 lb., and cavern nearly dried; improved during another month. Then neglected treatment one month and took cold, and weighed only 7 st. 5 lb. Temperature, 102. Lung breaking up.

Case 90.—Coughed up ½ pint dark blood yesterday, followed to-day by ¼ pint, florid, and not clotted; cough violent; considerable pulmonary congestion upper parts; after twelve weeks oil, emulsion, and evacuants, etc., maintained weight, and felt double strength.

Case 91.—Under oil and emulsion recovered two years ago, and kept well, but without regaining full weight. Five months ago pain in chest and cough set in, followed by profuse hæmoptysis. After six weeks oil and emulsion, no further loss of weight; after six weeks more, loss of 3 lb.

Case 92.—Under my care, off and on, five years. Weighed 9 st. five years ago, before hæmoptysis; 8 st. 6 lb. three years ago. Hæmoptysis only occurred with recurrence of cough, but weight was never regained; general catarrh and disintegration of right lung.

Case 93.—Heaviest weight known was three months before first hæmoptysis, and patient asserted there was no loss of weight or sign of illness till he broke the blood-vessel, lifting a heavy weight; since that frequent small bleedings, up to two months ago. Left lung dull, and disintegrating, not very active; right nearly normal. After six weeks oil and emulsion, gained 5 lb.; first hæmoptysis was three pints florid blood.

Case 94.—After cough and cold two months, spat up one pint dark blood; subsequently a little blood several times, after lifting. He lost more weight after bleeding than before, and has regained a little.

Case 95.—Had slight cough for ten years, but dates all symptoms from severe cough and cold six months ago. Heaviest weight 11 st. 3 lb., seven years ago; 10 st. 8 lb. three months ago; disintegration active.

Case 96.—Both parents died young, no consumption known in family. Hæmoptysis nine years ago, streaks with cough; three years ago, ℥j. with cough; and one week ago ℥j. with cough. Has gradually lost weight ten years; never regained original weight of 9 st. 5 lb. Weighed 9 st. at time of first hæmoptysis; 8 st. eight years ago, ill then with cough; got well with oil; 6 st. 12 lb. four years ago, before second hæmoptysis; with oil and change, got to 7 st. 4 lb., and averaged 7 st. 4 lb. to 8 lb. up to one year ago. Lungs catarrhal from end to end; general symptoms extreme. No cavity, but consolidation and softening upper lobes.

Case 97.—Second hæmoptysis followed immediately on first hæmoptysis. Had been weak twelve months, and lost 9 lb., when he spat ℥v. of blood, followed for a fortnight by streaks of blood. Got quite well, but never entirely lost cough, but regained former weight, 12 st. 7 lb., and kept it, till one year ago weighed 12 st. 3 lb.; four months ago ill with cough, and got well taking oil; now ill again, 12 st. 1 lb.; cracked pot sound and cav. râle left upper, consolidation right apex.

Case 98.—Growing while losing weight, no consumption in family.

Case 99.—After six weeks oil and emulsion, gained 6 lbs.

Case 100.—Weighed 9 st. 9 lb. one month after first hæmoptysis. He had brought up one pint in one day, and a little bleeding went on three days; he took oil and emulsion then, and ceased to lose weight; lost again, after leaving off treatment.

Case 101.—At 19 years old, while growing, weighed 7 st. 5 lb.; never weighed as much since 21, when he began to fade, and at 22 chest became delicate, and he spat 5 ozs. florid blood, coughed it up, having had previous cough a few months; coughed up congealed blood afterwards, and off and on since. Two years ago, under treatment, weighed 6 st. 6 lb. Has been better in health and stationary in weight since, and has taken cod oil off and on. Now is worse again, and sputa mixed with blood. Under treatment six weeks he gained weight up to 6 st. 12 lb.

Case 102.—Cough three years and three or four months; first hæmoptysis, half-pint suddenly coughed up after exertion; frequent slight hæmoptysis since; had lost flesh with cough, and taken cod oil at least three months before hæmoptysis. Patient at hospital two years and six months ago, took oil then; has recurrent diarrhœa; extreme emaciation.

Case 103.—Heaviest weight 10 st. at time cough began; seven months after weighed 8 st. 12 lb. time of first hæmoptysis, then came under me at hospital; took oil and emulsion and gained; has taken oil since, and kept weight up to 9 st. 4 lb.; never lost cough but lost sweats, and no return of hæmoptysis. Worse now; after four weeks emulsion, weight 9 st. 8 lb.; all sounds dry; consolidation right upper marked, left slight.

Case 104.—Exceptional case, carefully investigated; five years ago in good health; got cold and cough, and went out of town for it (1868); after cough had lasted five months, and while throat was sore, over-reached and exerted himself taking books from a case, and suddenly coughed up a half-pint of florid blood, after which cough rapidly went, and he remained in usual health till last December (1872); his wife is certain that he weighed 9 st. when cough came, and 9 st. when it left, no loss of weight following first hæmoptysis, and that he kept 9 st. up to date of second hæmoptysis, February, 1873. December, 1872, eleven months ago, the old cough returned, and in February, 1873 (two months after), profuse hæmoptysis occurred: one pint first day, half-pint other days, gradually diminishing for a week, during which he kept his bed; from this time he lost flesh, although taking cod oil ℥j. a day from December to July; in August he had lost 1 st. (weight 8 st.); cough and occasional slight hæmoptysis continued, but appetite kept up; no hæmoptysis now for three months; cough very bad, appetite good; loss of 2 lb. more since August; resting and feeding well, but no

oil. No consumption in family; both upper lobes consolidated and excavating; left, which he says was first affected, is depressed and least active; right most active. Under rest and emulsion, etc., he wonderfully improved in six days, and had gained 5½ lb.

Case 105.—Two years ago over-worked at baking, and lost much flesh, and has never regained; tried to get it back, and drank too much beer, but still lost weight; cough two years; sixteen months ago sputa streaked with blood, and fifteen months ago coughed up ℥ ij. in a day. Heaviest weight 11 st. four years ago, and up to two years ago; fifteen months ago weight 9 st. 8 lb.; took oil and emulsion, and gained up to 9 st. 12 lb. After leaving oil and emulsion lost slightly, although in country and taking care. Present weight, 9st. 4 lb.; no blood fifteen months except streaks. No consumption in family; right upper dull and contracted, nearly down to nipple, cavernous sounds nearly dry; left upper back consolidated slightly, and bronchal.

Case 106.—First hæmoptysis five months ago, streaks for a few days with cough, after loss of weight; second hæmoptysis two days ago ℥ j. per day, mixed with sputa, and followed by streaks. Has been under cod oil and other treatment, but loses weight. One maternal aunt, one maternal uncle, consumptive; one paternal uncle, one paternal aunt, consumptive; one sister consumptive.

Case 107.—Subject to coughs with cold four years, but continuous since two years, and losing weight and strength more or less during that time. First hæmoptysis, 5 ozs. florid, fourteen months ago, after walking in hot weather and coughing; second hæmoptysis, about 5 ozs. from similar cause, two months ago; last hæmoptysis one week ago, without cause, ℥ ij.; has lost 5 lb. last three months, in spite of cod oil and care. Cavity containing fluid, right upper; consolidation, left upper; some catarrh; sweat considerable. Went with wife to Australia.

With reference to the important question of the true position of hæmoptysis in the natural history of pulmonary consumption, it will be observed that the one hundred cases tabulated in Table 1, arrange themselves:—

First, into *three great clinical groups*, characterized by the relation in point of time between the *first hæmoptysis* and the *first loss of weight;*—

And again, into *three other groups*, characterized by the relation, in point of time, between the onset of *first cough*, and the occurrence of *first hæmoptysis;*—

And again, into *two groups*, characterized by the relation, in point of time, between the *onset of first cough* and the *onset of first loss of weight.*

First hæmoptysis occurred *before first loss of weight* in eight cases (8 per cent. Table 4). *At the same time* as first loss of weight in ten cases (10 per cent. Table 6). *After* first loss of weight in eighty-two cases (82 per cent. Table 5.)

First cough occurred *before* first hæmoptysis in eighty-seven cases (87 per cent.). *At the same time* as first hæmoptysis in twelve cases (12 per cent. Table 3.) *After* first hæmoptysis in two cases (2 per cent., Cases 26, 62, see details and analysis).

First cough occurred *before* first loss of weight in sixty-nine cases (69 per cent.). *After* first loss of weight in thirty-one cases (31 per cent.).

I have taken loss of weight as the most palpable sign of constitutional decline (see Parts IV. and V.), and it will be seen by Column 17, Table 1, that in every one of the hundred cases the patient weighed less than he had done at a previous period of his life; and a comparison of Columns 14 and 15, Table 1, shows that the average loss of weight per patient was 19·65 pounds. The rate at which this loss had occurred in the different groups of cases is shown in Table 8,* and in Table 9 I have given the total loss of weight, and the rate at which loss of weight had occurred at the different stages of lung disease.

Column 18, Table 1, shows that in only two cases out of the hundred had the *previous heaviest weight* been entirely regained at any time since the first onset of disease; and in these two the recovery of weight was altogether exceptional in its circumstances and meaning, as shown in the commentaries on the cases.

Column 29, Table 1, shows that in twelve cases cough began at the same time as first hæmoptysis; and I have constructed a special Table for this group of cases, in which the leading facts of each can be seen at a glance (Table 3).

It is a striking fact, that eighty-two of the hundred patients had lost weight *before the occurrence of the first hæmoptysis*—the average time during which this loss had taken place previous to the hæmorrhage being no less than 469·7 days. It is, then, self-evident that in these eighty-two cases some diseased condition existed before the occurrence of hæmorrhage, and that, therefore, we have no reason for seeking a cause in the hæmoptysis.

Our special interest is, therefore, concentrated upon the eighteen cases forming the two smaller groups arranged in Tables 4 and 6.

In Table 4, it is shown that first hæmoptysis *preceded first loss of weight* by 853 days on an average in eight cases.

In Table 6, first hæmoptysis and first loss of weight were coetaneous in ten cases.

In either of these groups, therefore, it is clearly possible to urge that the hæmoptysis was the cause of the constitutional decline, and of all the local disease accompanying it.

To each of these eighteen cases, therefore, I have devoted a special commentary, with a view to clearing up, as far as possible, the posi-

* In eighty-two cases in which loss of weight began *before* first hæmoptysis, rate about 7 lb. per 365 days.

In ten cases in which loss of weight began at *same time* as first hæmoptysis, rate about 8 lb. per 365 days.

In eight cases in which loss of weight began *after* first hæmoptysis, rate about 11 lb. per 365 days.

tion of the hæmoptysis as a cause of disease, and these commentaries may be examined immediately. (See page 28.)

First, however, it is necessary to point out that *one clinical fact*, in addition to *hæmoptysis* and *loss of weight*, at once assumes a right to prominence in discussing these cases. I mean the *onset of the first cough*, and its relation in point of time to the occurrence of first hæmoptysis. I have, therefore, given it a special place in Tables 4 and 6.* For, as cough must be taken to indicate some defective condition of the respiratory organs, if cough preceded first hæmoptysis by any appreciable period of time, it throws at once a grave doubt upon the claim of the hæmorrhage to be regarded as the first cause of the lung disease.

Now we have seen by Table 1 that out of the one hundred cases, first cough preceded first hæmoptysis in eighty-seven, and began after first hæmoptysis in only two.

And turning to the eighteen cases now more particularly under consideration, it is seen by Table 4 that first cough preceded first hæmoptysis by 924 days, on an average, in seven out of the eight cases, and thus indicated that some kind of mischief was going on in the respiratory tract before bleeding occurred. In the one case out of the eight (Case 62) in which the cough began eighty-four days after first hæmoptysis the circumstances were peculiar, and are fully discussed in the commentary on the case.

In Table 6, it is seen that in six out of the ten cases cough preceded first hæmoptysis by 1,600 days on an average, the minimum being 197 days. *In the other four cases, the cough, hæmoptysis and loss of weight began at the same time.* These were Cases 17, 42, 58, 93. In each of these cases it will be seen that there is room for the conclusion that the lung disease proceeded from the hæmorrhage, although in neither of them is this conclusion inevitable ; and the same may be said of Case 15, Table 4, in which *first hæmoptysis preceded loss of weight*. The commentaries on these cases therefore claim our special consideration. But in Case 93 our interest culminates, for in it there appears to be little room for any more rational conclusion than that the lung disease was produced by the damage done by the hæmorrhage. (See par. *d*, p. 26.)

Among the many subjects of interest contained in Table 1, which I have not yet had time to discuss, I may mention one intimately connected with the cases I have detailed. I mean the question whether the results of pulmonary hæmorrhage bear any relation *in respect* to *tissue damage*, to the *quantity* of blood expectorated in hæmoptysis. The materials for a separate table representing the facts relating to this question are to be found in Table 1. In this place I will only say that I am disposed to think that it is of more importance whether the

* Note that in recording "first cough" care was taken that some definite connection could be traced between the cough and the illness connected with first hæmoptysis.

hæmorrhage has taken place *into the respiratory tubes, or into the pulmonary tissues,* than whether the hæmoptysis has been profuse or slight. Analogy lends weight to this opinion, as seen in cerebral hæmorrhage and other instances of bleeding into the tissues and into the passages of internal parts.

It will be observed that in the commentaries on the cases I have studiously avoided entering upon questions of *pathological histology,* and the use of *debateable terms,* my object being to keep as strictly as possible to a simple analysis of the clinical facts presented by the cases produced.

But before we pass on from these cases and analyses, and while they are still in our memory, I will attempt to give them point and meaning by enumerating what appear to me to be some of the principal heads under which these clinical observations justify us in arranging hæmoptysis in the natural history of pulmonary consumption.

As a Symptom Hæmoptysis may be thus Classified:—

1. In a large number of cases it is simply a result of disintegration of a highly vascular organ in the course of a disease of constitutional origin. (*See* paragraph *e,* and note to it.)

2. In a large number of cases it is simply the result of congestion and disintegration of a highly vascular organ in the course of disease of local origin.

3. In a certain number of cases it is simply the result of accidents, temporarily over-distending the vascular system of the lungs, and leading to the rupture of vessels in the same way as similar over-distension leads to rupture of vessels in other parts of the body.

Whether such over-distension is competent to cause rupture of vessels, the walls of which are not previously diseased, is a very wide question, which must be argued with reference to the whole vascular system before it is specialised within the narrow area of the pulmonary circulation. Is it necessary to assume that a vessel was diseased because it burst under exceptional distension?

As a Cause of Lung Disease and Constitutional Decline:—

I am disposed to place hæmoptysis as only one item, and that a very exceptional one, in a large and important group, embracing *all foreign substances which find their way into the perivascular and perialveolar tissue of the lungs,* and, by their irritation there, set up lymphatic (adenoid) hyperplasia and cell-proliferation and their consequences.

Of this important group the following are some of the principal constituents:—

a. Lampblack, coal, flint, steel, stone, and other substances, inhaled by workers in different trades.

b. The products of inflammatory destruction of tissue.

c. The products of catarrhal affections, especially in scrofulosis.

d. The débris of tissue disintegrated by the extravasation of blood, and possibly the débris of the blood so extravasated.

e. Albuminoid tissue, disintegrated by abnormal oxidation in true tuberculosis. [This item involves that initial loss of weight characteristic of the true first stage of consumption.] In my opinion the disintegrated albuminoid tissue is the irritant which sets up the hyperplasia of adenoid tissue, and cell proliferation and its results, so well described by Portal, Virchow, Sanderson, Rindfleisch, and others. But whereas they place these processes first among the pathological changes of tuberculosis, I give precedence to disintegration of albuminoid tissue by oxidation, of which the hyperplastic and other changes are but the effects; the order of events being, according to my view:—

1. Deficiency of fat in the blood.*
2. Oxidation of albuminoid tissue.
3. The production of disintegrated albuminoid tissue, the result of oxidation.
4. Hyperplasia of adenoid tissue, cell-proliferation, &c., the effects of irritation set up by the disintegrated materials. (See full discussion of this subject at Part IV.)

Nearly all the paragraphs in this summary are evolved from the 100 cases here tabulated, and those which are not absolutely so evolved are necessary to give the proper position to the others. This especially applies to par. *e.*

* The cause of this deficiency of fat in the blood may be an open question. But if the defect is in the supply of fat from the food, it is obvious that it will be first, and especially, felt in the blood of the pulmonary artery, presented for aëration in the lungs. (See Part IV.)

KEY TO FURTHER ANALYSIS OF CASES IN SPECIAL GROUPS.

(For details of cases, see Table 1.)

Table 1, Column 18.—Cases 43, 97.

Two cases in which heaviest weight had at one time been entirely regained, although present weight was less than heaviest weight.

(Case 97 occurs also in Table 5, analysis of 82 cases of hæmoptysis after first loss of weight.)

(Case 43 occurs also in conjunction with 88, in analysis of two cases in which first hæmoptysis occurred before the period of heaviest weight, column 46.)

Table 1, Column 28.—Cases 26, 62.

Two cases in which first cough began after first hæmoptysis.

Table 1, Column 29.—Cases 7, 15, 17, 21, 27, 42, 64, 67, 82, 84, 88, 93.

Table 3, analysis of twelve cases in which first cough began at the same time as first hæmoptysis.

Table 1, Column 50.—Cases 7, 15, 21, 37, 62, 67, 88, 104.

Table 4, eight cases in which first hæmoptysis occurred before first loss of weight.

Table 1, Column 51.—Cases 1, 3, 4, 5, 8, 9, 10, 11, 13, 14, 16, 18, 19, 20, 22, 23, 24, 25, 26, 27, 28, 29, 30, 32, 35, 36, 38, 39, 40, 41, 44, 45, 46, 47, 48, 49, 50, 51, 52, 56, 57, 59, 61, 63, 64, 65, 66, 68, 70, 71, 72, 74, 75, 76, 77, 78, 79, 80, 81, 82, 83, 84, 85, 86, 87, 89, 90, 91, 92, 94, 95, 96, 97, 98, 99, 100, 101, 102, 103, 105, 106, 107.

Table 5, eighty-two cases in which first hæmoptysis occurred *after* first loss of weight.

Table 1, Column 52.—Cases 6, 17, 33, 34, 42, 43, 53, 58, 60, 93.

Table 6, ten cases in which first hæmoptysis and first loss of weight occurred at the same time.

Table 1, Column 48.—Cases 43, 88.

Two cases in which first hæmoptysis occurred *before* the period of heaviest weight.

(Case 43 occurs also in conjunction with 97. Column 18.)

Table 1, Column 67.—Cases 7, 104.

Two cases in which first loss of weight occurred at the *same time as last hæmoptysis.*

Table 1, Columns 5 to 10.—Cases 1 to 107. (In all 100 cases.)

Tables 2, 9, and 10 to 18 analyses of cases of lung disease in different stages. Showing the relation between loss of weight and hæmoptysis in point of time and rate.

Details of Cases in Special Groups, and References to Cases in Tables, with Commentaries.

Two Cases in which Heaviest Weight had at one time been entirely regained, although Present Weight was less than Heaviest Weight.

Case 97 occurs also in Table 5, eighty-two cases of hæmoptysis after first loss of weight.

Case 97, G. P.

Right lung consolidated, left lung excavated.
Heaviest weight, 12 st. 7 lb. Present weight, 12 st. 1 lb.
First hæmoptysis, nine years and ten months ago. Had been weak twelve months, and lost 9 lbs. weight (11 st. 12 lb.) when first hæmoptysis occurred. He spat ℥v. of blood, followed for a fortnight by streaks of blood. No hæmoptysis had occurred since that time.
He got quite well in health, and quite regained weight of 12 st. 7 lb., but never quite lost cough. Kept up weight till twelve months ago. Four months ago weighed 12 st. 3 lb.; was then ill with cough, but got well taking oil. He now feels ill again, and weighs 12 st. 1 lb. Has an empty cavity left upper, consolidation right apex.

Commentary.—It is clear that in this case constitutional decline preceded first hæmoptysis. The constitutional defect afterwards disappeared, but the damage to the lung remained a source of sufficient irritation to keep up a cough. Nearly ten years of health (except a cough) intervened between his first and second decline, and the second was unattended with hæmoptysis, and although ill and weak, and distressed by cough, he had not lost as much flesh as he had lost previous to first hæmoptysis.

There cannot be any justification in this case for an idea that the hæmoptysis was the cause of the lung disease; it was evidently only an accidental occurrence during the first attack of constitutional illness and advancing lung disease.

For full commentary on Case 43, see two cases in which hæmoptysis preceded heaviest weight, and ten cases, Table 6, in which first loss of weight and first hæmoptysis were coetaneous. The complete recovery of heaviest weight was quite exceptional, and occurred only during a voyage to Australia, at a time when the general health, appetite and nutritive functions much needed this rest and stimulus. The weight was rapidly lost after the voyage was over.

Two Cases in which First Cough began after First Hæmoptysis.

CASE 26, G. H. (See Table 5.)

Male, aged 27. Right lung softened and excavated, left lung normal. General symptoms moderate.

Total loss of weight, 18 lbs. First loss of weight began 365 days ago, 281 days before first hæmoptysis; and cough began, without assignable cause, twenty-eight days after first hæmoptysis.

The hæmoptysis was in small clots, over ℥ss. in twenty-four hours; it lasted one week, and did *not* recur.

There was no history of consumption in the family.

COMMENTARY.—This would appear to be a case of tuberculosis proper, the lung disease, cough, and hæmoptysis coming on with the advance of constitutional decline.

CASE 62, R. W. (See Table 4.)

Male, aged 27. Right lung softening, left lung softening. General symptoms, extreme.

Total loss of weight, 20 lbs. in 252 days. First loss of weight began twenty-eight days *after* first hæmoptysis, fifty-six days *before* first cough; so that first cough began eighty-four days after first hæmoptysis, and it had continued ever since.

The blood of first hæmoptysis was florid, profuse, over ℥x. in twenty-four hours. The cause assigned was dancing and getting over-heated, and then drinking iced ale. No recurrence of hæmoptysis.

No consumption in the family.

COMMENTARY.—In this case *hæmoptysis* would appear to have been the cause of the subsequent break up of health and lungs. The order of events was :—Hæmoptysis, 280 days ago; loss of weight, 252 days ago; cough, 196 days ago.—(See further Commentary p. 34.)

Eight Cases in which First Hæmoptysis occurred before First Loss of Weight. (*See* Table 4.)

CASE 7, T. P.

Male, age 26. Right lung softening, left lung consolidated, upper parts. General symptoms, moderate.

Present weight, 111 lbs. Heaviest weight, 121 lbs., three years ago. Present weight less than heaviest weight by 10 lbs. Time elapsed since first loss of weight, 112 days, same time as last hæmoptysis. Cause of first loss of weight assigned, hæmoptysis (last hæmoptysis). Loss of weight had been treated with cod oil.

Time elapsed since first cough began, 1,460 days, same time as present

cough and same time as first hæmoptysis. First cough began before first loss of weight, 1,348 days. First cough began at the same time as first hæmoptysis, 1,460 days ago. Cause assigned of first cough, *nil*. Present cough began at the same time as first cough, 1,460 days ago. Time elapsed since first hæmoptysis, 1,460 days, same time as first cough, 1,348 days before first loss of weight.

The blood of first hæmoptysis was florid. The quantity of blood in first hæmoptysis in twenty-four hours was over ℥ss., and lasted daily for a week. Cause assigned of first hæmoptysis, *nil*. First hæmoptysis occurred after the period of heaviest weight. First hæmoptysis occurred before the first loss of weight, 1,348 days. Hæmoptysis recurred. Time elapsed since last hæmoptysis, 112 days, same time as first loss of weight, 1,348 days after first hæmoptysis. The blood of last hæmoptysis was florid. The quantity of blood in last hæmoptysis was over ℥ss. in twenty-four hours. The last hæmoptysis occurred at the same time as first loss of weight, 112 days ago.

The family history was free from consumption.

NOTE.—This is one of the two cases (Col. 67, Table 1) in which first loss of weight occurred at the same time as last hæmoptysis. (See twelve cases, Table 3, in which cough began at the same time as first hæmoptysis.)

COMMENTARY.—The heaviest weight known by weighing occurred one year after first hæmoptysis, and it was positively declared that no loss of weight had occurred till the time of last hæmoptysis, and no hæmoptysis had occurred between those times (1,348 days). Under oil, emulsion and other treatment the heaviest weight was regained within 1 lb.

Apparently no constitutional decline occurred before or after first hæmoptysis, but cough began with it and continued after it, indicating the existence of some lung irritation. Decline of health began with the second and more profuse hæmoptysis, 10 lb. in weight being lost, but this was mostly regained under treatment. The family history was free from consumption.

In this case the constitutional defect was not very marked, even to the last. The small quantity of blood in first hæmoptysis, ℥ss. in twenty-four hours, and the 1,348 days' interval of comparative health, only disturbed by chronic cough, are more consistent with the idea of catarrhal damage to the lung causing slight hæmorrhage than of hæmorrhage causing lung disease, or of lung disease having a constitutional cause.

The *second and last hæmoptysis* was more profuse, and was followed by constitutional decline, and a loss of 10 lb. in weight. This might, therefore, be colourably set down as the cause of the softening and consolidation of lung; but it must be remembered that 1,348 days of cough, commencing with the first and slight hæmoptysis, had preceded the second attack, indicating that chronic lung disease was going on

before the second bleeding occurred. On the whole, therefore, although this is just one of those cases apt to be set down as of hæmorrhagic origin, I am disposed to regard it rather as one of accidental hæmorrhage in the course of catarrhal lung disease.

Case 15, R. W.

Male, age 29. Right lung excavated, left lung excavated, upper lobes. General symptoms, severe.

Present weight, 110 lbs. Heaviest weight, 146 lbs. Present weight less than heaviest weight by 36 lbs. Heaviest weight had never been regained. First loss of weight began 730 days ago, 365 days after first hæmoptysis. Cause assigned for first loss of weight—hæmoptysis. Loss of weight had been treated with cod oil.

First cough began 1,095 days ago, same time as present cough. The cause of first cough assigned was *nil*. First cough began before first loss of weight, 365 days. First cough began at the same time as first hæmoptysis, 1,095 days ago. Present cough began at the same time and from same cause as first cough.

First hæmoptysis occurred 1,095 days ago, 365 days before first loss of weight, same time as first cough. The quality of blood in first hæmoptysis was florid. The quantity of blood in first hæmoptysis was over ℥ss. in twenty-four hours. The cause of first hæmoptysis assigned was cough. First hæmoptysis occurred before first loss of weight, 365 days. Hæmoptysis had recurred. Time elapsed since last hæmoptysis, 504 days, 591 days after first hæmoptysis, 226 days after first loss of weight. The blood of last hæmoptysis was florid. The quantity of blood in last hæmoptysis was over ℥x. in twenty-four hours.

Father, mother, and one sister were consumptive, five brothers and sisters were healthy.

Commentary.—Although stated that first loss of weight was due to hæmoptysis, there is no evidence of the fact; no loss of weight occurred for 365 days after first hæmorrhage. Between first and last profuse hæmoptysis cough had continued, and the sputa had often been sanguinolent. This was a highly consumptive family, and the first hæmoptysis occurred at about 26 years of age—a period at which the constitutional complaint might be expected to culminate. But no constitutional decline, as indicated by loss of weight, occurred till 365 days after the hæmoptysis, although cough had continued ever since the hæmorrhage, and the sputa had been sanguinolent from time to time. It was, however, stated that the hæmoptysis was caused by cough, and weight had been maintained with the assistance of cod oil, although, after the loss began, 36 lb. had been lost in two years.

Had the family history been free from consumption, it would have been more open to the conclusion that the hæmorrhage caused the destruction of lung and constitutional decline—but with father, mother, and one sister consumptive, it is more probable that lung disease pre-

ceded the hæmorrhage. The explanation of the event is, however, open to a different conclusion.

Case 21, A. K.

Male, age 23. Right lung excavated, left normal, upper parts. General symptoms, moderate.

Present weight, 121 lb. Heaviest weight, 136 lb. Present weight less than heaviest weight by 15 lb. Time elapsed since first loss of weight, 168 days, 562 days after first hæmoptysis. Cause of first loss of weight assigned—cough. Loss of weight had not been treated with oil or emulsion.

Time elapsed since first cough began, 730 days, same time as present cough and first hæmoptysis. Cause of first cough assigned, *nil*. First cough began before first loss of weight, 562 days. First cough began at same time as first hæmoptysis. Present cough began at the same time as first cough. Time elapsed since first hæmoptysis, 730 days.

The blood of first hæmoptysis was florid. The quantity of blood in first hæmoptysis was over ℥ss. in twenty-four hours. The cause of first hæmoptysis assigned was cough. First hæmoptysis occurred before first loss of weight by 562 days. Hæmoptysis had not recurred.

There was no consumption in the family history.

Commentary.—730 days ago he coughed and spat up about ℥j. of florid blood, has coughed more or less ever since, and has never been quite well, but maintained his heaviest and average weight of 136 lb. up to 168 days ago. Lost rapidly since, and in spite of treatment lost 3 lb. more in nine weeks, reducing weight to 118 lb. A seton was placed over the cavity, which cleared out. Health much improved, and no further advance of disease occurred in the lung while under observation.

No constitutional decline, as indicated by loss of weight, occurred till 562 days after he had coughed up over ℥ss. of blood. The cough began with this hæmorrhage and continued ever afterwards, but hæmoptysis did not recur. Although not losing weight, he had not been well, and he attributed the loss of weight, which set in 168 days ago, to the wear of the cough; from this time loss of weight was rapid and obstinate—15 lb. being lost in 168 days in spite of care and treatment, accompanied by excavation of the right lung. After the clearing out of this cavity, and with the assistance of a seton, and of oil and emulsion, the progress of disease was arrested.

In this case the absence of any decline previous to first cough and hæmorrhage, and the abeyance of loss of weight during 562 days of subsequent cough, throw a doubt upon the constitutional origin of the lung disease, and favour the idea that, whether or not the hæmorrhage caused the lung mischief, the advancing lung disease led to the constitutional decline which set in most definitely 168 days before admission.

This idea is strengthened by the fact of the recovery of health which took place when the cavity had cleared out and the seton had removed irritation from the lung.

As it must be conceded that some cause for the hæmorrhage ought to be found, and is not apparently to be found unless in the cough which excited it, and as that cough could not be at once the cause and the effect of the hæmorrhage; and seeing that cough as a result of catarrhal congestion is a common and well-known fact—that tissue disintegration as a consequence of congestion is another well-known fact—and that hæmorrhage as a result of congestion of a highly vascular tissue is another well known fact; I think the most rational view to take of this case is that the hæmorrhage was an accidental accompaniment of a catarrhal attack from which the subsequent changes proceeded—although this again is a case which is apt to be set down as *prima facie* of hæmorrhagic origin.

Case 37.

Male, age 34. Right lung consolidated, left lung softening and excavating, upper parts. General symptoms, moderate.

Present weight, 143 lbs. Heaviest weight, 157 lbs. Present weight less than heaviest weight by 14 lbs. Time elapsed since first loss of weight, 168 days, 1,657 days after first hæmoptysis, same time as present cough. Cause of first loss of weight assigned—cough. Loss of weight had not been treated either with oil or emulsion.

Time elapsed since first cough began, 7,300 days (in winter), 5,475 days before first hæmoptysis. Cause of first cause assigned—*nil.* First cough began before first loss of weight, 7,132 days. First cough began before first hæmoptysis, 6,475 days. Time elapsed since present cough began, 168 days, just before or coetaneous with first loss of weight. Cause of present cough assigned—*nil.* Present cough began before (or coetaneously with) first loss of weight, 168 days ago. Present cough began after first hæmoptysis, 1,657 days.

Time elapsed since first hæmoptysis, 1,825 days, 1,811 days before second hæmoptysis. The blood of first hæmoptysis was in streaks. The quantity of blood in first hæmoptysis was under ℥ss. in twenty-four hours. The cause of first hæmoptysis assigned was "effects of pleurisy," which had occurred 365 days before. First hæmoptysis occurred before first loss of weight by 1,657 days. Hæmoptysis had recurred. Time elapsed since last hæmoptysis, 14 days, 1,811 days after first hæmoptysis. The blood of last hæmoptysis was florid. The quantity of blood in last hæmoptysis was over ℥ss. in twenty-four hours. (It was ℥v.) The last hæmoptysis occurred after first loss of weight, 154 days, and followed attack of pulmonary congestion.

The family history was free from consumption.

Subject to winter cough for twenty years or more (7,300 days), without interference with health till late years. Six years ago, 2,190 days, had pleurisy, and one year afterwards, 1,825 days ago, the

sputa became streaked with blood. No blood had been seen since till fourteen days ago, when he expectorated ℥v. of florid blood. His loss of weight he dated from his present cough, which began six months ago, 168 days.

On admission to hospital, before last hæmoptysis, patient had severe congestion of the right upper lobe, quickly relieved by leeches ; also large disintegration signs in the left upper lobe, which, during stay in hospital, became cavernous.

He had suffered long, and was still suffering, from severe post-nasal-catarrh, which may have been the source of the streaks of blood called "first hæmoptysis."

He greatly improved, and was sent to Eastbourne, and there he had the second and last-named hæmoptysis of ℥v. of florid blood, a fortnight after which, on return to hospital, disease was found to be advancing in the right upper lobe.

COMMENTARY.—In this case there can be no reasonable doubt that the hæmoptysis was a symptom, not a cause of the lung disease—a long standing catarrhal history existed, and the first hæmoptysis was only in streaks many years ago. The subsequent and recent profuse hæmoptysis succeeded a considerable loss of weight and cough, which would put it out of court as *a cause of* constitutional decline, and it accompanied symptoms of a distinct attack of pulmonary congestions, and disintegration of lung.

CASE 62, R. W.*

Male, age 27. Right lung softening, left lung softening, upper parts. General symptoms, extreme.

Present weight, 111 lb. Heaviest weight, 131 lb. Present weight less than heaviest weight by 20 lb. Time elapsed since first loss of weight, 252 days, 28 days after first hæmoptysis. Cause of first loss of weight assigned—*nil.*

Time elapsed since first cough began, 196 days, 84 days after first hæmoptysis. First cough began after first loss of weight by 56 days. First cough began after first hæmoptysis, 84 days. Present cough began at the same time as first cough, it had never ceased.

Time elapsed since first hæmoptysis, 280 days, 28 days before first loss of weight. The blood of first hæmoptysis was florid. The blood of first hæmoptysis was in quantity over ℥x. in twenty-four hours. The cause of first hæmoptysis assigned was "dancing and drinking iced ale." First hæmoptysis occurred before first loss of weight by 28 days. Hæmoptysis did not recur.

The family history was free from consumption.

COMMENTARY.—This is a typical case of consumption proceeding *primâ facie* entirely from hæmoptysis. Nothing could be extracted by

* This is one of two cases in which first cough began after first hæmoptysis.

the most careful enquiry to account for the hæmorrhage, except the cause assigned by the patient—viz. : that "while at his full weight and in usual health, he overheated himself with dancing, and then drank some iced ale, and immediately broke a blood vessel in his chest." No recurrence of hæmorrhage had taken place. Loss of weight began about a month after the accident, and 20 lb. had been lost in 252 days. *No cough set in till* 84 *days after the accident* and 56 *days after loss of weight*, but since it began it had never ceased. This is a remarkable circumstance, for if the decline in health, indicated by *loss of weight were due to local disease set up by the hæmorrhage*, it is very improbable that it should not have excited the ordinary symptom of lung disease or irritation, viz., cough.

When we regard these facts in relation with the condition of the lungs on admission, *i.e.*, softening of both upper lobes, it becomes still more remarkable that no cough should have been excited by the hæmorrhage, if it is supposed that the structure of both lungs was so much damaged by the effusion of blood as to lead to all that followed. My opinion is that these circumstances throw the greatest doubt upon such a supposition, and I am led to put aside the *primâ facie* conclusion that the whole case started from the hæmorrhage, and to conclude, instead, that the following is a much more probable solution. That at the time the accident occurred, the patient was not in such unquestionable health as he imagined ; that his habits of dancing, drinking and late hours were telling upon his constitution; that his subsequent decline was imminent at the time of the accidental hæmorrhage; that the hæmorrhage brought on by the temporary over-heating and excitement, instead of causing disease of the lungs *protected* them; that the relief to the vascular system, combined with the rest and ease enforced by the alarm caused by the hæmorrhage, actually postponed those changes in the lung which otherwise might have appeared much sooner than they did, and that thus it is that we have a history of 28 days in which no obvious change took place in the health. Then constitutional decline and loss of flesh for 56 days before sufficient irritation had occurred in the lungs to cause a cough.

If then we put aside the hæmorrhage as accidental, we see an ordinary case of consumption in which the events follow the usual order, viz.—

1. Damaged constitutional health (by fast living ?).
2. Decline of weight and strength.
3. Lung irritation and cough.
4. Advancing lung disease and constitutional decline.

Case 67, J. B.

Male, age 26. Right lung normal, left lung consolidated and softening, upper lobes. General symptoms, moderate.

Present weight, 143 lb. Heaviest weight, 156 lb. Present weight less

than heaviest weight by 13 lb. Time elapsed since first loss of weight, 1,095 days, 365 days after first hæmoptysis. Cause of first loss of weight assigned—cough. First loss of weight had been treated with cod oil.

Time elapsed since first cough began, 1,460 days. Cause of first cough assigned—*nil.* First cough began before first loss of weight by 365 days. First cough began at same time as first hæmoptysis, 365 days before first loss of weight. Time elapsed since present cough began, 56 days. Cause of present cough assigned, *nil.* Present cough began after first loss of weight, 1,039 days. Present cough began after first hæmoptysis, 1,404 days.

Time elapsed since first hæmoptysis, 1,460 days. The blood of first hæmoptysis was in small clots. The quantity of blood in first hæmoptysis was under ʒss. in twenty-four hours. Cause of first hæmoptysis assigned—cough. First hæmoptysis occurred before first loss of weight, 365 days. Hæmoptysis had recurred. Time elapsed since last hæmoptysis, twenty-eight days, 1,432 days after first hæmoptysis. The blood of last hæmoptysis was florid. The quantity of blood in last hæmoptysis was over ℥x. Last hæmoptysis occurred after first loss of weight, 1,067 days.

The family history was free from consumption.

While at his usual weight, 156 lb., 1,460 days ago, coughed up a few small clots of blood. This was repeated once or twice in the following two months. Does not think he lost any weight till 1,095 days ago, 365 days after first spitting of blood. Fifty-six days ago, with present cough, profuse hæmoptysis occurred, and he found on weighing at time of hæmoptysis that he had lost much flesh. Seven attacks have occurred since; the last twenty-eight days ago, over ℥x. in twenty-four hours.

Commentary.—Cough and hæmoptysis occurred 365 days before loss of weight. The hæmoptysis was under ʒss. in twenty-four hours. Small clots in the sputa. It recurred once or twice during two months, and then ceased; and at an interval of 365 days after the first appearance of blood, loss of flesh and constitutional decline commenced. Loss of flesh then proceeded during 1,039 days, when a fresh attack of cough occurred, and twenty-eight days afterwards it was accompanied by a profuse hæmoptysis of over ℥x. of florid blood in twenty-four hours. The total loss of weight in 1,095 days was 13 lb., and the bleeding recurred seven times in twenty-eight days.

Looking dispassionately at this series of events, I think it is more rational to regard them as a history of slowly-advancing lung disease, accompanied in two periods of its course by hæmorrhage, than to conclude that the hæmorrhage which accompanied the first onset of cough was a cause of the subsequent changes.

Case 88, F. T.

Male, age 26. Right lung consolidated. Left lung consolidated and softening, upper parts. General symptoms moderate.

Present weight, 140 lb. Heaviest weight, 154 lb. Present weight less than heaviest weight by 14 lb. Time elapsed since first loss of weight 1,825 days, 1,460 days after first hæmoptysis. Cause of first loss of weight assigned—*nil*. Loss of weight had been treated with cod oil.

Time elapsed since first cough began, 3,285 days, same time as first hæmoptysis. Cause of first cough assigned—nil. First cough began before first loss of weight, 1,460 days. Age 17 at time of first cough and first hæmoptysis. Completed growth and reached heaviest weight at 21, after first cough and hæmoptysis. First cough began at the same time as first hæmoptysis, 3,285 days ago. Time elapsed since present cough began, 1,825 days. Present cough began just before loss of weight, or at same time. Present cough began after first hæmoptysis, 1,460 days.

Time elapsed since first hæmoptysis, 3,285 days. The blood of first hæmoptysis was in small clots. The quantity of blood in first hæmoptysis was under 3ss. in twenty-four hours. Cause of first hæmoptysis assigned was cough. First hæmoptysis occurred before heaviest weight (at 17 years old). First hæmoptysis occurred before first loss of weight, 1,460 days. Hæmoptysis had recurred.

Time elapsed since last hæmoptysis, 1,095 days, 2,190 days after first hæmoptysis. The blood of last hæmoptysis was in small clots. The quantity of last hæmoptysis was over 3ss. in twenty-four hours. Last hæmoptysis occurred after first loss of weight, 730 days.

One or more sisters were consumptive.

COMMENTARY.—First hæmoptysis occurred with a cough at 17 years of age, before completing growth, 3,285 days ago. After that he completed growth and reached his heaviest weight between 20 and 21 years of age, 154 lb., no chest symptoms occurring from date of first hæmoptysis till then. Cough began again at 21, and although he had gained and lost often, the balance had always been a loss, and he never had regained his heaviest weight.

At 23, 1,095 days ago, the second and last hæmoptysis occurred with cough, and was in small clots as before, but in larger quantity.

At 25 cough had become much worse, and so continued at 26.

There was consolidation of both upper lobes and softening of the left upper.

Loss of flesh had been treated with cod liver oil without effect, and in six weeks' treatment with oil and emulsion he lost 5 lb. more of weight.

The increase of weight after first hæmoptysis, at the age of 17, was coincident with growth; and when growth was completed the loss of weight began. Growth had, in all probability, been made at the expense of fat, and the family being consumptive the constitutional tendency to disease culminated.

The first hæmoptysis, which was under 3ss. in twenty-four hours, was produced by cough, and 1,460 days elapsed between its occurrence and the setting in of active symptoms. It is not rational, looking at

the whole sequence of events in the case, to regard the hæmorrhage as the cause of the subsequent lung disease.

See also two cases in which first hæmoptysis occurred *before the period of heaviest weight.* Cases 43, 88.

Case 104, H. F.*

Male, age 32. Right lung excavated, left lung excavated, upper parts. General symptoms, extreme.

Present weight, 110 lb. Heaviest weight, 126 lb. Present weight less than heaviest weight by 16 lb. Time elapsed since first loss of weight, 252 days, same time as last hæmoptysis. Cause of first loss of weight assigned—hæmoptysis. Loss of weight had been treated with cod oil.

Time elapsed since first cough began, 1,460 days, 1,152 days before present cough. Cause of first cough assigned—cold and sore throat. First cough began before first loss of weight, 1,208 days. First cough began before first hæmoptysis, 169 days. Time elapsed since present cough began, 308 days, 1,152 days after first cough. Cause of present cough assigned—cold. Present cough began before first loss of weight, 56 days. Present cough began after first hæmoptysis, 983 days. Before last hæmoptysis, 56 days.

Time elapsed since first hæmoptysis, 1,291 days. Blood of first hæmoptysis was florid. Quantity of blood in first hæmoptysis during 24 hours was over ℥x. Cause of first hæmoptysis assigned—cough, sore throat, and over-exertion. First hæmoptysis occurred after period of heaviest weight. First hæmoptysis occurred before first loss of weight, 1,039 days. Hæmoptysis had recurred. Time elapsed since last hæmoptysis, 252 days, 1,039 days after first hæmoptysis. The blood in last hæmoptysis was florid. The quantity of blood in last hæmoptysis was over ℥x. The last hæmoptysis occurred after period of heaviest weight. Last hæmoptysis occurred at the same time as first loss of weight, 252 days ago.

The family history was free from consumption.

Commentary.—An exceptional case, carefully investigated, with unusually reliable evidence. In 1868, while in good health, he got cold and cough, for which he went out of town. After cough had lasted five months, and while throat was sore he over-reached himself taking some books from a shelf, and suddenly coughed up half a pint (or more) of florid blood, after which cough rapidly got well. He regained his usual health and so remained till December, 1872. His wife and he are certain that he weighed 9 st. when cough began, and 9 st. when it left; that no loss of weight either preceded or followed first hæmoptysis, and that he remained at 9 st. up to the date of second hæmoptysis, February, 1873.

December, 1872, the old cold and cough returned, as in 1868, and in the following February (1873) profuse hæmoptysis occurred—one pint

* This is one of two cases in which first loss of weight occurred at the same time as last hæmoptysis; Cases 7, 104. (See further commentary.)

the first day, half-pint on other days, gradually diminishing for a week, during which he kept his bed. From this time he lost flesh, although taking ℥j. of cod oil per day from December, 1872, to the end of July, 1873. During this time he had lost 1 st. (weighing 8 st. in August); appetite kept up, but cough and occasional slight sanguinolent expectoration continued. After three months, during which he had not taken oil, but had been feeding well and resting a good deal, he had lost 2lbs. more (weight in November, 1873, 7st. 12lb.). Both right and left upper lobes consolidated and excavating. The left, which he says was the first affected, less active than the right, and the chest wall depressed. A blister was applied, complete rest enforced, oil and emulsion taken, etc., and in six days he had remarkably improved and gained 5½ lb. in weight. The hæmoptysis was evidently a consequence not a cause of the lung disease.

Analysis of Ten Cases in which First Hæmoptysis and First Loss of Weight occurred at the same time. (Table 6.)

CASE 6, E. C.

Male, age 31. Right lung catarrhal, left lung softening, upper parts. General symptoms, moderate.

Present weight, 135 lb. Heaviest weight, 144 lb. Present weight less than heaviest weight by 9 lb. Time elapsed since first loss of weight, 168 days, same time as first hæmoptysis, 1,292 days after first cough, 166 days before last hæmoptysis. Cause of first loss of weight assigned—*nil.* Loss of weight had not been treated with oil or emulsion.

Time elapsed since first cough began, 1,460 days. Cause of first cough assigned—cold. First cough began before first loss of weight, 1,292 days First cough began before first hæmoptysis, 1,292 days. Present cough began at same time and from same cause as first cough.

Time elapsed since first hæmoptysis, 168 days. The blood of first hæmoptysis was florid and mixed with sputa (flesh-coloured sputa). The quantity of blood in first hæmoptysis was over ℥ss. in 24 hours. The cause of first hæmoptysis assigned was cough. First hæmoptysis occurred at the same time as first loss of weight. Hæmoptysis had recurred. Time elapsed since last hæmoptysis 2 days, 166 days after first loss of weight and first hæmoptysis. The blood of last hæmoptysis was mixed with sputa (flesh-coloured sputa), and in small clots. The quantity of blood in last hæmoptysis was over ℥ss. in 24 hours. Last hæmoptysis occurred after first loss of weight, 166 days.

The family history was free from consumption.

COMMENTARY.—Loss of weight and first hæmoptysis were declared to be coetaneous. But he had cough summer and winter for 4 years, 1,292 days before hæmoptysis, and he had not weighed for a long while till alarmed by hæmoptysis, when he found he was losing. Still he had only lost 9 lb. in 168 days.

Cough had existed 1,292 days before first hæmoptysis, although first loss of weight did not occur till the time of the hæmorrhage. This precludes the conclusion that the lung disease began with hæmoptysis.

CASE 17, A. G.

Male, age 31. Right lung normal, left lung consolidated, upper parts. General symptoms, moderate.

Present weight, 116 lb. Heaviest weight, 128 lb. Present weight less than former weight by 12 lb. Time elapsed since first loss of weight, 1,460 days, same time as first hæmoptysis. Cause of first loss of weight assigned—hæmoptysis. Loss of weight had not been treated with either oil or emulsion.

Time elapsed since first cough began, 1,460 days; it was accompanied by first hæmoptysis. Cause of first cough assigned—*nil*. First cough began just before loss of weight. First cough began before (or at the same time as) first hæmoptysis. Present cough began at the same time as first cough.

Time elapsed since first hæmoptysis, 1,460 days, 1,458 days before last hæmoptysis, 1,108 days before first profuse hæmoptysis. The blood of first hæmoptysis was florid. The quantity of blood in first hæmoptysis was over ℥ss. in twenty-four hours. Cause of first hæmoptysis assigned—*nil*. First hæmoptysis occurred after heaviest weight. First hæmoptysis occurred at the same time as first loss of weight. Hæmoptysis had recurred. Time elapsed since last hæmoptysis, two days, 250 days after first profuse hæmoptysis, 1,458 days after first hæmoptysis and first loss of weight. The blood of last hæmoptysis was florid. The quantity of blood in last hæmoptysis was over ℥ x. in twenty-four hours. Last hæmoptysis occurred after first loss of weight, 1,458 days.

The father was consumptive. The mother was consumptive. One or more brothers were consumptive.

COMMENTARY.—Without assignable cause, hæmoptysis to the extent of ℥ss. in twenty-four hours occurred with cough 1,460 days ago, and was immediately followed by loss of weight. Cough had continued ever since.

1,108 days after the first hæmorrhage a second and much more profuse bleeding occurred, and 230 days later a third and very profuse hæmoptysis took place. The only lung disease detectable by physical examination was consolidation of the left upper lobe. The total loss of weight was 12 lb. in 1,460 days. Consumption existed in both parents, and also in collaterals.

Seeing that the hæmorrhage occurred without any external cause, that 1,460 days had elapsed since the first bleeding, and 252 days since the last, and that no other lung disease than consolidation existed, it would be irrational to consider the constitutional decline as due to this amount of disease, and therefore irrational to look upon the hæmorrhage as the cause of both the lung disease and loss of weight. If the statement that no loss of weight had occurred up to the date of first hæmoptysis were correct, we should be without palpable evidence of constitutional decline as a cause of lung disease prior to the hæmorrhage; but the strong consumptive history in the family, and the absence of any external cause for the hæmorrhage, make it most probable that some hereditary defect existed. And as it appeared afterwards that the heaviest weight by weighing, was taken 2 years before the first hæmoptysis, it is open to question whether he had not unconsciously lost weight in that time. He was weighed at the hospital at the time of the second hæmoptysis, 250 days before he was weighed again, 2 days after the last bleeding, and on both of these occasions his weight was the same, but as he quickly gained 4 lb. under treatment, it showed that this weight had been a morbidly low one. Although, therefore, the case is obscure, there is no clear evidence that the hæmorrhage caused the lung disease.

Case 33, J. D.

Male, age 23. Right lung consolidated, left lung excavated, upper parts. General symptoms, severe.

Present weight, 147 lb. Heaviest weight, 174 lb. Present weight less than former weight by 27 lb. Time elapsed since first loss of weight, 617 days, same time as first hæmoptysis, 225 days after first cough. Cause of first loss of weight assigned—over-work and hæmoptysis. First loss of weight had been treated with oil and emulsion.

Time elapsed since first cough began, 842 days, 225 days before first hæmoptysis and first loss of weight. Cause of first cough assigned—cold. First cough began before first loss of weight, 225 days. First cough began before first hæmoptysis, 225 days. Present cough began at the same time and from the same cause as first cough.

Time elapsed since first hæmoptysis, 617 days, twenty-eight days before last hæmoptysis. The blood of first hæmoptysis was florid. The quantity of blood in first hæmoptysis was over ℥x. in twenty-four hours. Cause of first hæmoptysis assigned — cretaceous expectoration. First hæmoptysis occurred at the same time as first loss of weight. Hæmoptysis had recurred. Time elapsed since last hæmoptysis, 589 days, twenty-eight days after first hæmoptysis. The blood of last hæmoptysis was florid. The quantity of blood in last hæmoptysis was over ℥ss. in twenty-four hours. Last hæmoptysis occurred after first loss of weight.

One or more brothers, and one or more sisters were consumptive.

COMMENTARY.—He left the police force in 1868, 1,207 days ago, on account of difficulty of breathing on foggy nights, and in the winter of 1869, 842 days ago, cough and expectoration began. In 1870, when overworked and exhausted, he coughed up a lump of cretaceous matter. At this time, 628 days ago, he weighed 12 st. 6 lb. (174 lb.), and never weighed more.

Eleven days after the cretaceous expectoration, while coughing, he "broke a blood-vessel, and expectorated half a gallon of florid blood rapidly, and fell down fainting." After this he continued to expectorate blood for a month, gradually less and less, and has never seen any since. He was treated at the Royal Hospital, and took oil, which he has taken more or less for two years. In April, 1871 (after hæmoptysis), he was treated at the Royal Hospital again and took emulsion and gained weight, but never regained his heaviest weight. He dates loss of weight from July, 1870, about the time of hæmoptysis. The consolidation signs on right side extended over the mesial line of manubrium, as though from bronchial glands.

The disease appears to have been old and cured, (probably disease of bronchial glands in childhood, followed by cretaceous deposit), re-excited by exposure and overwork, with disintegration and dislodgment of cretaceous deposit, leading to hæmoptysis and extension of disease. This accounts for cavity in left side. In June, 1872, after note of case, he had diarrhœa, and lost weight. A seton was put in over cavity by my order, and kept open twelve weeks; by that time all active symptoms had subsided, but he had lost weight down to 10 st. (7 lb. loss since first note). He was, however, greatly improved on the whole, and started on a sea-voyage.

Although first loss of weight accompanied first hæmoptysis, the fact that cough had existed 225 days, and cretaceous expectoration three days before hæmoptysis, make it irrational to regard the hæmorrhage as the cause of the lung disease.

CASE 42, R. C.

Male, age 41. Right lung consolidated and softening, left lung normal, upper parts. General symptoms moderate.

Present weight, 143 lb. Heaviest weight, 159 lb. Present weight less than heaviest weight by 16 lb. Time elapsed since first loss of weight, 504 days (same time as first hæmoptysis). Cause of first loss of weight assigned—hæmoptysis from overlifting. Loss of weight had not been treated with either oil or emulsion.

Time elapsed since first cough began, 504 days, same time as first hæmoptysis and first loss of weight. Cause of first cough assigned—overlifting. First cough began just before loss of weight, he coughed and brought up blood, and then lost weight. First cough began at the same time as first hæmoptysis. Present cough began at the same time, and was assigned to same cause as first cough.

Time elapsed since first hæmoptysis, 504 days. The blood of first hæmoptysis was florid. The quantity of blood in first hæmoptysis was over ℥ss. in twenty-four hours. The cause of first hæmoptysis assigned was cough from overlifting. First hæmoptysis occurred at the same time as first loss of weight, *i. e.*, loss of weight dated from coughing up the blood. Hæmoptysis had recurred. Time elapsed since last hæmoptysis, 84 days, 420 days after hæmoptysis. The blood of last hæmoptysis was florid. The quantity of blood in last hæmoptysis was over ℥ss. in twenty-four hours.

Family history free from consumption.

Commentary.—He was certain he was in usual health and usual weight, 159 lb., 504 days ago, when he overlifted, coughed, and "broke a blood-vessel," and he had never been well since. In six weeks' treatment with oil and emulsion at the Royal Hospital, he gained six pounds, but was still ten pounds below his heaviest weight. Sixteen pounds weight had been lost in 504 days, commencing with first hæmoptysis and first cough. The cough is tabulated as beginning before hæmoptysis, because it was stated to have *caused* the hæmorrhage, but as no appreciable interval occurred between the two—the cough coming on, and hæmoptysis immediately following—it is quite possible that internal bleeding was the cause of the cough instead of *vice versa*.

The hæmoptysis was over ℥ss. in twenty-four hours, and was assigned to cough produced by overlifting. Hæmoptysis had recurred; the last attack being over ℥ss. in twenty-four hours, 84 days ago. There was no consumption in the family, and all illness apparently dated from the accident—the case is, therefore, fairly open to the opinion that the *lung disease was set up by the hæmorrhage*—although that conclusion is not inevitable.

Case 43, C. T.

Male, age 28. Right lung excavated, left consolidated, upper parts. General symptoms, moderate.

Present weight, 122 lb. Heaviest weight, 137 lb. (exceptional), average heaviest weight, 129 lb. Present weight less than average heaviest weight by 7 lb., less than exceptional heaviest weight by 15 lb. Heaviest weight occurred on voyage to Australia, after first hæmoptysis and first loss of weight, but was evanescent, and may be considered exceptional, the average weight being the better guide of normal weight. Time elapsed since first loss of weight, 365 days, same time as first hæmoptysis, 365 days after first and present cough began, 337 days before last hæmoptysis. Cause of first loss of weight assigned—cough and debility. Loss of weight was treated with a voyage to Melbourne, during which he gained up to 8lb. *over average* weight.

Time elapsed since first cough began, 730 days, 365 days before first hæmoptysis and first loss of weight. Cause of first cough assigned—de-

bility. First cough began before first loss of weight, 365 days. First cough began before first hæmoptysis, 365 days. Present cough was in all respects the same as first cough.

Time elapsed since first hæmoptysis, 365 days. The blood in first hæmoptysis was in streaks. The quantity of blood in first hæmoptysis was under 3ss. in twenty-four hours. Cause of first hæmoptysis assigned—cough. First hæmoptysis occurred before heaviest weight (exceptional heaviest weight). First hæmoptysis occurred after average weight (average heaviest weight). First hæmoptysis occurred at the same time as first loss of weight. Hæmoptysis had recurred. Time elapsed since last hæmoptysis, 28 days. The blood of last hæmoptysis was florid. The quantity of blood in last hæmoptysis was over 3ss. in twenty-four hours. Last hæmoptysis occurred after heaviest weight. Last hæmoptysis occurred after first loss of weight, 337 days.

Family history free from consumption.

COMMENTARY.—He suffered from debility three or four years, 1,460 days. Cough had lasted two years, 730 days, but he maintained an average weight of 9 st. 3lb. 129 lb., which was his usual healthy weight till one year ago (365 days), when he began to spit streaks of blood in his sputa, and to lose flesh. He then took a voyage to Australia (Melbourne), and on arrival found his weight 9 st. 11 lb., being 8 lb. more than he weighed before first loss. He could not maintain his weight, and eight months afterwards found he weighed 15 lb. less weight, 122 lb., when case was taken. During this eight months he had several times had returns of hæmoptysis, not in streaks, not profuse, but similar to the last hæmoptysis, florid blood over 3ss. in twenty-four hours. Right lung, upper part, signs of old cavity and chest wall depressed. Left upper lobe consolidated.

Under oil and emulsion he made great improvement, and gained weight up to 126 lb., only 3 lb. less than average weight.

In this case the hæmoptysis was evidently an accompanying symptom in the course of advancing lung disease ; there was no reason to look upon it as the cause of that disease.*

CASE 34, W. P.

Male, age 29. Right lung softening, left lung consolidated, upper parts. Catarrhal signs general through both lungs. General symptoms, moderate.

Present weight, 126 lb. Heaviest weight, 133 lb. Present weight less than heaviest weight by 7 lb. Time elapsed since first loss of weight, 140 days, same time as first hæmoptysis, 140 days after present cough began, 3,510 days after first cough began. Cause of first loss of weight

* For further commentary, see two cases in which heaviest weight had been entirely regained; also see two cases in which first hæmoptysis preceded heaviest weight.

assigned—hæmoptysis. Loss of weight had not been treated with either oil or emulsion.

Time elapsed since first cough began, 3,650 days, 3,370 days before present cough began. Cause of first cough assigned—*nil*. First cough began before first loss of weight. First cough began before first hæmoptysis.

Time elapsed since present cough began, 280 days, 140 days before first hæmoptysis, 3,370 days after first cough began. Cause of present cough assigned—cold. Present cough began before first loss of weight. Present cough began before first hæmoptysis.

Time elapsed since first hæmoptysis 140 days, same time as first loss of weight. The blood of first hæmoptysis was florid, and also mixed with sputa (flesh-coloured sputa). The quantity of blood in first hæmoptysis was over ℥ss. in twenty-four hours. Cause of first hæmoptysis assigned —cough. First hæmoptysis occurred at the same time as first loss of weight, *i. e.*, first loss of weight dated from first hæmoptysis. Hæmoptysis had not recurred.

The family history was free from consumption.

Commentary.—At the time of first hæmoptysis, 140 days ago, no loss of flesh had occurred. Loss of weight, disintegration of lung and hæmoptysis began coetaneously. A seton was put in by my order over the disintegrating part, and the case did well. But as cough had existed more or less for 3,510 days before this, and there was history of fresh catarrh and renewed cough for 140 days before hæmoptysis, it would be irrational to look to the hæmoptysis as the cause of the lung disease.

Case 53, J. E.

Male, age 46. Right lung normal, left consolidated, upper parts. General symptoms moderate.

Present weight, 117 lb. Heaviest weight, 136 lb. Present weight less than heaviest weight by 19 lb. Time elapsed since first loss of weight, 3,285 days, same time as first hæmoptysis, 4,015 days after first cough and present cough ; 3,117 days before last hæmoptysis. Cause of first loss of weight assigned—hæmoptysis. Loss of weight had not been treated either with oil or emulsion.

Time elapsed since first cough began, 7,300 days. Cause of first cough assigned—*nil*. First cough began before first loss of weight, 4,015 days. First cough began before first hæmoptysis, 4,015 days. Present cough was in all respects the same as first cough, *i. e.*, as to time and cause.

Time elapsed since first hæmoptysis, 3,285 days (4,015 days after first and present cough began. The blood of first hæmoptysis was florid. The quantity of blood in first hæmoptysis was over ℥x. in twenty-four hours. The cause of first hæmoptysis assigned was cough. First hæmoptysis occurred at the same time as first loss of weight (if hæmoptysis caused first loss of weight, it must have preceded it, although fractionally). Hæmoptysis had recurred.

Time elapsed since last hæmoptysis, 168 days, 3,117 days after first hæmoptysis. The blood of last hæmoptysis was mixed with sputa (flesh-coloured

sputa). The quantity of blood in last hæmoptysis was under 3ss. in twenty-four hours. Last hæmoptysis occurred after first loss of weight, 3,117 days.

The family history was free from consumption.

COMMENTARY.—Twenty-five years ago he weighed 9 st. 10lb. (136 lb.), and did not lose from this till nine years ago (3,285 days). He had had cough some twenty years or more, and nine years ago while coughing "broke a blood-vessel," and spat a pint of blood in forty-eight hours. He lost weight then, and has averaged about 8 st. 13 lb. (125 lb.) since then, till one year ago when he began to lose more perceptibly, and has lost 8 lb. since that time; present weight 117 lb. Ever since first hæmoptysis he has spat sanguinolent sputa, till six months ago (168 days). Since then no blood. Left upper lobe dull, with wheezing cough sounds towards sternal end (probably from diseased bronchial glands). The existence of more or less cough for 4,015 days before the first hæmoptysis and the general order of events in this case make it irrational to refer the lung disease to the hæmorrhage as a cause.

CASE 58, A. B.

Male, age 22. Right lung softening, left lung excavated, upper parts. General symptoms, extreme.

Present weight, 99 lb. Heaviest weight, 118 lb. Present weight less than heaviest weight by 19 lb. Time elapsed since first loss of weight, 1,095 days, same time as first hæmoptysis, same time as first cough, same time as present cough, 1,088 days before last hæmoptysis. Cause of first loss of weight assigned—*nil*. Loss of weight had not been treated with either oil or emulsion.

Time elapsed since first cough began, 1,095 days. Cause of first cough assigned—*nil*. First cough began just before first loss of weight. First cough began just before first hæmoptysis. Cough was said to cause hæmoptysis, and therefore preceded it by a fraction of time. Present cough began at same time as first cough.

Time elapsed since first hæmoptysis, 1,095 days. The blood of first hæmoptysis was florid. The quantity of blood in first hæmoptysis was over ʒx. in twenty-four hours. Cause of first hæmoptysis assigned—cough. First hæmoptysis occurred at the same time as first loss of weight. Hæmoptysis had recurred. Time elapsed since last hæmoptysis, 7 days. The blood of last hæmoptysis was mixed with sputa (flesh-coloured sputa). The quantity of blood in last hæmoptysis was under 3ss. Last hæmoptysis occurred after first loss of weight, 1,088 days.

One or more brothers were consumptive.

COMMENTARY.—It was just before first hæmoptysis that he weighed heaviest weight known, 118 lb., and he was certain no loss of weight had taken place up to that time, but he had been growing, and was only about 19 years old.

One or more brothers were consumptive, and as the break-down, con-

sisting in cough, hæmoptysis, and loss of weight without apparent cause, occurred just about the age of completion of growth, it is most probable that the symptoms were simply coetaneous indications of the hereditary disease assuming an active stage. It is, however, open to discussion, whether the hæmorrhage caused the subsequent changes in the lungs. The first hæmoptysis was over ℥ss. in twenty-four hours.

Case 60, T. P.

Male, age 22. Right lung softening, left lung excavated, upper parts. General symptoms, severe.

Present weight, 130 lb. Heaviest weight, 150 lb. Present weight less than heaviest weight by 20 lb. Time elapsed since first loss of weight, 168 days, same time as first hæmoptysis, 197 days after first cough, 161 days before last hæmoptysis. Cause of first loss of weight assigned—hæmoptysis. Loss of weight had been treated with oil.

Time elapsed since first cough, 365 days. Cause of first cough assigned—*nil*. First cough began before first loss of weight, 197 days. First cough began before first hæmoptysis, 197 days. Present cough began at the same time as first cough.

Time elapsed since first hæmoptysis, 168 days. The blood of first hæmoptysis was florid. The quantity of blood in first hæmoptysis was over ℥x. in twenty-four hours. The cause of first hæmoptysis assigned was cough. First hæmoptysis occurred at the same time as first loss of weight. Hæmoptysis had recurred. Time elapsed since last hæmoptysis, 7 days, 161 days after first hæmoptysis. The blood of last hæmoptysis was florid. The quantity of blood in last hæmoptysis was over ℥ss. in twenty-four hours. Last hæmoptysis occurred after first loss of weight, 161 days.

The father was consumptive.

Commentary.—Heaviest weight known, 150 lb., was just before hæmoptysis; he never weighed before, but declared there had been no loss till hæmoptysis occurred. The loss was rapid after hæmoptysis. Had cough 197 days before hæmoptysis. Then, 168 days ago, coughed up florid blood profusely for five days, and had continued to bring up smaller quantities almost daily till one week ago. Took oil at first, but not lately.

In this case, although first loss of weight occurred at the same time as first hæmoptysis, cough had existed during 197 days previously, and the family history was consumptive on the father's side. It would therefore be irrational to consider the hæmorrhage as the cause of the lung disease.

Case 93, A. B.

Male, age 28. Right lung consolidated, left softening, upper lobes. General symptoms, severe.

Present weight, 112 lb. Heaviest weight, 149 lb. Present weight less than heaviest weight by 37 lb. Time elapsed since first loss of weight,

365 days, just after or at same time as first hæmoptysis, same time as or just after first cough. Cause of first loss of weight assigned—hæmoptysis. Loss of weight had been treated with oil and emulsion.

Time elapsed since first cough began, 365 days. Cause of first cough assigned—hæmoptysis, cough accompanied and was said to cause hæmoptysis. First cough began before, or at same time as, first loss of weight. First cough began before, or at the same time as, first hæmoptysis. Present cough began at the same time, and was due to same cause as first cough.

Time elapsed since first hæmoptysis, 365 days. The blood of first hæmoptysis was florid. The quantity of blood in first hæmoptysis was over ℥x. in twenty-four hours. The cause of first hæmoptysis assigned was over-lifting a heavy weight. First hæmoptysis occurred at the same time as first loss of weight (if it was the cause of loss of weight, it must have preceded it, although by a fraction). Hæmoptysis had recurred.

Time elapsed since last hæmoptysis, 56 days, 309 days after first hæmoptysis. The blood of last hæmoptysis was florid. The quantity of last hæmoptysis was over ℥ss. in twenty-four hours. Last hæmoptysis occurred after first loss of weight, 309 days.

The family history was free from consumption.

Commentary.—The heaviest weight known was three months before first hæmoptysis, and he declared there was no loss of weight or sign of illness up to the moment of his lifting a heavy weight, when he began to cough up blood profusely ; the first hæmoptysis was said to be 3 pints of florid blood in twenty-four hours. From that time up to two months ago had had frequent smaller bleedings. Right lung very slightly consolidated ; left dull and chronically disintegrating, not very active. After six weeks oil and emulsion he gained 5 lb., making weight 117 lb., 32 lb. less than heaviest weight.

In this case, no consumption was known in the family, and everything appeared to date from an accidental strain in lifting a very heavy weight, which was immediately followed by extremely profuse hæmoptysis. An enormous loss of weight had occurred, 37 lb. (one-quarter of his total heaviest weight) in 365 days, in spite of treatment, and smaller bleedings had frequently occurred.

One lung was consolidated and the other slowly softening. Under oil and emulsion he gained 5 lb. but was still 32 lb. short of his normal weight.

This case approaches more nearly than any other out of the 100 here analysed to the description of Niemeyer of "galloping consumption, of which bronchial hæmorrhage is the immediate cause," but even here there is a history of 365 days, and the patient was still alive and beginning to gain flesh after his great loss, whereas Niemeyer speaks of such cases perishing in a few months.

This is the only case out of the 100 in which the most rational conclusion appears to be that the lung disease was caused by the hæmorrhage. (See p. 26.)

Two Cases in which First Hæmoptysis occurred before the period of Heaviest Weight. (Col. 48, Table 1.)

For full details see Table 6 (Ten Cases in which First Loss of Weight and First Hæmoptysis occurred coetaneously).

CASE 43, C. T.

(See also p. 43.)

COMMENTARY.—The increase of weight over average weight during sea voyage is accounted for by the fact that he had suffered from "debility" 730 days before cough began, and then from cough 365 days more before he lost weight and spat blood. So that having been for so long in chronically low health, the rest and invigoration of a sea voyage might easily give an exceptional start to his appetite and nutrition.

