Iconograms : a collection of colored plates illustrating interesting surgical conditions / explanatory text, with special reference to diagnosis and treatment, by Faxton E. Gardner.

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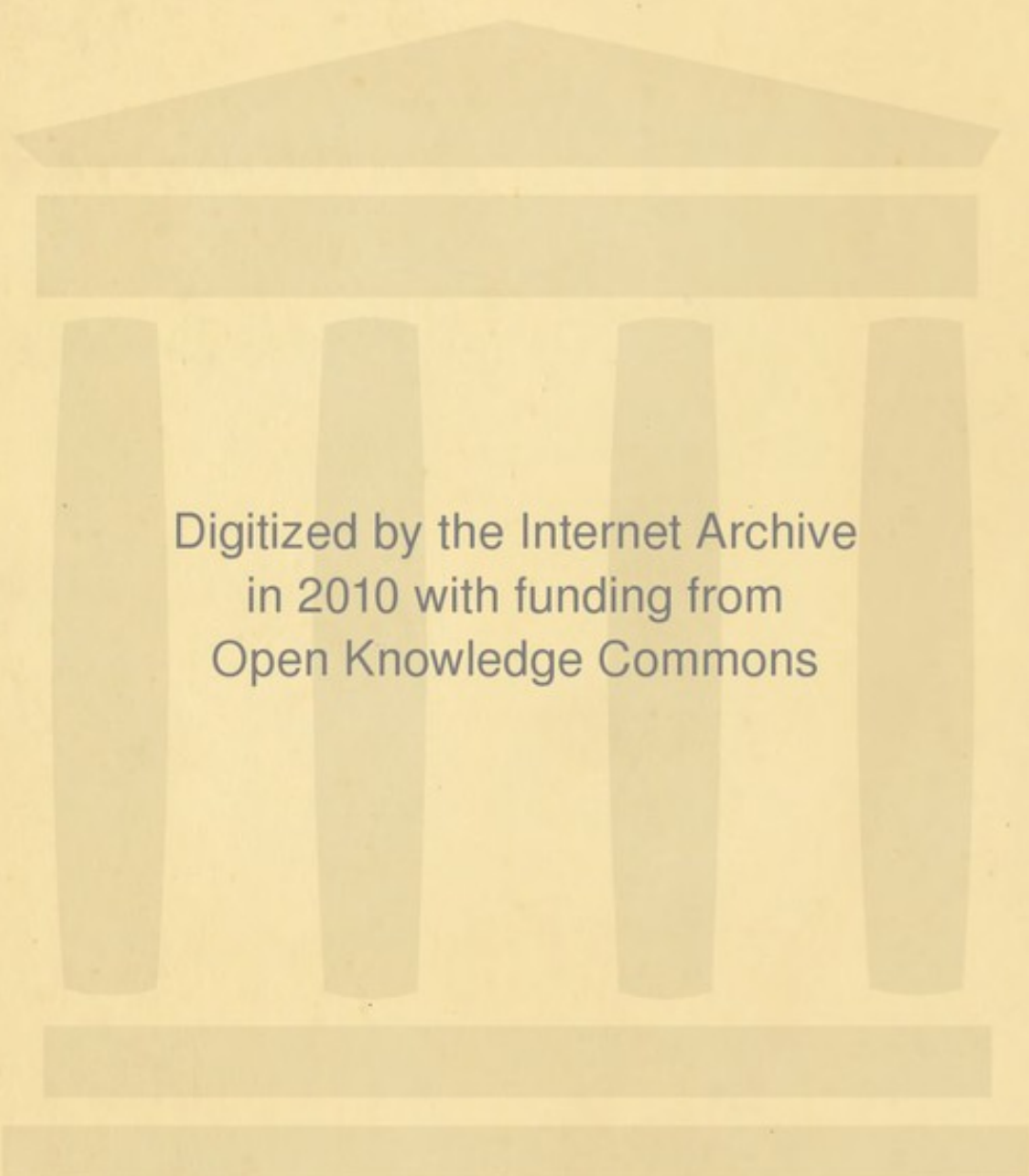
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ICONOGRAMS

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A Collection of Colored Plates

FROM

Prof. BOCKENHEIMER'S

"ATLAS DER CHIRURGISCHEN HAUTKRANKHEITEN"

ILLUSTRATING

Interesting Surgical Conditions

EXPLANATORY TEXT

WITH

Special Reference to Diagnosis and Treatment

ADAPTED AND REVISED BY

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FOREWORD.

This Atlas is not a text book, still less a series of extensive monographs.

No claim is made that any subject has been exhaustively covered.

The sole aim of the text is to set forth, in as concise a form as possible, the essentials of diagnosis and treatment, and, by comparison with the plates themselves, to establish in the reader's mind an association between the objective aspect of a condition and the two points just referred to—which association cannot but be helpful when a similar case occurs in actual practice.

As a basis the plates in Bockenheimer's Atlas of Clinical Surgery have been used. The order of the plates has not been changed. Considerable thought was, however, devoted to this point. At first it was believed that another grouping of the figures (by anatomical regions, for instance) might prove more didactic and thus enhance the practical value of the book, but, after several attempts, it was found that no arrangement was perfect—each one proved artificial in some respects and had its drawbacks as well as its advantages. The idea was then abandoned.

The text has been completely remodelled. Things move rapidly nowadays in medicine. There is no subject on which the past five or six years have not brought some enlightenment; a few have, indeed, been absolutely revolutionized. The rewriting has been done from the standpoint of American methods, reference to which is, unfortunately, very scant in most Continental treatises.

No theorizing has been indulged in, except when a direct relation to diagnostic or therapeutic principles was involved.

In a book of this kind, comparison of different plates with each other is always very fruitful. This is the reason why all references to plates have been printed in bold type, by which means their importance is clearly and duly emphasized.

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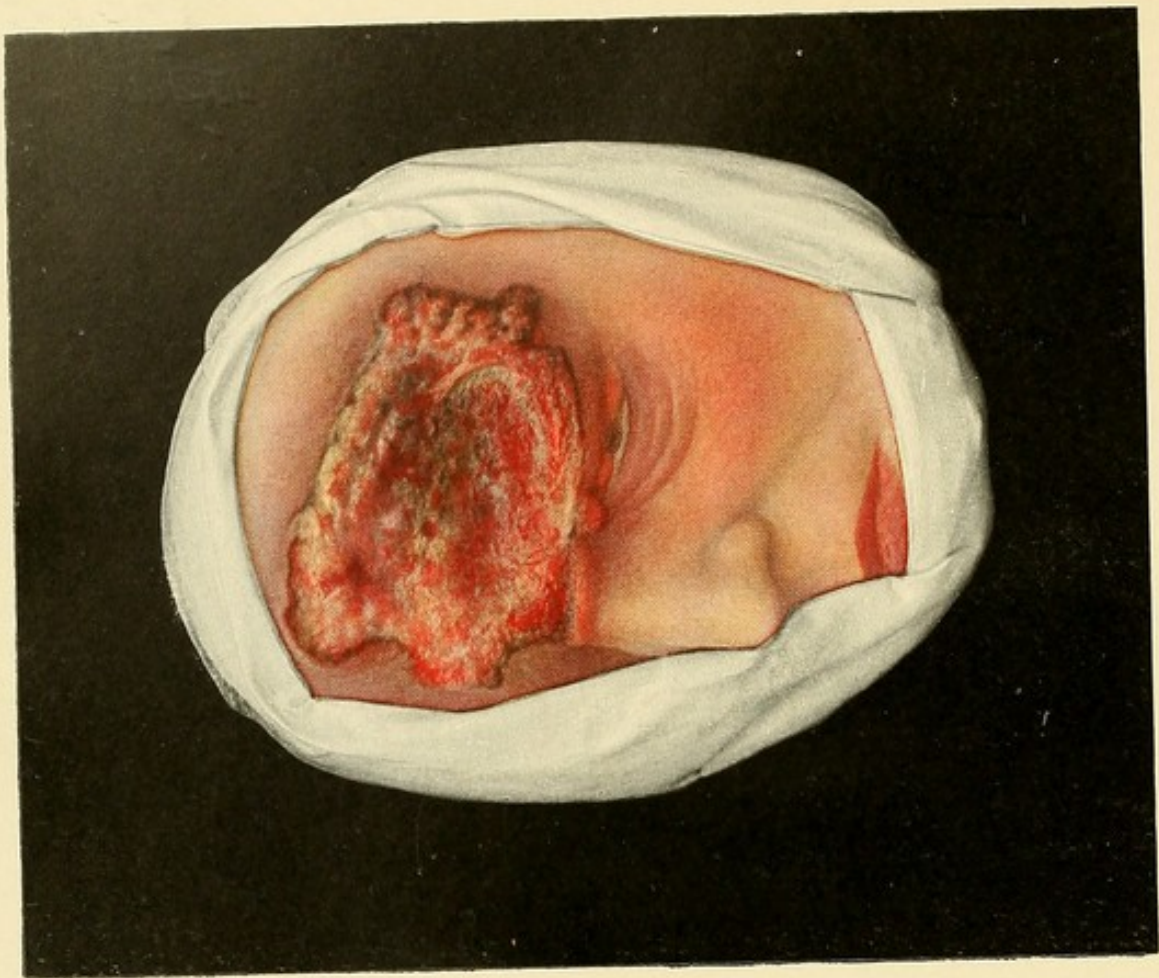
Fig. 2. Carcinoma frontis.
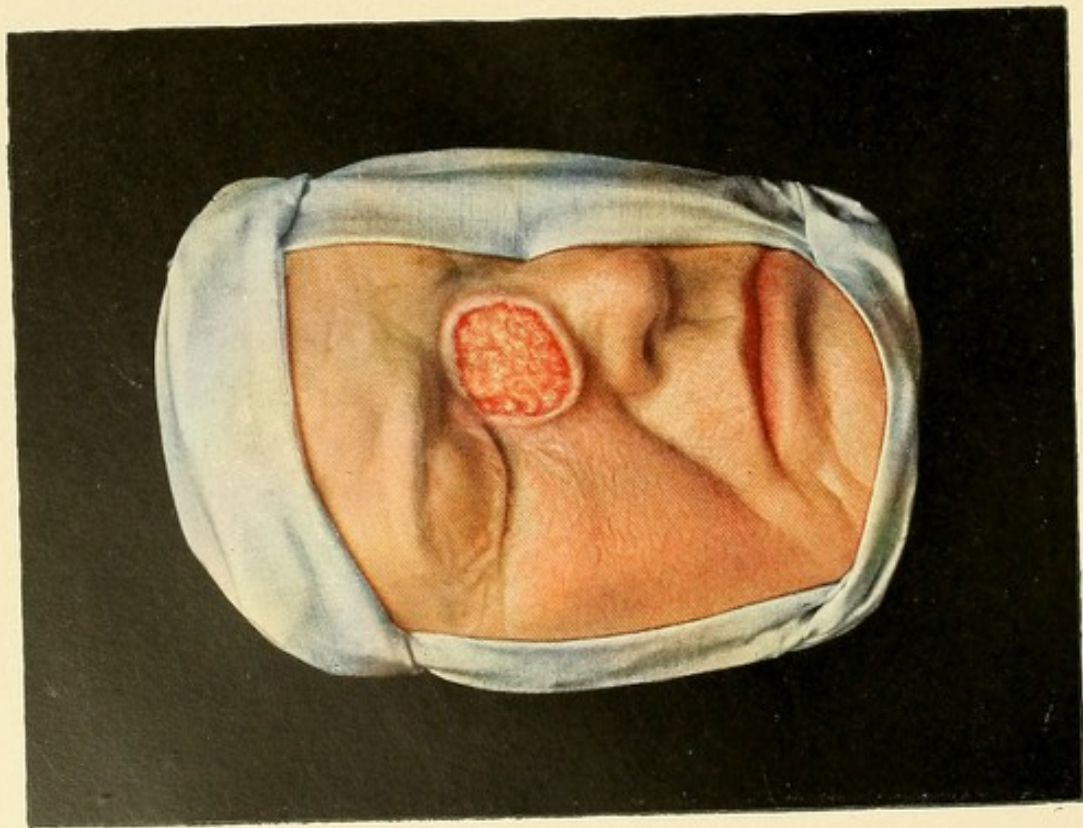
Fig. 1. Carcinoma planum faciei.

CARCINOMA

Figs. 1 to **5,** inclusive, depict types of **cutaneous carcinoma affecting the face.**

Cancers of the face are of great importance, because of their frequency. The nose, eyelids, cheeks, temples and forehead are the most common sites, while the chin and ears are least affected. In youth, these tumors are very rare; when they occur, they are malignant degenerations of a xeroderma pigmentosum (a disease described by *Kaposi,* and developing in the first years of life; characterized by multiple pigmentary spots on the parts exposed to sunlight, and atrophy of the skin. Epitheliomatous degeneration is common). In older people (fortieth to seventieth years), cutaneous carcinoma of the face is frequent and develops from pre-existing warts, cutaneous horns, adenomata (*see* **Fig. 38**), dermoid or sebaceous cysts (for a similar condition originating in a wen, *see* **Fig. 18**), or on a ground prepared by chronic irritations of the skin (erysipelas, eczema, seborrhea, excessive exposure to rough weather, sailor's skin).

In old country people the flat carcinoma, of the types shown in **Figs. 1** and **2,** is of common observation, being, as it is, favored in its development by early wrinkling of the skin, uncleanliness and senile seborrhea, causing an accumulation of dirty scales on the skin. When these epidermic scales are scratched, superficial, easily bleeding sores are formed, which, however, heal quickly as long as they are not cancerous.

Fig. 1 shows a so-called *"Rodent ulcer,"* that is, a flat, very superficial, cutaneous cancer, in a typical situation on the face; still clear of the subjacent tissues. Rodent ulcer, which, of course, may be observed not only on the face, but in any point of the skin, presents itself at first as a hard, flat, reddish nodule which, when scratched or broken, forms a shallow ulcer with little tendency to heal. Of slow growth, and never attaining a conspicuous size before several, and often many, years, it generally remains a long time unnoticed by the patient, especially as it causes no inconvenience or pain. When it presents itself as a growing superficial ulceration, this usually has a circular shape with hard, slightly raised edges of overlapping

thinned epidermis; while the floor of the ulcer is, for the most part, soft at first, and the whole growth is movable over the deeper structures.

It is characteristic of these cutaneous carcinomata that plugs the size of a pin's head can be squeezed from the yellow surface of the ulcer; microscopic examination shows these to consist of broken-down, fatty, cancer cells. The ulcer is often covered by a scab, so that the diagnosis is only possible after its removal. As the tumor extends there appear radiating contractions of the surrounding skin and consequent deformity (of the eyelids, for example). The original circular shape is then often lost, and the outline becomes irregular (**Fig. 2**).

At first superficial, the tumor may after some years extend to the deeper parts and cause extensive destruction; for instance, of the bones of the face (**Fig. 4**). This deep extension is especially seen in parts where the subcutaneous fatty tissue is not developed (the temples, bridge of the nose and zygomatic arch, etc., **Figs. 2** and **4**); and it is also evidenced from the beginning by the decrease in mobility of the tumor over the subjacent structures. However, the tendency to deep extension is very little marked: but the extension in surface is practically unlimited, old ulcers sometimes attaining a huge size.

On account of the spontaneous cicatrization, which may take place at different parts of the ulcer or over its whole surface, although it is not permanent, these growths were formerly wrongly placed in the group of benign tumors under the name of *cancroid*. Certainly metastases are by no means as frequent as in other cancers; the general condition of the patient for years remains unimpaired while the ulcer is but slowly expanding; recurrence seldom takes place after complete removal; so that, as a rule, ulcus rodens is not possessed of the high malignancy of the cancers of mucous membranes and glandular organs. It is, however, an unquestionably malignant lesion. Its microscopic structure is that of baso-cellular epithelial cancer.

Diagnosis

The appearance of rodent ulcer is typical and, oftentimes, the diagnosis is not difficult, provided care is taken thoroughly to remove the overlying scab to bare the real surface with its slightly raised and everted epidermic edge. However, particularly in the region of the chin, an *extragenital syphilitic chancre,* especially if modified by local caustic applications, might show a certain resemblance, but the early adenitis and the demonstration of the spirochetæ pallidæ would clear up the doubt. A *tertiary gumma* or an *atypical*

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lupus might also be mistaken for a rodent ulcer. The notion of age is very important, ulcus rodens being a disease of elderly people. In all doubtful cases, microscopical examination of an excised fragment is the quickest and best way to settle the question.

Transient epidermization can generally be promptly obtained in small flat ulcers by antiseptic dressings. A permanent healing is, however, not to be obtained by this means, and under the scar, columns of epithelial cells keep on proliferating.

Treatment

Early excision, about half an inch beyond the ulcer in healthy tissues, and of sufficient depth, is the treatment preferred and almost exclusively advocated by many surgeons; and was in fact, successfully applied in the case shown in **Fig. 1.** The defect was repaired by a pedunculated flap taken from the left side of the forehead, where the loss of substance was covered with *Thiersch* grafts.

Rodent ulcers are frequently situated in the vicinity of structures important to consider for cosmetic results (eyelids, nose): the gap created by the surgeon is often surprisingly large, owing to the tendency rodent ulcers seem to have to draw tissues toward them, so that a plastic repair is by no means a simple task in many cases. This is why many dermatologists prefer treatment by scraping with the sharp spoon, followed by cauterization with acid nitrate of mercury, and subsequent applications of X-rays. Such a treatment gives excellent results (*Sherwell*) provided it be **very thorough.** If incomplete, it simply accelerates the progression of the ulcer. It can be carried out under local anesthesia. Good results have been reported from the use of radium (*Abbe*). X-ray treatment alone is also efficient, especially the single dose method (*Mackee*) in which the ulcerative type requires about $1\frac{1}{4}$ *Holzknecht* units, and the nodular form about $1\frac{1}{2}$ of the same units. Finally, caustic pastes, tabooed by surgeons, have given favorable results in the hands of dermatologists (*Robinson, Pusey*).

Fig. 2 shows an advanced **carcinoma of the skin of the forehead** with irregular borders. The growth has already extended to the bones. The upper eyelid and the ocular conjunctiva are also involved. This is a case of the rare form of cancer of the skin first described by *von Bergmann*, which in its early stages appears in the form of small multiple nodules and may therefore be mistaken and treated for tuberculosis cutis (lupus). The raised, irregular, hard edges of the

ulcer point to the correct diagnosis, which in doubtful cases should be cleared up by removal of a piece for examination. Previous treatment with X-rays had caused a rapid extension of the carcinoma, so that the patient came to the clinic in an inoperable condition. When carcinoma of the face extends through to the dura mater, the patient may die of meningitis; operation is contraindicated and the palliative treatment alone comes under consideration. This treatment consists in disinfection by antiseptic dressings with potassium permanganate and hydrogen peroxide. Later on, cauterization with the actual cautery, or cautious fulguration (sparking with the high frequency current) may make the condition less unbearable for a time.

Fig. 4 shows a cutaneous cancer with extensive deep growth, having destroyed the bony framework of the nose and the ethmoid cells. This form of cancer in its early stage consists of subcutaneous nodules covered by unaltered skin. The skin gives way when the nodules break down and a very extensive and deep cancerous ulcer results. This may be mistaken for a **gumma,** but the latter is not so ragged and has a yellow core. (Cf. **Fig. 120.**) The presence of epithelial plugs is also characteristic of this form of carcinoma. Microscopical examination, the *Wassermann* reaction and antisyphilitic salvarsan treatment will decide the diagnosis in doubtful cases. The papillomatous forms **(Fig. 4),** which often give rise to deep cutaneous cancer through their rapid growth and metastatic formations, must be regarded as extremely malignant tumors.

In all cases of extensive carcinoma of the face the patients may die from septic pneumonia when the destructive process reaches the buccal cavity.

Permanent results may sometimes be obtained after radical operations which often necessitate removal of diseased bones, but these of course are very disfiguring. Inoperable cases may be somewhat relieved by the palliative treatment already mentioned.

Fig. 3 shows a *carcinoma involving the whole lower lip.*

Cancers of the lip resemble cancers of the skin in form and structure, for they have the structure of squamous-celled epithelioma and tend to cornification. Theirs is intermediate between the comparatively low malignancy of cutaneous cancer and that—very high— of lingual carcinoma. They arise as cauliflower-shaped, polypoid tumors on the mucosa of the lip or as deep, ragged ulcers. Both these principal varieties are found in cancers of all mucous mem-

Tab. II.

