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Original Articles

HEMOPTYSIS DUE TO TUBERCULOSIS.

A PRELIMINARY STUDY.*

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PHILADELPHIA.

The pathologic etiology should naturally engage our attention first and foremost in a discussion of the subject of hemoptysis dependent on tuberculosis. Broadly speaking, we may subdivide the cases in (a) those occasioned by congestion of the bronchial mucosa and lung texture and (b) those due to ulceration or erosion of vessels, or rupture of miliary aneurisms.

BLEEDING DUE TO PULMONARY CONGESTION.

When hemorrhage takes place before gross lesions are detectable by means of the physical signs or the x-ray in tuberculosis, we ascribe them offhand to congestion of the bronchial mucous membrane and of the lung texture. The earliest pathologic changes in this disease are not directly connected with blood vessels, although the adjacent tissues are the seat of inflammatory processes which may manifest a hemorrhagic tendency. The query is pertinent: Can we ascribe the principal hemorrhages of the earlier stages to congestion?

It may be reasonably questioned, as pointed out by Garland, whether in point of intensity the hyperemia of early pulmonary tuberculosis is equal to that of many other acute and chronic diseases of the lungs in which the tendency to free bleeding is decidedly less marked. It is probable that minute areas of necrosis are already in evidence and responsible for these early hemorrhages. At all events, in cases in which hemoptysis appears before the occurrence of any other initial symptoms of pulmonary tuberculosis, the x-ray shows the presence of the characteristic opacities, and tubercle bacilli can often be isolated from the sputa during and after the hemorrhage. That a high grade of congestion is a factor, however, is seen from the significant influence exerted by violent or prolonged physical exercise in the production of hemoptysis, both in tuberculous and non-tuberculous subjects.

This brings us at once to a realization of the fact that the heart can not be omitted from consideration in the pathogenesis of hemoptysis due to tuberculosis. As elsewhere remarked,¹ the cases of lung tuberculosis that are associated with chronic valvulitis affecting the mitral segments manifest symptomatic hemoptysis more commonly than the ordinary uncombined type of the disease, thus showing apparently the effects of marked

hyperemia. In the congestive bronchitis that attends chronic valvulitis and also that of the collateral septic processes in phthisis, the lumen of the vessels and capillaries is enlarged and this may lead to small hemorrhages, but the usual course is undoubtedly the pulmonary vessels (*vide infra*). The current opinion that the earlier bleedings in the course of pulmonary tuberculosis are dependent on congestion of the bronchial mucous membrane does not rest on secure pathologic grounds. It is to be recollected, however, that high tension in the pulmonary circuit leads inevitably to vascular sclerosis of the pulmonary vessels so that deficient nutrition at length favors leakage and consequent hemoptysis. Now, it is known that the walls of blood vessels are early affected, i. e., weakened by endarteritis in pulmonary tuberculosis; and, "while there are as yet few proteins and the living bacillus plays the chief rôle, the healthy wall of the smaller pulmonary vessels is apt to become invaded and eroded, giving rise to a hemorrhage" (G. Cornet). It is not improbable that hemorrhage may at times proceed from poorly supported capillaries or a want of outside pressure.

HEMOPTYSIS DUE TO ULCERATION OR EROSION OF VESSELS AND RUPTURE OF MILIARY ANEURISMS.

Rasmussen² first showed that fatal pulmonary hemorrhage is probably always caused by rupture of an aneurism in a lung vessel. Brown³ has also emphasized that few, if any, "well authenticated cases of fatal hemoptysis have occurred from rupture of the pulmonary vein or bronchial artery."

The method of growth of the tuberculous granulomata into the caliber of vessels explains satisfactorily how rupture of their walls, with ensuing hemorrhage, may occur, even at a comparatively early stage of the disease. The less frequent occurrence of pulmonary hemorrhage during the advanced stage of tuberculosis is ascribable to endarteritis which is slowly developed, with resulting thickening of the vascular walls, hence offering increased resistance to the invading forces.

R. E. Thompson⁴ has long since pointed out that no case of profuse hemorrhage proceeds solely from the bronchial artery. It must be remembered, however, that where there is a general tendency to hemorrhage a portion of the blood may come from the bronchial mucous membrane.

It is especially true in cases that pursue a rapid course or those in which the processes of softening and liquefaction progress rather acutely that bleeding from eroded vessels is most apt to arise. In these instances the vascular walls are not the seat of endarteritis owing to the short duration of the tuberculous process. In the more serious and fatal hemorrhages we may infer the exist-

* Read before the American Climatological Association, May 7, 1907.