It will be seen, also, that his first hæmoptysis was only in streaks, and it came on with the advance of a long-standing pulmonary catarrh of 365 days duration. The subsequent larger hæmoptysis (twenty-eight days ago, over 3ss.) occurred coincidently with advancing disease.

Even in the excavation stage of his disease he showed great power of recovering weight, and under oil and emulsion regained his average weight within 3 lb.

CASE 88, F. T.

(Commentary given at p. 37.)

No reason existed for considering the hæmorrhage to be the cause of the lung disease.

Two Cases in which First Loss of Weight occurred at the same time as Last Hæmoptysis. (Col. 67, Table 1.)

CASE 7, T. P.

(See Table 4, eight cases in which first hæmoptysis occurred before first loss of weight.)

In this case first hæmoptysis occurred 1,348 days before the first loss of weight, and was coincident with the beginning of first cough, which cough had continued to the present time.

First loss of weight began 112 days ago, coincidently with last hæmoptysis, 10 lb. having been lost in that time.

The hæmoptysis was assigned as the cause of the loss of weight. The quantity of blood in last hæmoptysis was over ℥ss. in twenty-four hours. In the first hæmoptysis the quantity was over 3ss., and lasted daily for a week. In both cases the blood was florid, and no hæmoptysis had occurred between the first and last, an interval of 1,348 days, but cough had continued throughout. The total time elapsed since first cough and first hæmoptysis was 1,460 days, and no cause was assigned for first cough and first hæmoptysis.

Under treatment with oil and emulsion, the heaviest weight known (121 lb.) was regained within 1 lb.

The right lung was softened, the left consolidated, and the general symptoms were moderate.

There was no consumptive history.

On the whole it appears most probable that this case was one of lung disintegration of catarrhal origin. Loss of weight and last hæmoptysis occurring only as a coincidence of advancing disease. (See Commentary at p. 30.)

CASE 104, H. F.

Male, age 32. Right lung excavated, left excavated. General symptoms, extreme.

The total loss of weight was 16 lb., which had occurred in 252 days, beginning coincidentally with last hæmoptysis, which was assigned as its cause.

The first cough had preceded first hæmoptysis by 169 days, and although hæmorrhage was profuse, 1,039 days elapsed before any loss of weight occurred. The present cough had preceded last hæmoptysis and first loss of weight by fifty-six days, and was attributed to cold.

The last hæmoptysis was profuse (over ℥x. in twenty-four hours), and the blood was florid.

The family history was free from consumption.

(See full details at p. 38.)

COMMENTARY.—On the whole it appears probable that this case was one of catarrhal disintegration of lung, not of tuberculosis proper.

It is a marked case. No lung symptoms followed upon first hæmoptysis, although profuse, in 1868, the break down dating from a second and more severe cold in December, 1872, followed in February, 1873, by profuse hæmoptysis. The hæmoptysis in this case was sufficiently profuse and continued to account for loss of strength and of weight, and as the severe cold and cough had lasted fifty-six days before hæmoptysis, there were sufficient evidences that pulmonary mischief of some kind preceded this hæmoptysis.

KEY TO TABLES.

TABLE 1. *Frontispiece.*
General Table—Analysis of 100 cases complete.

TABLE 2. *Columns 5 to 10.* (See also Tables 9 to 18.)
Shows the condition of the lungs in 100 cases (classified), and the per centage in which each class of condition occurred.

TABLE 3. *Column 28.*
Analysis of twelve cases in which first cough began at the same time as first hæmoptysis.

TABLE 4. *Column 50.* (See also Tables 7 and 8.)
Eight cases in which first hæmoptysis occurred before first loss of weight; average, 853 days.

TABLE 5. *Column 51.* (See also Tables 7 and 8.)
Eighty-two cases in which first hæmoptysis occurred after first loss of weight; average, 469·7804 days.

TABLE 6. *Column 52.* (See also Tables 7 and 8.)
Ten cases in which first hæmoptysis occurred at the same time as first loss of weight.

TABLE 7. (See also Tables 4, 5, 6.)
Table showing the relation in point of time between first hæmoptysis and first loss of weight, in three groups of cases analysed in Tables 4, 5, 6.

TABLE 8. (See also Tables 4, 5, 6.)
Table showing the total loss of weight, and the rate of loss per annum in three groups of cases analysed in Tables 4, 5, 6.

TABLE 9. *Columns 5, 6, 7, 8, 9, 10.* (See also Table 2.)
Table showing the total loss of weight; the time in which the loss had occurred; and the rate at which it had occurred in different stages of disease of the lungs.

CASES ANALYSED IN NINE GROUPS.

(See also Tables 2 and 9.)

TABLE 10. Both lungs excavated in eleven cases analysed.
TABLE 11. Both lungs softened in twelve cases analysed.
TABLE 12. Both lungs consolidated in eight cases analysed.
TABLE 13. Right lung excavated, left normal, in two cases analysed.
TABLE 14. Left lung excavated, right normal, in one case analysed.
TABLE 15. Right lung softened, left normal, in ten cases analysed.
TABLE 16. Left lung softened, right normal, in eight cases analysed.
TABLE 17. Right lung consolidated, left normal, in three cases analysed.
TABLE 18. Left lung consolidated, right normal, in three cases analysed.

TABLE 2.

Condition of Lungs in 100 *Cases of Hæmoptysis* (*in all cases the upper lobes affected*). (Cols. 5, 6, 7, 8, 9, 10, Table 1.)

	Per cent.	
Both lungs excavated (upper parts) ...	11	See table of these cases, showing the rate at which loss of weight occurred. They have been selected for this analysis as representing the most typical complications.
Both lungs softened „ ...	12	
Both lungs consolidated „ ...	8	
Right lung excavated, left normal ...	2	
Left lung excavated, right normal ...	1	
Right lung softened, left normal ...	10	In one of these cases there was considerable bronchitis in left lung.
Left lung softened, right normal ...	8	
Right lung consolidated, left normal ...	3	The left lung in each of these cases was catarrhal, but otherwise normal.
Left lung consolidated, right normal...	3	
Right lung excavated, left softened ...	9	These cases are not included in Table of rate of loss of weight in different stages of disease of the lung.
Left lung excavated, right softened ...	10	
Right lung excavated, left consolidated	4	
Left lung excavated, right consolidated	8	
Right lung softened, left consolidated	10	
Left lung softened, right consolidated	9	

	Per cent.
In eleven of above cases physical signs were entered under three or more headings, as follows :—	
Right lung consolidated, left consolidated and excavated	1
Right lung consolidated, left softened and excavated	1
Right lung consolidated and softened, left consolidated and softened	3
Right lung softened and excavated, left softened	1
Right lung softened, left softened and excavated	1
Right lung consolidated and softened, left softened	2
Right lung consolidated, left consolidated and softened	2
Catarrhal signs in both lungs, obscuring other signs (Case 13) ...	1
Catarrhal signs, complicating other signs detailed above	14

See also Tables 9 to 18.

TABLE 3.

Twelve Cases in which First Cough began at same time as First Hæmoptysis. (Col. 28, Table 1.)

Number of Case in General Table.	When First Cough and First Hæmoptysis began.			Total Loss of Weight.	Amount and Character of First Hæmoptysis.	Cause of Loss of Weight Assigned.	Cause of First Cough Assigned.	Cause of Hæmoptysis Assigned.
	Before First Loss of Weight.	Same Time as First Loss of Weight.	After First Loss of Weight.					
	Days.		Days.	lbs.				
J. P. 7.	1,348	—	—	10	Florid, over ʒss. daily for 7 days.	Last hæmoptysis (florid, over ʒss.)	Nil.	Nil.
R. W. 15.	365	—	—	36	Florid, over ʒss. in 24 hrs.	Hæmoptysis.§	Nil.	Cough.
A. G. 17.	—	1	—	17	Florid, over ʒss. in 24 hrs.	Hæmoptysis.	Nil.	Nil.
A. K. 21.	562	—	—	15	Florid, over ʒss. in 24 hrs.	Cough.‖	Nil.	Cough.
E. W. O. 27.	—	—	28	4	Florid, over ʒss. in 24 hrs.	Pleurisy.	Hæmoptysis.††	Cough.††
R. C. 42.	—	1	—	16	Florid, over ʒss. in 24 hrs.	Hæmoptysis.¶	Overlifting.	Over-lifting.
S. C. 64.	—	—	730	49	Florid, over ʒx. in 24 hrs.	Overgrowth.	Nil.	Over-exertion while weak from over-growth.
J. B. 67.	365	—	—	13	Small clots, under ʒss. in 24 hours.‡	Cough.	Nil.	Cough.
J. S. M. 82.	—	—	365	21	Florid, over ʒss. in 24 hrs.	Late hours and debility.	Late hours and debility.	Late hours and debility.
W. T. O. 84.	—	—	534	19	Florid, over ʒss. in 24 hrs.	Nil.	Cold.	Cough and illness.
F. T. 88.	1,460*	—	—	14	Small clots, under ʒss. in 24 hours.	Nil.	Nil.	Cough.
A. B. 93.	—	1	—	37	Florid, over ʒx. in 24 hrs.	Hæmoptysis.**	Accompanied Hæmoptysis.	Over-lifting.
12 cases.	5 cases.	3 cases.	4 cases.†	251 lbs., average 20·846.	Florid ... 10 Small Clots 2 Over ʒx. ... 2 Over ʒss. ... 6 Over ʒss. ... 2 Under ʒss. ... 2			

* First hæmoptysis occurred with cough at 17 years old, before completing growth. No chest symptoms recurred till 21 years old.

† In these cases, loss of weight had come before all other symptoms.

‡ After first loss of weight profuse hæmoptysis occurred.

§ 365 days elapsed before first hæmoptysis and first loss of weight, so there is no evidence that one caused the other.

‖ Hæmoptysis had not recurred, but cough had continued.

¶ While in usual health and weight, over-lifted, and broke blood-vessel; never well since. Hæmoptysis had recurred.

** While in usual health, lifted a heavy weight, and coughed up blood, 3 pints in 24 hours; frequent returns of smaller bleedings.

†† Cough said to be caused by hæmoptysis, and hæmoptysis to be caused by cough!

TABLE 4.

Analysis of Eight Cases in which First Hæmoptysis occurred before First Loss of Weight. (Col. 50, Table 1.)

(For further details, see Table 1.)

Number of Case in Table 1.	Column 14. — Present Weight.	Column 15. — Heaviest Weight.	Column 19. — Time elapsed since Loss of Weight began.	Column 38. — Time elapsed since First Hæmoptysis.	Time elapsed between First Hæmoptysis and First Loss of Weight.	Loss of Weight in the time elapsed since First Loss.	Time elapsed between First Cough and First Loss of Weight.	Time elapsed between First Cough and First Loss of Weight.
					Hæmoptysis before Loss of Weight.		First Cough before Loss of Weight.	First Loss of Weight before First Cough.
7	111 lbs.	121 lbs.	112 days.	1,460 days.	1,348 days.	10 lbs.	1,348 days.	
15	110 ,,	146 ,,	730 ,,	1,095 ,,	365 ,,	36 ,,	365 ,,	
21	121 ,,	136 ,,	168 ,,	730 ,,	562 ,,	15 ,,	562 ,,	
37	143 ,,	157 ,,	168 ,,	1,825 ,,	1,657 ,,	14 ,,	7,132 ,,	
62	111 ,,	131 ,,	252 ,,	280 ,,	28 ,,	20 ,,	—	56 days.
67	143 ,,	156 ,,	1,095 ,,	1,460 ,,	365 ,,	13 ,,	365 ,,	
88	140 ,,	154 ,,	1,825 ,,	3,285 ,,	1,460 ,,	14 ,,	1,460 ,,	
104	110 ,,	126 ,,	252 ,,	1,291 ,,	1,039 ,,	16 ,,	1,208 ,,	
Totals { Eight cases	989 lbs.	1,127 ,,	4,602 days.	11,426 days.	6,824 days.	138 lbs.	12,440 days.	56 days.
Averages	Eight cases 123·625 lbs.	Eight cases 140·875 lbs.	Eight cases 575·25 days.	Eight cases 1,428·25 days.	Eight cases 853 days.	Eight cases 17·25 lbs.*	Seven cases 1,777·142 days.	One case 56 days.
Minimum	110 lbs.	121 lbs.	112 days.	280 days.	28 days.	10 lbs.	365·0 days.	56 days.
Maximum	143 lbs.	157 lbs.	1,825 days.	3,285 days.	1,657 days.	36 lbs.	7,132·0 days.	56 days.

* Average loss in eight cases, 17·25 lbs.; in days, 575·25; about 10·95 lbs. per year.

TABLE 5.

Analysis of Eighty-two Cases in which First Hæmoptysis occurred after First Loss of Weight. (Col. 51, Table 1.)

(For further details see Table 1.)

Number of Case in Table 1.	Column 14. — Present Weight.	Column 15. — Heaviest Weight.	Column 19. — Time elapsed since Loss of Weight began.	Column 38. — Time elapsed since First Hæmoptysis.	Time elapsed between First Loss of Weight and First Hæmoptysis.
	lbs.	lbs.	Days.	Days.	Days.
102	84	114	1179	1095	84
103	130	140	365	196	169
105	130	154	730	477	253
106	122	131	365	140	225
107	125	136	730	421	309
1	145	193	814	730	84
3	124	128	1825	140	1685
4	125	142	504	112	392
5	139	148	42	14	28
8	120	140	364	308	56
9	120	144	2920	2190	730
10	128	140	730	616	114
11	122	140	365	358	7
13	135	154	730	168	562
14	129	158	224	84	140
16	121	131	42	2	40
18	117	131	730	365	365
19	107	140 (growing).	1825	168	1657
20	126	131	730	504	226
22	136	196	730	365	365
23	119	182	2555	2190	365
24	112	118	730	56	674
25	118	126	420	336	84
26	135	153	365	84	281
27	101	105	224	196	28
28	111	118	84	56	28
29	134	148	1516	1460	56
30	122	130	1628	1460	168
32	122	182	2555	365	2190
35	116	140	1516	730	786
36	131	145	1825	7	1818
38	103	131	898	561	337
39	100	122	730	365	365
40	107	145	252	21	231
41	152	172	112	28	84
44	132	154	5475	1095	4380
45	142	158	730	365	365
46	130	144	365	7	358
47	154	163	196	168	28
48	99	122	365	7	358
49	143	158	1095	365	730
50	99	117	730	365	365
51	158	177	308	140	168
Car. forward	5,325	6,201	40,618	18,880	21,738

TABLE 5 (*continued*).

Number of Case in Large Table.	Column 14. — Present Weight.	Column 15. — Heaviest Weight.	Column 19. — Time elapsed since Loss of Weight began.	Column 38. — Time elapsed since First Hæmoptysis.	Time elapsed between First Loss of Weight and First Hæmoptysis.
	lbs.	lbs.	Days.	Days.	Days.
Brt. forward	5,325	6,201	40,618	18,880	21,738
52	127	142	84	7	77
56	136	152	280	252	28
57	120	150	842	224	618
59	118	136	365	7	358
61	104	132	1460	533	927
63	116	120	730	449	281
64	119	168	4015	3285	730
65	136	146	504	21	483
66	118	124	224	28	196
68	114	138	730	168	562
70	142	186	1095	14	1081
71	104	126	1460	365	1095
72	106	120	730	280	450
74	125	140	112	84	28
75	123	148	1825	224	1601
76	105	130	1095	617	478
77	122	145	730	504	226
78	102	126	533	35	498
79	105	156	730	365	365
80	132	147	112	70	42
81	124	140	365	1	364
82	119	140	4745	4380	365
83	149	182	1825	224	1601
84	117	136	730	196	534
85	116	149	252	224	28
86	153	162	589	7	582
87	110	112 (grown since).	730	49	681
89	118	130	1460	1403	47
90	130	159	280	2	278
91	111	122	814	730	84
92	113	126	1825	1740	81
94	152	172	140	84	56
95	136	157	168	112	56
96	96	131	3650	3285	365
97	169	175	4015	3565	450
98	115	119	365	56	309
99	114	122	196	7	189
100	129	145	365	140	225
101	91	103 (at 19 years old).	1460	1095	365
Totals.	10,061	11,715	82,248	43,712	38,522
82 cases averages.	122·6951*	142·8658*	1003·0243	533·0731	469·7804
Minimum.	84·0	103·0	42·0	1·0	7·0
Maximum.	169·0	196·0	5475·0	4380·0	4380·0

* Average loss of weight in eighty-two cases, 20·1707 lb., in 1003·0243 days; rate about 7·34 lb. per year.

TABLE 6.

Analysis of Ten Cases in which First Hæmoptysis and First Loss of Weight occurred at the same time. (Col. 52, Table 1.)

Number of Case in Table 1.	Column 14. — Present Weight.	Column 15. — Heaviest Weight.	Columns 19, 38. — Time elapsed since First Hæmoptysis and since First Loss of Weight.	Loss of Weight in Time elapsed since First Loss.	Time elapsed between First Cough and First Loss of Weight and Hæmoptysis. First Cough before First Loss of Weight.	Time elapsed between First Cough and First Loss of Weight and Hæmoptysis. (No appreciable time elapsed).
6	135 lbs.	144 lbs.	168 days.	9 lbs.	1,292 days.	—
17	116 „	128 „	1,460 „	12 „	0	†
33	147 „	174 „	617 „	27 „	225 „	—
34	126 „	133 „	140 „	7 „	3,510 „	—
42	143 „	159 „	504 „	16 „	0	†
43	122 „	129 „	365 „	7 „	365 „	—
53	117 „	136 „	3,285 „	19 „	4,015 „	—
58	99 „	118 „	1,095 „	19 „	0	†
60	130 „	150 „	168 „	20 „	197 „	—
93	112 „	149 „	365 „	37 „	0	†
Ten cases Totals.	Ten cases 1,247 lbs.	Ten cases 1,420 lbs.	Ten cases. 8,167 days.	Ten cases 176 lbs.*	Six cases 9,604 days.	Four cases †
Average ...	124·7 lbs.	142 lbs.	816·7 days.	17·6 lbs.*	1,600·666 days.	†
Minimum	99 lbs.	118 lbs.	140 days.	7 lbs.	197 days.	†
Maximum	147 lbs.	174 lbs.	3,285 days.	37 lbs.	4,015 days.	†

* Average loss in ten cases, 17·6 lbs. in 816·7 days ; about 7·872 lbs. per year.
† Stand in Table as before first loss of weight, but interval was not appreciable.

TABLE 7.

Showing the Relation, in point of time, between First Hæmoptysis and First Loss of Weight, in three groups of cases, analysed in Tables 4, 5, 6.

First Hæmoptysis and First Loss of Weight.

100 Cases Compared.	Time elapsed since First Hæmoptysis.	Time elapsed since First Loss of Weight.
82 cases (Table 1, Col. 51) Loss of Weight began before First Hæmoptysis.	Average 533·0 days. Minimum 1·0 „ Maximum 4380·0 „	Average 1003·0 days. Minimum 42·0 „ Maximum 5475·0 „
10 cases (Col. 52) Loss of Weight began at same time as First Hæmoptysis.	Average 816·7 days. Minimum 140·0 „ Maximum 3285·0 „	Average 816·7 „ Minimum 140·0 „ Maximum 3285·0 „
8 cases (Col. 50) Loss of Weight began after First Hæmoptysis.	Average 1428·0 days. Minimum 280·0 „ Maximum 3285·0 „	Average 5752·0 days. Minimum 112·0 „ Maximum 1825·0 „

The average time elapsed since first hæmoptysis is least in the eighty-two cases (loss of weight before first hæmoptysis).

The average time elapsed since first hæmoptysis is greater in the ten cases (loss of weight same time as first hæmoptysis).

The average time elapsed since first hæmoptysis is greatest in the eight cases (loss of weight after first hæmoptysis). As follows :—

82 cases less than 10 cases by 283·7 days.
82 cases less than 8 cases by 895·0 days.
10 cases less than 8 cases by 611·3 days.

The average time elapsed since first loss of weight is in the reverse order of that of first hæmoptysis, viz. :—

Least in the 8 cases (loss of weight after first hæmoptysis) ;
Greater in the 10 cases (loss of weight same time as first hæmoptysis) ;
Greatest in the 82 cases (loss of weight before first hæmoptysis) ;
As follows :—

8 cases less than 10 cases by 241·5 days.
8 cases less than 82 cases by 427·8 days.
10 cases less than 82 cases by 186·3 days.

TABLE 8.

Showing the Total Loss of Weight, and the Rate of Loss per Annum, in three groups of cases, analysed in Tables 4, 5, 6.

TOTAL LOSS OF WEIGHT, AND RATE OF LOSS.—AVERAGES.

100 Cases Compared.	Averages.	
	Amount Lost in Whole Time of Loss.	Rate of Loss per 365 days.
82 cases (Col. 51) Loss of Weight began before First Hæmoptysis.	About 20 lbs. (20·17) in 1,003 days.	About 7 lbs. per year (7·34).
10 cases (Col. 52) Loss of Weight began at same time as First Hæmoptysis.	About 17 lbs. (17·6) in 816 days.	About 8 lbs. per year (7·87).
8 cases (Col. 50) Loss of Weight began after First Hæmoptysis.	About 17 lbs. (17·25) in 575 days.	About 11 lbs. per year (10·95).

RATE OF LOSS OF WEIGHT.

The rate (average) of loss of weight per 365 days is—
greatest in the 8 cases (loss of weight after first hæmoptysis) ;
less in the 10 cases (loss of weight same time as first hæmoptysis) ;
least in the 82 cases (loss of weight before first hæmoptysis) ;
As follows :—

8 cases rate greater than in 10 cases by 3·08 lbs.
8 „ „ 82 „ 3·61 „
10 „ „ 82 „ 0·53 „

The average total loss was nearly the same in the 8 cases as in the 10, about 3 lbs. more in the 82 cases than in either 8 or 10.

TABLE 9.

Showing the Total Loss of Weight, the Time in which the Loss had occurred, and the Rate at which it had occurred, in different stages of Disease of the Lungs. (See also Table 2.)

Reference to Cases in Table 1.	Number of Cases.	STAGE OF DISEASE. (The most advanced condition is that noted in each case).	Total Loss of Weight. Average.	Total Loss of Weight. Max. and Min.	Time in which Loss of Weight occurred. Average.	Time in which the loss occurred. Max. and Min.	Average Rate of Loss of Weight per 365 days.
			lbs.	lbs.	Days.	Days.	lbs. (about)
Cases 102, 104, 1, 8, 15, 23, 32, 64, 65, 81, 84.	11 cases.	Both lungs excavated.	33·363	Max. 63 Min. 10	1005·727	Max. 4015 Min. 252	12·108
Cases 106, 3, 9, 14, 16, 44, 45, 48, 61, 62, 96, 100.	12 cases.	Both lungs softened.	19·666	Max. 35 Min. 4	1472·750	Max. 5475 Min. 42	4·874
Cases 103, 20, 27, 63, 82, 90, 93, 95.	8 cases.	Both lungs consolidated.	16·5	Max. 37 Min. 4	950·875	Max. 4745 Min. 168	6·332
Cases 21, 26.	2 cases.	Right lung excavated, left normal.	16·5	Max. 18 Min. 15	266·500	Max. 365 Min. 168	22·600
Case 89.	1 case.	Left lung excavated, right lung normal.	12·0	Max. 12 Min. 12	1460·000	Max. 1460 Min. 1460	3·000
Cases 10, 30, 34, 35, 42, 47, 51, 83, 86, 99.	10 cases.	Right lung softened, left lung normal. In one of these ten cases there was considerable bronchitis in left lung (Case 51).	14·5	Max. 33 Min. 7	763·200	Max. 1825 Min. 140	6·934
Cases 6, 11, 49, 52, 56, 67, 72, 94.	8 cases.	Left lung softened, right normal. In three of these eight cases, right lung had catarrhal Rhonchi but no dulness (Cases 6, 11, 49).	15·0	Max. 20 Min. 9	494·875	Max. 1095 Min. 84	11·063
Cases 5, 28, 66.	3 cases.†	Right lung consolidated, left lung normal. In each case the left lung had catarrhal Rhonchi, but no dulness.	7·333	Max. 9 Min. 6	116·666	Max. 224 Min. 42	22·941
Cases 17, 53, 74.*	3 cases.‡	Left lung consolidated, right lung normal.	15·333	Max. 19 Min. 12	1619·000	Max. 3285 Min. 112	3·456
	58 cases.	In all cases the changes described were in the upper lobes.	For further details, see Tables.				

* Case 53. See analysis of ten cases in which first hæmoptysis and first loss of weight were coincident. Cough had preceded hæmoptysis many years. After profuse hæmoptysis, produced by cough, he lost 11 lbs., but remained nearly stationary after that for seven or eight years, and then he lost 8 lbs. in one year. He had expectorated sanguinolent sputa ever since first hæmoptysis. It was diagnosed as a case of probable disease of bronchial glands, the consolidation signs were especially marked at sternal end of left upper.

† The periods of loss of weight in these cases were 42 days, 84 days, 224 days: average 116·666. The rate of loss of weight, therefore, was on an average about 23 lbs. per year.

‡ The periods of loss of weight in these cases were Case 17, 1,460 days; Case 53, 3,285 days; Case 74, 112 days. In Case 17, the loss of weight from 128 lbs. to 116 lbs., occurred between first hæmoptysis and first profuse recurrence of hæmoptysis, a period of 1,108 days. During 252 days since profuse hæmoptysis (over ℥x. in 24 hours) weight remained stationary at 116 lbs., so that loss of weight had really occurred in 1,108 days. If we exclude Case 53, and give 1,108 days instead of 1,460 for Case 17, average 610 days, and take loss in Case 17 as 12 lbs., and loss in Case 74 as 15 lbs., average 13·5 lbs., the rate of loss becomes about 7·61 lbs. per 365 days. This is probably the fairest estimate.

CONDITION OF LUNGS.

TABLE 10.

Both Lungs Excavated, in Eleven Cases.

Number of Case.	Total Loss of Weight.	Time elapsed since Loss of Weight began.	Time elapsed since First Hæmoptysis.	First Hæmoptysis occurred before, at same time, or after First Loss of Weight.
	lbs.	Days.	Days.	
102	30	1,179	1,095	84 days after
104	16	252	1,291	1,039 „ before
1	48	814	730	84 „ after
8	20	364	308	56 „ „
15	36	730	1,095	365 „ before
23	63	2,555	2,190	365 „ after
32	60	2,555	365	2,190 „ „
64	49	4,015	3,285	730 „ „
65	10	504	21	483 „ „
81	16	365	1	364 „ „
84	19	730	196	534 „ „
11 cases	367	11,063	10,577	6,294 { 4,890 after (9 cases). 1,404 before (2 cases).
Average	33·363	1005·727	961·565	{ 543·333 after 702·000 before
Max.	63	4,015	3,285	Maximum 9,190 / Minimum 56 } after
Min.	10	252	1	Maximum 1,039 / Minimum 365 } before

TABLE 11.

Both Lungs Softened, in Twelve Cases.

Number of Case.	Total Loss of Weight.	Time elapsed since Loss of Weight began.	Time elapsed since First Hæmoptysis.	First Hæmoptysis occurred before, at same time, or after First Loss of Weight.
	lbs.	Days.	Days.	
106	9	365	140	225 days after
3	4	1,825	140	1,685 „ „
9	24	2,920	2,190	730 „ „
14	29	224	84	140 „ „
16	10	42	2	40 „ „
44	22	5,475	1,095	4,380 „ „
45	16	730	365	365 „ „
48	23	365	7	358 „ „
61	28	1,460	533	927 „ „
62	20	252	280	28 „ before
96	35	3,650	3,285	365 „ after
100	16	365	140	225 „ „
12 cases	236	17,673.	8,261	9468 { 28 days before (1 case) 9,440 days after (11 cases)
Average	19·666	1472·75	688·416	{ 28 days before (1 case) 858·181 days after (11 cases)
Max.	35	5,475	3,285	Maximum 28 / Minimum 28 } 1 case before
Min.	4	42	2	Maximum 4,380 / Minimum 40 } 11 cases after

TABLE 12.

Both Lungs Consolidated, in Eight Cases.

Number of Case.	Total Loss of Weight.	Time elapsed since Loss of Weight began.	Time elapsed since First Hæmoptysis.	First Hæmoptysis occurred before, at same time, or after First Loss of Weight.
	lbs.	Days.	Days.	
103	10	365	196	169 days after
20	5	730	504	226 ,, ,,
27	4	224	196	28 ,, ,,
63	4	730	449	281 ,, ,,
82	22	4,745	4,380	365 ,, ,,
90	29	280	2	278 ,, ,,
93	37	365	365	At same time as first loss of wt.
95	21	168	112	56 days after
8 cases	132	7,607	6,204	1,403 { At same time as (1 case) / 1,403 after (7 cases)
Average	16·5	950·875	775·5	200·428 days after (7 cases)
Max.	37	4,745	4,380	365 } 7 cases after
Min.	4	168	2	28 }

TABLE 13.

Right Lung Excavated, Left Normal, in Two Cases.

Number of Case.	Total Loss of Weight.	Time elapsed since Loss of Weight began.	Time elapsed since First Hæmoptysis.	First Hæmoptysis occurred before, at same time, or after First Loss of Weight.
	lbs.	Days.	Days.	
21	15	168	730	562 days before
26	18	365	84	281 ,, after
Average	16·5	266·5	407	562 days before. 281 ,, after.

TABLE 14.

Left Lung Excavated, Right Normal, in One Case.

Number of Case.	Total Loss of Weight.	Time elapsed since Loss of Weight began.	Time elapsed since First Hæmoptysis.	First Hæmoptysis occurred before, at same time, or after First Loss of Weight.
	lbs.	Days.	Days.	
89	12	1,460	1,403	47 days after

TABLE 15.

Right Lung Softened, Left Normal, in Ten Cases.

Number of Case.	Total Loss of Weight.	Time elapsed since Loss of Weight began.	Time elapsed since First Hæmoptysis.	First Hæmoptysis occurred before, at same time, or after First Loss of Weight.
	lbs.	Days.	Days.	
10	12	730	616	114 days after
30	8	1,628	1,460	168 ,, ,,
34	7	140	140	At same time as first loss of wt.
35	24	1,516	730	786 days after
42	16	504	504	At same time as first loss of wt.
47	9	196	168	28 days after
*51	19	308	140	168 ,, ,,
83	33	1,825	224	1,601 ,, ,,
86	9	589	7	582 ,, ,,
99	8	196	7	189 ,, ,,
10 cases	145	7,682	3,996	{ 3,636 days after (8 cases) { At the same time as (2 cases)
Average	14·5	763·2	399·6	454·5 days after (8 cases)
Max.	33	1,825	1,460	1,601 } 8 cases after
Min.	7	140	7	28 }

* In Case 51, acute bronchial congestion of left at time of examination, but no dulness.

TABLE 16.

Left Lung Softened, Right Lung Normal, in Eight Cases.

Number of Case.	Total Loss of Weight.	Time elapsed since Loss of Weight began.	Time elapsed since First Hæmoptysis.	First Hæmoptysis occurred before, at same time, or after First Loss of Weight.
	lbs.	Days.	Days.	
6	9	168	168	At same time as first loss of wt.
11	18	365	358	7 days after
49	15	1,095	365	730 ,, ,,
52	15	84	7	77 ,, ,,
56	16	282	252	28 ,, ,,
67	13	1,095	1,460	365 ,, before
72	14	730	280	450 ,, after
94	20	140	84	56 ,, ,,
8 cases	120	3,959	2,974	1713 { 365 days before (1 case) { At same time as (1 case) { 1358 days after (6 cases)
Average	15·0	494·875	371·750	{ 365 days before (1 case) { At same time as (1 case) { 226·333 days after (6 cases)
Max.	20	1,095	1,460	730 } 6 cases after
Min.	9	84	7	7 }

TABLE 17.

*Right Lung Consolidated, Left Normal, in Three Cases.**

Number of Case.	Total Loss of Weight.	Time elapsed since Loss of Weight began.	Time elapsed since First Hæmoptysis.	First Hæmoptysis occurred before, at same time, or after First Loss of Weight.
	lbs.	Days.	Days.	
5	9	42	14	28 days after
28	7	84	56	28 ,, ,,
66	6	224	28	196 ,, ,,
3 cases	22	350	98	252 ,, ,,
Average	7·333	116·666	32·666	84 ,, ,,
Max.	9	224	56	196 ,, ,,
Min.	6	42	14	28 ,, ,,

* In each case the left lung had catarrhal Rhonchi, without dulness.

TABLE 18.

Left Lung Consolidated, Right Lung Normal, in Three Cases.

Number of Case.	Total Loss of Weight.	Time elapsed since Loss of Weight began.	Time elapsed since First Hæmoptysis.	First Hæmoptysis occurred before, at same time, or after First Loss of Weight.
	lbs.	Days.	Days.	
17	12	1,460	1,460	At same time as first loss of wt.
53	19	3,285	3,285	,, ,, ,,
74	15	112	84	28 days after ,, ,,
3 cases	46	4,857	4,829	28 days after (1 case)
Average	15·333	1619·0	1609·666	28 days after (1 case)
Max.	19	3,285	3,285	28 days after
Min.	12	112	84	28 ,, ,,

See also Table 2.

PART II.

HÆMOPTYSIS AND PULMONARY CONSUMPTION.

OBSERVATIONS, OPINIONS, DOCTRINES,

ETC., ETC.

HÆMOPTYSIS AND PULMONARY CONSUMPTION.

PART II.

Researches, Opinions and Doctrines of various Authors discussed. — Laennec, Rokitansky, Lebert, Fuller, Walshe, Virchow, Rindfleisch, Niemeyer, Wymodzoff, Lipmann, Perl, Rasmussen, Sanderson, Fox, Moxon, Klein, Gerhardt, Lancereaux, Dittrich, Baümler, Weber, Powell, Williams, Austin-Flint, Osler, Heitler, Waldenburg, Sommerbrodt, Mazzotti, Hertz, Ziemssen, Nothnagel, Cordua, Charcot. —Before engaging in a battle every wise man will consider: 1. What he has to fight; 2. What he means to fight for; 3. What he has to fight with; 4. The best way to use the arms with which he has to fight.—Hæmoptysis considered under these heads.—Special Consideration of the Initial Hæmoptysis of Pulmonary Tuberculisation.—Hæmoptysis as a Symptom and as a Cause of Disease.

Up to the present place in this work our attention has been almost entirely confined to my own original investigations and their results.

We must now enlarge our field of vision, and demonstrate the firmness of the basis on which we have established our therapeutics by bringing the observations of others into focus with our own. For, as practical physicians, we must hold, that sound and enlightened therapeutics, directed to the maintenance of health, the prevention, management, and cure of disease, constitute the highest aim of all our work.

The revival, by the late Prof. Niemeyer, of the ancient doctrine of "phthisis ab hæmoptoe," by which great danger has been run that once more "the cart would be put before the horse;" the brilliant demonstration, by Prof. Rasmussen, of the frequency of aneurismal dilatations of branches of the pulmonary artery in the walls of cavities, as causes of profuse and fatal hæmoptysis; the doctrine of caseation, by which Laennec and his "tubercular infiltrations" have been thought to be deposed; the doubts thrown upon the existence of any substance entitled to the name of tubercle; the results of experimenters on the lower animals (forgetful of the fact that men are neither guinea pigs nor rabbits), which have been supposed to prove that tubercle not only does exist, but is capable of manufacture out of all sorts of "stuff," and of propagation by inoculation; the elaborate investigations of Virchow into hyperplastic and heteroplastic lymphomata, and his wide generalisations with regard to scrofula and tubercle; the immensely interesting and important anatomical disclosures of Sanderson, Wymodzoff, Klein and others, respecting the

peri-vascular and peri-bronchial lymphatic system of the lungs; the scarcely-noticed but most important contribution, by Dr. Moxon, of a case showing the probable identity of grey and yellow tubercle, notwithstanding all that has been said to the contrary; the valuable and still more recent contributions of Charcot :—these and many other puzzling and contradictory questions surround our subject at once with difficulty and interest.

But puzzling and difficult as they are, they must be confronted, and, somehow or other, the practitioner of medicine must make his way out of them, and find light enough to guide him in fighting boldly with the vital diseases which they seek to explain.

Before engaging in a battle every wise man will carefully consider—

1. What he has to fight.
2. What he means to fight for.
3. What he has to fight with.
4. What is the best way to use the arms with which he has to fight.

In the case of hæmoptysis the dread of it is so great that the question—"What the doctor has to fight" is popularly supposed to be completely answered by the words—"*The bleeding!*" While the question—"What he means to fight for?" is met by the equally laconic answer, "To stop it!" This, however, is not quite the view which, as scientific medical men, we have to take of the matter. Some notion of this may be gleaned from the following enumeration of the "conditions which are more or less frequently attended by hæmoptysis, trifling or grave," which Dr. Walshe has given with characteristic attention to detail :—

1. Hysteria.
2. Vicarious pulmonary congestion.
3. Diminution of barometric pressure.
4. Violent efforts and fits of passion.
5. Blood diseases, various, heteræmiæ, septicæmia, etc.
6. Wounds and injuries of the lungs.
7. Local diseases :—

(*a.*) Larynx: ulceration, phthisical, cancerous, syphilitic.

(*b.*) Trachea: ulceration.

(*c.*) Bronchi: bronchitis, simple and plastic; ulceration, cancer, fragile state of vessels of mucous membrane.

(*d.*) Bronchial glands: tubercle, cancer.

(*e.*) Lung: congestion, active, passive, and mechanical; pneumonia, acute and chronic, plastic and caseous; abscess, gangrene, cirrhosis, tubercle, cancer, hydatids.

(*f.*) Pulmonary arteries: atheroma of; aneurism of minute or large branches.

(*g.*) Air passages generally: perforative disease from without, cancerous or other.

(*h.*) Mediastinum: tumour, abscess.

(*i.*) Mitral disease: hypertrophy of the right ventricle, dilatation with feebleness of the left ventricle.

(*j.*) Aneurism of the aorta: aneurism of the pulmonary artery, quasi congenital co-arctation of the arch of aorta.

"But," adds Dr. Walshe, "although it is impossible to ignore in pathology the power of all these affections to produce hæmoptysis, in actual practice the symptom is so frequently connected with tuberculisation of the lung, that it comes to be one of the most significant symptoms of phthisis." ("On Diseases of the Lungs," 4th Edit.)

My distinguished coadjutor, Professor Austin-Flint, of Philadelphia, in his admirable series of clinical studies "On Phthisis" (1875), presents us with the results of his experience derived from a critical examination and analysis of 670 cases; and the following are the conclusions at which he arrives with regard to the meaning of hæmoptysis.

"1. Hæmoptysis, the hæmorrhage limited to the bronchial mucous membrane, and not dependent on disease of the heart, or on an injury of the chest, is always presumptive evidence of existing pulmonary disease.

"2. With the foregoing qualifications, the occurrence of a bronchial hæmorrhage, if there be no evidence of existing pulmonary disease, renders it extremely probable that, sooner or later, phthisis will become declared.

"3. In most cases, at the time when the hæmoptysis occurs, the physical signs of pulmonary disease are to be discovered by the careful and skilful employment of auscultation and percussion.

"4. Hæmoptysis occurs in some cases where not only the symptoms of pulmonary disease are wanting, but the result of physical explorations of the chest is negative. In a certain proportion of these cases it is probable that the hæmorrhage is connected with a small affection which is latent as regards both systematic phenomena and physical signs.

"5. In view of the foregoing propositions, prudence dictates that, in the cases in which hæmoptysis is the only evidence of pulmonary disease, it is wise to act as if phthisis either exists or is impending.

"6. Bronchial hæmorrhage in some cases occurs, and it may recur frequently and persistently for a long period without any apparent protological connection with phthisis.

"7. The pathological condition standing in immediate causative relation to bronchial hæmorrhage is a hyperhæmia or congestion limited to a section of the mucous membrane.

"8. Whenever, as is true in the majority of cases, this local congestion has some pathological connection with phthisis, it is probable that the latter is the primary morbid condition; the hæmorrhage being incidental to it.

"Dr. Flint considers that, in advanced phthisis, hæmoptysis usually occurs from bands of pulmonary tissue traversing tuberculous cavities becoming ruptured (cavernous hæmorrhage), and is rarely bronchial; whereas, in *early* phthisis, the hæmorrhage is generally bronchial. According to his experience death is rare from bronchial hæmorrhage, and if it occurs it is from suffocation or syncope, while it is comparatively common from cavernous hæmorrhage. The after effects of bronchial hæmorrhage he says are slight, while those of cavernous hæmorrhage are severe. The importance of bronchial hæmorrhage varies. In *a few cases* he says it has no marked effect either good or bad on the patient, but denotes simply the development of a morbid condition which speedily destroys life. But his experience is that *as a rule* its occurrence is not unfavourable; and he concludes by stating that hæmoptysis has no marked apparent influence on the duration of phthisis in those cases which prove fatal within a period of five years. Death from loss of blood is rare, while the chances of recovery or arrest with hæmoptysis, are greater than without it." ("Dr. Dobell's Reports on Diseases of the Chest;" vol. ii., 1876.)

The late Dr. Fuller enumerated the circumstances under which hæmoptysis might occur as follows:—

"1. Idiopathic congestion of lungs.

"2. Secondary, from engorgement due to diseases of the lungs.

"3. Consumption.

"4. Pressure of aneurismal tumours, or of tubercle, cancer, or other adventitious matter.

"5. Pneumonic congestion, abscess, gangrene.

"6. Ulceration of the larynx, trachea or bronchi, extreme congestion of the bronchial mucous membrane more especially connected with a spanæmic condition of the vital fluid."

"Careful observation," he says, "has led me to believe that, except in cases of diseased heart, or of purpura and other hæmorrhagic disorders, bleeding seldom arises from the bronchial mucous membrane, unless that membrane is the seat of ulceration, or is acted on by aneurismal or other tumours, or by tubercular or other adventitious deposit, which causes pressure upon the bronchial vessels, and thus mechanically leads to rupture of the capillaries." ("On Diseases of the Chest," p. 256.)

I have no intention of discussing all the exceptional conditions under which blood-spitting may by possibility occur, nor, on the other hand, can I limit my remarks to tuberculisation. Many of the items of the list given by Dr. Walshe (see p. 68), will be excluded by simply confining ourselves to cases in which *the bleeding is more abundant or in a different form than streaks in the sputa.* It is not often that Dr. Walshe makes a clinical mistake, but, according to my experience, he has made an important one here by confusing "streaked" sputa and "tinged" sputa as though they were of the same value;

thus he says "streaked or tinged sputa are rarely or never the first symptom of phthisis" (op. cit., page 401). The confusion of the terms streaked and tinged is a very natural one, because it is so constantly made by patients, and any conclusions drawn from the statements of patients in this matter will be utterly valueless unless the confusion between streaked and tinged is carefully eliminated by strict cross questioning. By streaked I understand thread-like marks of blood in the expectoration; by tinged I understand a colour of blood diffused through the sputa, giving it more or less deeply the tinge of diluted blood; and this distinction must be explained to patients when requiring a description of their expectoration.

Now, while I entirely agree with Dr. Walshe that streaked sputa are not worth much consideration as signs of lung disease, my experience leads me to a totally different conclusion with regard to tinged sputa; the occurrence of which should never be disregarded. In speaking of hæmoptysis, therefore, I exclude streaks of blood in the sputa, but not sputa tinged with blood. And here again a distinction must be made between tinged sputa and pneumonic or *rusty* sputa. No doubt rusty sputum is in a sense sputum tinged with blood, but practically there is a very definite difference, due probably to the character of the other ingredients (muco-epithelium, exudation cells, amorphous exudation matter, oil globules, granular fat, moulded exudation casts, &c., &c.) of the sputum, and of the way in which they are intermingled with the blood; such sputum is well described by Dr. Walshe, as "sanguinolent, or rusty in tint, viscid, semi-transparent, adhering to the vessel, but slightly aërated, passing through various shades of orange, apricot colour, faintly greenish, and lastly becoming white. In some cases the sputa are different, and of dull brown, or even faintly blackish hue—resembling liquorice or prune juice—conditions of evil augury." (Page 358.)

But when Dr. Walshe adds, "If the expectoration in pneumonia be actually more or less profusely bloody—if there be hæmoptysis, in short—the pneumonia is, according to my experience, tuberculous." Again, I cannot agree with him, for, although in a large number of cases it is undoubtedly so, yet, the combination with the pneumonia of disease of the heart, arrested menstruation, purpura hæmorrhagica, or hæmophylia (affections competent without pneumonia to produce hæmoptysis) will, according to my experience, easily convert typical rusty pneumonic sputa into "expectoration more or less profusely bloody," without any tuberculous complications being necessary. I have certainly seen, under such circumstances, quite free hæmoptysis in pneumonia where the subsequent history of the case proved that there was no tuberculosis.

With regard to cancer, although the frequency of its occurrence as a cause of hæmoptysis is only fractional, as compared with phthisis, yet, in any individual case in which the cause of the hæmorrhage is not

self-evident, the question whether it is due to cancer must always enter into our consideration. "*Primary Cancer of the Lung and Mediastinum* . . . . is very frequently attended with sanguineous expectoration, or pure hæmoptysis. . . . . The percentage of hæmoptysis *of all amounts* in cases of cancer is 72, in phthisis 80·92; while hæmoptysis *above one ounce* occurs in cancer and phthisis in the ratio of about 70 to 40. Hence 100 cases of cancer of the lung will be attended nearly as often with hæmoptysis of all amounts, and greatly more often with hæmoptysis above an ounce in amount at a time, than 100 cases of phthisis." (Walshe, p. 401.) It is, therefore, especially in cases of profuse hæmoptysis of obscure cause that we are to think of cancer as possibly one of "the things we have to fight," and we must, of course, investigate the question by searching for the signs and symptoms of malignant disease, not only locally but generally.

Aortic aneurism, like cancer, although but a rare cause of hæmoptysis as compared with phthisis, is sufficiently frequent and insidious, that, though statistically not of much import, yet, in any individual case of hæmorrhage from the air passages of which the origin is obscure, it must enter seriously into our consideration, and must be diligently searched for as another of "the things we have to fight," although, even when present, it may sometimes elude discovery.

"Aortic aneurism, opening into the trachea, may, without proving immediately fatal, give rise to hæmorrhage, indistinguishable by its own characters from profuse pulmonary hæmorrhage. The history of the case, the physical signs, the age of the individual, &c., commonly establish the diagnosis; but when the aneurism is small and so placed as to elude percussion, and pressure signs both concentric and eccentric are absent, the difficulty of *proving* the presence of aneurism may be insurmountable, the existence of the disease may be divined, but not demonstrated. It is to be remembered that the absence of notable signs of tuberculisation does not justify the inference that the hæmoptysis is not phthisical, seeing that a tremendous pulmonary hæmorrhage may occur when slight consolidation exists at one apex only, and that such consolidation might be supposed to depend on the pressure of an aneurism." (Walshe, p. 402.)

The late Dr. Vald Rasmussen ("Hospitals Tidende," 1868) reported three cases of hæmoptysis due to bursting of aortic aneurisms within the air passages. "In one case the aneurism had developed on the aorta descendens, and perforated the right bronchus of a man, *who also suffered from cavernous non-tuberculous pulmonary phthisis.* This as well as another observation is made prominent, in opposition to the common assumption that aneurisms in general, and particularly on the aorta, do not appear together with pulmonary tuberculosis in its older sense. (Rokitansky, Lebert.) Only in one of the cases death occurred suddenly during a violent hæmorrhage; in the two other cases the

hæmorrhage was intermittent." ("Dr. Dobell's Reports on the Progress of Medicine," etc., 1869.)

Notwithstanding the length of Dr. Walshe's list of "conditions which are more or less frequently attended by hæmoptysis," it does not include one which, although not of very frequent occurrence, is sufficiently common to deserve our notice; while the circumstances attending it involve so many interesting points of distinctive diagnosis, that the possibility of its existence should never be absent from our minds when called to investigate a case of hæmoptysis. I refer to *hæmorrhagic infarction of the lungs*, and more especially to that form of it which begins with thrombosis of a systemic vein, leading to embolism of a branch of the pulmonary artery, and consequent shutting off of a portion of lung from the pulmonary circulation, the hæmoptysis which follows being due to the reflux of venous blood into the affected portion of lung with consequent over-distension and rupture of vessels.

The hæmorrhage may be slight or profuse, the consequences to the lung may be (*a*) gangrene; (*b*) excavation; (*c*) shrinking and induration; or (*d*) restoration to a normal state; according to the completeness of the embolic obstruction, the size and importance of the obstructed arterial branch, the previous condition of the patient, etc., etc. The following case, to which I was summoned while writing these pages, is sufficiently typical in its characters and circumstances to form a good illustration of the present observations.

I was asked by Dr. B. to meet him in consultation on a case of profuse and obstinate hæmoptysis of 8 days' duration. On arrival, we found the patient, a married man of 26, too much exhausted to answer many questions, but I learned from Dr. B., that he had for some years lived a very rakish life, by which his health had become dilapidated, but he had not had syphilis. Twelve days ago, after coming from Scotland to London, he sent for Dr. B. in consequence of sudden and severe pains in the right leg. No other cause or explanation for the pain being discovered it was supposed to be neuralgia, and prescribed for accordingly. Two days later, Dr. B. was sent for again, the pain having suddenly seized the lower part of the back of the left side of the chest, accompanied by difficulty of breathing. The painful part was found to be somewhat dull over a limited area. The patient complained of creeping chills about the back, and a tendency to sickness; pulse 120, very feeble, and he seemed more ill than the doctor could account for, as *the temperature was normal.* He did not then tell Dr. B., but on enquiry I found, that about the time the chest pain set in he had an alarming attack of dyspnœa, accompanied by faintness and sickness. It was this, in fact, which frightened his friends and induced them to send for the doctor; but the severity of these symptoms having passed off on his arrival, this part of the attack was not

described. On the following day the sputa became "tinged with blood almost but not quite like the rusty sputa of pneumonia," and the area of lung dulness was somewhat extended and more marked; pulse 120, very feeble, breathing hurried and easily distressed by movement. The doctor thought it must be a case of asthenic pneumonia, but the temperature was 97·5°, which puzzled him. From this time the blood in the sputa became more and more abundant, varying in coloration—prune juice (purple, the patient said), liver colour, black—notwithstanding the use of a variety of hæmostatics.

When seen by me, the doctor thought the patient could not have lived through the preceding 24 hours, the sinking and distress were so markedly increasing; bleeding had then continued 8 days, the quantity in the past 12 hours had been about half a pint of dark thick blood, with very little admixture of air or mucus. The patient was pallid and extremely exhausted, anxious and restless, so that scarcely any examination could be borne, but the dulness below the left blade bone was confirmed, and beyond capillary crepitation in the dull part and larger râles in the bronchial tubes, there were no other signs of chest disease. The heart sounds were feeble and flapping, without bruit, pulse scarcely perceptible, 120. Respiration 40. He had sweated much the last few days, and still complained of queer quivering creeping chills, but not of distinct rigors. *The temperature was* 97·2°. During the last few days, pain had been felt in the *left* leg; no corded vein could be felt in either leg, but they were both tense, and white with œdema up to the abdomen, the walls of which were becoming œdematous, and the scrotum also.

The conclusion, of course, was that the "neuralgia" in the leg was the pain of thrombosis, that a clot from this had slipped off and become blocked in the lung, causing the attack of dyspnœa and faintness followed by pain in the chest, and that the quasi rusty sputa, which could not have been pneumonic as the temperature was 97·5, indicated the first stage of the hæmorrhagic infarction which had since caused the profuse hæmoptysis. The ordinary rule, that thrombosis of the left leg and embolism of the right lung are the first to occur, was in this case reversed, but when seen by me the thrombosis had either occurred in the left leg also, or more probably in the iliac veins.

My friend and coadjutor, Prof. Gerhardt, of Würzburg, has written a most complete and masterly essay on hæmorrhagic infarction, from which I am glad to be able to quote. He states that he has never himself seen pure blood (such as occurred in the case just recorded) in the hæmorrhage of infarction of the lung, but that Laennec had seen it in incredible quantities. But he specially notices the fact of the expectoration being "*deceptively like that of pneumonia*," as described by Dr. B. in the first stage of our case.

The following important passages from Prof. Gerhardt's essay are especially worthy of study in this place :—

"We observe these infarctions in cases of disease of the heart, mostly where their appearance introduces the last phase of a long illness, and often leads to one of the darkest scenes of human suffering. We may be tempted to look upon the infarction as a *modus moriendi* of heart cases, just as certain forms of pneumonia regularly develop in cases of slow suffocative death. . . .

"There are not a few anatomical results and certain convincing clinical observations which point to *cured* or curing infarctions in cases of heart disease. But if we leave out of consideration heart disease, and if we pass over pyæmia, we meet with the infarction, somewhat more rarely indeed, yet frequently enough to recognise it as a comparatively mild disease, and one which, under favourable circumstances, is eminently curable. . . .

"There seems to be little doubt that most of the infarctions arise from *the embolic closure of the supplying arteries*. The wedge-shaped piece of tissue, with its base at the pleura which the artery supplies, is, after closure of the artery, the seat of a hyperæmia, the blood flowing backward from the veins and infiltrating the part. The tissue is loosened in its structure and infiltrated with blood, which coagulates and converts the part into a swollen granular reddish black mass, over which the pleura is generally coated with a layer of fibrine. . . .

"You may divide the complete history of an infarction into, (1.) The *initiating thrombosis*; (2.) *The act of embolism*; (3.) *The infarction itself*. You will not always be able to make out clearly at the bedside the relation of the processes. . . .

"The *seat of the thrombosis* which gives off the embolus may of course be in any of the systemic veins, but not in the portal branches. . .

"At least we ought never to come to the conclusion that a clot from the *left* side of the heart or from a systemic *artery* has wandered over to the venous circulation, or that the infarction has arisen from the rupture of a pulmonary vessel, and the coagulation has afterwards arisen in the pulmonary artery, without a thorough examination of all the systemic venous system. . . .

The left lower extremity "is more frequently involved, on account of the less favourable course of the iliac vein on this side. . . .

"In these cases (mitral and tricuspid disease) it is not always the diseased valves or the dilated portions of the cavities of the heart which furnish the emboli, but much more frequently it is distant, highly dilated portions of the venous system. . . .

"The statement that *aortic stenosis* is peculiarly apt to cause *pulmonary hæmorrhage*, is explained, according to my experience, by the frequency of thrombosis of the right side of the heart and emboli arising there. The ultimate cause of the frequent occurrence of spontaneous coagulation of blood in heart disease is unknown. We can only guess that it is related to the diminution of the blood pressure in the arteries, the in-

crease of pressure in the veins, the retardation of the current, and the altered nutrition of the walls of the vessels. Among the infective diseases *typhoid fever and dysentery* are those which especially lead to infarction. . . .

"*The act of embolism* may occur almost or altogether without symptoms where only very small and few coagula pass into a perfectly sound lung, or where there is already a strongly-marked dyspnœa. Very large or massive emboli introduced simultaneously cause sudden death, or unconsciousness with convulsions, ending in death. Between these extremes lie the symptoms which, in very various degrees, indicate the obstruction of a portion of the pulmonary vascular system, and generally precede the proper symptoms of infarction. The more extensive the closure of the pulmonary artery the more certain is the *attack of unconsciousness* which opens the scene. The sudden diminution of the supply of blood to the brain is its cause. Under certain circumstances convulsive movements are added, and these are also explained by the cerebral anæmia just as they may occur along with embolism of several cerebral arteries. . . .

"The *embolus generally passes to the lower lobe*, directed thither by gravity and the stronger current; after the lower, the middle and upper lobes may be affected. Where there is no special circumstance directing it elsewhere, the embolus generally *passes to the right*, following the stronger impulse of the blood-current. . . .

"The earliest and truest of the *symptoms of the hæmorrhagic infarction* is *spitting of blood*. In my fifteen cases it was observed thirteen times. And it occurred $8\frac{1}{2}$, and a second time 24 hours after the shivering, which marked the occurrence of embolism, in other cases it was two or three days after the attack of dyspnœa. At the same time you will understand that it is by no means possible always to determine perfectly the length of time intervening. The bloody condition of the expectoration may be only transitory, and may be in the form of a mere trace, or it may last a long time when the attack is very violent—thus in two of our cases it lasted twenty-seven days. In infarction all forms of hæmoptysis which you can distinguish may occur, none is excluded. As a forerunner of the other forms we have often *streaks and spots of blood* in the muco-purulent mass ; in very anæmic persons this form of hæmoptysis continues the only one. For the existence of expectoration of pure blood we have not only the evidence of Laennec, who has seen almost incredible quantities of blood expectorated in infarction, ten pounds in 24 hours, thirty pounds in 14 days, but we have besides the observation of Dittrich of moulded blood clots in the broken-up lung tissue of the infarction. I have myself never met with pure bloody sputa in this disease. I consider this occurrence even more exceptional than the masses of mucus merely beset with bloody points. The sign which is commonly but falsely set down by many as pathognomonic of

the hæmorrhagic infarction is the sputa intimately mixed with blood. It may be *deceptively like the pneumonic sputum* in colour, transparency, toughness, etc.; but *as a rule it is more bloody than this*, and it contains no croupous coagula, certainly none of a light colour; it is rarely the only form present, but is mostly placed among the muco-purulent sputa. This latter circumstance is explained by the small size of most infarctions (according to Laennec one to four cubic inches, according to Rokitansky from a nut to a hen's egg in size), and by the existence of valvular disease, emphysema, and such diseases as readily lead to bronchitis. Bloody sputa may be yielded by one infarction for several weeks. They contain for more than a week unaltered blood-corpuscles. After two or three weeks there appear, however, in the expectoration granules and crystals of hæmatoidin, so that this form of bloody sputum is also met with in the course of the infarction. After a variable time of about one and a-half to two weeks the bloody sputa which have become scarcer assume a brownish red or dark red colour, similar to that of currant jelly. The expectoration of bloody sputa most frequently calls attention to the existence of the infarction; we learn often for the first time on putting direct questions that a shivering or dyspnoic attack has preceded. At the same time this form of sputum does not prove the existence of an infarction. It is not found in every case, and it is not always a sign of this affection. Just as in pneumonia, so here, the sputum is rich in an albuminoid substance, which is precipitated by alcohol and readily dissolved by water, similar to what has been described, among others, as paralbumin. . . .

"I have not often found that, contrary to expectation, a large infarction had produced no dulness; much more frequently *is it the reverse*, that at a place where there was extensive dulness an unexpectedly small infarction has been found. The reasons are easy to perceive. Œdematous lung tissue in the neighbourhood, increases the area of dull percussion. Then pleuritic exudation is very often associated with the infarction, and the dulness produced by this may be taken along with that of the infarction, at least where the latter has its seat near the lower edge of the lung. As the bloody expectoration may occur at a variable time after the embolic act, so the dulness on percussion, following as it does shortly after bloody expectoration, makes itself evident sometimes one, sometimes several days after the dyspnœa or shivering fit. . . .

"In a third of the clinical cases, but much more frequently in persons who observe their own symptoms carefully, there is found *a stitch in the side*, which corresponds approximately to the seat of the infarction. . . .

"Most of the pleuritic exudations of cardiac cases proceed from infarction. You should always ask, where there is pleurisy in cardiac cases, whether hæmoptysis was present at the outset. . . .

"*The Anatomical consequences of infarction* are:—1. Dissolution of the rusty brown portion of tissue, absorption of the blood in the alveoli with retention of pigment in the tissue. In this way does Rokitansky describe a perfect resolution of the lesion analogous to the resolution of a pneumonia. I have never been able distinctly to recognise any of the stages of this process, but I would not in the least throw any doubt on its existence. 2. Decolorization and contraction into a yellowish white or grey cicatrix of small dimensions. This appears to me to be the most frequent and most favourable transformation of the simple infarction, and its existence may often be proved a long time after the conclusion of the process. . . . 3. Softening and liquefaction of the structure to a brownish red or greyish red odourless fluid, which discharges into the bronchi and leaves a cavity. This fluid is remarkable for the abundance of hæmatoidin crystals which it contains, along with elastic fibres and large cells containing blood-corpuscles and blood pigment. We may guess that this condition is present when, after the existence of bloody expectoration for weeks there appear later on sputa of a striking greyish red colour. . . . 4. The hæmorrhagic infarction breaks down in the way of suppuration or gangrene. This consequence is generally determined by the constitution of the embolus, which carries with it some material which provokes suppuration and decomposition. With such infarctions there is of course a purulent and decomposing pleuritic exudation. . . .

"*The diagnosis of the hæmorrhagic infarction* is from these considerations, easy to make in most cases, especially in cardiac cases, and in cases of thrombosis of the crural veins or those of the calf. . . .

"Yet errors may very readily be committed if we look on the form of the expectoration as pathognomonic. Very similar forms of expectoration occur under the following circumstances:—1. *Frequent Ecchymoses of the bronchial mucous membrane*, such as often occur in heart disease with endocarditis of the left side. . . . 2. *Cancer of the lung* . . . furnishes in the majority of cases, a few bloody sputa in the midst of many mucous ones. The former are intimately mixed, but very bloody and somewhat of the colour of currant jelly. They resemble to a high degree the sputa which are discovered after an infarction has existed long. . . . 3. *Echinococcus* of the lungs. . . . 4. The diagnosis of the infarction from *Pneumonia* should never rest on an individual symptom. . . . Yet you may definitely hold the infarction as determined if there obviously exists embolic material and an embolic attack, and when the diagnosis is confirmed *by absence* of fever, dulness in the lower lobe lasting for some weeks and hæmoptysis." ("Clinical Lectures," translated for the "New Sydenham Society," vol. lxxi., 1877.)

Dr. E. Lancereaux says that the formation of pulmonary infarctus "entirely depends upon the seat occupied by the embolus.

Whenever this lies above the mouth of at least one anastomotic vessel, collateral circulation becomes established, and no blood extravasates. Whenever, on the contrary, the obstruction lies below the last anastomosis, that is to say, whenever no anastomosis is interposed between the seat of the embolus and the point where the plugged artery breaks up into capillaries, a backward flow develops behind the plug, and the production of infarctus ensues. This mechanism explains why the smaller embolic concretions are most likely to produce pulmonary infarctus. From all this it must not be concluded that every apoplectic nucleus found in the lung is an embolic infarctus. Many a case of pulmonary hæmorrhage, no doubt, should be referred to some cause other than embolism. But, in the absence of the latter, the apoplectic focus is much more extensive and is much more likely to occupy indifferently any point of the lung than it is in embolic cases. The preceding description merely refers to the mechanical effects of embolism. According as an embolus comes from a purulent, gangrenous, or other source, and is loaded with septic matter, the point of the arterial wall with which the coagulum is in contact first suppurates or sphacelates. Subsequently, this suppurative or gangrenous process implicates the neighbouring lung tissue. *Hence, according to the nature of the embolus, the production of purulent pneumonia, of abscess of the lung, or of pulmonary gangrene.* In these cases the effects of embolism are twofold, viz., those which are purely mechanical arise from the obstacle to circulation in the pulmonary artery; the others, which are irritative or specific, that is to say, which are special to the nature of the embolus, consist in some chemical irritation of the arterial wall and of the surrounding lung tissue. An embolus loaded with cancerous matter is far from possessing such active properties as those of a clot saturated with septic products. Nevertheless cancerous embolus in some instances appears to have been the starting-point of metastatic cancer of the lung. Again, embolus, in some cases, seems to have been the medium through which hydatids were deposited into the lung. As might be anticipated from the preceding description, the prognosis of pulmonary embolism depends entirely upon the circumstances of the case. Thus, whilst the obstruction of the trunk of the pulmonary artery would threaten rapid death, the presence of one or even of several emboli in the secondary, the tertiary, or the still smaller divisions of this vessel would, in most cases, produce no disturbance other than a slight degree of dyspnœa. If, however, such concretions were numerous, and occupied in both lungs several branches of the pulmonary artery, rapid death, as has been witnessed by M. Lancereaux, might possibly supervene on more or less violent exertion. Lastly, if the embolic plug came from some purulent or gangrenous focus, *the pulmonary tissue soon would undergo morbid*

changes, and these, in most cases, would ultimately produce death." ("Traité d'Anat. Pathol.," vol. i., 1875-77, p. 614, *et seq.*)

By far the most important contributions to the subject of hæmoptysis in modern times are those of my distinguished friend and coadjutor, the late Professor Rasmussen, of Copenhagen, of which the following brief abstract was prepared for me by his own hand, in 1869:—"*On Hæmoptysis, especially Fatal Hæmoptysis, in its Anatomical and Clinical Aspects;*" by Dr. Vald Rasmussen ("Hospitals Tidende," 1868, 1869; "Edinb. Med. Journ.," 1868, 1869; "Brit. and For. Med.-Chir. Rev.," Jan., 1869, etc.). "In the first communication the author states, in the historical view of the question, that Rokitansky is, no doubt, the only one who mentions similar causes of hæmoptysis in phthisis. Dr. Rasmussen gives nine sections in which he puts forth the bursting of dilated branches of the pulmonary artery continued in the wall of a cavity as the cause of the hæmorrhage. These vascular expansions he divides into two classes :—1. Smaller sac-like aneurisms; 2. Ectasias. Four of the specimens belong to the first, and five to the last class. The size of the aneurisms varies from that of a walnut to that of a pea, and even smaller; they are formed by the expansion of one of the vessels touching the inner wall of the cavity, as that part of the wall of the vessel expands, which at its point of contact comes close to the cavity. Thus the aneurism has the shape of a bag (*aneurisma sacciforme verum*), the surface being even; only a single one, of a very large size, has a small external protuberance. In the aneurism generally, only some fresh coagulated blood is to be found, and in one only of the four there were some old colourless coagulations in one of the protuberances. The walls of the unbroken aneurisms are of great thickness, while those of the broken ones are thin, especially towards the opening. This opening is always found on the most protruding part of the sac, and often forms a fissure-shaped chink from two to three millimètres wide, filled with fresh coagulations; the edges are thin, and of a yellowish colour (fatty degeneration, especially of muscular tissue). The number of aneurisms is different, varying from one to four; generally there is only one; once there were two, once four—two and two side by side. The branches of the pulmonary artery on which the aneurisms are situated are, as a rule, of one or two millimètres in diameter.

"The ectasias are smaller aneurismal dilatations of vessels in the walls of cavities. They appear in two shapes: one, either as shorter or longer cords, on the inside of the wall of the cavity, and when slit up they show a slight inward extension of their base towards the cavity; generally the wall is very thick, sometimes even to such a degree that the base of the vessels becomes perceptibly smaller. In cases where death had been caused by hæmoptysis these shapes were seldom observed, but they appeared frequently in cavities which had not caused hæmoptysis.

"The second shape is more frequent, namely, where the vessel touches the wall of the cavity only to a limited extent; here a small oblong protuberance is formed, which is partly due to the expansion of the bore of the vessel, partly to its wall growing thicker. The perforation takes place through a V-shaped chink in the wall of the vessel, whereby a vent or a lid-shaped flap is opened. The bursting takes place on the boundaries between the vessel and the wall of the cavity, and the point of the lid always lies in the direction of the blood-current. As a contrast to the regular aneurisms, the lid is sometimes thick, but as a rule it is thin.

"Only in half the number of the cases miliary tubercles were found in other organs, and in the other half nothing was to be seen but chronic pneumonia, with caverns of bronchiectasis. Dr. Rasmussen states that *every cavity in the lungs, the walls of which are formed by a compressed pulmonary tissue containing non-obliterated vessels, may become the seat of aneurisms or ectasias, with succeeding hæmorrhage; yet the thin-walled cavities immediately abutting upon air-containing pulmonary tissue, seem most frequently to become the seat of these vascular extensions.* The size of the cavity varies greatly, from that of an ostrich's egg to that of a nut, and it has no connection with the formation of the aneurism.

"The cause of the formation of the aneurism is not an atheromatous or any other change in the wall of the vessel, but consists principally in the want of support to that wall of the vessel which is directed towards the interior of the cavity, and in the heightened intra-vascular pressure, which becomes very considerable, partly because many of the branches of the pulmonary artery are obliterated, partly because the branches going out of the aneurism are very fine, and are quickly absorbed by the compressed pulmonary texture; thirdly, it is owing to an aspiration of the blood during coughing fits, which generally causes the fatal bursting.

"In a clinical respect, Dr. Rasmussen states that, while the fatal hæmoptysis generally occurs unexpectedly and suddenly, in three cases it was remittent (*recurrent*), accompanied by violent hæmorrhage, with an interval of several days; this lasted for nearly three weeks previous to death. These remissions are, perhaps, owing to reiterated small burstings of the wall of the aneurism, partly caused by the fatty degeneration of the wall, which, at least in one case, was proved by the post mortem examination of the body; or by the obturating coagulation, which might have been torn away during the violent hæmorrhage, although it could not be proved by the post mortem examination. The remittent hæmorrhage occurs only in the case of proper aneurisms, while the ectasias cause death immediately when the lid becomes loose.

"Concerning the question, as to what is the significance of this bursting of expanded vessels contained in the walls of the cavity, as the cause of pulmonary hæmorrhage in general, Dr. Rasmussen feels con-

vinced that the fatal and profuse hæmoptysis in phthisis is owing to such a bursting. He has, at least since he became aware of the existence of such aneurisms, never failed to notice them in any case of fatal hæmoptysis. Of course, it cannot be decided with certainty to what extent a considerable but not fatal hæmoptysis is caused by similar circumstances, but there is much in favour of this being the case. The cavity, as well as the vessel, may be very small, whereby there is a possibility of the hæmorrhage stopping of itself. This explanation of the origin of hæmoptysis is at least based upon certain anatomical facts; which cannot be said of the commonly supposed broncho-hæmorrhage, which is principally supported by the, at least in some instances, untenable doctrine of the absolute obliteration of the vessels in the walls of the cavity. It is not, however, the opinion of the author that an inter-parenchymatous hæmoptysis issues from a cavity; phthisis in its more acute forms is, no doubt, caused by the softening of an infiltrated substance.