Fig. 4. Carcinoma nasi.
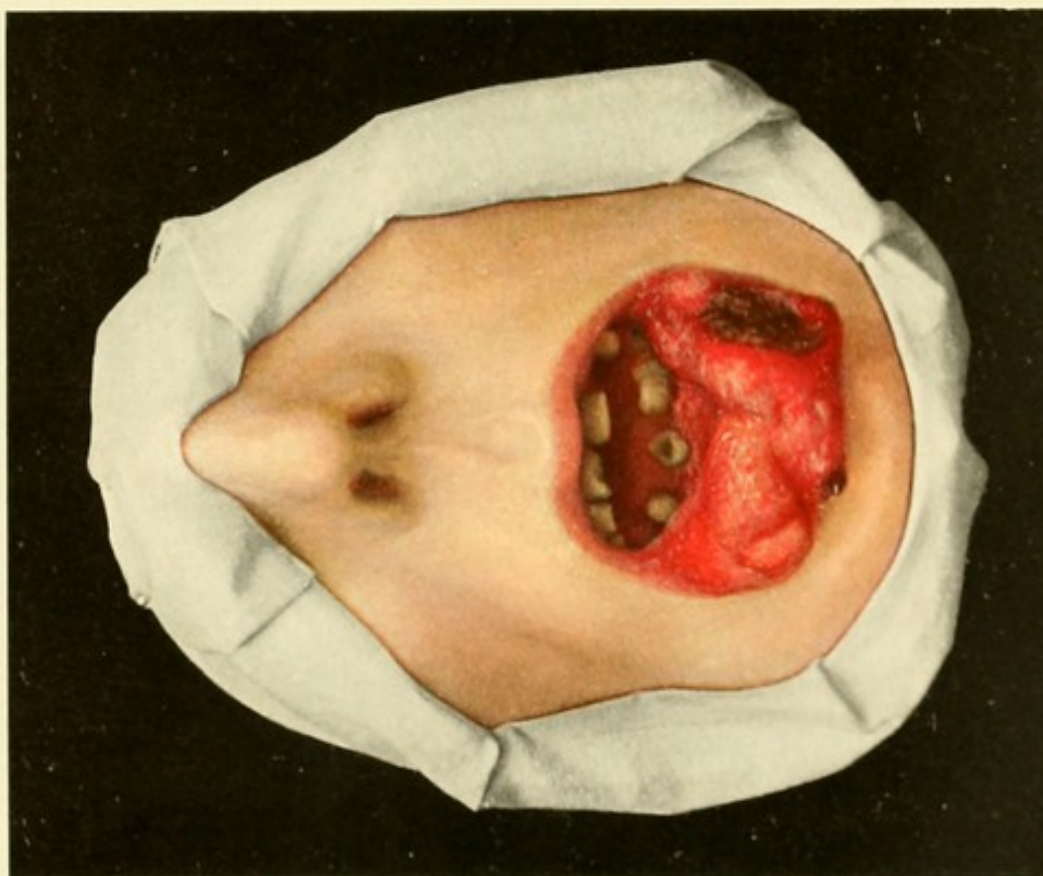
Fig. 3. Carcinoma labii inferioris.
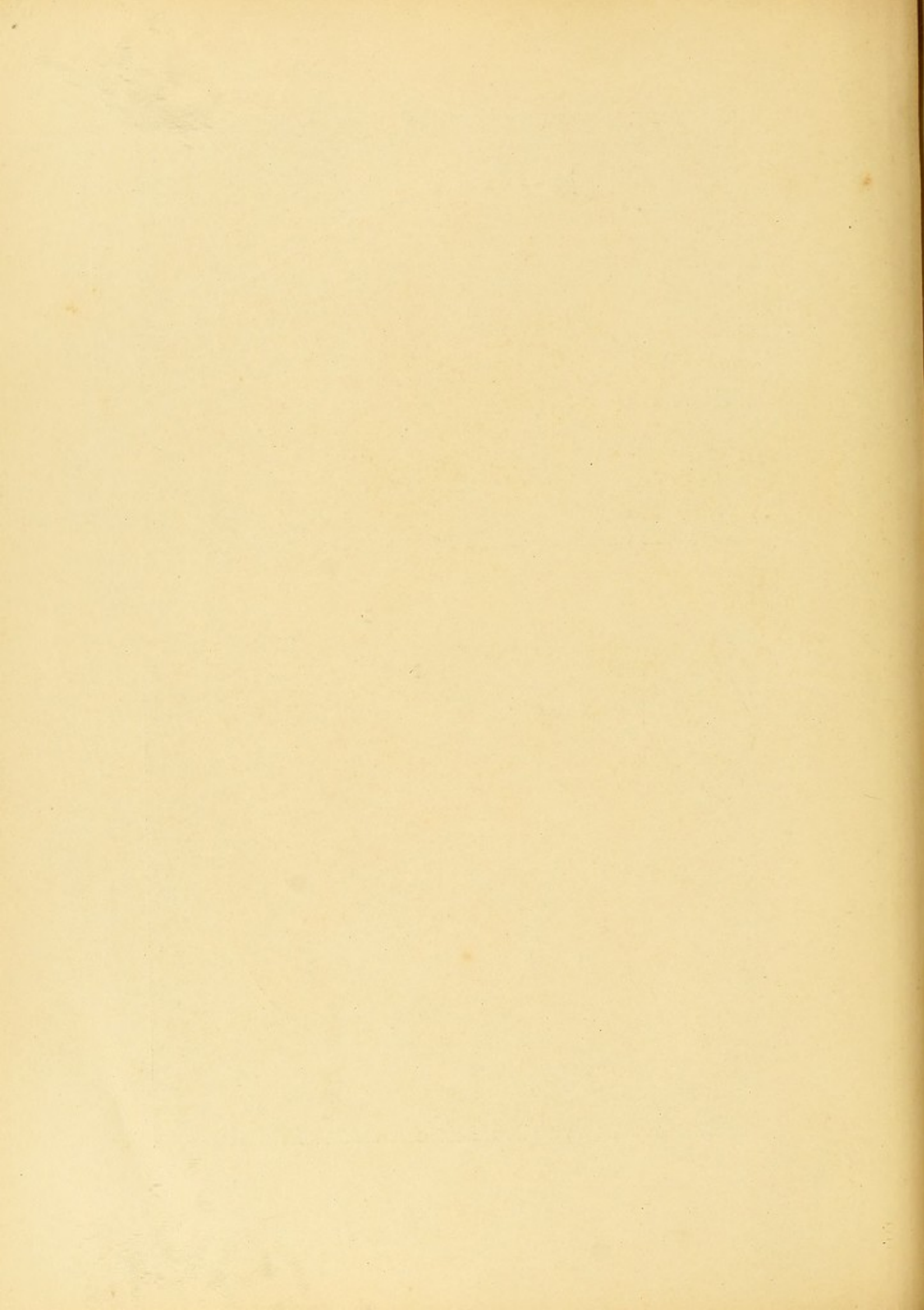
branes covered with epithelium (cheeks, tongue, penis, etc.). This is well shown in **Fig. 3,** where deep ulcerations alternate with papillomatous growths. In some parts there are scabs on the surface of the ulcers; in others isolated, yellow, epithelial plugs.

Carcinoma of the **upper** lip is very rare. A few cases have been published in which such a carcinoma developed after one of the lower lip, in a symmetrical position.

Carcinoma of the **lower** lip is almost exclusively a disease in the male sex, and seemingly more frequent in smokers, although the part played by tobacco, admitted by a majority of writers, is stoutly denied by others.

Labial leucoplakia, similar to that observed on the tongue (*Cf.* **Fig. 8**), and closely allied, as is the later, to syphilis (see page 8), may degenerate into cancer. Antecedent tuberculous disease also seems to be a cause favoring the development of cancer. (**Fig. 5** is an example of this mixed condition.)

Cancer of the lower lip often begins at the junction of the skin with the vermilion border of the lip, generally between the midline and the angle of the mouth, as a small, hard nodule at first covered by mucous membrane. The latter soon becomes broken and the nodule grows, infiltrating the surrounding tissues rapidly, while the mucosa breaks down more and more, and thus is formed an ulcer. The whole of the lower lip may be gradually destroyed (**Fig. 3**). Scabs and crusts form at several places on the ulcer, and when separated cause bleeding.

In its early stages the cancer is only an ulcer with hard, raised edges and a crateriform floor, but later papillomatous proliferations spring from this floor (**Fig. 3**). The more the carcinoma extends, the more it implicates the underlying bones and the mucosa of the cheeks and floor of the mouth, so that all these structures may be completely destroyed. The exudation of growing cancer of the lip gives rise to marked cachexia, gastritis and enteritis, and the secretion may reach the lungs and cause death from septic pneumonia. In such inoperable forms the submaxillary and submental regions are usually filled with hard, fixed glands.

Differential Diagnosis

Although these advanced forms, which are often neglected, especially in country people, are unmistakable, there may be difficulty in diagnosing a cancerous ulcer in the early stage, when it is most important. The irregular, ragged surface of the carcinoma is in

marked contrast to the smooth, raised surface of a **primary syphilitic sclerosis,** far from uncommon on the lip. The comedo-like epithelial plugs which are pathognomonic of squamous-celled epithelioma can be extruded from it by pressure. The submental and submaxillary glands are involved early in both cases, and as there are no character-istic clinical symptoms in either case sufficient to differentiate the adenitis, scraping of the surface of the ulcer and a *dark field illumin-ator search for spirochetæ,* or the excision of a piece of the tumor for miscroscopical examination, are imperative. This procedure is to be preferred to a test course of antisyphilitic treatment, because it wastes no time, a precious advantage when dealing with malignant growths.

An **ulcerated gumma** of the lip may resemble a carcinomatous ulcer. Here also, if anamnesis and a positive *Wassermann* reaction are not sufficient to clear any doubts, histological examination must be resorted to. The difficulty is still increased by the close relations between syphilis, leukoplakia and cancer and the existence of hybrid forms. In a general way, gummata are covered with a special, tenacious, yellowish deposit and do not exhibit the hardness of cancerous formations.

Primary tuberculosis of the lip, or the extension of a tuberculous ulcer of the buccal or lingual mucosa, is rare on the lip. The irregu-lar edges are not raised and remain soft. The surface of the ulcer has the anemic, reddish-gray color of all tuberculous ulcers; and at the periphery, there may be some small, non-ulcerated tubercles. The ulcer bleeds easily, is generally covered with a single large scab; no plugs can be expressed from it. Glandular enlargement is frequently absent: when it exists, it is soft and involves but few lymph nodes.

An ulcerated cavernoma (cavernous angioma) may look some-what like cancer, but the young age of the subject and the coexistence of other anomalies of the blood-vessels is conclusive.

The induration of fissures of the lips resulting from chronic eczema heals quickly under appropriate treatment.

Treatment

Much depends on an early diagnosis, because a small tumor may be easily removed by a **cuneiform excision,** while large growths neces-sitates difficult plastic operations; the results are much less certain, and the disfigurement is much more marked. This removal must always be **preceded** by a **thorough cleaning of the submental and submaxillary glands.** In extensive tumors, from half an inch

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Fig. 5. Carcinoma labii inferioris — Tuberculosis cutis.

to an inch of healthy tissue should be removed beyond the margin of the growth, and the neighboring parts suspected of disease, such as bones and buccal mucous membrane, should also be excised. In the case represented in **Fig. 3,** the extensive defect was repaired by double cheiloplasty and a cure was obtained.

Fig. 5 represents a *large cancerous ulcer,* originating from *tuberculosis* of the skin, involving half the *lower lip.* Such an association is not very rare; and most of the destructive forms of lupus described under the name of *lupus vorax* seem to depend on it. The hard, raised edges of the ulcer divested of mucous membrane are characteristic. The floor of the ulcer is irregular and ragged and beset with yellowish epithelial plugs. Cancerous ulcers developing on a previous tuberculosis of the skin have a great tendency to bleed. In contrast to hypertrophic lupus, which gives rise to soft, fungoid, slow-growing tumors, the hardness and rapid growth of lupus-carcinoma is characteristic. Excision of the carcinoma, removal of the glands, and repair of the defect by *Dieffenbach's* cheiloplasty led to a cure.

Fig. 5 also shows a characteristic picture of different forms of cutaneous tuberculosis (lupus) of the face. *Lupus* appears most frequently in this situation, usually begins on the nasal mucosa, and extends over the face in the shape of a butterfly. The sharp, irregular outline on the forehead, neck and behind the ears is characteristic. The disease begins with small reddish-brown, *apple-jelly like nodules* situated in the cutis and causing exfoliation of the epidermis (lupus exfoliativa); these become confluent and form flat, reddish-gray, easily bleeding ulcers (lupus exulcerans), which after healing leave radiating cicatrices, often after considerable destruction of tissue. (**Fig. 5,** ear). After a time papillomatous proliferations of soft and spongy consistency may arise, especially about the ear (lupus hypertrophicus). These three forms are often present in the same patient.

The characteristic lesion of lupus is the *ring of small apple-jelly tubercles* at the periphery, while the center has already healed. If those elements are found, the diagnosis is certain, unless we are dealing with some mixed lesion.

Treatment

The treatment of *small,* well-circumscribed lupic lesions may be **surgical excision** followed by simple suture or plastic repair. Scrap-

ing, scarifications, cauterization (thermocautery, hot air) and applications of strong caustics have been less used since phototherapy (*Finsen*) and radiotherapy have given brilliant results. These are now the real methods of treatment. Radiotherapy is more within the reach of all practitioners than is phototherapy. Freezing with a stick of carbonic acid snow has sometimes been employed with good success (*Pusey*) on small nodules or patches.

The general dietetic and hygienic antituberculous treatment is a great help; and injections of tuberculin (TR) in small doses, to avoid general reaction, may prove useful in refractory cases.

Figs. 6 to **9,** inclusive, represent **epithelial newgrowths of the tongue.** To be compared with **Figs. 36, 117, 118, 119).**

Fig. 6 shows a **flat papilloma** which was removed with the sharp spoon. (About the nature of papilloma, see page 61.)

Fig. 7 shows on the left half of the tongue an **extensive papilloma,** and on the right a superficially ulcerated **carcinoma.**

Fig. 8 shows a **deep carcinoma** developing under a patch of **leucoplakia:** it is not yet ulcerated and is characterized by its hardness and irregular outline. This central location is exceptional.

Fig. 9 represents the **most common form of cancer of the tongue:** a carcinomatous **ulcer** of the side of the tongue with extensive destruction, leucoplakia and glandular metastases.

Cancer of the tongue is seen almost exclusively in men after the fortieth year. Alcohol and particularly tobacco favor its development. The lesion shown in **Figs. 8** and **9** and called **leucoplakia** plays an interesting part in the production of lingual cancer.

Leucoplakia is a hyperkeratosis of mucous membranes, particularly frequent on the tongue and on the buccal mucosa, but which has also been observed in other mucosæ, *e.g.* bladder and glans penis (see page 20). It forms hard, white, opaline patches consisting of horny epithelium and raised above the surface of the adjacent mucous membrane. The nature of leucoplakia is uncertain. Formerly, it was considered as a special disease in itself (*buccal psoriasis*): more recently, chiefly under the influence of *Fournier*

Fig. 6. Papilloma linguae.
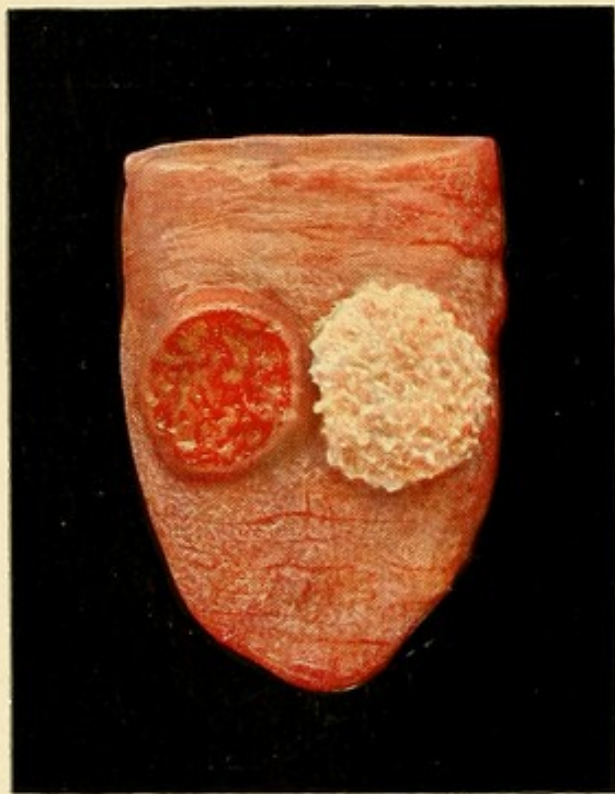
Fig. 7. Carcinoma et Papilloma linguae.

Fig. 8. Carcinoma linguae incipiens.
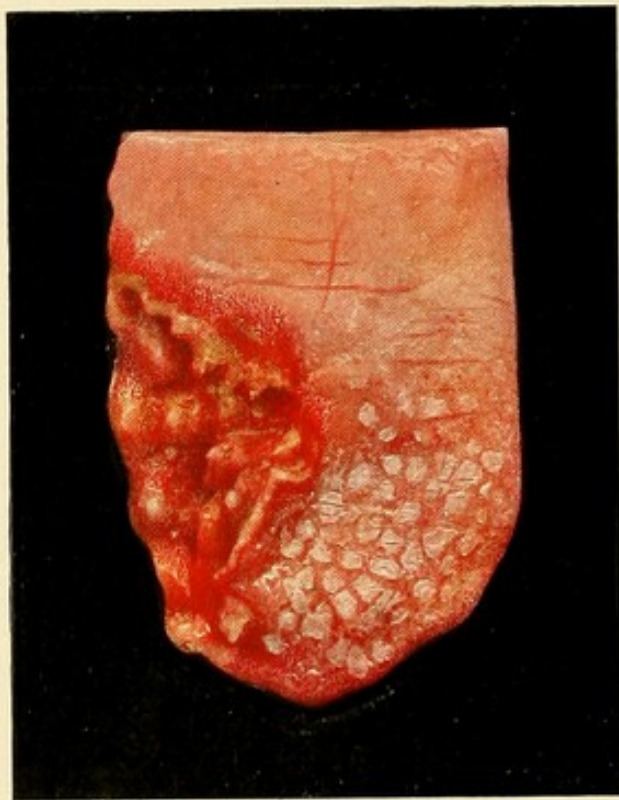
Fig. 9. Carcinoma linguae exulceratum. — Leukoplakia.

and his pupils, it has been considered as a parasyphilitic keratosis—that is, an affection of syphilitic *origin*, but not of syphilitic *nature*—developing under the combined influence of syphilis and tobacco. This view is now held by the majority of syphilologers. A third opinion contends that there are two kinds of leucoplastic lesions: first, *genuine leucoplakia*, hyperkeratosis of unknown origin, but having nothing to do with syphilis; second, *pseudo-leucoplakia*, much more frequent, which is the so-called parasyphilitic "leucoplaka" of syphilologers. This third opinion seems the most rational, but, practically, the point is not of much importance, because, be it pseudo-leucoplakia or genuine leucoplakia, all these patches of hyperkeratosis have an unfortunate tendency to undergo malignant epithelial degeneration. In fact, cancer is the natural outcome of leucoplakia of long standing; and lingual leucoplakia is found in the antecedents of one-half of the cases of lingual carcinoma.

The surface of the patch, at first smooth and painless, after a time becomes fissured, especially after excessive smoking, and the lesion becomes deeper and sometimes exceedingly painful.

The treatment of leucoplakia consists in the avoidance of tobacco, alcohol and spicy food, and in the removal of patches, either with the knife or with the thermo or galvano-cautery. This is possible only when the patches are not too extensive. Simple cauterizations which do not destroy the leucoplakia patch are worse than useless, because they merely irritate and tend to promote malignant degeneration, without being able to cure.

Besides leucoplakia, jagged carious molar teeth also act as exciting causes of cancer of the tongue, which explains the almost exclusive occurrence of cancer in the posterior part of the side of the tongue. Lingual carcinoma appears in two forms, according as it arises from the *superficial mucous membrane* or from the *glandular epithelium*.

The *first form* resembles a flat cutaneous carcinoma and soon gives rise to a small ulcer with hard, raised edges (**Fig. 7,** right half), the fissured surface of which has a yellowish or dirty-brown appearance. Although the carcinoma is only superficial, the submaxillary glands are soon affected, owing to the abundant lymphatics of the tongue.

Deep carcinoma begins as hard nodules over which the mucous membrane remains intact for a long time. After the breaking down of the nodules and destruction of the mucous membrane, an extensive crateriform ulcer is formed with hard, irregular edges and deep,

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fissures in the center. This often reaches as far back as the epiglottis. Numerous epithelial plugs can be expressed from the floor of the ulcer, and often from the papillomatous proliferations. The patients suffer great pain from the irritation of free nerve endings in the floor of the ichorous ulcer, and, in untreated cases, usually succumb within a year from glandular metastases extending along the carotid to the supra-clavicular region (as was the case in the patient represented in **Fig. 9**). Early diagnosis is, therefore, of the greatest possible importance.

Diagnosis

The superficial carcinoma (**Fig. 7**) is recognized by the characteristic features of flat cutaneous carcinoma and differs from **syphilitic chancre** (*see* **Fig. 120**) by its sharp, hard edges, the irregular floor of the ulcer with epithelial plugs, the absence of spirochetæ pallidæ in the scrapings examined under the dark-ground illuminator. As long as a flat carcinoma of the tongue is covered with mucous membrane it may in its earliest stages be mistaken for **papilloma** (**Fig. 6**), especially in the rare cases where it lies more in the center of the dorsal surface of the tongue. Papillomata, however, generally appear as multiple, soft elevations the size of a pin's head, so that the surface of the tongue may appear dotted with small points, or may assume a lobulated form; or there may be fungiform sessile tumors, like stalactites, which often form high projections and have a warty appearance (**Fig. 7**). A flat carcinoma and a papilloma of this kind may occur independently without microscopic transition into each other. Small papillomata cause the patient hardly any inconvenience and can be removed with the sharp spoon or *Paquelin's* cautery. Larger papillomata should be excised (**Fig. 7,** left half).

A small carcinomatous ulcer of the edge of the tongue is liable to be mistaken for **ulcerations caused by the irritation of broken teeth** (dental ulcers), especially when it is situated opposite a sharp tooth; however, the cancerous ulcer continues to grow after removal of the offending tooth.

Larger ulcerations which result from the breaking down of deep carcinoma may be confounded with a **gumma** on superficial examination. The latter, however, is almost always situated in the center of the tongue or in its anterior part, and has the characteristic dirty-yellow, gummatous core, which can be removed without bleeding (**Fig. 119**), in distinction to the easily bleeding reddish-brown proliferations of carcinoma. Moreover, the pain radiating to the ear

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which is constantly present in large carcinomata is absent in gumma; also the glandular metastases and the leucoplakia.

In cases of small, non-ulcerated tumors, the diagnosis is more difficult and may again hesitate between a cancer and a **gumma.** Here the *Wassermann* reaction is of little help when negative; it simply increases the suspicion of cancer without proving anything; a test course of antisyphilitic treatment wastes too much precious time so that *if the diagnosis is really impossible by clinical means, excision* of a piece and **immediate** *histological examination* by a com-competent pathologist is justified, but only if radical removal is to follow without delay, should malignancy be proved (as unfortunately happens practically in all "suspicious" cases); else such excision has all the inconveniences of partial ablation in cancer.

The diagnosis is also difficult when, as in **Fig. 8,** a hard, carcinomatous nodule develops under a patch of leucoplakia. The irregular, deep, hard infiltration and the rapid increase point to a commencing new growth, which should always be removed before it breaks through, especially as when there is leucoplakia over the nodule, malignancy must always be suspected.

Semi-chronic abscesses of the tongue, which result from injury by foreign bodies (steel pens, etc.), and form hard lumps in the substance of the tongue, are characterized by the early painfulness on pressure. **Actinomycosis** causes a more diffuse, woody infiltration of the whole tongue and very soon interferes with its motion.

Treatment

The treatment of lingual carcinoma is **always total removal of the tongue and its lymph glands.** Partial excisions are always insufficient, even in the rare cases of seemingly well localized lesions of the anterior half, and excision of the tongue without the lymph glands should never be performed.