1. Edinburgh Med. Jour., 1868.

2. Anders: American Jour. Med. Sciences, Jan., 1902.

3. Ibid., Aug., 1906.

4. Causes and Results of Pulmonary Hemorrhage, Smith, Elder & Co., 1879.

ence of rupture of vessels due to pathologic changes in their walls leading, as a rule, to aneurismal dilatation before the accident occurs. The development of the latter condition is favored by the removal of the tissues which serve as an external support to the vascular walls.

Finally, the dislodgment from quiescent or healed cavities of calcareous masses may be followed by slight hemorrhage, most probably occasioned by the rupture of capillaries.

EXCITING FACTORS.

It is undoubted that in a considerable percentage of cases of pulmonary tuberculosis there is a hemorrhagic tendency, and in such a veritable hemoptysis may be induced by increased pressure in the pulmonary circuit. That hemoptysis may manifest dependence on an hereditary taint is a matter of personal observation; this is especially true of families that show hemorrhagic proclivities. In several cases under my observation, early pulmonary bleedings (i. e., before the physical signs and the x-ray gave indications of pathologic changes) were associated with hemorrhage from other mucous outlets as the stomach and nose. Hemoptysis may have, as its exciting factor, an aggravation of the cough. Dobell found that not less than 76 out of a total of 90 cases of hemoptysis were excited by this symptom.

In my cases, muscular exertion, more especially if long continued or combined with fits of passion or mental excitement, has preceded the occurrence of symptomatic hemoptysis in a considerable percentage of cases and it deserves to rank higher as an exciting factor than is generally believed. In these instances we may attribute the leakage to congestion with rupture of diseased vascular walls provoked by exertion. Indeed, in one of my cases a fatal hemorrhage occurred in a female patient, aged 25 years, after a hurried walk to church about a mile distant. Although no postmortem examination was permitted to furnish positive proof, the existence of a small cavity in the apical region of the right lung explained the pathologic cause of the accident.

Franz Stricker made a statistical study of this symptom in relation to tuberculosis covering a period of five years (1890-95) in the Prussian army. The total number of cases admitted to the hospital was 900. In 480 cases the hemorrhage developed without recognizable cause, and, of these, 417 cases (86 per cent.) were certainly or probably tuberculous. Stricker draws the following, among other, conclusions, namely: "That soldiers attacked with hemoptysis without special cause are in at least 86.8 per cent. tuberculous. In the cases in which the hemoptysis follows the special exercises, etc., of military service at least 74.4 per cent. are tuberculous. In the cases which come on during swimming or as a consequence of direct injury to the thorax, about one-half are not associated with tuberculosis." As pointed out by Frick, "so long as a tuberculous subject has not spat blood he is able to persuade himself that he has not consumption, but after he has spat blood he takes it for granted that he has the disease." He continues: "There is really more reason for this popular verdict than we would at first sight think. Blood spitting is undoubtedly, in some cases at least, due to mixed infection and frequently occurs with the breaking down of tissue." Flick, Ravenal and others hold that hemoptysis is generally caused by a secondary infection with the pneumococcus. In many cases, however, exciting factors, if any be at work, are not obvious.

INCIDENCE.

Hemoptysis is universally held to be a frequent symptom in tuberculosis. According to a table given in the Second Annual Report of the Henry Phipps Institute, it was present at one time or another in 1,130 out of 2,344 cases. In proof of the extreme frequency of the occurrence of hemoptysis, I desire to present certain statistics which include 214 cases from my private case records, 300 from the out-patient department of the Medico-Chirurgical Hospital (covering a period of six years) and 78 from the practice of Dr. A. H. Lowenburg, who is in charge of the dispensary service, making a total of 589 cases; of these, 169 of the patients, or 28.7 per cent., manifested blood spitting. These cases, however, were not all followed until the death or recovery of the patient; hence the percentage in which hemoptysis occurs at some period throughout the entire course of the disease must be much higher. For example, if we consider only the cases from private practice, numbering 289, the percentage rises immediately from 28.7 to 41.8, but it is to be recollected that even these patients were under observation less than half of their duration; hence, I feel strongly that Osler is probably not far wrong in stating that it is a feature in from 60 to 80 per cent. of all cases of tuberculosis.