"In the second treatise, Dr. Rasmussen gives an account of two fresh cases of fatal hæmoptysis during the progress of phthisis, which in every respect confirm his former observations. In both cases the hæmorrhage was intermittent (*recurrent*), and was caused by the bursting of aneurisms. One of them is especially worth mentioning, because the cavity was only the size of a grey pea, and might easily have been passed over; and in the small aneurism was found an older colourless coagulation. These observations seem to justify the conclusion that the obturating coagulations are the cause of the remission of the hæmorrhage; but, as a rule, they are not to be discovered during the post mortem examination, because they have been torn away by the strong current of the hæmorrhage." ("Dr. Dobell's Reports on the Progress of Medicine," vol i., 1870.)

After these most important and unmistakable observations of Dr. Rasmussen, it was only natural that other observers in different parts of the world should seek to confirm or to refute them; and the result has been that their truth and exactitude have been abundantly confirmed by numerous subsequent observations; among these, a contribution by my friend Dr. Douglas Powell is deserving of special notice, from the clear and unmistakable character of its evidence. Dr. Powell submitted to the Pathological Society of London, in 1871, sixteen cases "illustrating the Pathology of Fatal Hæmoptysis in Advanced Phthisis," in which he tabulated fifteen of the cases, and gave the details of four; and he remarks, as I think with unquestionable correctness, that "the facts brought forward in the description of the above four cases, and also those briefly related in the table, show, I venture to think, conclusively, that *fatal pulmonary hæmorrhage in cases of advanced phthisis, almost invariably proceeds from rupture of a branch of the pulmonary artery in a cavity, either traversing its walls or crossing it imbedded in a trabecula.*

Of the fifteen cases tabulated (including the three above described), in all the twelve cases in which the source of hæmorrhage was discovered post mortem, it was found to be of this nature. In six of these twelve cases, a well-marked sacculated aneurism was found situated on the pulmonary branches, having caused death by rupture. These aneurisms varied in size from that of a large pea to a Maltese orange, had thin, very friable walls, and were usually devoid of coagula, or contained only a few fibrinous laminæ. Of the remaining six cases in which the source of the hæmorrhage was traced—in five the vessel was bulged at the seat of rupture on the exposed surface only, forming what might be called a semi-fusiform aneurism or ectasia. In these cases the wall of the vessel, at its bulged portion, was usually greatly thickened, though more brittle than natural. Dr. Rasmussen describes this thickening as due to hypertrophy of the muscular coat of the artery. I cannot think this is the true explanation, but shall have to refer to this point presently." (Dr. Powell afterwards says that he thinks the coat is thickened by inflammatory change set up by the increased strain upon the wall, as it is the exposed wall only which is so affected.) "In the remaining case (12 of Table), the hæmorrhage had resulted from *ulcerated erosion* of the wall of a branch of the pulmonary artery, on which, however, a little higher up, there was a small aneurism or ectasia situated."

For the purpose of noting the kind of cases in which these hæmorrhages occur, Dr. Powell, for practical convenience, divides cavities into "*old standing quiescent, old standing active, and recent cavities.*"

Dr. Powell had already noted, "It is perhaps well to mention that by the term *quiescence*, as applied to the walls of cavities, is here meant that condition of dryness, smoothness, and comparative avascularity, commonly seen in cavities which are old, circumscribed, and non-extending; while the term *activity* refers, not so much to the extension of a cavity by the breaking down of fresh pneumonic consolidations into it, as to that condition of active *ulceration* and *erosion*, which is apt to take place in cavities shut off by fibrous induration from the surrounding parts. Such cavities have long been *quiescent* when their walls become attacked by the ulcerative process, attended with profuse and exhausting purulent discharges, the occasional formation of gangrenous sloughs, or of fistulous canals, which may open into the pleural cavity, or through the thoracic wall. This process, which belongs to the class of ulcerations, not of parenchymatous inflammations, causes extensive exposure of vessels."

Of these three varieties, with the definitions of which I entirely agree, Dr. Powell says "the first is certainly that which is most favourable for the production of pulmonary aneurisms or ectasias; and there seem to be good grounds for saying that the more quiet and quiescent the cavity, and

the more unilateral the disease—the more nearly, in short, it approaches the type of what has been described as 'fibroid phthisis'—the more probable is it that hæmorrhage, if it occur in any quantity, proceeds from a pulmonary aneurism. . . . . In the second class of cases, the cavity is, I am inclined to think, usually of tolerably old standing; its walls are thick and indurated, but instead of being in an inactive condition, they are undergoing a rapid ulcerative process; such cavities are extremely vascular and roughly trabeculated, and in them the large vessels are extensively exposed and eroded, and may give rise to fatal hæmoptysis. . . . . Though hæmoptysis may be very considerable in such cases, *fatal* hæmoptysis is, I am inclined to think, not so common as in the first class."

"Of the third class, ... those in which hæmoptysis occurs from rupture of a vessel in the course of rapid breaking down of degenerated tubercular or pneumonic consolidations. . . . . Small vessels become broken across, but the softening, which is mainly due to pneumonic processes which have preceded, is in great measure the result of the obliteration of the small vessels involved in the inflammation. The tissue immediately surrounding the large vessels possesses great vitality. These arteries if exposed at all are so after being involved in and surrounded by acute inflammation, and are more likely to become entirely closed by coagula." ("Transactions of the Pathological Society," vol. xxii., 1871.)

Nothing, then, can be more striking than the different aspect which the observations of Rasmussen have given to the questions, "What we have to fight?" and "What we mean to fight for?" in cases of hæmoptysis. We can no longer wonder that in certain cases nothing can stay the fatal course of the hæmorrhage. At the same time, this knowledge of the aneurismal character of such hæmorrhages, by putting us on our guard as to their source and origin, enables us to save life in a much larger proportion of cases of profuse hæmoptysis than formerly, and thus we owe a double debt of gratitude to the distinguished Dane.

The little work of Professor Niemeyer ("Clinical Lectures on Pulmonary Consumption;" translated from the Second German Edition, by Dr. Baümler. New Sydenham Society, Dec., 1870), is so clearly the utterance of a man "who has the courage of his opinions," and is so perfect a model of what a clinical essay should be, that no one who has read it can wonder at the singular hold it at once took of the professional mind, both abroad and at home. It is in every way too important to be lightly passed by. Before considering it, however, I must call attention to the striking contribution of Dr. Moxon, by which the views of Niemeyer and his followers have, in my opinion, received a most serious, if not vital, home-thrust. The interest of Dr. Moxon's

case lies in "the strong contrast which two patches of disease, of which he gave drawings, . . . showed as spreading from their centre outwards . . . . the centre part being the older, and the peripheral the most recently formed. Now," says Dr. Moxon, "the centre part has in both exactly the same character and is identical; but the outer zone of one patch has the character of grey subpellucid tubercles of the most typical kind, while the outer zone of the other has larger yellowish white tubercles of the most scrofulo-pulmonic kind. Numerous microscopic sections showed that both of these kinds of tubercles were seated in the proper pulmonary texture, and not especially about the ends of the bronchial tubes. I believe in this case there is a kind of evidence bearing importantly on the question of the identity of the two forms of tubercle. The grey patches occur in the same lung, and have the same appearance, structure, size, and relation to the tissue, so that it is impossible to think that two such identical patches arose from dissimilar causes in the same lung together. Granting their identity, it is almost equally difficult to doubt that the tuberculous border around each of the masses stands to the mass as its most recently formed part. . . . . The identity of the two chronic patches establishes the identity of the yellow and grey tubercular border which are respectively the more recent parts of these patches." ("Transactions of the Pathological Society," vol. xxii., 1871.)

Now, let this short and simple but important case be constantly borne in mind while we review the assertions and opinions of Niemeyer and others.

Professor Niemeyer says—"Laennec's dogma, that every form of pulmonary phthisis is caused by a specific new growth (une espèce particulière de production accidentelle), and that the cavities in the lung take their origin alone in the softening and the excavation of this growth, was simply a *pathological hypothesis*, which, by the more recent researches in the field of pathological anatomy, has been entirely refuted. This being the case, the conclusions of Laennec have lost all support, and the assertions which are still so frequently repeated,—that a bronchitis, out of which phthisis is developed, is not to be considered as a primary genuine cold, but as a secondary catarrh caused by the irritation of the lung by already existing tubercles, and that the same explanation must hold good for those *attacks of pneumonia and hæmoptysis* which so frequently precede phthisis,—prove that in this field medical practice has almost entirely ignored the progress made by pathological anatomy.

"The error in which Laennec and his followers were entangled did not consist in their regarding tubercle as a new growth, but in the fact that they considered those condensations of the lung-tissue, which have quite a different origin, to be also products of a development of tubercles. This confusion originated, chiefly, in the *cheesy metamorphosis*

of the original grey and transparent tubercle being accepted as one of its specific peculiarities, and as a sign from which the tubercular nature of any substance which underwent the transformation might be inferred. From this point of view one was justified in regarding the extensive consolidations in phthisical lungs which were found side by side with miliary tubercles, and which, having been at first grey and transparent, had afterwards become yellow and cheesy, as a diffuse growth of tubercles, or as extensive infiltrations of the lung with tubercular material. But since pathologists, and especially Virchow, have shown *that substances of the most varied kind which have not the least relationship to tubercle*, such as old cancerous tumours, lymphatic glands swollen by hyperplasia of their cells, hæmorrhagic deposits, encapsulated masses of pus, etc., undergo the very same cheesy transformation as miliary tubercle, the fact that those diffuse consolidations of lung become yellow and cheesy can no longer be admitted as proof of their tubercular nature.* Laennec's whole theory of infiltrated tuberculosis, or tubercular infiltration, which was supported by that criterion alone, has thus lost its foundation. In the present stage of science there is but one kind of tubercle—miliary tubercle,—and but one form of tuberculosis—miliary tuberculosis,—and all those changes which, since Laennec, have been designated '*infiltrated pulmonary tubercle,' are the product of chronic, especially of catarrhal, pneumonia.* . . .

"But the doctrine of *miliary tubercle* has, no less than that of infiltrated tubercle, received a blow by the progress of pathological anatomy. It has been found that many formations which, at first sight, appear to be miliary tubercle, and which formerly were commonly considered as tubercle, are, on more careful examination, found to be transverse sections of bronchi with cheesy deposits, or whose walls are thickened and surrounded by alveoli filled with a cheesy infiltration. If such mistakes be avoided in judging of the post mortem appearances, we arrive at a result which, it is true, is in glaring opposition to the views prevailing in practice, namely, *that in very many cases there is not a single tubercle found in phthisical lungs*, and that the consolidations and destructions of the lungs are caused alone by inflammation leading to induration and softening."

It is here entirely ignored, but cannot possibly be denied by any practical pathologist, that in the destructive processes of phthisis the tuberculous deposit which set up these processes has often been removed in the general devastation of the affected parts, and therefore, of course, cannot be found on the post mortem table.

Concerning this "cheesy" transformation or "caseation" and its relation to "tubercle," Professor Rindfleisch says :—

"We must give up all hope of finding in the *ascending* series of morbid changes, any criterion which will enable us to distinguish

* The above is no proof to the contrary.

between chronic and acute inflammation ; in the descending series, however, we *do* find such a criterion, viz., CASEATION of the inflammatory products. The occurrence of caseation—a fatty degeneration of corpuscular elements, modified by the abstraction of water—implies the co-existence of various favourable conditions. Of these the following are the most important : excessive accumulation of cells in close contact with one another, owing to the absence of any intercellular fluid; a gradual arrest of the circulation, which serves as the proximate cause of the actual impairment of nutrition. . . .

"The *cheesy matter*, alluded to above, is identical with what used to be known as '*crude tubercle*,' and regarded as the sole starting point of phthisis. At the present day we must reserve the term *phthisis tuberculosa*, for those very numerous cases in which the presence of *miliary tubercle* in the connective tissue and in the vessels of the diseased lung can be actually demonstrated. We must admit that our forerunners had a very clear perception of a fundamental truth, when they asserted that pulmonary phthisis, at a certain stage in its progress, extended its ravages by means of a yellowish-white friable material. Nay, we may even go beyond this ; we may retain Laennec's distinction between 'tubercular infiltration and tubercular granulations' in so far as these terms correspond with the two chief naked eye appearances of catarrhal broncho-pneumonia." (Rindfleisch, vol. ii., pp. 28, 29.)

As a comment upon this we must revert to Dr. Moxon's case (p. 85), and also carefully consider the recent important communications of Prof. Charcot :—

"In the report of the meeting of the Société Anatomique published in the 'Progrès Médical' for December 15th, 1877, M. Charcot made the following communication on some cases of caseous pneumonia:—In the three cases in question there existed pseudo-lobar pneumonia which, in reality lobular in origin, had invaded the whole lobe of a lung. Clinically, the disease had developed with great rapidity, and with the phenomena of lobar pneumonia. Death supervened in one case on the fourth day. These were therefore cases of a lesion which has been designated caseous pneumonia, and which, according to Virchow, originates in a broncho-pneumonia developed apart from tubercle. Histological examination showed him that there was reason to reconsider the opinion emitted by Virchow and Reinhardt, and accepted too easily, that indeed in these cases there actually is a lesion of tubercular nature, and that we must accept the doctrine of Laennec modified slightly to suit the progress of histology. This opinion is not new in France. MM. Thaon, Grancher, and Renault have sustained it ; later, in England, Wilson Fox has confirmed it. The nodules visible to the naked eye in sections of the pulmonary tissue are constituted by an aggregation of miliary tubercles. It is to be remembered that tubercle, when isolated, presents at its centre a giant-cell surrounded by epithe-

lioid cells and numerous nuclei. In a section of a pulmonary lobule one sees two orifices, one of the artery, the other of the bronchus. Around the latter the lesions are grouped in the form of an irregular mass divided into two zones ; one, central, yellow, and caseous, is distinctly separate from the other, which is exterior. This, which has been well observed by Grancher, is formed of a series of tubercular granulations, each containing a giant-cell and nuclei. These giant-cells are ordinarily arranged in a single row, but may form several. Around this peripheral zone the pulmonary alveoli appear healthy, or at least have no lesions tubercular in character. The whole of these alterations constitute a tubercular mass with central caseation, for here as in the tongue they are always the central tubercules which first soften. There was no pneumonia in a part examined by M. Charcot. If there were fibrinous deposits in the pulmonary parenchyma, that was an accessory phenomenon, for the masses, caseating at their centre, were always surrounded by easily recognisable tubercular zones. It mattered little if pneumonic exudation were found in the lung-parenchyma ; caseation was never produced in these places. This always appeared at the centre of a distinct agglomeration of tubercles. Whether or not these were surrounded by various lesions, these latter were only secondary. Caseous pneumonia, the supposed result of caseation of a pulmonic exudation independent of tubercle, did not exist in the specimens examined : and as to the existence of caseous pneumonia, M. Charcot thinks there is need to support it by new facts." ("Lond. Med. Rec.," Feb. 15, 1878.)

After detailing his own views of the etiology of phthisis, Niemeyer goes on to say:—"From our point there is nothing at all strange in the fact that one man with a well-marked disposition to pulmonary consumption remains free from the disease, and reaches old age, whilst another who has no such predisposition becomes phthisical by external influences, by an intercurrent illness, etc., and may find an early death." And he thus sums up what he considers the most important points in the following conclusions :—

"1. The consolidations and destructions of the lung, which form the anatomical basis of pulmonary phthisis, are, as a rule, the products of pneumonic processes ; and the more abundantly cellular elements accumulate in the alveoli, and the longer this accumulation persists, the more readily does a pneumonia lead to phthisis, because the cheesy metamorphosis of inflammatory infiltrations is thereby favoured.

"2. Pneumonia resulting in cheesy infiltration occurs chiefly in delicate, badly-nourished persons ; this experience is partly founded on the great vulnerability of such persons, and partly on the fact that the inflammatory nutritive changes occurring in them show a tendency to an abundant production of cells, and thereby to a cheesy metamorphosis of the inflammatory products.

"3. Pneumonia of this character does not occur usually, even in delicate and vulnerable persons, before the age at which pulmonary diseases generally become more frequent; and it then takes the place of those inflammatory diseases of other organs which have prevailed during the preceding period of life. All the influences, indeed, which dispose to pulmonary consumption, from the conception by a consumptive father to the exhaustion of the body by a long and serious illness, become perfectly clear and intelligible by these propositions, the correctness of which can hardly be doubted. Nor can it appear at all surprising that the disposition to pulmonary consumption will be more or less clearly expressed in the poor and delicate habitus of an individual. . . . We can sum up the relation which, according to our opinion, exists between *scrofulosis* and *pulmonary consumption* in the following sentences:—

"1. Adults who in their childhood have been scrofulous have, unless the vulnerability on which scrofulosis depends has disappeared, a well-marked tendency to pneumonia, terminating in cheesy infiltration of pulmonary consumption. 2. In individuals who formerly were scrofulous, persistent cheesy bronchial glands give rise, in some instances, to the development of tubercles in the lungs and to a *tubercular* phthisis. 3. Individuals in whom an extinct scrofulosis has not left behind either an increased vulnerability or cheesy masses in the lymphatic glands possess no greater disposition to pulmonary phthisis than individuals who have never been scrofulous.

"I consider the almost universal opinion, that pulmonary consumption arises independently of accidental or immediate exciting causes, in consequence alone of a diathesis, to be as unproved as it is dangerous."

In this passage Niemeyer evidently misapprehends the opinion which he wishes to stigmatise—that opinion being that the consumption *excited by accidental or immediate exciting causes is a disease dependent upon a peculiar constitutional state;* a totally different thing from a disease "consequent alone upon a diathesis" independent of exciting causes, as he puts it.

Niemeyer immediately and naturally proceeds to apply these conclusions to the question of hæmoptysis and its relation to phthisis, and we shall see very plainly from his statements on this subject what he thinks "we have to fight, and what we have to fight for" in the treatment of pulmonary hæmorrhage.

"Among the various foreign bodies which, by direct irritation of the walls of the bronchi and of the parenchyma of the lung lead to phthisis, *the blood which, after a hæmoptysis or pneumorrhagia, remains behind and coagulates in the bronchi and alveoli, exerts this influence the most frequently.* . . . I do not hesitate to say, *that in the majority of cases hæmoptysis is followed by a more or less serious irritation of the lung and pleura.* Since

my attention has been directed to the occurrence of these consecutive attacks of pleuro-pneumonia, I have, almost without exception, been able to find, on the second or third day after the hæmorrhage, an increase of the temperature of the body and of the frequency of the pulse, a disturbance of the general health, more or less severe pains in the lateral regions of the thorax, and frequently also fine moist râles, pleuritic frictions, or a slight dulness, with diminished vesicular or with bronchial breathing. Even in cases in which a longer time had elapsed since the hæmoptysis, it was generally easy to make out that in the next few days succeeding the hæmorrhage, more or less clear signs of inflammatory changes in the respiratory organs had appeared."

The following statement must be particularly observed in order not to misunderstand Niemeyer's views; "I am far from asserting that the pneumonic processes following an attack of hæmoptysis leave behind them in all, *or even in the majority of cases*, cheesy infiltrations leading to pulmonary phthisis. On the contrary, their most common termination is in *resolution*. Frequently all symptoms disappear in a few days and the patient is again convalescent.

"In other cases," he says,—which it must be noted, after the last statement, can only be *a minority*, though Niemeyer himself seems afterwards to forget this, and his followers, still more, omit to notice this striking admission of their leader—"in other cases, however, the rise of temperature and of the pulse continue for a longer time, the general health remains impaired in proportion to the continuance of the pyrexia, while slight pains in the chest, which the patients talk of as rheumatic, persist, the respiration remains hurried, and there is cough and muco-purulent expectoration. If besides these symptoms the percussion sound is found dull in a more or less extensive region of the chest, if the respiratory murmur is of an indefinite character, and diminished or bronchial, and if the patients are rapidly consumed by the increasing pyrexia with evening exacerbations and abundant night perspirations, there is reason to fear that the pneumonic infiltration has undergone cheesy metamorphosis, and that the patients have fallen into consumption. . . . most of the cases in which previously healthy persons are, immediately after an attack of hæmoptysis, seized with galloping consumption, cannot be otherwise interpreted than by assuming that *the blood which remained behind in the bronchi and alveoli has led to a pneumonia undergoing cheesy transformation, the retained blood and the products of inflammation afterwards breaking down.*"

"But it is not in all cases that pneumonic processes set up by blood remaining in the bronchi and alveoli, and the inflammatory product undergoing the cheesy metamorphosis, lead, by a rapid breaking down of the lung-tissue, to death with the symptoms of phthisis florida. Even in these cases the cheesy masses may, as has been described above, become more and more inspissated, and may be encapsulated by

proliferating connective tissue, or may, at a later period, be liquefied and reabsorbed, connective tissue filling up the defect. This termination in shrinking and induration, with which the patient may enjoy a relatively good state of health, shows itself during life by flattening of the affected part of the thorax, the dulness being permanent, and the respiratory murmur absent or diminished. . . .

"Lastly, it must be mentioned that hæmoptysis may not only tend to destructive and indurating pneumonia, *but also, at a later period, by cheesy deposits remaining behind it, to true tuberculosis.* I have some very striking instances of this mode of termination among my notes of cases.

"To guard against misunderstanding, I must in conclusion remark that in the above description I have only drawn attention to that relation of *hæmoptysis and pulmonary consumption* which of late has either not been recognised at all, or has been too little observed; and that I am far from considering this relation to be the only one existing between hæmorrhage from the air passages and pulmonary consumption. On the contrary, I consider an attack of hæmoptysis, especially one which occurs from trifling causes, to be of bad omen, even apart from the danger that blood may remain behind in the alveoli, because experience teaches that the morbid friability (hæmorrhagic diathesis) of the branches of the bronchial artery terminating in the bronchial mucous membrane, is, as a rule, associated with a tendency to inflammatory diseases of the pulmonary tissue, where nutrition depends on the bronchial arteries.

"I also willingly concede that attacks of bronchial hæmorrhage occur even more *frequently in the course of* pulmonary phthisis, *and in all its stages*, than they precede it. I will briefly and concisely sum up my views on the relation of bronchial and pulmonary hæmorrhage and pulmonary consumption in the following sentences:—

"1. Abundant bronchial hæmorrhage occurs more frequently than is commonly admitted in persons who neither are consumptive at the time of the hæmorrhage, nor become so afterwards.

"2. In many cases abundant hæmorrhages of the bronchial mucous membrane *precede* the commencement of pulmonary consumption without *any causal* connection existing between the hæmorrhage and the disease of the pulmonary tissue. Here the two processes have their origin in the same source, namely, in the combined disposition of the patient both to bronchial hæmorrhage and to pulmonary consumption.

"3. Capillary hæmorrhage, either bronchial or pulmonary, does not unfrequently lay the first foundation for pulmonary consumption in persons in whose lungs neither tubercles nor pneumonic deposits previously existed; this is brought about by blood which remains behind in the *alveoli*, as well as the products of the inflammation which

the blood caused, *undergoing cheesy metamorphosis.* ("Niemeyer states that it is not the blood coagulated in the *bronchi*, but *that remaining in, or sucked into the alveoli*, which he has accused of being the cause of inflammation; and he mentions that according to the testimony of many pathologists, blood is found in the alveoli soon after an attack of hæmoptysis." Note to p. 39 of Translation, by Dr. Baümler.)

"4. In the same manner do bronchial pulmonary hæmorrhages not unfrequently accelerate the course of an already existing pulmonary consumption. But it is only in rare cases that they appear at a period when the lung-disease is still latent.

"5. In some rare instances the hæmoptysis is not the cause but the *consequence* of the pulmonic processes which in their further course lead to consumption. Such cases are easily recognised, because a generally high pyrexia, and other symptoms of inflammation, accompany the onset of the hæmoptysis, or even *precede* it.

"6. That portion of blood which remains behind in the alveoli, and which, together with the pneumonic infiltration, undergoes cheesy metamorphosis, not unfrequently *gives rise to an eruption of miliary tubercules.*" (Niemeyer, op. cit.)

Closely following in the steps of Niemeyer, Dr. Baümler in 1869 reported three cases of "hæmoptysis followed by inflammatory changes in the lungs," which, he says, "would seem to support Professor Niemeyer's views . . . The question where in such cases the blood comes from—whether from the mucous membrane of the air passages, or from the rupture of a large vessel in a cavity, or from lung-tissue breaking down, is not intended to form part of the present communication, as our cases *do not furnish any conclusive evidence on this point.* I only allude to it as Professor Niemeyer's theory has lately been opposed by Dr. Rasmussen ("Edinb. Med. Journ.," Dec., 1868), on the ground, principally, that a bronchial hæmorrhage has never yet been demonstrated by post mortem examination. Although the positive proof is still wanting, yet I think the possibility of hæmorrhage from the bronchi in the same way as from the mucous membrane of the nose in epistaxis, must be admitted. But this question seems to me altogether of less importance than the other one, whether blood which is not expectorated, or rather which has been driven into the air-vesicles by aspiration, may give rise to an irritative bronchitis or pneumonia. This question seems to be answered in the affirmative by the above cases. If once admitted that hæmoptysis, no matter in what way arising, may lead to pneumonic changes in the lungs, then it is easy to understand how it may bring on phthisis in lungs previously healthy, and how it may aggravate any lung-disease already existing. Whether hæmoptysis will be followed by such results, and to what extent, may be supposed to depend upon the amount of the hæmorrhage and the locality in which this takes place in the air-passages, or in the lung-tissue, as

well as upon external circumstances, as, for instance, whether the patient keeps quiet and lies down, or continues his avocation. The further course of such inflammatory changes, when once set up, is, probably, apart from different external circumstances, *principally influenced by the patient's constitution.* . . .

"The history of the above cases would seem to lead to the following conclusions:—

"1. Profuse hæmoptysis is, in some cases, followed by a more or less extensive bronchitis, especially of the smaller branches, and by inflammation of the lung-tissue.

"2. These inflammatory changes are capable of a complete resolution, or—

"3. They may lead to a permanent infiltration and induration, with their consequences." ("Trans. Med. Chir. Soc.," vol. ii., 1869.)

Now, on carefully examining the details of these cases upon which Dr. Baümler relies for his support of Niemeyer's views, I find Case 1 quite defective as to the state of the lungs previous to the hæmoptysis; Case 2, a baker, and therefore predisposed to lung disease by his trade, is also quite defective in many points; Case 3 had six weeks at least of chest affection before the hæmoptysis occurred; and so far from these cases proving that blood left in the air vesicles "may give rise to an irritative bronchitis or pneumonia," there is no evidence that the structure of the lungs was not broken down by the hæmorrhage.

Dr. Weber immediately supplemented Dr. Baümler's records by a paper "On Hæmoptysis as a Cause of Inflammatory Processes and Phthisis, with remarks on treatment" ("Trans. Clin. Soc.," vol. ii., 1869), in which he reported three cases. "These three cases," he says, "coincide with those related by Dr. Baümler in this important peculiarity, that the lungs did not exhibit any signs of organic disease at the time that the first hæmorrhage occurred." But here I must again point out that there was the *history of previous lung disease*, which is of much more importance than the negative evidence of an absence of physical signs, which might have been delusive. "It was not till several days afterwards," Dr. Weber continues, "that symptoms of inflammatory processes of the respiratory organs manifested themselves, accompanied by a corresponding elevation of temperature. These processes were principally pneumonic, but there was also a considerable addition of pleuritic and of bronchial irritation. The pneumonic affection differed from typical croupous or lobar pneumonia in the irregularity of its course, in its protracted duration, and in its being scattered over different parts of both lungs instead of involving the whole of one lobe. In all these respects it resembled rather lobular pneumonia. The first two cases show, as two of Dr. Baümler's, that complete absorption and recovery can take place, though the time occupied by this process is, as just

mentioned, much longer than that occupied by a common lobar pneumonia. In the second case pleuritis with effusion took place more than four months after the hæmorrhage, at a time when all the active symptoms of the consecutive inflammatory affection had apparently long subsided; this pleuritis occurred on the same side which had been the seat of the former inflammatory process, and I cannot help bringing the two in connection with one another; the more so, as the first sign of the pleurisy consisted of pain and friction over the second and third rib, where there was still some slight dulness from the former affection, and where probably a nodule on the surface set up the pleuritic process, which at first was local and then became more general. The case shows how distant the later effects may be from the original cause.

"The third case, which at last terminated fatally, and which may be regarded as an example of a form of galloping phthisis, is still more instructive by the result of the post-mortem examination. The first attack of hæmoptysis was followed after several days by inflammatory symptoms in the lower part of the right, and the upper part of the left lung.

"After about three weeks the fever had ceased, and the curative process seemed to progress satisfactorily, when, owing probably to excitement, a fresh attack of hæmoptysis occurred, which again was followed, after a few days, by an inflammatory affection in the left lower lobe. He again seemed fairly to proceed towards recovery, when, after a long and excited conversation, a fatal hæmorrhage occurred. The post mortem examination showed cheesy nodules surrounded by infiltrated tissue, with the commencement of the formation of cavities in the upper lobe of the left, and in the lower of the right lung, being of the same age, and corresponding to the inflammatory processes following the first hæmorrhage seven weeks before death; there were further the changes of a later date in the lower lobe of the left lung, with the plugged bronchial branch, corresponding to the second hæmorrhage seventeen days before death; and there was, besides, the fresh blood of the fatal hæmorrhage, not only in the large bronchi, but filling also some of the smallest ramifications and groups of air-vesicles which were felt from without as hard nodules. . . .

"According to Niemeyer's view, pulmonary hæmorrhage, from whatever cause it may take place, can give rise to inflammatory processes by blood aspired into, and remaining in, the finest bronchial ramifications and air cells, acting there the part of an irritant. The inflammation set up in the surrounding district may undergo the usual retrogressive changes, and the products may be absorbed sooner or later, together with the metamorphosed blood, but it may also lead to the formation of cavities and to phthisis."

"In two of the cases related there was a great tendency to epistaxis,

and we may regard the hæmoptysis as having arisen from the mucous membrane of the bronchi in a similar way as epistaxis from that of the nose. The same explanation may be applicable to the third case, but it certainly is not applicable to all the cases of hæmoptysis, for in a great many of them the hæmorrhage is probably due to various morbid conditions of the vessels of the lung-tissue in consequence of inflammatory processes in the latter. In such cases, owing to local and constitutional changes already existing, it is of course less easy to show whether an attack of hæmoptysis gives rise to fresh inflammatory processes or not; but in several patients it has been quite evident to me that fresh inflammation has been set up by the hæmorrhage, and this is probably not very rarely the case.

"With all gratitude to Niemeyer I am, however, inclined to think that he goes too far when he says 'that most cases of hæmoptysis are followed by more or less violent irritation of the lungs and pleura.' Since my attention has been directed to this question, I have had the opportunity of carefully watching nine cases of more or less severe hæmoptysis during at least ten days after the occurrence of the hæmoptysis, and in five of them the hæmoptysis was not followed by any increase of temperature or other sign of pleuro-pneumonia."

Dr. Weber thus summarises the results of his observations:—

"1. That more or less violent hæmoptysis can occur without the existence of any disease of the lung-tissue from congestion of the mucous membrane of the bronchi.

"2. That in many cases the hæmoptysis passes off without leading to any inflammatory changes in the lungs or to any constitutional disturbances.

"3. That in other cases, however, the retention of effused blood within the lungs gives rise to inflammatory symptoms, especially broncho-pneumonia, mostly lobular, from irritation.

"4. That the products of this inflammation may be readily absorbed under favourable circumstances, while, in other cases, they form the origin of a consumptive process, *i.e.*, subacute or chronic phthisis.

"5. That pulmonary hæmorrhages, occurring in already diseased lungs, may likewise, but do not always, give rise to fresh inflammatory processes." ("Transactions of Clinical Society," vol. ii., 1869.)

In order to estimate the actual clinical value of the views of Niemeyer and his school, it is necessary emphatically to recall the passage (quoted at p. 90), in which Niemeyer says :—"I am far from asserting that the pneumonic processes following an attack of hæmoptysis leave behind them in all *or even in the majority of cases*, cheesy infiltrations leading to pulmonary phthisis. On the contrary their most common termination *is in resolution*." He then goes on to describe what he considers happens in "*other*" cases, and it is in relation to these that his special views are detailed, as I have already pointed out. (p. 90.) Yet he

afterwards seems to forget, and his followers seem never to have remembered, that, according to his own showing, whatever the explanation may be of what happens in these "*other cases,*" they constitute but a small and exceptional class. It is extraordinary to find, after the above plain declaration that these cases are *exceptional,* and belong to a small minority, that in the summing up of his views he entirely reverses the order of things, and says—"*In some rare instances the hæmoptysis is not the cause but the consequence* of the pulmonic processes which, in their further course, lead to consumption."

In this place I must ask the reader to turn back to Part I., where it will be seen that in the analysis of my tabulated cases (Table 1), the first hæmoptysis occurred after the date of heaviest weight in 87 per cent., and after the ascertained *first loss of weight* in 82 per cent.; and in the commentary upon these facts I pointed out that in this 82 per cent. of the cases it is self-evident *that some diseased condition causing constitutional decline existed before the occurrence of hæmoptysis,* and that, therefore, in them at least, we have no excuse for looking to the hæmoptysis as its cause. By reference to my commentaries on the cases (18 per cent.), in which this was not so clear, it will be seen that there were explanations to be found in nearly all, without resorting to the hæmoptysis as the most probable cause of the subsequent advance of lung-disease.

Speaking of the relation of hæmoptysis to pulmonary phthisis, Dr. M. Heitler ("Pester Med. Presse," 1876), "asks what influence has hæmoptysis upon the development of pulmonary consumption, and can hæmoptysis be considered an etiological factor of pulmonary phthisis? On this question, the most contradictory views having been held, before giving his own opinion he made an historical review; he mentions that Hippocrates had admitted the possibility that blood emptied into the lungs may lead to pulmonary phthisis. He also quotes the views of Laennec, Andral and other investigators, down to the present time. Virchow, and after him Felix Von Niemeyer, were of opinion that hæmoptysis may be the cause of pulmonary phthisis—that the blood coagulates in the bronchi and alveoli after hæmoptysis, causing pneumonic processes. Traube and Skoda objected to this view. Dr. Heitler also mentions the different experiments and conclusions of Woldenberg, Perl, Lipmann, and Sommerbrodt.

"He then relates his own clinical observations, and as an instance, he names hæmorrhagic infarction, which he says never leads to true pulmonary consumption. But he cannot answer the question whether hæmoptysis occurring without palpable cause in a strong individual apparently quite healthy before, or happening in a person who has an aptitude to phthisis, will be the next etiologic event to a subsequent consumption. He thinks on the contrary that hæmoptysis is only an accidental manifesta-

tion, *an initial symptom*, whilst small deposits in the lungs have been present before the hæmoptysis. He narrates a very instructive case, and says that he adopts the views of those authors who do not consider hæmoptysis a productive factor of pulmonary phthisis." ("Dr. Dobell's Reports on Diseases of the Chest," 1877.)

From observations on 238 patients with pulmonary phthisis treated in the Ospedale Maggiore di Bologna in 1874-1875, Dr. Mazzotti concluded that "Hæmoptysis cannot be considered as a direct cause of phthisis."

A similar opinion has been carefully arrived at by Dr. Hertz, and is well stated in the following quotation from Ziemssen's Dictionary, vol. v.

"A critical examination, however, seems to impugn the correctness of this very plausible proposition, founded on such cases as the above. (Niemeyer's, Cases of Phthisis ab Hæmoptoë.) The perfect health of the patients is a very relative and subjective one, and every physician in large practice knows for how long a time persons with commencing phthisis go round with a slight cough, which they think nothing of, and, regardless of all exposure, do not seek medical advice. The hæmoptysis first arouses them from their indifference, and in many cases, by close questioning, they will acknowledge that they have coughed for a long time, have even at times felt feverish and have lost strength. A physical examination then not unfrequently reveals an old unsuspected focus of disease. Such cases as the above mentioned admit, too, of another interpretation in consequence of the careful observations of Buhl, whereby new light has been shed upon the different forms of pneumonia, and their signification as factors in the production of phthisis. Buhl justly makes the distinction between catarrhal (superficial) and desquamative (parenchymatous) pneumonia, that the *former* never gives rise to extensive destruction of lung-tissue, while the *latter* very frequently eventuates in phthisis. He also tells us that *desquamative pneumonia, resulting in caseous degeneration*, may, like croupous pneumonia, take place so very suddenly that it is sometimes found occupying a whole lobe, sometimes only a portion of one. Therefore the sudden change in the physical symptoms cannot be ascribed only to the hæmorrhagic infiltration, for it may just as well be, and more frequently is, dependent upon the inflammatory infiltration, of which the hæmorrhage itself was a consequence. Moreover, *cheesy degeneration never originates in catarrhal, but only in destructive desquamative pneumonia*. Now blood extravasated into the alveoli of the lungs only gives rise to catarrhal pneumonia, a fact which has been established by the experiments upon animals which have been instituted in different quarters for the elucidation of this question." . . . .

"According to this explanation (Perl, Lipmann, Waldenburg),

severe pulmonary hæmorrhages in man, by causing accumulations of blood in the bronchioles and alveoli, give rise to a condition of superficial irritation, which confines its action to the epithelium, and may lead to caseous metamorphosis of the contents of the bronchioles and alveoli. *This mass is, however, soon removed by absorption and expectoration,* without causing any real destruction of the alveolar and bronchial walls—in other words, phthisis. *We must, therefore, distinctly deny that bronchial hæmorrhage may give rise to pulmonary phthisis.* All those inflammatory symptoms mentioned by different authors—stitch in the side, increased frequency of pulse, rise of temperature, and general indisposition—which are observed to accompany, or to come on a few days after, an hæmoptysis, are due to disease of the lungs, independent of the hæmoptysis, and have no further connection with the latter." (Hertz, Ziemssen's Dict., vol. v., pp. 308-310.)

Now, I do not pretend to say that the present condition of our knowledge of etiology, pathology, or histology, would justify me in either positively denying or positively accepting the hypotheses and theories by which Niemeyer or Rindfleisch, or Rokitansky or Virchow, and their followers or opponents in this and other countries, attempt to explain the changes in the lungs and in the constitutional health which follow in the track of hæmoptysis, pneumonia, and catarrh, *in those exceptional cases* in which these phenomena appear to stand first in the clinical history of consumption. But at present my own very decided opinion is that, if hæmoptysis, pure and simple, is ever the fons et origo in the production of pulmonary consumption, it is in consequence of *the disintegration of the delicate lung-structure* by the blood, to an extent beyond the power of repair, and that it is this destruction of tissue, strangely ignored by Niemeyer, and not the presence of the blood itself, which is the primum mobile in the case. Assuming this to be correct, all degrees of coarseness, or of fineness, in the disintegrating process may exist, and the course and symptoms of the case will correspondingly differ, as we see in practice. Thus, in the case of hæmorrhagic infarction, gangrene, and excavation may rapidly occur in the infarct and spread to surrounding parts with symptoms simulating those of rapid consumption. "According as an embolus comes from a purulent, gangrenous, or other source, and is loaded with septic matter, the point of the arterial wall with which the coagulum is in contact, first suppurates or sphacelates. Subsequently the suppurated or gangrenous process implicates the neighbouring lung-tissue. Hence, according to the nature of the embolus, the production of purulent pneumonia, of abscess of the lung, or of pulmonary gangrene. (M. E. Lancereaux, " Traité d'Anat. Pathol.," 1875, 1877.)

In the case of sudden and accidental rupture of a large vessel, the rush of blood into the fine tubes and alveoli may at once

hopelessly break up the delicate structure of the lung, débris of tissue and débris of blood being then stirred up together with atmospheric air by every act of respiration, and decomposition and its consequences following as a natural result, accompanied by symptoms of pulmonary consumption. Such a case would doubtless be classed by Niemeyer as one of the cases in which a person "immediately after an attack of hæmoptysis, is seized with galloping consumption," and which, according to him, "cannot be otherwise interpreted than by assuming that the blood which remained behind in the bronchi and alveoli has led to a pneumonia undergoing cheesy transformation, the retained blood and the products of inflammation *afterwards* breaking down." Now, if we say instead that the hæmorrhage, by hopelessly breaking up the lung-tissue, produced inflammation and its consequences, we shall be just as correct clinically, and, as I believe, much nearer the truth pathologically.

In accordance with these observations are the admitted facts that "the after effects of cavernous hæmorrhage are severe." (Flint.) "Pulmonary hæmorrhages occurring in already diseased lungs may likewise, but do not always, give rise to fresh inflammatory processes." (Weber.) "Bronchial and pulmonary hæmorrhages not unfrequently accelerate the course of an already existing pulmonary consumption." (Niemeyer.) "When, however, we classify the deaths according to the stages the patients were in when the hæmorrhage took place, we perceive the significance which attaches to the state of lung at the time of the occurrence :—

Stage.	No. of Deaths.	Average duration. Years.	Months.
1	26	9	2·11
2	16	7	4·62
3	21	7	1·42

"We here see that hæmoptysis occurring in the second or third stages is more likely to curtail the duration of the disease than in the first." (C. J. B. and C. T. Williams, "Pulmonary Consumption," 1871.) The rational explanation of these facts, in my opinion, is, that in these cases the blood being poured into the midst of lung-tissue already broken down by disease, there is nothing to bar the surrounding parts from extensive and hopeless disintegration.

Again, the hæmorrhage may be much more insidious, proceeding from the finest capillaries, and insinuating itself among all the most delicate structures of the lung. In this case, supposing the blood to migrate from the vessels without rupture, or to proceed from simple rupture without disintegration, there can be no reason to doubt that it would be as easily and harmlessly removed, by the marvellously perfect

peri-alveolar and perivascular lymphatic arrangements of the lungs, as we so frequently see effused blood removed under much less favourable conditions in other parts. But, supposing this fine interstitial hæmorrhage to produce disintegration of tissue, breaking down the walls of vessels and alveoli; the products of such destruction—the fine débris of tissue mingled with blood and air—may have a very different effect; may lead to irritation and hyperplasia of the adenoid tissue of the lungs; and thus, possibly, may be simulated the effects of the disintegration of lung-tissue by oxidation, which I have suggested and described as most probably the first change of structure in true tubercular consumption of constitutional origin.

This last class of cases would, I suppose, be classed by Niemeyer under his sixth heading:—"That portion of blood which remains behind in the alveoli, and which, together with the pneumonic infiltration undergoes cheesy metamorphosis, not unfrequently gives rise to an eruption of miliary tubercles."

It is easy to see that by the above method of explaining *the exceptional cases*, they are brought into harmony with those which follow the general rule, at the same time that there is nothing inconsistent with the observations of the various authors referred to, so long as these are confined to clinical facts.

I have devoted so much space to the views of Niemeyer and his school, not only because they have been so much propagated of late, but because his observations are of the utmost importance to the questions, "what we have to fight," and "what we mean to fight for," in cases of hæmoptysis.

When Niemeyer says (see page 89), "I do not hesitate to say that in the majority of cases hæmoptysis is followed by a more or less serious irritation of the lungs or pleura," I do not hesitate to say that, according to my own very large experience, he is quite wrong. And Dr. Weber, doubting the correctness of this statement of Niemeyer's, says, (see page 95) "since my attention has been directed to this question, I have had the opportunity of carefully watching nine cases of more or less severe hæmoptysis, during at least ten days after the occurrence of the hæmoptysis, and in five of them the hæmoptysis was not followed by any increase of temperature or other signs of pleuro-pneumonia."

But, while denying that the occurrences described by Niemeyer *are the rule*, I am quite as certain that hæmoptysis is *occasionally* followed "on the second or third day after the hæmorrhage by an increase of the temperature of the body and of the frequency of the pulse, a disturbance of the general health, more or less severe, pains in the lateral regions of the thorax, and frequently, also, fine, moist râles, pleuritic frictions,

or a slight dulness, with diminished vesicular or with bronchial breathing, etc." (Niemeyer.) That statement is certainly clinically correct. In Dr. Weber's cases, "It was not till several days afterwards that symptoms of inflammatory processes of the respiratory organs manifested themselves, accompanied by a corresponding elevation of temperature. . . . These processes were principally pneumonic, but there was also a considerable addition of pleuritis and of bronchial irritation; the pneumonic affection differed from typical croupous or lobar pneumonia in the irregularity of its course, in its *protracted duration*, and in its being scattered over different parts of both lungs instead of involving the whole of one lobe" (see p. 93), such symptoms, in fact, as we might expect to follow the damage done—the bruises, destruction of tissue, and echymoses—by the hæmorrhage.

I wish to draw particular attention to these exceptional effects of hæmoptysis, as things which may occur in any case, for which, therefore, we ought always to lie in wait, and constituting, when they occur, some of the most serious "things we have to fight," and their prevention forming one of the most important "things we mean to fight for," in our treatment of Pulmonary Hæmorrhage.

I have reserved for this place the more serious consideration of that phase in the history of lung disease in connection with hæmoptysis, to which I have more than once referred in passing—I mean that stage, so critical in all its aspects, in which hæmoptysis, like the first shell thrown into a citadel, becomes the ghastly signal that an unappreciated constitutional difficulty has suddenly culminated in a vital war.

A vast traditional popular experience has but too justly estimated the significance of this alarming herald. Even Niemeyer is obliged to guard himself from misunderstanding, by saying, "I consider an attack of hæmoptysis, especially one which occurs from trifling causes, to be of bad omen, even apart from the danger that blood may remain behind in the alveoli."

Speaking of the value of symptoms as prognostics, in his admirable treatise "On the Elements of Prognosis in Consumption," my friend Dr. J. A. Pollock says:—"Hæmoptysis is often one of the earliest (symptoms), always the most alarming, to the patient. It occurs in about 63 per cent. of all cases of phthisis, and is more frequent (as 3 to 1) in the first stage. It is due at this period to extreme congestion of the pulmonary vessels, arising from the local inflammatory action accompanying the deposit of tubercle."

On this subject, too, Professor Austin Flint speaks from his large experience, and attempts an explanation. "Hæmoptysis," he says, "occurs in some cases when not only the symptoms of pulmonary disease

are wanting, but the result of physical exploration of the chest is negative. In a certain proportion of these cases it is probable that the hæmorrhage is connected with a small affection which is latent as regards both symptomatic phenomena and physical signs. . . .

"That there may be scattered tubercles in the lungs when their presence is not revealed by auscultation and percussion, must be conceded. Taking, now, into consideration the cases in which hæmoptysis is the initial symptom of disease, cough and other symptoms following directly after this event, it is a rational supposition that the *congestion supposed to give rise to the hæmorrhage is a primary effect of* the development of phthisis. . . . And again, it is to be considered *that a local congestion implies a local causative* condition of some kind; in other words, the occurrence of a local congestion as a primary condition is improbable. Moreover, that a local affection involves a local hyperæmia is rendered probable by the fact that, in general, it occasions a circumscribed bronchitis, believing, as clinical observation warrants us in doing, that the cough and expectoration in early phthisis proceed from a secondary bronchial inflammation. Accepting, as a conclusion justified by clinical facts, that hæmoptysis is in general an effect of a local congestion induced by the primary local affection in phthisis, this congestion is probably active, that is, the hyperæmia is due not to venous obstruction, but to an undue determination of blood to the congested portion of the mucous membrane.

"An explanation which has been offered of the dependence of hæmorrhages upon tubercles is as follows: the primary seat of tubercles (granulations) being the small arteries, the coats of the latter are thereby weakened, and rupture is, in consequence, liable to take place. Another explanation is, that the production of tubercle leads to the occlusion of small vessels, and hence the pressure of the blood in the proximate unobstructed arteries (collateral fluxion) leads to rupture." (Op. cit., p. 82.)

The late Dr. Fuller says that, according to his experience, "if those cases are excluded in which the sputa are slightly specked or streaked with blood—as in certain instances of severe congestive bronchitis—or are rusty-coloured from admixture with it, as is often seen in pneumonia; the only exceptions I am disposed to admit to the fearful significance of hæmoptysis are those in which it results from blows on the chest, or from mechanical injury to the lung, or in which it occurs in women vicariously to the menstrual discharge. In these cases undoubtedly it seems occasionally to take place independently of structural pulmonary disease." (Op. cit., p. 258.) And Dr. Walshe's experience is that "there is no established morbid anatomy of the most frequent variety of hæmoptysis—that depending on the process of tuberculisa-

tion. Molecular ruptures of the capillary vessels of the parenchyma enclosed within softening tubercular infiltrations are, doubtless, the cause of the discharge of blood in phthisis, except in those cases where a vessel of some size undergoes breakage." (Op. cit., p. 395.)

I must confess that, after a careful review of all the explanations that have been attempted by various authors of this *initial hæmoptysis*, I am obliged to return to the one originally suggested by myself, as affording the most rational and satisfactory mode of accounting for the phenomenon (See Part IV. for amplification); I mean, the damage to the finest vessels of the pulmonary alveoli, by oxidation of albuminoid tissue during the interchange of gases between the air in the alveoli and the blood deprived of its normal amount of fat; *i.e.*, by the first stage of pulmonary tuberculisation. Whatever may ultimately prove to be the correct explanation of the loss of fat, in what I have called "the true first stage of consumption"—that which *precedes* tuberculisation,—there is ample proof that such a loss occurs, and that the deficiency is first felt in the blood which reaches the right heart; and there is every reason, physiologically, to believe that the extremely delicate albuminoid tissue, which alone separates the air from the blood and through which oxygenation takes place, would be the first to become disintegrated; while it is admitted by all pathologists that this is the locality in which tuberculisation begins.

That such a conjunction of circumstances would produce hyperæmia, and that hyperæmia would increase the oxidation, cannot be doubted;* and all the clinical facts are entirely in accord with this suggested explanation of the occurrence of hæmoptysis at this stage of consumption, preceded, as it has been shown to be, by loss of weight in so large a majority of cases. (See Table 1.)

The practical lessons pressed upon us by this view of the case are of the utmost importance, viz.—1. That the hæmorrhage itself in these cases may be advantageous, by temporarily relieving the hyperæmia. (That it is so is proved by clinical experience.) 2. That if the hæmorrhage is moderate it is better not to check it by merely hæmostatic remedies. 3. That, whether the hæmorrhage is moderate or profuse, the most important remedies are such as diminish the activity of the pulmonary circulation and diminish the influx of highly oxygenated atmospheric air into the alveoli, *i.e.*, stay the process by which the vascular tissue is being disintegrated; and 4thly and chiefly, that the constitutional state, the deficiency of fat in the blood, claims our

* The late Dr. Barlow observed that "there almost always exists a considerable hyperæmia of the bronchial mucous membrane at the commencement of phthisis." ("Guy's Hospital Reports," Series I., vi., p. 210.) "And, in almost all cases, a considerable hyperæmia of the alveolar capillaries." (Dr. Shepherd, "Goulstonian Lectures," 1877.)

most prompt and active interference, if we mean to prevent a recurrence of the hæmorrhage and the further progress of that war upon the lung-tissues of which it is the signal. "What we have to fight and what we mean to fight for" are thus plainly set before us in theory; and clinical experience tells us that treatment so dictated is that which is attended with the most brilliant results. (See "Treatment of Loss of Weight and Lung Disease," Part V.)

In concluding this part of the subject I must refer to what we have already said concerning the etiological investigation of consumption, and the position of hæmoptysis as a symptom and as a cause of lung disease. (See Part I., pp. 25, 26.)

PART III.

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HÆMOPTYSIS AND PULMONARY CONSUMPTION.

TREATMENT.

HÆMOPTYSIS AND PULMONARY CONSUMPTION.

PART III.

"The arms with which we have to fight and how best to use them."—Remedies used by different Practitioners in this and other countries: Aperients, Atomised Fluids, Alum, Antiseptics, Acids (Gallic, Sulphuric, Tannic, etc.), Canada Balsam, Copper, Climate, Cold, Counter Irritants, Dry Cupping, Diet, Digitalis, Emetics, Ergot and Ergotine, Eucalyptus Rostrata, Elastic Compression, Heat to Spine, Hypodermic Injections, Iron, Ipecacuanha, Kashanda, Leeches, Ligation of Limbs, Lead, Mineral Waters, Quinine, Rest, Salt, Stimulants, Syncope, Sedatives, Turpentine, Urine, Veratrum Viride, Venesection.

Treatment recommended by—Austin-Flint, Annelli, Baglivi, Drasche, Fuller, Graves, Gerhardt, Hertz, Hirschfeld, Laennec, Lassalas, Massari, Massina, Muller, Oppolzer, Peter, Richelot, Rogers, Trousseau, Williams, Weber, Williamson, Waters, Ziemssen.

Treatment recommended by the Author—Avoid Amplification of similar remedies, Dangers of Elastic Compression, Dangers of Freezing Chest, Dangers of Internal Use of Iron, Modus Operandi of Remedies and their appropriate Adaptation to Special Cases.—Practical Summary.

HAVING, then, formed a fair notion of "what we have to fight, and what we mean to fight for," we come to the questions—"What are the arms with which we have to fight? and What is the best way to use them?"

If we turn to that remarkable monument of persevering industry lately published by the New Sydenham Society, "The Medical Digest" of my friend Dr. Richard Neale, we shall find, from the following list of remedies enumerated under the head of "Treatment of Hæmoptysis," that we have no reason to complain of lack of arms :—

"1. Heat to the spine. 2. Increased respiratory efforts. 3. Aperients. 4. Atomised fluids. 5. Emetics. 6. Digitalis. 7. Gallic acid. 8. Secale. 9. Veratrum viride. 10. Tinct. larici. 11. Tannin. 12. Turpentine. 13. Ozonized turpentine. 14. Plumbi acet. 15. Ferri persulph. 16. Ferri. tinct. 17. Cupric sulph. 18. Urine (used by Chinese and Malays). 19. Climate."

How these remedies are usually adapted and administered by our own countrymen, and by our American and Continental neighbours, may be well seen by the following abstracts and quotations:—

The late Dr. Fuller, in his comprehensive work on diseases of the chest, 1862, says—

"If the hæmorrhage is profuse . . . the sole point to be decided

is—How to stay the bleeding, and obviate the tendency to death. A variety of remedies have been employed for this purpose, amongst which I may mention, as having received the greatest amount of testimony in their favour, venesection, cupping, dry cupping, the application of ice down the spine, tartar emetic, acetate of lead and opium, gallic acid, sulphuric acid, alum, matico, ergot of rye, and turpentine. My own experience leads me to testify most strongly in favour of repeated dry cupping, aided by the application of ice down the spine, and by the internal administration of digitalis (ʒj. to ʒij. of tinct., or grs.vj. to grs.viij. of the powder). If this fails, full and frequent doses of gallic acid, or of lead and opium, may be given *if the circulation is much accelerated*, and of spirit of turpentine if the bleeding is unattended by vascular excitement. The gallic acid should be given every hour, in 8 or 10 gr. doses, until the hæmorrhage is subdued, or until a dark green colour in the sputa indicates its action on the system; or, if lead is employed, it should be given in doses of two or three grains, combined with dilute acetic acid and laudanum, after the method recommended by the late Dr. Anthony Todd Thompson, and the dose should be repeated every two or three hours. . . If turpentine is had recourse to, it should be administered at brief intervals, in ʒss. doses. . . When the hæmorrhage is very profuse, and infiltration and rupture of the pulmonary tissue takes place, the blood may not only break up the tissue of the lung, but may burst through its serous envelope, and escape into the pleural cavity. Many cases are on record of death produced in this manner." (See Dr. Paterson's "Observations on Pulmonary Apoplexy proving Fatal by Rupture of the Periphery of the Lung by Effusion of Blood into the Pleura." "Edin. Med. and Surg. Journ.," Jan. 1846.)

Dr. Weber, in the paper already quoted at p. 95, sums up by saying that "with *regard to treatment*, perfect rest is the most important element, and in many cases sufficient. . . In others, especially if the hæmoptysis does not soon cease, and remedies like gallic acid and ergotin do not succeed, and fresh attacks follow one another, the *emetic* is a remedy of great value as well in arresting the hæmorrhage as in clearing the bronchi from effused blood, and thus preventing inflammatory processes. . . . In the treatment, as well of the tendency to hæmoptysis as of the effects of the latter, the Alpine climates deserve the more general attention of the profession."

With regard to the use of emetics we find Trousseau ("Lectures on Clinical Medicine," vol. iii., New Sydenham Society, 1870), whose clinical experience was exceptionally large, speaking in the following very strong terms, "When the parenchymatous hæmorrhage is obstinately recurrent, ipecacuanha is a remedy which seldom fails. . . . As *an emetic* it is more to be relied upon in the treatment of what is called bronchial hæmorrhage. . . . You remember an old

man aged 62, who lay in bed 7, St. Agnes Ward. He was resident in the hospital from the beginning of 1863; and during the preceding year, he had asked my advice on account of his having serious symptoms of tubercular disease. For several years he had been phthisical; and from time to time, the upper lobe of the right lung, *in which were large cavities,* became the seat of acute inflammation, by which life was placed in jeopardy. Twice within the space of five months he had frightful hæmoptysis, twice it was immediately arrested by four grammes (rather more than a drachm) of powder of ipecacuanha administered within the space of half-an-hour, in such a way as *to cause violent vomiting.* A similar result was obtained, you remember, in the young man who occupied bed 8 in the same ward, and also in another patient now occupying bed 16. . . . Some months ago, I was summoned in consultation to a provincial town, in the case of a tuberculous man, aged 42, who had had hæmoptysis going on for forty days. A great diversity of very rational plans of treatment had, in succession, been fruitlessly employed. I recommended three grammes (46 grains) of ipecacuanha to be divided into four packets, one of which was to be given every ten minutes. The hæmoptysis had ceased before the last vomiting took place, and from that time, when it did recur, it was only to an insignificant extent. . . .

"Should, however, there be a relapse of the hæmoptysis, the use of the ipececuanha must be resumed. I never hesitate in such circumstances to return to it two or three times, if necessary, and I have never yet seen the least inconvenience result from this proceeding. Gentlemen, this is not a new method of treatment. For the last two centuries, physicians have lauded the Brazilian root as a remedy in all forms of hæmorrhage, and Baglivi says:—'Radix ipecacuanhæ est specificum et quasi infallibile remedium in fluxibus dysentericis, aliisque hæmorrhagiis.' Nevertheless, gentleman, the hand trembles when it administers this remedy for the first time in the treatment of hæmoptysis." Trousseau goes on to describe his further treatment as follows: "We are accustomed to prescribe the greatest possible quietude to our hæmoptoic patients; we counsel them to keep absolute silence; we tell them to restrain the slightest effort to cough; the very most we allow them to do is to breathe, and so frightened are we of congestion, even passive congestion of the lung, that we act as if we placed them in peril by permitting them to make the very slightest effort. Yet here we are giving a medicine which produces vomiting, during which the face swells, the blood stagnates in the veins by which it is being conveyed to the auricles, and consequently the pulmonary veins become distended. One might expect that such treatment would cause the hæmoptysis to return in a much more profuse degree; but in place of this it is stopped in nearly every case. Here is one proof more

of the small reliance to be placed on theoretical explanations, and of the value of empirical facts, without which, indeed, therapeutics would be a nullity."

Concerning the treatment of hæmorrhagic infarction and its attendant hæmoptysis, Professor Gerhardt says :—

"The infarction once established, if the embolus has not brought with it any provocatives of decomposition, it requires only favourable external circumstances in order to its cure. The most important of these are pure air and rest of the body. . . . With the body at rest the diminution of the pulmonary circulation may still allow of sufficient aëration of the blood, but every muscular exertion stirs up a dyspnœic attack with the most violent respiratory movements. We recognise, therefore, in a state of bodily rest, and in the injection of morphia at the onset, the proper conditions for the cure of infarction. . . . You will treat violent hæmorrhages with inhalation of *perchloride of iron*, and the use of tonics will be, in various ways, indicated. Under certain circumstances you will seek to hinder the gangrenous softening of the infarction *by antiseptic inhalations*. But on the whole this proposition is applicable :—"*The infarction, which is not infected either by the embolus or by the inspired air, cures itself. The danger lies in the embolism. If the infarction forms, the danger is already past.*" (Op. cit.)

Dr. Austin Flint, speaking from the results of his very wide clinical experience, says with reference to the hæmoptysis of phthisis, "The mental disturbance which an attack of hæmoptysis is apt to occasion, especially a primary attack, renders it desirable for the physician to make such encouraging statements as are warranted by clinical facts. Assuming either that phthisis is not established or that it is not advanced, we are authorised, in the first place, to assure the patient, even when the hæmoptysis is profuse, and repeated for many successive days, that immediate danger from the hæmorrhage is almost *nil*. The very great infrequency of the cases in which death is caused by the loss of blood is to be borne in mind. In the second place, we may say, if there is evidence sufficient to show the existence of pulmonary disease, that with the occurrence of hæmoptysis the chances of recovery, arrest, or tolerance are greater than if hæmorrhage did not occur. Here, too, a fact is to be borne in mind, namely, that in rare instances an attack of hæmoptysis is accompanied and followed by symptoms of great severity, and death takes place within a short period. Exclusive of these cases, the symptoms following hæmoptysis sometimes show either the development or an increase of the disease, and sometimes, if the disease already exist, the symptoms denote improvement. The statement that relief may follow a hæmorrhage will sometimes tend greatly to diminish undue apprehension. . . . The physician should consider that, as a rule, sooner or later phthisis becomes developed, and the importance of giving proper heed

to the attack of hæmoptysis is to be impressed. It is wise always to act as if phthisis were threatened, adopting such hygienic measures as may be likely to ward off danger in that direction.

"In an attack of hæmoptysis the patient is usually intensely anxious to have the hæmorrhage arrested. To arrest the hæmorrhage is a therapeutical object, in view of the results of the foregoing clinical studies, for these studies have developed no facts leading to the conclusion that the continuance of the profuseness of the hæmorrhage is ever desirable. So common and widespread is a confidence in the use of common salt, that the physician generally finds it has been used more or less freely before he has seen the patient. The usual mode of using it is to take the fine salt in substance into the mouth. . . . Repose is to be enjoined, although the instances in which persons who become accustomed to attacks, and do not pay much attention to them, are sufficiently numerous to show that the hæmorrhage is not always kept up or renewed by physical exertions. The common idea is, that an attack generally depends on some exciting cause (an idea which the study of cases does not sustain), and hence the importance of avoiding all exertion is exaggerated.

"This idea leads not infrequently to too prolonged quietude and the observance of other needless precautions against renewed attacks after the hæmorrhage has ceased. It is customary, and with reason, to direct bland articles of diet, which are to be taken cold. Milk should be the basis of the diet during the continuance of the hæmoptysis. If persons are not afflicted unpleasantly by opiates, these are useful by allaying nervous excitement. In general they should enter, more or less, into the treatment during an attack of hæmoptysis.

"The more active measures for the arrest of the hæmorrhage are those, first, which have reference to the general and the pulmonary circulation; second, the introduction into the blood of remedies supposed to have a hæmostatic operation; and, third, topical styptic application.

"Venesection was formerly employed, and sometimes largely, for the arrest of bronchial hæmorrhage. Several of the cases in my collection were recorded so long ago that the employment of this measure enters into the histories. It was also customary to employ local blood-letting by means of cups and leeches. It would require not a little hardihood to advocate bleeding for this end at the present time. I have no disposition to do this. But it may be remarked, that the injudiciousness of abstracting blood for this purpose is perfectly consistent with its efficaciousness in certain cases. The obvious explanation of this statement is, that the evils of blood-letting may outweigh the advantage gained by accomplishing the object. With the views respecting blood-letting which now prevail, it is not necessary to discuss this measure. This is the more unneces-

sary, because it is probable that whatever efficaciousness blood-letting may have, may be obtained without the abstraction of blood. Ligation of the limbs, so as to detain blood in the veins beyond the ligatures, secures the effect of venesection. I have witnessed an immediate arrest of hæmorrhage by resorting to this measure. The patient, whom I saw with Dr. Varsch, of Jersey City, had profuse hæmoptysis, recurring after short intervals for several successive days. Loose ligatures were applied to the four extremities, and these were tightened whenever the hæmorrhage returned. A hæmorrhage occurred during my visit, and it ceased almost instantly on tightening the ligatures. Detaining thus the blood in the four extremities should only be done by the physician. The effect on the circulation is very great, and without watching, fatal syncope might be induced." (Op. cit.) Dr. Flint does not seem to have thought of the danger of producing clots in the veins which, on loosening the ligatures, might rush into the right heart. Cases have been recorded of death from this cause after ligature of the limbs.

Dr. Massari ("Gazetta Medica Italiana," March 17, 1877,) points out a serious inconvenience in the methodical compression of the extremities by elastic bandages; and mentions the case of a pregnant lady who became very anæmic from considerable losses of blood occasioned by a placenta prævia. To arrest the hæmorrhage, the plan of Dr. Muller was adopted, and an elastic bandage applied to compress both legs, after which the anæmic condition markedly changed for the better; but soon, owing to local pains, the elastic bandages had to be temporarily removed, and on re-application of the pressure, it was well borne for thirty-two hours after the child's birth, when the patient suddenly collapsed, had difficulty of breathing and palpitation of the heart; the immediate removal of the elastic bands gave no relief whatever, and the patient died in two hours. Post mortem: large clots were found in the pulmonary artery. Dr. Massari therefore counsels caution in the prolonged application of elastic bands in all cases." ("Embolism of the Pulmonary Artery in consequence of Elastic Compression of both Lower Extremities," "Dr. Dobell's Reports," 1877.)

Dr. Flint says, "Of course the ligation is to be continued for a short time only, the effect on the pulse being constantly watched. The effect of the abstraction of blood by cups or leeches may be obtained by dry cupping, and to a certain extent by large sinapisms. These substitutes for blood-letting, more especially the ligation of the limbs, are admissible only when the pulse has a certain degree of fulness and strength; they are contra-indicated if the pulse is small and weak. There is a marked difference in different cases with respect to the activity of the circulation, or, to speak more definitely, of the heart, as represented by the character of the pulse. In attacks of hæmoptysis with a full, strong,

pulse, the *direct cardiac sedatives* may be employed with advantage, namely, aconite, digitalis, and veratrum viride; also the *indirect sedatives*, namely, saline purgatives and nauseants. The employment of these remedies is to be regulated by the circumstances proper to individual cases, such as the amount of hæmorrhage, the tendency to its renewal, the strength of the patient, etc. In the category of measures having reference to the circulation, belongs the application of cold to the chest. This may be resorted to especially if the hæmorrhage persist despite the employment of other measures. The most convenient and effective mode of applying cold is by means of compresses wet with iced water, which are to be renewed every few minutes, or as soon as they acquire warmth from the body. The applications may be continued for several hours, if they do not cause discomfort. They will diminish the heat of the body if this be morbidly increased. It is hardly necessary to add that none of the potential measures are indicated when the hæmorrhage is slight or moderate.

"The more prominent of the hæmostatic remedies are the tannic or gallic acid, the acetate of lead, the pernitrate or persulphate of iron, and ergot. After a pretty large experience in the use of these remedies, I find it difficult to form any positive opinion as to their value severally and relatively. They often seem to have no effect as hæmostatics, and when the hæmorrhage ceases under their use, there is always room for the supposition that the cessation is due to an intrinsic tendency thereto, rather than to the remedies.* I am far, however, from being disposed to deny that they exert more or less influence in the arrest of bronchial hæmorrhage. It may seem that I dismiss the consideration of these remedies with undue brevity; but I am unable to add further remarks which would have importance in my own estimation.

"As regards the topical application of styptics to the bronchial mucous membrane, I shall dismiss the consideration of them with a few words. The application is, of course, to be made by means of atomized liquids. The inhalation of spray from liquids holding in solution alum, gallic acid, and astringent preparations of iron has been employed with success for the arrest of bronchial hæmorrhage. My practical acquaintance with this mode of treatment is too limited for me to speak of its merits. I have seen apparent success from the inhalation of the vapour of turpentine in arresting a persisting slight hæmorrhage. I have also known the inhalation of atomised solution of persulphate of iron promptly efficacious in profuse hæmoptysis.

"After bronchial hæmorrhage has ceased, it is not judicious to continue the use of remedies with a view to forestall the possible recurrence of hæmoptysis; nor is the continuance of rigid rest and a low diet advisable. The danger which an attack of hæmoptysis opens up

* This might be said of all remedies!

when it occurs in one apparently well, is the development of phthisis, and this danger points to the importance of active habits of life, together with full alimentation, and, perhaps, the use of alcoholic stimulants within certain limits. The more the system is invigorated by a hygienic course adapted to this end, and the more the danger of phthisis is lessened thereby, the less the liability to recurring attacks of hæmoptysis. If, as is true in most instances, hæmoptysis has either occurred in connection with already existing tuberculosis, or the evidence of the latter immediately follows, the danger from an increase of the tuberculous affection is vastly greater than that incident to the liability to renewal of the hæmorrhage. Under this view it would be highly injudicious to employ any measures of treatment with reference to such a liability, if the measures conflicted with those indicated by the tuberculous affection. Moreover, it is to be borne in mind in this connection that the chances of recovery, arrest, and notable tolerance, in cases of tuberculous disease, as our clinical studies have shown, *are greater when hæmoptysis occurs than when this event is wanting.*"

I have not hesitated to give Dr. Flint's views in such detail, because they fully represent the widest and ripest experience of our American confrères ; neither shall I hesitate to give, in equally full detail, the views of our Continental brethren as represented in the treatise by Dr. Hertz, written for the fifth volume of Ziemssen's New Dictionary, under the head of " Treatment of Hæmorrhages from the Lungs ":—

" Blood-letting.—By a single operation or repeated ones, this favourite means among the old-time physicians in the treatment of bronchial hæmorrhage has been, in recent times, more and more laid aside, because we possess means which are better, and less injurious to the constitution. Where formerly it was the custom to bleed from the foot, apply leeches to the inner surfaces of the thighs and to the ankles, in the case of pulmonary hæmorrhage following an absence of the menses, we now use other remedies if they are indicated, such as sinapisms to the thighs and loins, and stimulating lye or mustard foot baths.