The best surgical technique for the removal of the tongue is that in which, as a preliminary step, **all the lymph glands of the sub-maxillary and submental regions are cleaned out and both lingual arteries are ligated.** This step is wholly aseptic. The extirpation of the tongue is effected a few days later. It is almost bloodless, owing to the previous ligation of the lingual arteries, and, the tongue being already markedly shrunken, its removal is much easier. There is less chance of being compelled to divide the lower jaw, either in the midline or laterally; however, such a division remains necessary if the carcinoma extends far back.

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Even after extirpation of extensive portions of the tongue the patients, after a few months, can make themselves well understood. Permanent cures are, however, unfortunately rare, even after radical operations performed before any hardened lymph nodes can be felt in the submental region, so that some surgeons content themselves with the local palliative treatment of carcinoma by applications intended to relieve pain. X-ray, radium, and high frequency treatment does not yield results in lingual carcinoma. Early and extensive removal is the only hope, and it is none too promising.

The treatment of cancer of the buccal cavity, which often develops from leucoplakia, with the same symptoms and objective aspect as lingual carcinoma, is carried out on the same principles. The closure of extensive defects of the cheek is no easy task in most cases. A mucous lining must be provided for the buccal side. Cicatricial retraction often hinders almost completely motion of the lower jaw; section of the ramus of this bone at the time of operation prevents this cicatricial ankylosis (*Bodine*).

Figs. 10 to **16,** inclusive, show a number of types of **carcinoma of the breast.**

(Other lesions of the breast, see **Figs, 29. 30, 37, 85** and **86.**)

Of the carcinomata of glandular organs those of the female mammary gland are among the most common (they take the third place). A division into soft, many-celled, rapidly growing tumors of which the medullary cancers represent the most malignant, and slow-growing scirrhous forms with few cells, is of clinical importance.

The exciting causes include inflammatory irritation, puerperal interstitial mastitis, eczema of the nipple, antecedent benign tumors (fibro-adenoma, cysts), injuries, mechanical irritation, frequent parturition with prolonged suckling of infants. Cancer of the breast is attributed by the laity to injuries (blows), but these are often too recent to be accepted as an etiological factor, considering the slow growth of the carcinoma.

The activity of the gland in lactation is a predisposing cause of cancer. Only 10 per cent. of the cases of breast carcinoma are seen in sterile women. The weakening action of pregnancy and lactation is well shown in the type known as **carcinoma mastitoides** (*Schurman*) or **mastitis carcinomatosa** (*Volkmann*) (*see* **Fig. 16**), which is the most malignant form of all.

Women are most often affected at the menopause (fortieth to

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Tab. V.
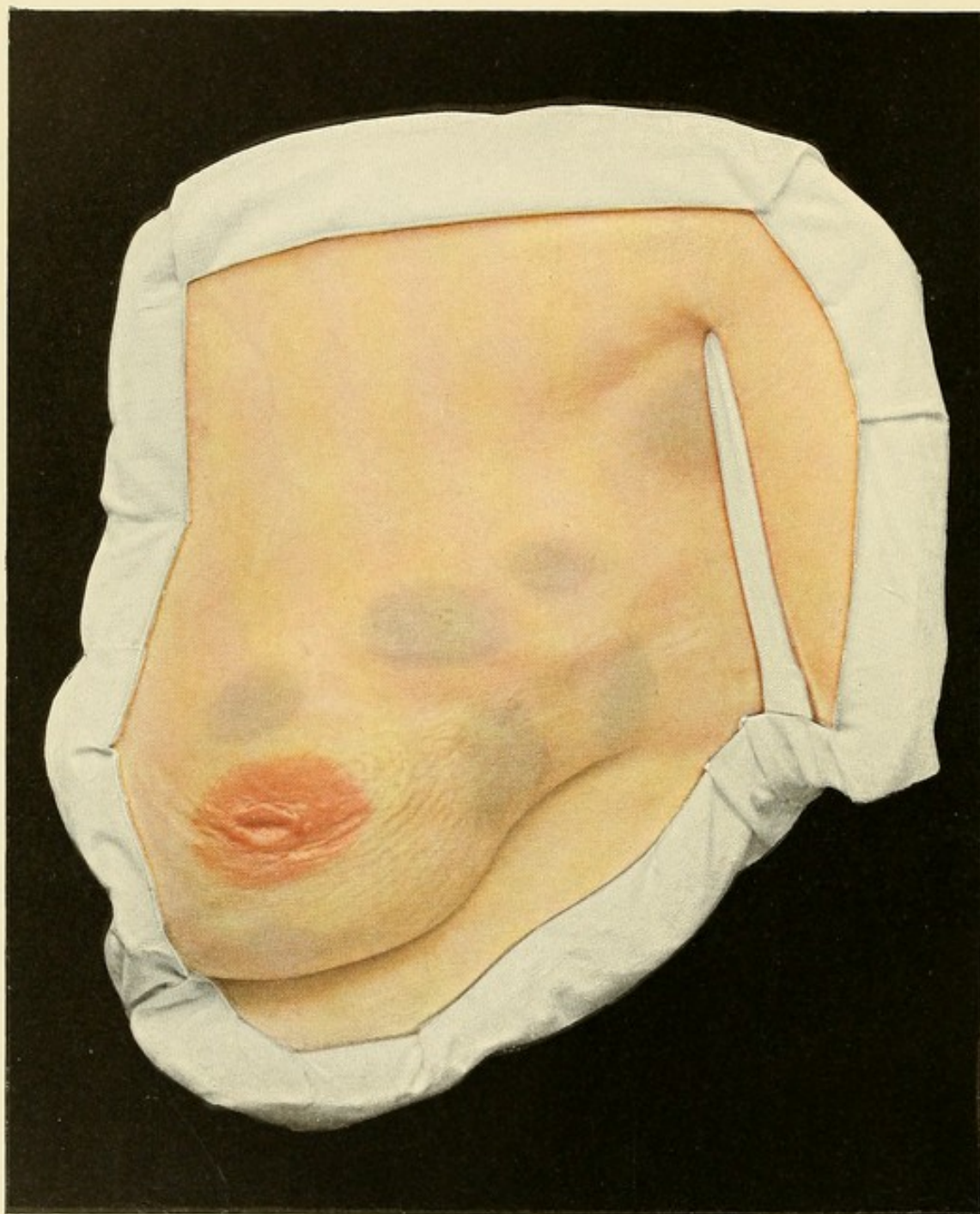
Fig. 10. Carcinoma mammae — Lymphomata carcinomatosa.

fifty-fifth years), and come to the surgeon with nodules in the breast which have been hitherto painless and are only accidentally observed. These nodules very soon form a malignant growth of hard consistency and irregular surface.

The most important sign of a malignant new growth is the **absence of any demarcation or encapsulation.** The tumor cannot, like all benign tumors, be separated from the mammary tissue and moved freely, but is fixed immovably in the glandular tissue, with ill-defined boundaries, and is anchored in the meshes of the mammary tissue by numerous offshoots. The nodules, which at first appear harmless, thus soon show their malignity.

Progressively the tumor sends its destructive extensions in all directions into the neighboring tissues, without limit or restraint, and reaching the surface adheres to the skin and causes **dimpling of the skin, retraction and fixation of the nipple.** Finally, it gives rise to a hard inflammatory infiltration of the whole of the overlying skin. At the same time the tumor extends deeply and soon infiltrates the lymphatics beneath the pectoralis major muscle and also the regional lymphatic vessels and glands of the axilla (**Fig. 10**), which are usually affected about a year after the formation of the nodules in the breast, and take the form of hard, solid, painless lumps, which are often difficult to feel in corpulent women. Extensive glandular involvement gives rise to radiating pain and edema of the arm (supra-clavicular glands). Although the cancer usually arises as a single nodule, there are cases in which several nodules develop simultaneously (**Fig. 10**) and extend through the whole breast to the axilla (**Fig. 10**). The prognosis is unfavorable in these cases, and in disease of both breasts (**Fig. 15**).

Cancer is very frequently situated in the upper and outer quadrant of the breast, especially on the left side. The tumors situated in the outer half of the mamma towards the axilla, wrongly called paramammary carcinomata, are really glandular cancers, for they originate in the offshoots of the mamma, which extend toward the clavicle, sternum, axilla and twelfth rib in the form of long, thin cords.

Cancer of the breast, like all cancers rich in cells (acinous, tubular), grows rapidly, especially during pregnancy, and causes destruction of the skin. A cancerous ulcer results, characterized like cutaneous carcinoma by its hard, raised, fixed borders, crateriform base and sanious discharge. A hard infiltration develops around the tumor, which is usually firmly adherent to the thorax. Small nodular thickenings of the adjacent unbroken skin sometimes constitute the first

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sign of commencing general cutaneous dissemination (**Fig. 11**). In this way the whole mamma may be transformed into a large ulcer (**Fig. 15**).

In other cases the tumor is gradually developed and involves the whole breast without breaking through externally. The skin, however, may be infiltrated and the redness may be mistaken for inflammatory infiltration (**Figs. 14** and **16**). These leathery infiltrating forms of breast cancer finally develop the whole mammary region like a cuirass (**Fig. 15**).

In the infiltrated skin there often appear small, pin-point, disseminations of the carcinoma (**Fig. 15,** right side), which by confluence give rise to a nodular infiltration of the whole thorax (**Fig. 14**).

In scirrhous cancers, which are poor in cells, the mammary gland is often diminished in size by shrinking and the skin becomes puckered over the tumor by cicatricial contraction (**Fig. 10**).

Differential diagnosis

An advanced ulcerated cancer of the breast, or one with hard and raised infiltration, is easy to recognize, but those are not the interesting cases to diagnose accurately, because they have already reached a stage where chances of a permanent cure after operation are rather slight.

Small, young tumors, on the other hand, are often difficult to diagnose properly. ***Benign tumors*** (fibro-adenoma, cysts and mixed tumors), ***chronic interstitial mastitis,*** abscesses in which there frequently is deceptive induration, ***tuberculosis, galactocele*** are as many stumbling blocks.

The age of the patient has nothing characteristic; particularly in tumors of the breast, age is *not* a factor that allows to rule out malignancy.

The continuous growth of the nodules, the appearance of glandular enlargement, still more the cachexia are things for which we should not have to wait in order to be able to make a diagnosis.

As regards "benign" tumors, the only safe rule is always to view them with suspicion. Benign tumors in the breast are rare as compared with malignant; the proportion being less than 1 to 10. Many tumors, long classified among the benign, have now passed to the malignant group: *e.g.,* cystic disease of the breast, now called papillary cystadenoma, and unquestionably malignant in many instances. Finally, a really benign tumor is liable to undergo malignant degeneration after years of benign evolution. As a rule, every breast tumor
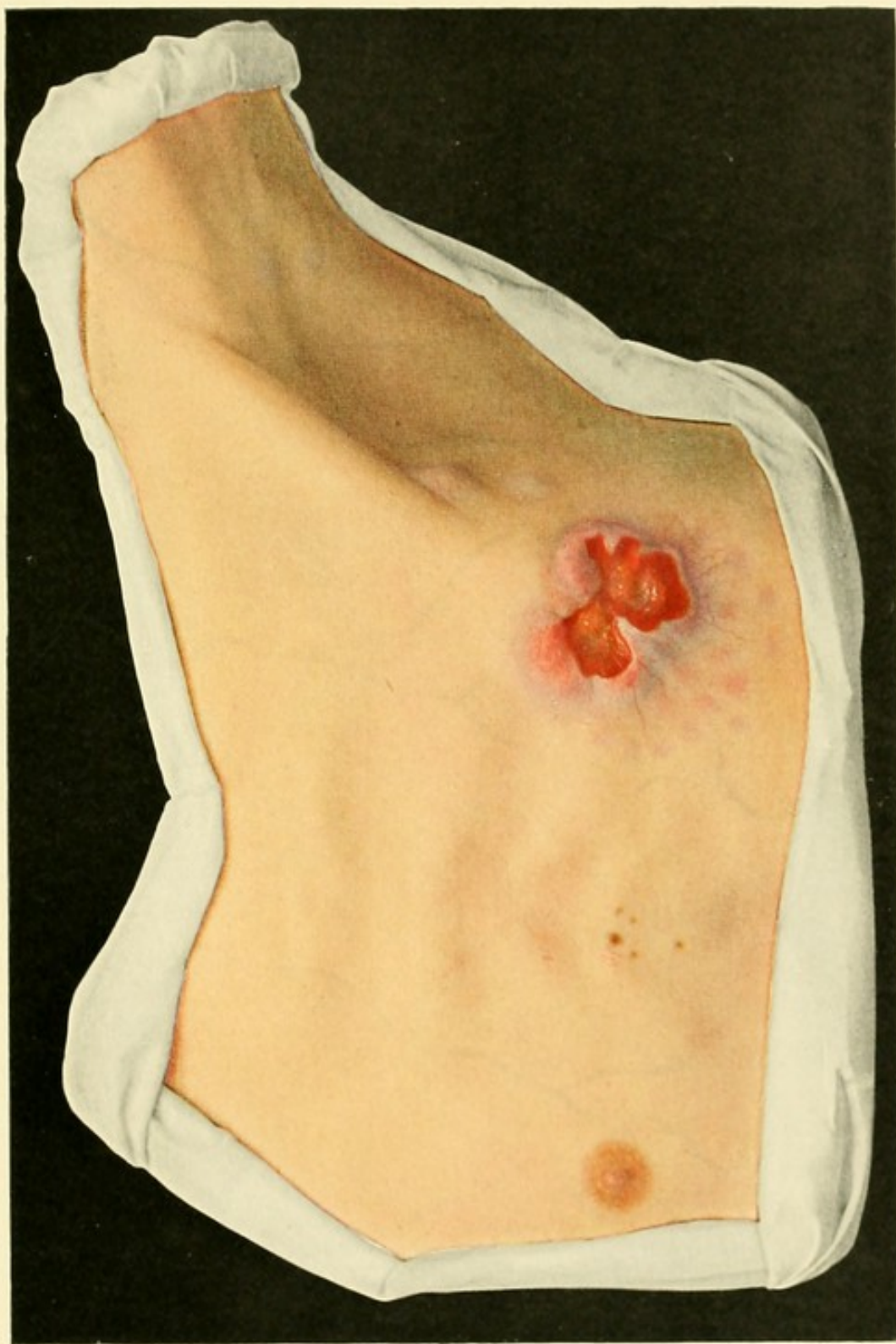
Fig. 11. Carcinoma mammae exulceratum.

about which the surgeon has even a vague suspicion of malignancy turns out to be malignant. Hence, **we must deem malignant every breast tumor the character of which does not establish beyond doubt its non-malignancy,** and act accordingly.

The great anatomical feature of non-malignant tumors is *encapsulation.* A perfectly well limited growth, movable in the gland tissue, non-adherent to any structure, is likely to be benign. Any tumor that is felt to send offshoots in glandular tissue **is cancer.** The diagnosis of chronic interstitial mastitis is particularly difficult; perhaps only because there exist between this condition and cancer intimate relations not as yet elucidated. *Microscopical examination* of an excised piece must be performed in all doubtful cases; and if even the microscope (as will happen sometimes) is unable to give a definite answer, the breast must be removed. Rather remove a chronically inflamed breast than give cancer too long a chance.

Sarcoma occurs at an earlier age and has a fairly typical appearance (**Figs. 29** and **30**); but a mistake in diagnosis is not very important, since both conditions, sarcoma and cancer, call for the same treatment.

The same may be said of infiltrating forms of **tuberculosis of the breast,** which are not exceedingly rare.

Treatment

The treatment of breast carcinoma is **radical excision of the whole breast and its processes** as early as possible with removal of the pectoralis major and minor muscles, and **complete cleaning out of the axillary glands** (*Halsted's* or *Willy Meyer's* technique). As in all operations for cancer, squeezing of the breast during removal must be carefully avoided, so as not to spread cancerous seed over the operative wound. In fact, recurrences are much more common in the scar than anywhere else. If small, they can be excised again.

Operation is contraindicated in all cases with extensive dissemination in the skin, diffuse infiltrating cancer, "*cancer en cuirasse*" (**Figs. 15** and **16**), also when the supraclavicular glands are involved, in slow growing scirrhus of very old people, and in cases where organic metastases are present.

X-ray treatment is good in the post-operative period, but cannot cure cancer without operation. A carcinomatous nodule may disintegrate and disappear under its influence, and surface epidermization occur, but the cancerous process continues in the deeper tissues. The

same is true of rádium and fulguration. Nevertheless, X-rays, fulguration, scrapings, cauterizations are useful in the palliative treatment of inoperable cancer.

No cancer serum has as yet gone beyond the experimental stage or given durable results.

Fig. 10 shows an **acinous carcinoma** forming several nodules in the breast, already infiltrating the skin. The axillary glands are felt as hard, fixed, indolent lumps, and a chain of nodules can be easily traced from the mammary gland to the axilla. The nipple is retracted and fixed, and the whole breast is diminished in size. Operation was performed in the usual way. The patient was already emaciated.

Fig. 11. A **single cancerous nodule in a male breast.** The skin has broken down and shows a cancerous ulcer with hard, raised, jagged edges, which has destroyed the nipple. The floor of the ulcer is irregular and the whole tumor is fixed to the pectoral muscle. At the edge of the ulcer the skin is radially contracted and shows isolated cancerous nodules. The axillary glands are hard, visible and hardly movable. In spite of the small size of the tumor, there was already cachexia. After removal of the mamma with the pectoralis major and the axillary glands the wound, which could not be completely closed by suture, as is frequently the case in women, but almost the rule in men, was repaired by *Thiersch* (*see* **Fig. 55**) grafts.

Cancer of the male breast (about 1 per cent. of all mammary carcinomata according to *Schuchardt*) generally arises as a small, hard nodule (scirrhus) in the neighborhood of the nipple and gives rise to a typical cancerous ulcer. The tumor occurs between the fortieth and seventieth years.

Fig. 12 shows a very rare case of **carcinoma arising from the nipple (squamous-celled .epithelioma).** This is more common in men than in women. It begins as a hard infiltration of the nipple, in the same way as does incipient carcinoma of the navel. The nipple is much retracted and the whole areola is transformed into a rigid wall. A cancerous ulcer soon develops which destroys the nipple and areola. At first there is no connection between this cutaneous cancer and the mammary gland.