AGE AND SEX.

Of 1,084 cases of tuberculosis studied by Thompson, the female sex was found to show greater liability to hemoptysis than the male "in the relation of 65 to 62 per cent." Of the total number in which the sex was given (589 cases), my figures show the following: 1. That the liability to tuberculosis is shown at an earlier period of life in the female than in the male, in the proportion of 10 to 6 before the twentieth year and 7 to 1 prior to the tenth year (Table 1 and Fig. 1). 2. That hemoptysis is manifested earlier in life, probably dependent on increased incidence in the female sex during the first two decades as compared with the male (Table 2 and Fig. 2).

TABLE 1.—AGES BY DECADES AT WHICH TUBERCULOSIS OCCURRED.

	0-10	10-20	20-30	30-40	40-50	50-60	60-70	70-80
Males	1	32	154	106	45	12	6	0
Females	4	32	87	61	25	9	2	0
Total	5	64	241	167	70	21	8	0

TABLE 2.—AGES BY DECADES AT WHICH HEMOPTYSIS OCCURRED.

	0-10	10-20	20-30	30-40	40-50	50-60	60-70	70-80
Males	0	7	53	40	15	3	2	0
Females	0	11	30	22	10	4	0	0
Total	0	18	83	62	25	7	2	0

My observation and statistical studies go to show that profuse and fatal hemorrhages are somewhat less common in the female than the male sex, although the sexual differences in this respect are less pronounced than certain writers would lead us to believe.

SEASONAL INFLUENCE.

The occurrence of hemoptysis considered in relation to the seasons has been the subject of statistical study. My own figures (Table 3 and Fig. 3) give a summary of 157 cases for each month of the year.

TABLE 3.—SHOWING SEASONAL INFLUENCE ON HEMOPTYSIS.

Jan.	Feb.	March.	April.	May.	June.	July.	Aug.	Sept.	Oct.	Nov.	Dec.
11	9	14	19	13	16	14	16	16	8	11	10

A glance at the above figures can not fail to show a seasonal influence on the prevalence of pulmonary hemorrhage. It will be observed that the spring and summer months give the highest coincidence and this is in consonance with accepted opinion regarding the relax-

ing and enervating effects of heat as well as the influence of the marked oscillations of temperature and humidity during the spring months as causative factors. Suddenly, extreme changes of weather bring about intercurrent acute bronchitis and an aggravation of the cough which tends to excite bleeding.

Conversely, the months exhibiting a steady, stimulating cold—December, January and February—have a preventive influence, as shown by their smaller numbers. I should add that the above figures concerning the influence of the seasons on pulmonary hemorrhage strongly corroborate the points brought out by Thompson in his statistical inquiry in England, although the latter was not confined to hemoptysis in the course of tuberculosis.

The strong tendency to recurrences of the hemorrhage is shown by my statistical inquiries. Thus of the totality of cases 589, a second outburst occurred in 292, or about 50 per cent. Considered with regard to the stage of the tuberculous process, my figures indicate that in 75 per cent. of the instances hemoptysis appeared in the first and early part of the second stage, or that of softening. In this respect, our collective investigations support the

I and others have since confirmed, that hemoptysis, even when considerable in amount, does not seem to exert any influence on the reduction of temperature in the majority of cases at least. Advanced cases sometimes show a fall of some extent, which may last for a period of several days. Now, while in some instances no variations of temperature occur and in a few a slight lowering, it is a well-known thermometric observation that fever and often decided pyrexia develops during and immediately after the blood spitting. Moreover, a moderate depression of temperature may be promptly followed by a notable rise, lasting a number of days.

The explanation of these elevations of temperature offers notable difficulties. When of brief duration they may be occasioned by alarm and demoralization of the patient, as shown by cardiac palpitation, mental agitation and other nervous concomitants. In the case of large hemorrhages which induce marked symptomatic anemia, the ensuing restlessness and febrile disturbance have as an added causative factor the anemia. These traditional explanations for the occurrence of fever, however, need to be further investigated. But the query arises, What new theory can be advanced on rational, clinical and pathologic grounds? May not fever, when a sequel of hemoptysis, be symptomatic of a fresh area of tuberculous inflammation occasioned by the liberation of infective material which is conveyed partly by grav-