" Blood-letting should be reserved for certain cases only ; for instance in those in which otherwise healthy and robust persons are attacked with intense pulmonary hyperæmia, tumultuous action of the heart, great oppression, and sense of anxiety, followed by pulmonary hæmorrhage, or, when in a case of serious passive hyperæmia from heart disease, the stasis of blood in the lungs is proportionately very great when compared with the propulsive power of the heart. Oppolzer, moreover, advises a small venesection, from six to eight ounces, as an excellent means of combating hæmorrhage in those cases in which hæmoptysis has continued days and weeks in spite of all other remedies.

" Whenever the hæmorrhage is due to an altered condition of the

blood, and consequent perverted nutrition of the walls of the vessels, the prophylaxis requires that special attention be paid to the patient's diet and mode of life. For in these, as in weak and slightly-built patients, in those affected with tuberculosis and chronic inflammatory lesions of the lungs, such as precede phthisis, even a very slight exposure is sufficient to cause hyperæmia of the lungs, followed by hæmoptysis.

"These patients should be very careful to avoid every bodily exertion that is in the least degree fatiguing (dancing, riding, running), out-of-door walking when the weather is cold, or the winds blow from the north or east, or during great heat; further, every mental excitement, hot and highly spiced foods and warm stimulating drinks, such as coffee, strong wines and liquors. On the other hand, moderate out-of-door exercise during fine weather may be encouraged, and a residence in the country during the summer, and a warm climate for the winter and inclement seasons recommended.

"Whenever there is a well-marked *condition of anæmia*, mild preparations of iron are indicated, such as the lactate, or phosphate, or the reduced iron; also the internal and external use of *chalybeate waters*, bearing in mind the stimulating effects of the carbonic acid, which, in the case of those waters very rich in this gas, may be modified by the application of heat or by the addition of milk or whey. The food should be nourishing and easily digestible, and the taking of milk should be particularly urged; special attention should also be paid to the regularity of the bowels.

"During *an attack of hæmoptysis* complete *repose of the body* should be allowed the patient, best attained by the half-sitting position in bed, and all tight or uncomfortable articles of clothing should be removed. The patient should be strictly forbidden to speak, and all mental excitement should be as far as possible avoided. The room should be cool and airy, and the patient covered with a single covering; only cold food and drinks should be taken. To allay the *desire to cough*, *narcotics* are indicated, especially morphine or opium, and to regulate an excited action of the heart digitalis in infusion or tincture.

"The most effective remedy for the *hæmoptysis* itself is *cold*, applied externally, as an ice bladder or cold wet compress to the chest, and internally in the shape of bits of ice. Oppolzer recommends the gradual application of cold as more suitable, especially in the cases of such patients as are sensitive and dread the cold.

"Among the customary remedies is the solution of chloride of iron (strong solution of perchloride of iron, B.P.) taken internally, or better, by inhalation, diluted from 25 to 100 times. Another remedy is ergot in infusion (from 9 to 18 gramms of ergot to 4½ oz. of water, one tablespoonful every one or two hours). It is less judicious to prescribe large doses in powder, because it irritates the throat and easily provokes

cough. In severe cases the aqueous extract of ergot (Bonjean) may be given subcutaneously (aqueous extract of ergot, ℥ij., alcohol and glycerine, two drachms each : of this inject from one-half to one syringeful). Worthy of mention, however, are tannin and gallic acid, nine grains, every one, two or four hours; in less severe cases an alum whey by the cupful. Rogers recommends the tannate of alumina, and, as a more powerful remedy, ferric alum. Furthermore, acetate of lead, best combined with a grain of opium, every hour or half-hour. The *balsams*, especially oil of turpentine, five, ten or twenty drops in gelatine capsules ; the copaiba, best in the *potio Choparti*, as modified by Wolff (copaiba, syrup of tolu, peppermint water, alcohol, of each an ounce ; spirit of nitrous ether thirty minims, in teaspoonful doses). The *mineral acids* are frequently given, especially in mild cases, such as the dilute sulphuric or phosphoric acids, or Haller's acid elixir (pure sulphuric acid one part : add by drops while stirring to three parts of alcohol. German Ph.) from 10 to 15 drops in a mucilaginous vehicle every two hours ; occasionally combined with tincture of digitalis if the action of the heart is very excited; and if there is much cough, with bitter almond water, or with morphine.

"A popular remedy and indeed one of some efficacy, and always at hand, *is common salt*, which, in case the physician and the more appropriate medicines cannot be had at once, should be given in doses of from a half to one tablespoonful, either dry or mixed with a little water. The effect, according to Skoda, is caused by the salt acting as an irritant upon the gastric mucous membrane, producing through the sensory nerves of the stomach a reflex action upon the small pulmonary arteries whereby the latter are contracted.

"The administration of *emetics* in hæmorrhages, especially ipecacuanha in nauseating doses, so highly recommended by Graves and Trousseau, has lately received new advocates (Peter, Massina, Weber). Peter refers the effect to the diminution and size of the pulse, and Weber suggests that besides this, the expulsion of the blood from the bronchi, where it has accumulated and become a source of inflammation, is thus facilitated. There is really nothing to be feared from the blood thus accumulated; and the effect of the ipecacuanha upon the heart and vascular system is as yet uncertain, and but little understood. On the other hand, the danger that new hæmorrhages may be produced by the jarring of the body during the act of vomiting, is so apparent that this mode of treatment should meet with disapprobation in spite of the high standing of its advocates.

"If the bronchial hæmorrhage merely consists in the daily expectoration, after coughing or hawking by the patient, of two or three bloody sputa, or in the raising of sputa streaked with blood every morning; a simple dietetic treatment is all sufficient, the avoidance of all bodily exertion, of hot and exciting food, etc., and no special medica-

tion is required. But out of regard for the patient's anxiety, it will frequently be necessary to do something, and then the mineral acids may be prescribed.

"Notwithstanding Laennec's advice, that in *hæmorrhagic infarction* the bleeding should be combated during the first or second day by 'copious' blood-letting—from twenty to twenty-four ounces—this remedy is no longer employed so freely. Venesection in infarction is not indicated to subdue hæmorrhage, but is of use only where there is an intense degree of passive congestion in the pulmonary circulation, threatening œdema of the lungs.

"In many cases where there is increased frequency of the heart's action, with incomplete contraction of that organ, acids, and more especially *digitalis*, are indicated. With a little precaution, and more especially by reserving this latter remedy for the urgent cases, the danger feared by Gerhardt and Renzolds, that it favours thrombosis of the right side of the heart, and gives rise to new emboli, may be avoided.

"In many cases embolism of a large vascular branch is quickly followed by collapse, when stimulants should be administered, such as wine, camphor, musk, and the preparations of ammonia internally and externally, hand and foot baths, embrocations with spirits of mustard (oil of mustard 1, alcohol 50. Germ. Ph.), mustard poultices to the breast and the inner aspect of the thighs.

"If the hæmorrhage is considerable in quantity and requires special treatment, the indications are the same as those mentioned for bronchial hæmorrhage.

"It is impracticable in the case of *metastatic abscesses* to make use of any special treatment, therefore the latter should coincide with that of the primary disease. *For pulmonary apoplexy*, if the physician arrive in time, cold should be made use of externally and internally, the subcutaneous injection of ergotin, and internally, perhaps, the solution of chloride of iron. If collapse threatens, analeptic remedies should be given. Furthermore, all those indications are applicable which have already been enumerated in the treatment of bronchial hæmorrhage." (Hertz, Ziemssen's Dictionary, pp. 316-321.)

The Drs. Williams include the whole treatment of hæmoptysis in the following passages ("On Pulmonary Consumption," 1871):—

"Hæmoptysis, when so slight as not to amount to a teaspoonful, hardly requires the use of styptics, but may be treated by rest, by avoiding excitement and alcoholic stimulants, and by mild counter irritation. Where, however, the quantity expectorated exceeds that amount, or continues to recur, it should be promptly checked; and there is the more reason for doing so if a cavity is known to have formed, or to be forming, in either lung; as such hæmorrhage is likely to be more profuse, and if not checked may end fatally. A common, and generally a

very effective, styptic, is gallic acid, given in powders, either alone or combined with acid tartrate of potass (℞ Acidi gallici pulv., sacchari āā grs. x.; potassæ tartratis acidæ, ℈j.; pulv. cinnamomi, gr. j.), and continued every three or four hours while the bleeding lasts. . . .

"Tannic acid is a stronger remedy, which we have found more useful in other kinds of hæmorrhage, as epistaxis, hæmatemesis and hæmorrhœa; but in hæmoptysis it is useful to combine it with gallic acid if the latter prove insufficient to check the blood-flow.

"A more powerful styptic is the acetate of lead, but the dose and mode of administration require some care and attention. In order to produce a decided effect on the bleeding, it should be given, not in two or three grain doses, as many practitioners are in the habit of doing, but in doses of five grains at a time, in the form of a mixture, with a little excess of acetic acid, every three or four hours, and where the hæmorrhage is very profuse it may be given every two hours, or indeed every hour. To prevent the constipation, colic and cachexia, consequent on the accumulation in the system of so large a quantity of lead, a draught of sulphate of magnesia and sulphuric acid should be administered every morning. With these precautions large doses of lead have been given for several days with the effect of checking very profuse hæmoptysis and without any bad results.

"Oil of turpentine in doses of ten minims and upwards is by no means a pleasant remedy, but may be resorted to occasionally, and the taste be covered by an aromatic infusion of peppermint or cloves. The tincture of perchloride of iron is recommended in cases of extreme weakness.

"The above styptics, as also alum and sulphuric acid, check the hæmorrhage by causing coagulation of the blood. We will now notice some which probably owe their astringent power to their contracting the blood-vessels. Digitalis, in doses of a half-a-drachm and upwards, exercises a marked effect; but its use is more adapted to hæmoptysis from cardiac disease, than to this form of hæmorrhage. In cases of consumption, however, where the heart's action is violent, or where the complication of cardiac disease exists, digitalis succeeds best, and in smaller doses it may be well combined with other styptics, as with gallic and tannic acids. Our experience of ergot of rye has been most satisfactory. We have often tried it when other styptics have utterly failed, and with prompt and decided effects. The dose should be at least a drachm of the fluid extract, and a few repetitions of it will soon test its effects, after which, if success has followed, the quantity had better be reduced, and soon discontinued.

"Another way of treating profuse hæmoptysis is by dry-cupping the chest, generally in the inter-scapular and scapular regions; and this measure has the advantage of immediate and decisive action, though the effects are not always lasting. A slower process of derivation, but

one that answers well when the hæmorrhage is somewhat reduced by strong styptics, is the application of a blister, and we have, by this means, spared the patient several doses of astringent medicine, which for the stomach's sake, must be considered highly desirable.

"But we must not forget to enforce the common sense measures for arresting hæmorrhage, which will sometimes prove efficacious without the administration of medicines, and always greatly assist their action. The patient should be kept in bed, with his body and mind free from all excitement; his room should be cool, as also his beverages, which may be iced, and, except in cases of great exhaustion, must be quite free from all alcoholic stimulant. His diet should be restricted to nutrient liquids, such as cold beef tea, chicken broth, milk, etc., and he can suck ice freely, but we cannot sanction the application of ice to the chest—a practice which has been known to induce pneumonia and consolidation of the lung of a phthino-plastic kind.

"The bowels should be kept freely open, and tonic medicines, for the time, discontinued."

A writer in the "New York Medical Record," June 1, 1874, says: —"Hæmoptysis, a symptom which alarms the patient, is not often the immediate cause of a fatal termination (in consumption); on the contrary, patients in whom this hæmorrhage occurs are more likely to have their phthisis arrested. Those cases that do recover almost always have this hæmorrhage before recovery. It is in the early stages that hæmoptysis is beneficial. In the latter stages it may have an opposite effect. If a patient have slight hæmorrhage, put him in bed and keep him quiet, and if he bears opium well give him a moderate dose, which will diminish the moral effect of the hæmorrhage, and also diminish irritability; then a small teaspoonful of salt, followed by a little water, may do good. Where the hæmorrhage is only slight and occasional, *do not* give inhalations of nitrate of silver or persulphate of iron.

"Hypodermic injections of ergotine may be given in doses of about one grain as a hæmostatic, or the liquid extract may be administered by the mouth in doses of ♏ xx. to ʒ j.

"Quinia may be given in 5 grain doses every hour, until the pulse and temperature are brought down. Turpentine is a most powerful hæmostatic in all internal hæmorrhages; it may be given in ♏ x. doses every two hours, or it may be easily and beneficially administered by inhalation, and is then especially useful in those cases where small hæmorrhages last for some days. Strangury as a result is of rare occurrence.

"If the hæmorrhage is profuse, the withdrawal of blood from the general circulation is indicated. This may be done by dry cups applied to the chest anteriorly and posteriorly and to the thighs. Another and more effectual plan is to apply a ligature to one of the extremities,*

* See a caution as to this treatment, p. 112.

and thus arrest the venous circulation, allowing the blood still to pass through the arteries ; the ligature must not be applied to one limb for more than six or eight minutes, and then must be gradually loosened so that the blood is only slowly re-admitted to the general circulation. A second ligature may be applied to another limb two or three minutes before the one previously applied is loosened." ("Dr. Dobell's Reports," 1875.)

There is no subject in medicine upon which so much trash is written as upon the effects of baths and waters; it would almost seem as though they exercise a diluent influence on the brain-power of those who live amongst them. The following account of the Mont Dore treatment of hæmoptysis is, however, worthy of note; although one cannot help remarking upon the curious fact that the experience of M. Lassalas, the new comer at the springs, should be diametrically opposite to that of his long-resident predecessor, M. Bertrand, on a vital point.

M. Richelot ("Union Médicale," 1877) considers Mont Dore treatment a sedative to the nervous system and to the circulation. "It also obviates pulmonary congestion. . . . By obviating the tendency to pulmonary congestion and to subsequent hæmoptysis this treatment causes feverishness to subside. In addition to these effects it promotes the more or less complete resolution of pre-existing pneumonic patches and inflammatory thickening. . . . The inflammatory form of consumption, marked pyrexia, nervous *erethism and hæmoptysis*, should be considered so many indications for the Mont Dore medication. . . . In treating strumous or anæmic subjects, and in cases of acute tubercule, or of galloping consumption, this medication should be precluded. Its application, therefore, requires close watching."

"At the Congress of Clermont, M. Lassalas ('Revue Scientifique,' No. 9, 1876) recommended the inhalation of the Mont Dore water for hæmoptysis. Under the influence of this treatment, the pulse, without losing its strength, becomes less frequent; the respiratory movements increase in extent, and the blood-spitting is directly arrested. This result, (he says,) should not be attributed to the effect of altitude, which is 1,050 metres. In fact, it is not at all uncommon at Mont Dore to see hæmoptysis occurring in patients who never before had been subject to this accident. However, a course of inhalations suffices to suppress it. M. Lassalas is by no means prepared to say that every case of hæmoptysis must necessarily yield to this treatment. Nevertheless, *although his predecessor at Mont Dore, M. Bertrand, held blood-spitting to be a decided contra-indication to the use of the mineral water, M. Lassalas, out of* 120 *cases of hæmoptysis thus treated, did not meet with one failure.*" ("Dr. Dobell's Reports," 1877.)

It may be thought that the preceding details of the various means of treatment in hæmoptysis should have fairly exhausted the subject.

But I cannot regard the matter in that light, and I think I ought to attempt to give it some further elucidation, if only by putting the materials into form. We have, it is true, passed in review a number of conditions under which more or less profuse hæmoptysis may occur, various phenomena which may come after it, and many remedies which may be used in its treatment; but I hardly think that enough classification has been given to these remedies with reference to their special functions, and to the occasions on which one or other is the most appropriate.

It is of the first importance to keep clearly before our minds the main objects to which our treatment is to be directed, the special aptitude of our different remedies for attaining those objects, and the most unequivocal forms and modes of using the remedies selected.

I would particularly advise practitioners *not to unnecessarily amplify their lists of remedies having the same properties;* but to select those known to be most potent, and to use them in larger or smaller doses, or in greater or less dilution, according to the severity of the case. Thus, it is pure waste of time to try first one and then another of a long list of infusions and tinctures having gallic or tannic acid for their active principle. It is far better to use the acids themselves, or those tinctures or infusions which contain them in the most potent form; and *if these fail after a thorough trial,* to turn to *some other active principle* without further loss of time.

It is an old axiom that "he is a bad workman who complains of his tools." And certainly in the practice of medicine this warning is most necessary. It is astonishing to see how badly some of our best tools are often used, and therefore how signally they fail; and, on the other hand, how skilfully some of our worst tools are used, so that success is attained by the most unpromising means. We have plenty of good weapons if we know how to use them.

The main objects to which our treatment of hæmoptysis is to be directed are:—

1. To clot the blood about the bleeding part.
2. To relieve the vascular tension in the bleeding part.
3. To contract the vessels in the bleeding part.
4. To stay the rush of blood to the bleeding part.
5. To maintain the above conditions for a sufficient length of time to prevent a recurrence of the bleeding.
6. To remove or avoid the causes which excited the hæmorrhage.
7. To remove or avoid the causes which predispose to the hæmorrhage.
8. To prevent inflammation and disorganisation of the parts implicated in the bleeding.

To "clot the blood about the bleeding part" requires that we should "contract the vessels," "stay the rush of blood into it," and "remove or avoid the causes which excited the hæmorrhage."

We have, then, to consider what are our most direct means of clotting the blood. They are simple and powerful, viz., cold, rest, and styptics or astringents; and all of these may be used both topically and generally. First let me speak of cold,—so potent for harm as well as for good. The topical and general effects of cold are all favourable to the arrest of hæmorrhage; by constricting capillaries, stagnating circulation, and clotting the blood; cold therefore is a valuable adjunct to all other means of stopping hæmorrhage.

But I must make a most emphatic protest against the way in which cold is ordered to be used in hæmoptysis by many authors of authority, and in which, I regret to say, I see it used almost daily in practice. I refer to the continuous application of bags of ice to the surface of the chest, by which every dictate of reason and common sense is violated. When we hear of cases in which destructive lung-disease had followed hæmoptysis, and of grand pathological theories invented to account for it; when we learn that such cases had been treated by bags of ice continuously applied for hours to the chest-walls of patients who had been previously guarded from every breath of cold air, and that windows had been kept open to secure a draught across the chest, from which all covering but the ice bags had been removed; when we find that these influences are absolutely ignored as causes of the destructive lung-disease which naturally follows in their track; when we turn to the writings of the same and of other authors, and find "exposure to cold" set down among the most common and potent causes of bronchitis, pleurisy, and pneumonia in the previously healthy—in those, therefore, who have not previously received any special protection from cold; we can only wonder at the blindness, folly and prejudice which sometimes stultify the best intelligences. In the name of common sense, then, never attempt to freeze the human chest.

The air round the patient should be cool without draught; the upper parts of the body should be kept cool without chill. Ice may be sucked in moderate quantities, so as to keep a cold stream down the œsophagus, and the lotions used as spray may be made with iced water, so as to lower the temperature of the contents of the air-tubes. A lump of ice may be now and then grasped for a few seconds in the palm of each hand alternately, by which a thrill of cold is sent through the body; but care should be taken that it is not held longer than a few seconds:—and with these measures the use of cold should end in the treatment of hæmoptysis. In fact, it is with this as with all other treatment—we must not relieve the patient for the moment at the price of killing him afterwards by the effects of our remedies used in the emergency. The wise physician is always considerate and judicial, at the same time that he is prompt and energetic,—looking to the future as well as to the present. The watchword of his life should be *σωφροσύνη*, in its widest interpretation.

The next most important agent in our treatment is rest;—rest to the whole body, so as to calm and slow the general circulation ; rest for the bleeding part, to avoid movement of its structures, and that hyperæmia inseparable from functional activity. Our conception of rest must be taken in a wide sense. It must not be confined to absence of locomotion, favourable position, and silence; but must include, as an important item, restriction of the flow of new nutritive materials through the pulmonary circulation. If we allow copious supplies of food to be thrown into the system, we necessitate activity in the oxygenating processes in the lungs, and hence destroy functional rest in the inmost recesses of the wounded organ. We must, therefore, put first among our conditions of rest for hæmoptysis a restricted diet. In selecting position we must remember the effects of gravity, and thus the chest and shoulders should be raised to prevent static congestion. We must remember, also, how greatly syncope promotes coagulation of blood, and therefore, although we do not wish for actual syncope because of other reasons, the dread that the patient may become faint must not be allowed to go too far, a certain amount of faintness being one of our most potent auxiliaries in arresting hæmorrhage. And after hæmorrhage has ceased, rest and a favourable position are among the most necessary precautions for preventing its return. Still more, this rest should be maintained long enough to be secure against the inflammatory and disintegrating effects—not, as Niemeyer would have it, of the unexpectorated blood in the alveoli of the lungs—but, of the torn and broken up lung-tissue, mingled with blood and air, to which I have already fully referred. (See 8 in the list, p. 121.) A few days of rest and careful watching of the temperature are, therefore, necessary after the hæmorrhage has ceased. So long as the temperature is above the normal, this rest should be maintained, and any symptoms of inflammation should be promptly met by appropriate treatment. (See pp. 99—101.)

I entirely agree, therefore, with Dr. Baümler in the practical conclusion at which he arrives, in his paper to the Royal Medical and Chirurgical Society, although I differ from him as to the reasons by which he arrives at that conclusion. He says, "Patients who have brought up blood from the air passages or lungs ought to be very carefully watched, especially by means of the thermometer, for some days afterwards; even if the hæmorrhage has quite ceased, and however slight the symptoms following the hæmoptysis may be, the patient ought to be kept quiet till all febrile symptoms have disappeared."—("Trans. of the Royal Med.-Chir. Soc.," vol. ii., 1869.)

In the light of the foregoing facts I cannot help expressing my surprise that so experienced a physician and pathologist as my friend Dr. Wilks, should write as follows:—

"When we are treating a case of hæmoptysis, we insist on the

patient being absolutely quiet, to lie in bed, to make not the slightest movement of the arms, such as putting on a coat, which we suggest might seal his fate. Believing that such practice has been determined entirely on *à priori* considerations, I have for some time ceased to adopt it." ("Med. Times and Gazette," May 23, 1868.)

But, while I entirely differ from Dr. Wilks on this point of treatment during the active stage of the case, I must protest against the absurd and injurious prolongation of the period of enforced rest after hæmoptysis, which seems to be thought necessary by many practitioners. I hear with astonishment, almost daily, of patients being kept on low diet and rest *for many weeks* after an attack of hæmoptysis, although they have been long free from any symptoms of inflammation, or of recurrent hæmorrhage, and are obviously suffering severely in constitutional health from this ill-advised and debilitating treatment.

It is chiefly by rest *immediately* after an attack of hæmoptysis that we shall fulfil the object of treatment which I have placed eighth in the list (p. 121), viz., "to prevent inflammation and disorganisation of the parts implicated in the bleeding." But if, in spite of such precautions, inflammation and disorganisation set in, they must be met by counter-irritation (blisters), and antiseptic inhalations.

On the subject of Antiseptic Treatment, Dr. Annelli remarks ("Il Morgagni, Napol," 1877), "that the development of phthisis after hæmoptysis is neither due to the irritant action of the blood entering the bronchi, nor to the action of the air upon the effused blood, but to the entrance of infected matter into the pulmonary tissue, which has become looser in its continuity by the hæmorrhagic matter. . . He therefore concludes that in hæmoptysis not only hæmostatic, but also antiseptic treatment should be adopted." ("Dr. Dobell's Reports," 1877.)

In arresting the hæmorrhage, we must add to "cold" and "rest" the use of topical and general astringents or styptics. "Styptic is a term sometimes used synonymously with astringent, but generally applied to a substance employed to arrest hæmorrhage. A *mechanical* styptic is one that arrests the flow of blood by being applied immediately to the mouth of the bleeding vessel, or by inducing a coagulation mechanically in it. A *chemical* styptic is one which coagulates chemically the blood around the bleeding orifice." (Dunglison).

Dr. Headland says, "As neurotics act directly on nerve, so these (astringents) act directly and especially on muscular fibre. They cause this to contract, whether it be striped and voluntary, or of the involuntary or unstriped kind.

"Their action is more readily understood, because it can actually be seen. It takes place out of the body, or in the body—externally or internally. Nearly all astringents have the power of coagulating or precipitating albumen. By virtue of this power they are enabled to constrict many dead animal matters. They affect fibrinous tissues in a

similar chemical way. But they seem to possess a further dynamical influence over living tissue, which possibly depends in some way on this chemical property. This dynamical influence is, as I have said, to cause the contraction of muscular fibre. *By this, all their operations can be explained.* Taken into the blood in a state of solution, they pass through the walls of the capillaries to the muscular tissue. By inducing the fibre of the voluntary muscle to contract, astringents may brace the system, and simulate the action of tonics. But as the contraction of voluntary muscles is short and brief, it requires for its maintenance a continual excitation; and unless the medicine is thus continually repeated, the tonic effect subsides. *But astringents* further *contract involuntary* muscle. This contraction is slower, and more durable and important in its results. Unstriped muscular fibres exist in the middle coat of arteries, in the walls of capillary vessels, in the lining of the ducts of glands generally, and in the substance of the heart, and in the coats of the stomach and intestines. Astringents are irritants and poisonous in large doses. But in small doses they constrict and stimulate to a healthy condition those tubes that contain in their coats the unstriped fibre. By diminishing the calibre of the capillary vessels generally, they promote health and counteract a lax state of the system. *By the same action on the extreme vessels they prevent hæmorrhages.* By constricting the ducts of glands, they diminish the secretion of those glands, because denying it an exit. By acting on the stomach and intestines, they are able to give them tone, to diminish their secretions when excessive, and thus to promote digestion. . . .

"Such appears to be the simplest and most rational explanation of the action of astringent medicines.

"As this action appears to be quasi-chemical, we can understand how it is that dilution with the mass of the blood impairs their activity. They act best when they can immediately touch the point to be acted on ; next best, when they are excreted by a gland which is to be influenced, for then the dose again undergoes concentration; least, when diluted by the whole mass of blood." ("On the Action of Medicines in the System," by F. W. Headland, M.D., etc., 4th ed., 1867.)

The following are the astringents upon which we must rely in the treatment of hæmoptysis :—

1. For topical use, as spray, douche, lotion, or fine powder : alum, sulphate and perchloride of iron, eucalyptus rostrata, tannic acid.

2. For internal administration and absorption into the blood : gallic acid, acetate of lead, sulphuric acid, alum.

If the bleeding surface is in any visible portion of the naso-pulmonary tract, we may effectively use our astringents as lotion, douche,

or impalpable powder. When it is deeper seated, the form of spray is the only one which can reach it, and is most efficient. I have seen profuse pulmonary hæmorrhages that have resisted many other potent means of treatment stopped within an hour by the use of ice-water spray, containing ℈j. of alum to the ounce. Dr. Poggiale ("Rev. des Sciences Méd.," 1874,) demonstrated clearly on animals, and in the case of a woman with a tracheal fistula, that the spray of inhaled liquids reaches the bronchial tubes,—a fact hitherto denied by Pidoux and Durant-Fardel.

But whatever doubt there may be as to the extent to which atomised fluids reach the finest air-passages when there is no hæmorrhage, it is quite certain that in profuse hæmoptysis, when the air-passages are wet with blood, the atomised fluids mix with this blood ; and as it has been proved that such blood is drawn into the finest tubes and into the alveoli during inspiration, so it is clear that, mixed with this aspired blood, the atomised fluids reach the same structures. Therefore, the use of astringents in this form, for the arrest of profuse hæmoptysis, is a simple dictate of common sense; and in my practice alum has proved the most suitable and efficacious. But should this fail, iron, tannic acid, or red gum may be tried. The red gum is a remarkably pure and pleasant form of tannin, the taste of which is seldom objected to, and it is particularly unirritating; the only disadvantage is its colour, which, when mixed with saliva and mucus, is apt to be mistaken for blood. I remember a medical friend who was using atomised red gum with excellent effect for an attack of moderate hæmoptysis, being desperately frightened one day because he thought the bleeding "would not stop," till I convinced him, by examining the sputa with the microscope, that the colour was due to his lotion and not to blood. There had, in fact, been no blood for several days, but the expectoration was quite red with eucalyptus rostrata.

For internal use I am sure that gallic acid is by far the best. That it reaches the contents of the air passages is proved by the discoloration of the expectorated blood which sometimes occurs, and which steadily increases with the continued use of the remedy if given largely; and that it thus effects its object is equally clear, from the rapidity with which the hæmorrhage ceases after this change in the colour of the expectorated blood occurs.

As there is no evidence that tannic acid exerts any influence through the blood except when converted into gallic acid, never being found in the secretions in any other form than that of gallic or pyrogallic acid (Parkes), I always avoid it and select gallic acid in its place; except where it is wanted for topical use. "When the solution (of tannic acid) is heated in the air or taken into the human system, the elements of grape sugar are oxidised into carbonic acid and water, and gallic acid is set free. It is thus gallic acid which passes out into the secre-

tions after exerting an astringent action on distant parts of the system." (Headland.)

Our next most useful ally is sulphuric acid. Owing to its high diffusive power it passes readily into the blood (Ringer), and long experience has convinced all practical men of its utility, not only as an astringent but as a restorative ; it is wise, therefore, at least to combine it with our other astringent remedies.

It is necessary to remember that all these remedies disagree with milk, and if used at the same time as a milk diet, they may produce great gastric discomfort by the extreme hardness of the curds formed in the stomach. While these astringents are being used, therefore, *whey* should be ordered instead of milk. Or if milk is given, it should be first turned into curds and whey, and the curds beaten into fine particles, and some peptodyn added before they are swallowed. I have found this plan answer perfectly, whereas I have seen most distressing stomach complication produced by the neglect of such precautions. (See p. 154 of the sixth edition of my work "On Diet and Regimen" for different forms of curds and whey.)

In acetate of lead we often find a potent hæmostatic when other remedies fail. "It is not an active poison, although commonly supposed to be so. . . As to the dose of sugar of lead capable of producing acute poisoning, it is difficult to speak with any degree of certainty, as a drachm of acetate of lead has been taken daily for ten days before any symptoms of poisoning manifested themselves : one ounce has been taken without any result whatever, whilst in other cases two drachms have produced alarming symptoms. . . The lead line has been produced after 18, 21 and 42 grains of this salt. . . It is due to the formation, in the minute capillaries of the gums, of a sulphide of lead, probably by the action of the sulphuretted hydrogen derived from the fluids of the mouth on the lead circulating in the capillaries." (Woodman and Tidy's "Handybook of Forensic Medicine.")

This assurance, on such high authority, with regard to the large doses required to produce acute lead-poisoning may give practitioners confidence in using the remedy in what I consider to be the proper way, viz., in full doses rapidly repeated for a short time, until its specific effect on the hæmorrhage is obtained, after which it should be cleared out of the system, by sulphuric acid and sulphate of magnesia, as quickly as possible. The thing to guard against is *chronic* lead poisoning with its depressing effect upon the general health; and this is much more likely to result from the protracted use of small doses of a grain or two, three or four times a day, combined with opium, by which constipation is produced and the lead accumulated in the system, than by doses of five grains, protected by an excess of acetic acid, repeated every one, two, three or four hours, according to the urgency of the case; taking care that the accumulated dose in twenty-four hours does not exceed

60 grains for an adult; stopping the drug as soon as possible; and eliminating it, as directed above, while the hæmostatic effect is maintained by other remedies.

It must be remembered that alum is chemically incompatible with acetate of lead, and must therefore be withheld while the lead salt is being administered; but when the lead is stopped alum comes in well as an adjunct to the eliminative and astringent mixture of sulphate of magnesia and sulphuric acid.

This is perhaps the most appropriate place for observing that the object of treatment which I have placed second on the list (p. 121), viz., "to relieve vascular tension in the bleeding part," is best attained by sulphate of magnesia purging, which can be so conveniently combined with other treatment.

For my own part, I much prefer turpentine to lead, over which it has many important advantages to set against its one disadvantage, viz., its nasty taste. I have often seen it succeed when lead has failed, and I never prescribe lead till I have tried turpentine, unless the patient refuses to take the latter, or unless—and this is an important proviso—there is great vascular excitement; in which case lead is eminently suitable, and turpentine quite the contrary.

Turpentine is a thoroughly wholesome medicine in every sense. It is restorative, antiseptic, anti-inflammatory, anti-catarrhal; the system may be rapidly impregnated with it—as seen by its appearance in the breath and in the urine—either by administering it by the mouth, or by using it for injection, inhalation, or liniment. It has also the advantage of acting as a counter-irritant, when applied to the chest-walls, at the same time that its fumes are being inhaled. It is very nasty, there is no doubt; but then people who have profuse bleeding from the lungs will gladly swallow almost anything to stop it; and there are several ways of disguising the worst of the taste. Thirty minims every half hour for the first hour or two, and then every two or three hours as the bleeding diminishes, is the best dose and mode of administering it, supplementing the internal dose by its external use, and its consequent inhalation. Larger doses than this are apt to produce sickness. The turpentine may be rubbed up into an emulsion, strongly flavoured with liquid extract of liquorice; or, when used extemporaneously, may be stirred in gin, peppermint water, or peppermint cordial, or—and this I consider by far the best—it may be taken in orange or lemon juice, a piece of orange or lemon peel being masticated directly after it.

The *modus operandi* of turpentine in stopping hæmorrhage has never been satisfactorily explained. Headland classes it (with a mark of interrogation) among vegetable astringents; and adds, "These volatile oils (turpentine, cubebs, copaiba) are probably incapable of exerting a true astringent action. But it should be observed that turpentine when in large proportion coagulates albumen, and on the supposition that it

is really an astringent, turpentine has been often prescribed as a remedy for hæmorrhages in different parts of the body."

Dr. Ringer does not attempt any explanation of the mode in which it acts, but he says:—"Turpentine passes readily into the blood, and may be detected in the *breath* and sweat, and in an altered state in the urine, giving to this excretion an odour of violets or of mignonette.... It is very efficacious in bleeding from the various organs of the body, as the *lungs*, nose, uterus, kidneys, and bladder. A drachm should be given every three hours, a dose which sometimes causes sickness, diarrhœa, and even blood in the urine; but on discontinuing the drug the blood soon disappears. Given to check bleeding from the kidneys, or in Bright's disease, it must be administered in very small quantities. It is also reputed to possess the power of checking bleeding in the *hæmorrhagic diathesis*, and to be useful in purpura." ("Handbook of Therapeutics," 6th edition.)

Through an accident, I once had a good opportunity of observing the wonderfully energetic action of which turpentine is capable in stopping internal hæmorrhage when given in large doses. A tall, fat licensed victualler was suffering from purpura; his skin was covered with purpuric blotches, and he was bleeding freely from the lungs, kidneys, and bowels. I prescribed a teaspoonful of sp. of turpentine in a glass of gin every three hours; but by some blunder of the nurse, probably explained by her being in a public-house, a *tablespoonful* of turpentine was given for each dose instead of a teaspoonful; and when I saw the patient the following day he had taken four doses with the happiest effect. The bowels had been violently purged of a quantity of clotted blood; his urine, breath, and perspiration were redolent of violets and mignonette; bleeding had ceased at every point; and from that day forth he got rapidly well without any other remedy.

Iron.—It will be observed that although I have included iron in the list of astringents for topical application, I have omitted it from the list of those for internal administration. This I have done advisedly, because I prefer to trust to less equivocal friends than the preparations of iron. We all know that "In addition to their astringent action, they tend to restore the deficient red colouring matter of the blood. . .

"It is probable that the increase in the hæmatosin is the first change; that this improves the condition of the blood corpuscles, increases their number, and through them betters the condition of the blood, and of the system generally." (Headland.)

"Oxide of iron possesses an ozonising power. . . The iron of the blood corpuscles acts in the same way, converting oxygen into ozone, thus promoting oxidation." (Ringer.)

These are the grounds on which I prefer other hæmostatics to iron as an internal medicine in hæmoptysis. In so large a proportion of cases pulmonary hæmorrhage is connected with tuberculosis, that in

all our treatment this probability must be borne in mind, and the use of iron avoided as a general rule. If in any special case there is certainly no connection with tuberculosis, my objection to the use of iron is removed. The theory on which my objection rests was stated by me in 1867. My practical experience before that date had already prepared me for the theoretical conclusion. But I have had abundant opportunities since that time of watching the effects of iron prescribed by other physicians in tuberculous patients, with the result of substantiating in the most unequivocal manner my belief in its injurious effects, and, especially, in its tendency to predispose to hæmoptysis. In 1867 I wrote as follows: "Another question which is in my opinion of the greatest importance, and which according to my experience is generally decided in the manner exactly contrary to what is best, is that of the administration of iron. If, as we have the best reasons for believing, iron when taken into the system increases the number of red corpuscles in the blood, and if, as we have equally good reason to believe, these corpuscles are the means by which oxygen is distributed throughout the tissues, it is surely totally at variance with common sense to give iron, and thus increase the activity of the process of oxidation, so long as there is the slightest deficiency of carbon in the organism. Yet iron, being regarded as an excellent tonic, is commonly given to consumptive patients with an idea of strengthening them, regardless of its incongruity with the leading points of treatment. The fact is, as I have already pointed out, that so long as there is a deficiency of carbon in the blood, anæmia (in the sense of white blood) is a protection against abnormal oxidation of tissue. Let me caution you therefore, never to give iron to a consumptive person until you have effectually supplied all deficiencies of fat in the system, and never to give iron to anæmic persons, until you have ascertained that there is no defect in the supply of fats to the blood." And I went on to say in a foot note, "While writing the above my attention has been called to the remarks of M. Trousseau and of Dr. Millet on this subject. It is a striking coincidence that I have predicted as necessary to the nature of tuberculosis, a fact which *they had learnt from observation but could not explain.* . . .

"In a lecture delivered at the Hotel Dieu, by M. Trousseau, and reported in the 'Medical Circular,' January 25, 1860, after relating two very marked cases in which rapid consumption followed the apparent cure of chlorosis by preparations of iron, he goes on to say, 'This time I had received a lesson not to be overlooked. I had occasion also to see in the practice of my colleagues very serious pulmonary symptoms succeed to relapsed chlorosis treated by iron, so that it seemed to me extremely probable that in individuals predisposed to tubercular phthisis, iron administered and continued for a certain time, only favoured and hastened the development of the accidental productions.

This great probability has become for me a well-founded certainty, and I have become convinced of this, more especially for this reason: I have seen chlorotic patients spit blood after using ferruginous preparations, and become more chlorotic than ever; and I have remarked that the more the chlorosis becomes confirmed, the less does the tuberculisation show itself, so that, during more than twenty years, I have stood by the opinion that chlorosis *in some sort of way* excludes phthisis; or, rather, that it is a safety-valve against the ulterior explosion of tuberculisation. Not only do I not give iron in chlorosis when there is a marked disposition to pulmonary phthisis; but every time I am consulted on the subject of chlorotic disease, I inquire with the greatest care into the family history, and when I happen to find suspicious precedents (of consumption in the family) I energetically proscribe the use of martial preparations.'

"In the 'Bulletin Général de Thérapeutique Médicale et Chirurgicale' (1862), Vol. lxii., p. 507, Dr. Millet, Physician to the Colony at Mettray, speaks strongly of the danger of giving preparations of iron in early phthisis. He gives notes of sixty cases, and says he has seen a great many more, in which the treatment of anæmic girls with iron has been quickly followed by fatal galloping consumption. He arrives at the conclusions:—(1.) That iron should never be given without previous careful auscultation of the patient, and must always be withheld if there is the least suspicion of tubercle. (2.) That iron never does any good in consumption, but always accelerates it." ("On the True First Stage of Consumption.")

As already stated (p. 121), one of the main objects to which our treatment of hæmoptysis should be directed is "to remove or avoid the causes which predispose to the hæmorrhage." Let us then at least take care that we do not add to them instead. We are not obliged to employ iron in hæmoptysis, for we have plenty of surer arrows in our quiver. Unless, therefore, it is absolutely certain that there is no danger of tuberculosis we must not put iron into the blood.

We come now to the last weapons in our armoury, and happily they deserve to be called "arms of precision" as well as of power.

Digitalis, Ergot.—With these we are able "to stay the rush of blood to the bleeding part," and "to contract the vessels in the bleeding part." (See p. 121.) The "rest" and "cold," of which we have already spoken, are our reliable auxiliary forces in this important part of the battle. Sometimes they are sufficient of themselves to do the work. But where the danger is imminent, "sure must be made doubly sure," and digitalis and ergot are "doubly sure" remedies.

The late Dr. Brinton stated as the result of his experience, that in bleeding from the lungs treated with digitalis, as soon as the frequency of the pulse is diminished the bleeding ceases.

But, "though most authors who have written on the subject mention that the bleeding stops when the peculiar *slowing of the pulse* has been produced, there is much doubt whether the mere reduction of the frequency of pulsation is the curative agent; it is much more probable that the cessation of hæmorrhage is chiefly due to the contraction of the smaller arteries, and the consequent prevention or diminution of venous stagnation." ("Materia Medica and Therapeutics," by C. D. F. Phillips, M.D., 1874.)

Few medicines have excited more lively discussion or more careful investigation of their physiological and therapeutical action than digitalis; and, notwithstanding many conflicting statements, there is at the present time a fair consensus of opinion with regard to the leading points of practical importance.

Its effects on healthy persons, as shown by pulse tracings, have been found to be (1.) Diminution in the frequency of the heart's beats. (2.) Increase in the force of each beat. (3.) Increase in the arterial tension. (4.) A co-ordinating influence of the drug in restoring the regularity of the heart's movements.

When ordering so potent a drug it is necessary to bear in mind its poisonous effects, and thus to be on our guard when to continue it and when to withdraw it.

Drs. Woodman and Tidy in their recent "Handybook of Forensic Medicine," say, "Experiments on animals prove that however digitaline is administered, it is an active poison. Mr. Blake found that three drachms of the leaves arrested the action of the heart in five seconds. . . Digitalis is what is called a cumulative poison, that is, after a series of small doses have been taken, apparently without any effect, the poisonous action of the drug may break out suddenly and with great violence. . . The prominent symptom to be observed in digitalis poisoning is the depression of the heart's action, the pulse becoming irregular and almost imperceptible. . . Towards the end of the case, the faintings become constant, and death usually occurs from syncope induced by the patient making some slight exertion, such as sitting up in bed. . . . Homolle found his pulse reduced by small doses of digitaline taken at short intervals, to a $\frac{1}{4}$ (17) the number of beats that was normal. Doses of the $\frac{1}{30}$ to the $\frac{1}{15}$ of a grain lowered the circulation in 24 hours, and caused vomiting and purging. Further it was noticed that doses above the $\frac{1}{15}$ (equal to 8 grains of the powdered leaves) had a strong emetic and purgative action. . . . We may, from experiments, conclude that a dose of from $\frac{1}{4}$ to $\frac{1}{2}$ a grain of digitaline would prove fatal. Two or three grains of good powdered fox-glove will often produce serious symptoms, though patients have recovered after taking as much as a drachm. Altogether it must be acknowledged that digitalis is a very uncertain, and because uncertain, a very dangerous drug."

It is true, no doubt, that digitalis is somewhat uncertain, and in that sense dangerous; but it is natural that medical jurists should be somewhat unduly impressed with this side of its character; while, on the other hand, medical practitioners, who are prescribing it every day of their lives without any serious consequences, may be apt to forget its possible dangers. But, on the whole, those who have the largest experience of its use therapeutically have the greatest confidence in its power for good and the least fear of its going seriously wrong, when used with due caution as to the quality and form of the preparation and the intervals at which moderate doses are repeated.

When large or frequent doses of digitalis are being given, the following warning of Dr. Headland may be wisely borne in mind:— "Certain precautions are necessary in its administration. The patient should be generally confined to the recumbent position; for if, while under the influence of digitalis, he should suddenly rise, and the heart, already weakened, have further to contend with the force of gravity in the propulsion of the blood upwards, it may actually stop." Drs. Woodman and Tidy mention a case in which, on the sixth day after digitalis poisoning, a woman died from merely rising up suddenly in bed.

Dr. Phillips says, "Taking the digitaline of Homolle and Quevenne as a standard, the following may be said to be the true physiological action of digitalis, so far as authorities are agreed about them:—

"1. It is admitted on all hands that digitalis is a cardiac poison; given in large doses, it brings the heart to a standstill.

"(2.) In doses which just fall short of a fatal effect, digitalis produces faintness, diarrhœa, nausea, and vomiting, with irregularity of the heart's action.

"(3.) In still smaller doses the heart's pulsations are much reduced in frequency, and the arterial blood-pressure is remarkably raised.

"(4.) Doses large enough to slow the heart's action usually reduce the temperature. . . .

"It must not be supposed that the slow pulsations of the heart indicate a diminished rapidity of the blood-current; on the contrary, their increased vigour causes the blood to circulate with abnormal swiftness." (Op. cit.)

"Blake, Brunton, Foster and others find that digitalis sometimes considerably increases arterial tension. Digitalis injected into a vein causes the tension to reach its maximum in four to ten minutes, and this heightened blood-pressure Brunton attributes chiefly to contraction of the arterioles." (Ringer.)

Professor C. Peyrani in his experiments at his Parma Laboratory, found that the hypodermic injection of digitaline produced in the frog diminution of the heart's beat from 19 to 4 per minute; in the rabbit from 140 to 73.

"M. Lombard believes that the constant and gradual fall of arterial

tension produced by digitalis shows that, through its direct influence on the cardiac ganglia, digitalis acts on the heart primarily." ("Dr. Dobell's Reports," 1877.)

"In estimating the effect of digitalis on the heart we must be careful to discriminate its influence on that organ from its influence on the pulse. Under the influence of digitalis the pulse, no doubt, grows fuller and less compressible; but it must be borne in mind that digitalis slows the heart, and therefore, if the heart does only the same amount of work, each beat must be stronger. Again, digitalis contracts the arterioles, thus increasing arterial tension, and rendering the pulse larger and less compressible. . . .

"Dr. Brunton believes that digitalis 'acts on the regulating apparatus of the heart chiefly through the vagus, thus causing slowing of the heart, and stimulates the musculo-motory apparatus (ganglia and nerves of the heart), causing increased force of the cardiac contractions. This primary stimulation then gives way to paralysis, at first partial, then complete. Subsequent observations led him to conclude that digitalis slows the heart by its contracting influence on the arterioles, thus heightening arterial pressure. . . .

"The irregularity of the pulse is the capital indication for giving digitalis" in heart disease. (Ringer.)

One point more must be mentioned, and that a very important one, viz., that a poisonous dose of digitalis after a time *paralyses the arterioles*, which then dilate, and the arterial tension falls; therefore, if we are pushing the drug to its extreme effects in restraining hæmorrhage, we must beware lest the arterioles suddenly collapse.

I have called digitalis and ergot "arms of precision as well as of power;" but in doing so I limit the word *precision*, as applied to digitalis, to its effect in *slowing and regulating* the heart in those perturbations of its action so often found in hæmoptysis; and in my own practice I restrict its use in hæmoptysis to these two notable, reliable, and important functions.

It is to the action of *ergot* that the term *precision* may be applied without qualification. It is remarkable that its value in hæmoptysis should have been allowed to be almost forgotten until ten years ago, when I published the following note:—*On the Treatment of Hæmoptysis by Ergot of Rye.*—"In common with other physicians who, like myself, are connected with hospitals for diseases of the chest, I see every year a large number of cases of severe pulmonary hæmorrhage resulting from a variety of causes. . . .

"But every medical man of experience considers himself perfectly qualified to treat hæmoptysis; and it is almost the rule, therefore, that, when called to these cases in consultation, one of the first remarks of the doctor in attendance is, that 'everything possible has been done, and every remedy tried, but in vain.' It is assumed, in fact, that the

only object of the consultation is to sanction the inevitable death of the patient. Yet, according to my experience, it is exceedingly rare for a patient to die of hæmoptysis. In these remarks, I confine myself to cases of pulmonary hæmorrhage due to tuberculous disease of the lungs, which make up the large majority of all cases of severe hæmoptysis. . . .

"I attribute the success of my practice in this respect mainly to the use of ergot of rye; because it is quite true, as already suggested, that, in nearly every consultation-case of appalling hæmoptysis that I have seen, 'everything has been done, and every remedy tried,' *with the one exception of ergot of rye;* and the use of this remedy has generally turned the fate of the patient. It has always struck me as a singular fact, that general practitioners, who are so well acquainted with the effects of ergot in uterine hæmorrhage—who use it more frequently than any other class of practitioners—with whom, in fact, it is almost a 'pocket-companion'—never seem to think of using it in pulmonary hæmorrhage. I find, from frequent inquiry of my medical friends, that this is explained by the prevalence of the idea that ergot only acts by inducing contractions of the muscular tissue of the uterus; its remarkable power of inducing contraction of the blood-vessels being lost sight of. This is so generally the case, that I never (1868) met with but one general practitioner in the London district (in the country, it seems to be better known) who was at all aware of the power of ergot to control hæmoptysis. This exception was Dr. Betts, formerly of Watford, and afterwards of Ventnor, who has as much confidence in the remedy as I have myself, and dates his confidence from the occurrence that, when a student at Guy's Hospital, many years ago, suffering from profuse and intractable pulmonary hæmorrhage, under the care of the late Dr. Addison, his case was on the point of being given up as hopeless, when the bleeding was suddenly brought to a standstill by a large dose of ergot, administered at his own request; the idea having occurred to him, that, as it so often arrested uterine hæmorrhage, it might also answer in hæmoptysis.

"But I have said that, in the appalling cases to which I have referred, 'everything else had been done, and every remedy tried,' before I ordered the ergot; and I desire to attach the greatest importance to this fact. Ergot is only competent to do one of the many things necessary to stop a severe pulmonary hæmorrhage; viz., to contract the vessels. It is necessary to do much more than this.

"1. The vital power must be supported by brandy, iced milk, and beef-tea, if indicated by the general symptoms.

"2. The heart must be kept steady by digitalis.

"3. Congestion must be relieved by saline purging.

"4. Clotting of the blood must be promoted by styptics, and by the free admission of cold air.

"5. The bleeding part must be kept at rest by position, by enforced silence, and by soothing the cough.

"In spite, therefore, of the fashionable outcry against complicated prescriptions, I venture to give the following as the most efficacious, and, as it seems to me, the most rational, combination of remedies for a case of profuse tubercular pulmonary hæmorrhage. It has served me many a good turn, and I hope it may do the same for my professional brethren.

"℞ Ext. ergotæ liq. ʒ ij (to contract the vessels); tincturæ digitalis ʒj (to steady the heart); acidi gallici ʒj (to clot the blood); magn. sulphatis ʒ vj (to relieve congestion); acidi sulphurici diluti ʒj (to assist the rest); infusi rosæ acidi ad ℥ viij (to make a mixture). A sixth part every two or three hours till hæmorrhage is arrested.

"In any given case, either of the ingredients may be omitted, if the symptoms indicate that it is not required, or that it has already done its duty." ("British Medical Journal," 1868).

This article was immediately copied into various medical journals; and in Dr. Napheys' work on "Modern Medical Therapeutics" (Philadelphia) it soon acquired a world-wide circulation, and became a household word with many practitioners. In the "Casa de Salud" (Principal Sanitary Institution) at Havanna, I am informed by Dr. Sullivan that "Dr. Dobell's mixture for hæmorrhage" has a conspicuous label in the dispensing department, and is confidently relied upon by the medical officers.

From the date of the above communication to the present time ergot has steadily risen in reputation as the sheet anchor in hæmoptysis. It will be observed, however, that in my mixture it is carefully combined with the other remedies on which I have shown that we have reason to rely.

My friend, Dr. James M. Williamson, of Ventnor ("Lancet," Nov. 13, 1875), has recorded very satisfactory results in treating hæmoptysis with ergot; and while Resident Medical Officer to the "National Hospital for Consumption, Ventnor," he had abundant opportunities of observation. "Forty minim doses of the *liquid extract* were given: two in the first hour, and one every two hours afterwards, if necessary, the dose being diminished as the hæmorrhage subsided. No disagreeable effects followed; even if large doses were given in a short period; but, generally speaking, if four or five doses do not materially affect the hæmorrhage, another remedy should be tried. The preparation of the drug should be fresh and sound. The subcutaneous injection of ergotine is seldom necessary.

"Of fifty cases, in about one-fourth of which the hæmorrhage was profuse, in forty-four all bleeding was rapidly checked. *In forty of these it was the only drug used;* in two others it was effectual after the failure of alum, gallic acid, and dilute sulphuric acid; in another it succeeded after the ineffectual use of acetate of lead and opium; in the

other case it was used with success after the failure of both these plans.

"In six cases ergot failed, and in three of these hæmorrhage was arrested by gallic acid; in one, acetate of lead and opium availed; in one, oil of turpentine was successful after the failure of gallic acid; in the last case, gallic acid, ergot, acetate of lead with opium, perchloride of iron, ammonio-sulphate of iron, the mineral acids, and oil of turpentine were all used to no purpose.

"Ergot has the distinct advantage over gallic acid of not causing griping or constipation, and of not interfering with the liberal use of milk."—("Dr. Dobell's Reports," 1876.)

I have never seen the slightest harm or inconvenience result from the proper use of ergot administered by the mouth. In the human subject the general symptoms of a poisonous dose are "nausea, vomiting, delirium, and stupor, the pulse being diminished both in frequency and in force." (Drs. Woodman and Tidy.) "In large doses it produces nausea, vomiting, colic, diarrhœa, giddiness, headache, dilatation of the pupil, great retardation and slight weakness of the pulse." (Ringer.) If, therefore, we find a tendency to this train of symptoms, we may suspect that we are either overcharging each dose, or repeating it too quickly.

The well-known and frightful condition called Ergotism is never seen except as the result of chronic poisoning "either by the long continued administration of the drug in medicinal doses, or by eating bread made from corn which has been infected by it while growing. This dreadful disorder is manifested under two distinct forms, the convulsive and the gangrenous, the former not terminating in gangrene, whereas the latter is always distinguished by gangrene. The convulsive form commences with malaise, irritation of the whole surface of the body with formications, numbness and coldness of the extremities, often accompanied by cramp and by pains in the head and loins. Some time later, the digestive organs become affected. There is a sense of tightness and oppression about the epigastrium, heartburn, a feeling of lightness in the head, difficulty of hearing, with faintness, abnormal twitchings of the muscles of the face often attended by strabismus and irregular contractions of the joints. These symptoms are generally accompanied by delirium, bordering upon mania, with cold sweats, and the whole body is pervaded by a sense of great heat. . . .

"The gangrenous form of ergotism commences with a sense of pain and weariness in the limbs, the countenance becomes heavy-looking and stupid, and the skin acquires a leaden or cadaverous hue. Afterwards there is malaise with exhaustion, coldness of the whole body, and numbness of the hands and arms, while formication is perceived in every part of the surface; the lower extremities become similarly affected with the upper ones; later on the abdominal

muscles are spasmodically contracted; and about the sixth day from the commencement, nausea, vomiting, and diarrhœa make their appearance, with severe colicky pains in the bowels and bladder." (C. D. F. Phillips.)

I once saw gangrene of the extremities set in with other symptoms, very similar to the above, in a man, for whom a medicine containing a full dose of ergot had been prescribed for fatty degeneration of the heart, and who had continued to take the medicine, without further consultation with his doctor, for many consecutive months. But such cases are extremely rare, and there can be no reason or excuse in cases of hæmoptysis for prescribing ergot in such a manner as to run the slightest risk of chronic ergotism. As soon as the hæmorrhage has ceased the ergot may be withdrawn, and, if it does not quickly cease, some other remedy should be substituted.

Ergot is very quick in its action, and this is another of its great virtues in treating hæmorrhage.

In Dr. Ord's cases of thoracic aneurism, treated with ergot administered by the mouth, the pulse tracing was found to indicate increased tension of the arteries *in twelve minutes* after the patient had swallowed ℥30 of liquid extract of ergot, and this tension went on increasing till it attained its maximum in twenty minutes after the dose had been taken. ("Medical Examiner," March 8th, 1877.)

"Absorbed into the blood ergot causes contraction of the blood-vessels, and especially those of the cord." (Brown-Séquard.)

Dr. Ringer says:—"Whether administered by the stomach, or hypodermically, ergot causes contraction of the arteries and veins, by its influence, it is said, on the sympathetic system; administered in either way, it is most valuable in hæmorrhage; indeed, few, if any, remedies rival its efficacy in this respect. . . .

"In severe bleeding, when it is urgently necessary to check it at once, the hypodermic application must be used, in from *two to five grain doses*. I have seen this injection in many instances produce a good deal of swelling and pain, which, however, always subsided without suppuration; hence it is well to warn the patient that this temporary untoward accident may occur. In less urgent bleeding, administration by the stomach is very successful. It is very useful in hæmoptysis in doses of thirty or forty minims of the liquid extract every three or four hours, indeed, hourly in severe cases." (Op. cit.)

This brings us to the question of the best method of administration, *i.e.*, whether by the mouth or by hypodermic injection. I have never joined the crowd who have "gone cracked" over the hypodermic method of administering medicines; and I do not think the "Report of the Committee appointed by the Royal Medical and Chirurgical Society to investigate the hypodermic method of administering medicines,"

—when carefully considered—at all justifies the rage for this method that has prevailed of late years. The results arrived at are:—

"1. That, as a general rule, only *clear neutral solutions* of drugs should be injected.

"2. That, whether drugs are inserted under the skin or administered by the mouth or rectum, their physiological and therapeutical effects are *the same in kind*, though *varying in degree*.

"3. That symptoms are observed to follow the subcutaneous injection of some drugs which are absent when they are administered by other methods; and, on the other hand, certain unpleasant symptoms which are apt to follow the introduction of the drugs by the mouth and rectum are not usually experienced when such drugs are injected under the skin.

"4. That, as a general rule, to which, however, there are many exceptions, neutral solutions of drugs, introduced subcutaneously, are more rapidly absorbed and more intense in their effects than when administered by the rectum or mouth.

"5. That no difference has been observed in the effects of a drug subcutaneously injected, whether it be introduced near to, or at a distance from, the part affected.

"6. That the advantages to be derived from this method of introducing drugs are—*rapidity of action, intensity of effect, economy of material, certainty of action, facility of introduction in certain cases,* and, with some drugs, *avoidance of unpleasant symptoms.*"

This report, while stating that "certain unpleasant symptoms which are apt to follow the introduction of the drugs by the mouth and rectum, are not usually experienced when such drugs are injected under the skin," omits to notice the important facts, (1.) That very unpleasant and dangerous symptoms sometimes follow the introduction of the drugs by injection, which are avoided by administration by the mouth. (2.) That when such unpleasant symptoms set in, they are more out of the reach of antidotes and remedies than when the drugs have been put into the stomach. (3.) That hypodermic remedies cannot be repeated without the presence of the doctor—a very serious objection in all emergencies, and in country practices, and in the night; unless, (4.) he teaches the patient or nurse how to use the injection; a piece of information which, it is now too well-known, is apt to be frightfully abused.

Rapidity of action is the one prominent feature of hypodermic injection to set against these objections to its use; and there are certainly cases in which this is worth almost any risk and any inconvenience; but if properly eliminated, such cases are very few and far between. In regard to ergot, when we learn that its physiological and therapeutical effects, when administered by the mouth, are detectible in the pulse-tracing in twelve minutes, and culminate in twenty minutes, we must see that there are very few cases, even of hæmoptysis, in

which this action is not rapid enough to answer all practical ends. Still, I say, we are right to be ready with even more expeditious means in case of need, and then we may use the ergot hypodermically. It is important to select the form for injection which not only possesses the greatest precision and potency, but which is least likely to produce the painful after-inflammation of the skin, referred to by Dr. Ringer.

Dr. Josef Hirschfeld ("Mittheilungen der Wiener Med. Doctoren-Collegiums," 1877) recommends as the best remedy, the hypodermic injection of *Ergotine* in concentrated solution (1 part in 10 of glycerine) as already used by Dr. Drasche in Vienna in 1871. The solution in glycerine is more complete and fungi do not develop so early as in the aqueous preparations. The author injects morphia either before or at the same time as the ergotine, in order to diminish the desire to cough. When given with morphia, he considers that the hæmostatic effects of ergotine are better developed.

Mr. Alfred Tanner (Chemist), of Fairfield, Liverpool, has introduced a very satisfactory "permanent solution of ergotine for hypodermic injections," the advantages of which are quite fairly set forth in his prospectus as follows:—

"This solution has been designed with a view of overcoming the objections usually experienced in the use of the so-called ergotine in the solid form, that substance as ordinarily met with being merely an aqueous extract from the ergot, without any attempt at purification or separation from the inert and non-diffusible substances which invariably accompany it; hence its use is attended with many disadvantages to the operator and much inconvenience to the patient.

"These may be briefly summarized thus :—

"1. Trouble and loss of time in dissolving it in water.

"2. Uncertainty of dose so prepared.

"3. Pain and irritation produced by the insoluble and non-diffusible matter contained in it.

"4. The impossibility of keeping such a solution free from decomposition and from becoming inert.

"The proprietor believes all the above difficulties to be overcome in his 'permanent' solution of ergotine. It contains the *full activity* of the drug, will *keep* almost *indefinitely* without change, is always *ready for use* and *uniform in strength*. It contains neither acid nor alcohol, and consequently is little liable to form abscesses or cause pain.

"The form in which the 'permanent' solution of ergotine first appeared has been much improved, being now sent out in sealed tubes, which it is believed will offer greater convenience in use, with less liability to change.

"The strength remains the same, viz.—Three grains in ten minims, and the dose for hypodermic use will average *five or ten minims*; this however may be greatly increased in urgent cases."

In my own practice I prescribe the Ext. Ergotæ liq. of the British Pharmacopœia, in doses of from twenty to forty minims, every one, two, three, or four hours, according to the urgency of the case; and I believe that in this way we may obtain all the important effects which ergot is capable of producing, without any disadvantages; and I know no more generally useful form in which to prescribe it than that of the mixture already described. (p. 136.)

I have never tried ergot in the form of spray; but Dr. Ashe has injected the uterus with a 12 per cent. solution of ergotine without producing irritation, and with the effect of arresting hæmorrhage; and it seems possible that it might be used locally with advantage in hæmoptysis.

Dry cupping, and hot sponges to the dorsal spine, and sedatives, must be borne in mind as important additions to our treatment. Dry cupping and hot sponges to the spine to relieve local congestion, while other remedies are taking effect. Sedatives to allay needless coughing. I say "needless coughing," advisedly, because it is a mistake to suffocate the patient with retained blood by stopping the cough with stupefying doses of sedatives. He should be allowed to cough enough to clear the larger air tubes; but irritable, teasing, tearing, or jarring cough should be soothed by poppy, lettuce, gelseminum, bromide of potassium, or even morphia if necessary; but, as a general rule, the sucking of ice and of lemons answers this purpose, while doing good in other ways.

I have recorded the ipecacuanha treatment so much vaunted by Trousseau (see p. 109), without at present commenting upon it. I do not doubt its efficacy in well chosen cases, or its risk in others. Had Trousseau known as much as we now know of the occurrence of small aneurisms in the walls of cavities, as causes of profuse hæmoptysis, I think his usual sagacity would have led him to use ipecacuanha less incautiously in its emetic doses, and to restrict it more to its nauseating effects. The stage of overpowering faintness, preceding violent ipecacuanha emesis, is unquestionably favourable to the arrest of hæmorrhage by clotting; but I think few would have the hardihood deliberately to excite violent vomiting, if they could see that the hæmorrhage was pouring out of a ruptured aneurism, on a branch of the pulmonary artery, in the wall of a gaping cavity. In my opinion the emetic treatment, if used at all, should be restricted to the profuse hæmoptysis of the earliest stages of tuberculosis, *i.e.*, to capillary hæmorrhage.

The powerful effect of veratrum viride in depressing the pulse has led to its being suggested as a remedy in pulmonary hæmorrhage. But it is too fitful in its action to be relied upon, and the results of

experiments, show that, like that of aconite, its effect upon the small arteries is rather to relax than to contract them; and this alone should be fatal to its adoption in hæmoptysis.

I have already expressed my opinion and advice that we should avoid unnecessary amplification of remedies, and select a few that are reliable, and use them with vigour and judgment. Of these I have now given an amply sufficient list. Therefore in concluding this part of our subject I only add the following as curiosities :—

Benicassa cerifera.—Dr. Baboo Udhoy Chand Dutt, Civil Medical Officer, Nookhally, says ("Indian Med. Gaz.," 1874), "this plant belongs to the natural order *Cucurbitaceæ*. In English it is called Squash or Vegetable Marrow. Its Sanscrit name is Kashánda. The large fruit or gourd is a culinary vegetable, and is extensively cultivated all over India for that purpose. *The pulp of the ripe fruit is a specific for hæmoptysis*, for which it is extensively used by the native physicians. I will now give an account of two of the preparations in common use, and the virtues ascribed to them. . . .

"1. Kashánda Khanda, or confection of squash.—In preparing this, old ripe gourds (the older the better) are selected, those not at least a year old are rejected. The fruits are longitudinally divided into two halves. The pulp is then scooped in thin flakes by an iron comb or scratcher. The watery juice that oozes out abundantly during this process is preserved, the seeds being rejected. The pulp is boiled in its water till softened. It is then tied up lightly in a cloth, and the juice, or watery portion, allowed to strain through. The softened pulp is dried in the sun, and the strained juice preserved for subsequent use. For preparing the confection, take 12½ chittacks of the prepared pulp ; fry it in 4 chittacks of ghee, or clarified butter ; add the strained juice, and boil again till the whole is reduced to the consistence of honey. Then put in 12½ chittacks of refined sugar, and heat over a gentle fire till the whole assumes such consistence as to adhere to the ladle. Now remove from the fire, and add the following aromatic substances, finely powdered: long pepper and ginger, of each half a chittack; carraway-seeds, cardamoms, cinnamon, *tejpat* (leaves of laurus cassia), black pepper, and coriander, of each half chittack. Stir briskly with a ladle till cool, then add honey 2 chittacks, and preserve in a new clean earthen pot. The dose of this medicine is from one to two tolahs, according to the age and strength of the patient. . . .

"Chakradatta thus describes its uses: It is useful in hæmoptysis, phthisis, marasmus, cough, asthma, giddiness, dyspepsia, thirst and fever. It increases the strength, improves the colour, and removes the effects of age. It heals ulceration of the lungs, cures hoarseness, and acts as a general tonic.

The other preparation is called Basa Kashánda-Khanda. Dr. Chand Dutt says:—"In my own practice I have often found these

medicines succeed in putting a stop to bleedings from the lungs, and relieving the cough and expectoration of phthisis when our ordinary medicines failed. . . . In a patient under my care . . . who suffered from several attacks of profuse hæmoptysis during the course of a strongly-developed disease, Kashánda-Khanda was the only medicine which seemed to have any beneficial effect on the bleeding. In the last attack of hæmoptysis he had, the bleeding was so copious that I was quite alarmed, and, unwilling to trust to native medicines, I pushed on vigorously with our ordinary medicines, such as gallic acid, sugar of lead, turpentine, stupes, etc. Days passed on without relief, and every morning I had the mortification to find from half-a-pound to a pound of blood preserved for my inspection. The patient now suggested that I should prescribe Kashánda-Khanda, which had relieved him on previous occasions. The medicine was prepared and given to him, and from that date the blood began to decrease as if by a charm, and quite disappeared in about a week." ("Dr. Dobell's Reports," 1875.)

I am informed that the Indians in Western America smoke Canada Balsam with their tobacco to stop hæmoptysis, and that it is effectual.

Dr. Richard Neale, the author of "The Digest" already referred to, tells me that, when he was practising in Java, he often saw obstinate hæmoptysis stopped by Malays and Chinese, after his own remedies had failed, by the administration of their favourite native remedy, viz., half-a-pint of the urine of a child. He says it does not act as an emetic, and his notion is that the urea enters the blood, and produces contraction of the arterioles.

I have now shown the very efficient means which we have at our command for the attainment of the "main objects to which our treatment of hæmoptysis is to be directed," viz., 1. To clot the blood about the bleeding part. 2. To relieve vascular tension in the bleeding part. 3. To contract the vessels in the bleeding part. 4. To stay the rush of blood to the bleeding part. 5. To maintain the above conditions for a sufficient length of time to prevent a recurrence of the bleeding. 6. To remove or avoid the causes which excited the hæmorrhage. 7. To remove or avoid the causes which predispose to the hæmorrhage. 8. To prevent inflammation and disorganisation of the parts implicated in the bleeding.

I have referred to my prescription published in 1868, as a generally useful form in which to administer many of the most potent of the remedies enumerated for the attainment of the above ends. But it must not for a moment be supposed that I advocate an *ad captandum* treatment of all cases of hæmoptysis, either by this or by any other medicine or combination of medicines.

Those who have read the foregoing pages will have found abundant

evidence of this; and now I wish to emphasise it, by pointing out in a few words the SPECIAL ADAPTABILITY OF OUR SEVERAL REMEDIES TO SPECIAL FORMS OF HÆMOPTYSIS.

Casting aside, for the moment, the elaborate and minute questions of diagnosis and pathology, which have already been fully discussed, the special forms of hæmoptysis and the remedies most suited to each may be clinically arranged under the following heads:—

1. Profuse and dangerous cavernous hæmoptysis, from rupture of aneurisms formed on branches of the pulmonary artery, in the walls of cavities, and from ulcerative erosion of large vessels traversing cavities. (See pp. 80—83.)