The treatment consists in early extirpation of the mammilla with

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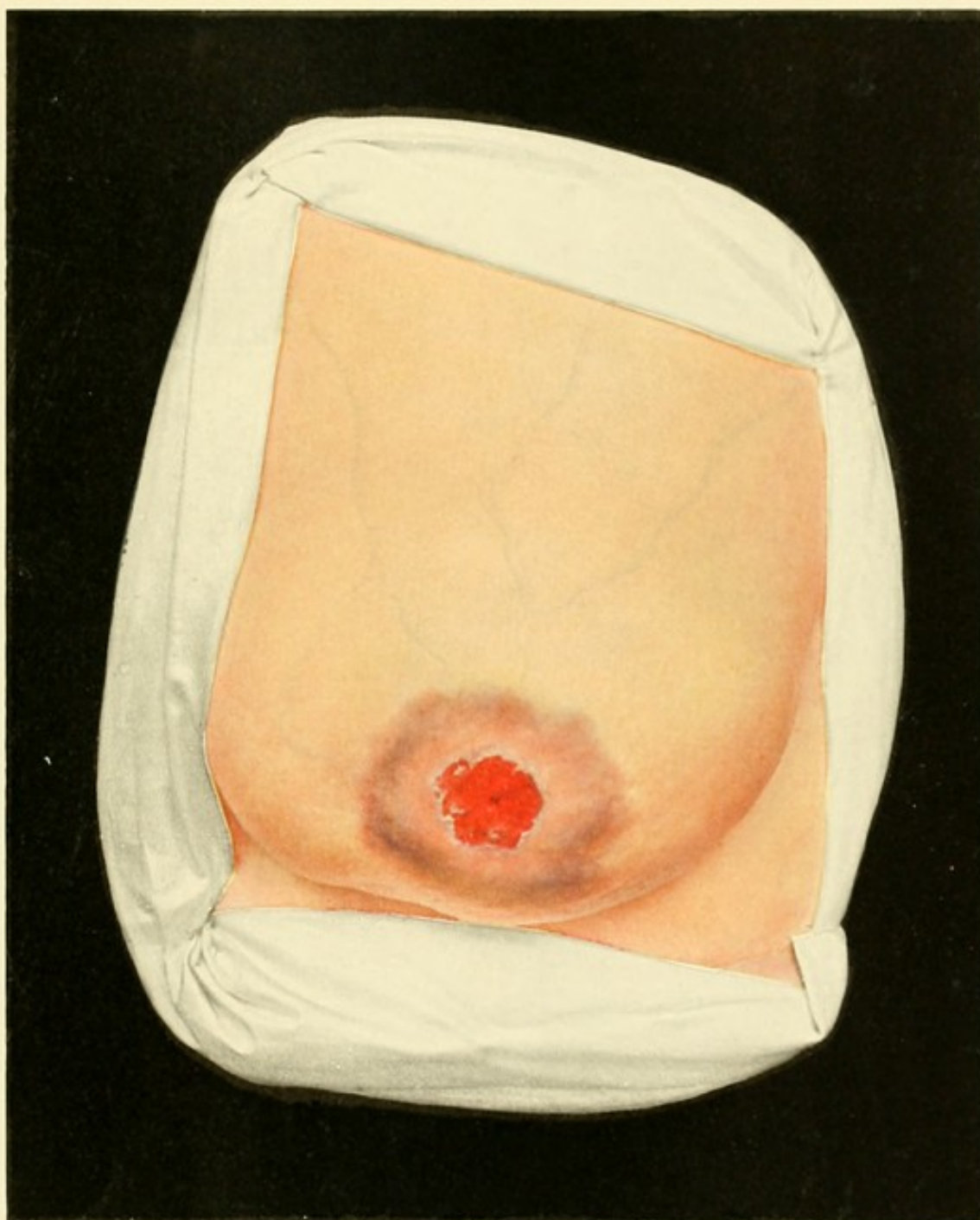
Fig. 12. Carcinoma mammillae.
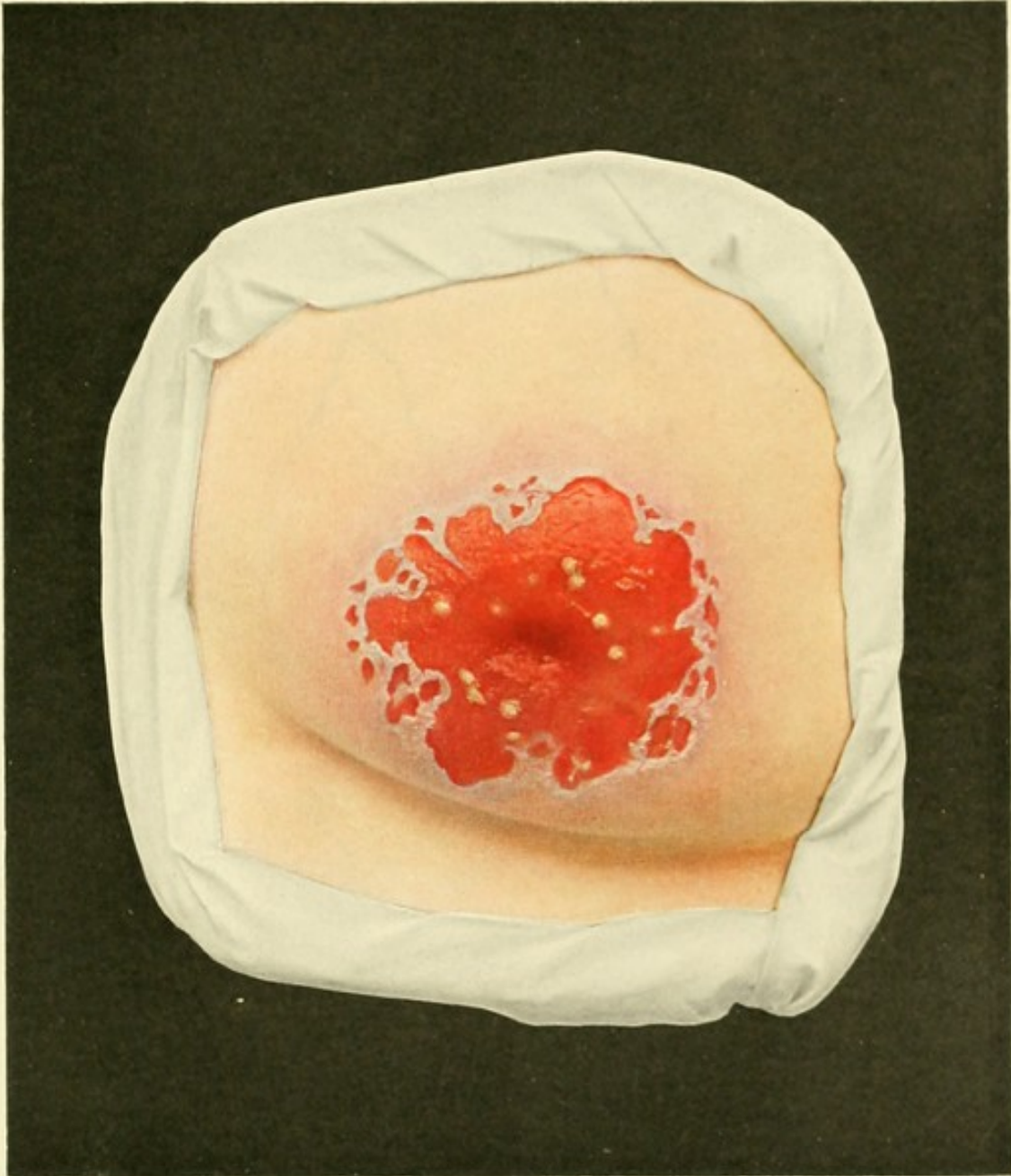
Fig. 13. Carcinoma mammae — Paget Disease — Eczema chronicum mammillae.
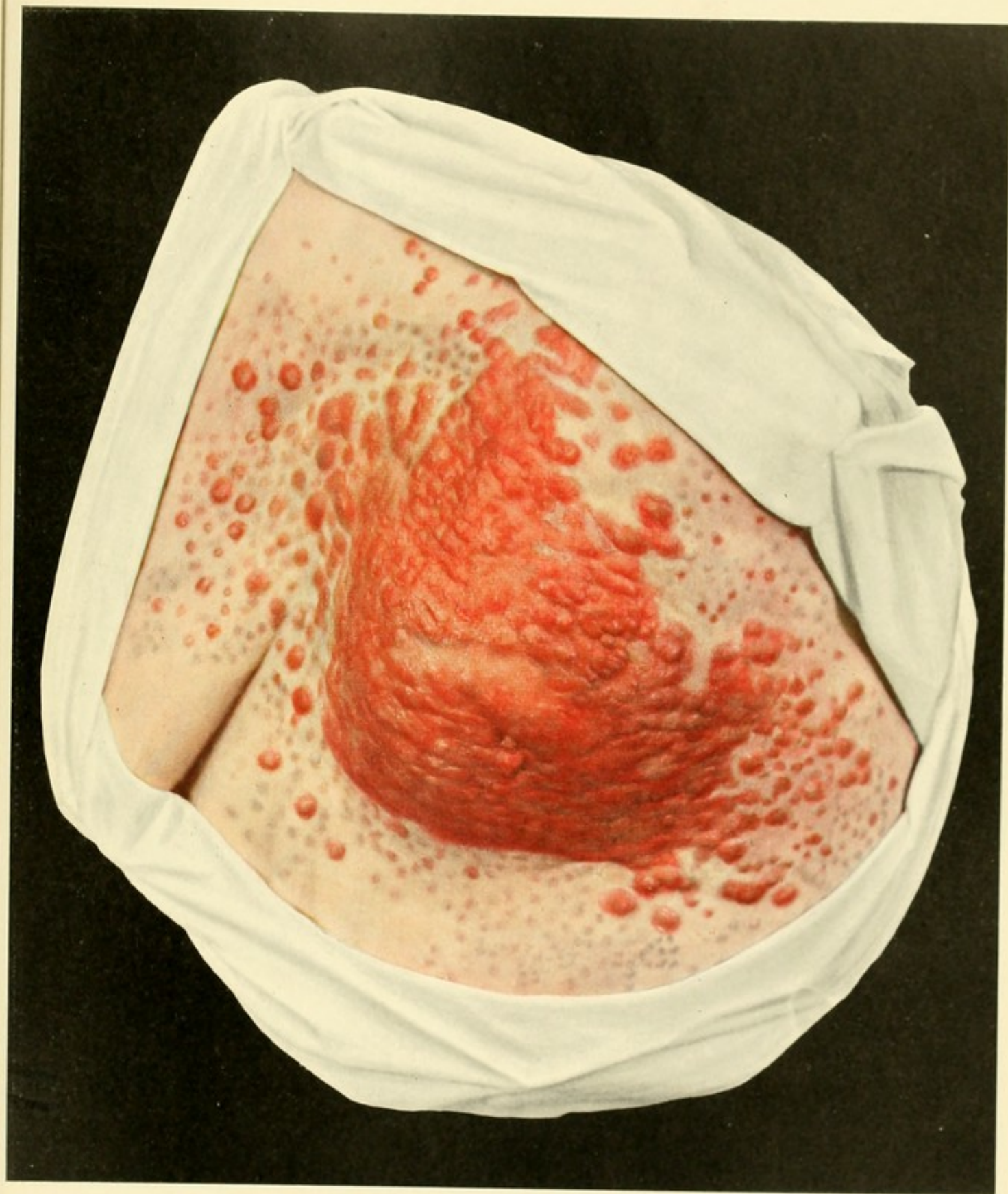
Fig. 14. Carcinoma mammae — Disseminationes.

Tab. X.
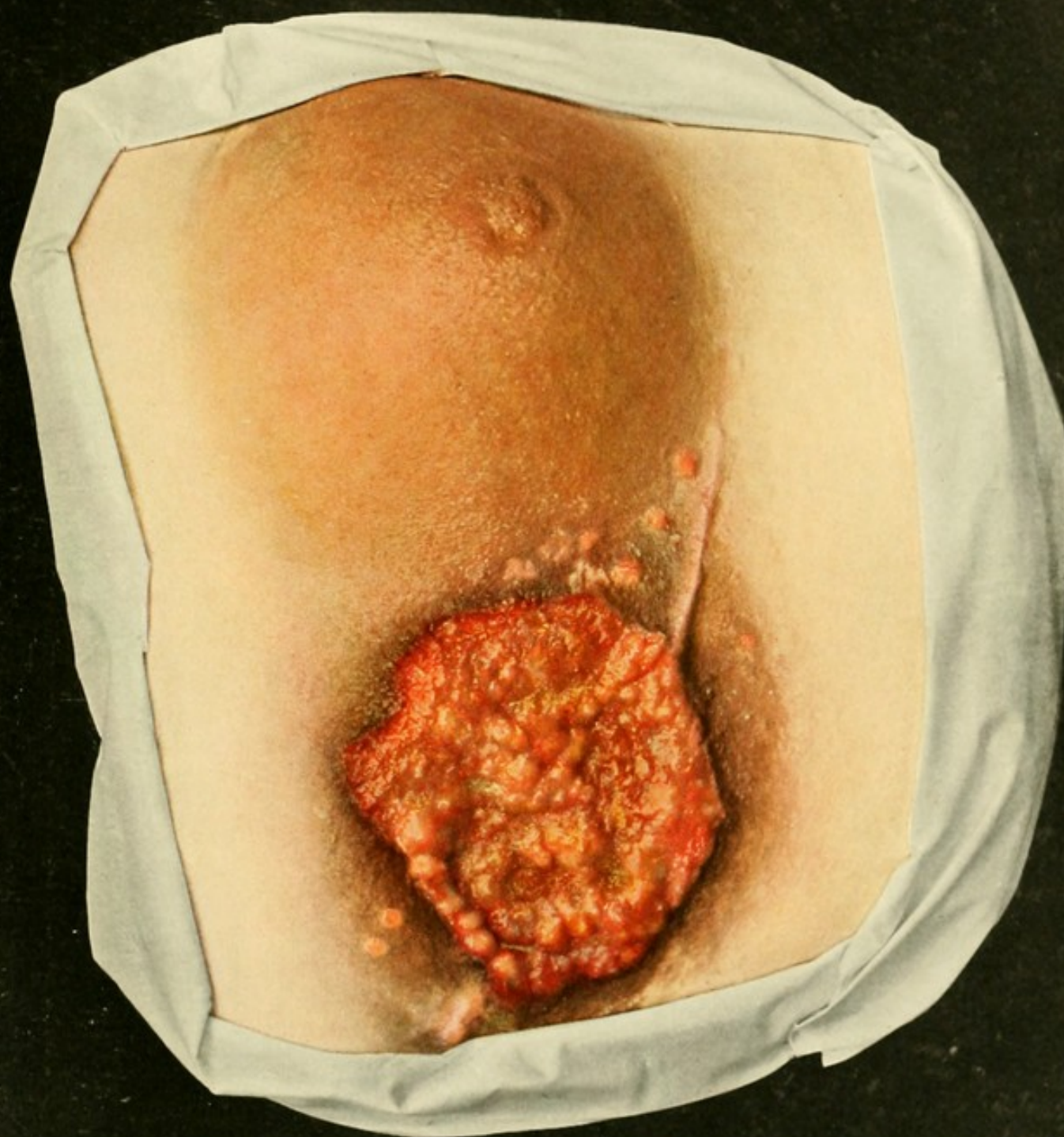
Fig. 15. Carcinoma mammae utriusque. — Cancer en cuirasse.

the subjacent mammary tissue, by means of an oval incision with subsequent suture. Recurrence is rare after early treatment. In doubtful cases with induration of the mammilla excision should always be performed.

Fig. 13. *Paget's disease,* or *chronic eczema of the nipple,* which is refractory to all treatment. The eczema begins on the nipple and gradually extends to the areola and surrounding skin. Retraction of the nipple and dragging pains are caused by the presence, under the nipple, of carcinoma (cylinder-epithelioma), which at first has no connection with the nipple, but later on may become attached to it. The mammary gland in this case shows hard infiltration around a nodule. In the normal parts of the skin there are small dimples. Obstinate eczema of the nipple accompanied by a tumor in the breast, with infiltration of the axillary glands and early cachexia, make the diagnosis clear and indicate removal of the whole mammary gland with the axillary glands. In cases of chronic eczema of the nipple resisting all treatment, excision of the mammilla is advisable. Out of 884 cases of mammary carcinoma in *v. Bergmann's* clinic there were only seven typical cases of Paget's disease. Two of the author's cases showed cancer of the mammary gland without connection with the eczematous nipple.

Fig. 14. This is a case of *tubular carcinoma* with cutaneous dissemination which has extended in all directions and spread over the thorax. The development of nodules in the skin occurs early. These appear at first as punctiform, bluish, glistening elevations, which increase in number and size and coalesce, forming a kind of cuirass inclosing the thorax in a rigid mass (*"Cancer en cuirasse"*). These cases are inoperable.

Fig. 15. This is a case of *inoperable cancer "en cuirasse,"* in which both mammæ are affected with carcinoma. On the right side there has been a recurrence of the growth in the scar soon after operation, where a soft, fungating, easily bleeding ulcer is seen. In the surrounding skin there are several isolated nodules. The left mammary gland is involved in a hard, immovable, carcinomatous infiltration. The transmigration of a carcinoma from one side to the other is possibly explained by the persistence of congenital lymphatics.

Fig. 16. At first sight this appears to be a pyogenic inflammation. (Compare with **Figs. 85** and **86.**) However, the bluish color, the retraction of the nipple, the hard, immovable breast forming a large tumor, and the extensive metastases in the axillary and supraclavicular glands lead to a diagnosis of carcinoma. *Volkmann* has named this not very rare form of cancer **mastitis carcinomatosa** and *Schurman* **carcinoma mastitoides**. That we have here to deal with an affection of the lymphatics (lymphangitis carcinomatosa) is shown by the punctiform red spots between the two breasts, the larger punctiform or circular spots below the clavicle and the changes in the region of the neck. The latter is of a blue color and the seat of a hard infiltration, which is not inflammatory, but due to plugging of the lymphatics with cancer cells, and consecutive edema. This form of cancer is hardly seen except during pregnancy or lactation, when there seemingly exists a special vulnerability of the breast cell. Therefore this acute form of carcinoma is seen more frequently than the other forms in young women; and in not a few instances, it is bilateral.

The last three plates (**Figs. 14, 15** and **16**) show the terrible effects of advanced cancer of the breast, so that the necessity for the **earliest possible diagnosis and radical removal** by operation must once more be set forth emphatically.

Fig. 17 shows a rapidly growing tumor developed, in a man aged 37 years, on a congenital nævus. Degeneration of such a nævus, of an old scar, ulcer, wart, sebaceous cyst (**Fig. 18**) or mole, is the origin of all **cancers of the scalp,** which are very rare. Papilloma, sarcoma and melanoma are other possible evolutions.

The cutaneous covering of the nævus was quickly destroyed and the tumor was formed by cauliflower growths separated by deep fissures. The ulcerated surface was covered with sanious secretion, so that the naked-eye appearance was not sufficient to establish whether the case was one of sarcoma or carcinoma. However, the malignant character was not doubtful, on account of the rapid growth, the cachexia and the lymph-gland metastases, which soon extended along the large vessels of the neck down to the supraclavicular fossa. These lymphatic metastases favor the diagnosis of carcinoma as against that of sarcoma.

This case was inoperable. A less advanced tumor, without any

Fig. 16. Carcinoma mammae. — Lymphangitis carcinomatosa.

Tab. XII.
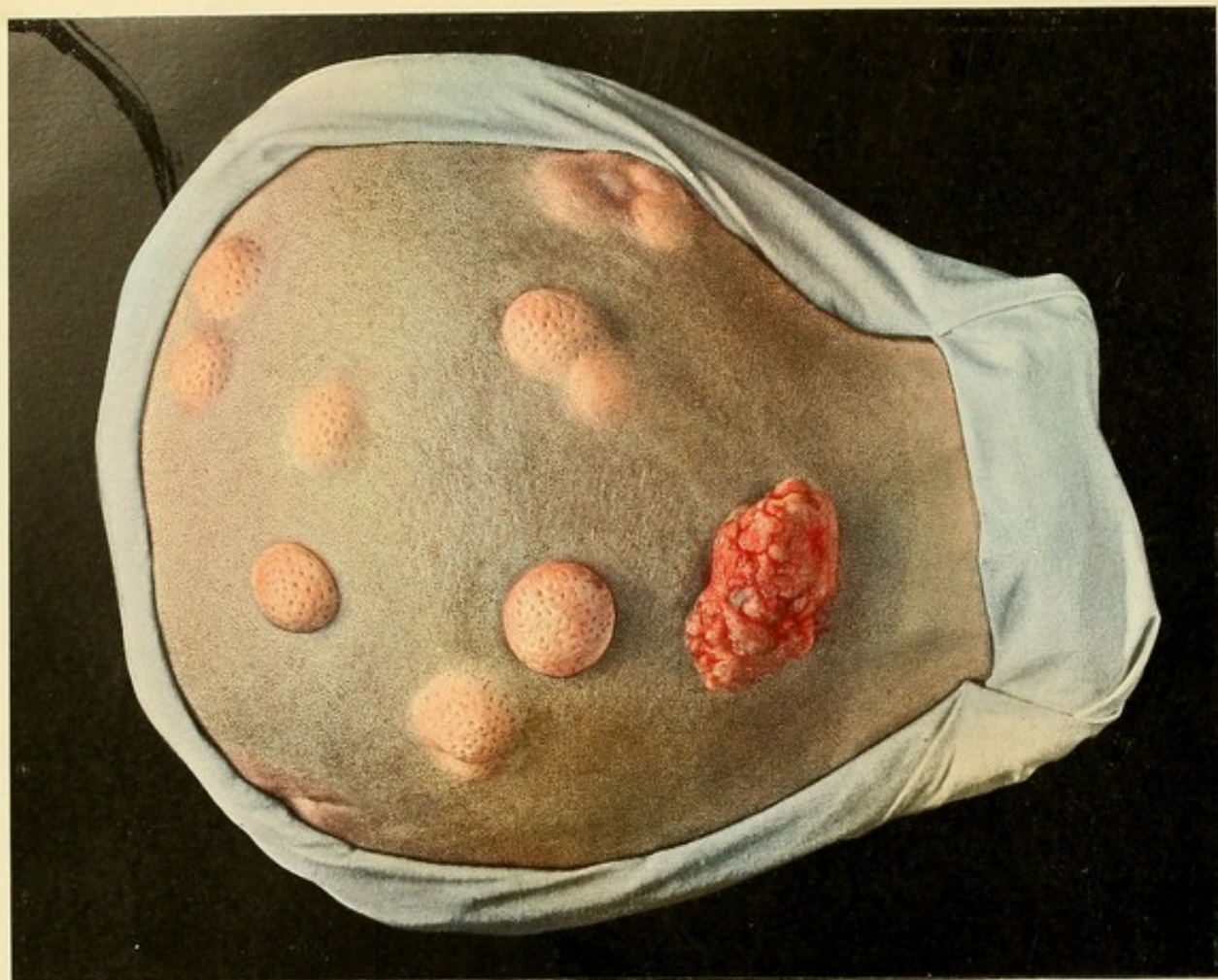
Fig. 18. Atheromata — Carcinoma cutis.

Fig. 17. Naevus verrucosus — Carcinoma cutis.

palpable lymph glands, would require a wide excision of the tumor and of the nævus. (For the treatment of nævi, *see* **Figs. 67, 68, 76** and page 104). As soon as changes of any kind appear in a nævus, it is important to remove it forthwith. It is best to excise all pigmentary nævi because they are too often the starting point of fatal melanotic growths (*see* **Figs. 23** and **28**).

Fig. 18 shows a *carcinoma of the scalp originating in a sebaceous cyst.* Ordinary, non-degenerated, sebaceous cysts are scattered over the whole scalp. Beginning as small nodules inlaid in the skin, these cysts slowly grow into large tumors with a broad base and smooth surface. They are fixed to the skin, but easily movable over the subjacent bone, and have a doughy consistency often resembling fluctuation. If this mobility of the cyst over the subjacent tissues ceases and the originally soft tumor becomes a hard nodule with an irregular, rough surface, malignant degeneration is to be suspected; apart from the occurrence of calcification in its walls, in which, moreover, the spherical, smooth surface is generally preserved. This suspicion becomes a certainty when the skin gives way and there appears a rapidly growing nodular tumor characterized by multiple lobulation and secreting a fetid discharge. Such a carcinoma resembles in many ways an ulcerated sarcoma (**Fig. 33**), and often causes severe pain owing to inflammation around the tumor. Cachexia occurs early, and the patients are usually of advanced age.

The dignosis of carcinoma depends on the characteristics mentioned above and later on the hard multiple glandular enlargement, which affects the whole nape of the neck. This usually occurs late and is not so hard in sarcoma.

Treatment

This consists in *extirpation* of the carcinoma, and involves removal of part of the external table of the skull on account of the tumor being fixed to it. The extensive space left by removal of the tumor can be sutured after making two long lateral incisions over both ears and undermining the scalp. The spaces left by the lateral incisions can be repaired by *Thiersch* grafts. The glands in the nape of the neck must also be removed.

On account of the early appearance of glandular metastases the excision of especially indurated sebaceous cysts is indicated. Moreover, as there is always a possibility of malignant degeneration, it

is advisable to remove every sebaceous cyst by dissecting it out, so as to avoid recurrence.

Fig. 19 shows the ordinary clinical appearance of **carcinoma of the penis.**

Carcinoma of the penis begins on the glans or in the coronary sulcus as a squamous-celled epithelioma, generally between the fiftieth and seventieth year. Predisposing causes are all chronic irritations of the region; for instance, congenital phimosis with preputial concretions, leucoplakia preputialis (white, glistening patches similar to leucoplakia of the tongue and cheek, see page 8), warts, long-standing tuberculous and syphilitic ulcerations. Old fistulæ, which occur especially in eunuchs after removal of the scrotum, testicles and pendulous part of the penis, near the symphysis or perineum, also predispose to carcinoma. "Chimney sweep's cancer" is a cutaneous cancer observed on the scrotum and is due to the irritation of soot and dirt.