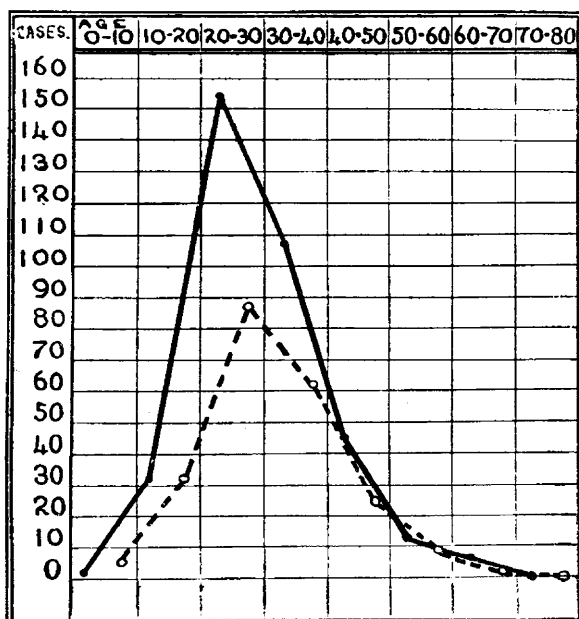


Fig. 1.—Chart based on Table 1 showing sex-ages by decades at which tuberculosis occurred. Solid line indicates males; broken line, females.

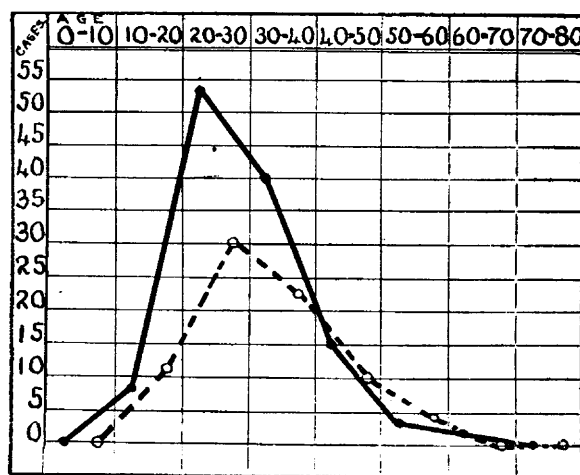


Fig. 2.—Chart based on Table 2 showing sex-ages by decades at which hemoptysis occurred. Solid line indicates males; broken line, females.

views expressed above on the subject of the pathogenesis of the condition.

CLINICAL PECULIARITIES.

We must look on hemoptysis as being the resultant of existing tuberculosis, although neither symptoms nor physical signs may betray the presence of the latter disease prior to the occurrence of the blood spitting. This dictum was originally advanced by Laennec and Louis and later by Traube. Many of these patients have no recurrence and subsequent symptoms of tuberculosis do not develop; such may be regarded as instances of spontaneous arrest or even cure of the affection. Again, hemoptysis is commonly followed by the characteristic evidences of chronic phthisis or it may be after the lapse of months and even years that the rational symptoms and physical signs of lung disease first appear.

Wilson Fox⁶ made the interesting observation, which

ity, but chiefly by aspiration to the surrounding bronchioles and air cells? The daily exacerbations of temperature, sometimes observed for days or even weeks after a hemorrhage, may also, it seems to me, be attributable to localized structural alterations, which permit the absorption of toxic substances at the seat of the ulceration or erosion. As before stated, the majority of the bleedings occur after secondary infection with the streptococcus has occurred.

I hold it to be exceedingly difficult to determine the source of the blood in a minority of the cases of pulmonary hemorrhage, and in a case seen recently the blood was thought for a time to come from the stomach. The points of detection to be found in text-book literature are: A tickling about the fauces, a saline taste, blood brought into the mouth by the act of coughing, a bright red color appearance, frothy and mixed with mucus. The character of the blood expectorated may not give unfailing proof of its origin, however, and this is espe-

6. Temperature of Phthisis, *Medico-Chir.*, Trans. lvi, 399.

cially true of a sudden, copious, pulmonary hemorrhage. In these cases, the color of the blood may be dark, not having been exposed to the air in the lungs long enough to be changed to arterial brightness. In dubious cases of bleeding of moderate grade, rigid search is to be made for bits of mucus, from which either bacilli or elastic tissue may be isolated. A blood-tinged sputum during the subsequent few days—a common sequel—is a point of great support in hemoptysis and the same is equally true of evidence of pre-existing lung disease.