Our first and main object here should be, to block up the cavity with clotted blood; our chances of saving life in the first emergency turn upon our succeeding in this. Rest and Posture are our first remedies. We must take advantage of the well-known fact that patients with open cavities can sleep best on the cavernous side, because the secretions from the cavity are thus prevented from continually draining into the bronchi and exciting cough. Therefore, in cavernous hæmorrhage, place the patient semi-recumbent on the same side as the cavity from which the bleeding proceeds. This position will favour the accumulation and stagnation of blood in the cavity, and its clotting should be at once promoted by the *topical use of styptics* in atomised fluids, or—in the absence of a spray producer—by the aspiration of impalpably powdered alum. It is to these cases that the *topical use of stypics* is specially suited.

Cold, ergot, and digitalis are next in value, from their rapid action; and to save every moment that is possible, hypodermic injection of ergotine is here eminently suitable. Faintness, short of syncope, should be encouraged, as it specially promotes our object; and, therefore, stimulants should be withheld till urgently required. All our other remedies may also be brought to help in turn.

These are the cases which have surrounded the idea of "breaking a blood vessel" with all the terrors of sudden death. And it is unfortunately true that we may sometimes find it impossible to avert it. The vessel may be too large, and the rupture too extensive for any remedies to act before the patient is overwhelmed.

The same remarks, and the same treatment are applicable to—

2. The profuse hæmorrhage of aortic aneurisms; and in a less degree to those of cancer.

3. Capillary hæmorrhage of the first stage of pulmonary tuberculosis. Unless very profuse this need not cause alarm. (See p. 103). The hæmorrhage may even be advantageous by temporarily relieving the hyperæmia, and this end should be assisted by rest—general bodily rest, and local functional rest—as already described (p. 123), and by the eliminative action of sulphate of magnesia. It is only when the hæmor-

rhage is profuse that other remedies should be used, and then ergot or lead are specially suited, and iron specially to be avoided. (See p. 129.) The constitutional condition of oxidation, and lack of fat in the blood, is the point to which our attention must be turned directly the emergency has been met; for this is the *primum mobile* in the case, the pre-disposing as well as the exciting cause.

4. The hæmorrhages occurring in the course of advancing pulmonary disintegration as distinct from "*cavernous hæmorrhage*," are best met by the combination of styptics, saline aperients, ergot, and digitalis, and by topical styptic inhalations of atomised fluids. It is in these cases, also, that lung-tissue is most in danger of being hopelessly broken down by the hæmorrhage (see p. 99); and in which, therefore, the after watching of the temperature, counter irritation and antiseptic inhalations are most likely to be called for. If the hæmorrhage is obstinate, and vascular excitement absent, turpentine is here a specially well suited remedy, for the patient is generally feeble and cachectic, and the turpentine answers several purposes at once. (See p. 128.)

5. In hæmorrhagic infarction, bleeding should not be stopped unless it is severe, and in that case atomised styptics are the best suited. The most important point is, to find the seat of the thrombosis from which the embolus sprang, and to protect the patient against a recurrence of the accident. The next point is to lie in wait for pulmonary gangrene, and if this is indicated by the character of the expectorated blood, to use turpentine externally and internally, and carbolic acid in vapor or spray. Professor Gerhardt says, "You will treat violent hæmorrhages (in hæmorrhagic infarction) with inhalation of perchloride of iron, and the use of tonics will be in various ways indicated. Under certain circumstances, you will seek to hinder the gangrenous softening of the infarction by antiseptic inhalations. . . But on the whole, this proposition is applicable; the infarction which is not infected either by the embolus or by the inspired air, cures itself. The danger lies in the embolism. If the infarction forms, the danger is already past."

6. Purpuric hæmorrhage is best treated with turpentine and iron.

7. The hæmoptysis of retrograde congestion specially requires digitalis, sulphate of magnesia purging, rest, and appropriate position.

8. Vicarious hæmoptysis, due to the excessive vascular tension associated with the menstrual period, is best treated by cholagogues and aperients, and by the usual means of restoring normal menstruation. Its recurrence should always be anticipated and prevented by timely remedies. If normal menstruation is obstinately absent in spite of the usual means for its restoration, and the pulmonary hæmorrhage insists on periodically recurring, a leech or two applied to the sacrum, or near the anus, or, as Trousseau recommends, on the knees, when the monthly period threatens, is an efficient means of treatment.

According to my experience, this form of hæmoptysis is very common, and of itself does no serious harm. It need not, therefore, cause any alarm, were it not for one circumstance, and unfortunately that is a very serious one—viz., that the reason why the lungs become the seat of the vicarious hæmorrhage instead of some other vascular organ, may be that they are rendered vulnerable by pre-existing or impending disease. It is with this view that the case must be carefully investigated, and both at the time and afterwards, vigilantly watched; and it is especially under the guidance of this view that we must select our remedies.

PART IV.

LOSS OF WEIGHT & PULMONARY CONSUMPTION.

GENERAL DISCUSSION OF THE SUBJECT.
TWO GOOD WORKING HYPOTHESES.

(See pages 161 to 169.)

"It is clear that fat exerts a protective influence over the albuminous tissues, sparing their consumption or oxidation by its own greater affinity for oxygen. . . . When fat is added to the food, its direct combustion takes up the oxygen, and prevents its action on the nitrogenous tissues."—Carpenter's "Principles of Physiology," 6th edition, page 265.

"I quite agree with you that there are very strong grounds for believing that such an oxidation (of fibrin or albumen) will not take place in the protecting presence of fat. The view of muscular action which I have advocated lends great support to your theory. The oxidation of fibrin or albuminoid substances *in situ* is an abnormal or morbid condition of things."—Professor E. Frankland, F.R.S., etc., Royal College of Chemistry, London, 1867.

"I quite agree with you that tubercle is the result of excessive oxidation, and not the reverse."—Professor S. Haughton, M.D., F.R.S., etc., Trinity College, Dublin, 1868.

"Many spectroscopic examinations of de-oxidised hæmatin spectra have shown that in the case of phthisical blood a *third band* is frequently observable. This band is found when the original blood is in a hyperoxidised condition, this hyperoxidised state being capable of imitation by artificial means. It may, therefore, be concluded that Rokitansky was right in his statement, made before the discovery of spectroscopic analysis, that *the blood in phthisis is in a hyperoxidised state.*"—C. Meymott Tidy, M.B., F.C.S., Professor of Chemistry, London Hospital, etc., etc., 1878.

"I was asked only yesterday by a medical friend . . . what I thought of your hypothesis of tuberculosis, and I can only say to you what I said to him, that it *was very ingenious and very possibly true.*"—G. E. Day, M.D., F.R.S., Professor of Medicine, St. Andrew's University, Editor of Simon's, Rokitansky's, and Lehmann's Works on Chemistry and Pathology, 1866.

"It is recognised as an axiom amongst scientific speculators that the best tests of a scientific hypothesis are found in the simplicity of its conception and the universality of its application. If the solution of all observed phenomena is found in a proposition, unencumbered with provisoes and additions to meet special cases, the proposition proves itself."—"Times," April 15, 1874.

Compare the above with the following;—

"We might state as broadly as is consistent with courtesy that it (Dr. Dobell's hypothesis as to the nature and cause of tuberculosis) can scarcely be true; *on the ground that a state so complex as tuberculosis can scarcely be accounted for by reference to any single condition.*"—"Medical Times and Gazette," Review, Feb. 2, 1867.

LOSS OF WEIGHT & PULMONARY CONSUMPTION.

PART IV.

The Maintenance of a Stationary Weight a Wonderful Evidence of the Perfection of Vital Existence.—Immense Influence of Loss of Weight on the Possible Continuance of Life.—Great Value of Means of Preventing or Remedying Loss of Weight.—Expectoration, Sweating, Diarrhœa, High Temperature, Defective Quantity or Quality of Food, Excess of Exercise over Food Supply, as Causes of Loss of Weight.—Loss of Weight Independent of all these Causes Typical of the True First Stage of Consumption.—Cause of this Loss of Weight explained.—Why Loss of Weight in Consumption is so Difficult to Restore.—Importance of Fat, and the Relative Value and Properties of Liquid and Solid Fats.—Essentials of a Diet capable of maintaining Heat, Mechanical Force and Histogenesis.—Emaciation in Different Stages of Consumption explained.—The Relations between Loss of Flesh and Tuberculisation, and the Order of Precedence in Tuberculisation of Organs explained.—The Explanation of Loss of Flesh before Tuberculisation and the Explanation of Tuberculosis and Tuberculisation are Two Distinct Hypotheses.—Importance of Two Good Working Hypotheses in suggesting Proper Treatment.—Singular Neglect of the Subject of Consumption by Scientific Men, after Laennec.—Its Revival in the Present Day.—The Author's two Hypotheses submitted to Rigid Criticism in the Light of Modern Research, and the Probability of their Correctness established thereby.—No other Hypotheses attempt to explain the Typical Clinical Fact of Consumption, viz., Loss of Weight Antecedent to Tuberculisation.—Answer to the Question, What is Tubercle?—Tubercle classified according to its Causes.—Critical Examination of the Views of different Authors, with Comments.—The Importance of the Pancreas, so long Neglected, now Established.—Causes of Defective Pancreatic Function Compared with the Causes of Consumption, and Classified in Groups.—Categorical Answers to the Problem set forth, Part I., p. 8.—Tuberculæmia.—Septicæmia.—Contagion.—Antiseptics.

THE fact that it is possible for a human being to maintain from year to year a stationary weight, has always appeared to me one of the most wonderful evidences of the perfection of vital existence.

When we consider the vicissitudes of rest, of activity, and of food, both bodily and mental, to which an average human being is subjected in average daily life, and the myriads of intricate processes concerned in nutrition, development, maintenance, growth, and repair; it is simply marvellous that waste and repair should be equally balanced, not only from day to day, but from year to year. Yet it is not uncommon to meet with both men and women whose average weight has not appreciably altered for many years.

A proper weight having been attained, after the complete growth

and development of the organism, it is no doubt the normal condition of things that it should be equally maintained through a large portion of adult life; and that it has been so maintained without artificial intervention is one of the most unequivocal proofs of health. While, on the other hand, any marked tendency either to gain or lose should always be a subject for serious investigation by the physician. Many and diverse circumstances may necessitate a change of weight without any necessary divergence from a healthy state, and all of these must be considerately investigated before we decide that loss or gain in weight is a sign of disease.

The immense importance of loss of weight is strikingly shown by the experiments of Chossat on the effects of inanition in animals. ("Recherches Expérimentales sur l'Inanition," Paris, 1843.) He found that, without the existence of any disease, death always occurred in simple inanition when the total loss of weight reached 45 per cent. of the normal body weight. According to these experiments therefore it is impossible to live, even without the intervention of disease, when half the normal weight has been lost. Now suppose a person's average weight to be 144 lbs., and he is losing weight at the rate of 24 oz. per week, or 6 lbs. per month; it is inevitable that, unless this is stopped, he must die in less than 12 months, that is, before he has lost 72 lbs. or one half his usual weight.

In the recent case of Harriet Staunton, who died of supposed starvation at Penge, the weight after death was 74 lbs. ("The Times," September 21, 1877); her average normal weight was not stated, but as her height was 63 inches, her normal weight, according to Dr. Woodman's tables, would have been about 120 lbs.—so that at the time she died she had lost about 46 lbs., or 14 lbs. less than one-half her probable average weight. In another recent case in which death occurred from "starvation from non-assimilation of food," due to melancholia ("Lancet," October, 1877); the patient, a woman of 55, weighed at death 55 lbs., and her height was 63 inches; her normal weight was not given, and supposing it to have been up to Dr. Woodman's average of 120 lbs., she would have lost 5 lbs., more than one-half her average body weight; but as a melancholic person, and 55 years old, she probably was naturally a light weight, and may not have exceeded 110 lbs., which would make the loss equal to one-half her weight.

It will be seen by my analysis of the weights of one hundred cases of hæmoptysis (Tables 8 and 9) that the average loss of weight per patient had been at the rate of between 8 and 9 pounds per year. In two cases the rate of loss averaged 22·6 pounds per 365 days; in three cases, 22·941 pounds; in eleven cases, 12·108 pounds. The total loss of weight per patient averaged 33·363 pounds in eleven cases, and in one of these the total loss had reached 63 pounds; it averaged 20 pounds in eighty-

two cases, and 17 pounds in eighteen cases. It must be borne in mind that all these patients were still living at the time the weights were taken.

According to Chossat's results it would be impossible for a patient whose normal weight was 120 pounds, to live three years with an annual loss of 20; and we see therefore how desperate must be the condition of a patient who is suffering, not from simple inanition, but from an exhausting and distressing disease, and is losing weight at the rate of 22·941 lbs. per annum, or even at between 8 and 9 pounds per annum,—the average rate in all of my one hundred cases.

In the light of these facts, we cannot fail to be impressed with the immense and beneficent importance of any medicines or medicinal foods, which have the power to prevent, to overcome or to mitigate this deadly symptom.

I am sure that, as a rule, we do not set sufficient importance upon remedies which assist, in other ways than oil and fat, in staying waste; such, for example, as those which check excessive perspiration and excessive expectoration and diarrhœa. The dire inroads upon vitality made by these excessive excretions will be best appreciated by considering them in conjunction with the foregoing facts.

Let us remember that if a patient, weighing 120 pounds, spits up one pound per twenty-four hours, the expectoration alone will amount to a loss of half his body-weight in sixty days; or if he spits half-a-pound per twenty-four hours, it will be equal to half his body-weight in one hundred and twenty days; or if one-quarter of a pound, it will be equal to half his weight in two hundred and forty days. The loss of weight in health that may be produced by sweating is a fact of every-day observation with trainers. It has been supposed to be due chiefly to the loss of fat by the sebaceous secretion, and to the large quantity of salts which escape with the perspiration. The great accession of emaciation which accompanies excessive sweating in disease is equally familiar to medical men; while nothing reduces weight so rapidly as diarrhœa.

The subject of the balance between waste and repair in the living organism having been one of intense interest to my mind from an early period of my professional career, in the hope of discovering the secret of the wasting in the early stage of consumption, I devoted many years to a rigid investigation of every point that I thought could throw light upon it. These studies formed no inconsiderable portion of the work to which I referred when I stated in 1866, that "the greater part of my leisure during the last sixteen years has been devoted to the study of the natural history of pulmonary consumption and other forms of tuberculosis."—("On the Nature, Cause, and Treatment of Tuberculosis.")

It is, of course, plain enough that, supposing assimilation and nutrition

to be normal in quantity and quality, if there exists any abnormal drain upon the system there must be, either a compensatory increase in the amount of food assimilated and utilised in nutrition, or a loss of body-weight.

If, therefore, we find loss of weight coincident with diarrhœa, sweating, profuse expectoration, abnormally high temperature, profuse mucous or muco-purulent discharges, and so forth—although ingestion and digestion of food are going on at the usual rate; there can be no secret as to the cause of the loss of weight. Or if, on the other hand, we find loss of weight unattended by any excessive or abnormal secretion or excretion, but coincident with diminished ingestion of food; there can be no secret as to the cause of the loss. Or again, if we find normal average but not increased ingestion of food, normal secretion and excretion, but excessive bodily labour coincident with loss of weight, there can be no secret as to its cause.

But how shall we explain a case in which none of these causes of extra waste exist, and in which there is no diminution in the quantity of ingested food, and no apparent difficulty in digesting a normal or even an increased quantity of average food, and yet in which a steady loss is taking place in weight and in strength. Yet, that is the conjunction of circumstances typical of the true first stage of consumption.

In 1853, while engaged in the investigation of the causes of fatty-liver in phthisis, I was struck with the fact that the following group of apparently incongruous phenomena may co-exist in a case of advanced consumption; and it gives the clue to the explanation of the cause of the loss of weight in the early stages of the same disease, *i.e.*, before any lung substance has been damaged.

(*a.*) A considerable portion of the lungs deprived of decarbonising and of oxidising function.

(*b.*) A fair appetite and the ingestion of a normal quantity of food.

(*c.*) A steadily progressing diminution of the carbon of the tissues.

(*d.*) Blood not surcharged with carbon, but the contrary.

Reliable observations on the blood in phthisis are very rare, but those of Becquerel and Rodier ("Animal Chemistry," 1857, Speer's translation) showed that in phthisis the blood is more deficient in fat than in any other disease; and Rokitansky pointed out that the blood in phthisis is in a hyperoxidated state. But the most recent and most interesting contribution to our knowledge of this subject is due to spectroscopic analysis. My distinguished coadjutor, Dr. C. Meymott Tidy, informs me, as the result of many spectroscopic examinations of deoxidised hæmatin spectra, "that in the case of phthisical blood a *third band* is frequently observable. This band is found when the original blood is in a hyperoxidised condition, this hyperoxidised state being capable of imitation by artificial means." It may, therefore, be concluded that Rokitansky was right in his statement, made before the

discovery of spectroscopic analysis, that *the blood in phthisis is in a hyperoxidised state.*

(*e.*) No sickness, diarrhœa, or other flux,—there may be neither sweating nor expectoration of any amount.

(*f.*) Arrested menstruation in females.

Menstruation in health has a definite relation to the quantity of carbonic acid discharged from the system by other means. From the occurrence of puberty to the cessation of menstruation, so long as healthy nutrition is maintained, everything which arrests menstruation increases the discharge of carbonic acid from the lungs ; and the recurrence of menstruation reduces that discharge. It is evident, therefore, that the menstrual flux is a means of eliminating carbon. Now, it is well known that, previous to the appearance of local symptoms of tubercle, the catamenia usually become irregular, and gradually diminish in quantity until, as the disease goes on, they entirely cease ; that permanent arrest of menstruation is a usual occurrence in confirmed and progressing tuberculosis ; that any prolonged arrest in the disease is usually attended with a restoration of menstruation ; that, when menstruation coincides with confirmed and progressing tuberculosis, all the symptoms of the disease are usually increased at the monthly period; that a large number of cases of phthisis commence at the age of puberty ; and (which appears at first very curious) that at this age, among those attacked, there is a large preponderance of males over females.

As I read these facts, they have the following meaning. In females, the balance of their nutritive functions is so arranged that, at the age of puberty and during the whole generative period, there shall be, normally, a surplus of carbon in the system. At the age of puberty, this superfluous carbon protects them from some of the dangers of tuberculosis to which boys, who have no such surplus to fall back upon, are exposed. Thus, a girl at the age of puberty, temporarily deprived of some of the normal supply of properly prepared fats, simply suffers a delay in the appearance of menstruation, appropriating her surplus carbon for the protection of the tissues ; whereas a boy, similarly placed, has no such means of escape.

The disappearance of the catamenia when tuberculosis has set in is explained by the deficient supply of fat to the blood cutting off the surplusage of carbon for excretion. The restoration of the catamenia during periods of arrested tuberculosis is the expression of a return to the normal proportion of carbon in the system. With this clue, many other analogous phenomena may be easily explained.

All attempted explanations of the conjunction of circumstances (*a*, *b*, *c*, *d*, *e*, *f*,) above detailed, failed, when rigidly scrutinised, except the assumption that the fats of the food, or the fat-producing elements of food, did not find their way into the blood of the right heart in normal

proportions and conditions, and that this was due, not to the failure in the ingestion of these materials, but to defect in their assimilation when ingested.

This hypothetical explanation I published in the beginning of the year 1866 ("British Medical Journal," Jan. and Feb.), and up to the present time no one has been able to offer any more reasonable explanation of the facts. On the contrary, the accumulated clinical experience of numerous observers has abundantly confirmed the correctness of my suggestion; but I shall return to this subject directly.

Taking it for granted, for the present, that we have to deal with a case in which loss of weight has occurred simply through defect in the passage of fat from the food into the blood; that the patient has no difficulty in taking the normal proportion of fat and fat-forming materials with the food; and assuming that we have at our command a means of supplying the defect in the natural powers of digesting and assimilating these materials, that is to say, that we can, by an artificial manœuvre, place the patient again in a normal position with regard to the power of taking fats into the blood:—taking all this for granted, nothing is more natural and reasonable than to ask for some explanation of the frequent failure of our attempts to keep up that favourable change which so often occurs in case of consumption when cod liver oil is first administered. We all know how frequently it happens that a consumptive patient makes remarkable progress for a certain time while taking cod liver oil if it is well digested—a progress which might well lead us to hope that it would end in a cure; and we all know equally well how frequently this progress stops at a certain point, beyond which recovery does not seem able to advance, and from which it too often happens that a gradual descent takes place. In explanation of the above difficulty, I suggested, in 1865, "that, assuming a defect to exist in the natural powers of digesting and assimilating fats, it would be irrational to expect anything else to happen than that which we witness. . . .

"According to the careful estimate of Dr. Lyon Playfair ('The Food of Man in Relation to His Useful Work,' 1865), the quantity of fat required by an adult in twenty-four hours to keep up healthy nutrition is from 1 oz. to 2·5 oz.; and according to the estimates made from numerous and carefully selected data by Mr. Farrants and myself ('A Manual of Diet and Regimen,' 1864), the quantity is from 2 oz. to 3·5 oz. . . .

"We may fairly assume, then, that not less than two ounces of fat per day, on an average, is required to keep up healthy nutrition in an adult. We have next to bear in mind, that before a case of consumption ordinarily attracts attention, and begins to be treated as such, many pounds weight, principally consisting of fat, have been gradually removed from the body. . . .

"In this condition—1, a deficiency of fat throughout the organism; 2, a loss of the power to assimulate ordinary fats; 3, a constant demand for two ounces per day to maintain healthy nutrition,—we administer cod-liver oil, in the belief that this form of fat will assimilate when other forms will not. Supposing that it agrees and that some or all of it is utilised, a rapid improvement takes place in the patient, from the supply of some of that for want of which life was steadily fading—very much as a cut flower, that has drooped for want of its supply of sap, rallies and recovers freshness for a time when put into water. But there are very few persons who can take more than from half an ounce to one ounce of oil per day—few who can even take this steadily from week to week without intermissions. But supposing an ounce or an ounce and a half per day to be taken regularly, how is this to supply, not only the two ounces per day required for healthy nutrition, but all the extra ounces of arrears that were lost before the treatment was begun? But assuming the possibility of two ounces per day of oil for nutrition and another two ounces for arrears being taken and utilised, even then the *whole thing may be unstable and may break down*, from the fact that we are supplying oil and *not solid fat*—a body rich in olein and poor in stearin and margarin, in the place of bodies rich in stearin and margarin and poor in olein, such as the fats taken in normal food. . . .

"The practical conclusion from these considerations appears to be, that if we are to give a fair chance of recovery to a patient deprived of the natural powers of digesting and assimilating fats, we must, by one means or another, secure that two ounces of fat of average solidity are utilized every day for the purposes of nutrition, and an additional ounce or two to make up for arrears." ("Lancet," Nov. 11 and 18, 1865.)

With regard to the distinction which I have here drawn between fats rich in olein, and those rich in stearin and margarin, it may be well that I should in this place introduce the following extracts from a paper which I published in the "Chemical News" of Sept. 4, 1868, after a full investigation of the subject.

"The most usual objections to my views, as to the importance of distinguishing between solid and liquid fats, with which I have met among medical men, have been based upon the similarity in the chemical formulæ for stearin, margarin, palmitin, and olein. Such objections will of course have little weight with those acquainted with isomerism; on the contrary, it appears to me that the peculiar isomeric modifications of which stearin and palmitin are susceptible, as shown by Duffy, pointedly distinguish them from olein, which, so far as at present known, has not this susceptibility—a distinction which is supported by the different behaviour of oleic acid towards chlorine and bromine, from that of stearic or margaric acids (Lefort),

and by the different action of bile upon stearic acid and upon oleic acid (Marcet). . . .

"But I think we ought to be prepared to learn that solid and liquid fats differ in some important physiological properties, by the first general fact concerning the constitution of all natural fats—viz., that they are mixtures in varying proportions of at least four different bodies, of which the melting points so widely differ—stearin melting at 144° F., palmitin at 114·8° F., margarin (probably a compound of stearin and palmitin) at 116° F., while olein remains fluid at 32° F.

"That the different degrees of solidity of fats depend upon the proportions in which the solid ingredients are mixed with olein; that olein has a peculiar power of dissolving the solid ingredients; and that the melting point of the mixture is thereby reduced; appear to me to be facts pointing in the same direction as the foregoing, especially when we remember that the affinity of oleic acid for oxygen is much greater than that of the other fatty acids. . . .

"The fatty bodies obtained from warm-blooded animals are generally solid at ordinary temperatures, whilst those from fish and from cold-blooded animals are liquid. And when we consider the high melting points of the solid fats as compared with the temperature of the body in warm-blooded animals, it is evident that the fat in them would be solid at the temperature of their blood, but for the mixture of olein, by which the melting point is reduced. Therefore the solidity or fluidity of the fat in living animals is determined by the proportion of olein, which is able to be mixed with the stearin, palmitin, and margarin in each individual; and we are forced to conclude, *either that it is of no importance whether the fats of the body during life are in a solid or liquid state, or that it is important in what proportion the olein, stearine, etc., shall be combined.* . . .

"It has been already proved, by experiments on the fattening of cattle, that the solidity or fluidity of the fat in the body varies with the food—that cattle fattened upon linseed cake, for example, accumulate, in their adipose tissue, an oily material of unusual fluidity (Draper), and that the consistence of butter is dependent upon the kind of food given to the animals from which it is produced (Fownes). . . .

"The fat in animals is particularly liable to accumulate immediately beneath the cutis, in the omentum, and around the kidneys; and the fat found in the latter situation, where it is subject to a more uniformly elevated temperature than in the integument, is well known to be of a more suety character—that is to say, it contains a smaller proportion of olein, and has a higher melting point. These familiar facts point again to some importance in the animal economy, attaching to the melting point of the fat and the consequent degree of fluidity in which it should exist during life.

"With regard to the fat of the integument—the principal deposit of

adipose tissue in the body—it appears to me self-evident that the fluidity of this fat must vary with the temperature of the atmosphere in which the animal is placed ; to what extent this is the case, is, in my opinion, a most important subject for enquiry, and although the experiments to determine the question are yet deficient, I hope soon to be able to supply them. . . .

"What I now suggest, as a general proposition, is this:—That in all probability, *the stability of the fats of the animal body in resisting too rapid oxidation is dependent upon the degree of solidity which they possess at the temperature of the living animal at any given time;* that alterations in external temperature may affect the solidity of the adipose tissue of the integument, and consequently its power of resisting oxidation; and that, therefore, in all probability, it is of great importance that *the food of an animal shall contain a certain proportion of material capable of supplying the adipose tissue with solid fat.* . . .

"I wish, however, to submit the following questions to chemists, as requiring to be settled by their experiments :—

"1. What is the relative facility for oxidation of the solid and liquid fats at similar and at different temperatures ?

"2. Is the facility for oxidation inversely as the melting points ?

"3. Is it the same for all fats at their melting points ?

"4. After the melting point of a fat has been attained, is the facility for oxidation affected by further increments of temperature ?

"5. Is there a temperature at which fats cease to be oxidisable, and if so, what relation does this bear to the melting point in each instance ?"

In order fully to appreciate the facts already stated,—viz., the impossibility of life continuing after a certain proportion of body-weight is lost; the circumstances under which such loss may take place in addition to the simple deprivation of food; the means by which it may be restored; the difficulties in the way of carrying out these means with permanent success; and so forth;—the following facts must all be carefully considered :—

1. A supply of fat, *per se*, to the blood is essential for histogenesis and for the protection of the tissues, and is also of importance for general use as a source of heat and mechanical force.

2. The carbo-hydrates and albuminoids may supply heat and mechanical force, but they cannot take the place of fat in histogenesis and protection of tissue.

3. Fats may be supplied by absorption into the portal system, by absorption into the general lymphatic system, and by absorption into the lacteal system. But *the latter is the means by which the principal supply of solid fat is carried into the blood,* and is the most important.

4. The mean consumption of oxygen by an adult man of average stature (weight 150 lbs.) taking ordinary exercise, is about 30 oz. in

twenty-four hours, and the heat evolved by each 1 oz. of oxygen, in combining with carbon, hydrogen, etc., is about 340 British units. Hence 10,000 British units of heat will be evolved every twenty-four hours by the combination of 30 oz. of oxygen with carbon, hydrogen, etc.; therefore the food of an adult man under ordinary circumstances, should be such as may, in addition to other purposes, evolve at least 10,000 British units of heat.

5. Practical experience in the dieting of large numbers of men, and other means, have enabled us to establish the fact, that such an average man as I have spoken of requires, for the maintenance of health, a diet which shall contain about 4 oz. of plastic materials, 3 oz. of fat, and 10 oz. of carbo-hydrates ; and, on careful analysis of this diet, we find that it can supply the required 10,000 British units of heat, viz., 2,516 from the plastic, 3,357 from the fat, and 4,150 from the carbo-hydrates ; total, 10,023.

6. Assuming these statements to be approximately correct,* the point which especially concerns our present subject, and which is of extreme interest and importance, is the mode in which these 10,000 British units of heat are disposed of, and the purposes which they serve. This may be seen in the following calculation which has been made as nearly correct as possible. 8,000 British units are required as sensible heat, to raise the temperature of the inspired air to the temperature of the body, to vaporise the pulmonary halitus, and to maintain animal heat. The mechanical equivalent of 2,000 British units (equal to 690 foot-tons) is expended in actual work, more than half of which is employed in internal vital work (the mechanical work of the heart, of respiration, and other vital movements), leaving about 290 foot-tons available for external work, which may be represented by the labour of walking sixteen miles ; but of course only so much is available for actual walking as is not used in those other external movements of the body which we daily perform.

7. From these calculations it is clear that the demand for 8,000 British units of heat—to raise the temperature of the inspired air, to vaporise the pulmonary halitus, and to maintain animal heat—may be diminished by so much as we can maintain these increments of temperature by artificial means. For example, if, by the combustion of carbon in the form of wood or coals, we can keep up the external temperature of the body, and warm the food and drink which enter the stomach, and if, by any means, we can raise the temperature of the inspired air to that of the body; we shall save the combustion of carbon, etc., within the body by the combustion of carbon, etc., out of the body, and to that extent diminish the demand for a supply of carbon,

* Although some of the details may be subject to alteration with the advance of science, no material error can exist, as the calculations are based on well observed facts.

etc., to the blood from the food. We must, however, bear in mind that it is the oxygen, brought into the blood by respiration, which makes the demand for carbon and hydrogen ; we must therefore diminish this supply of oxygen, at the same time that we supply the heat artificially, if we wish to attain our end of reducing the demand for a supply of carbon and hydrogen from the food. This reduction in the supply of oxygen can be made by rarefying (by altitude) and by diluting (with aqueous vapour) the air supplied for respiration, and by reducing the amount of air respired, by means of rest, etc. (See Altitude, Part V.)

8. Of the 690 foot-tons of force available for mechanical work, we have seen that 400 are employed in the internal vital work of the body; very little can be done therefore to reduce the amount of this demand. Even here, however, *something can be done* by tranquillising respiration and the action of the heart as much as possible. But we still have left the 290 foot-tons of force expended during ordinary health and under ordinary conditions of life upon the external work of the body. It is over this that we have the greatest control. We have, in fact, the power to save about 18 foot-tons of force by every mile of walking, or its equivalent in other work, of which we can deprive our patient.

9. Supposing, then, for, the sake of example, that we could, by artificial means, reduce the demand for sensible heat from 8,000 British units to 6,000, we should not only save the whole amount of heat the mechanical equivalent of which (400 foot-tons) is required for the internal work of the body, but we should have a balance equivalent to 290 foot-tons. And supposing that we could also stop all the *external* work of the body we should thus gain another 290 foot-tons. So that by the combined influence of rest and artificial heat we should save all the carbon and hydrogen required for producing 580 foot-tons of mechanical force, or about 1,680 British units of heat ; that is, as much as would be evolved by the combination of about 5 oz. of oxygen, or one-sixth of the whole quantity required in twenty-four hours under ordinary circumstances.

10. If then we have a patient suffering from a defect by which his supply of carbon and hydrogen is cut off from the blood at its principal channel, we shall best protect him from destruction by placing him in those conditions of rest, warmth and diminished respiration of oxygen which I have pointed out, until the deficiency in the supply of carbon and hydrogen is artificially provided for, or the defect itself removed. And let me especially call attention, in this place, to the fact that by the rest from mechanical work, whether external or internal, we save the wear and tear of the frame, and thus diminish the demand for that histogenesis for which *fat*, and fat only, will suffice. By these combined means, therefore, we not only reduce to its lowest point the demand for carbon and hydrogen, but we especially reduce the demand for them in the form of fat.

The recent case of the Welsh miners presents a good illustration of some of the foregoing statements:—"An influx of water from an old working of a Welsh coal mine caught nine men and imprisoned them in one of the higher levels of the pit. Outsiders, believing that some of the men were alive, fitted large pumps to the pit, but without avail. Next it was decided to cut through forty yards of solid coal lying betwixt the imprisoned men and an accessible point. This took some days, but the men were finally reached, and all recovered alive. For ten days they had been immured absolutely without food, except a little grease licked off their candle-box. They had some muddy water which they drank occasionally. Some of the men were able to walk away themselves on being liberated. As they were about to go to dinner when caught by the water, they began their fast on an empty stomach. What were the conditions favourable to the retention of so much vigour after so long a fast? (*a*) The men made almost no exertion. (*b*) They were in a pit of high temperature. (*c*) Until the last few hours they were kept dry. (*d*) They were in total darkness after their candles went out. (*e*) The compressed air which they breathed had no deleterious effect on the lungs." ("New York Med. Journal.")

Finally, let me impress, that all the important measures of protection which I have enumerated—are but provisions necessary for the purpose of placing the patient in a position of safety, in which the means for restoration of the healthy functions of the stomach, liver, pancreas, and intestinal glands may be pursued without risk.

With the preceding facts and ideas in our minds, we cannot be the least surprised that a patient rapidly emaciates who is in the second or third stage of consumption. For in such a patient, we find the concurrence of the following causes of waste:—

1. A defect in the supply of fat from the food to the blood.
2. Diminished or capricious appetite, by which the total weight of food ingested per day is diminished.
3. Hurried respiration and pulsation, by which the internal vital work is increased.
4. Accelerated combustion, as indicated by increase of temperature.
5. Discharge of solid products from the body by expectoration.
6. Loss of saline and fatty matters by excess of perspiration.
7. Occasional attacks of diarrhœa.
8. In addition to all these, a destructive disease going on in a vital organ, with pain, loss of rest, and blood contamination from absorption of septic matters.

In a case of loss of flesh from advancing pulmonary consumption, this concatenation of circumstances is "what we have to fight," and "what we have to fight for" is their removal.

Now it is entirely consistent with physiology and pathology that all the conditions enumerated here, from 2 to 8 inclusive, should proceed directly in the natural sequence of cause and effect from No. 1. If by any means whatever, the supply of fat from the food to the blood is stopped, sooner or later the tissues must become disintegrated by oxygen. Of course the commencement of this destructive process will be delayed so long as the fat accumulated in the body can be spared for fuel and for histogenesis. That it will be at once utilised in this way when the supply from the food ceases, and that it can be spared up to a certain point for this purpose, we know from daily observation of man and other animals who temporarily lose fat and regain it without damage. But the elaborate care taken in nature to prevent the possibility of this supply being exhausted teaches us its immense importance. "In some animals in tropical climates the fat is collected in large masses at certain parts, as in the zebra, the yak, the buffalo, and the camel. In the camel the hump is firm and solid when the animal is well fed, but when insufficiently supplied with food it becomes loose and flabby. Whenever the fat assimilated is insufficient for the wants of the economy, the subcutaneous fat becomes absorbed in order to supply the waste of more important tissues. Fatty, starchy, and albuminous articles are all capable of being converted into fat. . . The fats themselves are partially emulsified . . and passing through the lacteals, mesenteric glands, and thoracic duct with the general blood current, increase the amount of fat in the blood." ("Absorption of Fat and its Uses," by Dr. Lauder Brunton, "Lancet," Dec. 15, Report of Medical Society Meeting, Dec. 10, 1877.)

If then, it is correct, as I assert, that all the enumerated causes of waste which we have to fight in a case of consumption, follow necessarily upon No. 1, we ought to imitate the Homeric warriors, and begin by confronting our chief antagonist face to face. But to do battle with him with the zeal essential to victory, we must have no doubt about his deadly powers.

Before collecting the armaments at our command, and considering how best to use them, let us then reconnoitre the camp of our foe.

This foe is oxidation,—the yielding up of the tissues to combustion by oxygen, in consequence of a defect in the supply of fats from the food into the blood. Thus the blood becomes deficiently and defectively supplied with fat elements from the food, is unable to afford those required for direct combustion; does not replace those taken up during interstitial nutrition, but, on the contrary, takes up more to compensate the deficient supply from the food. This having gone on up to a certain point, the fat elements of the albuminoid tissues are seized upon, and those tissues are minutely disintegrated in the process. They are in fact reduced to ashes.

It is evident that this disintegration by oxygen will take place first

wherever the following combination of conditions coexists in the most marked degree:—

1. Greatest activity of interstitial oxidation.
2. Smallest amount of fat elements able to be spared by the tissues.
3. Oxidation of blood deficient in materials for oxidation, through the medium of tissues also deficient in such materials.

I need hardly point out that under ordinary circumstances the lungs will be the organs most markedly fulfilling these conditions; and we cannot, therefore, wonder that when the blood flowing through the pulmonary artery for oxidation in the lungs is deprived of the fat-elements sought for by the oxygen, those elements will be seized from the materials of the vascular and alveolar walls which constitute the medium of communication between the oxygen and the blood; and if the tissues of these walls have been already nourished by blood deficient in fat, they will the more readily be reduced to ashes by the oxygen.

There is no other part of the body traversed by fluids essentially the same as those which traverse the lacteals, mesenteric glands, thoracic duct, and pulmonary artery; and there are no other parts of the body in which the relations between pancreatised fats, albuminoid tissues, and oxygen, are so similar as in the small intestine and mesenteric system and in the lungs. In the lungs the oxygen is in the air, and the material to be oxidised in the capillaries of the alveoli. In the small intestine and mesenteric system, the oxygen is in the capillaries of the glands, and the material to be oxidised traverses the glands. Hence we find that this disintegration by oxygen—which occurs first in the lungs of both children and adults—is next most frequent in the small intestine and mesenteric system; except when it falls upon the bronchial glands of children—an exception susceptible of an explanation consistent with the foregoing statements, as I have shown in the second edition of my work on Tuberculosis, p. 22. (See Part II., pp. 101–3, Initial Hæmoptysis in Tuberculosis.)

It will have been observed that I have already (Part I., p. 26, Part II., p. 103,) especially referred to this disintegration of albuminoid tissue by oxygen. But it is in the present place that we have to view it in its all-important relations to loss of flesh on the one hand and to pulmonary tuberculisation on the other—as occupying the medium ground between the two—the point at which one passes into the other.

Having already treated this subject in some detail in my two works, "The True First Stage of Consumption," 1867 ; and "The Nature, Cause, and Treatment of Tuberculosis," 2nd edition, 1866, I should hardly have thought it necessary to enlarge upon it in this place were it not for the following circumstances :—

1. Both the above works are nearly, if not quite out of print, and it is not my intention to reproduce them.

2. They were written with the object, first, of suggesting an explanation of the action of pancreatic emulsion, which I had recently brought before the profession without an explanation, in the pages of the "Lancet;" and second, of explaining the course of ideas which led to my invention.

The attention of the profession was naturally fixed much more on the question of the function of the pancreas in preparing fats for assimilation than upon the far more vital question of the origin of tubercle by oxidation of tissue. It thus most naturally came to pass that what I had merely suggested as a plausible hypothesis explanatory of the deficient supply of fats to the blood—viz., the possible paralysis or perversion of pancreatic function—obscured from notice *the more important pathological and etiological hypothesis which had been the outcome of sixteen years of labour*—viz., the suggestion that tuberculosis was oxidation of albuminoid tissue, the result of deficiency of fat-elements in the blood and tissues:—an hypothesis which I wish to be kept quite distinct from—as it is quite independent of—the further suggestion, that this deficiency may be caused by defect of pancreatic function.

3. For many years before I ventured to publish my hypothesis, the subject of tubercle and consumption had been almost dead in the professional mind. The genius of Laennec and Louis, and their remarkable works had overshadowed the medical and scientific world; and —as it has ever been in those "piping days of peace" which follow upon long and troublous wars—no one seemed to dare to disturb the calm.

When I entered upon the practice of my profession, in 1849, and for many years afterwards, the man who ventured to whisper—as Niemeyer and others have of late so loudly cried—that consumption could be caused by a cold, would have been hissed down as a quack; and even to write upon the subject of consumption was looked upon with suspicion; it being assumed that the science of the subject was so completely settled, that a writer upon it could now have no other object than to produce "a bait for practice."

Now all this is changed. In 1859, the late Dr. Hughes Bennett published his book on Consumption. In the "Lancet" of September, 1864, I published my first Report on "The Treatment of Consumption by Pancreatic Emulsion;" in June, 1865, the Second Report; in November, 1865, the Third Report; in November, 1866, the Fourth Report. In January and February, 1866, I published my papers on the "Nature and Cause of Tuberculosis" in the "British Medical Journal." In 1865 and 1866, my friend and coadjutor, M. Villemin gave to the Academy of Medicine in France, the startling results of his experiments on the inoculation of tubercle, to be followed in 1868, by his extensive work, "Etudes sur la Tuberculose."

In consequence of these and other productions—or at least coincidently with their appearance—the whole profession began to stir on the subject of tuberculosis, and from that time to this the interest and agitation have acquired yearly increasing impetus. Happily, the result has been the addition to our knowledge of a vast mass of observations, many of them of great interest and importance; so that the whole subject is extraordinarily changed in its conditions since I published my hypothetical explanation of tuberculosis in 1865-6.

In the war of discussion to which I have referred, I have purposely remained vigilantly silent. I had plainly stated my views, and it was for others to disprove the correctness of my conclusions, and to demolish my hypothesis if they could. I must confess that I expected nothing less than absolute refutation of all my ideas and conclusions in the light of the great flood of new observations which were pouring in from every part of Europe, from the greatest pathologists of our time. I felt it to be my duty, carefully and candidly to analyse all these observations as they were produced; to compare them with my own; to use them as arguments against myself whenever this was possible; to keep my own ideas and hypotheses, as it were, constantly in a crucible, and to submit them day by day to the fire of other men's work,—holding myself always ready to discard them. Over and over again have I thought I saw them melting away. Especially was this the case while the puzzling inoculation experiments were going on, and when Virchow promulgated his views on adenopathy.

It was in the careful and anxious study of these that, at last, I found what I thought to be the confirmation, instead of the refutation, of the correctness of my hypothesis. It depended almost "on the turning of a straw." In my original statement I had said, "The blood becomes deficiently and defectively supplied with fat elements from the food; is unable to afford those required for direct combustion; does not replace those taken up during interstitial nutrition; but, on the contrary, takes up more, to compensate the deficient supply from the food. This having gone on up to a certain point, the fat elements of the albuminoid tissues are seized upon, and these tissues are minutely disintegrated in the process." (In the 2nd edition I added a note that "the word tissues must be understood to include albuminoid materials employed in the construction and repair of tissues, for it is probable that the fat elements may be waylaid during the process of tissue formation.") Then I added: "*This disintegrated albuminoid tissue is nascent tubercle, and this process is tuberculisation.*"

Now, the whole of the above holds good in the light of modern research, except the giving the name tubercle to the disintegrated albuminoid material. I had guarded myself by the use of the word *nascent*, but I should rather have said "this disintegrated albuminoid tissue causes tubercle."

This, it must be observed, is no change of front; but it is an important change of form, which enables the whole of my hypothesis to assimilate with the results of modern pathological research, as I shall endeavour to show further on.

These debris of the combustion of tissues set up adenoid hyperplasia, angio-plastic, epithelial, and other cell proliferation—and all that follows in their track. In this relationship they fall into the ranks with other causes of similar changes; and thus, I venture to think, is the great riddle solved—only hypothetically of course—but hypothetically solved as it has not been solved by any other hypothesis or suggestion yet put forth—solved as neither Virchow's nor Villemin's pathology, nor any other that I know of, has the least claim to having solved it.

And why? Because no hypothesis but mine *attempts to explain how loss of flesh precedes all other changes.* I am astonished that so palpable a clinical fact, as the loss of flesh premonitory of tuberculisation, should have been entirely lost sight of by all modern pathologists. All their ideas seem framed upon the basic clinical fallacy that tubercle precedes constitutional decline instead of following it. Thus, to take the most recent illustration, Dr. Shepherd sums up his "Goulstonian Lectures" (1877) by saying, that "the commonest form of pulmonary consumption in this country is due, in the first instance, to an intra-alveolar and intra-bronchial catarrh, accompanied by proliferation of the epithelium more or less rapid, followed generally by impermeability and disappearance of the alveolar capillaries, and by changes in the septa of the alveoli, and resulting in the destruction of lung-tissue, the *whole process giving rise to the clinical symptoms of phthisis.*"

It is evident that nothing stated in the above quotation can give rise to loss of weight antecedent to catarrhal or other lung symptoms; and, therefore, the typical symptom of consumption is entirely ignored in Dr. Shepherd's view, which is one taken exclusively from the dead-house, instead of from observation of the living patient.

My hypothesis, therefore, based, as it originally was on clinical data, framed to explain these data, and entirely consistent as it is with these data, must take precedence of all hypotheses in which they are disregarded.

But while such hypotheses are thus deposed from any claim to explain *the cause* of consumption, they fall into their places as learned and lucid explanations of changes *subsequent to the onset* of disease.

I am not forgetful of the promise, made in the early pages of this book, that I would not enter into abstruse questions in pathology further than was necessary to give point and force to practical conclusions. Therefore, in order to make the following statements as concise and

forcible as possible, and at the same time to give them the necessary point, I have selected abstracts of the most important observations, theories, and ideas, of recent years, on the subject of tubercle, and I have interpolated within brackets my own comments on the text. These are not made in any spirit of dogmatism, but simply to lead the mind of the reader into my own course of ideas at apposite places for comparison and criticism, and in this way to enable him to judge how far my own researches and hypotheses assimilate or otherwise with the researches and hypotheses of others.

First then to state the question.—Explaining my conclusions as to the position of hæmoptysis in the natural history of consumption (Part I., p. 25), I have stated that, as a cause of lung disease and of constitutional decline, I am disposed to place hæmoptysis as only one item (*d*), *and that a very exceptional one*, in a large and important group, embracing all foreign substances which find their way into the peri-vascular and peri-alveolar tissue of the lungs, and by their irritation there set up lymphatic (adenoid) hyperplasia and cell prolifieration with their consequences.* Of this important group, the following are some of the principal constituents:—

a. Lamp-black, coal, steel, stone, flint, and other substances inhaled by workers in various trades.

* Those who have not devoted much study to the mechanism of this large and important group may derive some assistance in understanding it from the following abstract of recent investigations on the subject:—"A question in pulmonary pathology of considerable scientific interest and direct practical bearing is raised by some observations published by Professor Nothnagel in Virchow's 'Archiv.' They relate to the passage of cells from the alveoli of the lung into the interstitial tissue. When extensive hæmorrhage occurs into the trachea of a rabbit, such as takes place when the trachea and carotids are divided simultaneously, the lungs are found gorged with blood. The bronchial tubes are full, and the alveolar tissue is of a dark brown-red colour. The blood is not equally distributed; there is always most in the vicinity of the hilus. On microscopical examination of the hardened lung, it was found that the blood was mainly in the bronchial tubes and air-cells, which were most of them stuffed with corpuscles, a few being free. But there was also a large amount of blood in the interstitial tissue of the lung, between the alveoli, and even in the interlobular septa. In places this accumulation was so dense as to mask the normal structure of the lung. Although some of the interalveolar septa contained no corpuscles, the nodal points of the septa invariably presented accumulations. In these spots the connective-tissue framework is normally more highly developed. The appearance of the lung suggested an interstitial hæmorrhage, with rupture, and escape of the blood into the air-cells. Indeed, so considerable was the interstitial accumulation that it seemed possible that the process had actually been of that character—that the convulsions which preceded death might have led to interstitial hæmorrhage. Some observations were therefore made on rabbits killed by dividing the carotids only, the trachea being left intact. No interstitial accumulation could, however, be discovered, and, of course, there was no blood in the air-cells. Hence it seemed clear that the blood had passed into the interstitial tissue from within the alveoli.

"A remarkable feature of the phenomenon was the rapidity with which the passage occurred. The anæmic convulsions came on in three-quarters of a minute after the

b. The products of inflammatory destruction of tissues.

c. The products of catarrhal affections especially in scrofulosis.

d. The debris of tissues disintegrated by the extravasation of blood, and possibly the debris of the blood so extravasated.

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severance of the carotids, and ceased in a minute and a-half. Immediately afterwards the thorax was opened, and the lungs removed and thrown into absolute alcohol. The whole period occupied was not more than five minutes. Whatever be the mechanism, all movement of cells or fluids must cease as soon as the alcohol acts upon the tissues.

"That particles passed with readiness from the air-cells into the interstitial tissue had been previously shown by Ins in his experiments to ascertain the mechanism by which grinders' phthisis is produced. He found that cinnabar dust, introduced into the air-passages, could be discovered within the nodal points of the interalveolar septa in from six to twelve hours after its introduction, and that some granules had passed into the bronchial glands.

"The path which the blood-corpuscles traverse in their migration could not be discovered. But we know that channels exist capable of affording ready ingress for solid particles from the air-cells into the interstitial tissue. Wymodzoff, Klein, and others have shown that the commencement of the lymphatic channels are in immediate communication with the alveolar and bronchial spaces. Nothnagel's observations show that these openings must be extremely numerous to permit the rapid passage of so many corpuscles into the interstitial tissue. He suggests that it is possible that the dilatation of the air-cells by the blood may dilate the stomata which exist in their walls; since in every case the chief interstitial accumulation took place where the air-cells were the most distended with blood.

"The immediate application of these observations to the elucidation of many processes of disease will be sufficiently obvious. The light similar facts throw on the occurrence of interstitial lung changes, as the result of inhalation of fine particles of stone, &c., was pointed out by Ins. Another important consideration, which flows more immediately from Nothnagel's observations, is the question of the absorption of the exudation in croupous pneumonia. It is probable that in the process of the resolution of pneumonia, only a small part of the exuded material which fills the air-cells is expectorated. A greater quantity must be absorbed. The common assumption is that the contents of the alveoli, white and red corpuscles, and fibrin, undergo fatty degeneration, and that the products of this degeneration are absorbed. Nothnagel suggests that the cells may find their way back into the lung-vessels without undergoing degeneration. He admits, however, that the interference with the circulation in the inflamed lung may constitute a difficulty in the acceptance of this view, and we think that the facts of the post-mortem room are opposed to it. All pathological evidence goes to show that destruction by degeneration of the exudation precedes its removal from the air-cells, by whatever process that is effected. Cordua, in his investigation into the method by which extravasated blood was removed from the tissues, was able to demonstrate its return into the vessels only so long as it remained in a fluid state.

"The destination of blood effused into the air-cells is a very important element in the pathology of the origin of phthisis from hæmoptysis. It must be confessed, however, that these observations do not increase our knowledge of the pathological processes which underlie such a condition; indeed they diminish rather than increase its probability. Nothnagel, in some early but unpublished observations, had failed entirely to produce lung changes by injecting blood into the air-passages of the lungs of the rabbit. No caseous pneumonia was found either days or weeks after the injection; neither was there any blood in the minute bronchial ramifications, in spite

e. Albuminoid tissue disintegrated by oxidation in true tuberculosis.

It must be observed that this item (*e*) is the only one in the list which *involves that initial loss of weight* characteristic of the true first stage of consumption.

In my opinion, this disintegrated albuminoid tissue is the irritant which, in true tuberculosis, sets up the hyperplasia of adenoid tissue, the cell proliferation, angioplasia, and their results, so well described by Portal, Virchow, Sanderson, Rindfleisch, Charcot, Malassez, and others. But whereas they—ignoring the clinical fact of initial loss of weight—place these processes first among the pathological changes of tuberculosis, I, on the other hand, *believing it to be essential to take cognisance of the initial loss of weight*, give precedence to the disintegration of albuminoid tissue by oxidation, of which the hyperplastic, proliferative, angioplastic and other changes are but the effects.

According to my view, therefore, the order of events is as follows:—

1. Deficiency of fat in the blood (the deficient supply of fat from the food is necessarily first felt in the blood of the pulmonary artery presented for aëration in the lungs.)

2. Oxidation and disintegration of albuminoid tissues, with the consequent production of molecular debris.

3. Hyperplasia of adenoid (lymphatic) tissue, the effects of the irritation caused by the molecular debris of the disintegrated albuminoid tissue.

According to Professor Sanderson ("Edinburgh Medical Journal," Nov., 1869), "The tubercle produced artificially is in a certain sense hyperplastic, that is, an overgrowth, not a new growth, and thus parts apt to be affected with tubercle are those in which the structure in question exists naturally. . . . The masses of new growth in the lung are

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of the fact that rabbits neither cough nor expectorate. These observations entirely agree with the negative results of Perl and Lipmann. The experiments of Sommerbrodt were less conclusive. He believed that under some conditions hæmorrhage into the lungs would cause a catarrhal pneumonia and phthisis. All, however, agreed that the contents of the bronchi and air-cells were very small in quantity compared with the amount of blood they must have contained, and the explanation of this is completely afforded by the observations of Nothnagel. Admitting the fact that hæmoptysis may, in man, sometimes lead to phthisis, these observations—if we may transfer them to the human lung—show very clearly why such a consequence so seldom follows the hæmorrhagic infiltration into the air-cells which must very often take place."—("Lancet," Jan. 26, 1878.)

[See a very interesting case of "Miners' Lung," in which the clinical, postmortem and microscopical phenomena are most carefully given by my friend Dr. Osler, of Montreal. ("Dr. Dobell's Reports on Diseases of the Chest," 1876.) See also the introductory chapter "On the Perivascular System," in the third edition of my work "On Winter Cough"; Dr. Noble Smith's beautiful plates of Anthracosis, in Dr. Shepherd's "Goulstonian Lectures," 1877; and Dr. Klein's "Anatomy of the Lymphatic System."]

overgrowths of masses infinitely smaller, which (as proved by Professor Sanderson) existed before, scattered through the lungs, . . . viz., adenoid tissue, which only requires to be increased in size to be indistinguishable from miliary tubercle."

If we add to this adenoid hyperplasia, epithelial and connective tissue cell proliferation, angioplasia and other attempts at repair of disintegrated tissue, and, also, the results of degradation and decomposition of these new formations, we shall see that *we make up a foreign mass* answering to all the requirements of the antagonistic pathologists from whom I am about to quote.

It is of little consequence, in my opinion, whether we apply the term "Tubercle" to this heterogeneous mass of materials, or to the molecular debris of disintegrated albuminoid tissue from which it springs, so that we clearly appreciate and keep in mind which is the cause and which the effect, *and strictly adhere to the clinical order of events.*

As I have already pointed out (page 164), in my previous works I called the disintegrated albuminoid tissue "nascent tubercle," as being the essential initial formation in tuberculosis; but, as the ordinary meaning of the word "tubercle" is "a little tuber or kernel," I think it is better to apply the term to the heterogeneous adenoid and cell mass I have just described, which is much more like "a tuber or kernel" than the molecular debris of tissue alone. But it must be emphatically observed that if we thus apply the term, it will equally belong to similar "tubers" of hyperplastic adenoid tissue, proliferated and angio-plastic cells, etc., *by whatever cause produced.* And thus we introduce a very wide generalisation, requiring careful classification of its elements. The tubercle resulting from oxidation and disintegration of albuminoid material, and preceded by initial loss of weight (*e*), must stand first in such a classification—as representative of True Consumption of constitutional origin; while tubercle resulting from other causes, such as those which I have enumerated under *a*, *b*, *c*, *d*, must follow.

Now, in the light of the foregoing statement of the question let us read the following quotations and comments:—

In 1871, Dr. Walshe attempted the following *reductio ad absurdum* of the state of opinion at that time:—

"It will probably be conceded by impartial lookers-on that the attempt to fix the microscopical nature of tubercle has reached but an early stage of advancement. How short a time has passed since the *specific tubercle-cell* figured as the cherished cardinal fact in the micrology of the disease! While now the doctrine of the hour not only stigmatises the distinctive cell as a pure myth, but invites us to adopt articles of faith antagonistic to all specificness. Thus according

to Virchow 'the cells present in tubercle may be relics of epithelial cells, *or* of some other natural product, or they may be related to, *or* identical with exudation cells, *or* pus-globules, *or* to and with the cells occurring in cancerous growths, *or* in typhoid infiltrations.' ('Rankin's Abstract,' vol. xviii.), and from the observations of Dr. Burdon Sanderson (loc. cit.) it would follow 'there are no elements in the induration of phthisis, which are not to be found in chronic bronchitis,' nay more, 'it is even possible there are no anatomical elements in the induration of phthisis which are not to be found in the healthy lung.'

"Now practically, to what issue do the latter propositions point? Presumably, not to the equations 'tubercle=lung substance' and 'bronchitis=phthisis;' not a whit more to the paradox 'hypertrophy of the lung and tubercle mean one and the same thing.' But inasmuch as they teach microscopical identity, 'actual' or 'possible,' between objects so utterly dissimilar to unaided sense, there seems to be no escape from one or other term of the following dilemma. Either this corpuscular identity is *the* dominant and essential pathological fact, and men must be wrong in considering phthisis and bronchitis different diseases, or the seeming identity that captivates the micrologist of to-day, is a quality of mere secondary and quasi-accidental importance, and the arrangements, the site and mode of accumulation, the chemical conditions, vital tendencies, &c., of these elements, really give them their specific characters, and their distinctive potentiality." ("On Disease of the Lungs," 4th edition, p. 413.)

Writing "On the Pathology of Pulmonary Phthisis," Dr. T. H. Green says:—"The microscopical examination of the *yellow* nodules shows that most of them differ somewhat in their constitution from the grey. The latter may, as has been stated, undergo retrogressive changes and become caseous, so that some of the smaller yellow growths may be simply the grey which have degenerated. The majority of them, however, do not originate in this way."—[Assumed, but not proved.]—"When examined with a low magnifying power, it is seen that they are not like the grey nodules, simply inter-alveolar growths involving merely the alveolar walls, but that their more external portions, at all events, are made up of *accumulations within the alveolar cavities.*"*—[No evidence that this is not a secondary change due to irritation.]—"The outline of the nodules is much less defined than that of the grey, and the alveoli with their contents, which are quite distinct in the peripheral portions of the growth, gradually become lost as one passes towards the centre; whilst in the central parts there is no indication of structure such as is seen even with a low power in the grey nodule"—[the effect of time].—"When examined with a somewhat higher magnifying power, the constitution of the nodule at once becomes apparent. Its central portions consist for

* The italics in the quotations are frequently mine.

the most part of a structureless granular *débris*"—[*débris* of albuminoid tissue disintegrated by oxidation]—"in which, perhaps, here and there, traces of formed elements are visible. More externally are seen indications of alveoli which are filled with partially degenerated epithelial-like cells, with, in some parts, small tracts of a small-celled tissue resembling that met with in the grey nodules; whilst in the peripheral portions of the growth, the alveoli are quite distinct, *their walls are infiltrated* with a small-celled growth,"—[probably the first change which led, by irritation, to cell proliferation within the alveoli,]—"and their cavities are filled with large nucleated cell-forms, similar to those which are always to be found in the alveolar cavities of the healthy lung. In short, it is clear that in most of the yellow nodules we are dealing with a structure which more closely resembles a lobular catarrhal pneumonia than that met with in the grey growths; and that although the *walls of the alveoli are thickened by a small-celled tissue* similar to that which constitutes the *grey growths*, and small tracts of the same tissue may be found in the *central portions of the nodule*, the nodule consists in the main of alveoli stuffed with epithelial-like cells which have a *special* tendency to undergo rapid retrogressive changes.

"Respecting the pathology of acute miliary tuberculosis, I presume it to be an infective disease resulting from the dissemination of *infective substances* (*probably minute particles*) *from some primary inflammatory lesion*"—[or from combustion by oxidation].—"The results obtained from the artificial production of tubercle in the lower animals by Villemin, Wilson Fox, Sanderson, Cohnheim, and others, the *general dissemination of the secondary lesions*, together with the clinical characters of the disease, appear to render this view tolerably conclusive, and I shall not therefore discuss further this part of the subject. What I am particularly desirous of bringing somewhat prominently under notice are certain differences which we have seen to exist between many of the grey and yellow nodules. These nodules we must admit to be *inflammatory growths* resulting from the irritation of substances derived *from the infective centre, and distributed by means of the blood vessels or lymphatics*. Their dissemination is characteristic of their infective origin. Dr. Sanderson, to whom we are indebted for so much of our present knowledge on this subject, terms the nodules secondary or infective inflammatory growths, inasmuch as *the focus of infection is itself the product of inflammation*."—[This is begging the question, no cause for inflammation being shown.]

"What, then, is the relation between the grey and yellow nodules? The latter *are certainly not* in all cases simply an advanced stage of the former,"—[see plate of Dr. Moxon's case]—"grey nodules which have become caseous. On the contrary, although the grey nodules may, as we have seen, undergo retrogressive changes, so that *many of the*

smaller yellow growths may be simply degenerated grey ones"—[why not all?]—"*the majority of the yellow differ anatomically* from the grey. Whereas the grey nodules consist of a small-celled tissue often known as 'adenoid,' the greater number of the yellow growths are largely constituted of *nucleated epithelial-like cells crowded together within the pulmonary alveoli.* This *association with some of the miliary nodules* of more or less accumulation of epithelium within the alveolar cavities has long been recognised, but *the epithelial accumulation has generally been regarded as a secondary part of the process*—as being either a merely mechanical result of some obliteration of the terminal bronchioles, or as resulting from an irritating influence exercised by the primary nodule upon the adjacent pulmonary tissue"—[rather of the irritative cause of the nodule].—"It is any such explanation of the occurrence of the epithelial accumulation which so frequently constitutes a part of the miliary lesions in the lungs in acute tuberculosis that I *now venture to question.* I believe the accumulation of epithelium within the alveolar cavities to be *the result of a catarrhal process* ; a process, however, which is not secondary to any primary adenoid nodule, but one which is the *direct result of the irritant*"—[Yes]—"derived from the focus of infection. My grounds for this conclusion are mainly two. Firstly, in the larger nodules, in which the epithelial accumulation in question is most frequently met with, *it is not common* to find any distinct central growth of small-celled (adenoid) tissue which might be regarded as the cause of the peripheral epithelial growth. *It is true that in many of the nodules small tracts of small-celled tissue are met with, and the alveolar walls themselves are thickened by it;* but this is so intimately mingled with the accumulation of epithelium within the alveolar cavities that *I cannot help* regarding them both as the direct result of one common cause."—[This can hardly be called argument.]—"Again, it is exceedingly common to find the small-celled growth *almost entirely absent*"—[if not entirely absent this is no argument],—"the nodule consisting merely"—[*almost*]—"of accumulated epithelium. Secondly, in a large number of cases of acute tuberculosis the nodules *consist simply of the small-celled tissue,* any accumulation within the alveoli being entirely absent. It cannot be argued in these cases that the disease has run a rapid course, and that death has ensued before the nodules had had time to cause any epithelial proliferation in the adjacent alveoli, because the fibrillation of many of the nodules is so marked that their development must evidently have extended over a somewhat lengthened period. These considerations appear to me to be *tolerably conclusive*"—[not to me in the sense intended],—"and I submit, therefore, that the small-celled growth and epithelial accumulation do not stand to one another in the relation of cause to effect, but that *both are the direct result of one common cause—the irritant derived from the focus of infection.*"—[There is no reason why the

irritant should not be one or other in different cases. Probably much depends upon the accidental more or less implication of the epithelial (mucous membrane) element in the irritation. This will naturally occur in catarrhal diatheses, and under catarrhal excitement.]

"Granting that both the small-celled and the epithelial growth are the direct result of the irritant derived from the focus of infection, it may be asked how it is that this irritant produces in some cases the one form of lesion, and in others the other. To this I reply that *the nature of the lesion depends upon the severity of the irritant.* The *more* severe the irritant, the greater is its tendency to produce epithelial proliferation; the *less* its severity, the more does its influence tend to be limited to the elements immediately adjacent to the blood-vessels and lymphatics."—[Is it not rather the effect of constitutional and like causes than of less or more?]—"*The firm grey nodule, which consists* of small-celled tissue, and which is in the *main an inter-alveolar growth,* I believe to be the result of an irritant of less severity than that which gives rise to the majority of the yellow nodules,"—[why should not the grey nodule be an irritant of such severity as to produce the yellow epithelial nodule?]—"*i.e.*, those in which an accumulation of epithelium within the alveolar cavities constitutes so large a portion of the growth. Further, inasmuch as the greater the severity of the irritant the greater is the injury sustained by the tissue, and the *less is the tendency of the newly-formed elements to undergo further development,* we find that those nodules which consist largely of epithelial elements, and therefore presumably result from an irritant of considerable severity, *tend to undergo retrogressive changes and to become* caseous ; whereas the nodules which consist entirely of small-celled tissue, being the result of an irritant of less severity, have a *much greater tendency to become fibrous.*"—[Hypertrophied adenoid tissue is of course less subject to decay than dead epithelium.]—"The differences which we have seen to exist in the intervening pulmonary tissue in those cases of acute tuberculosis in which only the firm grey nodules are met with, and in those in which the majority of the growths are of the yellow variety, appear to me to support this view. In the former, the influence of the irritating substances being limited mainly *to the inter-alveolar elements, it is exceedingly common to find the tissue between the miliary nodules perfectly normal ;* in the latter, on the other hand, in which the irritant is more severe, and exercises its influence over a wider area, the intervening tissue is almost invariably either much congested or consolidated. In these latter cases, indeed, extensive tracts of consolidation are sometimes found associated with the miliary lesions—consolidation which, although *mainly consisting in a catarrhal pneumonia,* is occasionally partially the result of an exudation of *liquor sanguinis and leucocytes into the* pulmonary tissue (croupous pneumonia). Such *diffuse* lesions are much more common in the artificially-

induced tuberculosis in the lower animals than they are in man."—[In these cases inflammatory products are set up or introduced.]

"If the above view of the cause of the differences in the anatomical characters of the pulmonary lesions in acute tuberculosis be correct, it tends, I think, very materially to increase *the analogy, which is already by many admitted to exist, between these lesions and those which result from other inflammatory processes.* If we study the process of inflammation as it occurs in the several organs and tissues, we must admit that the severity of the injury upon which the inflammatory process depends, and the duration of its action, determine to a great extent the *nature of the resulting anatomical changes.* We know that in many inflammations, both primary and infective, the injury sustained by the blood-vessels is so great as to give rise to an abundant exudation of liquor sanguinis and emigration of *leucocytes, the latter often being so numerous as to form an abscess.*"—[These are accidental complications.]—"This occurs in pyæmia. In those inflammations, however, in which the injury sustained is less severe, and at the same time for the most part more prolonged in its action, the exudation from the vessels constitutes the least prominent part of the process, the principal change consisting in *an increase in the nutritive activity of* the elements of the inflamed tissue —an increase which usually leads to the production of *new elements*, which either become the seat of retrogressive changes or undergo progressive development"—[according to their nature and site].—"Our knowledge of these less severe and more chronic forms of inflammation is at present somewhat incomplete; but I think it is sufficient to justify the conclusion that the *less intense the irritant, and the more prolonged its action, the more does its influence tend to be limited to the elements immediately adjacent to the blood-vessels and lymphatics; whereas it is only in inflammation of somewhat greater severity*"—[irritation is not always inflammation]—"*that the more distant elements become involved.* We see this, for example, in inflammatory processes in the kidney and in mucous membranes. In the former, the less severe and more chronic inflammations"—[irritations]—"are characterised anatomically by the development of a *small-celled tissue around* the inter-tubular vessels—a tissue which *tends to become fibrous;* whilst in the more *acute forms* of inflammation, the earlier stages, at all events, are attended either by *swelling or proliferation* of the tubal epithelium. The same is true of mucous membranes. Here, also, we have both epithelial proliferation and small-celled growth in the submucous tissue, *the latter being an invariable consequence of prolonged inflammatory action.* Thus this association of epithelial proliferation and small-celled growth *occurs both in primary inflammations and in the disseminated infective inflammations of acute tuberculosis;* and in both I believe"—[no proof]—"the epithelial growth to result from an irritant of greater severity than that which gives rise to the small-celled development. Lastly, it

must be borne in mind that, whereas the small-celled growth tends to become fibrous—although this tendency is undoubtedly less marked in the lungs than in many other organs—the epithelial elements, being incapable of organisation, tend to undergo *retrogressive changes*"—[of course].—"Having thus considered the anatomical characters and pathology of acute tuberculosis, we are in a position to endeavour to answer the question, *What constitutes tubercle?* In the first place, then, I think it is evident it must be *impossible to frame a definition of tubercle upon a purely anatomical basis.* The miliary lesions in the lungs resulting from the infective process known as tuberculosis present different anatomical characters, some consisting simply of a small-celled tissue, whilst others are constituted largely of epithelium. A similar anatomical difference is observable in the miliary nodules in other situations. In the omentum the nodules consist of a *mass of small cells covered by endothelial* elements. In the kidney and spleen the nodules are constituted solely of the small cells. It would thus appear that the precise anatomical constitution of the nodules varies somewhat with the tissue in which they originate"—[this must be so if tubercle is not a specific growth, but an effect of an irritant upon surrounding parts, the elements of which are involved].—"It would, I think, on many accounts be advantageous to get rid of the term 'tubercle' altogether, as it implies no definite anatomical characters."—[There is no reason why it should. If it identifies a concrete idea it is sufficient.]—"At the same time it must be admitted that it has a certain practical value as indicating the anatomical result of a definite infective process. *By tubercle, then, I understand a miliary nodule resulting from an infective chronic inflammatory process; a nodule which, although it does not possess any absolutely definite anatomical characters—inasmuch as it is the product of a more or less chronic inflammation*"—[irritation]—"*usually consists largely of small lymph-like cells which are associated with a varying amount of fibrillated tissue.* Such a definition is, I must admit, by no means satisfactory, as it implies a knowledge of the process by which the nodule was produced; but if we are to continue the use of the term 'tubercle' at all, I think we must do so in this somewhat indefinite sense. The *small-celled tissue, which is the most typical constituent of the miliary nodules, is, I believe, by no means characteristic of tubercle, but is the invariable result of all chronic inflammatory processes, whether these be limited to minute circumscribed areas, as in tuberculosis*"—[adenoid tissue being already present, I suppose]—"or whether they involve wide tracts of tissue, as in the chronic indurations of the liver, kidney, and mucous membranes. The microscopical characters of the tubercular grey granulations, and of the new tissue met with in the early stage of those chronic inflammatory *processes are so similar that in most cases* it is impossible to distinguish between them. This was pointed out long ago by Dr. Bastian. The fact that the miliary lesions in tuberculosis originate from *elements*

which have been shown in many organs to have a close relation to the lymphatic system, will, I suspect, prove not to be peculiar to this disease; but as our knowledge of other chronic inflammatory processes becomes more complete, it will be found that in this respect they all resemble one another.