The usual form of penile carcinoma is that represented in the figure, a warty carcinoma which destroys the prepuce and soon forms a cauliflower growth. Between the separate hard nodules destitute of skin appear crateriform excavations which are characteristic. Epithelial plugs can be expressed from the growth, and in other parts the surface is cornified. Thus, continuous growth alternates with permanent disintegration. The rapidly developing nodules often cause exhausting hemorrhage, while the breaking down of the carcinoma gives rise to a fetid sanious discharge. The borders of the growth are hard, raised and prominent. The whole penis may be transformed into a large tumor, which may extend to the scrotum, testicles and pelvis. The growth may destroy the urethra and cause much pain on micturition.

A more rare form of carcinoma arises as a small ulcer, generally on the corona glandis. It is hidden by the resulting phimosis, but its characteristic hard borders can be felt distinctly and there is a sanious secretion. The inguinal glands are affected early and point to the diagnosis of carcinoma. The growth at first causes the patient little inconvenience, but quickly leads to severe cachexia, so that the patients often present themselves with extensive metastases of the inguinal and retroperitoneal glands, and are in an inoperable condition. A saying of *Kauffmann's,* "In old men with phimosis and offensive discharge the possibility of cancer is always to be borne in mind," merits special consideration.

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Tab. XIII.
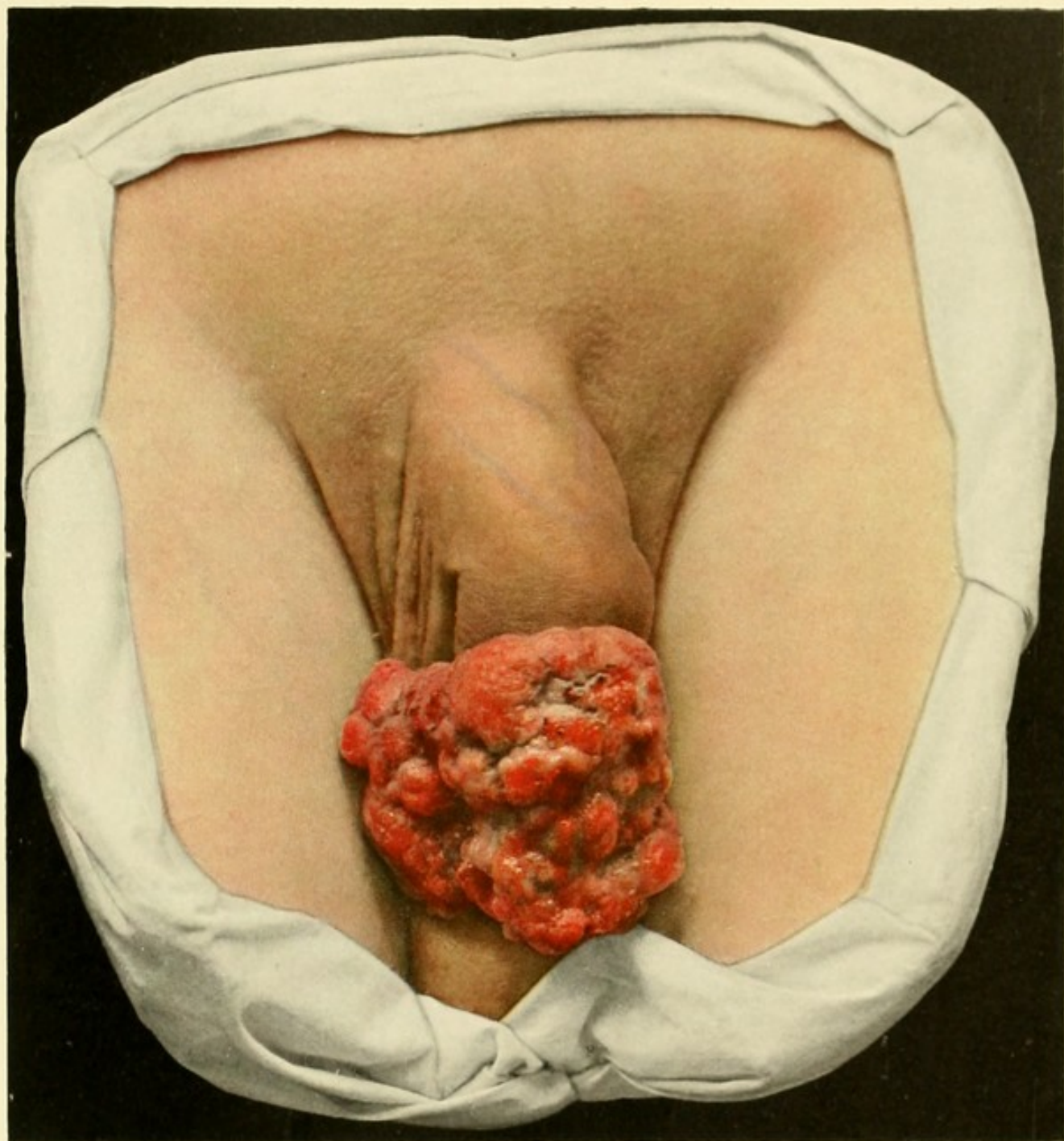
Fig. 19. Carcinoma penis — Leukoplakia.
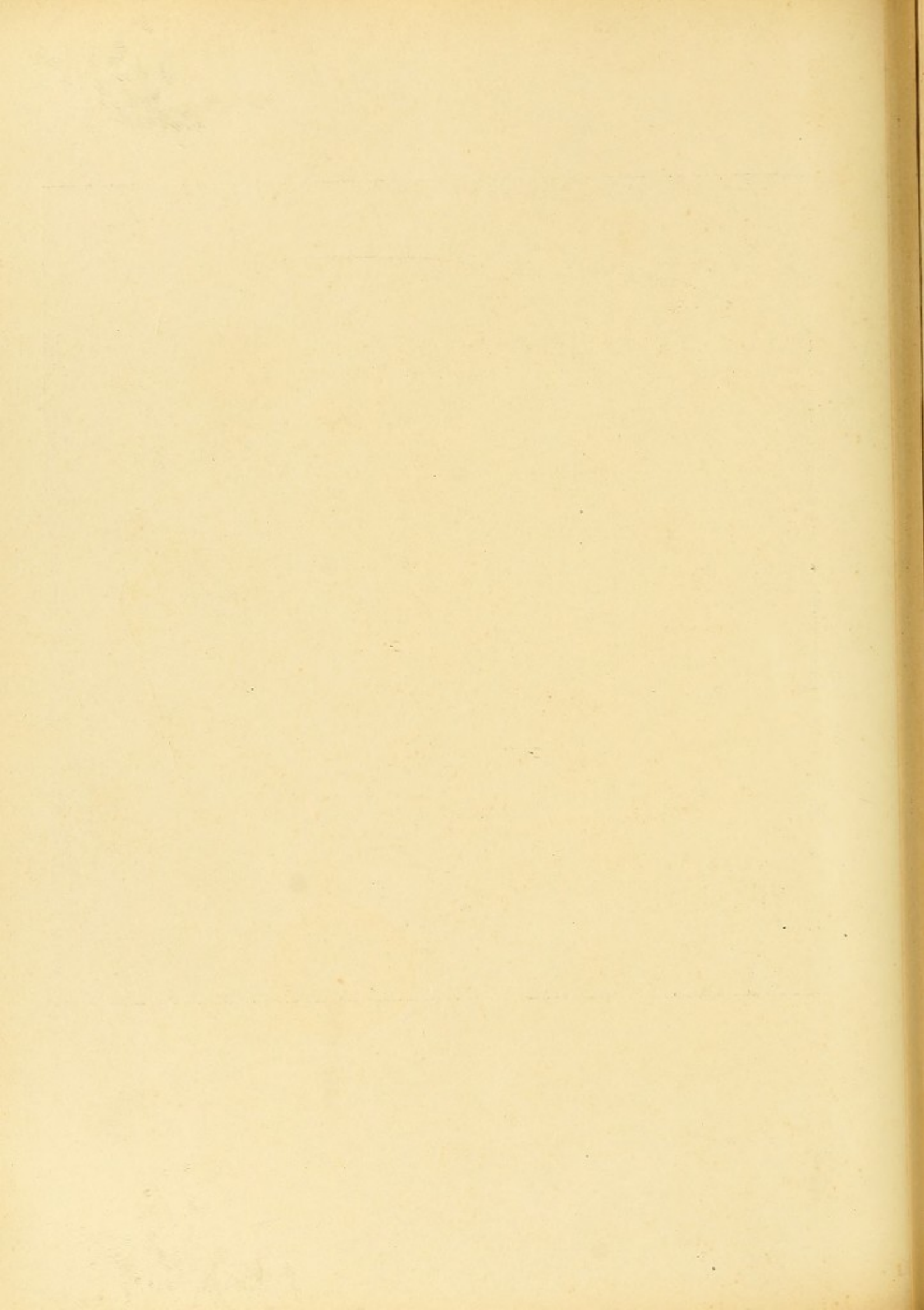
plugs. A cauliflower tumor grows, which soon becomes fixed to the fascia (**Figs. 20** and **22**).

Warts, old-standing ulcers of the leg and lupoid changes in the skin also lead to carcinoma of the extremities. Eczema of the skin occurring in chimney-sweeps and workers in paraffin has often led to multiple carcinoma of the extremities and scrotum.

Fig. 20 shows a *papillary carcinoma of the skin* of the leg arising from the scar of a burn. The smooth, partly white and partly brownish, shiny scars of the burn are seen over the whole leg. The carcinoma has extended above and below and has extended around the whole circumference of the leg. The soft, cauliflower proliferations have given rise to severe hemorrhages. From the depth of the growth oozes a sanious discharge. The borders of the tumor are very hard and raised, and are immovable over the fascia. The inguinal glands were already involved.

Amputation was performed through the thigh, and the inguinal glands were removed. Though adenitis, in cancer of limbs, may, perhaps, not be a formal contraindication of operative inference, it considerably increases the chances of a recurrence. Therefore, here also, as in all cases of tumors, an early diagnosis is essential. As soon as a chronic ulcer of the leg begins to show marked induration of the borders and proliferation, the suspicion of carcinoma is justified, and it is best to remove the whole ulcer as soon as possible.

For **X-ray carcinoma,** see **Fig. 138** and page 219.

Fig. 21 shows a *carcinoma* in a common situation, the *back of the hand,* arising from a wart and forming a characteristic carcinomatous ulcer. As the growth was still movable over the fascia, and there were no glandular enlargements, it was excised and the gap repaired by a pedunculated flap from the forearm. The rapid growth of these small tumors with hard borders makes early diagnosis and removal necessary, so as to avoid recurrence. Compare with *rodent ulcer* of **Fig. 1.**

Fig. 22 shows a very extensive *carcinoma* arising from the *scar of an injury* two years before. In this case the irregular, wall-like,

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Tab. XV.
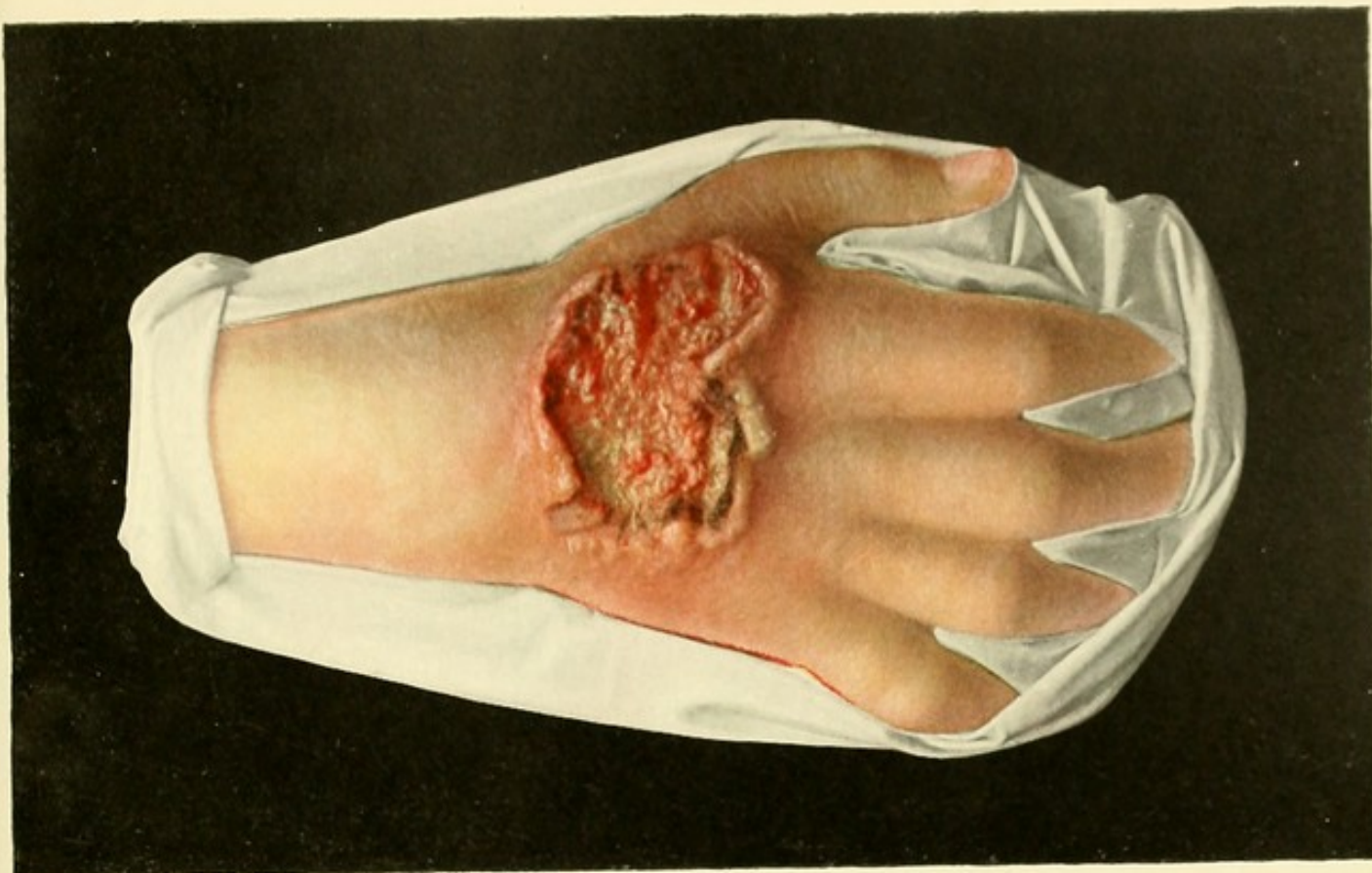
Fig. 22. Carcinoma cutis ex cicatrice.
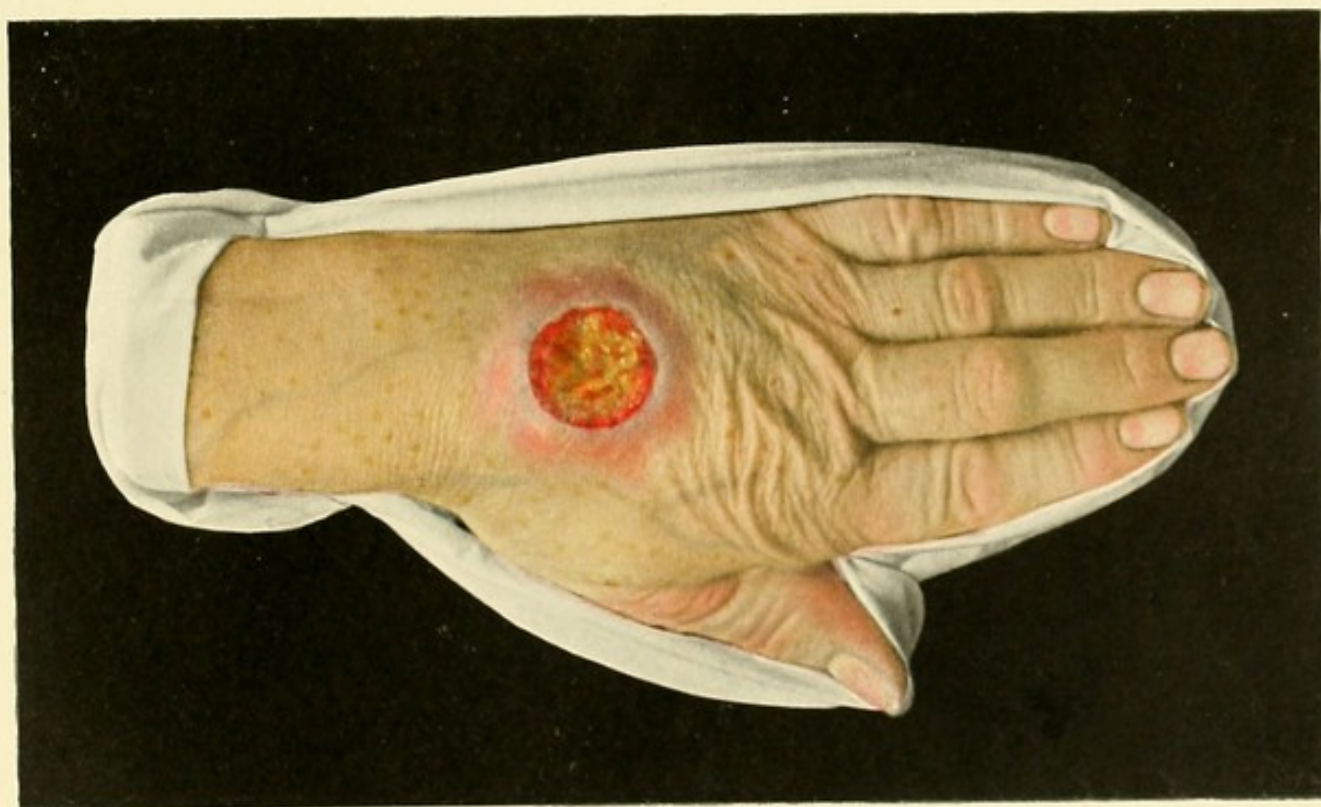
Fig. 21. Carcinoma cutis ex verruca.
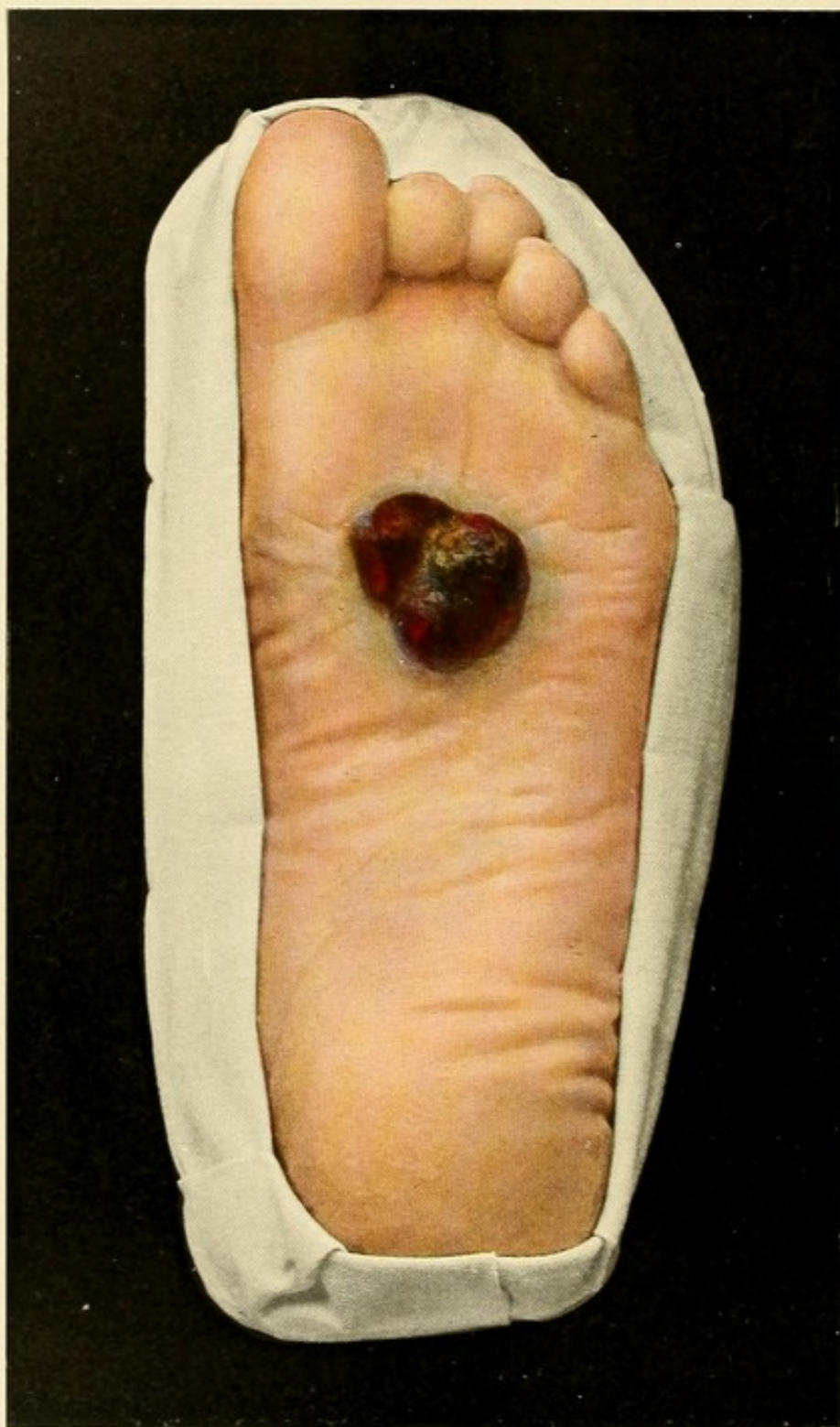
Fig. 23. Melanocarcinoma cutis ex verruca.

hard, irregular borders are very marked. The floor of the ulcer is in some places cornified and is covered with crusts and sanious secretion. The carcinoma has already extended through the fascia to the bones, interfering with the function of the hand. The glands of the elbow and axilla are hard and nodular. The rapid growth of the tumor has led to severe cachexia. This was treated by amputation through the arm and removal of glands, and calls for the same remarks as **Fig. 20,** about early excision of a scar showing suspicious symptoms of degeneration.