According to personal observation, it is beyond doubt that there is a form of hemoptysis, which recurs at long and regular intervals of time; this might appropriately be termed recurrent or, since all cases tend to recur, periodic hemoptysis. Although far from common, brief reference should be made to it for prophylactic and therapeutic reasons. It is clear that unusual watchfulness at the time of the expected hemorrhage would be effectual in preventing recurrences. Whether this clinical variety is separated from the usual type either etiologically or pathologically, I am unprepared to state. In the cases observed, the intervals between attacks have

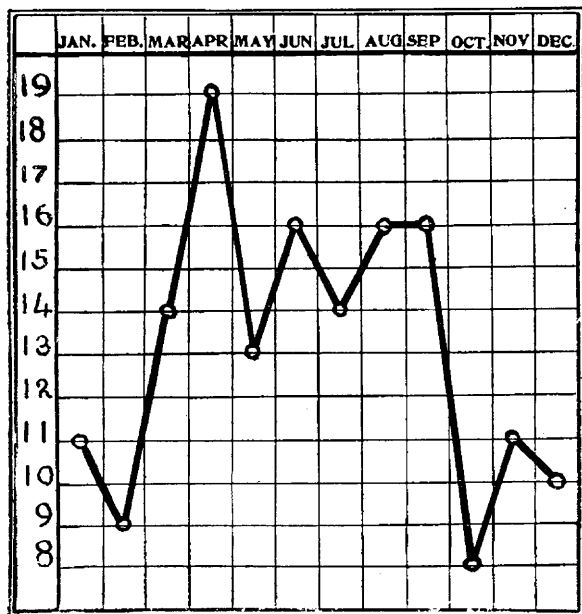


Fig. 3.—Chart based on Table 3 showing seasonal influence on hemoptysis in a total of 157 cases.

been one year, as a rule, and the most favored season, spring. The physical signs of adhesive pleuritis at the base were found in association in two of these cases and the pulmonary lesions slight and practically non-progressive. Indeed, the rather free bleedings were invariably followed by decided improvement which lasted for months together.

SUMMARY OF TREATMENT.

A retrospective view of the various methods of treatment employed during a comparatively brief period of time indicate clearly a notable diversity of opinion on the subject. It must be admitted also that certain well-founded principles and facts are but dimly recognized by the present generation of physicians. For example, the bleeding from the hyperemic lung texture affords decided relief to the local pathologic condition, but more than this symptomatic improvement lasting weeks and months may be a secondary result. Again, the profession has, it seems to me, an imperfect notion of the

therapeutic value of absolute rest in this condition, as well as of appropriately restricted feeding, with light, unwarmed articles of diet.

Prophylactic measures of signal importance to the sufferer from hemoptysis are, I fear, systematically and inexorably neglected by the mass of the profession. The avoidance of undue muscular exertion, of the free use of stimulants and strong mental excitement is to be strenuously enjoined. Certain climatic elements exercise a favoring influence and these are to be adversely considered in making a selection of a suitable habitat for the individual patient. Curtin long since pointed out that a residence far removed from the sea coast is best for patients suffering from hemoptysis, and, further, "that a rarified, but also a cold, dry, aseptic air would be most useful."

The remedial treatment is unsatisfactory and unpromising in view of the fact that, while numerous medicinal agents have been recommended, but few have given reliable evidence of their efficacy. It is in order that we put into force the few whose curative virtues have been proven. Unless there be obvious danger of inundation of the uninvolved lung tissue, as in cases of profuse hemorrhage, the cough should be arrested by the use of codein or, if troublesome, by morphin administered hypodermatically. It is necessary to stop disturbance of the bleeding point by coughing in order to give opportunity for the formation of a clot—Nature's own method of arresting hemorrhage. Hot drinks and alcoholic stimulants, if previously taken, must be intermitted.

My own best results during the early active stage of hemoptysis have been obtained from the strict enforcement of absolute recumbency and quiet, the patient not being allowed to speak aloud nor to put forth any muscular exertion, and an arrest of the cough by codein in the milder forms and morphin hypodermatically in the severer cases. The single contraindication to the use of opium has been pointed out, and if we accept the small class of cases in which it obtains, morphin or opium in some form, rightly administered, is the most serviceable single remedy at our command; its signal virtue, I repeat, being ascribable to the remarkable enhancement of a coagulum at the seat of bleeding. I feel confident that the perils, near and remote, incident to copious or otherwise protracted bleedings can be most successfully obviated by its judicious employment. My earnest plea is for a wider use and closer attention to the practical application of this sovereign agent. The importance of controlling the cough receives striking confirmation from the investigation of Dobell, previously mentioned. J. B. Walker⁷ also emphasized the value of opium in the treatment of hemoptysis in a paper read in 1889.