"I have entered thus somewhat fully into the subject of acute tuberculosis, not with the object of attempting to make any fine anatomical distinctions between 'grey' and 'yellow tubercle,' but with the hope that, by pointing out the close analogy which subsists between this and other inflammatory processes, I might do something towards clearing up some of the difficulty with which the pathology of tubercle is still involved, and with the belief that the proper understanding of this subject is a necessary preliminary to the study of phthisis itself. In order to be perfectly clear, and to avoid any misunderstanding, I will conclude this part of the subject by summing up the conclusions which the facts appear to warrant under the following heads :—

"1. The lesions in acute tuberculosis are inflammatory growths" —[this is a pure assumption ; inflammation has not been proved as a necessary element]—"resulting from the dissemination of infective substances derived from some primary inflammatory product."—[Where is the primary inflammatory product? That is the essence of the whole question.]

"2. Inasmuch as these lesions are the result of infection"—[this has not been proved to be invariable]—"they usually occur as disseminated miliary nodules, although in exceptional cases they may consist of diffuse tracts of consolidation.

"3. Anatomically these lesions in the lungs present certain differences, depending upon the severity of the irritant"—[it has not been proved to depend on the severity of the irritant, but has appeared more likely to depend upon other circumstances]—"by which they are produced.

"4. The less the severity of the irritant, the more does its influence tend to be limited to the elements immediately adjacent to the blood-vessels and lymphatics, from which elements a small celled tissue is produced, similar to that which results from chronic inflammatory processes in other organs"—[rather irritative than inflammatory].

"5. The greater the severity of the irritant, the more liable are the epithelial-like cells contained within the pulmonary alveoli to become involved, in which case the nodule is made up of alveoli stuffed with these epithelial elements, together with a varying amount of small celled tissue.—[See preceding comments.]

"6. The small celled tissue tends to become developed into a fibrillated structure, although it very frequently undergoes retrogressive changes. The latter is especially the case when it is associated with epithelial accumulations within the alveoli. The epithelial-like ele-

ments, on the other hand, being incapable of further development, quickly degenerate.

"7. The lesions in acute tuberculosis are precisely analogous to those which result from other chronic inflammatory"—[irritative]—"processes. They differ merely in their extent and in their mode of origin"—[which mode of origin is not demonstrated]. ("Lancet," Oct. 26, 1872.)

In connection with the subject of "irritative causes," and the "multiplication of diseased foci" in tuberculisation, the following contribution by Dr. Reginald Thompson is of value, as proceeding from one to whom, as he says, "ample opportunities of observation have been given from the Pathological Department of the Hospital for Consumption at Brompton, of which I have the supervision."

"The presence of morbid products in the lungs at a distance from the original infecting source occurs so as to show that the lungs may appropriate, under certain circumstances, material which is thrown off in the course of destructive disease, and ought to be thrown out from the body by expectoration. Such translation of matter from one part of the lung to another, and from one lung to the other, is fraught with hazard, and, in some cases, may hasten materially the fatal termination of the disease. And the term 'translation' is used designedly to indicate that a disease, by transferring its resultant morbid products to another region, may induce lesions of a secondary nature expressed in pathological terms differing from the primary process.

"Disease may spread by simple extension or by propagation. Extension may be illustrated by a bronchitis beginning in the large tubes, which may pass down to the smaller tubes, and eventually, in some cases, involve the alveoli; this is extension by continuity of channel. Propagation of disease is illustrated by pyæmia, where there is multiplication of diseased foci from a primary source of infection. Disease is translated in artificial tuberculosis where the injection of septic matter procures a peculiar kind of irritation. It is very doubtful whether this condition ought to be called a tuberculosis, and, indeed, it has been produced in animals admittedly but little prone to tubercle . . .

"I will endeavour to adduce evidence to show that morbid products, the resultants of pulmonary disease, change their position and emigrate to a distant region of the lung, and to the opposite lung. . . .

"The plainest evidence on the point is derived from cases in which a fluid of a peculiar colour secreted from one lung is found in the opposite lung. Two lungs were examined, of which the left was extensively excavated to within two inches of the lower border by a large trabecular cavity, evidently the result of a low and non-tubercular type of inflammation, freely secreting sanious matter of a dark claret colour. The base was thickened and indurated, and a few minute

groups of tubercle were found here. The right lung was found to contain two old contracted and quiescent cavities, each as large as a walnut, and the surface above them was puckered and drawn in, as usual with old cavities near the surface. These cavities were found half full of the claret-coloured fluid secreted from the left lung, and besides this there were found, some way below these cavities, a broad band of the same colour (due to the same fluid) running across the lower part of the lung an inch above the lower margin; in the middle of which band were a number of isolated lobular or racemose groups of tubercle, comparatively recent and of unusual colour, for they were slightly stained. Evidently, from the position of this band, which was horizontal and transverse, as regards the erect posture which is always assumed in anatomical descriptions, it could not have happened after death. It must have collected there with the help of inspiration and gravitation, and hastened the fatal termination by clogging the useful portion of the lungs.

"Another case may be mentioned which was under observation during life. The physical signs showed that the left lung was firmly and completely adherent and extensively excavated by a large trabecular cavity. There was very little respiration on the left side. Harsh respiration and dull percussion were noted over the right lung. The patient came into the hospital and died about one month after the above examination was made. At the autopsy the left lung was found firmly and universally adherent; extensively excavated by a large non-tubercular cavity, secreting in quantity purple sanious fluid. The lowest portion of this lung, about one-fifth of the whole, was found remarkably sound—I will not say healthy, for it was in a condition of hyperæmia from irritation. Two minute groups of racemose tubercle were found close to the excavated portion; the rest of the lung was quite free from tubercle, the bronchial tubes supplying this portion (which was not active in consequence of the firm adhesions which surrounded it) were transparent and in a healthy condition; the bronchial tubes above being extremely thickened, opaque, and inflamed. The right lung (which had been doing all the work of respiration) was found much enlarged, studded with tubercle, infiltrated throughout with the purple fluid which had found its way from the opposite side and induced ultimate suffocation.

"The evidence from cases of pulmonary hæmorrhage bears strongly upon this point. In examining cases of fatal hæmorrhage arising from the rupture of a pulmonary aneurism (which occurs frequently in phthisis, and is without doubt the most common cause of fatal pulmonary hæmorrhage*), it is often very difficult, if we look to the evidence afforded by inhalation of blood only, to say in which lung the aneurism will be found—so generally are both lungs stained by patches

* See Part II., p. 80.

of inhaled blood. In cases of hæmorrhage into the lungs from aortic aneurism the blood may be found in both lungs. Such a case occurred recently, in which an aortic aneurism was found compressing and nearly occluding the left bronchus. Having examined the man some time before his death, I established the fact that oozing into the left lung was going on. The man died from rupture of the aneurism, and at the autopsy it was found that the whole of the right lung was distended and filled with blood inhaled during the last half-hour of existence. In the left lung hæmorrhage of older date was found, forming a coloured band at the apex and a horizontal band at the base, with a few patches in the middle of the front axillary border of the lung.*

"In cases where matter is transferred from one lung to the other the influences which compel the matter to assume any region are evidently two—the force of gravity and the force of inspiration. Gravitation compels a preference for the most dependent parts, the position of which of course varies with the attitude generally assumed by the patient during the day and during the night.† Inspiration compels a preference for the apex, the base, and the region of the chest where there is greatest action of the ribs—viz., the front axillary border under the fifth and third ribs.

"The next point to which I will refer is *the irritation which appears to result from the translation of morbid matter*. The simplest evidence of this is to be found in cases where an eruption of tubercle immediately surrounds a cavity. In such instances small grey tubercle is found closely studded round the floor of secreting cavities, diminishing in quantity and closeness of position in direct proportion to the distance from the cavity. This condition has been observed and noted by others. Rindfleisch remarks: 'In the immediate neighbourhood of a large cavity occupying the apex of one lung we very often find an enormous number of nodules of uniform and literally miliary dimensions embedded in a parenchyma otherwise but little altered. Should the section be a happy one, we further notice that the nodules are not scattered irregularly through the tissue, but according to a certain definite order—*e.g.*, in branching lines, or in lines radiating from a central point where the nodules are thickly set.' He shows a little hesitation in affirming this to be ordinary tubercle, but he concludes the consideration of the matter in these words: 'We may regard this as a mere local modification of the ordinary development of tubercles.' And, indeed, there seems to be no reason to prevent such a conclusion; it is apparently a secondary irritation of lymphatics.‡

"But there is another peculiar form of tubercle which appears to invade the lung under certain conditions of irritation. I mean that

* See Part II., p. 72. † See Part III., p. 144.
‡ See Part IV., pp. 166–169.

grouped or racemose form of tubercle which affects isolated lobules, and which, from resemblance to a cluster of grapes, is sometimes known by the name of Carswell's grapes. This kind of tubercle is found in peculiar positions, and, if I may be allowed the expression, in peculiar soil—namely, in a highly irritated condition of lung. This irritation does not appear to me to be the result of tubercle, as some suppose. It is not due to the effect of tubercle, because it occurs without the presence of tubercle, and out of all proportion to the tubercle which may be present. I believe it to be a preliminary stage of irritation, which may be developed, by chronicity of irritation, into tubercle under some conditions at present not clearly made out. As this irritation (which is an alveolar catarrh or catarrhal pneumonia) affects lobules, we are allowed to surmise that the irritating matter has been inhaled, and this surmise is strengthened by some experiments on animals, in which catarrhal pneumonia resulted from the inhalation of irritating air. It is in the midst of a region thus irritated that these solitary lobular groups of tubercle are found. They are found sometimes outlying, in a dependent portion of lung, extensive ulceration of the lung, or they may be found in the neighbourhood of a cavity of which the supplying bronchial tube as it passes upwards towards the root of the lung gives off a little branch from its under surface, but above the cavity, and this branch is found to lead to the infected lobule. The matter which has infected this may have passed upwards from the cavity, to be drawn downwards into the lobule by another inspiration.

"Some attention to the preferential regions in which this form of tubercle is found leads me to the conclusion that the matter which thus irritates is influenced by gravity and by inspiration : by gravity because it is found generally at the most dependent parts—viz., below the level of the root of the lung, and also at the posterior parts ; and by inspiration because, in cases where the greatest power of inspiration is called into play in consequence of the extent of disease, this form of tubercle is found just where the lung has the greatest play, and close under the pleura.

"A remarkable instance evidencing the effect of gravitation came under notice a little time ago. Two lungs were examined, of which the left lung was excavated by non-tubercular ulceration. The right lung was sound in the sense that there was no ulceration in it, but it was in a highly irritated condition from apex to base. Scattered through the lung in numbers were found racemose groups of tubercle, but almost entirely below the level of the root, and assuming a general grouping round the main descending bronchus. On a first section of the lung it seemed as if no tubercle at all occupied the upper lobe above the level of the root, but a more careful inspection showed one or two groups at the back, close to the main bronchus.

"But is there any evidence to show that the matter passes down the

air tract? I believe there is. First, there is the fact that the tube supplying the lobule is inflamed, and often opaque; sometimes this irritation is confined to the small tube, but at least it has appeared evident that the bronchial tubes supplying the affected tract are much more inflamed than those which lead to a simply irritated portion of lung. Secondly, there is the evidence that if there is pressure on the bronchial tubes, or adhesions preventing inspiration, those parts affected are not invaded by these groups of tubercle, or are proportionately affected to the amount of inspiration permitted. In evidence of this I will aduce two cases: one which I have already described, where the lower portion of one lung, which was prevented from action by adhesions to the thorax and the diaphragm, was only affected with a few minute groups of tubercle in the near vicinity of a large cavity, the lowest part being quite free, the supplying bronchial tubes being found quite transparent; the other case being one in which the deposit of tubercle was apparently prevented by the presence of a large quantity of fluid between the base of the lung and the diaphragm, which compressed the base of the lung, all the upper portion being infected.

"The opinion that I hold with respect to the nature of tubercle is that it is the result of irritation set up in the lungs or elsewhere by the presence of morbid matter, and I have endeavoured to show, by pathological evidence, that such matter, the result of destructive disease of the lung, may pass down the bronchial tract under the influence of inhalation and gravitation, and set up an irritation that may result in a certain form of tubercle. It must not be supposed that I imagine that all tubercle is formed exactly in the same manner. Although I believe that it is a secondary product, pathological evidence appears to point to the conclusion that there is more than one channel through which irritative matter may be carried to distant parts, resulting also in, but in different forms of, tubercle."*—("Lancet," July 13th, 1878.)

The following "History of the Doctrine of Pulmonary Tubercle," appeared in the "Med. Press and Circular":—

"Dr. Cornil, of Paris, ("Le Progrès Méd.," Nov. 8, 1873), remarks that Laennec gave a complete description, as far as the naked eye could serve, of the lesions and of all, or almost all, the physical signs of pulmonary tuberculosis. He showed how the grey granulations, transparent at first, soon became opaque and yellowish at their centre; he fully assimilated with these the larger masses, which he named miliary tubercles, and which are either agglomerated granulations forming a nodosity larger than usual, or lobules of cheesy pneumonia, and the yellow tubercles, which are nothing more than the advanced stage of the same lesion. Besides these more or less voluminous nodules, Laennec described the gelatiniform tuberculous infiltration and grey

* See Part IV., pp. 166-9 and 204-5.

infiltration, which correspond to our cheesy pneumonia or lobar pneumonia. Then came cavities and other older lesions.

"For Laennec, all these alterations depended on the same process, on the same cause or general disposition of the economy; for him their characteristic was the cheesy condition.

"These ideas of Laennec were forced upon the generations of medical men that followed him, as if they were reasoned out, and it is a fact that there was nothing to change until microscopic studies had introduced a new instrument of analysis and discovered new points of view.* But the opinions of the author of mediate auscultation were so rooted in men's minds that the first observers with the microscope had no other aim but to seek out in the cheesy state the elements characteristic of tubercle.

"Such was the object of Lebert, who, in parts of the lungs which were completely degenerated, always finding fragments of granular elements, small, irregular, and angular, gave these forth as the corpuscles characteristic of tubercle.

"In the year 1850, Reinhardt described the internal modifications of the lung parenchyma, which he referred to pneumonia, and Virchow indicated the characters of the granulation as seen by the microscope. But it is useful to remember this; for long after this, the opinion of Lebert was still so dominant in France, that the first examinations of tubercular granulations by Robin, together with Lorain and Bouchut, made so great a difference of structure from that given by Lebert, that these observers took them for fibro-plastic tissue. This is the explanation why Empis, under the inspiration of Robin, has been able to describe *granuile* as a disease constituted by the presence of granulations, and as being altogether different from the tubercule of Lebert or from tuberculosis. We must, before all, understand well what terms we use; thus, I must forewarn you that, in the language of Empis, the word 'tubercle,' which we consider with the great majority of authors as the synonym of granulation, means cheesy pneumonia. With the exception of this difference in terminology, Empis belongs to that series of authors who consider as two distinct diseases the tubercular granulations and pneumonia.

"The actual head of this school of dualism in anatomical characters and in etiology is Virchow. The Berlin professor has confirmed the labours of Reinhardt in so far as the various appearances of cheesy pneumonia are concerned; and since 1852 he has established the structure of the granulation, and the differential character which distinguishes it from pneumonia. For Virchow the granulation develops itself solely at the expense of the connective tissue, and in the lung it is never situated in the alveoli. Virchow, who had showed that inflammatory lesions, whether simple or specific, such as those found in

* See Part IV., p. 163.

scrofula or in syphilis, or other tumours, may all become cheesy, later has replaced the expression of cheesy pneumonia by that of scrofulous pneumonia. What is the meaning of scrofula when applied to pneumonia? Would that mean perchance that the scrofulous or those who are affected with suppuration of the glands have a predisposition to cheesy or scrofulous pneumonia? If such were the sense of this designation it would not resist criticism, for very often I have seen the autopsy made of such subjects as have died with an acute attack of generalised granulations in all the organs, and especially in the pia mater, and thus granulations show themselves related to scrofula as pneumonia is. I have already remarked that the terms cheesy or tuberculous applied to the pneumonia of the phthisical were not rigorously good in the sense that the cheesy condition is only their termination, and that the word tuberculous, implying the presence of tubercles, was too absolute in the sense that every knot of lobular pneumonia is not always developed around a tubercle. But the word scrofulous is still worse, because phthisical patients with pneumonia are far from always being connected with scrofula, and, on the other hand, the typically scrofulous have often, as the final lesion, generalised tubercular granulations.

"The anatomical dualism established by Virchow, sustained by his pupils in all lands, by his teaching, and by the immense success of his book on cellular pathology, has made its way.

"M. Villemin had fully adopted it in his first work issued in 1861. Later, when Villemin saw tubercle inoculated on rabbits and guinea-pigs by means of caseous pneumonia, as well as by granulations, he could no longer admit an anatomical dualism when experiment showed an indivisible pathology. Thus in his work on tuberculosis, published in 1868, the part of pneumonia and of pulmonary inflammations in phthisis disappears almost completely. And, as on the other hand, M. Villemin no longer recognised the existence of epithelium in the pulmonary vesicles; he classed the large cells with nuclei which are comprised among the exudations filling the pulmonary vesicles around the granulations among the first period of evolution, or among the increase of tubercular granulations. Pneumonia in its different periods of congestion, red or grey hepatisation, or cheesy stage, no longer exists in phthisis, and M. Villemin hardly admits that the bronchitis of phthisical patients is a simple inflammation.

"We have already explained on these points that which we cannot agree to, and demonstrated clearly the existence of pulmonary epithelium, and the work of M. Villemin is important enough in the history of tuberculosis to require criticism.

"Our work on pulmonary phthisis, made together with M. Hérard in 1867, admitted as conclusive the etiological and pathogenetic unity of phthisis pulmonalis, which did not prevent us admitting and describ-

ing the various lesions, granulations, and pneumonias, and the different part these play in the various cases under our observation. We have given the pre-eminence to the granulation, which is found everywhere the same, which exists alone in acute generalised consumption. We have regarded the cheesy or interstitial pneumonias as consecutive or concomitant lesions. We have hardly been able, even in a few cases of caseous pneumonia, to admit the absence of granulations. In such facts we had to do with chronic cheesy pneumonias when the fatty degeneration of the elements was so advanced that we hesitated whether the degenerated elements might belong to pneumonia or to granulations first of all. It is certain that this distinction is very difficult when we have to do with old masses, and we then preferred to remain in doubt rather than to affirm that we had to do only with a process of pneumonia. We have even risked this hypothesis, that some granulations might have existed at the commencement in the part of the lungs now occupied by cavities."—[This must often be the case.*]—" In the numerous autopsies of tuberculous patients completed by microscopic examination which I have had occasion to make since that time, I declare to you not to have met with one fact of tuberculosis where the absence of granulations has been clearly demonstrated to me."—[A very important statement.]—" You will see, gentlemen, that such is also the conclusion formulated in the thesis of two very competent observers who have devoted themselves to the study of the lungs microscopically, with all the materials, and with all the previous knowledge requisite—MM. Grancher, director of the Laboratory of Histology at Clamart, and by M. Thaon, whose work has been written under the eye and in the laboratory of M. Ranvier, in the Collége de France.

" The pupils of Virchow have accentuated more and more the separation of tubercular from cheesy pneumonia, and they have pursued this division not only in the domain of pathological anatomy, but in etiology, in symptoms, diagnosis, prognosis, and treatment.

" With regard to pathological anatomy, which we are now chiefly occupied with, this dualism is chiefly the result of incomplete knowledge and insufficient methods.

" I myself (Dr. Cornil) worked in Virchow's laboratory in 1862; at that time it sufficed that the morbid products were in the midst of the infundibula and surrounded by the alveolar walls to make them called pneumonia. They were looked on as pneumonia, although the elements were small, agglomerated, and glued together by a very coherent substance uniting them, and although their *ensemble* and grouping, and the alteration of their cellular constituents made of them perfect granulations, developed in an infundibulum. This error was discovered and soon rectified in the laboratory of Berlin; but at first the tubercular granulation therein was a myth in the lung, and it was only found at the sur-

* See Part I., p. 6; and Part II., p. 86.

face of the pleura or around the bronchi or vessels, where there exists a quantity of connective tissue sufficient to develop it.

"Virchow subsequently described peribronchitis as having nothing of affinity with the tubercles. The little indurated knots which are there met with around the bronchi, and the interstitial inflammation, with thickening of their external coats which accompany them in their whole course were separated from tuberculosis to enter into the class of broncho-pneumonias. It was under the influence of these ideas that Rindfleisch wrote the chapter of his book consecrated to bronchitis and broncho-pneumonia. The granulations of tubercle in the lungs became thus rarer and rarer. I have told you what I thought of peribronchitis; I have shown you that along the vascular canals or other canals surrounded by connective tissue, and in particular along the vessels of the pia mater, where this is easily observed, there exists, besides granulations visible at various distances, a zone of connective tissue in proliferation, or inflamed; I have shown you that this is the same everywhere, and that the granulations are always like other neoplasms, surrounded by a zone of inflammation"—[secondary].

"It is now easy for you to understand how generations of anatomo-pathologists reared in the same school have become *accustomed to never finding tubercular granulations in the lung.* In the other organs they were easily found amid the inflammatory zone, *but in the lung they only saw the pneumonia.*"—("Med. Press and Circular," December 10, 1873.)

On June 6, 1877, at Edinburgh, Mr. D. J. Hamilton read a paper on tubercle in the human lung. "From the time of Laennec," he said, "till a few years ago, there had been great confusion about tubercle."—[Does it not still exist?]—"As much of this confusion had arisen from an inaccurate use of terms, recent observers had tried to define what a 'tubercle' was. It would be his object to show that a tubercle really had a definite structure. There was no naked-eye characteristic of tubercle, and, therefore, no distinction could be made between tubercular and tuberculoid structure by the naked eye. In this country, tubercle was known as a lymphoid or lymphadenoid structure. If so, it was inflammatory."—[Not necessarily.]—"The first really good definition of tubercle was by Wagner. To the naked eye, true tubercles were about the size of a pin's head, elevated, rounded, not confluent, with no definite distribution, and seldom any caseation, as they were supplied with blood-vessels. Each tubercle, on section, was found to be made up of several giant-cell systems. The structure of a giant-cell system was as follows:—A large giant-cell was a rounded piece of protoplasm with from ten to one hundred nuclei, a vacuole, and many anastomosing processes, with cells in their meshes, of an epithelial nature, or leucocytes. Surrounding this there was a delicate band of fibrous tissue with leucocytes. In children, the tubercle was developed near the branches of the pulmonary

artery, which might give it small twigs. Mr. Hamilton stated that miliary tubercle was, he believed, as common in the adult as in children. As no distinction could be made in the early stages between catarrhal pneumonia and tubercle, the term miliary tubercle was accordingly only a naked-eye one. In their development, catarrhal pneumonia and tubercle were alike. In both, there was a development of the epithelioid cells lining the alveolus. Thus far they were identical."—[Common results of a local irritant.]—"The cells, however, in tubercle organised, while those of catarrhal pneumonia caseated. In the former case, one or more of the epithelial cells developed into a giant-cell"—[this is not proved]—"with processes entangling the others. Then the concentric band of fibrous tissue appeared, and in this way a 'tubercle' was formed. Thus, just as epithelial cells were now admitted to be the source of cancers, so epithelioid cells would probably soon be regarded as the source of 'tubercle'"—[or both ideas prove to be wrong].—"Tubercle might be secondary, and was, therefore, found in chronic catarrhal pneumonia, in fibroid phthisis, around dilated bronchi, in pleurisy, around cavities, and so on. These secondary tubercles were developed from the lymphatics of the interstitial tissue by the irritation of caseous material. The same views held for other organs. Phthisis of the kidney and testis were catarrhal."—"Dr. Wyllie said that Mr. Hamilton had alluded to the many changes in the use of the term tubercle. Long ago, any form of disease, if nodular, was considered tubercular. Laennec then limited it to those forms which were small, as in an ordinary phthisis. Virchow and his followers examined these, and, finding many of them inflammatory, *limited the term to those not so.* The next step was the investigation of tubercle artificially produced. Villemin, Wilson Fox, Burdon Sanderson, Cohnheim, and others, showed that the lymphatics were the parts involved. This accordingly seemed to settle the lymphadenomatous nature of tubercle"—[or rather showed an adenoid complication].—"The last series of observations was by Wagner, Schuppel, Buhl, Klein, etc., showing that tubercle was identical with catarrhal pneumonia."—[This has not been shown, but only that there are catarrhal complications in the products.]—"Mr. Hamilton had said that the cells in catarrh were larger and more actively proliferating than in tubercle. This, however, was difficult to settle, and, therefore, the question came to be, Is tubercle a catarrhal pneumonia? Probably true tubercle was at first a mere catarrhal pneumonia in the vesicles of the lung, forming giant-cells and caseating. Mr. Hamilton had said that tubercle of the lung did not undergo caseation. He had had no opportunities during the last year or two of investigating the subject; but he thought that Mr. Hamilton might be correct in saying that it did not occur in the lung. In a recent paper in Virchow's 'Archiv,' it had been shown that, in the testis, true tubercle did caseate. He himself believed that tubercles

were really due to catarrhal pneumonia; indeed, in children, Mr. Hamilton had said that the so-called miliary tubercles were really catarrhal pneumonia. From what had been said, it would be seen that he did not take the same view as Mr. Hamilton; but he thought that a great step had been made in the recognition of giant-cells in tubercle. . . . Dr. W. T. Gairdner agreed with the previous speakers as to the ability of Mr. Hamilton's paper. All of them, in their remarks, had gone back to Virchow. The time, however, at which he would begin was that of Lebœuf, when nothing was tubercular that did not contain tubercle-corpuscles. Thus all of them for many a year went in pursuit of these little angular bodies. Since that time, there had been many revolutions, and now they were getting definite views. Nothing hitherto had been so satisfactory as the views of Mr. Hamilton and Dr. Wyllie, and they probably ought to be regarded as complementary. But how to reconcile the new views with the old history of a disease so destructive in its tendencies; how to connect the history as given by Louis with the modern views, was a much more difficult task, which he would not attempt. The views advanced horrified him when they showed that cases apparently of tuberculosis were not that at all. All, however, reaped great advantage from free opinion in such cases; and he hoped that all would come right in the end."—"Mr. Hamilton replied that, in regard to Dr. Wyllie's remarks, he had, in his paper, said that the corpuscles formed at first by proliferation were the same as those in catarrhal pneumonia. He knew, however, that it was very difficult, indeed impossible, in the first stage, to separate tubercle from catarrhal pneumonia."—[Two effects of irritation upon two different histological elements, the further progress depending upon which predominates.]—"Very soon, however, the differentiation took place, because in catarrhal pneumonia there was caseation, but in tubercle organisation, and a distinct tubercular structure in many or all cases. The appearances were so remarkable that students could see the difference. In the third stage, there might be caseation in tubercle of the lung, but it was rare, limited, and after the tubercle had been produced. It should be kept in mind that tubercle was by no means a non-vascular structure." ("Brit. Med. Journ.," Sept. 1, 1877.)

At the Forty-fifth Annual Meeting of the British Medical Association (1877), "Dr. Andrew Clark exhibited a series of portraits of phthisical diseases of the lungs, and read a paper explanatory of his views with regard to the pathology of phthisis. He discussed three of the portraits at length, and gave particulars of the cases from which they were taken as illustrative of what he considered the three forms of phthisis: pneumonic, tubercular, and fibroid.—Dr. Wade (Birmingham) alluded to the importance of Dr. Clark's remarks, in their bearing on

the treatment of phthisis. He considered the local origin of this disease highly necessary to be remembered. He believed in the reality of hæmorrhagic phthisis, and mentioned some cases in illustration.*—Dr. Bradbury (Cambridge) could not agree with the threefold division of the varieties of phthisis made by Dr. Clark. He would like to have his opinion concerning the nature of tubercle. He was convinced that pleurisy often originated phthisis; and remarked that tubercle is frequently found in pneumonic cases. He regarded all forms of phthisis as products of inflammation.—The President (Sir William Jenner) observed that the microscopic specimens demonstrated by Professor Charcot were entirely contradictory of the views enunciated by Dr. Clark. From the earliest time that he (the President) taught medicine, though he had not used the term 'fibroid phthisis,' the pathological change thus characterised had been well known, and had been fully described by Addison.—Dr. Clark, in reply, said that he had, in his paper, particularly acknowledged what former observers had done. He drew a picture of the different forms of phthisis, constrasting their clinical history, and insisted that, although the anatomical elements were the same, the forms which the disease assumed, pathologically and clinically, were different, and justified different names. *As to the nature of tubercle, he could give no answer.* It could not be defined; its origin was not known, but all knew what it meant. The several forms of phthisis were not found in their simplicity at *post mortem* examinations, but this was due to the fact that each begot secondary changes of a different kind." ("Brit. Med. Journ.," August 25, 1877.)

The demonstrations referred to by Sir Wm. Jenner at the meeting just noticed, gave rise to the following leader in the "British Medical Journal,"—"On the Relations of Tuberculosis and Caseous Pneumonia." . . . "On this subject, there is a great want of agreement among pathologists. M. Charcot has studied it very thoroughly in his lectures this year at the Faculty of Medicine, and lately gave a summary of his conclusions at the Société de Biologie.

"Clinically, under the name of caseous pneumonia, pulmonary phthisis, is designated a subacute inflammation of the lung, which has often the characters of a fluxion: it commences then with a pain in the side, fever, etc.; presents stethoscopic signs which need not be described here; and in two or three months an enormous destruction of the lung is produced. By some, these characters and the acute course of this affection are made to distinguish it from tuberculosis properly so called.

"According to the doctrine of Laennec, tuberculosis in the lung is manifested sometimes under the form of grey miliary granulations, sometimes in more or less voluminous yellow masses. That learned

* I have asked Dr. Wade for these cases, but he is not yet in a position to produce them for criticism.

clinician thus attributed to the same process the miliary granulations and the caseous masses. In 1850, Reinhardt and Virchow substituted for the doctrine of the French physician the following formula. After pneumonia, bronchopneumonia, etc."—[in such a case there can be no initial loss of flesh; if loss of flesh preceded the pneumonia, then pneumonia was not the initial disease (see p. 165)]—" when resolution does not occur, the exudation accumulated in the bronchi and the alveoli degenerates, undergoes caseous transformation, and constitutes more or less voluminous masses. These play then the part of a foreign body determining a peripheral inflammation: then their centre breaks down and is eliminated, and a cavern is formed. In this case, tubercle does not exist: it is only accessory. Caseous pneumonia is, in a word, for Virchow, only a degenerated pneumonia: it is not a tuberculous pneumonia.

" By the kindness of his hospital colleagues, M. Charcot has been able to study the process in the lung from a great number of cases of caseous pneumonia. According as death is more or less rapid, the processes more or less generalised, there are found disseminated caseous *foci* of the size of a pea, of a small nut, or of a filbert, or confluent masses ; sometimes even a lobe or an entire lung forms only a mass resembling a piece of Roquefort cheese. With the aid of his pathological assistants, he has successively studied all these varieties. In particular, in one case, which ran a very rapid course, he made a very minute examination of the mode of formation of the little disseminated caseous *foci*. The patient had succumbed in fourteen days under a stroke of intercurrent diphtheria. It was thus possible to seize the pneumonic phthisis at the commencement of its evolution, which is rare "—[see remarks on morbid anatomy, Part I., p. 6,]—" that is to say, before it had undergone its phases, and the masses had become confluent. In all these cases, he finds, and can demonstrate, he believes, as the fundamental and exclusive *processus*, tubercle with all its characteristic attributes.

" What is, in fact, tubercle according to Virchow and the most critical persons ? The common tubercle, the grey microscopic granulation, is only an agglomeration of many follicles or elementary tubercles. To understand the constitution of this pathological corpuscle, it must first be studied in the organs in which it shows itself in the most simple state, as in the fungosities of ' white swelling,' or in the tongue. In these fungosities are found little whitish masses smaller than a millet-seed.

" Under the microscope, are observed : 1. In the centre one or more large cells, called giant-cells (these are constituted by a protoplasmic mass relatively voluminous, having scattered nuclei in its periphery) ; 2. Peripherally, an agglomeration of embryonic cells, which are strongly coloured by reagents. When degeneration occurs, the large

cells are the first to undergo the caseous transformation : the centre of the elementary granulation is no longer coloured by the carmine solution. In the tongue, it is agglomerated tubercles which are observed. One sees first many follicles or elementary tubercles, such as those which have just been described, develop separately in the little muscular bundles : they are placed in the little muscles, in the very centre of the myolemma, in the fibrous sheath, of which they gradually crowd back the contents. Then the follicles of neighbouring fascicules join, and together they form a veritable aggregation. They have then a common life: they are surrounded by a common zone—the embryonic fibrous zone."—[All these may be correct descriptions of effects of a molecular irritant, but no explanation of their occurrence is contained in their microscopical character. What we want to know is the initial process. (See pp. 168-9 and 194.)]

"Caseous degeneration occurs first in the most central follicles. In the lung, almost always, the follicles or elementary tubercles are agglomerated. The grey granulations—those which are met, for example, in the lungs of children who have died of generalised tubercle—are already very complex masses. With the naked eye, or with the pocket-lens, it is easy to observe that the grey granulation is installed in the network of the lung ; the alveolar walls are blended with it. It seems to be a sort of invasion of the tissue"—[as would be the case if due to molecular disintegration by oxidation].—"From the microscopic point of view, the grey granulation is formed of numerous elementary tubercles or follicles, like those which M. Charcot describes in the fungosities of white swelling or in the tongue ; that is to say, a centre formed of giant-cells and an embryonic periphery. When caseation occurs, these follicles are the first to undergo it. If one of these grey granulations be treated with caustic soda, the elastic framework of the lung reappears, and it thus may be recognised that the neoplasm occupies at once the alveoli and their walls. The framework of the lung is incorporated with it, and the elastic fibres remain. [The albuminoid tissue is disintegrated, *not* the gelatinous.]

"Such is tubercle, the classic tubercular granulation. Thus far, the description agrees with that of Virchow. M. Charcot proceeds to show that caseous pneumonia is an exclusively tuberculous pneumonia ; that is to say, that the caseous masses are enormous tuberculous conglomerations. He points out here that he has been preceded by distinguished histologists (who have recognised this nicety in phthisis) in France and in England. (He cites Grancher, Thaon, Renaud, and Wilson Fox.) He considers, however, that his predecessors have been too timid in their statements. He completes their thought and synthesises their description by saying that tubercle is not recognised in caseous pneumonia because it is of such great size. It is formed of a multitude of associated elementary tubercles"—

[a combination of proliferation, hyperplasia, etc., of the histological elements around the initial irritant. (See pp. 168-9.)]

"This he demonstrates as follows. Evidently it is not in a lung of which one lobe is completely caseous that we must seek for tubercle; it is in that stage of caseous pneumonia when it is still constituted of small disseminated masses"—[that is, less complicated by the results of subsidiary processes].—Subsequently, all the intermediate stages may be followed, from nodules of the size of a pea to total degeneration.

"By the aid of a schema, which Professor Charcot gives in his 'Lectures on the Histological Characters of the Lung,' it is always easy to find one's way over a preparation. With a very slight magnifying power, the intralobular bronchi are rapidly and easily recognised, in that they are joined to an arteriole. This is more easy than guiding oneself by delicate particulars of their structure. In the case of pneumonic phthisis which he was able to examine on the fourteenth day, and which has been already mentioned, it was easy by this mode of examination to recognise that the lobular bronchus and arteriole were here and there surrounded in caseous islets; otherwise, under this slight enlargement, the lung appeared relatively sound.

"Professor Charcot proceeds to study successively, by higher magnifying powers, the constitution of the 'islets' and the changes of the part of the lung which surrounds them, and which he compares—to continue the image—to the intermediate sea. The islets present in the centre an arteriole and a bronchus more or less completely obliterated, and whose walls are more or less altered"—[by oxidation of albuminoid tissue].—"They are surrounded by a yellow uniform mass, which does not become coloured by carmine. In this mass, soda reveals elastic fibres which outline the alveoli. The zone which surrounds the yellow mass, and which might be called 'the riparian zone,' becomes rose-coloured with carmine-solution; it is composed of embryonic cells." [the result of irritation and of attempts at repair].—"It is at the most external part of this riparian zone of the islets that the giant cells may be observed here and there, disposed like detached forts on the coast, circulating and forming the advance-guard"—[attempted repair of vessels, vaso-formative cells ?].—"There, each one of them is surrounded by a little special embryonic zonule. These giant-cells seem to be a centre of formation of elementary follicles, as a centre of crystallisation would be in mineralogy. In a word, each of these islets is only an agglomeration of elementary tubercles; its growth takes place at the periphery by the formation and adjunction of elementary follicles, each with their central giant-cells. There is, therefore, the greatest analogy between these caseous masses and the grey tuberculous granulation. They are simply grey granulations united in mass. Now, the largest caseous masses, whether they have the size of a nut or occupy the whole of a lobe, are always constituted in the same man-

ner. There are always to be found at their periphery the riparian zone of embryonic follicles and the giant cells."—[It is evident that we are dealing here only with the *effects*—of a cause not made apparent to the senses.]

"What has interfered with the conception and demonstration of the analysis of pulmonary tuberculosis and caseous pneumonia? According to Professor Charcot, it is the exclusive adoption of the idea of Virchow, that tubercle is a special neoplasm. The caseous masses, however, it has been already stated, have absolutely the same constitution as the grey granulation. The only difference lies in the size. [See Dr. Moxon's case, p. 85.]

"The attentive examination of what M. Charcot calls the intermediate sea—that is to say, of the pulmonary parenchyma around the islets—establishes yet more clearly the error of Virchow in alleging that caseous pneumonia is only a degenerated pneumonia. Indeed, in neighbouring alveoli, the most various lesions are found; epithelial catarrh, fibrinous exudations, purulent mucus. Now, these pathological products never undergo real caseous transformation. The epithelial cells, as everywhere else, become granulo-fatty, form granular bodies, etc.; crystals of fatty matter, etc., are found. Thus, it is not exact that these elements form a magma, as Virchow had alleged. Histologists have, in the view of M. Charcot, put too blind a confidence in the words of the great pathologist, and have admitted, without serious examination, what was rather a conception of his mind than a complete anatomical description. We must, therefore, in this view, return to the theory of Laennec—caseous pneumonia or pneumonic phthisis is of a tubercular nature."—[Its products are the result of a local irritant, and if this irritant is the result of oxidation of albuminoid tissue, loss of flesh will have preceded the attack of pneumonia. But the constitutional state may have been removed, and the irritant remain latent, till catarrh occurs. (See p. 204.)]—"It is constituted by conglomerations of tubercles often very voluminous, or, if one prefer it, by gigantic tubercles.

"M. Malassez partakes the general views of M. Charcot on the analogy of caseous pneumonia and tuberculous pneumonia. But he thinks M. Charcot's elementary tubercle still too complicated. The giant-cells are an extraneous attribute of tubercle; they are not found in the tubercles of the great omentum."—[No, the composition of tubercle must vary with the histological elements surrounding the initial irritant.]—"These are constituted solely by rounded or fusiform foci of embryonic cellules surrounding these connective bundles. In the second place, these giant-cells are not special to tubercle; they are also found in certain tumours, fibro-sarcomata, etc. They have intimate relations with the vascularisation."—[and may therefore appear wherever capillary repair is required].—"M. Charcot recognised the fact,

indeed, that the giant-cells are not essentially characteristic of tubercle; the researches of M. Malassez indicate that they are in direct communication with the vessels. They are angio-plastic prolongations which become hypertrophied. This is also the opinion of Brodowski."* —("Brit. Med. Journ.," Sept. 1, 1878.)

With reference to the nature of *giant-cells in tubercle*, "The Lancet" says in an editorial note:—"There is much difference of opinion amongst pathologists. Some have ascribed to them an especial importance in the formation of tubercle; others have regarded them as accidental productions; but few now deny their pretty constant occurrence. M. Cornil has recently brought before the Société de Biologie a communication designed to show that they are formed in the interior of obliterated vessels. He showed specimens from a tubercular infiltration of the pericardium, prepared by hardening with osmic acid and alcohol, and staining sections with picrocarminate of ammonia. From sections thus prepared he succeeded in isolating a large number of free giant-cells, consisting of masses of granular protoplasm of various shapes, with numerous prolongations, some of which were bifurcated, and containing many nuclei. These nuclei were always ovoid or budding, showing an active process of growth. They usually occupied the more peripheral part of the cell, and varied from two or three to twenty or thirty, or more, in number. M. Cornil believes that he was able to trace the process of growth of the cells in these and many other preparations of tubercle of serous membranes, and he describes it as consisting in a special inflammation of a limited part of a vessel, with coagulation of the fibrin, the accumulation of leucocytes in the clot, an active process of growth, and multiplication of these and of the endothelial cells, softening of the walls of the vessel, which, by infiltration with cells, become indistinguishable from the surrounding tissue.

"In opposition to this view, M. Malassez brought forward evidence to show that the true giant-cells of tubercle are not due to obliteration of vessels. He objected to the views of M. Cornil, that the number of giant-cells was often enormous, that they presented prolongations which could not arise in a vessel, that they were often much larger than the vessels in the part, and that they contrasted with obliterated vessels in the fact that their protoplasm was actively growing, not mere altered coagulum, and that they lacked all trace of a muscular wall. He himself urged that many of them were cells which should form vessels, but failed to do so, corresponding to the 'angioplastic' or vaso-formative cells of new growth. This view has before been advocated by Brodowski,* and adopted by Professor Charcot. But M. Malassez holds that other methods of formation may also be observed." —[Supposing the walls of capillaries to be disintegrated by oxidation, it is consistent with what we know of nutrition and repair, that

* Virchow's "Archiv.," vol. lxiii.

vigorous attempts should be made to replace or restore the damaged parts, and thus we might expect to find vaso-formative cells as an almost necessary accompaniment of such processes.]—"There can, we think, be little doubt that such is the case; that many of the so-called giant-cells are only obliterated vessels, but that true giant-cells are also found, and that these may be formed either by angioplastic cords or by accumulation of actively growing, but not yet differentiated, protoplasm, derived usually from endothelial cells."—("Lancet," June 29, 1878.)

Those who have taken the trouble to go critically through the preceding pages must have been struck by the comprehensive manner in which the doctrine of oxidation due to defect in the passage of fat into the blood throws light upon all other contending doctrines and observations respecting tuberculosis, tuberculisation, and tubercle, while it accounts for clinical facts which they ignore :—

1. It accounts for the *initial loss* of weight and strength which every other doctrine ignores.
2. It brings into place the loss of weight attendant upon lung-disease, but *not initial.*
3. It accounts for the *order* in which organs are affected with tubercle at different periods of life.
4. It accounts for the *order* in which the tissues of these organs are invaded.
5. It accounts for all the elements of the heterogeneous masses called *tubercle*—the bone of contention among pathologists of all nations.
6. It explains the *clinical history* of pulmonary consumption from its first symptom to its last.
7. It clears up the difficulties which have so confused and confounded pathologists as to the *local or general origin* of pulmonary consumption.
8. It explains the *action of all remedies* known to have any appreciable effect upon the disease.

It is as undeniable as it is satisfactory, that no remedy and no plan of treatment for pulmonary consumption has had any fair pretensions to success that is not consistent with the therapeutic dictates of my hypothesis.

For these reasons it must certainly be allowed to be the best working hypothesis yet presented, and, from "the simplicity of its conception, and the universality of its application," it may, in the words of the axiom which I have placed at the head of this chapter, be said almost "*to prove itself.*"

With regard to the question, Why the supply of fats is cut off from

the blood, no one has pretended that a better explanation can be found than the one suggested by myself, viz., *defect in the action of the pancreas, and of its coadjutors the intestinal glands, and the liver.*

When I brought forward the importance of the secretion of the pancreas, in 1864, it was quite natural that suggestions giving dignity to this organ should have met with incredulous smiles from a large number of medical men; for at that time the very existence of the pancreas was almost ignored in medical literature, so insignificant was thought to be its rôle in the organism. Even in 1872, in my paper "On the Use of Pancreatic Emulsion in the Wasting Diseases of Children," I was able to point out that, notwithstanding all I had written about the pancreas, "the word Pancreas does not occur in the indexes of the last editions of West, Tanner, Vogel, Meigs and Pepper, Eustace Smith, or Churchill on the diseases of children. . . Looking through these works and their indexes, one is led to the conclusion that their authors are not aware that there is such an organ as the pancreas." Nevertheless, so long ago as 1849 Professor Bernard ("Archives Générales de Medicine") had published his invaluable experiments showing the power of the pancreatic juice to emulsify fats, and still longer ago ("Med. Chir. Trans.," 1833) Dr. Bright had shown that in obstructions of the pancreatic duct undigested fat is excreted by the bowels. But ignorant doubts had been thrown upon the validity of Bright's conclusions, and the physiological results obtained by Bernard had taken no hold upon the mind of the profession as having the least bearing upon practical medicine.

This is the more astonishing if we read, in the light of subsequent events, the admirable little brochure of Mr. Jonathan Hutchinson—"On the form of Dyspepsia which often precedes and attends Phthisis," dated May 2, 1855.

He says, with regard to the difficult assimilation of fatty matters, "I am inclined to believe that this symptom may be made great use of for purposes of prognosis. Exceptions undoubtedly occur, but as a general rule, it might probably be safely laid down that the severity of the tubercular dyscrasia is measured by the difficulty with which cod liver oil is borne. The need of that remedy is mostly in exactly inverse ratio to the facility with which it is digested."

Passing on to speak of the treatment of the dyspepsia of phthisis, he says, "it must not be assumed from what has been laid down as to its essential characteristic being the *non-assimilation of hydro-carbonaceous matters*, that the forcible administration of such is the measure indicated. It would be perfectly useless to order the subject of confirmed dyspepsia of this kind to take fat; in the first place his palate would refuse it; and, secondly, if swallowed, *his stomach, pancreas and liver are quite incompetent to its digestion.*" And again, he says, "All who have investigated the dyspepsia of tuberculosis have arrived at

nearly the same conclusions as to its treatment, and the conclusions of all are in active support of the theory of *impaired hepatico-pancreatic functions.*" At the time this was written, Mr. Hutchinson's name was not known to fame, and his wise words excited little attention.

I am happy to think that all this apathy on the subject of the pancreas and its functions has passed away. At the present time there is no organ in the body the name and functions of which are more familiar to the whole body of the profession throughout the world than the once despised pancreas.*

Now I particularly wish, in this relationship, to make prominent the fact which I pointed out in 1865-66, viz., *that the pancreas is a remarkably sensitive organ*, comparable more in this respect with the mammary gland than with any other, and that its secretion can be deprived of its normal properties without leaving traces of structural disease in the pancreas—just as the mammary gland can secrete milk unfit for nutrition, or cease to secrete at all, and yet return to its full

* "THE PANCREAS IN FISHES.—Very imperfect, to judge from the vague descriptions given in works on Comparative Anatomy, seems to have been, until recently, our knowledge regarding the nature and varieties of the pancreas in fishes. Now, however, some addition to our knowledge, or rather enlightenment of our ignorance, has been furnished by M. Legonis, in a paper 'On the Pancreas in Osseous Fishes,' a translation of which, from the 'Comptes Rendus,' appears in a recent number of the 'Annals and Magazine of Natural History.'

"The so-called 'pyloric appendages' had been generally supposed to subserve in some way or other the functions of a pancreas, until it was observed that they co-existed in certain fishes—*e.g.*, the trout—with this organ, and could not, therefore, be homologous with it. Furthermore, certain ducts—'Weber's canals'—running from the liver to the intestine in company with the biliary channels, were believed to be carriers of the pancreatic secretion; their seeming origin leading Weber to suppose that the liver fulfilled a double function, an hypothesis rejected by Claude Bernard.

"According to M. Legonis, besides the Plagiostomes—viz., the sharks and rays, which have a pancreas similar to that of other vertebrates,—all osseous fishes possess some representative of this organ, however variable it may be in form and position.

"Three forms are described :—

"1. *Disseminated*, found in the barbel, loach, and lump-fish, consisting of glandular lobules dispersed through the laminæ of peritoneum.

"2. *Diffused.*—This form, which resembles the pancreas of the rabbit, occupies the interstices of the viscera, and is found in the conger, gurnard, stickleback, and others.

"3. *Massive*, resembling the organ in higher vertebrates; such occurs in the pike, eel, and Silurus.

"The ducts of the first two forms are the afore-mentioned 'Weber's canals.' Their course, which is not well defined, terminates at the duodenum, in the neighbourhood of the gall-ducts. The apparent extension of the pancreas into the liver, which was the cause of Weber's faulty conclusion, may be accounted for by the fact that the development of these two organs still goes on in the adult fish.

"To any who are striving to find in higher vertebrates bodies homologous with the pyloric appendages of fishes, we may suggest that the glands of Brunner, which are so well developed in some mammals—*e.g.*, the mole and shrew—may be possibly truly represented by the follicles which stud the lining of these saccular organs."—("New York Medical Gazette," Sept. 3, 1870.)

and normal function without any coarse disease being detectable. The careful and often repeated experiments of Bernard show that when the nerves of the pancreas are acted upon, either by excitation of the cerebro-spinal or by section of the sympathetic fibres, the secretion grows abundant and uninterrupted, while a profuse diarrhœa is constantly established. Extirpation of the semilunar ganglion produces similar effects; and under special conditions the pancreas pours forth a peculiar fluid *which no longer exhibits the physiological properties of the secretion.* When the secretion is thus rendered continuous, the characteristic active principles are no longer produced within the gland, and the watery vehicle alone escapes from the secreting apparatus. Bernard particularly insists upon the important fact that "*the general perturbations of the economy*" exert a powerful influence upon the functions of the pancreas, and the least degree of inflammation in its neighbourhood perverts the properties of the pancreatic juice. (Bernard's "Lectures on Physiology," "Med. Times and Gaz.," 1860.)

From the effect of nervous influences upon secretion generally, especially upon that of the salivary glands, the stomach, the liver, and the lachrymal and mammary glands (see Carpenter, 6th edition, pp. 738-744), we might reasonably have expected by analogy that the pancreas would suffer in the manner shown by Bernard. It is evident, then, that *the pancreatic secretion can be deprived of its normal properties by causes acting through the nervous system, both cerebro-spinal and sympathetic; and by affections of neighbouring organs.* Such causes may be of a temporary or of a more permanent nature, and unless of long duration, may not leave traces of structural disease in the organ.

It is impossible to shut our eyes to the importance of this influence of "the general perturbations of the economy" upon the functions of the pancreas, when regarded in conjunction with our knowledge of the importance of fat in the economy, and of the necessity for healthy pancreatic function to bring that fat into the blood. We cannot be surprised that loss of fat and strength should follow in the track of such causes of perturbation in the economy as are ranked by popular experience among the causes of consumption.

But there are many other causes of perverted pancreatic function than "the general perturbations of the economy."

Defect in the function of the pancreas may be produced :—

1. By any cause which for a prolonged period greatly reduces its activity, by diminishing the normal demand for carbonaceous matters in the blood.

2. By prolonged loss of absorbing power in the small intestine by which the function of the pancreas is rendered useless.

3. By the action of powerful or prolonged depressing influences.

4. By inflammatory and other abnormal conditions of neighbouring parts.

The normal function of the pancreas may be restored (within certain limits as to the duration of the existence of the cause of its defect) by means which—make a healthy demand upon its active functions; remove the depressing influences from the nervous system, and permit the absorption of properly prepared fats by the mucous membrane of the small intestine.

In my papers "On Tuberculosis," in 1866, I showed that all the supposed causes of consumption given in the following list, selected from various authors of authority, might be brought back to the pancreas, and arranged as causes of perversion of its functions.

1. Disappointment; home-sickness; longings after the objects of affection, either absent or lost; and other depressing influences.

2. Whatever depresses vital power—exhausting passions, poverty, and the vicissitudes of life.

3. Persistent defective expansion of the chest, and defective exercise of the respiratory powers.

4. Persistence of young persons in a diet deficient in milk.

5. Inattention to a due preservation of the cutaneous function.

6. Chronic alcoholism.

7. Neglect of exercise in the open air: deprivation of sunlight; congregation of numbers in a close or insufficiently ventilated place. "If disease of a given form be so associated with certain conditions that, in two-thirds of the instances which present themselves to our notice, these conditions be present, we cannot avoid referring the diseased result to the antecedent error. In other words, deficient ventilation and crowded apartments are eminently productive of tubercular disease. . . . The effects of posture are chiefly observable in bootmakers, tailors, hand-weavers, and others whose work necessitates the stooping posture. The results are mechanical hindrance to the free entrance of air to the chest, restricted expansion of the bony wall, and an imperfect respiration. In the case of bootmakers, there is the addition of pressure on the epigastrium, which gives rise to the well-known neurosis. But, on the whole, all the errors of this class are to be referred to insufficient expansion of the chest.' (Pollock, 'Elements of Prognosis in Consumption,' p. 368.)

8. 'Rebreathed air.' 9. Cyanosis. 10. Diabetes. 11. Acute diseases, especially continued fever. 12. Childbirth. 13. Superlactation in mothers. 14. Weaning in children. 15. Hereditary taint.

All these supposed causes of phthisis may be assembled under four heads.

1. Causes which *act directly* upon the pancreas, either specially, or in common with other internal organs; *e.g.*, hereditary tendency to disease of the pancreas; occupations causing pressure upon the abdo-

men in the pancreatic region; acute diseases, especially continued fevers; inflammatory affections of neighbouring parts.

2. Causes which *act indirectly* upon the pancreas, by diminishing the elimination of carbon from the blood and thereby reducing the normal call for the introduction of fat from the food into the blood. By these means, the pancreas is kept in a state of inactivity and low nutrition, until in time its secreting powers are depraved or destroyed, and the organ itself becomes degenerated, in accordance with the laws governing all other secreting organs.

It is evident that this heading will include a large number of the causes of phthisis which I have enumerated; *e.g.*, all those which diminish respiratory blood-changes for protracted periods, whether it be simply through deficient expansion of the chest, or through the hypercarbonised condition of the air presented for respiration; and all causes of deficient execretion by the skin, especially if combined with defective respiratory action. It will also include cyanosis and chronic alcoholism.

Cyanosis is a condition which would, at first thought appear to be antagonistic to my hypothesis; for, if tubercle is produced by the combustion of the fat elements of the albuminoid tissues, it would seem that a disease in which the blood is persistently surcharged with carbon would, *par excellence*, be protective against tuberculosis. But the contrary is the fact; and it is easily explained. It is only protracted cyanosis that ends in tuberculosis. In these cases the long continuance of hypercarbonised blood paralyses the function of the pancreas. But as it is not carbon only that is requisite to maintain nutrition and protect against tuberculosis, but fat properly prepared by the pancreas, after a time the albuminoid tissues are invaded to supply the fat-elements which the pancreas has lost the power to prepare, and tuberculisation results.

In chronic alcoholism, the mode of operation is somewhat similar to that in cyanosis. A form of hydrocarbon is thrown into the circulation through the portal system, which substitutes the normal supply of fats by the lacteal system. The affinity of oxygen for alcohol being greater than its affinity for fat, respiration is supplied from this artificial source with carbon for direct combustion; an artificial nutrition is kept up, in which the natural call for fat is stopped, and the function of the pancreas is reduced to supplying the minimum quantity necessary for histogenetic purposes. To this inactivity of the organ is added the usual tendency to degeneration due to alcoholism. In course of time, the pancreas loses that minimum amount of function which it had been allowed to exert, and fails to supply even so much fat as was necessary to protect the albuminoid tissues; and tuberculosis results. Or, as frequently happens, the toper ceases to obtain his supply of alcohol, either from inability to get it, or from inability to absorb it or to retain

it on the stomach. His artificial supply of carbon, upon which he has been depending, is thus cut off ; a sudden call is made upon the pancreas for that which it has now lost the power to give ; the tissues are disintegrated to supply the required fat-elements ; and tubercle is produced.

3. Under a third head may be included causes which deprive the system of carbon to such an undue extent that the pancreas cannot keep pace with the demand made upon its function; the supply of fat to the blood becomes insufficient to keep up the waste; and the albuminoid tissues are invaded. This may be the case when those who inherit a tendency to disease in the pancreas, or who have acquired a feeble secreting power in the organ, are exposed to drains upon their fat-elements which in healthy persons might be met by increased function. It is thus that, in tuberculous families, childbirth, superlactation, and profuse purulent discharges, especially when not protected by proper diet and regimen, precipitate the patient into tuberculosis; and as the mother is deprived of fat-elements by lactation, so is the child deprived of them by a persistence in a diet deficient in milk. In the case of a child thus deprived of fat, a double injury is done—first, by cutting off the supply of fat-elements necessary for the protection of the tissues; and, secondly, by paralysing the function of the pancreas by prolonged inactivity. (See Part V., "Wasting Diseases of Children.")

Under this heading must also be placed diabetes. Tuberculosis occurs in diabetes when the excessive drain upon the carbon by the excretion of the carbo-hydrates, as sugar, is not duly supplemented by a corresponding supply of fat in the food; or when the demand for pancreatic action has become so excessive that the pancreas is no longer able to keep up to it. Then the albuminoid tissues are attacked, and tuberculisation occurs.*

* "*Pulmonary Phthisis in Diabetes.* — According to M. Bourchardat, pulmonary phthisis, which so commonly supervenes on diabetes, is always tubercular. It is the result of general exhaustion, bad digestion of feculent food, the presence in the blood of an abnormal amount of glycose, the elimination of glycose through the kidneys, and the replacement of this excreted glycose by the slow destruction of the constituents of the blood, muscles, and the other organs." ("Revue des Sc. Med.," Tome vii., 1876.)

"*Diabetes Mellitus with Changes in the Pancreas.*—M. Lancereaux read a paper on two cases of diabetes mellitus. In these two cases a certain number of points in common were remarked, viz., rapid and considerable emaciation, general atrophy of all the systems, with perhaps the exception of the nervous system, insatiable appetite, unappeased thirst, excretion of a large quantity of urine and sugar, and death at the end of two or three years. At the necropsy, atrophy or even almost entire destruction of the pancreas, due to obstruction of Wirsung's duct and the accessory pancreatic duct, by numerous calculi of carbonate of lime, hypertrophy of the glands of the stomach, and of the duodenum, increase in the size of the spleen, and even of the lymphatic glands of the abdomen, and pulmonary lesions, were found. M. Lancereaux had found, in medical literature, ten similar cases, which he grouped according to the nature of the pancreatic change. Moreover, rapid decline with emaciation is a constant

"4. A fourth heading will include all causes which act powerfully in depressing the nervous system, either suddenly or gradually, and by this means pervert or paralyse the secreting function of the pancreas. It is in this way that "the vicissitudes of life" bring on tuberculosis, especially in those who by hereditary transmission have a tendency to depraved pancreatic function, or in whom the causes of nervous depression are conjoined with some of the other causes of affections of the pancreas which have been enumerated. Under this fourth heading, therefore, we must place all forms of shock to the cerebro-spinal or ganglionic nervous systems, and all such causes of mental and nervous depression, as disappointed love or ambition, excessive grief, unrequited longings, hope deferred, and the like.

Now, whether we admit or not that perverted pancreatic secretion is the initial cause of consumption, no one can deny that the suggestion that it may be so is a "good working hypothesis;" for, as I have shown with regard to my oxidation hypothesis (p. 194), it is consistent with all our knowledge of the *clinical history of tuberculosis;* it is consistent with all our knowledge of the means of treatment which have proved most useful, in the hands of all, by whatever theories or experience they may have been dictated.

I have not hesitated to bring it thus prominently forward in this place, because we cannot deal with the great question now before us,

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symptom; and it is worthy of remark that the emaciation and the voracity are symptoms of which the existence has been noted in animals in which the pancreas has been destroyed by physiologists. There is, therefore, reason for making researches as to whether there is not a causal relation between serious changes in the pancreas and that form of diabetes which is distinguished by a relatively sudden commencement, and especially a rapid evolution." ("Lond. Med. Rec.," Dec., 1877.)

"*Tubercle and Diabetes.*—Although M. Lécorché ('Traité du Diabète,' 1877) believes that many of those pulmonary cavities which are found in diabetic people are of a nature merely inflammatory, he teaches that, in a still greater number of cases, the origin of these lesions is tubercle. It is more usually in patients aged from fifteen to twenty that diabetes gives rise to tubercle. In children and in aged people this complication, as a rule, does not develop until two or even three years from the beginning of diabetes. In adults it supervenes as early as from five to six months after the onset of the glycosuric affection. It is also quite true that tubercle is far more liable to complicate diabetes in the case of poor than in that of well-to-do patients. Nevertheless, the opinion of those who deny its occasional occurrence in the latter is erroneous. The only feature special to tubercle connected with diabetes is the rapidity of its evolution. The duration of the disease rarely, however, is less than six, and it may very possibly exceed eight months. Instead of making incessant progress, the tubercular disease sometimes proceeds by successive deposits, and in the meantime the lesions remain quiescent, hence the comparatively protracted duration of the disease. In diabetic tubercle, provided the fever do not run too high, the appetite in many instances remains good almost to the last. Extreme emaciation, nevertheless, is invariably present." ("Dr. Dobell's Reports on Diseases of the Chest," Vol. III., 1877.)

—Loss of Weight—in any satisfactory manner, as practical physicians, without reference to the functions of the organs by means of which fat is introduced into the nutritive fluids of the organism.

I think it must be conceded, then, at the very least, that whether or not these two hypotheses entirely hold their ground in the light of future research and experience (and the history of science and of medicine forbid us to hope that they will not need revision from time to time)—I say, I think it must be at least conceded that they afford us two good working hypotheses, which taken together cover an immense amount of ground; both consistent with, and explanatory of, all known pathology and etiology of phthisis; and both consistent with, and explanatory of, the results of all enlightened treatment of phthisis.

The great importance of this to us as practising physicians is, that hypotheses which are consistent with, and explanatory of, so large a number of the established facts connected with any disease *may be trusted, better than any other guide we have, to suggest further successful treatment.*

It was thus that I was led to the invention of pancreatic emulsion; and to the use of this and of pancreatine in consumption and other wasting diseases,—remedies which have proved of such inestimable service to the world. And there is every reason to hope that in the same way other means of treatment may yet be suggested under the guidance of these hypotheses. Indeed, I may fairly add in this place that it was under the dictation of this train of ideas that I was led to insist upon the importance of local antiseptic treatment by means of inhalation, and to make it a leading feature of my treatment of phthisis at the Royal Chest Hospital and in private practice long before its importance was as generally accepted as it may be said to be at the present day—long before "antiseptic surgery" had opened the mind of the profession to the importance of antiseptic medicine. (See Part V.)

We have now arrived at a position in which we are able to answer the somewhat complicated set of questions which I propounded at Part I., p. 8, as follows :—

"If it is found that in a *vast majority of cases* of pulmonary consumption (destructive lung-disease with constitutional decline), local disease is preceded by constitutional disease by an unequivocal interval, and yet that *in a minority* of cases local disease precedes constitutional disease by an unequivocal interval ; the conclusion is almost inevitable that there are at least two modes in which pulmonary consumption may commence. It then becomes of the greatest interest and importance to ascertain whether these two classes of cases are to be considered as *absolutely distinct throughout*, or whether there is a period in their causative history at which they meet on common ground. That is to say, Is there a stage at which the constitutional disease sets

up the local? And is this first stage of the local disease in these cases the same as the first stage of the local disease in the cases not preceded by constitutional disease? And again, starting from this point, are the subsequent symptoms of constitutional decline in the two sets of cases due alike to the effects of the progress of the local disease? Or are they, in the first case, due to the progress of the original constitutional disease, plus the effects of the local disease? And in the second case, are they due to the effects of the local disease alone? Again, does this local disease *per se* set up a state of constitutional disease of *the same nature* and leading to the same local effects as that which precedes the local disease in the first set of cases? Or is a new constitutional disease of special character set up by the local disease in the second set of cases? And if so, is *the local disease of the first set of cases* also competent to set up a special constitutional disease *the same as that of the second set?* Finally, are the symptoms of constitutional decline which follow the establishment of local disease in each set of cases devoid of all special character, and only such as accompany the progress of any local disease proceeding to a fatal termination?"

Answers :—

1. It is found (see my Tables) that in a vast majority of cases, local disease is preceded by constitutional decline by an unequivocal interval.—Constituting Class 1.

2. In a minority of cases, local disease precedes constitutional decline by an unequivocal interval.—Constituting Class 2.

3. These two classes must be considered as absolutely distinct throughout.

4. There is no stage at which these classes meet on common ground. Class 1 is always plus Class 2.

5. There is a stage in which the constitutional disease sets up the local disease in Class 1, *i.e.*, when oxidation of albuminoid tissue begins.

6. The first stage of the local disease in Class 1 is not the same as the first stage of local disease in Class 2. In Class 2 there is no initial oxidation of albuminoid tissue.

7. Starting from this point, the subsequent constitutional symptoms of decline are not alike due to the effects of the progress of the local disease. In Class 1 they are due to the progress of the constitutional disease, *plus the effects of the local disease*, and in Class 2 they are due *only to the effects of the local disease.* The first will always, *therefore, unless arrested, beat the second in the race of constitutional decline.*

8. The local disease in Class 2 does not *per se* set up a state of constitutional disease of the same nature, and leading to the same local effects as Class 1.

9. A new constitutional disease of special character may be set up

by the local disease in Class 2, and the local disease of Class 1 is competent to set up a constitutional disease of the same special character as in Class 2. In both cases this disease is due to the absorption of products of the local disease.

Finally, as already stated, the symptoms of constitutional decline which follow the establishment of local disease in each set of cases are not "devoid of special character, and only such as accompany the progress of any local disease proceeding to a fatal issue"—but are of a distinct and special character, due, in Class 1, firstly, to the defective supply of fat to the blood; secondly, to consequent disintegration of albuminoid tissue; thirdly, to consequent irritative hyperplasia and proliferation; and fourthly, to absorption into the blood of morbid products; while, in Class 2, the symptoms of constitutional decline are due to (1) irritative hyperplasia and proliferation, due to inflammatory and catarrhal processes; (2) exhausting discharges; (3) absorption into the blood of morbid products.

It is competent for Class 2, through "perturbations of the economy," to set up Class 1, by arresting the supply of fats into the blood, in which case—from the addition of this cause of loss of weight—a sudden urgency of constitutional decline will take place, and then, Class 2 approximates in its later stages to the characteristics of Class 1.

It is also competent for the original cause of constitutional decline in Class 1, viz., arrested passage of fat into the blood, to be overcome in the course of the disease, and thus the cause of loss of weight characteristic of Class 1 may be removed; in which case Class 1 will approximate in its later stages to Class 2.

The nearest approach to common ground by the two classes of cases, is where the following peculiar conjunction of accidents occurs, viz., both Class 1 and Class 2, having advanced to the stage mentioned at No. 9, viz., the establishment of a new constitutional disease due to the absorption of the products of the original local disease; and the cause of the original local disease in both sets of cases having been arrested and overcome, the two sets of cases may then run a similar course—and constitute what I described in my treatise on tuberculosis as "tuberculæmia." Thus, speaking of the debris of disintegrated albuminoid tissue (the irritative cause of tubercle in true constitutional tuberculosis), I said it "may be allowed to remain on the spot where it is formed, constituting *a primary deposit at the point of origin;* or it may be carried away by the lymphatics, and either arrested in the lymphatic glands or carried on into the blood to be deposited from it, constituting a *primary deposit distant from the point of origin;* or having been primarily deposited in either of these ways, it may be taken up by the lymphatics and deposited in the lymphatic glands, or carried into the blood and deposited from it, constituting a *secondary deposit.* In the *advance* of these diseased processes, any part of the body in

which nutrition is going on in the albuminoid tissue may become both the source and seat of tubercle, and any part, whether albuminoid or not, if supplied with lymphatics or blood vessels, may become *the seat of tubercle.*

"*A secondary state is superadded when tubercle has been carried into the circulation, which constitutes tuberculæmia or tuberculous blood poisoning.* . . . The fat elements in the blood having been restored, tuberculosis will cease; but *tuberculæmia*, with all the difficulties due to the presence of foreign, decomposable, and absorbable matters in the tissues and blood, may remain."

"In progressing tuberculosis, after the occurrence of tuberculization, tuberculæmia is added to and combined with the original disease. An ordinary case of consumption is a combination of these three states."