Fig. 23 shows a tumor arising from a pigmentary wart of the sole of the foot, the alveolar structure on microscopic examination showing it to be a **melanotic carcinoma.**

(Compare **Fig. 28, Melanosarcoma.**)

Malignant melanotic tumors (sarcoma, endothelioma and, more rarely, carcinoma) occur most frequently in the skin and adjacent mucous membranes, and in the choroid and iris. In the skin, they arise from pigmented benign tumors, flat, pigmentary nævi, and from warts subjected to repeated irritations. Warts on the sole of the foot, the toes and the fingers often degenerate in melanosarcoma. A tumor develops, *black, bluish*, or *brownish-yellow* in color. The skin soon becomes ulcerated, the tumor breaks down and a *deep, ragged, black or blue ulcer* is produced. A melanosarcoma remains soft. A melanocarcinoma produces an ulcer with hard edges.

All melanotic tumors are **extremely malignant** because they propagate and disseminate with the utmost rapidity. Small black nodules appear in the neighboring skin; soon the lymphatics are involved, cachexia develops and a miliary crop of small growths is found at the autopsy in most organs, particularly the brain, the lungs and liver. Some of the growths are full of pigment as in the mother tumor; others, younger, do not yet contain any, and are white or pinkish on section. The deposit of melanin in the growth is, therefore, secondary.

Melanocarcinomata may be seen in children as multiple growths in the skin in connection with xeroderma pigmentosum. (See page 1). The rapid growth and frequent hemorrhages lead to severe anemia.

The appearance of melanotic growths is so typical that no confusion is possible.

The best treatment is prophylaxis, which consists in the precautionary removal of pigmentary nævi, warts, especially those that are subject to constant irritation. Repeated cauterization of nævi and warts is to be condemned.

Once a melanotic tumor has developed, a radical operation may be performed, if the tumor is situated on a limb and the regional glands are not yet involved. But, in spite of this (in the case represented in **Fig. 23,** amputation of the leg and removal of the inguinal glands), early recurrence is the unvarying rule. No kind of new growth possesses as high a dissemination power as malignant melanoma.

Tab. XVII.

Fig. 24. Lymphosarcoma colli.

SARCOMA

Figs. 24 to **35**, inclusive, represent *different types of sarcoma*.

Sarcomata (thus named from their fleshy appearance on section) are tumors developed from connective cells and which, therefore, may originate in any organ containing connective tissue, that is, practically everywhere in the body.

Owing to the often very rapid growth, the newly formed cells do not attain complete maturity, so that the sarcoma consists of imperfectly developed connective tissue. In its early stages it often resembles, microscopically, inflammatory granulation tissue, but by its rapid growth it soon assumes the character of a malignant tumor. The bulk of the sarcoma is formed of various connective tissue cells, while the interstitial fibrous tissue is scanty. *There is in sarcoma an abundant formation of new blood-vessels, which is characteristic;* so that any new growth accompanied by a marked collateral circulation or possessing itself a great vascularity is very likely sarcoma.

The transition of fibroma, especially of such as arise from the connective tissue of fascia, and of other connective tissue tumors, *e.g.*, chondroma, into sarcoma has been demonstrated.

Patients often attribute these growths to various injuries, but there is no direct proof of this, though *Phelps, Coley, Segond* admit the possibility of a causal relation between both.

Pure sarcomata are classified according to their microscopic structure into round-celled, spindle-celled and giant-celled sarcomata. Those formed of various tissues are known as lympho-, myxo-, fibro-, chondro-, angio-, and glio-sarcomata. Pigmentary or melanotic sarcomata are placed in a special group.

Clinically, sarcomata are best divided into soft, many-celled, quickly growing, very malignant, easily recurring medullary sarcoma (usually small, round-celled sarcoma); and hard, few-celled, slow-growing, less malignant forms (spindle-celled and giant-celled sarcoma). In the first form the soft consistence is due to the richness in cells and the scanty development of interstitial tissue. As compared with carcinoma, sarcoma is more circumscribed and is at first almost completely encapsulated, with borders as soft as the rest of the tumor.

Frequently, owing to hemorrhages and softening in the interior of the sarcoma, cystic cavities are formed which can be recognized by the presence of fluctuation (**Figs. 25** and **30**). Sarcomata situated under the skin gradually destroy and break through the latter and proliferate on its surface in various forms. Fleshy reddish-brown parts alternate with yellowish-white, pulpy parts in these tumors. There are usually blood extravasations, both old and recent. The whole tumor has the appearance of a fungoid mass (**Figs. 26, 27, 29** and **33**). After a time these superficially proliferating growths break down and become septic, so that the characteristic appearance of the sarcoma is lost, and, on the scalp and extremities, for example, it cannot be distinguished from an ulcerated carcinoma. As the sarcoma usually breaks through the skin and proliferates on the surface, so may it extend into all the deeper tissues, so that finally an enormous tumor is formed which may destroy the bones (**Figs. 25, 27** and **33**).

The second form, the slow-growing, few-celled tumors, resemble fibroma and often represent transitional forms (fibro-sarcoma). The latter sometimes occur as multiple nodules in the skin.

Sarcoma often occurs in robust people in middle life (between 30 and 50). Sometimes it is congenital or appears in infancy (kidneys and testicles), or soon after puberty (mammary gland). The earlier the tumors appear, the more malignant they are, as a rule. Multiple sarcomata are seen in the skin as pigmentary sarcomata (**Fig. 31**) and in the bones.

Soft sarcomata lead to metastases much more frequently than hard forms. Metastatic deposits are formed by growth of the tumor into the large veins and the formation of emboli, which are carried to the lung, spleen, liver and brain. Dissemination by way of the lymphatics is usually absent. The latter, however, are certainly often involved, especially in ulcerated sarcoma, in melanotic forms and in osteosarcoma.

Sarcoma is accompanied by a **thermic ascension,** either general or local, or both, due to the resorption of toxins.

In many cases the body is so quickly filled with metastases that the patients soon succumb from severe anemia and toxemia. Unfortunately, they often do not apply for treatment before metastases render the condition hopeless.

Differential diagnosis

Sarcoma may be mistaken for a **benign tumor,** for a **gumma,** for a **carcinoma.** The latter is a *hard* tumor, while sarcoma is gen-

erally *soft;* however, it does not matter if a mistake be made in this direction, since both conditions call for the same radical surgical treatment.

The other mistakes are more serious: a benign tumor is distinguished by its slow evolution; but, in doubtful cases, it would be most unwise to delay making a diagnosis till the latter has become evident.

Exploratory incision with direct inspection, and excision of a piece for immediate microscopical examination is the best procedure, care being taken not to submit the tumor to unnecessary manipulation. It goes without saying that such a partial excision with microscopical examination is only for those organs the conservation of which is important (a limb for instance). In others, such as the breast or the skin, immediate complete excision of all suspicious tumors is indicated.

A **gumma** may be mistaken for sarcoma; but here the antecedent history, the *Wassermann* reaction and the quick influence of salvarsan treatment help clear up all doubts. In former times, the mistake seems to have been frequent. *Esmarch* claims that many growths were extirpated as sarcoma that might have been cured by antisyphilitic treatment.

A **local hyperthermia,** evidenced by the color of the skin and the sense of touch, and an **abundant development of collateral venous circulation,** mapping out a bluish network under the skin (*see* **Figs. 25** and **32**), are always particularly suspicious symptoms when an underlying newgrowth is felt by palpation. So is the *withdrawal of only pure blood from a tumor* by exploratory puncture.

Treatment

All tumors in which there is a suspicion of sarcoma must be removed as early and as radically as possible. Conservative operations have no place in the treatment of ordinary spindle or round-celled sarcoma except as stated below, even if the latter seems still to be encapsulated. Sarcoma of the limbs demands exarticulation or amputation high up above the newgrowth. Recurrences is very frequent; it is almost without exception in the soft varieties. The harder a sarcoma, the fewer, and the less embryonic in nature the cells it contains, and the less malignant the tumor; so that in the very hard varieties of bone sarcoma, conservative operations (*e. g.,* resection) sometimes give permanent results.

Bloodgood goes so far as to say that giant-celled bone sarcoma is not a malignant tumor, and that it can be cured permanently by

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resection or scraping followed by treatment of the cavity with boiling water. This opinion is accepted by many surgeons, and there is no doubt that this hard giant-celled variety is at least comparatively benign.

Many cases have been treated by the X-rays. While some authors contend that the X-rays transform the embryonic cells of sarcoma into adult tissue, and, therefore, sarcoma into fibroma, others say that the action is only superficial and similar to that caused by an intercurrent erysipelas, which often brings about a temporary shrinking of sarcoma.

It is interesting to note, in this respect, that *Coley,* treating inoperable cases of sarcoma by injections of a mixture of toxins of the microbe of *erysipelas* and of the *Bacillus prodigiosus,* claims to have obtained remarkable results (sometimes permanent cures) in about 10 per cent. of cases. He also advises injection of the mixed toxins after operation as a prophylactic against recurrence. *Coley* always begins by operating largely, and uses toxins alone only when operation is refused.

Fig. 24 shows an **extensive tumor** involving the whole right side of the neck, and made of several nodular, irregular formations. The skin is broken in one place, in other places it is thin and bluish-red in color. There is a fistula discharging a sanious secretion. This tumor is a **lymphosarcoma.**

The region of the neck, where lymphatics are abundant, is the seat of predilection of three different malignant processes involving the lymph glands.

1. **Lymphadenoma** (also called *malignant lymphoma, Hodgkin's disease, pseudoleukemia*).
2. **Lymphosarcoma,** sarcoma of the *lymphoid cells* of the lymph glands.
3. **Sarcoma** of the connective tissue of the same lymph glands.

The latter two are often not distinguished from one another. A hard, diffuse, nodular tumor quickly develops from a group of small, hard, movable glands and shows its malignancy, especially in young individuals, by the continual formation of fresh nodules at the periphery, which coalesce with the main tumor and cause it to attain a considerable size. The unlimited growth into the neighboring tissues is characteristic. The capsule of the gland is quickly broken through, a fact which does not happen in other lymph gland tumors of the neck. The cervical fascia is destroyed and the sterno-mastoid

muscle invaded. The skin is at first reddish, then bluish-red or livid; it then becomes thin and gives way over the tumor. The exposed parts of the tumor rapidly break down while the sarcoma grows into the deeper parts, especially into the internal jugular vein, giving rise to fatal organic metastases. The vagus nerve and the common carotid also become ensheathed in, and may be destroyed by, the tumor. Dyspnea and dysphagia may be caused by pressure on the larynx and esophagus. This occurred in the case represented in **Fig. 24.** The tumor extends downward into the mediastinum and may even destroy the vertebræ.

Diagnosis and Treatment

To recognize that a tumor of the neck is made of lymph glands is not difficult. Its situation in regions where lymph glands normally exist, and the palpation of *rounded nodules* is sufficient. To decide what is the nature of the condition is not so easy.

Lymphosarcoma is distinguished from other lymph gland tumors of the neck *by its rapid growth in all directions,* its *breaking through to the exterior* and its fungating masses. But, in the earlier stages, it has no pathognomonic symptoms, and microscopical examination itself may be inconclusive, so that the nature of the affection can only be suspected.

Lymphadenoma (*Hodgkin's* disease), which usually begins in the neck, consists of small, multiple, encapsulated nodules, which do not break down nor extend into the neighboring organs. There are generally also glandular enlargements in the axillæ, groins and mediastinum, and changes in the spleen and bone-marrow.

Tuberculous glands resemble *Hodgkin's* disease so much that many of the cases reported as being the latter have been found either to be purely tuberculous or, at least, to contain tuberculous lesions. The confusion is not possible when there is a history of several years' duration, other symptoms of scrofula, and groups of glands of different consistency, some hard, some soft, some fluctuating. But the diagnosis is exceedingly difficult—in fact, sometimes not possible—when there are but few hard, movable glands involved.

Syphilitic glands are at first hard, later on soft; but are not so extensive, and there are other symptoms, which soon clear the diagnosis.

Branchiogenous carcinoma (*Volkmann*), arising from the remains of the epithelium of the branchial clefts, is very rare and appears as very hard, spherical tumors in the carotid fossa.

The tumors affecting the sheaths of the blood-vessels of the neck,

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first described by *Langenbeck,* and considered by some as lympho-sarcoma, or rather as sarcomata of the lymph gland stroma having involved at an early period the vascular sheaths, are regarded by many as *peritheliomata,* that is, tumors developed from tissue much resembling endothelium, but situated outside of the blood vessels (about endothelioma, *see* **Fig. 39** and page 48).

Metastatic carcinoma and sarcoma can be diagnosed by the presence of the primary tumors (scalp, *see* **Fig. 28,** esophagus, parotid, maxilla). It must be said that lymph gland metastases may exist in the neck while the primary tumor in the scalp, a mole for instance, does not show any apparent activity.

Actinomycosis may also cause hard infiltration of the neck, but the infiltration is diffuse and uniform, not nodular, and extends over the whole region of the neck. If there is a fistula, the pus contains the characteristic yellow bodies in which the microscope demonstrates the ray-fungus (*see* **Figs. 115** and **116** and page 175).

The only treatment of lymphosarcoma is *radical extirpation,* if the diagnosis is made early enough. Even then, the operation is very extensive and often entails dissection or excision of the big blood vessels of the neck. Recurrence is very frequent. The results of surgical treatment being a very doubtful compensation, some are content with X-ray treatment and high doses of arsenic internally. One may obtain a temporary remission. This treatment was followed in the case shown in **Fig. 24,** but without any noticeable success.

Fig. 25 shows a soft, partly fluctuating growth with fungating borders, which has begun to extend over both eyes. It is fairly symmetrical on both sides of the middle line, a feature which denotes the origin from the basilar process, in distinction to the more lateral swelling of retro-maxillary tumors. In some places the skin is so thin that it appears livid and transparent; in others it shows the marked vascularity so often observed in sarcoma. The tumor is on the point of breaking through.

The disease was of ten years' duration. Several non-malignant polypi had been removed previously at intervals, and also a larger tumor, after partial resection of the upper maxilla.

The lesion now represented is an **epipharyngeal sarcoma,** or malignant nasal polypus, which has grown forward and destroyed the whole bony framework of the nose. On digital examination the whole nasal cavity and naso-pharynx were found filled with soft,
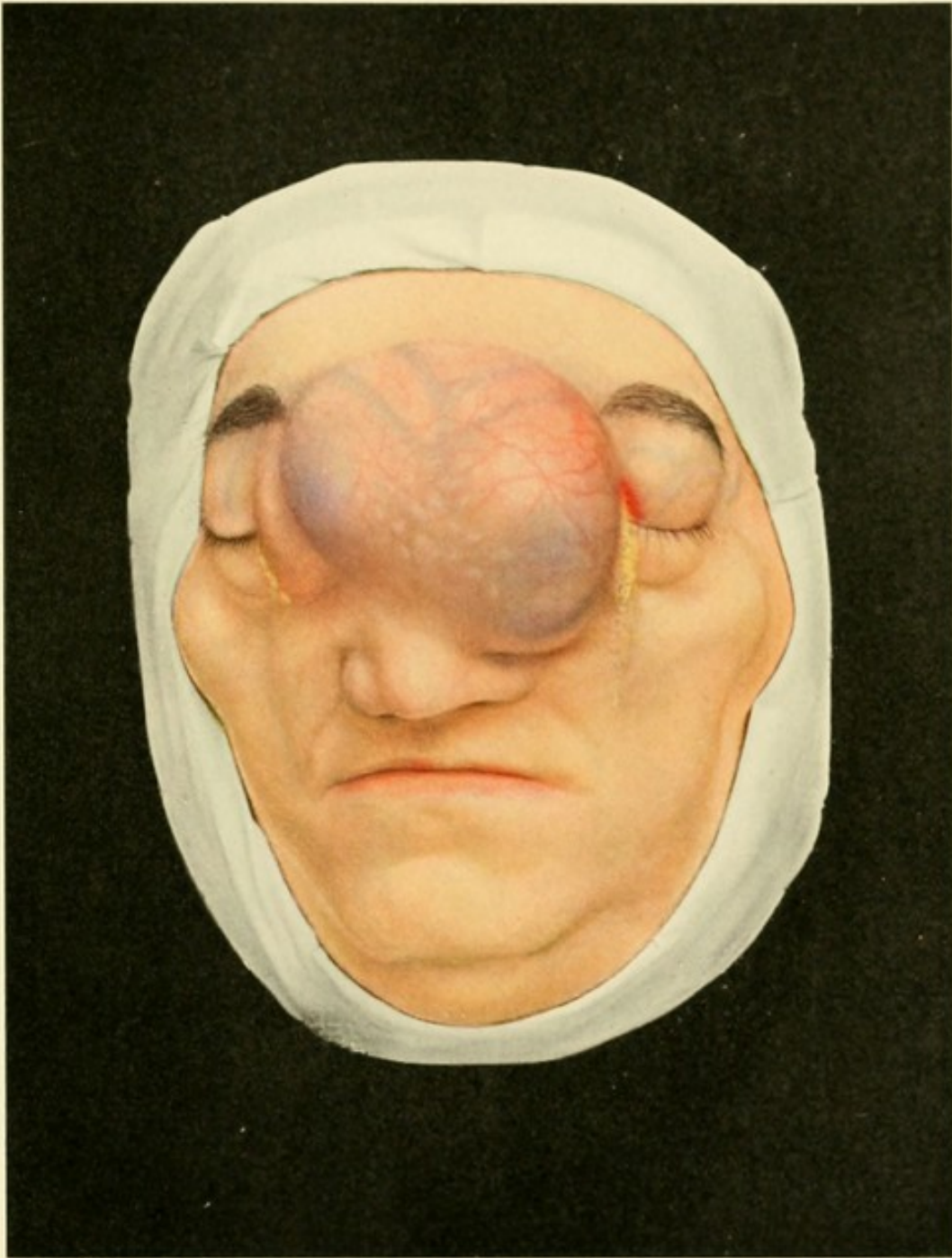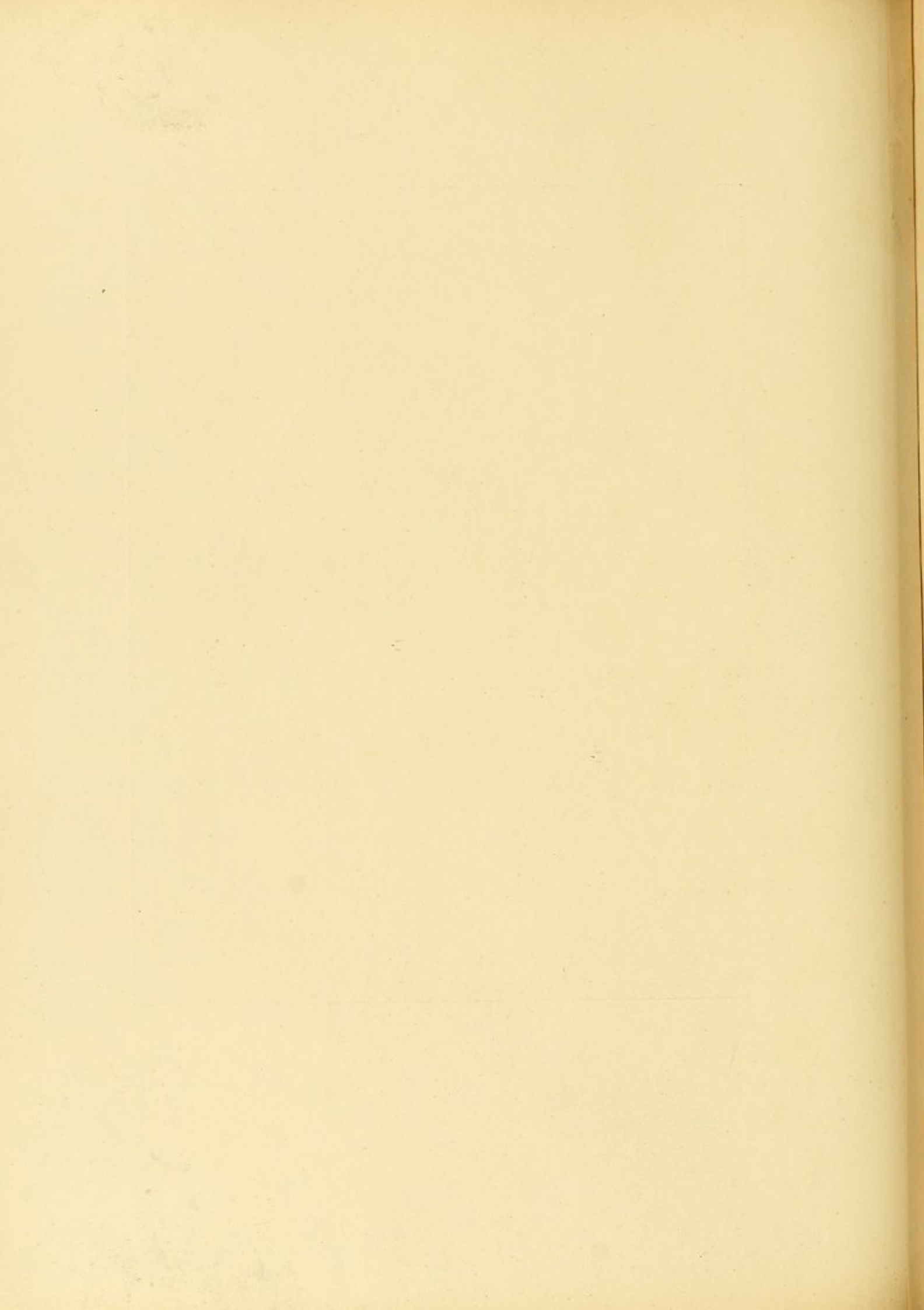
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Fig. 25. Sarcoma epipharyngeale — Polyposis nasi maligna.

infiltrating tumor masses, which had displaced the palate downward and forward. The tumor had also extended through the base of the skull.

In the naso-pharynx two kinds of growths claim special attention—*fibromata,* usually occurring in males between the twenty-fifth and thirtieth years, arising from the basilar process, also called nasopharyngeal polypi—and *sarcomata,* which appear between the thirtieth and fiftieth years. Tumors arising in the spheno-palatine fossa are sometimes described separately as retro-maxillary tumors, but after further extension they cannot be distinguished from the two mentioned above.

Fibromata, which occur earlier in life, generally arise from the connective-tissue cells of the periosteum as pedunculated or sessile encapsulated tumors, which by extensive growth fill up all the spaces and apertures of the naso-pharynx, especially the posterior nares, cause atrophy of the bones by pressure, and break through into the nasal cavity, maxillary antrum and cranial cavity. On account of their great vascularity, these growths, which in some places often take the character of cavernous tumors, are of much softer consistency than other fibromata. They may ulcerate on the surface and give rise to exhausting hemorrhages. On account of their tendency to increase and the frequent sarcomatous degeneration, they are to be dealt with as malignant growths.