During the first twenty-four or forty-eight hours, according to the size of the hemorrhage, the patient should be kept well under its influence, the object being to stop the cough. Meanwhile the thorax must be carefully and frequently auscultated, and should abundant, moist or bubbling râles be audible over the previously uninvolved portions of the lung—a rare event except in case of rupture of a miliary aneurism—it is to be regarded as a signal for the withdrawal of the opiate.

While the arterial pressure in the lesser circulation is less than in the greater or the arterial system, it is subject to decided variations. The hypertrophy of the right ventricle which develops in the course of pulmonary tuberculosis it is fair to assume maintains an abnor-

7. Treatment, Other Than Climatic, of Hemoptysis in Chronic Pulmonary Disease, Trans. of the Amer. Climatological Assn., 1889.

mally high degree of pressure in the cardiopulmonary arc. This increased pressure may be also brought about by remedies that produce vaso-constriction in the peripheral vessels, such as ergot, and it is most probably accentuated by the forcible action of the heart in consequence of nervous excitation occasioned by hemorrhage. It is clear that a pure cardiac sedative, e. g., aconite, by lowering the tension in the lung vessels, must meet an important indication, and it may be prescribed with much confidence in its efficacy. I have found aconite to be highly serviceable in one-drop doses every third hour; it is especially useful in cases presenting fever. Lawrason Brown⁸ suggests "that the blood pressure be frequently observed, that morphin be used when necessary to quiet the patient and so equalize the blood pressure, that sodium nitrite be exhibited to reduce when necessary the blood pressure, and that in case of a sudden hemoptysis amyl nitrite be administered at once when possible, to produce a marked fall in the blood pressure and so aid in a temporary cessation at least of the hemoptysis."

N. A. Johanson⁹ states that, in his experience, atropin in doses of gr. 1/50, repeated every twelve hours, has stopped hemorrhage when all other remedies have been of no avail. R. H. Babcock gives an immediate injection of atropin sulphate (gr. 1/50 to 1/25) when hemorrhage occurs from a cavity.

Successful treatment of the case in question must always be preceded by its reference to one of two categories previously mentioned, to-wit: (1) in which the hemoptysis is due to congestion or erosion of the smaller pulmonary vessels (most common); (2) in which there is profuse hemorrhage due to erosion of the larger pulmonary vessels or rupture of a small aneurism (comparatively rare).

The differentiation is greatly aided by remembering that the first class is composed principally of the early-appearing cases, while the second class represents the cases which, in addition to showing profuse hemorrhage, occur at an advanced stage of this disease, as a rule. As stated in other connections in this article, the point of highest importance in the treatment of hemoptysis according to the nature of the lesion producing it is that in profuse hemorrhage proceeding from one of the larger vessels or a ruptured aneurism, the cough is to be encouraged rather than stopped. Opiates are contraindicated.

SYSTEMIC BLASTOMYCOSIS AND COCCIDIOIDAL GRANULOMA.

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INTRODUCTION.

Since the description by Gilchrist in 1894 of blastomycetic dermatitis numerous instances of this infection have been recorded by American observers. The clinical course, the characteristic double-contoured, budding organisms, and the distinctive gross and microscopic anatomy—especially the marked epithelial hyperplasia, the minute epidermal abscesses, and the peculiar granulomatous infiltration of the corium—early established the entity of blastomycetic dermatitis so firmly that denial of its existence, especially by certain German writers, never has received serious notice. H. G. Wells discov-

ered the first case in Chicago, and the large number of cases observed in this city, especially by Professor Hyde and his associates, is noteworthy. Whether this apparent relative frequency of the disease here is in any way due to accidental conditions or to our being in the midst of an especially heavily infested territory remains to be seen.

The interest in blastomycetic infection enlarged when it became known that this infection may be systemic. Since 1902, when J. W. Walker and F. H. Montgomery reported the first case, nine cases of systemic blastomycosis have been reported from Chicago and its vicinity, and it is my purpose in this short communication to present in summary form the essential facts concerning these and certain other cases and to make certain comparisons with the so-called coccidioid granuloma of California. Busse's well-known case of "saccharomycosis hominis," reported in 1894, and studied also by Buschke, clearly resembles, in many important details the cases reported from Chicago under the general name of systemic or generalized blastomycosis, and it is therefore placed first in the tabular summary. Curtis' case of human saccharomycosis also appears to belong to this group, and is therefore included in the list.