To the above I added the following note:—"It is not certain whether or not absorbed tubercle has a zymotic action. I suspect that it has. From my own observations, I am disposed to think, that laryngeal phthisis will be found to occur more frequently in persons who have slept, night after night, with patients suffering from the last stage of consumption, than in other persons; and that molecules of tubercle may, in this way, be occasionally transplanted by the breath from the lungs of one person to the respiratory passages of another; then being carried by the lymphatics may produce 'a deposit distant from the point of origin,' or if carried into the circulation, producing tuberculæmia and secondary deposits. I am not prepared to say this is the case; but to suggest it as an important subject for further clinical and pathological observation." ("On Tuberculosis," 2nd Edit.)

A learned article entitled "Is Phthisis Pulmonalis Contagious?" by Dr. W. H. Webb, of Philadelphia, will be found in the American "Journal of the Medical Sciences," April, 1878, in which the author concludes by saying, "That phthisis is a contagious disease, and therefore belongs to the zymotic group, the evidence and proof, as herein presented, are, the writer believes, decisive and irrefragable."

The "Lancet," June 8, 1878, says:—"*Is Consumption Contagious?*—From the time of Aristotle to the present, various medical writers have urged that phthisis may be propagated from one individual to another through the medium of a material cause. Although we feel bound to say that the supporters of this doctrine have been in the minority, yet they include amongst them many of the celebrities of our profession, and there can be no doubt whatever that many of the most illustrious pathologists and clinical observers of our day coincide in this view. In Dr. R. Morton's 'Treatise of Consumptions,' published in 1694, and Dr. T. Young's 'Practical and Historical Treatise on Consumptive Diseases,' published in 1815, these opinions are set forth with great force and vigour. It is curious to notice that the contagiousness of phthisis is, and has been, very generally held in Southern Europe, that it has been doubted, as a

rule, and especially during the past century, in North Germany and Great Britain, whilst French authorities have been much divided on the question. Since the investigations of Villemin, Wilson Fox, Sanderson, and others on the inoculability of tubercle, and the spread of the disease from local infective centres, physicians are much less inclined than they formerly were to underrate the importance of contagion as a factor in the causation of this disease. Dr. Walshe, in 1860, considered the influence of contagion anything but proven, but in 1871 he had considerably modified his views, for he said, 'My belief in the reality of such transmissibility has of late years strengthened, I have now met with so many examples of the kind that *coincidence* becomes itself an explanation difficult of acceptance.' Cases due to a supposed contagion are generally of an inflammatory character, and very rapidly run on to destruction of the lungs and a fatal termination. We are frequently asked why, if consumption is contagious, wives and husbands who have been in close attendance on their diseased consorts do not inevitably become affected? The answer to this query is, that even when experiments have been carried out on animals, the tuberculising process occasionally fails to take place, and the direct transmission of infecting particles from the lungs of one individual to another is obviously much more liable to failure. As a matter of practice, we think that it cannot be too strongly enforced that it is a very dangerous proceeding to regularly share the bed of a phthisical patient, and to be habitually in close contact with, and attendance on, such a person."

PART V.

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LOSS OF WEIGHT & PULMONARY CONSUMPTION.

TREATMENT.

LOSS OF WEIGHT & PULMONARY CONSUMPTION.

PART V.

Loss of Weight and Lung-Disease.—Treatment of the First and Worst Cause of Loss of Weight, viz., a Defective Supply of Fats to the Blood—1, When it is hopeless to restore the Lost Functions of the Stomach, Liver, Pancreas and Intestines; 2, When this is not Hopeless, *i. e.*, Essentially Curative Treatment.—Treatment of Tuberculosis, of Tuberculisation, of Tuberculæmia.—The True First Stage of Consumption.—Initial Loss of Weight Antecedent to Tuberculisation, Difficulties of Treatment and the Means of Overcoming them.—The Arms with which we have to Fight.—Pancreatic Emulsion.—Pancreatine.—Peptodyn.—Malt Extract, Cod Liver Oil, Local and General Exercise and Rest.—Climate, How and When to Use Special Climates in Special Cases.—Altitude, Special Views as to Exercise at High Levels.—Quinine.—Arsenic.—The Salfatara at Pozzuoli.—La Bourboule.—Chloride of Calcium.—Wasting of Children (Marasmus), and the Use of Fat and Starch.—Nascent Milk and Pancreatic Emulsion.—Intravenous Injection of Milk.—Principles of Diet in Disease.—What we have to Fight in the Lung-Diseases connected with Loss of Weight and Blood-spitting.—The Author's Opinions with regard to Dictating the Details of Treatment for Individual Cases.

SUCCESSFUL treatment is the end and aim of all practical medicine; whether preventive, curative, or palliative, still it must be treatment. Let us then proceed at once to consider what are the arms at our command, and how it is best to use them in our fight with loss of weight, the antagonist which now confronts us face to face.

I think I have sufficiently fulfilled the promise made at page 161, that we should "reconnoitre the camp of our foe," in order to remove all doubt as to the *deadly power of the first and worst cause of loss of weight—a defective supply of fats to the blood.* So dire, so manifold are the ills traceable to this apparently small functional derangement that it might well have given rise to the trite old axiom, "great events from little causes spring." And as the treatment of all other causes of waste is simple and easy as compared to this, we shall give it precedence of the rest.

Supposing the supply of fat to the blood to be arrested, through defective pancreatic function, defective absorbing power in the intestines, and defective digestion, the following are the principles of treatment indicated.

1. If the case is so far advanced that it is considered hopeless to aim at restoring the functions of the pancreas and of the digestive tract, two principles must guide our treatment.

A. To supply the greatest amount of fat to the blood that is possible by other means than through the action of the pancreas:—

a. By giving pancreatic emulsions of solid fat, or, if these are rejected, pancreatic emulsions of oil, in the hope that some absorbing power for such matters may yet remain in the lacteal system.

b. Chiefly, by giving olein, with a view to its absorption by the portal system of vessels: and by rubbing it into the surface of the body and limbs, with a view to its absorption by the skin.

c. By giving highly albuminous food in conditions in which it may be most easily utilised, so as to supply an excess of material as a source of carbon by disintegration. (See p. 230.)

B. To save the albuminoid tissues from disintegration to the greatest possible extent—

a. By supplying a surplus of carbo-hydrates, so as to economise as much as possible the consumption of hydro-carbons.

b. By diminishing the proportion of oxygen in the air presented to the lungs.

c. By diminishing the demand for the generation of animal heat, by supplying it artificially.

d. By reducing respiratory and cutaneous action to the lowest point consistent with maintaining what remains of appetite and digestive power.

It is obvious that *persistence* in these means must be considered as utterly at variance with curative intentions, and simply as the expedient best fitted to prolong the process of gradual death.

2. If the symptoms are only what is commonly called "premonitory" —that is, if they are those of commencing tuberculosis ("the true first stage of consumption" as I have called it), and no reason or sign is discoverable which justifies the suspicion that tuberculisation has commenced; if a sufficiency of fat-elements remains, without calling upon the albuminoid tissues—*the principles of treatment are quite opposite to those last detailed. They are now entirely curative in their intention.*

A. To restore the function of the pancreas as quickly as possible, by placing the patient under those conditions which call for pancreatic action:—

a. An atmosphere rich in oxygen.

b. A climate at once cold and dry.

c. Exercise of the respiratory and cutaneous functions.

d. Cheerfulness of the mind and quiet to the emotions; exhilaration of the animal spirits.

e. A good mixed diet, easy of digestion, and free from substances irritating to the mucous tract.

f. Pancreatic emulsions of solid fat, and pancreatic juice in the form of pancreatine and peptodyn, in quantities sufficient to ensure protection to the albuminoid tissues while the process of restoration is going on in the pancreas and digestive tract, but *not in such quantities as to entirely supplant* the natural pancreatic function and thus to counteract the measures adopted for calling it into action. (See p. 259.)

These two classes of cases represent the two extremes in the history of tuberculosis—the greatest and the least hope of cure.

3. A third class, intermediate between these extreme points, includes the bulk of all cases brought under the care of the physician. They require a combination of the two principles of treatment already stated. The nice adjustment of these two plans of treatment—the hopeful and the hopeless, the palliative and the curative—calls for all the intelligence and judgment that a medical man can possess. His watchword, —"*σωφροσύνη*"—must never be forgotten. (See p. 122.)

If tuberculisation, even in the smallest degree, is going on, the first and most urgent need is to supply fats. Olein should be given for absorption by the portal system—thus to afford, so far as it is able, materials for combustion. But, above all, FATS ACTED UPON BY PANCREATIC JUICE are called for, and must be supplied until the deficiency is removed, the just balance restored, the process of tuberculisation stopped. Until this point is arrived at—until the balance is turned in favour of the albuminoid tissues—*everything which favours the reception of oxygen into the blood, everything which increases the wear and tear of the body, everything which calls for the generation of animal heat, directly favours tuberculisation, and precipitates the patient into the very catastrophe we wish to avert.**

On the other hand,—as soon as the balance has been turned in favour of the albuminoid tissues, and tuberculisation has been artificially arrested,—*everything which postpones the restoration of the functions of the pancreas and of the digestive tract, directly favours the conversion of a temporary into a permanent disease.* But moderation is essential to success. Too great and too sudden a demand upon weak and defective digestive organs may only paralyse the organism in its attempts at restoration of function. It is, however, impossible to magnify the importance of *periods of arrested tuberculisation*, whether artificially or naturally produced. They are the bright opportunities for the permanent restoration of pancreatic function. Too often these opportunities are wasted. The patient and his friends, pleased with the marked improvement that has taken place under some plan of treatment, either continue this treatment long after its proper time and place have passed by, or give up treatment altogether, content to wait till signs of active disease return. In either case, the opportunity for establishing a permanent recovery is thrown away, and perhaps it may never return.

* See Part IV., p. 152, "Spectroscopic Examination of Blood."

It must not be forgotten that, when tuberculisation and tuberculosis have been stopped, when the functions of the pancreas, liver and stomach have been restored, and the intestinal mucous membrane brought back to a normal condition, there may yet remain *tuberculæmia.* From time to time, fresh blood-poisoning may occur from the absorption of deposited tubercle; and, if there is any considerable quantity deposited, this chronic blood-poisoning may become a tedious and dangerous disease. The absorbed tubercle may be again deposited and again absorbed, and thus keep up a ceaseless repetition of morbid changes, somewhat analogous to those witnessed in pyæmia. Treatment directed to the blood-poisoning will, therefore, be imperatively called for in most cases of tuberculosis which have advanced to the stage of tuberculisation.

It should be our constant object by the use of antiseptics to stay decomposition and disinfect the products of decomposition *in situ* wherever they can be found and reached. It is with this view that I have always made creosote, carbolic acid, and turpentine inhalations a leading and essential part of my treatment of destructive lung disease. (See Index, "Antiseptics;" see also "Winter Cough," 3rd edition, p. 202.)

We have now a most agreeable and potent addition to our list of such remedies in Thymol and in the preparation of it called "Shirley's Thymolene."

As I may not find a better opportunity, I must not omit to mention in this context the pains which my talented and zealous friend, Dr. Clifford Allbutt, of Leeds, is taking to test the *antiseptic properties of climate,* and his interesting articles on Davos am Platz, in the "Lancet" of October 20 and 27, 1877, and June 8, 1878. In the latter article he says—

"In my former paper I gave some account of the sitution and climate of Davos, and of the curative work I saw in progress there. I did not hesitate to say that the results there obtained, especially in phthisis, seemed to me to be far in advance of those secured at any other health-resort, and I ventured to suggest an interpretation of the results apparently obtained. For many years I have held that the majority of phthisical patients die of septicæmia, and that the arrest of this daily repoisoning is a primary object of treatment. To reach, cleanse, and dress ulcers of the lungs by surgical methods seems impossible, and the effects of antiseptic inhalations are disappointing; if, however, there be *an antiseptic climate,* we may hope to counteract this secondary blood-poisoning, by sending our patients to live in it. Such antiseptic or aseptic climates are found in Switzerland, in Upper Egypt, and in other districts; and in the mountain air of Switzerland there is found also a tonic virtue which no doubt largely aids the physician in his work. The aseptic state of the air on alpine heights has been proved by Professor Tyn-

dall's experiments at the Bel Alp; but the Bel Alp, in common with most other alpine resorts, is insufficiently sheltered.

"In Davos alone, so far as we yet know, is found an air at once aseptic, bracing, and still. Upon these points I must refer the reader to my former paper. Very few English invalids have as yet found their way to Davos, and observations based upon the cases of English invalids are therefore not many." [See Dr. Dobell's "Reports," 1875, p. 243; and 1876, pp. 209—274.] "During the past winter my friend Dr. Rüdi has treated nineteen cases among the English; some of these were patients of my own, and others had been seen by me. Dr. Rüdi has kindly sent me notes of all. Of these nineteen, nine were cases of pulmonary disease.* Shortly put, they fared as follows :—

"Case 1.—Arrived on August 6th, 1877. Large cavity in upper lobe of left lung below the clavicle, infiltration of the whole lobe, and moist râles all over the left lung. Ordered to keep quiet, sit out in the fresh air, take abundant food and milk, and wine twice daily. Soon strength was restored, perspirations ceased, fever ceased, and appetite returned. At the end of December came on an attack of bronchitis in the whole left lung and renewed fever; this attack lasted four weeks. Recovery was satisfactory; respiration in the lower lobe became normal, and the cavity almost ceased to secrete. The surrounding tissue is still infiltrated, but shows no sign of irritation. The cavity is contracting. Next winter it will close. Although this patient has to return to Davos, good progress was made; for on arrival the disease was still progressing and liquefaction rapidly at work.

"Case 2.—Came with infiltration of the right apex and adhesions of the right lung to the diaphragm after pleuro-pneumonia. Perfect recovery in five months.

"Case 3.—Had a very large cavity in the right lung, extending into both upper lobes; moist râles all over the right lung, infiltration being extensive. In four months expectoration diminished to one-third, and strength and weight were gained. There was no inherited disposition. The process being now arrested and the cavity closing, recovery may be expected next winter.

"Case 4.—Convalescent from a pneumonia. No infiltration. Very feeble breathing, and imperfect expansion of left lobe. Douches used. Recovery perfect.

"Case 5.—Course exceptional. The appetite improved directly on arrival, sleep returned, fever and night-sweats ceased, and there was no diarrhœa. The state of the lungs also much improved. But this patient lost constantly in weight. He is a medical man, and says of himself, 'in every other respect I made a splendid cure, only the

"* These cases are purposely divested of personal data, as in a society like that of Davos each is known to all. Full particulars of the cases are, however, on record, and will be gladly supplied to any medical man who wishes for them."

constant loss of weight frightens me.' [I may add that this gentleman saw me late in the year, and I would have dissuaded him from going so late to Davos ; but he, knowing too well the probable course of his malady, stopped me, saying quietly he had determined to go. Both lungs were diseased, but I have not the exact notes at hand.—T. C. A.]

"Case 6 is one in which I (T. C. A.) am deeply interested. I shall give hereafter some extracts from letters this gentleman has written to me. I urged him decidedly to spend the winter at Davos rather than proceed to Egypt as he had intended. He had cavities at one apex and infiltration at the other. He had been a *poitrinaire* for four years, and when he reached Chur could scarcely walk up to his bed-room. His hectic was continuous and severe. Dr. Rüdi now reports of him:—'After staying here another winter he may return to England, although the infiltration at the back part of the left apex will not have fully disappeared.' I shall, as I have said, speak further of Case 6.

"The two following cases died.

"Case 7.—Had come to me (T. C. A.) for many months in 1876-7, in hopeless phthisis. Both lungs were extensively involved, and he had been repeatedly in bed with intercurrent pneumonia. There were other reasons for despairing of his state, among them great anxiety and pressure of business. As his life was very valuable I named to him last October the chance Davos might give him, and I put fairly before him the risks and the hopes. He decided to go on his own responsibility, and I described his going to his friends as a bold stroke for life. The patient wrote to me several times in good spirits, and assured me how much better he was. His appetite and his strength improved; his fever diminished, but *never ceased.* It was normal some days, but it would then run up in an evening to 100° to 101° F., and Dr. Rüdi always found the physical signs gaining ground rather than giving way, and wrote to me without holding out any hope. He died in the middle of February, but his departure from England is not wholly to be regretted, as he certainly suffered less than he had done at home.

"Case 8.—Came to Davos on December 20th, 1877, with both lungs infiltrated, the right more extensively. In right middle lobe a cavity. Fever considerable, 102° to 104°, commonly 103·5°. This never diminished. Death occurred on March 20th following. It was a lost case when patient arrived, and should have been sent much earlier in the year, if sent at all.

"Case 9.—Arrived August 6th, 1877, very ill. Insufficiency of mitral valves after rheumatic fever; there was also a catarrhal state of the right apex. This was therefore a complicated case. Patient gained weight, recovered well, and was sent home cured as regards his lung.

"The rest of the cases contain no phthisis."

While fully appreciating the lovely atmosphere of the Swiss Alps, the importance of antiseptics, and the value of high altitudes (see pp. 252-6), in the treatment of tuberculosis, I must confess that I am at a loss to see what these patients of Dr. Allbutt gained by their troublesome exile at Davos that we do not daily see obtained by suitable treatment nearer home.

Let us then return to the treatment of that more hopeful and curable condition—the initial loss of weight—which I have called "the true first stage of consumption," and devote to it a more detailed consideration.

Assuming that we have been able to determine that the function of the pancreas is defective,—that it is either depraved, deficient, or both, and that, as a consequence, normally pancreatised fats are not passing into the blood in proper quantities—what is to be done for the patient?

To answer this question in the simplest form first, I will assume—that the defect has been detected at its very onset, that there is no hereditary cause, that there is plenty of fat in the blood and in the systemic reserve to last for some time, and that, therefore, there is no *immediate* danger of tuberculisation,—that there is in fact nothing but a temporary defect in the functions of the pancreas, liver, and stomach.

Narrowed within these limits, the object of treatment is simple enough. We have nothing to do but to correct the defect in digestion and assimilation, by rectifying the functions of the pancreas, the liver, and the stomach. When we have done this, we shall have saved the patient from all the catalogue of miseries and difficulties included under the head of consumption. We shall have actually cured the disease in its "true first stage."

The means by which this simplest form of cure is to be effected, must vary with the circumstances under which the defective function of the digestive and assimulative organs has occurred. All depressing influences must be removed;—in some cases, in which the defect has arisen simply from some sudden depressing emotions, a sudden revulsion to joyous spirits will set all right again. The stomach, liver, intestines, and kidneys must be unloaded of their contents, and their secreting glands and cells freed from accumulations, and stimulated to fresh action; this is especially necessary with the liver and mucous glands of the intestines. The portal system of veins must be thoroughly relieved from congestion and obstruction, so that absorption by this route may be accelerated. The skin must be stimulated into action. The pulmonary circulation and the right heart must be freed from all impediments. Respiration must be set vigorously to work, and the diet must be regulated. In fact, every function of the body must be looked to and called to its account; and, above all, *everything must be done promptly*

with as little cost to the strength as possible. The true remedy for defective or arrested pancreatic function must then be applied freely and without delay, that is, cold dry air without wind, and plenty of it; exercise in such air, and exhilarating society. *Clear, dry, windless cold, out-of-door exercise, and good spirits, constitute the essence of stimulation to the pancreas.* It is astonishing how the organ leaps into action under these influences. Immediately it does so and the call comes for food, this should be supplied to any extent demanded, only providing that it must be unirritating and unstimulating in its nature, and that it must contain plenty of fresh fat, starch, and milk. No more striking example of this treatment and its effects can be found than is seen in America. The New Yorker who has jaded his appetite and his nerves, till he has lost all relish for his complicated table d'hote, has failed to answer to his "liquoring-up," sees no more charm in crowded rooms and gossip, and when even dollars cannot stir his gaunt and fatless body out of its apathy, at last consigns himself to the railway car for St. Paul. And there, in the cold, dry, clear, windless air of Minnesota, he wakes to the excitement of "the hunt," soon calls ravenously for food, longs for fat, and in a few weeks regains his wonted energies and relish for the world. I wish there was a Minnesota nearer home, to which I could despatch the jaded Londoner. It would be the saving of many valuable lives, who now dawdle over physics, quackeries, and intended changes of occupation, till the day for cure has past.*

Promptitude in this case is everything. I cannot too strongly describe the value of every day lost, after the flow of fat into the blood is interfered with; for every day brings nearer, at a rapidly increasing rate, the hour when successful treatment will have lost all its simplicity and become beset with difficulties.

Having now sketched, in as few words as possible, the essence of all treatment for the simple restoration of pancreatic function, I shall pass on to consider some of those *complicated difficulties* with which nearly every case is unhappily more or less beset, when it comes before the physician.

Hereditary predisposition. Of all "the difficulties" perhaps none is equal to that presented by hereditary predisposition. The hereditary defect may be in the pancreas itself or in the whole organism. In the first case we may start with a pancreas that was never made to act consistently and efficiently—that has been either defective from birth, or has become so in the course of development after the model of its ancestor,—a defect analogous to that in the cerebral organisation of persons who inherit insanity. Such a pancreas may have kept up

* See a very interesting little work on "Minnesota as a Home for Invalids," by my friend Dr. Brewer Mattocks, President of the Board of Health, St. Paul. Philadelphia: 1871.

fairly to a limited function—sufficient for the purposes of the individual while all went smoothly in its favour—but is inadequate to any emergency. Such an organ is easily turned from its duty, its secretion perverted or stopped by the slightest unfavourable influence. In the second case, the pancreas, *per se*, may be sound enough, but the individual may inherit such a susceptible sympathetic system that every passing ruffle in the affairs of life paralyses or perverts the pancreatic function. For, as I have already shown (p. 197), the pancreas, like the mammary, the salivary and the lachrymal glands, is singularly susceptible of sympathetic influences; and, as we see the secretion of milk in women perverted in a number of ways, gradually lost, or suddenly stopped, under the influence of defects of health, mental anxiety, excitement of the passions, emotional shocks, and a host of other influences, so do we see the secretion of the pancreas affected by similar and by other causes. Thus among the forms in which this organ may be affected by family predisposition, must be included as very important, *an undue susceptibility to sympathetic perturbations.* Without saying more, I think it must be clear that the difficulties introduced into treatment by hereditary predisposition are very great.

Another very serious difficulty is found in the state of the mucous lining and absorbent system of the alimentary canal. Before the existence of tuberculosis is identified, or at least before treatment is begun, the pancreas may have been performing its functions insufficiently or abnormally for a considerable time. In consequence of this, improperly prepared chyle has been passing through the digestive tract, irritating the delicate grandular structure of its lining and damaging its absorbing and secreting powers, the lacteals have been taking up imperfectly prepared fats, and have thus been irritated and obstructed. Added to all this, the stomach has often suffered by an attempt to supplement the deficiencies of the small intestines, by digesting an undue amount of albuminoid materials; and, by the forced absorption of fluid fats, the portal system has become overloaded, and the condition of the liver materially deranged. Hence it commonly happens that when cases of early consumption first come under our treatment, all this has to be set right before proper digestion and absorption would be possible, even if the pancreas could be made to resume its functions at once. In the mean time, *tuberculosis is going on, and may advance to the stage of tuberculisation before the normal absorption of fats is restored.* Yet the inconveniences attending this state are not very demonstrative. They may amount to no more than a certain sort of dyspepsia (see Mr. Hutchinson's observations at p. 195), and the general symptoms of disease are so unimpressive that the imminent importance of promptitude and energy in carrying out the necessary treatment is with difficulty impressed upon the patient and his friends,—who are very apt to think

that "too much fuss" is being made about what appears to them a trifling derangement of health. This places the greatest difficulty in the way of the conscientious physician.

Another and a most frequent "difficulty" in the way of adopting the means requisite for restoring pancreatic function is presented by the susceptibility of almost all delicate persons "*to take cold*" on slight exposure to changes of temperature. Fears of catarrh, of laryngeal sore throat, bronchitis, congestion of the lungs, pneumonia, pleurisy, rheumatism, constantly haunt the minds of the friends of these delicate persons, and stand in the way of the adoption of the means calculated to restore digestion, assimilation, and vigorous health. These fears are often well grounded. In a large number of cases, the patient is already suffering from more or less bronchitis, tracheitis, or pneumonic congestion when first brought under the physician's notice. All this has to be disposed of before any attempt can be made at enforcing those conditions which I have pointed out as the best restorers of pancreatic function. Indeed, when the colds have gone, the susceptibility to their return still stands in the way. In many other cases, though there may be no catarrhal complications present, the patient has been kept like a hot-house plant, in the hope of avoiding colds, or has been engaged in occupations requiring such a condition, so that it is only by months of the most cautious management that this hot-house life can be broken through without accidents. All this causes the most dangerous delay in adopting the main treatment of tuberculosis. Before curative treatment can be put into action, such a serious advance in the progress of disease may have taken place as to render such treatment impracticable.

Another of the difficulties constantly presented to us occurs in those cases where the defect in pancreatic function has been brought on by the mental or emotional shock of some great and irremediable anxiety or grief—blasted fortune—disappointed ambition—blighted love. What can the vigilance of friends or the skill of the physician do to remove such causes of disease? Yet these are among the daily vicissitudes of life, and we are called upon to treat the disease which they have caused, while the cause itself is exerting its full influence upon the case. Every medical man must have seen cases illustrative of the power of these vicissitudes of life to produce consumption, and of the rapid arrest or cure of the disease if happily the cause be removed in time. Cases, of girls suddenly falling into consumption when "crossed in love," and as suddenly recovering when the course was unexpectedly smoothed; of wives and husbands, in the midst of health, dying rapidly of consumption after the loss of those to whom they have been devoted; of strong men, vexed and worn by struggling with unconquerable difficulties, breaking up and dying of consumption when at last all hope was blasted—these are no dreams or inventions of the novel-

ist; but the stark realities of every day life. With these the physician has to deal, while the power to avert or to remove them is in other hands. (See p. 198.)

I need not do more than suggest the often insuperable difficulties placed in our way by a variety of *bad habits* to which consumptive patients in common with other persons may be addicted ; habits which in many cases have played their part in the causation of disease. It is hard enough at any time to induce persons to break bad habits, and even if they give all their energies to the task, time is necessary to success. How much more this is the case when the physical and mental powers are enervated by sickness, must be familiar to all.

Again, we must call to mind the numerous states of health and of disease which are found as *accompaniments* of consumption, and stand most seriously in the way of such treatment as I have pointed out for the restoration of pancreatic function. Among these none are more puzzling than climacteric changes, pregnancy, and the puerperal state; they are often insuperable bars to the adoption of proper treatment. So are we stopped by spinal affections, diseases of joints, and many other complications, which I need not further enumerate.

Unfavourable occupations and dwellings are at once the most common and the most serious of the difficulties which beset the curative treatment of consumption in all its stages. At page 197 I have placed first among the producers of defect in the function of the pancreas "any cause which for a prolonged period greatly reduces its activity, by diminishing the normal demand for carbonaceous matters in the blood," and I have described (p. 198) a large group of the admitted causes of consumption as "causes which act indirectly upon the pancreas, by diminishing the elimination of carbon from the blood and thereby reducing the normal call for the introduction of *fat* from the food into the blood. By these means the pancreas is kept in a state of inactivity and low nutrition, until, in time, its secreting powers are depraved or destroyed. . . It is evident that this heading will include a large number of the recognised causes of phthisis, *e.g.*, all those which diminish respiratory blood-changes for protracted periods, whether it be simply through deficient expansion of the chest, or through the hyper-carbonised condition of the air presented for respiration; and all causes of deficient excretion by the skin, especially if combined with defective respiratory action. It will also include cyanosis and chronic alcoholism." It will at once be seen how great is the difficulty presented by this class of causes of defective pancreatic action. The essence of the difficulty consists in the slowness of the process of deterioration. The pancreatic function is slowly and stealthily spoilt, the organ is, as it were, *cheated into inactivity* until it has lost the power to act.

What is to be done? Secretion of healthy and sufficient pancreatic juice is out of the question; the habit of producing it has been lost, the

organ is degenerated. Nothing can restore its healthy structure and functions without time; and time cannot be allowed, for every hour is precipitating the patient further into the depths of tuberculosis. If tuberculisation has not yet begun it is dangerously near; there is not a moment to spare. And we must remember what it is we want our patient to do, and the class of persons most often subjected to unhealthy occupations, ill-ventilated work-rooms, and unhealthy dwellings. We want to make our patient relinquish the pursuits upon which a family are depending for subsistence, leave a dwelling inseparable from those pursuits, and engage in a variety of hygienic measures calling for an unusual supply of the very means which we are proposing to take away his power to earn. And all this about an indisposition which appears to the patient and his friends utterly unimportant in comparison with the sacrifices we demand.

Although these remarks apply most especially to the case of the poor man or woman whom we wish to take from the workshop and the cellar, they are also applicable, to a large extent, to persons in the higher walks of life. Close offices, chambers, committee-rooms, law-courts, schools, libraries, museums, assembly-rooms, churches, and engrossing sedentary occupations, may imprison those who are far removed from the workshop or the cellar.

I have now pointed out so many serious difficulties in the way of adopting the best, easiest, and simplest means of stopping consumption in its "true first stage," that I will not stay to enumerate the many other obstacles which really lie in our path. There is one, however, which is so intimately related to those we have been considering, that I have already been obliged to refer to it more than once; and I must not pass it by, because it is the one which presents the leading indications for that treatment by which we can best escape from the defeat with which these complicated difficulties threaten us in practice,—I mean the *imminence of tuberculisation.*

There is a point in nearly every case, and it happens to be the one at which even the earliest cases of consumption are most often brought to the physician—a point when it is clear that tuberculosis has gone so far that, if tubercle is not yet formed, if albuminoid tissue is not yet invaded, it may be invaded—tubercle may be formed—at any moment. (See p. 161.)

This may be illustrated by the case of an oil lamp. If the oil is not renewed, we may watch it gradually disappear as the flame burns, till at length a moment arrives when the last drop has left the wick, and the wick itself begins to burn. The wick may be saved at any time previous to the disappearance of the last drop of oil, by supplying fresh oil to the lamp. I limit the "true first stage of consumption," then, to this period *before the wick begins to burn.* It is impossible to exaggerate the importance of this stage when rightly understood.

The normal supply of fat to the blood has stopped, histogenesis is at a standstill, the fat stored up in the tissues is being steadily consumed, every hour brings nearer the moment when this store will fail, and the first particle of albuminoid tissue will be yielded up for combustion. At that moment the die is cast, tuberculisation has commenced, and no one can prognosticate the end. Who shall say that the oil will be replenished before the wick is spoilt? We cannot blow out the lamp of life while we pour in fresh oil. It must continue to burn, and all the time required for restoring fats to the blood may be occupied in disintegrating albuminoid tissue and in the consequent formation of tubercle. (See pp. 161-2.)

For practical purposes, we cannot do better than keep before us this comparison of the lamp, for the main points of the simile are singularly near the truth; and they are so simple and familiar that they strike the mind at once, and keep the attention upon the leading indication for treatment, viz., *to throw fats into the blood* without a moment's delay.

It is in this emergency, in the hurry of this moment, that cod-liver oil is such a God-send to the patient. It is the kind of fat that can be hurried most rapidly into the pulmonary circulation ; it is the fluid oleinous kind of fat that can pass by the portal instead of by the lacteal route (see pp. 155-7); it is, as I have said in another place—and I repeat it, because I can find no simile so apt—like water to the uprooted flower. If you have a flower that has been uprooted and left out of the ground till it has begun to droop, you save it by plunging its roots into water, and you plant it in the earth when it has revived. Put it into the earth at once, instead of into water, and it will probably die before its withered spongeoles can convey nutriment to its vessels. On the other hand, if you keep it in the water after it has revived, instead of planting it in good soil, it will droop again and die for want of materials on which to live.

Patients in the True first stage of consumption advanced to near its end, when *tuberculisation is imminent*, must be treated like the drooping flower, only that oil must take the place of water. It would be absurd, if it were possible, to set about the restoration of pancreatic function at this juncture. This must be our next consideration ; but, first, we must adopt artificial means, by which the blood can be charged with fats much faster than the pancreas could be made to act, even if it were possible to adopt at once those hygienic means which I have pointed out as promoters of pancreatic function. (See p. 215.) But I have already shown that this is not possible, in consequence of the "difficulties" by which almost every case is beset. (See p. 216.)

It is in this place that *pancreatic emulsion of solid fat* comes to the rescue, and assumes such incalculable importance in the treatment of the true first stage of consumption. My experience of its

action is now so large, and my observations have been so cautiously and doubtingly made, that I dare to speak with a confidence which I trust may be distinguished from dogmatism. Pancreatic emulsion of solid fat is a natural substitute for the inactive or perverted pancreatic function. It supplies the lacteal system with solid fat in a condition fit for absorption, fit for transmission through the lymphatic glands, fit for combustion in the pulmonary blood for the protection of tissues, for histogenesis, and for general utilisation throughout the organism. By an artificial expedient we supply the missing elements of normal nutrition in a natural form. Thus time is gained, *the imminence of tuberculisation* is removed, and the means for the restoration of the normal functions of the pancreas, and of the other digestive and assimilative organs, can be adopted at leisure and in safety, under conditions favourable to success.

From these remarks it will be easy to appreciate the position which I assign to cod-liver oil and pancreatic emulsion of solid fat in the curative treatment of the early stages of consumption. I have attempted to depict the case of a person in whom tuberculosis is detected at its very onset, and I have endeavoured to show that in such a case the cure is so simply confined to the restoration of pancreatic function that there is no need for either oil or emulsion. But, practically, treatment is never commenced in this stage, and the means for restoring pancreatic function, that is the *cure proper*, cannot be safely attempted without the aid of the artificial introduction of fat—Cod-liver oil, if it can be taken, to supply what fluid fat it may by the portal route ; and pancreatic emulsion of solid fat to provide a normal supply of properly prepared fats by the lacteal route. (See pp. 157 and 210.)

But let us never forget that when diseased changes have been arrested and prevented by these means, when the patient is thus placed in a position of safety, he is scarcely better off than a leaking ship when by means of constant working at the pumps she has been brought safely into a harbour of refuge. Unless advantage is taken of this temporary security effectually to mend the leak, the ship will go down the first time the pumps are stopped. If means are not adopted to restore pancreatic function while the blood is being supplied with fat artificially, the patient will sink the first time an accident occurs by which the artificial supply of fat is stopped. It is through ignorance or neglect of this vital object of treatment that cases of early consumption, apparently progressing steadily towards recovery, so often relapse just when the improvement ought to have been made permanent, and thus the best chance of a radical cure is lost.

If means for the restoration of pancreatic function could always be adopted directly the need for them is discovered, and if we could be certain of speedy success when we employ them, there might be no particular need for any other remedy than cod-liver oil, at least for

those persons who are able to take it.* But, unfortunately, as I have attempted to show, it is very seldom that the means for the restoration of pancreatic function can be adopted at once, there are so many difficulties in the way; and still more unfortunately, the normal function cannot always be restored at all (see pp. 199, 200); and even when it can be restored, it often takes a long time to do it, and relapses into inactivity and perverted action are particularly apt to occur. Hence, under all circumstances, we are thrown upon the necessity of keeping up an artificial supply of fat to the blood for protracted periods of time. And *here it is that cod liver oil so signally fails.* Oil, even when it agrees and passes into the blood, does not completely represent the solid fats of the natural food, and therefore cannot permanently take their place. (See p. 155.) As a temporary substitute for natural fats introduced by the natural route, it answers admirably; but, sooner or later, in some cases very soon indeed, the portal system becomes choked and refuses to absorb more oil, the oil disagrees with the stomach, it rises, it spoils the appetite, and thus not only ceases to do good, but does positive harm by preventing the patient from taking as much food as the stomach might otherwise call for and digest. None of these disadvantages occurs with well made pancreatic emulsions of solid fat. The consequence is that an artificial supply of natural fat by the natural route can be kept up for an indefinite time, if required, while the appetite is usually improved and the digestion also; and, at the same time, a very large quantity of amylaceous food is rapidly converted into dextrine and sugar by the pancreatic action of the emulsion, and thus a most important assistance in the economy of fat is given, by the increased supply of carbon from the carbo-hydrates, at the same time that fat is being thrown into the blood by the emulsion. (See pp. 157 and 210.)

From the date of its first introduction in 1863, up to 1872, at the Royal Hospital alone, I had prescribed the emulsion in more than 6,000 cases, and since that time I have prescribed it *in all suitable cases* of consumption or of wasting from defective assimilation of fat that have come under my care, both at the Royal Chest Hospital and in private practice, and I have had numerous opportunities of witnessing its effects, when prescribed by my colleagues at the Hospital, and by other medical men, who have communicated to me their opinions on the subject or sought my assistance in consultation.

The general results of my thus extended experience have been confirmatory of the statement of my opinion, extracted at page 225 from the report which I published in 1867.

Happily, to my own experience may now be added that of thousands of my professional brethren in different parts of the world, by whom

* Of all the cases reported by me in which cod-liver oil and emulsion were both tried, the oil disagreed in about 52 per cent., whereas in the same cases the emulsion disagreed in less than 4 per cent. (See "Lancet," November 17, 1866.)

pancreatic emulsion and pancreatine are daily prescribed, and from whom I constantly receive testimony of its value.

I am informed on the best authority, that as much as sixty thousand pounds weight of the emulsion (made in London) has been consumed within a single year.

While there are certainly a few persons who cannot possibly take or assimilate the emulsion, although able to take cod-liver oil, they are but very few indeed, now that the emulsion has been made so perfect a preparation; whereas the number of persons who can take and assimilate emulsion, but not cod-liver oil, is very large. (See note, p. 223.) In either case—the rejection of cod oil or the rejection of emulsion—it is necessary not to be too easily persuaded by our patients to desist from prescribing the remedy.

I frequently find that patients, who assert that they cannot possibly, and never could, keep down the oil, will manage to do so when informed that it is the only thing that will stay the progress of their disease, and when assisted by being instructed in the variety of ways in which the oil may be disguised, and its assimilation facilitated. I may here point out that the addition of a dose of pancreatine to each dose of oil will, in a large number of cases, at once get over the difficulty in keeping it down—when that difficulty is that it eructates for some time after it is taken and is then rejected, not that it comes up directly. Many persons who have long taken quantities of oil without difficulty, but with scarcely any good effect, will gain flesh, and obtain all the advantages which assimilated cod-liver oil can give, if they add the pancreatine to their dose.

With the emulsion—patients, who after a first trial say they cannot take it, will nearly always find out that they can, when it is explained to them that there is nothing but pancreatic emulsion and cod-liver oil which can really stay the progress of their wasting; and when they are assisted by being shown the best way of taking the remedy and by having any faults in their primary digestive functions set right. The usual defect in primary digestion with such patients, is the combination of an excess of acidity with a feeble stomach, and this should be corrected by an effervescing draught of potass and soda, with citric acid, quinine and strychnia; or where there is much flatulent distension as well as epigastric pain soon after food, by a stomachic powder of bicarbonate of soda, bicarbonate of potass, powdered ginger, and powdered calumba, in half a tumbler of water, a quarter of an hour before the meal.

At the same time it is frequently necessary to unload the bowels, and to relieve the portal system, which has often been long over-loaded with cod-liver oil olein, absorbed by the veins. It is to be remembered that, whereas olein is, in some measure at least, taken up by venous absorption, the solid fats have to pass by the lacteal route; and thus,

EXTRACT FROM REPORT TABULATED FROM SCHEDULES, 1867. (Referred to p. 223.)

QUESTIONS.	1.—In how many cases of Consumption have you prescribed the Pancreatic Emulsion, prepared by SAVORY and MOORE?	2.—In what doses, at what interval after food, and for how long a time, did you administer it in each case?	3.—What is your opinion of its effects upon: — 1. Digestion? 2. Nutrition? 3. Weight of the patient?	4.—Have you found that patients can take the Emulsion when they cannot take Cod-liver Oil?	5.—What is your opinion of the effects of the Emulsion in:— 1. The true first stage of Consumption (Pre-tubercular)? 2. The stage of Tuberculisation? 3. The stage of Softening? 4. The stage of Excavation?	GENERAL REMARKS.
ANSWERS.	Up to this date, 1867, I have prescribed it in about 2,500 hospital cases, with results which, I think, may be fairly represented by those obtained in the 187 cases of which careful notes were taken, and which have been published in the "Lancet."	From one to four teaspoonfuls once or twice a day, from one to two hours after the principal meals, for periods of not less than eight weeks at a time, in milk, water, or ginger wine and water; a little brandy or rum usually being added.	1. Assists greatly in the digestion of fat and starch, and improves digestion generally. But it is necessary to use common sense in correcting any special derangements of digestion, which may be present, by other remedies. 2. Supplies fat for oxidation, and for histogenesis. 3. Maintains or increases weight according to the amount of weight previously lost, the power of taking food, and the quantity of Emulsion administered.	Yes; in 187 published cases, Emulsion agreed in 180; disagreed in seven. Cod-liver Oil agreed in seventy-five, disagreed in ninety-eight; was not tried in fourteen. In the 187 cases of which I have published results, no Cod-liver Oil was given during treatment with Emulsion, so as not to confuse the effects of the two remedies. But in daily practice, I recommend both Oil and Emulsion to be taken, if the stomach will bear them. The proportion of cases in which Emulsion agrees will not be as large as here stated, unless care is taken to correct obvious defects in digestion by other remedies.	1. That it materially contributes towards effecting a radical cure, as explained by me in my work "On the True First Stage of Consumption." 2. In forty-five cases, results measured by general symptoms, forty-four improved, one stationary; measured by physical signs, thirty-one improved, thirteen stationary, one worse. 3. In sixty-nine cases, results measured by general symptoms, fifty-nine improved, five stationary, three worse, two not noted; measured by physical signs, fifty-two improved, eleven stationary, four worse, two not noted. 4. In seventy-three cases, measured by general symptoms, fifty-five improved, three stationary, fourteen worse; measured by physical signs, thirty-five improved, twenty-two stationary, thirteen worse, three not noted. For details see "Lancet," Sept. 10, 1864; June 10, 1865; Nov. 11 and 18, 1865; Nov. 17, 1866.	In the true first stage, my experience is derived principally from private practice, such cases not often appearing at hospitals. Of the 2,500 hospital cases who have taken the Emulsion, many have been in an extremely advanced stage of disease; and in some of these the Emulsion has appeared to prolong life in a remarkable manner, being retained on the stomach long after all other kinds of food had ceased to be tolerated. Whereas oleinous fats, and especially Cod-liver Oil, are absorbed into the blood through the portal system, and serve an important purpose by rapidly presenting themselves for combustion and histogenesis; they do not, and cannot, take the place of the solid fats—rich in margarin and stearin, fusible at higher temperatures than olein, and less easily oxidisable—which can only be absorbed by the lacteal system after pancreatisation. This accounts for the remarkable stability of the improvement which accompanies and follows the administration of Pancreatic Emulsion, after Cod-liver Oil has been given without success, or with very evanescent success.

when substituted for oil, in the form of pancreatic emulsion, they relieve the tax upon the choked-up venous system. (See p. 156.)

Then, the period after a meal at which the emulsion is taken may be varied, to meet the differences in the rapidity with which different stomachs dispose of their contents. Many who cannot keep the emulsion comfortably down when taken two hours after food, can do so if they take it from half an hour to an hour after. This particularly applies to young children. They generally manage it best within half an hour of a meal; and when their diet consists of farinaceous foods and milk it is best to mix it with a portion of the food.

With regard to menstruum:—milk is certainly the nicest and best, where milk agrees with the stomach and taste; but many persons have an old-standing aversion to milk, or have long ceased to be able to digest it comfortably. In such cases it is absurd to insist on the emulsion being taken in milk. The best substitute, and indeed one of the best menstrua for pancreatic emulsion, is thin water-arrowroot, or barley-water. The emulsion mixes well with it, and by converting the starch into glucose, gives it a pleasant, nutty sweetness. Many persons, however, prefer the emulsion mixed in water to about the consistence of new milk, and then flavoured with wine, especially Tokay,* ginger wine, or orange wine. In whatever menstruum it is mixed, it should not be made much thicker than new milk, and I advise a sufficient quantity of wine, brandy, rum, orange-brandy, ginger-brandy, or clove-brandy to be added, to give it a decidedly cordial character both to the palate and to the stomach.

One of the most important points in getting the emulsion well taken, is to have it *smoothly* mixed in the menstruum; this is easily done by putting the emulsion first into a cup, or glass, and then adding the menstruum little by little, and beating them together with a spoon, till the whole is made of about the consistence of oil, or of good cream; after which any quantity of fluid may be mixed simply by stirring; but if the emulsion is put in a lump into the menstruum, it may be difficult afterwards to get it smoothly mixed, especially in cold weather. The mixing may be facilitated by having the cup or glass made warm, or the chill taken off the menstruum, but it is not well to make it decidedly hot.

Some patients have asked to be allowed to take the emulsion "as it is" with some brown sugar over it, and declare that they like it in that form. This is better than not taking it at all, but I prefer its being mixed into a more fluid state. A good many mothers have told me that their children take it spread on bread, instead of butter, and sugared. This is not a bad way of giving it, because

* The "Imperial Tokay" of Max Greger, is an admirable menstruum for Pancreatic Emulsion, and, in itself, is a fine restorative.

in masticating it with the bread it gets well mixed with the food and saliva; I may mention also, that treacle-posset, after it has cooled, is a very good menstruum, and pleases children when they are fond of sweets.

One more detail is worth alluding to, viz., to order a biscuit or a piece of bread to be eaten after the dose, so as completely to clear the palate of its taste and influence.

It is of the utmost importance to impress upon patients that cod liver oil and pancreatic emulsion are *not to be regarded as medicines, but as articles of diet—Medicinal foods—without which they, in their state of health, will as surely starve as persons in health would do if deprived of the most nutritive part of their food.* They should be ordered to be weighed whenever they leave off either the oil or the emulsion, and to weigh every week, to see if they lose or gain. So long as they do not lose any weight they may desist from their dose, but must at once return to it if there is the slightest diminution in weight. I always order patients on leaving off either oil or emulsion, to make an equivalent increase in the quantity of fat they eat, and to see whether by this means their weight can now be maintained, and thus to learn whether the power of digesting and assimilating a sufficiency of fat *without artificial assistance* has been restored.

A point of great interest and of vital importance in treating all wasting diseases is to ascertain what was the normal average weight of the patient before any wasting set in. (See Part I., p. 10.) It requires considerable care and circumspection to avoid being deceived on this matter by the patients themselves, who almost always date their wasting from a period considerably later than that at which it actually commenced. They attach no importance to the loss of weight till it begins to tell upon their personal appearance, either in face or figure. Yet, in tolerably plump persons many pounds may be lost before the change is obvious. In cases which do not start from a definite acute illness, but simply depend upon failing digestion and assimilation, the first few pounds are generally lost very gradually. The best plan is, if possible, to ascertain what is the heaviest weight ever reached in the patient's previous lifetime (See Table 1), or when this cannot be done, to catechise the patient very closely with regard to the year or two preceding the date at which they fix the beginning of loss of flesh. Supposing the patients say that loss of flesh began twelve months ago, they should be asked, "Were you just as fat and just as firm in flesh at that time as you were six months before that?" The answer will generally be, "No, not quite;" and then they should be carried back another and another six months, until a time is reached at which they have not the least doubt, either from actual weighing or from general impression, that they were at their full weight and full firmness. It must be remembered that loss of firmness, which is, chiefly, loss of solid fat replaced

by olein, often precedes actual wasting by a considerable period. (See p. 156.) This is a change which careful and watchful mothers will observe in children as the first sign of degrading health, and those who are judicious enough to take warning by it, will often, by change of air and of diet, save their children from any further mischief. Increase of fat with loss of firmness in the fat is a certain sign of improper feeding or of impaired assimilation, and such fat will disappear, like snow before the sun, on the occurrence of the first attack of even a trifling acute disease.

Having ascertained as clearly as practicable about the average weight of the patient when in normal health, the next thing is to endeavour by every possible means to recover that weight completely. Where the loss has been considerable, this may prove a most difficult task, and when the loss has gone beyond a certain point, it may be quite impossible to recover the original weight. (See p. 155.) In very extreme cases of exhaustion and emaciation, with stomachs so irritable that nothing can be kept down, I have been able to put the first stop to the downward progress by mixing pancreatic emulsion with nutritive enemata, and it has been administered in this way till sufficient restoration of strength and of digestive power has been gained to allow of food and medicine being given by the mouth. And in cases where the stomach tolerates food and medicine, but in which the loss of weight has been very great, it is well to supplement the treatment by the stomach by extra doses of emulsion given in enemata ; and thus to get a larger daily supply of fat into the system than the stomach could possibly take.* In this endeavour to restore the normal weight, the influence of exercise and of temperature must always be kept before the mind ; a certain amount of exercise and a certain coldness of temperature may be essential to keeping up the appetite and digestion, without which no progress can be made with the feeding and fattening process ; but all exercise involves waste, and all cold involves waste ; therefore, so long as the weight of the patient is in any degree below the normal average, exercise and cold must be kept at the lowest point consistent with maintaining appetite and digestion. A little over-

* "NUTRITIVE ENEMA.—Take of cooked beef or mutton finely grated ¼ lb. ; pancreatic emulsion (Savory and Moore's) 1 oz. ; pancreatine powder (Savory and Moore's) 20 grains ; pepsine (Porci) 20 grains. Mix the whole in a warm mortar quickly and add brandy one table-spoonful and enough warm water to bring the mixture to the consistence of treacle. Inject from an elastic enema bottle, as quickly after the mixture is made as possible, and let it be retained. When nutriment is given in enemata the quantity should not exceed from 2 to 4 oz., and the temperature should be about 80°. The bowel should be first washed out with half a pint of warm water.—An elastic bottle holding the required quantity is better for nutritive enemata than the ordinary enema syringe. They should be given while the patient is lying on the back with the hips raised on a pillow." ("On Diet and Regimen in Sickness and Health," Sixth Edition.)

fatigue every day may entirely frustrate all attempts to recover weight, and a little excess of demand for heat-producing materials to maintain the temperature of the body may just consume all the little surplus we are sending into the blood. Here lie our great difficulties in the restoration of weight, the rocks upon which so many promising cases are "wrecked in port." (See pp. 157-9 and 254-6.)

I must not neglect this favourable opportunity to speak of the nature of the *Diet* which should be ordered in consumption. The objects to be kept in view are very simple:—

1st. To give the diet in forms that shall be unirritating to the alimentary mucous tract.

2nd. To supply enough carbon and hydrogen *in other forms than that of fat*, to combine with the 30 oz. of oxygen introduced every twenty-four hours by respiration, and thus to provide for the evolution of the 10,000 British units of heat per day, which I have shown to be required as sensible heat and mechanical force. So that whatever fat gets into the blood by natural or artificial means may be available for those purposes in the organism, for which nothing but fat will suffice.

3rd. To supply plastic material in sufficient quantity for the renovation of the albuminoid tissues.

4th. To supply enough fat, already pancreatised, for histogenesis and the protection of the tissues against oxidation.

5th. To supply mineral matters and water.

These conditions will be found, as nearly as practicable, provided in the Diet (Table I., p. 230), which I have employed both in hospital and in private practice, with unmistakeable advantage to the patients, for many years.

The 2nd Diet (Table II., p. 230), provides for a large amount of the carbon from the albuminoid articles of food, and is intended for those cases in which albuminoids are more easily digested than carbo-hydrates. In some consumptive persons, when the defect or deficiency in the supply of pancreatised fats to the blood first occurs, there is an immense effort made by the nutritive powers to save the patient from the disastrous effects of this loss of a direct supply of fat. The appetite for all other foods, and especially for albuminoid foods, becomes ravenous, and, in those who have the means of freely supplying this demand, carbon is procured by the disintegration of albuminoids in sufficient quantity to replace that which ought to be supplied in the form of fat, while the fat absorbed from the tissues is made to suffice for those histogenetic processes which cannot be carried on without the help of fat. This acts as a sort of temporary cure; and I have taken advantage of the expedient, thus adopted by nature, in the treatment of certain appropriate cases, by supplying them with a diet in which the carbon, which in a normal diet is supplied by the fats, is made up from the excess of albuminoids. This, however, can only be a provisional remedy for the

DIETS FOR CONSUMPTION.—TABLE I.—CARBO-HYDRATE.

Oz.	Food for 24 hours.	Analysis.			Carbon from		Total Carbon.
		Plastic.	Fat.	Saccharine.	Nitrogenous.	Non-Nitrogenous.	
6	Cooked Meat	1·350	·534	..	0·732	0·420	1·152
10	Bread	1·000	·070	4·530	0·540	1·930	2·470
8	Potatoes	0·136	..	1·840	0·072	0·760	0·832
2	Sugar	..	..	1·800	..	0·848	0·848
2½	Milk 20 fluid ozs.	1·000	0·700	0·840	0·540	0·900	1·440
2	Liebig's Food for Infants ..	0·300	0·116	1·064	0·162	0·558	0·720
6	Farinaceous Foods	0·300	0·020	4·900	0·160	2·190	2·350
..	Fermented Liquors*	..	..	..	..	1·000	1·000
2	Pancreatic Emulsion	..	1·000	..	..	0·740	0·740
37½	Totals	4·086	2·440	14·974	2·206	9·340	11·552
	Deduct Carbon from Non-pancreatised Fats as waste ..				..	..	0·945
	Total available Carbon				..	..	10·607

* Either—Half a pint (Imperial) of Port, Sherry, or Marsala; or, one pint of Burgundy, Claret, or other similar Wine; or, one pint of good Ale or Stout; or, a quarter of a pint of Rum, Whisky or Brandy, diluted with one pint of water.

DIETS FOR CONSUMPTION.—TABLE II.—ALBUMINOID.

With this Diet Hydrochloric Acid and Pepsine should be given to assist in digesting the very large quantity of Plastic Matter.

Oz.	Food for 24 hours.	Analysis.			Carbon from		Total Carbon.
		Plastic.	Fat.	Saccharine.	Nitrogenous.	Non-Nitrogenous.	
8	Cooked Meat	1·800	0·712	..	0·976	0·560	1·536
6	Pigeon or Game	1·300	0·830	..	0·740	0·090	0·830
3	Dried Fish	1·310	0·055	..	0·710	0·035	0·745
1	Cheese	0·308	0·256	0·024	0·166	0·200	0·366
3	Vermicelli	1·425	..	1·164	0·777	0·516	1·293
4	Bread	0·400	0·030	1·810	0·220	0·770	0·990
6	Rice or Arrowroot	0·300	0·020	4·900	0·160	2·190	2·350
3	Sugar	..	..	2·700	..	1·270	1·270
2½	Milk 20 fluid oz.	1·000	0·700	0·848	0·540	0·900	1·440
6	Green Vegetables	0·060	0·012	0·468	0·030	0·204	0·234
..	Fermented Liquors*	..	..	..	..	1·000	1·000
2	Pancreatic Emulsion	..	1·000	..	..	0·740	0·740
43½	Totals	7·903	3·615	11·906	4·319	8·475	12·794
	Deduct Carbon from Non-pancreatised Fats as waste ..				..	..	1·410
	Total available Carbon				..	..	11·384

* Either—Half a pint (Imperial) of Port, Sherry, or Marsala; or, one pint of Burgundy, Claret, or other similar wine; or, one pint of good Ale or Stout; or, a quarter of a pint of Rum, Whisky, or Brandy, diluted with one pint of water.

defective supply of fat; and unless the normal supply is quickly restored, the patient makes a disastrous descent. For, first, there is overtax of the digestive and assimilative processes by the inordinate consumption of one element of diet; then there is overtax of all the vital processes necessary to obtain, by a complicated route, the carbon from the albuminoid materials; and, lastly, there is the overstrain upon the organs of excretion in getting rid of the excessive waste of nitrogenous matters. And, after all, how totally different is the result from that constant and abundant flow of fat from the lacteals into the right heart, which occurs in the normal state, keeping the delicate albuminoid tissues of the lungs constantly bathed in fatty blood, and thus protecting them from the influence of oxygen. (See pp. 148 and 161-9.)

Nevertheless, this inordinate consumption of albuminoid food does take place in certain cases, with the effect of deluding the observer by keeping up the weight of the patient. In a certain number of picked cases I have found this albuminoid diet of great service, but for general use I much prefer the carbo-hydrate diet (Table I.).

A third diet (Table III., p. 232), consisting of 78 oz. of milk, 6 oz. of arrowroot, and 1 oz. of pancreatised fat yields nearly the same results as the the former two, except that the quantity of carbon reaches only 8 oz., or enough to evolve 7,250 British units of heat. The quantity of carbon, therefore, is not up to the required amount, but it is a diet only intended for a very temporary purpose, viz., until solid food can be borne by the stomach. During this period of treatment the patient may be precluded from engaging in external mechanical work, and some sensible heat may be artificially supplied. (See p. 159.) I have used this diet in some peculiarly unmanageable cases with very excellent effect, giving the milk in divided doses every four hours, or in half the quantity every two hours, and mixing the arrowroot with it. I find that 78 oz. of milk per twenty-four hours is the largest quantity that the adult stomach will take, and in the average of cases of irritable stomach it is difficult to get beyond 60 oz. The emulsion is best mixed with a little water and given directly after the dose of milk and arrowroot, not mixed with them. To this diet brandy may be added in some cases, and thus the quantity of carbon may be increased.

In these "DIETS FOR CONSUMPTION" it is assumed that no fat is assimilated except that artificially pancreatised.

In Table I. The required amount of carbon is supplied by an excess of carbo-hydrates.

In Table II. The required amount of carbon is supplied by an excess of albuminoids.

In Table III. The amount of carbon is kept low, because it is only intended as a temporary diet to be used during periods of rest in a warm room. The arrowroot and some of the fat of the milk are pancreatised by mixture with the "pancreatic emulsion."

DIETS FOR CONSUMPTION.—TABLE III.—TEMPORARY FLUID DIET.

Oz.		Analysis.			Carbon from		Total Carbon.
		Plastic.	Fat.	Saccharine.	Nitrogenous.	Non-Nitrogenous.	
78	Milk 78 fluid oz. containing 10 oz. solid	3·900	3·730	3·276	2·106	3·510	5·620
6	Arrowroot	0·300	0·020	4·900	0·160	2·190	2·350
2	Pancreatic Emulsion	..	1·000	..	..	0·740	0·740
18	Totals	4·200	4·750	8·176	2·266	6·440	8·710

This diet is to be given as follows:—

8 ozs. of Milk and 1 oz. of Arrowroot every four hours (six times in twenty-four hours) for twenty-four hours.

10 ozs. of Milk and 1 oz. of Arrowroot every four hours for twenty-four hours.

12 ozs. of Milk and 1 oz. of Arrowroot every four hours for twenty-four hours.

13 ozs. of Milk and 1 oz. of Arrowroot every four hours for twenty-four hours.

The last quantity to be continued until solid diet can be borne by the stomach.

One-third of an oz. of Pancreatic Emulsion is to be mixed with a little water, or with a portion of the milk, and given directly after each dose of Arrowroot and Milk, not mixed with the whole bulk.

I cannot speak too highly of the following "*Special Nutritive*" as a means of feeding and fattening patients unable to take a sufficiency of solid food. I have seen emaciated patients grow fat upon this alone.

Special Nutritive.—Beat up an egg, both white and yolk, quite smooth and free from stringy particles, stir it well into half a pint of hot milk in which enough arrowroot has been boiled to make it about as thick as cream; add a wine-glassful of sherry or a table-spoonful of pale brandy, five grains of pancreatine powder (Savory and Moore's), and some fresh nutmeg and sugar; mix all thoroughly by pouring from cup to cup. On this food alone, repeated every four hours, a patient can be well supported for a considerable time. A person living upon this diet alone should drink fresh made lemonade when thirsty.

The use of Diastase in the form of Peptodyn (Pancreatine Pepsine and Diastase) or in that of powdered malt, or of malt extract will assist in the supply of fat-forming and heat-producing materials as recently impressed by the "Brit. Med. Journ.," July 13, 1878, in the following editorial note:—

"Mr. J. J. Coleman, having lately been a sufferer from a serious illness, a prominent symptom of which was inability to digest food, was ordered to take a malt extract. The usual dose—a wine-glassful twice or thrice a day—was taken with the result that food, which had hitherto escaped undigested, was assimilated, and the power of producing animal heat and storing up fat was increased. Other persons—thin, cold, or aged—were induced to become the subjects of experiment, and reported that it was far more sustaining than most alcoholic liquors. These

facts led Mr. Coleman to make an extended series of experiments, which he has embodied in a paper read before the Glasgow Philosophical Society, and which is abstracted in the 'Chemist and Druggist' of May 15th. Analysis of the extract proved that, in composition, it closely resembled other malt liquors, differing chiefly by yielding a rather larger percentage of extract. In appearance it resembles porter. An explanation of the sensation of being lifted from a feeling of semi-starvation to the condition of being effectually warmed and nourished, seemed to be required. . . . Now the diastase contained in malt is able to convert the starch of four or five times its weight of barley into soluble substances. Starch forms a very large proportion of our daily food, and it seemed possible that, by rendering this more soluble Hoff's malt extract might produce the effects which had been experienced. Experiments were, therefore, instituted to decide the question; and it was conclusively proved that all malt liquors exert a more or less powerful solvent action on bread and other starchy foods, and that Hoff's liquid possesses four times the power of Burton ale, and half as much again as London porter. This action is so great as fully to account for the good effects observed to follow their use."

A most important question arises in the treatment of consumption in all its stages, viz., the amount of fresh air and of exercise to be allowed or enforced. The progress of the patient, upwards or downwards, will often be determined by the decision of the physician on this point. (See pp. 228-9.)

Experience has taught me that in coming to a decision on this vital question, the physician ought to be guided by physiological reasoning, and that his advice will be right or wrong just in proportion as he is shrewd and discriminating in measuring the exact condition of his patient, and in the application of his physiological knowledge to each particular case. The following leading facts must be borne in mind:—

1. That the labour of walking one mile (if the weight be 150 lbs.) equals 17·678 foot-tons, equivalent to 51·295 British units of heat, the evolution of which requires the oxidation of 0·0566 oz. of carbon.

2. That every 1 oz. of oxygen will combine with 0·375 oz. of carbon, the combination being accompanied by the evolution of 339·84 British units of heat, equivalent to 117·12 foot-tons of mechanical force. (See "Altitude," p. 252.)

3. That fat is essential to histogenesis and to the protection of the tissues from oxidation; that increased muscular exertion necessitates increased oxidation, and requires increased histogenesis.

If the patient has lost enough fat to make an impression upon his strength, that is to say, if heat is being generated by the combustion of materials which ought to be available for mechanical force and histogenesis, it is madness to make any avoidable demand upon either

mechanical force or histogenesis until all, and more than, the deficient fat has been restored; and it is equally mad to increase the supply of oxygen to the blood beyond the smallest quantity consistent with life. If, while pouring fat into the blood, we could actually stop the supply of oxygen and stop the demand for renovation of tissue, mechanical force, and sensible heat, without arresting those phenomena upon which the maintenance of life and nutrition depend, we should have the patient as much under our control as we have the lamp, which we blow out while recharging it with oil and thus save its wick from combustion. (See p. 220.)

But although we cannot do this in the case of the patient, our object should be to approach as nearly to this condition as is consistent with the maintenance of life and nutrition.

Another question which is in my opinion of the gravest importance, and which according to my experience is generally decided in the manner exactly contrary to what is best, is that of the administration of iron. My reasons for objecting to iron in tuberculosis have been given at pages 129, 130, and I need not repeat them here.

It has been under the dictation of the principles already stated, and further detailed under the head of climate and of altitude, that I have been accustomed to prescribe REST IN PULMONARY CONSUMPTION, whenever tuberculisation has commenced or is already imminent. First, rest of the whole body; secondly, rest of the whole of both lungs; thirdly, rest of one lung; fourthly, localised rest of diseased portions of lung.

It was in November, 1872, that, at my request, Mr. Heather Bigg made the first "lung-splint," and I asked him to call it at once by this name, as conveying an unmistakable explanation of its objects. He arranged it with his usual skill; it answered admirably, and he has made many others since, adapting each to the special requirements of the case. Before this, I had been accustomed to procure rest for portions of lung by other devices, principally by keeping the arm of the affected side flexed upon the walls of the chest, so as to restrain expansion by its weight and by the absence of muscular action; and it was the difficulty of sufficiently localising the pressure by these means to suit special cases that led me to suggest the "lung-splint;" but, whether it be partial rest of the whole of one lung, or more complete rest of a portion of one or both lungs that is desired, the greatest caution is necessary; because whatever local means secure rest to one part of the lungs, throw extra work upon the other parts, and may, therefore, easily do more harm than good.

For this reason, I have always used the greatest circumspection in selecting cases for this kind of treatment, and I trust that, if anyone is led to follow my example in one part of this treatment, he will

most scrupulously do so in the other. With this precaution, nothing can be more satisfactory or more common sense, in the treatment of lung-disease, than the use of lung-splints, bandages, and the like; whereas without it nothing can be more foolish.

Those who enter into my views as to the importance of avoiding disintegrating oxidation of lung-tissue in consumption (pp. 166-9), will at once see how dangerous may be the effect of throwing exaggerated action upon one portion of lung by the attempt to control the action of another portion. If the disease by which the one portion was damaged had been purely local, there would be no danger worth considering, in taxing the sound parts to save the unsound; but, when we consider consumption as "an abnormal physiological state" of the constitution, exposing all parts of the lungs to destructive changes, we at once see how much caution is required in determining that it is safe to throw exaggerated action on parts which may be only needing this excitement, to subject them to the same destructive changes as those taking place in the portion of lung we wish to save by localised rest.

The rules, for *for the cautious application of localised rest* in lung-disease, which I recommend, as dictated by a consideration of the nature of tuberculosis, and justified by the results of my own practice, are as follows:—

1. If one lung, or a portion of one lung, or a portion of each lung, has become diseased, under circumstances which make it certain that there is no constitutional cause of lung-disease, then it is safe to secure localised rest for the diseased part, and to throw the extra work upon the sound parts; but even then it is necessary to be cautious that the extent of lung so rested is not too large in proportion to the extent of sound lung upon which the extra work is thrown. If there is any question about this, rest of the whole body must be secured in addition to the localised rest of lung, so as to save the sound lung from as much work as possible.

2. If there is a constitutional cause of lung-disease, but only a small area of lung at present suffering, and that in the upper lobes, while there is a capacious chest with large areas of lung in the lower portions quite sound and insufficiently used; then it is safe to secure localised rest for both upper lobes, and to make the lower portions do a fairer proportion of the work; but even under these circumstances, the respiration should be kept at as low a point as practicable. A case illustrative of this rule has just occurred to me. A fine young man, with a very capacious thorax, who has practised all sorts of gymnastic exercises with his arms while restricting the lower parts of the chest by dress, has thus acquired a habit of breathing almost entirely with the upper portions of the lungs.* He has a tuberculous family history,

* As those do who wear tight waists at lawn tennis.

and, after foolish overtraining, by which he reduced his flesh considerably, he overtaxed his lungs in a race, and he has since become the subject of partial consolidation of the apices and recurrent hæmoptysis. Finding that he has large tracts of scarcely utilised lung at the lower parts of the chest, I have not hesitated to get Mr. Bigg to apply mechanical restraint, by means of lung-splints to both upper lobes ; but I have, at the same time secured rest for the whole lungs by sending the patient on a long sea-voyage to a warm climate, under careful watching against over-exercise. (See pp. 250-3.)

3. If a portion of lung has become disintegrated, under the influence of constitutional causes, and remains obstinately unhealed after all constitutional symptoms have been arrested, and, for some time past, no other portions of lung have shown a tendency to yield,—then I think it is quite safe to secure localised rest for the disintegrated portion, so as to give it a fairer chance of healing ; while an amount of air and exercise may be allowed to the patient, for the purpose of improving his reparative powers, which could not be permitted while the damaged lung was exposed to the same amount of action as the sound parts. But even here the utmost caution is required not to carry the exercise beyond a very limited amount.

4. If the constitutional tendency to lung disease—"the abnormal physiological state"—is strong, and signs of impending mischief in the lungs are scattered, no localised rest should be attempted, but every means should be brought to bear upon the important object of maintaining respiration at its lowest point, consistent with life and nutrition, until the constitutional tendency has become passive and the local symptoms have been removed. (See p. 211.)

In conclusion, to prevent misapprehension on so vital a point, let me remind my readers that, in urging "the importance of rest in consumption," I am referring to cases in which the lungs are already damaged, or in which the constitutional disease has declared itself in sufficient force to render tuberculisation imminent. If the symptoms are only those of commencing tuberculosis, and no reason or sign is discoverable which justifies the suspicion that tuberculisation has commenced; if a sufficiency of fat remains without calling upon the albuminoid tissues, the principles of treatment are quite opposite to those above detailed. It must be admitted that the proper regulation of this matter is one of the greatest trials of the astuteness of the physician, and it is almost impossible, unless he can make the patient and his friends comprehend its meaning and importance.

But not less does it test the skill and judgment of the physician to decide upon the moment when these restrictions upon fresh air and exercise ought to be removed. (See pp. 210, 211.) The argument so often used when a patient appears to be "doing well," that "it is the best to let well alone," may be fatal if applied to this

case. The very fact that he is doing well" may be the sign that he must not be "let alone;" that he is now in a state in which it is safe to make a call upon his mechanical force, to accelerate histogenesis, to supply fresh oxygen—in a word, to set about the restoration of active nutrition. And then, again, how scrupulously these new tasks should be set; how carefully watched in their effects, lest even now they cannot be continued with safety! On the first sign of their being badly borne, they should be moderated or promptly stopped.*

Quinine is one of our most valuable tonics in consumption; it generally increases the appetite and thus promotes the ingestion of an in-

* It is only fair to Dr. M'Crea, of Belfast, to insert in this place his letter to the Editor of "The Lancet," October 25th, 1873:—

"Priority in the Treatment of Phthisis by Rest.