In older individuals, in the majority of cases, we have to do with true sarcoma, arising from the periosteum or fascia (malignant nasopharyngeal polypi), which extends to the posterior nares, the sphenomaxillary fossa, Eustachian tubes and larynx; not, however, as encapsulated tumors like fibroma, but as soft, fungoid, sessile, firmly attached growths with an irregular outline. Later on they grow very rapidly, destroy the neighboring bones, and reach the external surface through the frontal sinus, nasal cavity and orbit; internally they extend to the brain (**Figs. 25** and **27**).

Disintegration of the tumor goes hand in hand with the increase in size and the patient succumbs from the results of hemorrhage, septic infection, anemia and organic metastases.

The clinical symptoms in fibroma and in commencing sarcoma arise from obstruction of the naso-pharynx. Continual mouth-breathing is suggestive of disease of the naso-pharynx. Owing to obstruction of the posterior nares the patients snore during sleep; they acquire nasal catarrh (often atrophic rhinitis) and have a nasal voice. As the tumor grows, obstruction of the Eustachian tubes

causes deafness and pain in the ear; extension to the cranial cavity gives rise to headache, somnolence and choked optic disk; extension to the orbit is attended by disturbance of vision, *e.g.,* diplopia. Pressure on the facial and trigeminal nerves results in paralysis and severe neuralgia.

Diagnosis and Treatment

The diagnosis of these advanced cases presents no difficulty. The **soft, fungoid consistency** of the whole tumor, the **tendency to bleeding** and the rapid growth are characteristic. In extensive sarcoma with commencing disintegration and discharge soft metastases are found in lymph glands. Incipient sarcoma can be recognized by digital and rhinoscopic examination as an irregular, rough, infiltrating tumor, which differs from the nodular, encapsulated fibroma.

It is only in young individuals that other conditions can be confounded with true tumors of the naso-pharynx. *Hypertrophied tonsils* and *extensive adenoids* cause somewhat similar symptoms, but direct examination by the finger and rhinoscopy will make the diagnosis clear. In very young children *teratomata* are seen (**Fig. 146**), which may be mistaken for sarcomata arising from the basilar process and extending to the face. However, teratomata are usually more or less encapsulated and only appear on one-half of the face.

Retro-maxillary tumors manifest themselves at first by unilateral pain in the face, swelling of the cheek and fixation of the corresponding maxillary joint, but on further extension they cannot be distinguished from advanced tumors of the naso-pharynx, or from large tumors of the upper maxilla or orbit.

In large naso-pharyngeal tumors, excision of a piece for microscopical examination is dangerous on account of severe possible hemorrhage, and is useless, because, whatever its nature, the tumor will have to be removed.

In fact, the mode of approach is much more dictated by the size of the tumor and its connections than by its nature. It goes without saying that the smaller the tumor, and, therefore, the earlier the diagnosis, the better are the operative conditions (less anemia on account of less frequent hemorrhage and less interference with blood oxygenation), and the better are the chances of a complete and permanent cure. Consequently, a complete **rhinoscopic examination** is indispensable in all cases of mouth breathing, and all tumors, even benign, must be removed as early as possible.

Hypertrophied tonsils are removed by tonsillectomy, not simply partially sliced off by tonsillotomy. Adenoids are scraped away, and
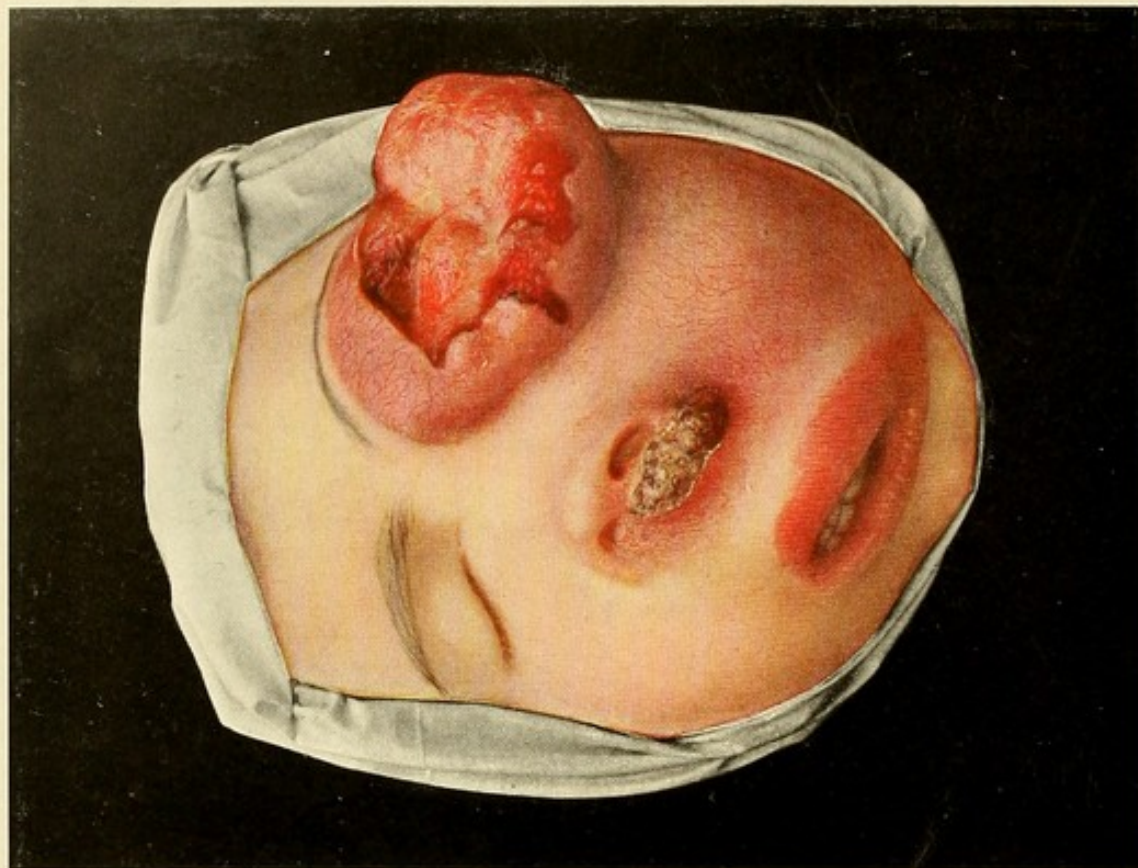
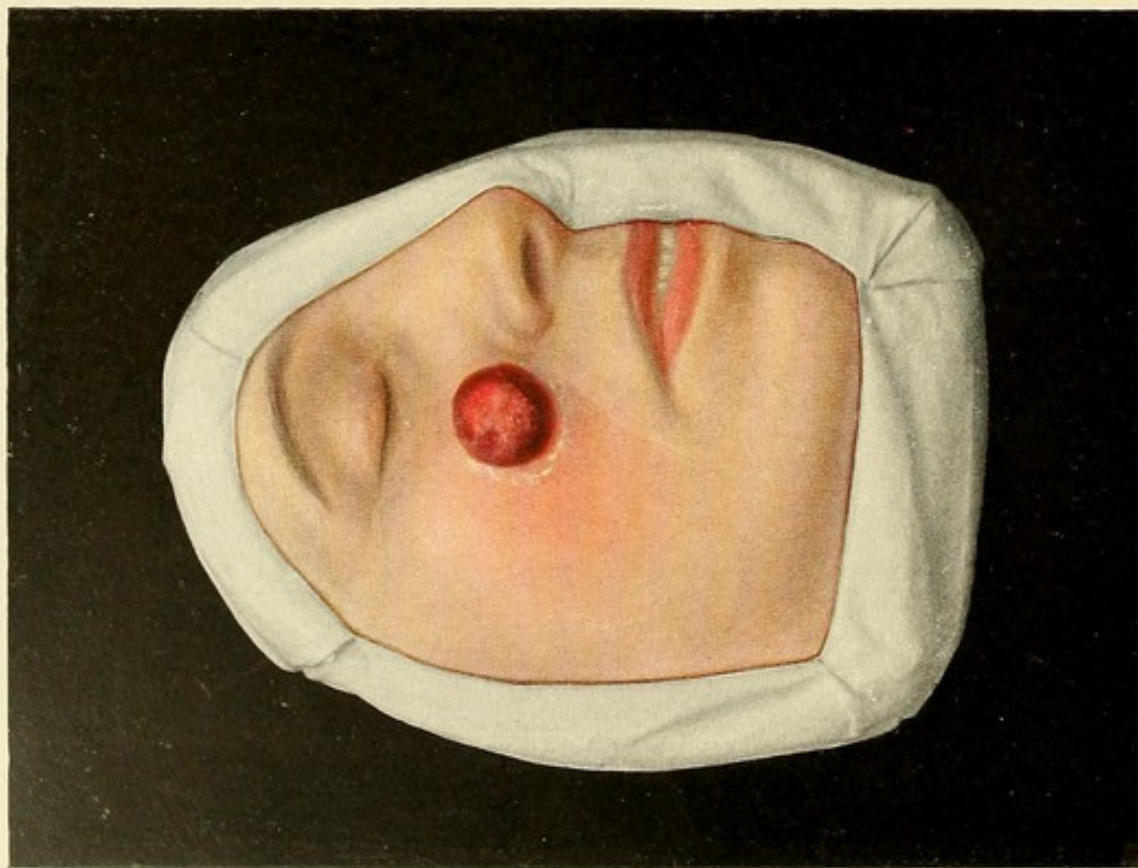Fig. 27. Sarcoma fungoides orbitae.

Fig. 26. Angiosarcoma cutis.

not left alone under the pretense that they may spontaneously disappear at puberty.

Small fibromata may be removed through the mouth after splitting of the soft and part of the hard palate, or by temporary division of the lower maxilla. The tumors should always be removed by incision into healthy tissues with the knife and not simply torn from their attachment. In extensive fibromata and in all tumors suspected of sarcoma, the naso-pharynx must be freely laid open, by temporary resection of the hard palate together with the alveolar process (*Partsch*), or by temporary resection of both upper maxillæ and upturning of the nose (*v. Bergmann*). Preliminary tracheotomy and ligation of the external carotid on one or both sides (*Kocher, König*), are expedient in these bloody operations.

Inoperable tumors may be treated by the X-rays, and, when ulcerated, disinfected with moist antiseptic dressings. In the last stages tracheotomy becomes necessary to save the patient from death by asphyxia.

Fig. 26 shows an **angio-sarcoma of the face.** (Compare with **Fig. 1,** *rodent ulcer* in the same situation, and **Fig. 31,** *multiple sarcoma of the skin.*)

Round-celled and spindle-celled sarcoma of the face is rare; angio-sarcoma is more common. In this case the tumor is pedunculated and is characterized by its concentric, spherical disposition. The base of the tumor is surrounded by a ring of epidermic scales. The surface is red, slightly uneven, not unlike that of exuberant granulations. On a whole, the tumor somewhat resembles a strawberry; it is of very soft consistence, easily bleeding at the slightest touch. The malignancy is shown by its rapid growth. It is distinguished from carcinoma by the absence of glandular enlargement.

Differential diagnosis

The tumor resembles in appearance two diseases—*frambœsia tropica* (or *yaws*) and *botriomycosis*. The initial lesion in yaws is, however, soon followed by a general eruption of similar frambœsiform growths, covered by cheesy-like scabs. The disease is seen only in certain countries. It tends to spontaneous recovery and heals very quickly under salvarsan treatment. Botriomycosis is simply infected granulation tissue. The granular growths in both yaws and botriomycosis remain superficial, while sarcoma extends into the deeper tissues.

In *mycosis fungoides* multiple growths occur which may develop into tumors resembling sarcoma. (See page 37).

Treatment

Early and free excision. In the face the defect may be repaired by a plastic operation.

Fig. 27 shows a very extensive **sarcoma involving the left half of the face** and already extending to the right half. Protruding from the orbit are fungoid masses characteristic of sarcoma (sarcoma fungoides). The soft edges have the typical reddish-brown color of sarcoma. In the places where the skin is destroyed, soft masses with a fairly regular surface protrude, which differ from the ragged, irregular ulcer of carcinoma. The whole of the tumor situated in the orbit is of soft, almost fluctuating consistency. In some parts the fungoid masses are breaking down and covered with sanious discharge. Blood crusts form on the ulcerations owing to the frequent hemorrhages in the tumor. The brown-colored skin is almost atrophied from pressure of the tumor. Sarcomatous masses protrude from both nostrils, and the whole buccal cavity and the naso-pharynx are full of tumor masses, which have caused complete destruction of the bones of the face. The tumor has also extended through the base of the skull, causing extreme drowsiness. It is no longer possible to decide whether it is a case of malignant naso-pharyngeal polypus, a retro-maxillary tumor, a maxillary tumor, or a periosteal sarcoma of the orbit. The last is the most probable, as the tumor was first observed in the orbit. Such a growth is, of course, inoperable.

Fig. 28 shows a hard, rough, movable, brownish-black **tumor of the scalp,** which rapidly developed from a pigmentary nævus in a man of 19. (Compare with **Fig. 23.**) The hardness and rapid growth reveal a malignant tumor the nature of which (melano-carcinoma or melano-sarcoma) can only be decided by microscopic examination, for carcinoma and sarcoma of the scalp are very similar. The tumor has remained small and is covered by unbroken, pigmented skin.

The malignancy of the tumor is strikingly shown by the enormous enlargement of the regional lymphatic glands. Not only the glands of the nape of the neck, but also all the glands on the right side of the neck to the supra-clavicular fossa are transformed into soft nodular tumors. The consistency of these glandular tumors is so soft
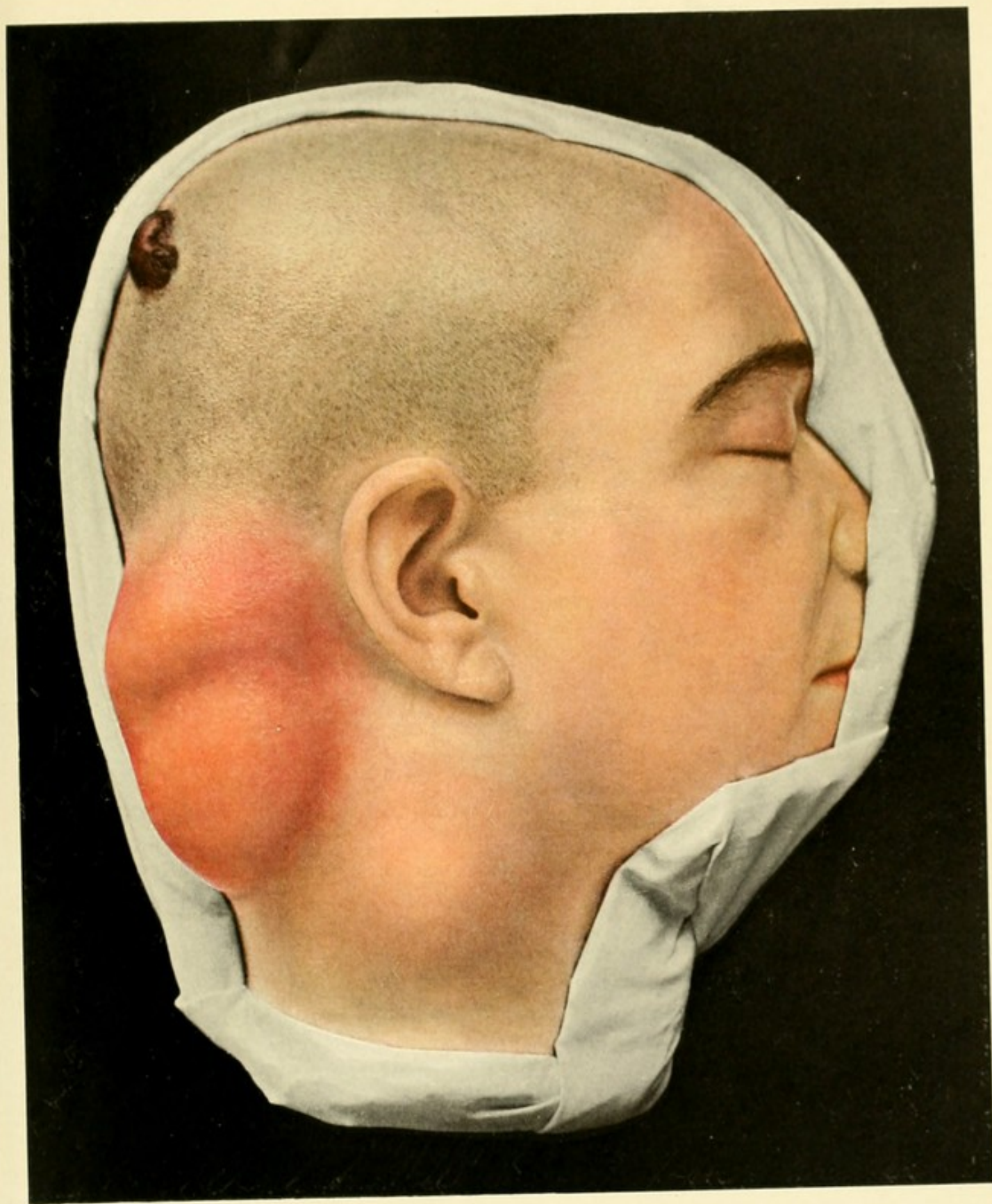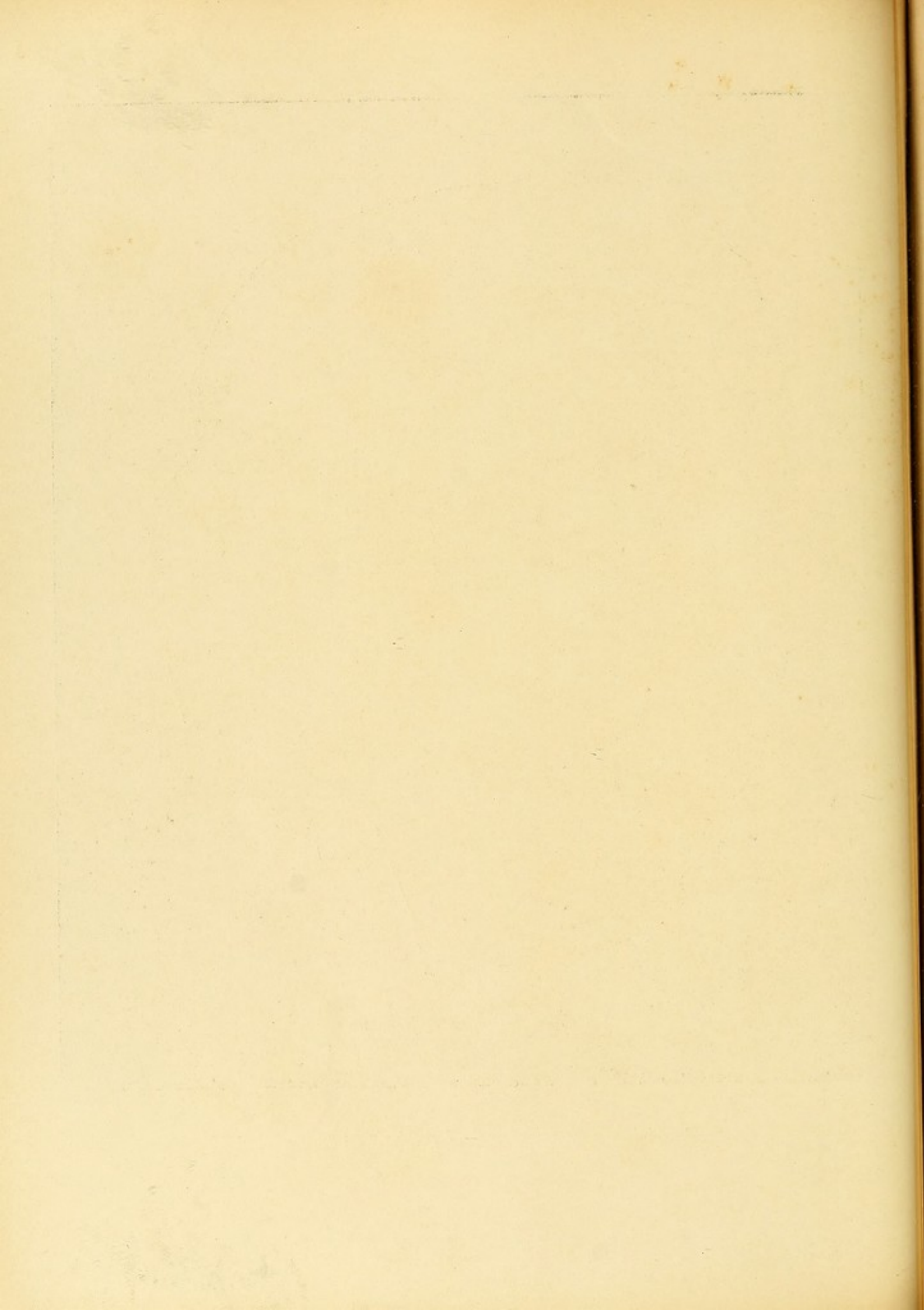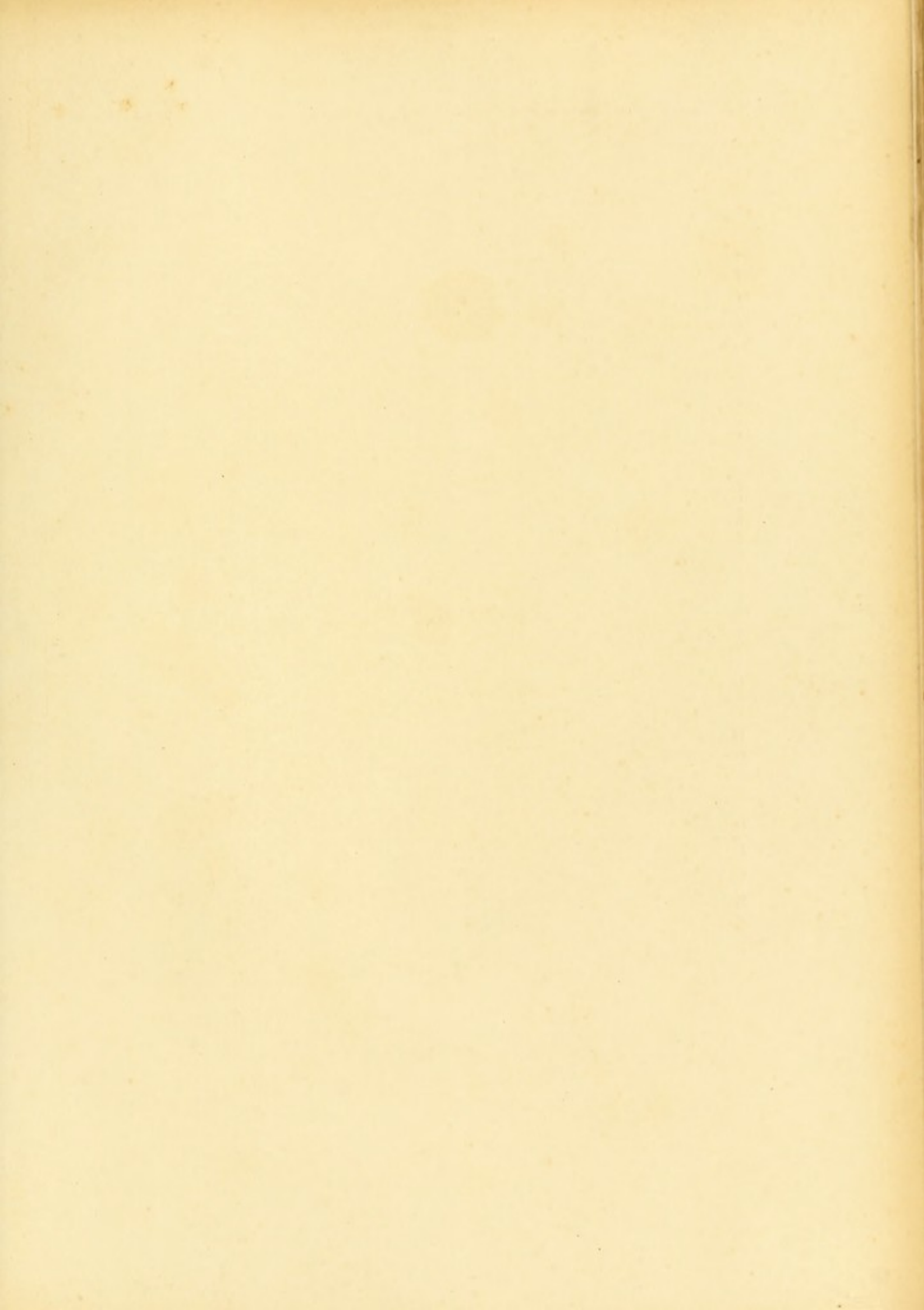
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Fig. 28. Melanosarcoma cutis — Lymphomata sarcomatosa colli.
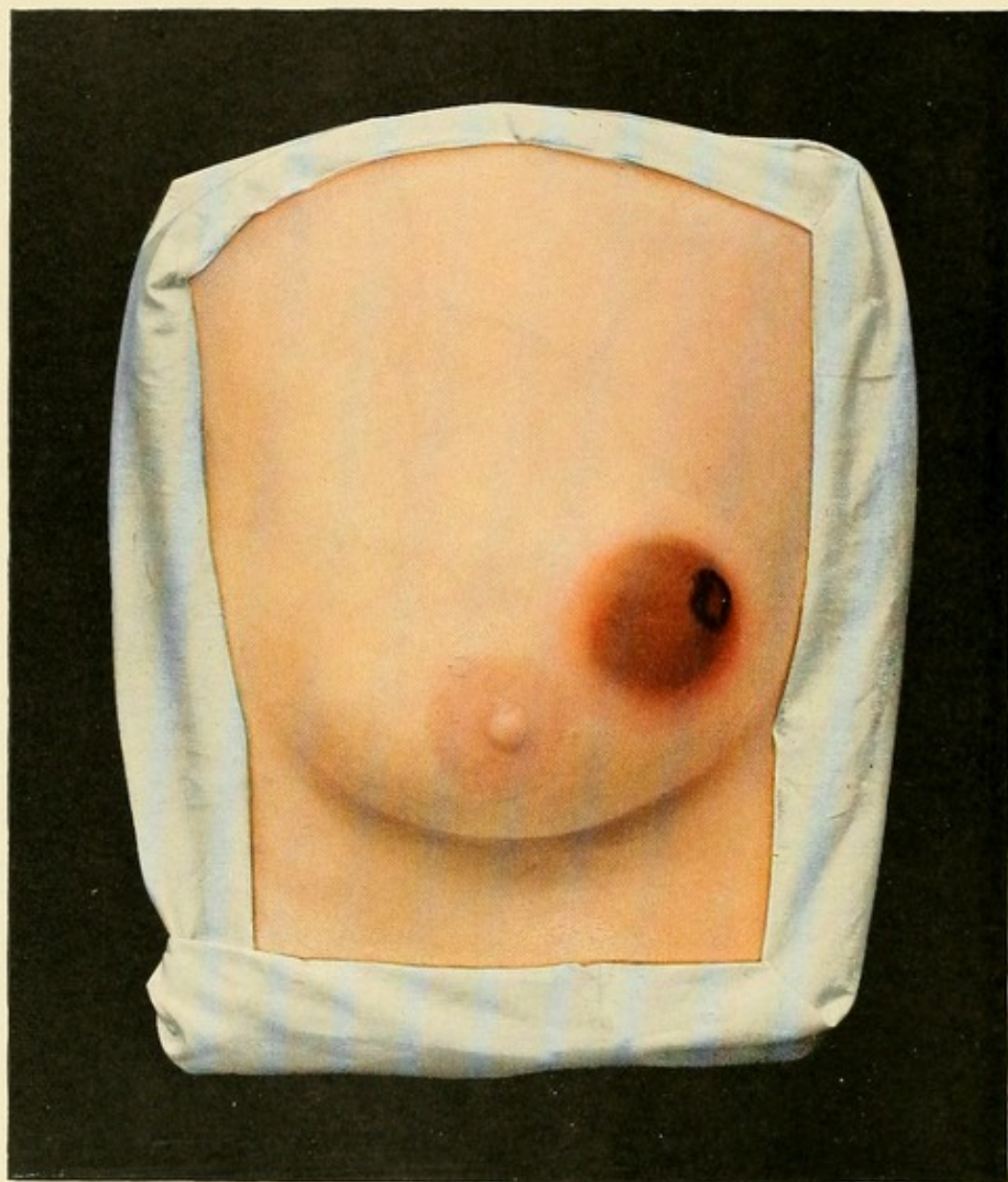
Fig. 29. Sarcoma mammae exulceratum.