SUMMARY OF RECORDED CASES OF SYSTEMIC BLASTOMYCOSIS.

1. BUSSE-BUSCHKE (Busse, *Virchow's Archiv*, 1896, cxliv, 360; Die Hefen als Krankheitserreger, 1897; Buschke, Die Hefemykosen bei Menschen und Tieren, *Falkmann's Sammlung klin. Vorträge*, No. 218, 1898; Buschke, Die Blastomycose, 1902.); Woman, aged 31, housewife, born and lived in Germany.

Onset and Course of Disease.—Soft tumor below the knee containing glairy, reddish fluid; later disease extended to knee joint; abscess near elbow, also in left sixth rib; according to Buschke, ulcers, sometimes healing spontaneously, would appear on neck and forehead previously to development of tibial abscess. Busse describes ulcers on face as small and round with raised borders; coalescence formed larger sensitive ulcers; subcutaneous foci would form ulcers. Death.

External Lesions.—Borders of cutaneous ulcers sharp, irregular, undermined and infiltrated, surrounded by livid zone; bases composed of soft granulation tissue; numerous double-contoured parasites, often budding, in sections which on surface showed fibrin, polymorphonuclear leucocytes and red corpuscles, and a little deeper, giant cells, leucocytes, and small abscesses; epithelium either destroyed or proliferating.

Internal Lesions.—Abscesses in the right ulna, tibia, left sixth rib; abscesses and minute nodules in lungs, left kidney and spleen. No tubercle bacilli.

Microbiology.—Beginning cutaneous ulcers covered by crust, always gave pure culture of fungus. Pure cultures from abscesses in ulna and rib and from certain skin lesions, the organism proliferating chiefly by budding; Buschke, however, speaks of mycelium with cornidia; no endospores. Fungus not found in urine during life, but eight weeks before death a pure culture was obtained from the left median vein (Buschke). Organism virulent for dogs, rabbits, guinea-pigs, cats, white mice, causing inflammatory changes, the multiplication being by budding. Alcohol and carbon dioxide produced in grape sugar solution. Typical ulcers produced experimentally on skin of patient by rubbing in pure cultures (Buschke).

2. CURTIS (*Ann. de l'Inst. Pasteur*, 1896, x, 448); Male, aged 20.

Onset and Course.—Tumors in various parts of trunk, neck and extremities arising in the course of a few months. Death presumably from internal growths or meningitis after one and one-fourth years. No autopsy.

External Lesions.—Skin in places intact, in places ulcerated over tumors or abscesses, some of which were soft, others firm. Tumors composed almost wholly of organisms, intracellular and extracellular, the latter often double-contoured.

Microbiology.—Cultures obtained with which could be produced in white rats growths resembling those in the patient as well as disseminated internal nodules. No sporulating forms.

3. J. W. WALKER AND F. H. MONTGOMERY (F. H. Montgomery, *Jour. Cut. Dis.*, 1901, xix, 26; J. W. Walker and F. H. Montgomery, *THE JOURNAL A. M. A.*, 1902, xxxviii, 867); Man, 33 years old, resident of Chicago, carpenter.

Onset and Course.—Seven years before coming under observation pimple developed in skin of right shoulder following irritation and scratching; crusting and slow increase in size up to about silver dollar when stationary for years, then growth again particularly between scapulas. Recurring attacks of chills and fever lasting a few days, with signs of pulmonary involvement began 43 days before death, soon after curetting of original lesion. During this period lesions occurred under the right eye, in left temple, and in back. Lesions at first subepidermal, elevated, red, semiglobular, later epidermis fell off, leaving deep red surfaces soon assuming verrucous appearance. Emaciation, dyspnea, death. Diagnosis: Miliary tuberculosis.

External Lesions.—Original lesion raised, firm, well-defined, surfaces in places smooth, cicatricial, in others warty. Microscopically cellular infiltration in the corium with minute focal accumulations, epithelial proliferation, giant cells, and double-contoured, large organisms.

8. *Amer. Jour. Med. Sci.*, August, 1906.

9. *Denver Med. Times and Utah Med. Jour.*, June, 1906.