"Sir,—I have read in the current number of your journal an able paper by Dr. Berkart on the importance of securing rest of the affected portion of lung in phthisis. As I have been working at this for several years, I ask to be allowed to say a few words on the subject.

"Whoever reads Dr. Berkart's paper will see that it might have been written almost in its entirety years ago by anyone to whom the idea of resting the lung might have occurred. Almost the whole paper consists of theoretical considerations, very ably put, it is true, but still theoretical. The same line might have been taken by myself more than three years ago; but I preferred to put the matter to the test of actual observation. This is customary in other branches of science, and an experimenter or observer to whom a "happy thought" may have occurred is usually expected to make his experiments or observations on this a prominent feature of anything he may publish. Such is not the course which Dr. Berkart has adopted in his ingenious paper; but he has adopted another, which gives him an unfair advantage over more patient observers. The corrected proof of a paper of mine is in the hands of the printer of the "Dublin Monthly Journal"; and so anxious have I been to fully test the method that I take no account of any cases which have occurred since the beginning of the present year. Although, however, I have not formally published, I have exhibited the treatment over and over again to several members of the profession here, including my colleagues at the Belfast Dispensary. I also mentioned what I was doing to several members of the profession that I met at the Birmingham meeting of the British Medical Association. And at an early stage of my investigations I took the opportunity, in the course of a discussion on another subject at the Ulster Medical Society, to state the high opinion which I felt myself forming of the advantages of rest in some lung affections. This was all that I deemed justifiable without extended observations.

"I think, then, that in putting theory to the test of practice, my claim to priority can hardly be disputed; for the sentence or so in which Dr. Berkart tells what he has actually done is so very vague as to give no ground whatever on which one could form an opinion as to either his method or his results. As to priority in suggesting the idea, if it be said that my not having published before anyone else in the "Journal" destroys my claim, the same remark will apply to Dr. Berkart; for, however long and however publicly he may have been giving expression to his line of thought in the presence of all comers to the institution with which he is connected, he also has been anticipated in the matter of full publication. At a meeting of the Royal Medical and Chirurgical Society, November 12th, 1872, in a discussion on Dr. Ransome's paper on the 'Respiratory Movements of Man,' Drs. Anstie and Powell were reported in the

creased quantity of force-producing materials. If it interferes with appetite it must not be given. It assists in preventing and arresting that terribly distressing source of waste—excessive perspiration. (See p. 151.) None of the objections to the use of iron apply to that of quinine. (See p. 129.) Given cautiously, with an acid or an alkali according as one or the other is indicated by the state of the digestion, it is of infinite service in nearly every stage of consumption, and as a general rule, the patients who have once taken it, crave for its continuance.

From what has already been said of the importance of all means of arresting waste of tissue, and also of the assistance to be obtained from antiseptics (see p. 212), it will be readily understood that *I attach great value to Arsenic* in the treatment of loss of weight and lung disease. I have always thought that (probably in consequence of the form in which it was written) too little attention was given by the profession to a little book published in 1857, by "Dr. John Gardner, Editor of Liebig's Letters and Lectures on Organic Chemistry," in which he says :—"The great remedy upon which, after much experience of its effects, I rely to arrest the formation of tubercle, is the arseniate of soda, the mildest and most manageable form of this agent. It should be administered in such a manner as just to tinge the system, and this may be done securely and safely, inasmuch as the symptoms of its action are definite and immediately become obvious. We have not in the whole list of our Materia Medica a safer or more manageable remedy, and I have had sufficient experience to recommend it to the adoption of the profession. But I respectfully warn all who would make trial of it, to satisfy themselves of the exact nature of the case, to be sure it is tubercular consumption they are treating, before pronouncing for or against it. Most cases of tubercular consumption require to be carefully watched, and brought under the influence of this remedy several times at intervals, and the result will, I feel con-

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journals as having made observations showing that they had a notion of the importance of restraining respiratory movement in phthisis and other chest affections.

"In my paper above alluded to I have almost excluded theoretical considerations; and if this letter had been shorter I had intended, with your permission, to slightly supplement in this direction what Dr. Berkart has said. Perhaps you will allow me to direct attention to Laennec's view, that extensive pleuritic effusion prevented pneumonia; to the fact that, in a lung partly bound down by pleuritic adhesions, the free portions are more liable to phthisical processes than the confined portions; to the effect of pleuritic adhesions in the neighbourhood of cavities in promoting contraction and cicatrisation; to Copland's opinion that in some cases of pneumothorax, the air by its pressure might promote the healing of the cavity; and to the evil effects of certain constrained positions of the body, which limit the motions of the lower masses of lung by pressing the abdominal viscera up towards the thoracic cavity.

"Your obedient servant,

"Belfast, October 18th, 1873. "John M'Crea, M.D."

fident, justify my assertion of its being the most powerful, if not the only means of arresting tubercle." When he goes on to say :—"In many cases a single grain divided into forty-eight doses, and one given every six hours, has been sufficient to arrest the disease and change the whole aspect of the patient," he spoils his case by excess of zeal, and tempts one to recall the axiom, "What proves too much, proves nothing."

But Dr. Gardner was not the first by a great many to recommend arsenical preparations in the treatment of phthisis,—although he says with regard to the use of arseniate of soda, "I have only met with one author who has even hinted at this application of it before I first recommended it."

At the Royal Chest Hospital, a mixture of liq. arsenialis ♏ ij. to ♏ iij., ext. cinchonæ liq. ♏ x. to ♏ xx. in an ounce of water twice a day after food, was largely used by me as a tonic in the treatment of phthisis; and both in hospital and private practice I have had great and increasing reason to be satisfied with the effects of arsenical preparations. Old cases which have done well are constantly turning up, in which I find that a combination of arsenic and bark, prescribed by me some years before, has been the favourite "*tonic*" to which the patient has returned again and again with confidence and advantage.

Of late years the waters of La Bourboule, which contain arseniate of soda, have been used by me with satisfactory results both as spray and as a diet drink ; the water being taken with an equal quantity of hot milk for breakfast and tea, or mixed with wine at luncheon and dinner.*

From my experience of arsenical treatment, I am not at all surprised to read in the "Boston Med. and Surg. Journ.," June 27, 1878, the following eulogy on the arsenical water of Pozzuoli, by my friend Dr. Horatio R. Storer, of Newport, R. I., President of the Gynæcological Society of Boston:—

"For the four years, 1872-76, and in part at the request of the American Medical Association, I was continuously engaged in investigating the relative claims of certain of the health resorts of Central and Southern Europe. . . .

"During 1873 and 1874 my attention was chiefly given to the ordinary considerations of local climate and a study of the Neapolitan chain of mineral springs, extending from Meta, adjoining Sorrento, through the whole circuit of the gulf, and ranging in temperature from 15° C., or thereabouts, at Meta, Vico Equense, and Castellamare, through from 17° C. to 21° C. in the springs of the city of Naples, to from 30° C. to

* I have just learnt with much pleasure that Dr. Brandt, of Oporto, will in future reside at Bourboule during "the season." This will secure for the patients the intelligent skill and kindness of an accomplished English-speaking physician.

95° C. at Pozzuoli, Baiæ, and the adjacent island of Ischia. I then became aware, from chance statements of my friends Dr. J. A. Menzies, of Naples, and Signor Saggese, a skilful chemist, that several of the more noted Neapolitan physicians were commencing to claim for Pozzuoli an exceptional excellence of a wholly different character, asserting that a portion of it had an atmosphere of its own, perceptibly charged not with sulphur merely, but with arsenic, from the semi-extinct volcanic crater known as the Solfatara, which, from but slightly rising above the level of the adjoining country, is easily accessible on foot, by donkey, or by sedan-chair to the most feeble invalid. The breathing of this *sulpho-arsenical atmosphere*, it was stated, not only theoretically promised to be of benefit in cases of threatened or actual pulmonary tuberculosis, after repeated visits to the crater extending over a longer or shorter period, but had in fact been proved so by actual experiment.

"Such assertions, in such a country, were to be received with great caution, but occasion offered for me to make a practical test of the matter during the winter of 1874-75. A lad of eighteen—my own son—extremely tall for his age, feeble, and ill-nourished, had passed the preceding two winters at Mentone under the supervision of Dr. Henry Bennett, and at Sorrento, going steadily from bad to worse, so that his medical friends had become extremely solicitous as to the result. The patient was now confided to Dr. Menzies, by whose directions, after the late portion of the autumn of 1874 had been spent in the westerly extremity of the Corso Vittorio Emmanuele at Naples (the very sunniest and most sheltered portion of the whole city), he was removed, still failing, to Amalfi. He passed some weeks at this place without noticeable benefit. It was therefore determined to make trial of Pozzuoli, despite the difficulty of obtaining a passably comfortable lodging. An apartment, such as it was, was hired, furnished, and a cook was taken out from Naples. The patient was unable to walk even the short distance from the house to the Solfatara, and was therefore carried into it by a couple of porters, breathing its direct exhalations for a gradually increasing period. Almost immediately, he commenced to improve. In addition to the prolonged respiration of this special atmosphere he took also, in minute quantities, the water of a spring arising from within the crater, and very unlike the prevailing mineral sources of the neighbourhood, which are alkaline, while this is sharply charged with sulphuric acid, and in use requires large dilution. The spring, and also the atmosphere in the vicinity of the greater vent of the crater, contain by analysis very appreciable quantities of arsenic. Other and ordinary measures, demanded by the alarming exhaustion of the patient, were meanwhile as hitherto pursued, and a certain amount of the improvement which then set in and became continuous was possibly attributable to them. There could, however, be no question that

this improvement began with and was rendered uninterrupted by the special influences of the Solfatara. In the case now reported marked physical signs had been wanting. All the rational symptoms, however,—profuse night sweats and a host of others,—had long been those of progressing pulmonary, or at any rate strumous, disease.

"Though now greatly interested in the subject, I could as yet—in view of a possible relapse in the instance referred to,* the impropriety of forming a conclusion from so limited an experience, and the total unfitness of Pozzuoli for the residence of invalids who had been accustomed to average American and English comforts—only say the following in my report to the American Medical Association, made in the spring of 1875: 'Regarding the Solfatara at Pozzuoli, which has been recommended as a residence for certain classes of invalids, the Neapolitan physicians are of opinion, and the question is one worth considering, that the arsenical and other emanations given out by the still-smoking crater sensibly and beneficially modify the neighbouring atmosphere in a medicinal way.'

"In proceeding to investigate the matter still further, I found that perhaps the first attempt to utilize the local atmosphere of the Solfatara was made by Dr. Abele Franza in 1871, as recorded in 1874 by Prof. Sebastiano de Luca, of the University of Naples.† Franza's patient, a Russian, was attended in consultation with Professors Manfrè and Lauro, and the diagnosis was advanced tubercular disease. He had been treated in Naples for four months without benefit, and the case had been pronounced a hopeless one. He was removed to Pozzuoli, and remained there for six weeks, being merely from time to time carried into the Solfatara, and respiring its vapours. The improvement is declared to have been immediate and to have remained permanent. A similar case, of even more interest, was reported at the same time by Dr. Annecchini.‡ The patient was seen in consultation with Prof. A. de Martini, of Naples, and considered clearly one of acute tuberculosis (*tisi galoppante*). A year after, the disease having in the mean time greatly progressed, this lady commenced to inhale the air of the Solfatara, and 'solely in consequence' (*per virtù esclusiva della potenza medicatrice dell' aria della Solfatara*) she was pronounced 'to have been radically cured' (*essa era in uno stato di salute il più soddisfacente, come se nulla avesse sofferto*).

"The admirable work upon the medical geography of Italy by Prof.

* No relapse, however, occurred. The patient was able to pass the winter of 1875-76 continuously in Naples, living upon the Vomero, above the Corso Vittorio Emmanuele. He returned to America in October, 1876, convalescent, and went through that winter safely in the harsh climate of Boston. He is now a student in the Massachusetts Institute of Technology.

† "Ricerche Sperimentali sulla Solfatara di Pozzuoli." Naples; 1874. Page 13.

‡ *Ibid*, page 43.

Luigi Marieni, of Milan, which so far as the mineral springs of that country are concerned, must long remain a chief authority, was published in 1870,* a year before these facts occurred. In 1868 and 1869 Prof. de Luca had presented three papers to the Academy of Sciences of the Royal Society of Naples upon the Composition of the Thermo-Mineral Water of the Solfatara, and the Temperature of the Greater Throat (*fumarola*) of its Crater, whence the arsenical exhalations escape, and within a limited though constant radius of which they are appreciable by Marsh's test. Other papers, chemical and therapeutical, upon the same subject, to the number of twelve or more, have followed from this gentleman, and are to be found in the *Comptes Rendus* of the Naples Academy; still others have been communicated by him to the Academy of Science at Paris, and he has also published the *brochure* already quoted, entitled Experimental Researches upon the Solfatara of Pozzuoli. The effect has been widely to attract professional attention in Southern Italy, and indeed throughout Europe, guaranteed as Professor de Luca's statements have been by the testimony of many of the physicians and surgeons to the great hospital Degl' Incurabili at Naples, some of whom are also attached to the University.

"Whether the hopes of these gentlemen are well grounded can be proved only by continued experiment. They are now, however, even more sanguine than at first, and at the present moment, after, it is said, 'the complete and radical cure' of quite a number of additional cases of advanced phthisis, who had been made to reside continuously for several weeks at the Solfatara, a branch of the Incurabili hospital has been established within the crater for the special treatment of pulmonary disease. To make the collateral test upon English-speaking patients has, as already stated, now for the first time been rendered possible at Pozzuoli. It is not unlikely that in the future the crater of the Solfatara may become as famous for its effect upon the prolongation of life as the neighbouring Grotto del Cane has been for imperilling it.

"It will be very interesting to test for arsenic the atmosphere of the various *stufe*, or volcanic vent-holes of steam and dry air, existing upon the neighbouring island of Ischia (a famous place of resort for invalids, in the Bay of Naples), which does not yet seem to have been thought of, though they were studied with care by the late Dr. Chevalley de Rivaz, of Casamicciola;† and it would be also well that the same should be done with those at Calistoga and the Geysers, already noted in the treatment of disease, in the vicinity of San Francisco, California, and the similar fumaroles of the Yellowstone.

* "Geografia Medica dell' Italia." Milan. 1870. Pp. 665.

† "Description des Eaux minero-thermales et des Étuves de l'Ile d'Ischia." Naples. 1859.

"Whether equal advantage to that mentioned above can be gained by a partial return to the old way of treating phthisis by artificial preparations of arsenic, this time with sulphur or sulphuric acid,—a combination that seems never yet to have been used in medicine,*—and employing them by inhalation, in atomized solutions, either cold or conjoined with steam, is as yet a point to be determined. My own impressions are in their favor.

"It may be argued that the, as it were, camping-out life at the Solfatara has something to do with the effect produced. The freest and most open air is never too pure for consumptives anywhere, but in Italy the ordinary life is an out-of-doors one, and these Solfatara cases had been subjected to it before being carried to Pozzuoli, and yet had rapidly declined.

"It may also be said that by the Solfatara method we are not treating a local disease by constitutional measures, but a constitutional one with a local tendency by means that are wholly localized. Though seemingly true in cases of pulmonary disease, the remark cannot be applied to other forms of struma, which appear to derive similar benefit. It would seem probable that the arsenic produces its effect, granting the premises that in Naples are now assumed, in a manifold way: (1) by direct action, in diseases of the respiratory passages; (2) by increasing nutrition or suspending waste, or both, for which it has long been noted; (3) as a general alterative; and (4) as a general tonic.

"In the case observed by me, the arsenical water of the Solfatara was used in conjunction with the respiration of its atmosphere, and may have increased the action of the latter, while its own, like that also of the atmosphere, may have been enhanced by the sulphuric acid that it contains. In many of the successful Italian cases, however, the inhalation of the arsenical atmosphere has alone been employed, thus narrowing the question to its ultimate merits. In either case, however, the

* In answer to an inquiry that I addressed to him, Mr. Theodore Metcalf, of Boston, has kindly given me the following *résumé* of our present knowledge upon the subject:—"The only arsenical preparations used in medicine are the liq. potassæ arsenitis, liq. sodæ arsenitis, liq. arsenici chloridi, and a solution of chloro-phosphide of arsenic, recommended by Dr. Hammond, of New York. The arseniates of ammonia, potassa, and soda are in use; also Donovan's solution, iodide of arsenic, and arseniates of antimony, copper, iron, quinia, and strychnia. Pastes of arsenic I have never had any occasion to make, nor any fumigations, except in one instance, when I had an order to saturate cigars with liq. potass. arsen. The arsenious acid in pills is also considerably used, and arsenic combined with quinia, iron, etc. Orpiment, a tersulphuret of arsenic, consisting of one equivalent of arsenic and three of sulphur, is used only as a pigment and a depilatory. Realgar, a bisulphuret, one equivalent of arsenic and two of sulphur, is used only as a pigment. Neither of the sulphides, that is sulphurets, of arsenic are supposed to be so poisonous as the arsenic itself, but so far as I know they are never prescribed. The most recent medical literature that I have at hand gives nothing new in arsenical remedies."

treatment would chiefly have been constitutional, in accordance with Dr. Henry Bennet's well-expressed formula: 'The most advanced minds in the profession more and more recognize the fact that the local manifestations of chronic chest diseases, tubercular or inflammatory, are mere epiphenomena. They require treatment, of course, but their treatment is of secondary importance when compared with the treatment of the constitutional state of the patient, which is at the root of the mischief.'

"Remarks upon the effect of the Solfatara in purely gynæcological cases, functional and organic, I must reserve for a future occasion. Meanwhile the whole question will be still further investigated, from an English stand-point, by Dr. Cerio."

Chloride of calcium, another old favourite remedy of mine—the beneficial effects of which, on a family of children with strumous glands, made a very strong impression upon me many years ago—has lately cropped up into notice and deserved favour, chiefly through the following article by Dr. Robert Bell, of Glasgow. My own experience of this salt enables me to confirm to a great extent, the somewhat strongly-drawn eulogium of Dr. Bell. I certainly agree with him that "In tubercular disease of the cervical glands I know of no remedy which can equal it," and by analogy we ought to expect that it would have a beneficial effect upon that phase of tuberculous disease of the lungs which implicates the adenoid tissue, and it is probably in this way that it proves an important auxiliary to other treatment in pulmonary consumption. (See p. 166-9.)

Dr. Bell says:—"I trust that a short time spent in considering the therapeutic properties of a salt which has been till quite recently thought of such little value in the treatment of disease, that in some works on materia medica it is not even classed as a therapeutic agent, may be of advantage to the readers of 'The Lancet.'. Chloride of calcium is an agent which, I am sure, only requires to be more thoroughly understood to be appreciated as a remedy, and to be valued as a pharmaceutical preparation. That it has not hitherto entered largely into the prescriptions of medical men in this city may be inferred from the following incident. I wrote a prescription, the principal ingredient of which was the chloride of calcium; this was made up at a most respectable dispensing druggist's shop. In a day or two after, the mother of my little patient came to me complaining that her daughter had only been able to take the medicine once or twice, as it produced such obstinate vomiting. What was my horror on finding that chlorinated lime had been used instead of what was ordered, and a most sickening compound was the result. On my going to the principal of the firm where the mistake had been made, he coolly told me that he knew of no other chloride of calcium than the one he had

used. I mention this incident to show how very rarely this potent remedy has been taken advantage of.

"Chloride of calcium possesses a most wonderful power in controlling if not actually curing, many forms of tubercular disease. In my experience I have found no remedy on which so much reliance can be placed in tuberculosis as on this salt; more especially, however, this remark applies to the wasting diseases of children. It has been most extensively used by me during the past four years, and with most gratifying results. Having prescribed it in every form of tubercular disease that has come before me during this period, perhaps a short account of my observations on the effect of the drug may not be uninteresting.

"In the wasting diseases of childhood, be these tubercular or not, chloride of calcium has proved itself to be, in my experience, a therapeutic agent of inestimable value. The conditions which indicate the probable usefulness of the salt in children are, first of all, a falling off in flesh. The child may take his food heartily enough—nay, his appetite may be better than usual—yet he becomes more attenuated every day, he is languid, oft-times sleepless, and the pupils are always very much dilated. When sleep does come on, the little patient frequently starts up in a fright, grinds his teeth, and convulsive twitching of the muscular system will often be observed—these symptoms being evidently due to a large amount of undigested food in the lower bowel. Oft-times there is a craving for stimulants, and a most extraordinary liking for potatoes and other articles of diet containing a large amount of starch. If the mother is questioned, the remark will often be made that the child takes his food so well as to make it quite beyond her power to understand how he does not thrive, but, on the contrary, is falling off every day. And it is remarkable to note how rapidly a fat and healthy-looking child will become a mere shadow of his former self when such a train of symptoms is present. When we come to examine the patient, the face may appear to be pretty plump, but the arms and legs are miserably thin, soft, and flabby, while the abdomen is greatly distended, having the cutaneous veins very much engorged. The evacuations should always be examined, when it will be observed that they are much greater in quantity than they ought to be, that undigested food can be largely traced in them, and that their fetor is excessive. Such a state of things distinctly points to great defect in the powers of digestion and assimilation. In fact, none of the food appears to have entered the child as nourishment, it having seemed to pass away in a state of putrid fermentation, while the body has been preying on its own tissues. It is in circumstances such as these that the beneficial effects of the chloride of calcium can be appreciated. If a child is brought to me with the symptoms that have just been enumerated, or if any symptoms indicating a want of assimilating power

are observed, I immediately insist on the medicine being administered and continued for several weeks. Its powers in arresting such symptoms, in my opinion, are superior to cod-liver oil or iron; and what is of no little advantage, very young children soon get to take it quite readily. Of course, when one is prescribing in disease of this kind, it is absolutely necessary to observe strict dietetic rules. In addition to the internal remedies, it will be of immense service if the abdomen of the patient is gently rubbed, night and morning, with olive oil, and afterwards a flannel bandage applied. With reference to diet, I insist upon a large quantity of milk, and the avoidance of starchy food and sweets.*

"The history of one or two cases may perhaps be of service as evidence of what has just been said.

"J. F——, three years of age, was a most miserable-looking object when he came under my observation about four years ago. His legs and arms were most attenuated, and the abdomen was distended to an extraordinary degree; moreover, symptoms of ulceration were present at the umbilicus. This gradually increased till, in a very short time, a false anus became established at this point, from which passed the most fetid and offensive of stools. To such an extent did the evacuations exude from this opening that very little passed through the rectum. An area extending for several inches round the umbilical orifice was excoriated by the acridity of the discharges. This was counteracted by the application of charcoal poultices, and other antiseptic applications. In consequence of the emaciated and exhausted condition of the patient, the slightest movement caused him intense pain. In spite of all these untoward symptoms, the chloride of calcium worked a cure, for in fifteen months the fistulous opening healed, the child gained in weight and strength, and now he is a strong and apparently healthy boy, not presenting at this time any appearance outwardly of having been in such a precarious state of health three years and a-half ago.

"Another example of the curative effects of this salt in the wasting diseases of children will suffice; and this time I will relate the history of three children of one family who were considered to be in a dying condition from tabes mesenterica. Two children of the same family had, previous to my having been called to attend, been cut off by the same disorder. The three children—all boys—to whom I refer, had all the symptoms of tuberculosis, and appeared to be literally dying on their feet. Their ages varied from three to eight years. Within seven months of the commencement of the treatment by chloride of calcium, combined with a judicious diet, the children were plump and healthy-looking. The abdomen in each individual was reduced to a normal size, the stools had become healthy in appearance and quantity, and had lost their offensive stench. In short, the children were cured. It

* See p. 256, "On Fat and Starch in the Nutrition of Children."

is now eighteen months since they passed from under my care, and I believe they are still enjoying most excellent health.

"Before passing from this part of my paper, I think it would be remiss in me were I to omit mentioning the effect of the chloride upon the children of the Cambridge Street Orphanage for Girls, of which institution I have the medical charge. The majority of these children are in a most pitiable condition when admitted, and in more than half the cases signs of tubercular disease are very apparent. The chloride of calcium has been here put to a crucial test, but it has shown itself to be worthy of the trust reposed in it, for in none of the cases has it proved of no benefit. All the children to whom it has been administered have improved under its use, and the majority of those affected appear to have shaken themselves clear of the disease altogether. These facts appear doubtless very extraordinary, and so they are, but if anyone would like to prove the matter for himself, let him visit the Orphanage, which is always open to visitors, and the matron will gladly show the patients who have been under treatment.

"In the tubercular diseases of bones and joints of children the beneficial effects of the chloride of calcium are very marked. I could recount numerous instances of cure effected by this means, but will content myself by mentioning one. A lad about thirteen years of age was brought under my notice some two years ago. At that time there were eight running sores in different parts of his body each having connection with diseased bone. A rib was affected, the bones of the forearm, and the metatarsal bones. He was in a most emaciated condition, but under the chloride of calcium treatment he rapidly gained flesh. He took the medicine for fifteen months, at the expiration of which time the lad was perfectly well.

"In tubercular disease of the cervical glands, I know of no remedy which can equal it. Numberless instances of tuberculous cervical glands yielding to its power have passed under my eye, and, indeed, it is in these affections that the chloride of calcium most unmistakably displays its therapeutic properties. A single case may be sufficient to show how it acts in such circumstances. A young man came under my care about eighteen months ago. His condition was as follows :—A running sore proceeded from each parotid and the submaxillary glands, and there was enlargement of some of the cervical glands. His left testicle was also very much hypertrophied, and it was the seat of an abscess. I should add that all these abscesses had been discharging for a period of several months. He was ordered twenty grains of chloride of calcium three times a day after meals, and in a few months the abscesses in the neck healed, the enlarged glands became reduced in size, the testicle also recovered, and the patient gained rapidly in weight ; and now he is in the enjoyment of good health, though I still advise a continuance of the treatment.

"I have treated several cases of phthisis pulmonalis by this means, and with most gratifying results when the disease was got at in the early stage. In such cases I have had distinct cures—at least, the disease has been checked, emaciation has ceased, the cough has disappeared, and the patients have recovered their healthy appearance. In conjunction with the chloride I have used a spray containing one-fifteenth of a grain of arsenious acid to one drachm of water thrice a day, and I think with the effect of expediting the cure.

"Before concluding, permit me to add that the only case of tubercular peritonitis that has come under my notice during the past four years has yielded completely to this remedy.

"It will be seen from the cases that I have cited that the medicine requires to be perseveringly used. Let me urge on my professional brethren to give it a lengthened trial, and not to be discouraged by an apparent failure. Chloride of calcium can do no possible injury to the economy, while in properly selected cases it will be of incalculable service." ("Lancet," Aug. 25, 1877.)

The next point on which I ought to speak is Climate or Change of air. What is the proper kind of climate for Consumption? There is no subject on which more conflicting opinions have been expressed by experienced authorities. There certainly have been few means of treatment which have been so whimsically employed—by which such remarkable injuries and remarkable benefits have been conferred upon consumptive persons, as by change of climate. And up to the present time no one has been able to give a rational explanation of the good or of the harm often done to different persons by the same climate. The whole mystery appears to me to be explained by the views I have enunciated of the physiology and pathology of that "Abnormal Physiological State"—tuberculosis. (See Part II.)

In order to make this clear as briefly as possible, let us consider the objects of climatic treatment in the True first stage of consumption. These may be divided under three heads:—

1. The restoration of the normal supply of fats to the blood, *i.e.*, the radical cure of tuberculosis. (See pp. 194—201.)

2. The economy of fat and carbon in the organism and the protection of the lungs from undue oxidation, *i.e.*, provisional protection against tuberculisation. (See pp. 220-2.)

3. Removal or prevention of catarrhal affections of the air-passages, of chills to the general surface, and of local congestions, *i.e.*, the collateral treatment of tuberculosis. (See p. 218.)

Now it happens that the climates necessary for the 2nd and 3rd of these objects are utterly different from those required for the 1st, and that the climate required for the 2nd object, is frequently unfit for the attainment of the 3rd.

No wonder, then, that attempts to cure consumption by change of climate, undertaken without any clear appreciation of these important distinctions should so often fail, and that, when they succeed, it should appear to be attributable to a sort of "good luck."

I will attempt in as few words as possible, to explain these incongruities. The first object—restoration of the normal supply of fats to the blood, *i.e.*, the radical cure of consumption—is the one about which there is the greatest misunderstanding. Places are picked out for the consumptive, because they are "mild and equable," or because they are "sunny and stimulating," or because they are at once "warm, equable, and bracing," and the like; whereas none of these qualities is the one especially called for by the necessities of the case. It is true, that in those cases where pancreatic function has been perverted by some mental or emotional shock, a sunny stimulating atmosphere may be of some use by its exhilarating effects upon the depressed spirits. (See p. 218.) It is true, that in a warm and bracing air a weak person may be rendered less conscious of the sense of weakness, and that an equable climate may improve the digestion and the appetite, by allowing a delicate person to be more constantly out of doors ; and thus all these are good in their way. But the real exciter of pancreatic function is clear dry cold. It is neither the equability, nor the stimulating, nor the bracing quality which makes the demand upon the pancreas, and wakes it into life, but the cold, *per se*. The cold must not be damp and must not be accompanied by darkness, because damp and darkness interfere with the special action of the cold, producing what we commonly understand by *chill*, and depressing at once the animal spirits and the processes of life. The cold therefore must be clear and dry and light, and in this cold, exercise must be taken, so that carbon may be peremptorily demanded for the supply of both heat and mechanical force. Typical of this treatment I may mention "hunting" in Minnesota. (See p. 216.)

But remember how dangerous all this must be if tuberculosis has advanced far enough to make tuberculisation imminent. (See p. 220.) Such means can only be adopted with safety while the tissues are still well protected by fat, or when the blood, at least, is fully charged with it. They are utterly at variance with all those precautions upon which I have insisted for protection against tuberculisation. Again, no one suffering from catarrhal complications or local congestions can be exposed to these hygienic conditions without risk to life. But this is the treatment to be constantly kept in view, to be adopted promptly whenever other conditions will permit. Whatever modifications and dilutions of this treatment may be required by circumstances, we should never lose sight of the fact, that our treatment of consumption only becomes truly curative in proportion as it includes these means for restoring the healthy functions of the stomach, liver, and pancreas.

The second object of climatic treatment—economy of fat and carbon in the organism, and protection of the lungs from undue oxidation, *i.e.*, provisional protection against tuberculisation—is of the greatest importance. It is the object of treatment which most often demands our first anxious attention in cases of the true first stage of consumption as ordinarily presented to us in practice. Tuberculisation is imminent, and nothing else must be thought of until we have safely provided against it. (See pp. 210-11, and 220-2.)

It is evident that if this object is to be met by climate we must look for an atmosphere which, while pure, may yet convey as little oxygen to the lungs as is consistent with the continuance of life and nutrition. It must therefore, be either rarefied, or diluted with some unirritating matter.* The combination of a certain amount of rarefaction with a considerable dilution with aqueous vapour is most readily to be found. Again, we must look for an atmosphere sufficiently warm to save some of the demand for carbon to supply animal heat, and we must look for a place where this warm diluted air can be freely breathed with as little exercise as possible, so that histogenesis may be passive, and the demands for mechanical force reduced to their lowest degree. (See p. 159.) At the same time, we must try to combine a bright and cheerful landscape and pleasant society, to encourage the pursuit of a vegetative life without depressing the spirits. I cannot better typefy such a place in this country than by mentioning Torquay.

The third object—removal or prevention of catarrhal affections of the air passages, of chills to the general surface, and of local congestions, *i.e.*, the collateral treatment—may sometimes be attained in the same climates as the second object ; but very often this is not the case. These collateral affections are frequently dependent upon collateral diatheses, independent of the consumptive, such as the gouty, rheumatic, syphilitic, hæmorrhagic, etc., and can only be benefited by climates specially suited to each case. Thus in the commonest catarrhal and rheumatic complications, the aqueous vapour with which the warm air is diluted, in such places as I have referred to under the second heading, may be positively injurious, and a warm, dry, stimulating air may be absolutely necessary to their removal ; hence if we are bent upon climatic treatment, we may have to seek for different classes of climates to any yet mentioned, such for example, as Cannes, or Upper Egypt. (See the chapter on Climatic Treatment, in the sixth edition of my work "On Winter Cough.")

On the whole, for patients in whom tuberculisation is imminent or has commenced, I consider a Nile boat and a residence in Upper Egypt the nearest to perfection possible. And it is an increased advantage that we have now, through the enterprise of Messrs. Cook, a first rate hotel established at Luxor, Thebes, with every comfort for the residence of invalids in that superb climate.

* See Altitude in Treatment of Consumption, p. 252.

Thus it is seen how diverse are the reasons why one consumptive patient may seem to be cured by the same climate that kills another. While we are curing our patient's catarrhal complications at Cannes or Thebes, tuberculisation may be taking place, for want of the conditions to be found at Thibet or at Torquay; and while we are protecting him against tuberculisation at Thibet or Torquay, his defective assimilation of fats may be becoming permanent and incurable, for want of the conditions to be found at St. Paul.

When we consider how little these distinctions enter into the reasons which guide consumptive persons in resorting to this or that change of climate, I fear we must attribute the cures which occasionally take place more to "good luck" than to anything else; this "good luck" consisting in the spontaneous resumption of pancreatic function during some favourable phase of the case—some period of arrested tuberculisation, during which the weather fortunately turned dry, windless, and cold, or during which the patient, feeling better, indulged in more air and exercise just at the fortunate moment, and set the pancreas and digestive organs into action. Or, as I have suggested before, the perverted pancreatic function may have been simply due to the influence of some depressing mental or emotional cause, which happened to be removed in time.

If we could send our patients here or there exactly at the right moment, and each time to a place accurately adapted to the passing phase of his case, we ought unquestionably to select those strongly marked distinctive climates which I have described. But, practically, how seldom we can do this! And let us remember what happens every day in practice, to the infinite risk of the life of the patient, and of the reputation of the physician. Patients consult their physician, and receive, we will assume, advice as to the choice of climate cautiously and wisely adapted to their condition at the time; and after such advice is given, circumstances occur which alter their plans, and delay their seeking the prescribed change, until their state has so far altered, that *if they consulted their physician again, he would certainly reverse his former judgment.* But instead of giving either themselves or him this chance, they carry out, at great expense and inconvenience, a plan of climatic treatment utterly unfit for the, then, conditions of the case.

On the whole, therefore, under ordinary circumstances, more good and less harm may often be obtained by climates which to some extent combine the qualities I have described—even though they have not either of them developed in perfection—provided they are near home, within reach of friends, and under the management of a judicious physician who can watch the changing aspects of the case, and point out the time and mode in which to take advantage of this or that feature in the climate of the place.

It is in this way that such places as Mentone, San Remo, Bournemouth, the Isle of Wight, and Hastings, retain their hold upon the public and upon the medical profession. In each of these places there are the means, to a considerable extent, of pursuing the second and third objects of climatic treatment; and when the time comes for adopting the first, patients can come out of their warm quarters, and on the cliffs and hills can find some amount at least, of the *clear dry cold* necessary to rouse their pancreatic functions.

But whether the treatment is conducted at home or abroad the principles are the same, and must be steadily borne in mind by the physician, who must carry them out in the best way that the means and opportunities of his patient render practicable.

The question of treating tuberculosis by Altitude is too full of interest and promise to be passed by without special remark, but it is beset with so many conflicting difficulties that, practically, its applicability, as yet, is very limited.

It is a conclusion which strikes the mind at once, that if we have to treat a patient in whom there exists—1. A constant flood of fresh oxygen brought into contact with the pulmonary blood by respiration, and seeking materials for oxidation; 2. Deficient supply of materials for normal oxidation, and blood deficient in such materials flowing through the lungs; 3. Excessively oxidised blood; 4. Impending or progressing abnormal disintegration of tissue by oxygen,—two measures are urgently called for: *a*. To supply the necessary materials for normal oxidation, by specially adapted medicinal foods, etc.; *b*. To diminish the influx of oxygen to the blood from the air. (See pp. 152, 159—169.)

The latter of these measures would seem *primâ facie* to be easy enough to carry out by placing the patient in an atmosphere rarefied by altitude; and we have enough facts to justify us in the belief that such a plan of treatment is of the utmost value in protecting the lungs from oxidation, while other measures are adopted to remove the abnormal physiological state which has placed them in danger. (See p. 250.)

But altitude, if great enough to have any material effect on the proportion of oxygen, and if long enough continued to have any abiding influence on the patient, almost inevitably means exposure to low temperature, with its attendant dangers, and it means increased afflux of blood to the lungs, at least for a time, with its attendant dangers to injured or morbidly vulnerable vessels. (See p. 25.)

I am strongly of opinion, however, that much of the good with but little of the evil effects of altitude may be obtained, if only the main physical and physiological facts of the case, both with regard to the nature of the abnormal physiological state to be remedied, and of the

effects of altitude upon the animal organism, are kept clearly before the mind, and boldly enunciated.

Under their dictation, we shall *strictly forbid exercise to the consumptive patient while exposed to the effects of altitude.* I say "boldly" because this question of exercise is the rock upon which most cases are wrecked. Under the influence of sentimental and mystical notions about "pure air and exercise," patients are sent into high altitudes, with orders "to invigorate their bodies, and their lungs especially, by exercise in the pure and lovely atmosphere;" and it is such deeply ingrained and popular twaddle as this which requires boldness on the part of the physician to upset it.

The late Dr. Edward Smith proved by experiments upon himself that "walking in health at one mile per hour increases the respiration from 500 cubic inches to 800 cubic inches per minute; at two miles per hour, to 1,000 cubic inches; at three miles per hour, to 1,600 cubic inches; and at four miles per hour, to 2,300 cubic inches per minute. Whilst with running moderately at six miles per hour, it is more than 3,000 cubic inches per minute. The depth of inspiration is likewise increased from 35 cubic inches to 100 cubic inches per respiration; and hence both the mechanical distension of the lung, and the chemical and physical changes of respiration are greatly increased.

"Riding in a carriage increases respiration, but the degree varies with the oscillation of the carriage. In an omnibus the increase is fully half of the original quantity. On horseback at a walking pace, the increase is more than half, in trotting, three-and-a-half times, and cantering, three times the original quantity.

"Walking at the rate of four miles an hour will increase the rate of pulsation to about 130 per minute, and running at six miles per hour, to about 180 per minute, and even in gentle exercise there is some increase in the rate of pulsation." (On "Chronic Phthisis," by Dr. Edward Smith.)

It is palpably absurd, therefore, to place a patient in a special climate with a view to diminishing oxidation, and then to order, or permit that riding, driving, or walking should be practised, by which respiration, pulsation, and oxidation are all strained to their utmost.

The very feature of the climate for which we seek its influence, viz., the deficiency of oxygen in a given volume, excites the respiratory and circulative systems to over-action in vain attempts to obtain the customary supply of oxygen to the blood, and they need constant care to restrict them within proper limits. The effects of altitude upon persons in health, are well illustrated by the description given by Mr. Webber, who writing to "The Times" from the Grindelwald in Switzerland, states that "in Thibet he had lived for months together, at a height of more than 15,000 feet above the level of the sea, and that the result was as follows:—His pulse, at normal height only 63

per minute, seldom fell below 100 per minute during the whole time he was at that level. His respirations were often twice as numerous in the minute as they are at ordinary levels. A run of 100 yards would quicken both pulse and respiration more than a run of 1,000 yards at the sea level, and the higher the level, the greater the difficulty of walking or running fast. He crossed the shoulder of the Gurla Mandhâta at a height of some 20,000 feet measured by the barometer, and found the greatest difficulty in getting his breath quickly enough, had frequent and violent headaches, and found that his native guides and companions suffered much more even than he did." ("Spectator," Aug. 3, 1878.)

"Mr. F. Cresswell made some observations on two naval officers during three days at an altitude of 2,950 feet.

"A's normal vital capacity—325 cubic inches—decreased to 290·5 cubic inches on the first day, gradually rising to 313·8 cubic inches on the third day.

"B's normal vital capacity of 270 cubic inches was reduced to 241 cubic inches on the first day, gradually rising to 265·5 cubic inches on the third day.

"The temperature varied from 61° Fahr. to 52° Fahr., and the barometric pressure from 27·78 to 27·6 inches.

"Both the men were accustomed to the process, so that the progressive increase in the vital capacity during the three days was due to the lungs accommodating themselves to the decreased pressure; the causes of the first diminution being removed or neutralised.

"The explanation given by Mr. Cresswell is, that the depressed pressure on the pulmonary tissue causes an increased quantity of blood in the lungs, and hence diminishes the vital capacity; that either this hyperæmia gradually subsides or else compensatory dilatation of the pulmonary air-cells occurs, and, as a necessary result, the vital capacity gradually regains its natural standard."—("Dr. Dobell's Reports," vol. ii., p. 282.)

In 1869, Dr. Treutler and Dr. B. Stewart of the Kew Observatory kindly provided me with the following information for publication in the first volume of my "Reports on Practical and Scientific Medicine":—

One hundred cubic inches of dry atmospheric air, temp. 32° Fahr., contain—

At the sea level,				7·147	grains of	oxygen.
„	500	feet above	sea level,	7·01	„	„
„	1,000	„	„	6·87	„	„
„	2,000	„	„	6·61	„	„
„	3,000	„	„	6.38	„	„
„	4,000	„	„	6·18	„	„
„	5,000	„	„	5·90	„	„

Supposing these figures to be correct, and assuming that the same number of cubic inches of air per 24 hours are respired by the same individual at each altitude (which is questionable), and that 30 oz. avoirdupois is the weight of oxygen consumed in 24 hours by such an individual at the sea level, the following results are obtained by calculation :—

Altitude.	Grains of Oxygen per 100 cubic inches of dry air 32° Fahr.	Ounces (Avoirdupois) of Oxygen consumed in 24 hours.
Sea level.	7·147	30
500 feet above sea level.	7·01	29·4249
1,000 „ „	6·87	28·8372
2,000 „ „	6·61	27·7459
3,000 „ „	6·38	26·7804
4,000 „ „	6·18	25·9409
5,000 „ „	5·90	24·7656

It will be observed that, according to these calculations, the average diminution in the weight of oxygen consumed is about 1 oz. per 1,000 feet of elevation, the temperature being the same, viz., 32°.

All these calculations are open to fallacy from the difficulty of accommodating them to all the circumstances ; but, taking them as approximately correct, we shall not be surprised at the distress experienced by Mr. Webber and his friends when we consider that they were deprived of oxygen at the rate of 1 oz. per 1,000 feet of elevation, so that at 15,000 feet in Thibet they were deprived of 15 oz. of oxygen per twenty-four hours, or one-half their total normal consumption, and when on the Gurla Mandhâta, 20,000 feet above the sea-level, they were deprived of two-thirds of their normal supply of oxygen. No wonder that they were gasping for more oxygen, like fish out of water.

I have said that these calculations are open to a variety of fallacies difficult to avoid or estimate, and one of the chief of these is the question of *temperature*. Dr. Treutler and Mr. Stewart attempted to make their calculations at different altitudes comparable by taking a fixed temperature of 32° for all, but as a matter of fact there is a rapid diminution of temperature with increasing altitude. In his balloon experiments in 1862 Mr. Glaisher found a loss of about 2° Fahr. for every 1,000 feet of ascent; thus, he left the earth at 1 p.m., and in an hour reached 30,000 feet. At starting the temperature was 59°, and at 30,000 feet it was 61° lower, or 2° below zero. At this rate, therefore, Mr. Webber would have found a temperature of only 19° at 20,000 feet, supposing it to have been 59° at the sea level.

But the balloon experiments of Professors Wahl and Wise in 1874, starting at a much higher sea level temperature, gave results differing

from those of Mr. Glaisher.—(See "Lond. Med. Rec.," Oct. 14, 1874.) The maximum temperature observed by them at starting was 97°, and this continued at 1,300 feet. At 2,000 feet it was 96°, at 3,800 feet 85°. The fall went on until at the greatest altitude attained, viz., 8,743 feet, the temperature was 68°, or a loss of 17° in the last 4,943 feet of ascent. Supposing this rate to have continued during another 9,886 feet, there would have been a loss of 71° at 18,629 feet; whereas Mr. Glaisher, starting at a sea-level temperature of 59°, found a loss of only 61° at 30,000 feet; and supposing Mr. Webber to have made his observations on the Gurla Mandhâta when the sea-level temperature was 97°, as in Professor Wahl's and Wise's experiments, he would have found the temperature at 20,000 feet of altitude about the same as when reckoned by Mr. Glaisher's standards; but the loss upon the sea-level temperature would have been about 78° in the one case, and only 40° in the other. This example well illustrates the difficulties and sources of fallacy, complicating our speculations on the influence of altitude on disease, to which I have referred.

Another source of difficulty has been pointed out by M. A. Sanson ("Comptes Rendus," April, 1876). His experiments show that the exhalation of carbonic acid by respiration increases with *in*crease of temperature, and with *de*crease of barometric pressure. He concludes, therefore, that the greatest possible elimination of carbonic acid will result from the combination, a *high temperature* and a *low barometric pressure.* But supposing this to be the case—as we have seen that low barometric pressure consequent upon altitude necessitates low temperature, it is evident that they may neutralise each other in their influence upon the elimination of carbonic acid. These results point to the great differences which may exist in physiological effects between such climates as are obtained by the altitudes of the Swiss Alps, the mountain plateaux of Mexico, or of the Bolivian Andes.

Amidst all the difficulties of the subject, however, there is one practical conclusion which we may safely draw, and which I wish most emphatically to impress, viz., that if we seek to save oxidation by the influence of altitude we must *forbid exercise.* Our patients should be carried in the quietest possible manner to their destination, and when there they should simply vegetate till they have become fat and well. (See p. 159.)

Next in importance to the initial loss of flesh characteristic of constitutional tuberculosis is that Wasting of Children which contributes so largely to the Registrar's records of deaths under the head of "Atrophy and Debility," and which is commonly described as "Marasmus;" and I venture to sum up what I wish to say on the treatment of this condition, by the following quotations from a paper "On Fat and Starch in the Nutrition of Children," which I contributed to

the "Practitioner," in 1872, only adding that my subsequent experience, and that of numerous medical friends, has abundantly confirmed what I then wrote :—

"A very interesting article by Dr. Prospero Sonsino, of Pisa, in the September (1872) number of the 'Practitioner,' 'On the Physiological Dyspepsia for Starchy Food in Infancy,' revives an intention, from which I have been diverted by other occupations, of publishing a few words on an allied subject.

"I propose to speak of the class of cases constituting that wretched form of 'atrophy and debility' and 'marasmus' in children, in which every part of the body wastes away except the abdomen; the state described by Dr. Druitt, in the last edition of his 'Vade Mecum,' in the following few and graphic words:—'Emaciation and voracity; the belly swelled and hard; the skin dry and harsh; the eyes red; the tongue strawberry-coloured; the breath foul; the stools clay-coloured and offensive, sometimes costive, sometimes extremely relaxed; the patient usually dies hectic' (p. 75).

"I wish to bring prominently forward the fact that this state, provided there is no advanced lung disease, *is rapidly cured by Pancreatic Emulsion given in doses of a teaspoonful every four hours, and regularly persisted in till fat and flesh are restored.* It is, of course, necessary that a proper diet should be insisted on at the same time ; but proper diet without the Pancreatic Emulsion will not do. This I have found over and over again in cases where everything judicious in the way of feeding and cod-liver oil had been carefully and perseveringly tried without avail, but which, on the addition of the Emulsion to the previous diet, began at once to improve.

"This fact has been familiar to me for a long time; and considering how largely Pancreatic Emulsion is now used in the wasting diseases of adults, I am surprised to find that it is not even referred to in the latest works on the diseases of children. (See p. 195.) . . . Yet scarcely a week now passes but some medical man relates to me cases of the successful use, in his own practice, of Pancreatic Emulsion in the wasting of delicate children.

"Dr. Prospero Sonsino's paper will, I hope, excite more general attention to this important subject. He, however, has laid all the stress of his observations upon the influence of the salivary and pancreatic juices on the digestion of *starch.* This is unquestionably a point of the greatest importance in the case of very young children brought up by hand, as showing the absurdity of attempting to nourish them upon starchy food, not artificially digested, before the period of life at which the saliva and pancreatic juice attain their functional activity. And even then, as Dr. Sonsino afterwards remarks, 'good reasons make us now believe that really it is not proper to feed infants with copious starchy matters, however these may be rendered digestible.' The

principal results of Dr. Sonsino's investigations are summed up in the two following conclusions, which, however, are not new :—1. 'Pancreatic juice in dogs, cats, and rabbits, in the first week of life—perhaps for some days more—is devoid of any digestive action on starch.' 2. 'In the early life of man, probably till the beginning of dentition, infants offer a true physiological dyspepsia for starchy aliments, caused by the inactivity of one at least—possibly of all—the humours that concur in the digestion of those aliments' (saliva, gastric juice, pancreatic juice, enteric juice).

"No doubt, when wasting occurs in these early periods of life, it is very often due to foolish attempts to nourish children upon farinaceous foods, by which dyspepsia and diarrhœa add to the exhaustion of partial assimilative-starvation. But, as a matter of fact, farinaceous food is seldom depended upon without some addition of cow's milk or some assistance from lactation; and we see children suffer from wasting who are fed entirely upon cow's milk or nursed by their mothers, and in such cases the 'physiological dyspepsia for starchy food' will not account for their decline. Therefore, we must not forget, that although normal saliva only acts upon starch, *normal pancreatic juice acts also upon fats;* and it is probable that these two functions of the pancreas are sufficiently independent of each other that they may exist separately. This I pointed out in my paper to the Royal Society in 1868, 'On the Special Action of the Pancreas on Fat and Starch.' It is there stated as the results of my experiments, that 'in addition to the influence of the pancreas upon fat, it has the power of converting starch into glucose by simple mixture. This property remains to a certain extent *after the pancreas has exhausted its property of acting upon fat.* The quantity of pancreas which before mixture with fat will convert about eight parts of starch into glucose, after saturation with fat will still convert about two parts of starch into glucose.' It is possible, therefore, that in different states of depraved health one or other of these properties of the pancreatic juice—that for the digestion of starch, or that for the digestion of *fat* may be deficient. And thus the depraved nutrition due to such deficiency will not be limited to the period of life anterior to that at which, under normal conditions, the proper functions of the pancreas should be developed. It is evident that, when the power of digesting fat fails to be developed at its proper time, the defect must tell with double force upon children already suffering from deficient digestion of starch.

"The children who become the subjects of the kind of wasting of which I am now treating are especially: (1) those who are suckled by mothers whose milk, though abundant in quantity, is extremely deficient in nutritive properties; (2) those who are brought up by hand; and (3) those who, at a later period of childhood, have been subjected

to similar chronic defects in diet. Now, it is especially when the mother's milk is poor in fat and lactin that the child becomes 'dissatisfied' and 'craving,' and in the majority of cases it is this which leads to the introduction of farinaceous food, under the popular nursery belief that it is '*satisfying*;' and, as Dr. Sonsino states, if this be given before the power of digesting starch is established, of course nothing but mischief can result.

"But organs, like individuals, do not rise to the full performance of their duties unless called upon by the necessity for their activity; and, as I pointed out in 1866 ('On Tuberculosis,' p. 40, second edition), 'As the mother is deprived of fat-elements by lactation, so is the child deprived of them by a persistence in a diet deficient in milk. In the case of the child thus deprived of fat, a double injury is done, first, by cutting off the supply of fat elements necessary for the protection of the tissues; and secondly, *by paralysing the functions of the pancreas by prolonged inactivity*.' I venture to think that this is a point deserving far more attention than it has yet received.* It accounts in a great measure for the impossibility of restoring these ill-nourished wasted children by any kind of *natural* diet after they have been allowed to remain in a chronic state of defective nutrition. A child that has been long fed upon diet deficient in fat fails to develop the fat-digesting properties of the pancreatic secretion, and thus, when proper food is at last presented, cannot make use of it for nutrition.

"It is probable, therefore, that it is due to this conjunction of circumstances that these wretched cases of fatal infantile wasting occur: the food deficient in fat not only fails to nourish the child, but fails to develop the function of the pancreas for the digestion of fat at a later period of life; the craving of the child due to the deficiency of assimilated fat is met by starchy food, which it has not the power to digest, and which, if digested, cannot supply the place of fat. Thus it is literally starved from first to last of those elements of nutrition especially essential in early life. We cannot, therefore, be surprised that such cases have proved obstinately fatal, neither is it anything but what one might expect *a priori*, that they get rapidly well when Pancreatic Emulsion of fat is added to their diet, for by this means they are enabled to assimilate both fat and starch. (See pp. 157-9, 194-7, and 225.)

"Certainly, of all the satisfactory remedial effects of Pancreatic Emulsion, none equal the almost magical recoveries of some of these miserable wasted children. The cases in which I have seen it administered within the last eight years (1872) are too numerous to relate, and I will only briefly mention three of those which first especially excited my attention.

* See some excellent papers by Dr. D. J. Brakenridge, "On the Influence of a Digestive Habit," etc., in which he has elaborated my views on this subject. "Medical Times and Gazette," June, 1868.

"1. A poor woman came to the Royal Hospital for Diseases of the Chest with a child presenting the most exaggerated features of emaciation of every part, except the abdomen, which was large and hard. She was very excited at having succeeded in gaining admission, and explained in great haste that 'all she wanted was some Pancreatic Emulsion, which she had learned could be obtained at the Royal Hospital.' She said the child had been even worse than I saw it, that everybody told her it was a hopeless case, and that she had carried it to her mother's home in the West of England, where it appeared to be slowly dying, when a charitable visitor came in and gave her a bottle of emulsion, saying that he had seen just such a case cured by it. She gave the emulsion, and the child began to improve so wonderfully that she was able to bring it back to London, where it continued to mend till her bottle of emulsion was finished, when it rapidly fell back, and became nearly as bad as ever, before she could find out where to procure more of the remedy. This she had just done, and hurried off to the Hospital. The child had diarrhœa, but she said she knew that would stop if I gave her emulsion, as it had done so before. I did as she asked—let her have as much emulsion as she wished, and the child got absolutely well. I have seen it this year, a well-grown, plump, hearty little girl. This woman has since had two other children, each of whom has in turn shown signs of marasmus like its elder sister; in one, when brought to me, the lungs presented small crepitation from end to end; but both of these children were put upon emulsion at an early stage of their wasting, and made easy recoveries.

"2. Soon after these cases occurred, Dr. Dingley, of Argyll Square, consulted me about a little patient of his in Soho, who was wasting in the same way; and as all the usual remedies, both in medicine and diet, including cod oil, had quite failed to arrest the downward progress of the case, we agreed to try the Pancreatic Emulsion. I did not see the case again, but Dr. Dingley has since informed me that from the time of commencing the Emulsion the child began to improve, and steadily progressed till it got perfectly well; and it remains well to the present day. Dr. Dingley was so impressed with the success of the remedy in this apparently hopeless case, that he tells me he has since resorted to the same treatment in all similar cases with equally satisfactory results.

"3. At the Oxford meeting of the British Medical Association, Dr. Langdon Down told me of a case that had made a great impression upon him, and it is especially important as coming from a man of his large and intimate experience in all that relates to the affections of childhood. The following note from Dr. Down graphically indicates the outline of the case :—

"'The patient at Reigate was seen by me in consultation with Mr.

Steele, in the spring of 1867. She was in the most attenuated condition I ever remember seeing. It appeared to be the extreme marasmus of mesenteric disease. The lungs were healthy. The treatment had been most judicious and exhaustive. As something which had not been tried, I suggested the Pancreatic Emulsion. The improvement was coincident with the altered treatment and was very progressive. Five months after I was asked to see her by her father, to test whether I could recognise her. She was playing croquet, and I could hardly believe that the one pointed out to me was our patient, the change was so great. She has ever since had excellent health.'

"These cases, which are well known to many persons besides myself, may appear somewhat 'sensational,' but they are only samples of numerous others which have occurred in my own practice. The fact is, that when these cases are properly selected for the treatment, they are all 'sensational;' for the rapidity with which it takes effect, and the completeness of the restoration to health of children, who appeared to be hopelessly dying, is simply startling.

"I have proved over and over again that, whether in children or adults, no amount of milk or cream, however good, will do instead of Pancreatic Emulsion, and I have tried to discover why this should be. Milk, so far as this part of its composition is concerned, is simply an emulsion of fat; and Pancreatic Emulsion, as I have shown in the paper to the Royal Society already referred to, is not, as formerly supposed, a chemical combination, but a true emulsion. Why, then, does not milk answer as well? I believe the explanation to be very simple, and that it turns upon the following points:—

"1. The fineness of the particles of fat, and the absence of albuminous envelope.

"2. The permanent character of the molecular mixture of fat and water.

"3. The proportion of fats having high melting points.

"(*a.*) In my first paper on Pancreatic Emulsion ('Lancet,' September 10, 1864), I gave the measurements (made for me by the late Mr. Farrants, President of the Microscopical Society) of the particles of fat in cod oil and beef fat emulsions, as then prepared for me; showing that the majority of the particles in the cod-oil emulsion ranged from the 16,000th to the 1,200th of an inch in diameter, and those in the beef-fat emulsion from the 10,000th to the 2,500th of an inch; and, according to Bowman ('Practical Handbook of Medical Chemistry,' p. 174), 'The size of the globules in healthy milk varies from a mere point to about the 2,000th of an inch.'

"Since I published Mr. Farrants' measurements, Pancreatic Emulsion has been made by a much more equal and satisfactory process than at that time, and I have just examined a chance specimen procured from Messrs. Savory and Moore, in which the large majority of the particles

of fat range from the 21,600th to the 14,400th of an inch in diameter, the prevailing size being the 18,000th of an inch : while in a specimen of good new milk (cold), which I have also just examined, the large majority of the particles of fat range from the 7,200th to the 3,600th of an inch in diameter, the smallest being the 10,800th.

"(*b.*) The permanent character of the Pancreatic Emulsion is very remarkable, far exceeding that of milk. It 'differs entirely from all other kinds of emulsion of fatty matter, whether chemical or mechanical. All other emulsions of fat are destroyed by ether, the fat being restored at once to its original condition. The influence exerted by the pancreas upon fats, therefore, appears to operate by breaking up the aggregation of the crystals of the fat. It alters the molecular condition of the fat, mingling it with water in such a way, that even ether cannot separate the fat from the water. *A permanent emulsion* is thus formed, ready to mix with a larger quantity of water whenever it may be added.' ('Proceedings of the Royal Society,' already referred to).

"(*c.*) In the "Chemical News," September 4, 1868, I stated my reasons for believing in the importance of fats of high melting points, such as stearine, margarine, and palmatine, over those of low melting points, such as oleine, as elements of food and medicine ; although further experiments and investigations are still needed on this interesting subject. (See p. 155.)

"Pancreatic Emulsion of solid fat, consisting principally of stearine, margarine, and palmatine, is therefore quite a different thing from milk, the fat of which is principally oleine.

"Now, the nearest approach to a pancreatic emulsion is what may be called *nascent milk*, by which I mean milk just secreted—milk that flows from the mammary gland as it is formed, or, as mothers term it, 'as the draught comes in.' In this the emulsification is finest and most perfect; but every minute that elapses after the milk is secreted deteriorates this perfection of emulsification, until, as we know, whether retained in the lactiferous ducts or in an artificial vessel, but especially in the latter, and when allowed to cool, the cream separates from the water of the milk, never again to be susceptible of the same emulsification with water in which it first existed, *except under the influence of pancreatic juice.*

"I submit that this is the secret of the superiority of lactation, and especially of lactation at the time 'the draught comes in,' over every other kind of infant-feeding, whether in man or in the lower animals. It forms an important distinction between milk-diet, supplied by the natural process of suckling, and milk-diet administered artificially, and affords some reasonable colour to the old-standing belief in the efficacy of 'new milk warm from the cow' for delicate children, and to the remarkable recoveries recorded in ancient times of old persons nourished by lactation when everything else had failed."

A remarkable confirmation of the above view with regard to *nascent milk*, which I first published in 1872, has just occurred in the results obtained by Dr. Gaillard Thomas, of New York, from his important experiments on "The Intravenous Injection of Milk as a Substitute for Transfusion of Blood." He found that—"(1) the injection of milk into the circulation in place of blood is a perfectly feasible, safe, and legitimate process, which enables us to avoid most of the difficulties and dangers of the latter operation. But (2) in this procedure none but milk removed from a healthy cow, within a few minutes of the injection should be employed. Decomposed milk is *poisonous*, and should no more be used than decomposed blood." ("New York Med. Journal," May, 1878.)

Dr. C. Meymott Tidy has furnished me with the following important case which occurred to him in 1874. I believe it is the only instance in which the milk of a suckling mother has been tested by a competent analyst and physician during treatment by pancreatic emulsion, and the results are very striking. Dr. Tidy says:—"Mrs. H., wife of bank clerk. January 15th, 1874.—Called on me complaining of considerable prostration. She was suckling her child, which was two months old. She remarked that it seemed dwindling away as though the milk would not nourish it. The quantity of milk was abundant. The question she specially consulted me about was whether, under the circumstances, it was wise for her to continue nursing. There was evidence in Mrs. H. of slight dulness at the apex of left lung. I examined some of the milk and it gave as follows:—

Total solids	8·9 per cent.
Fat	1·8 per cent.

"I prescribed iron, and advised *cream* to be taken freely.

"February 21st.—Mrs. H. called again, signs of slight improvement both in mother and child, but not very marked. I again examined the milk, which gave per cent.—

Total solids	9·46
Fat	2·30

"I then advised *Pancreatic Emulsion* to be taken three times a day.

"March 29th.—Mrs. H. called again, she said the child had made 'great strides' since the previous month. It seemed very much better. The milk was again tested and yielded—

Total solids	12·24
Fat	3·84

"I saw this patient again in July. The child was plump and fat, she had at that time commenced feeding it, but she still nursed it twice a-day, and took some Pancreatic Emulsion. The milk yielded—

Total solids	11·80
Fat	3·75"

It must be remembered that the normal average composition of woman's milk is—

Total solids	10·92
Fat	2·66

("On Foods," pp. 315–16, by Dr. Edward Smith).

It will be seen, therefore, that when this patient came under treatment, January 15th, the milk which gave on analysis—

Total solids	8·9
Fat	1·8

was defective in total solids to the extent of 2·02 per cent., and 0·86 per cent. deficient in fat; whereas, after taking Pancreatic Emulsion from February 21st to March 29th, the total solids were 1·32 per cent. in excess of normal, and the fat 1·18 per cent. in excess of normal, although during this time the woman had continued to suckle her child, which had thriven instead of wasting; and this improvement in both mother and child continued up to July, when she began to wean; and the lung disease had made no progress after the supply of fats was restored to the blood.

In the treatment of these cases of wasting in children the chloride of calcium may be used as a valuable auxiliary, as suggested at p. 244.

Having now considered the treatment of the two most important forms of Primary loss of weight—(1) the true first stage of consumption, or the initial loss of weight due to defective supply of fat from the food to the blood, and (2) the wasting of children (leading to marasmus), due to defective assimilation of fat and starch—we pass on to those forms of Secondary loss of weight, in which the supply of food and the power to carry it into the blood are intact, but in which some abnormal drain exists in the organism, such as sweating, expectoration, mucous, or purulent discharges, diabetes, diarrhœa. The treatment of these consists in supplying the materials necessary to make up the properties lost by the drain in the most readily assimilable form, and in using means to stop the drain as fast as possible. In some cases, such as diabetes and diarrhœa from ill-digested food, a medium course must be pursued, and no food must be given in the form which gives rise to the drain, while means are taken to re-establish the normal digestive and assimilative power for these forms of food.

The following rules of diet to be observed in such cases as I have been describing were laid down by me in a lecture "On some Principles of Diet in Disease," delivered at the Royal Hospital for Diseases of the Chest, in 1865:—

Rule 1.—When the power of appropriating any essential ingredient of a normal diet is lost to the organism, the lost function must be substituted by some artificial process, or the ingredient in question must

be withdrawn from the diet till the normal function is restored. In obedience to this rule we administer Pancreatic Emulsions of fat to patients, who have lost the power of assimilating fat without this artificial assistance, while we adopt all practicable means of restoring the normal function. (See pp. 225—232.)

Rule 2.—Is inseparable from the first, and it is this :—No essential of a normal diet must be withdrawn, without an attempt being made either to supply to the organism in some other way the ingredient of which it is deprived, or to suspend those functions which call for a supply of this ingredient. Thus, to take a simple illustration :—Suppose the power of digesting meat to be lost through a deficient secretion of gastric juice, meat must be withdrawn from the diet till the lost function is restored, or else an artificial digestive fluid must be introduced ; or if it is impossible by these means to maintain the digestion of meat, the physiological ingredients of meat must be supplied in the form of some albuminoid solution ; or, finally, if this cannot be done, then those functions which principally waste the albuminoid tissues of the body must be placed as far as possible in a state of rest ; muscular action must be suspended until the function is restored.

Rule 3.—If an undue waste of any elements of normal nutrition is found to be going on in the organism, and the means remain of appropriating those elements from the food, they must be supplied in the food in quantities as much in excess of those proper to the normal diet of health as will be sufficient to supply the waste, until it is stopped. In Bright's disease of the kidney, for example, there is no loss of the power to appropriate the albuminoids from the food, whereas a constant loss of albumen is going on through the kidneys, which must be met by proportionate increase of the albuminoids in the diet. But in following this rule, in this particular case, it will be necessary to observe certain precautions, dictated by a very important Principle of Dietetics, viz., to obtain rest for every organ while it is suffering under active disease, by removing from the diet such elements as increase its functions. These are conditions which it is not easy to fulfil consistently with maintaining healthy nutrition, for the healthy nutrition of the kidney requires a supply of albuminoid materials, while its function is increased by any surplus of these materials in the organism; and when its function is interrupted by disease, a proportion of albuminoids in the diet, necessary to the healthy nutrition of the organism generally, will be tantamount to an excess as regards the function of the kidney, and the accumulation of retained excretory matters will press injuriously upon the affected organ. Other medical aids than diet must, therefore, be brought to bear; some auxiliary organs which are not damaged must be stimulated for the time, to save the diseased part from undue pressure upon its functions.

Rule 4.—When through any defect in the organism, the elements of

a normal diet are lost to nutrition if presented in the usual forms, those forms must be changed; but care must be taken that in the altered forms all the essential elements of a normal diet are supplied in their proper quantities and proportions. Nothing can illustrate this better than the use of milk as a substitute for solid or mixed foods in diarrhœa or sickness. (See p. 232.)

Rule 5.—Has to deal with more complicated difficulties. If such a defect exists in the organism that *some* of the essentials of a normal diet are misappropriated, so that the organism is deprived of one or more of the normal elements of nutrition, and at the same time a disease is constituted out of the misappropriated food, then we have a double duty in interfering with the diet. First, disease must be stopped at its source by withdrawing that part of the diet out of which it is constituted; and, secondly, the elements of nutrition thus removed must be supplied by some other means or in some other form. Thus, in diabetes, the saccharine and amylaceous elements of the diet are misappropriated; they do not serve their normal function of supplying carbon for the evolution of heat, and by passing off through the kidneys they constitute an exhausting disease. It is necessary, therefore, to stop the source of this disease by cutting off the saccharine and amylaceous ingredients of the diet till normal nutrition is restored. But, in the meantime, as carbon must be obtained by some means, it is taken from the fat stored up in the body, so long as that lasts, and when it is gone, from the albuminoid tissues themselves, till the whole organism is disintegrated; unless at the same time that we cut off the starch and sugar, we increase the quantity of *fat* supplied in the food as much in excess of the proportion proper to a normal diet as shall fully supply the demand. (See p. 200.)

The modern dietetic treatment of diabetes may be taken as a good example of the way in which increased knowledge of the nature of disease and of the physiology of food enables us to avoid any unnecessary reduction in the number and variety of the forms in which food can be taken. In former days the poor parched diabetic was forbidden to drink water lest he should increase his flow of urine; now we are able to let him quench his thirst as much as he pleases, so that he takes nothing which contains starch or sugar; and again, by preparing his articles of food in such a manner as to exclude the injurious ingredients, and by selecting those which are known to contain them in the smallest quantities, or not to contain them at all, we are able to present the diabetic with a fairly tempting and varied diet, so that he is able to keep to it for months and years with comparatively little difficulty. (See "Diet for Diabetes," at p. 104 of the Sixth Edition of my work on "Diet and Regimen in Sickness and Health.")

Treatment of the Lung Diseases connected with Blood-Spitting and Loss of Weight.

We have seen that in our battle (see p. 68) with the lung diseases connected with loss of weight and blood-spitting, it is principally with the following conditions that we have to fight :—

1. Abnormal physiological states, primarily connected with the digestive and assimilative processes, producing constitutional decline.
2. Disintegrations of tissues and organs dependent upon abnormalities in the nutritive processes.
3. Abnormal growths of adenoid, epithelial, and connective tissue, and the results of abortive formative processes.
4. Catarrh, hyperæmia, inflammation, gangrene.
5. Decomposition of the débris of disintegrated tissues, of the products of disease, and of the attempts at repair above referred to.
6. General septic disease.
7. Secondary local diseases of septic origin.

Associated with the discussion of these conditions will be found the principles and means of treatment.

My reasons for abstaining from dictating the *details of treatment for individual cases*, I have often stated in other places, and in the following passage of my work "On Winter Cough, Catarrh, Bronchitis, Emphysema, Asthma," (1866), I especially emphasised my opinion on this point:—

"I do not believe in the possibility of adapting the exact details of treatment to particular cases, without taking into consideration and carefully balancing all the circumstances of each case to an extent which it is impossible to do in lectures and books, or in any other way, *if the patient is not before us.* I will not pretend, therefore, to direct the exact cases in which this or that remedy, or combination of remedies is to be used. To do this is, in my opinion, very much like the folly sometimes perpetrated by Governments of issuing, from their offices at home, orders for the exact mode in which their generals, abroad, shall conduct their battles. It has always ended in defeat."

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