as to give the sensation of fluctuation (pseudo-fluctuation), which is characteristic of rapidly growing sarcomatous metastases. The patient rapidly succumbed after the appearance of metastases in the lungs, which caused abundant pleural effusions. (Compare with **Fig. 24.**)

The glandular metastases and innumerable nodules in the lungs and heart were white in color, the pigmentation of the mother tumor often being absent in the rapidly developing metastases of melanotic tumors, as already stated on page 23.

Figs. 29 and **30** show two examples of **Sarcoma of the Breast.**
Compare with **Figs. 10-16,** *carcinoma of the breast,* and **85** and **86,** *inflammations of the breast.*

Sarcoma is much less common in the mammary gland than carcinoma (one hundred carcinomata to ten sarcomata, and half of these cysto-sarcomata, *v. Angerer*). All cell forms of sarcoma may be represented as well as mixed forms, such as myxo-, angio-, and melano-sarcoma.

They occur most often in young women. According to their composition they have different clinical signs. Spindle-celled sarcoma is of firmer consistency and of slower growth than the soft, malignant, round-celled sarcoma and melanosarcoma. Cysto-sarcoma soon leads to extensive tumors, which transform the breast into a large sac with fluid contents. The typical characteristics of sarcoma are generally present in the mammary tumors (**Fig. 29**).

Differential diagnosis

Carcinoma is distinguished by the absence of any demarcation from the mammary tissue, while sarcoma is often encapsulated. Moreover, the clinical signs of carcinoma are so characteristic (*cf.* **Figs. 10-16**) that confusion is hardly possible.

Cysts of the mamma are usually situated behind the nipple, multiple (in one or both mammæ), and not so large as true cysto-sarcoma.

Fibroadenoma, which originates in the glandular tissue and contains many connective tissue cells, is the type of the slow-growing, encapsulated, benign tumor.

The treatment is extirpation of the whole mamma as early as possible, with free exposure of the axilla. After early complete operations, local recurrence is less common than in carcinoma.

Fig. 29 shows a rapidly growing, **round-celled sarcoma** in a young girl. The tumor forms a soft, fairly circumscribed nodule in the mammary gland. That the part of the tumor lying in the mamma is considerably larger than the external appearance indicates is shown by the prominent veins. As already stated, this collateral circulation is always suggestive of sarcoma. The tumor is near the nipple, but there is no retraction of the latter. It is freely movable over the pectoralis fascia. Externally it has involved the skin, which has the usual brownish-red color of sarcoma, has become very thin and is already ulcerated in one spot, from which repeated hemorrhage has taken place. The fungoid tumors, in distinction to carcinoma, have a smooth, uniform surface and resemble exuberant granulation tissue. There were no glands to be felt in the axilla. The case was treated by extirpation of the mamma and free exposure of the axilla.

Fig. 30 represents a **cystic tumor** occurring in a young woman, which has begun to displace the whole breast. There is no alteration in the nipple. The tumor is movable over the pectoral fascia, and in several places distinctly separate from the mammary tissue. The veins are enlarged from pressure of the tumor. The tumor has already invaded the skin, which has become very thin, and in some places fluctuating. The skin is colored brownish-red and bluish-green, and shows a network of vessels. As long as the skin is intact it can never be definitely ascertained, except by exploratory puncture, whether we have to deal with actual cavities filled with fluid, or the pseudofluctuation of gelatinous or mucoid sarcoma. Rapid growth and commencing soft glandular swellings in the axilla point to the diagnosis of **cysto-sarcoma,** which was treated by extirpation of the mamma and removal of the axillary glands.

Fig. 31 shows a case of **Multiple Sarcoma of the Skin** affecting the whole of the thorax, abdomen and back.

In sarcoma of the skin, all histological types are found. It may be pigmented (melanotic sarcoma, see **Figs. 23** and **28**) or not. It may be single or multiple, primary or secondary: when secondary, it is generally multiple. Next to **true sarcoma,** we find the lesion first described by *Kaposi* as multiple pigmented hemorrhagic sarcoma, but better named **multiple hemorrhagic sarcoma,** because it is not pigmented, the color being exclusively due to hemorrhage. Next to these really sarcomatous groups exists the group of **Sarcoid** lesions,

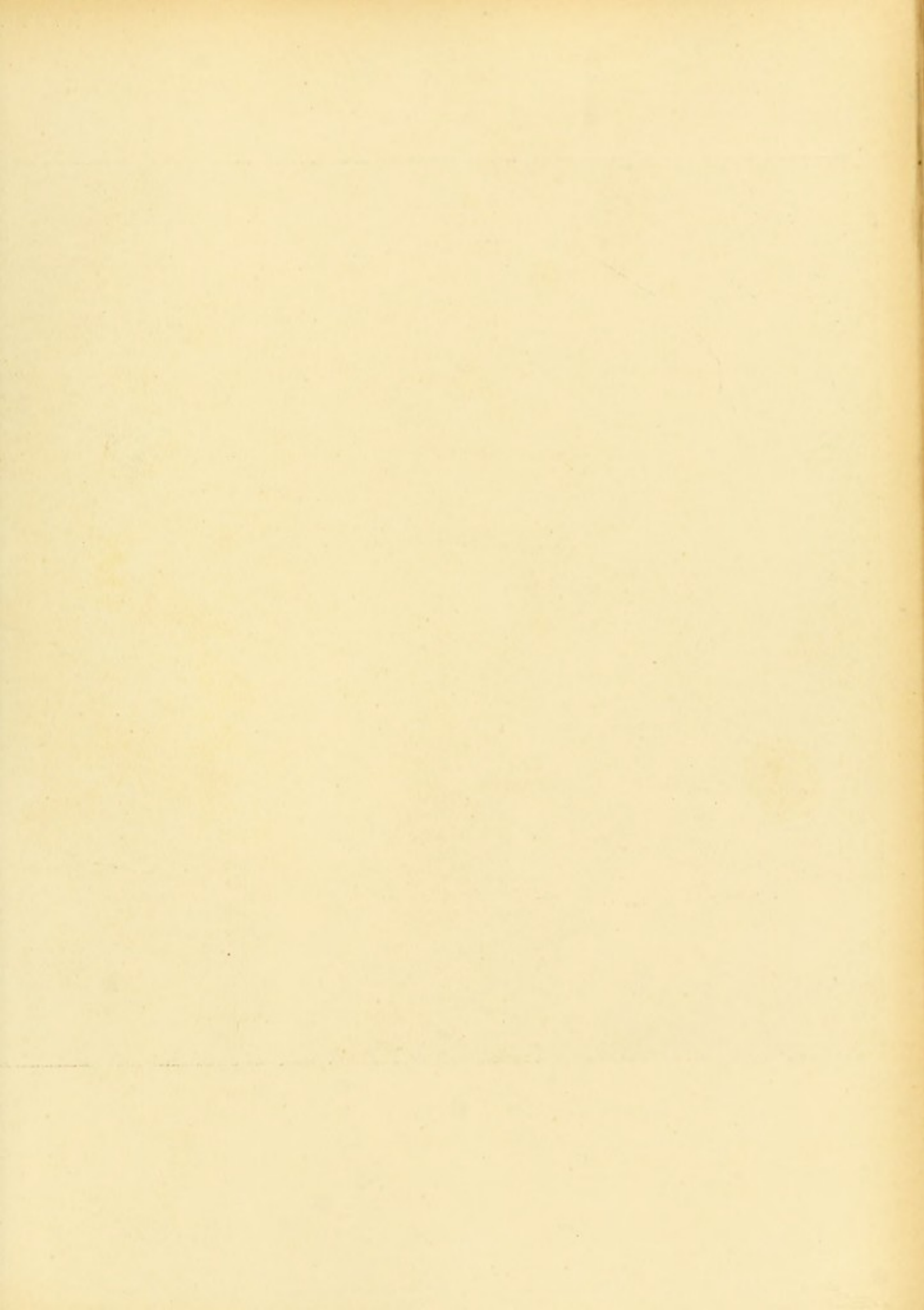Fig. 30. Sarcoma mammae cysticum.
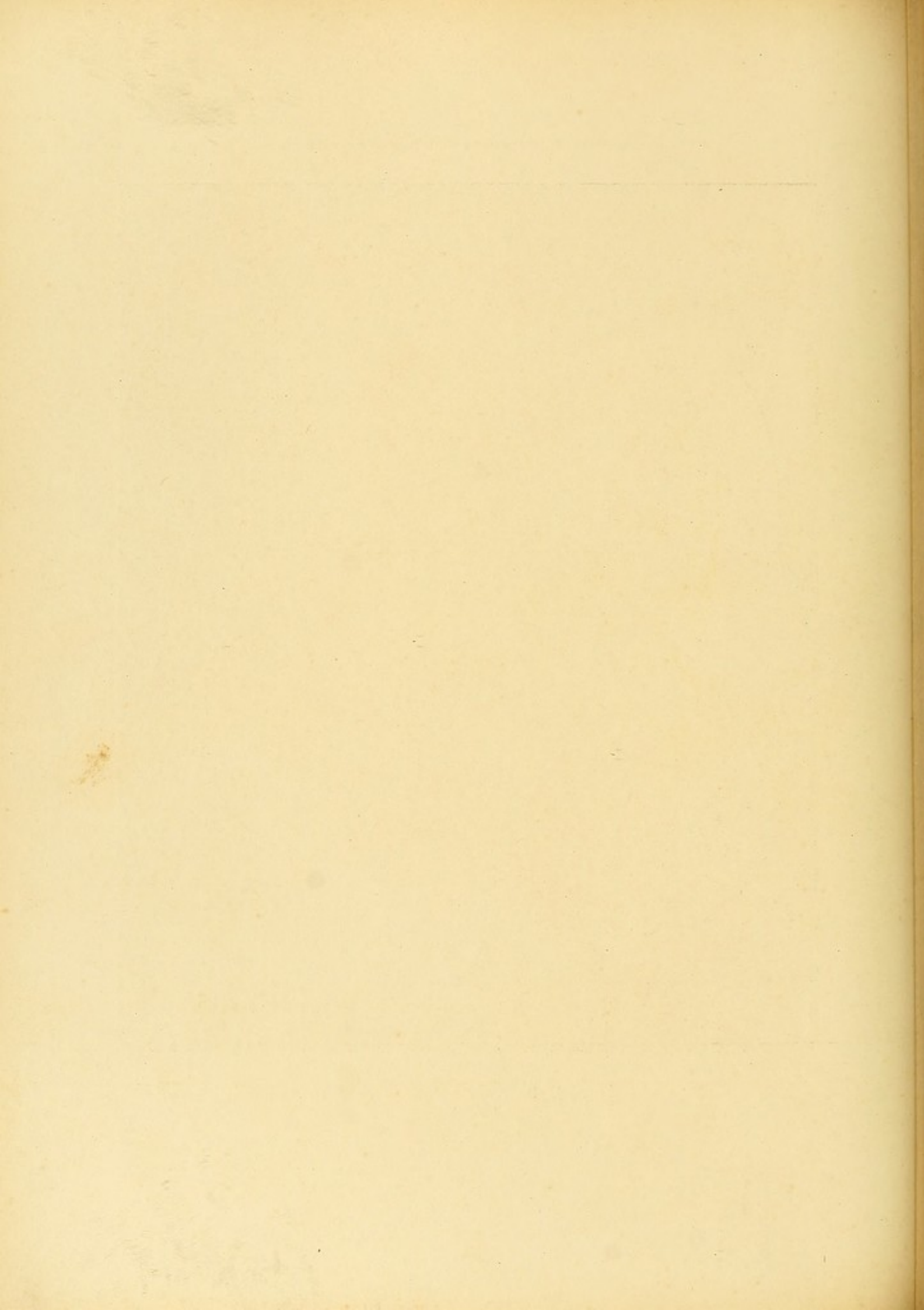
Fig. 31. Sarcoma cutis multiplex.

among which we shall mention *Bœck's Sarcoid* and *Mycosis fun-goides,* the true nature of which is, as yet, unknown.

Multiple sarcoma of the skin always appears in a characteristic form, as red spots, which soon become nodules. The nodules increase in size and become confluent, thus forming a tumor which is at first movable over the underlying tissues. Later on the skin desquamates and becomes red, bluish or livid, then browner after repeated hemorhages, and may finally ulcerate. The skin over pigmentary sarcomata is bluish black. (**Figs. 23** and **28.**)

Besides the ulceration of the nodules, spontaneous resolution is possible, complete or partial, leaving a cicatrix. The nodular tumors may in some cases remain the same size for years. The tumors are always circumscribed, and are of soft or firm consistence according to their composition. Soft nodules tend to disintegration, hard nodules to atrophy and cicatrization. The former are very malignant and soon lead to death from glandular and organic metastases; the latter, by their multiplicity, after some years cause cachexia, which with me-tastases leads to a fatal issue. This happened in the case shown in **Fig. 31,** where some of the cicatrices left by previously existing nodules may be seen. Cachexia, however, kept on progressing till death. The skin of the whole body between the nodules is often of a dirty sallow color (**Fig. 31**). Small spots and elevations on the skin point to the development of fresh sarcomatous nodules.

Multiple hemorrhagic sarcoma appears in the form described above, but first of all on the lower extremities, in the form of reddish nodules which often cause much itching. Tumor formation goes hand in hand with edematous infiltration which extends over the whole leg and prevents the patient from walking. Desquamation of the skin on the surface of the nodules occurs along with cornification of the epidermis. Cicatrices form in the skin from atrophy of the nodules. Other regions of the body are unaffected, except the peripheral parts of the upper extremity. There is no enlargement of the lymphatic glands. The disease runs a progressive course, and in spite of the spontaneous resolution of some of the tumors, finally causes death by marasmus. The average duration is about 5 years, but some cases have been followed for 20 to 30 years.

Microscopic examination shows a pure sarcoma with abundant blood-vessels, which often gives rise to organic metastases. As this form occurs often in old people, arteriosclerosis may, perhaps, play a part in the origin and course of the disease.

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Primary multiple sarcomata must not be mistaken for secondary sarcomatous growths in connection with a primary cutaneous sarcoma or a sarcoma of the internal organs. So a careful search must always be made for a possible primary tumor. The tumors of **mycosis fungoides** are more likely to be mistaken for sarcoma, as they also develop from red, uneven spots, and form granulation tumors of a brownish-red color which in the later stages tend to ulceration and cachexia; but mycosis fungoides is of much slower growth than sarcoma and there generally coexist in different parts of the body of the same patient the three different stages of mycosis fungoides, namely, the premycotic itching patch, the infiltration and, finally, the fungating lesion. The association of these three types of lesion is characteristic. **Syphilitic and tuberculous granulomata** can hardly be taken for sarcoma on careful examination.

Carcinoma of the skin is clinically so different from multiple sarcoma that no hesitation can arise, except for the melanotic form, which can be distinguished only by microscopical examination.

Preventive treatment of multiple sarcoma consists in the removal of all nævi which begin to take on rapid growth. In already existing multiple pigmentary sarcoma excision is generally useless, and should only be performed when the tumors are very few in number if not single, and the blood-vessels free from melanin. After excision of multiple sarcomata, especially melanosarcomata, death often follows from rapid dissemination and organic metastases. Hence the X-rays, large doses or arsenic (internally or subcutaneously) have been employed for multiple cutaneous sarcoma, in the same way as for mycosis fungoides. They have given temporary improvement. A permanent cure, however, is not to be expected and the prognosis of these multiple sarcomata is always bad.

Fig. 32 shows a **peripheral sarcoma of the upper end of the humerus** in a young individual.

The soft tumor has extended under the skin, in which the brown coloring and *extensive network of dilated veins* are very marked. The lower borders of the fusiform tumor are irregular and send processes here and there into the muscles. The tumor has destroyed the head of the humerus and has broken through into the joint, in which there is an effusion. The function of the joint and upper arm is destroyed. The supra-clavicular glands are enlarged. Posteriorly
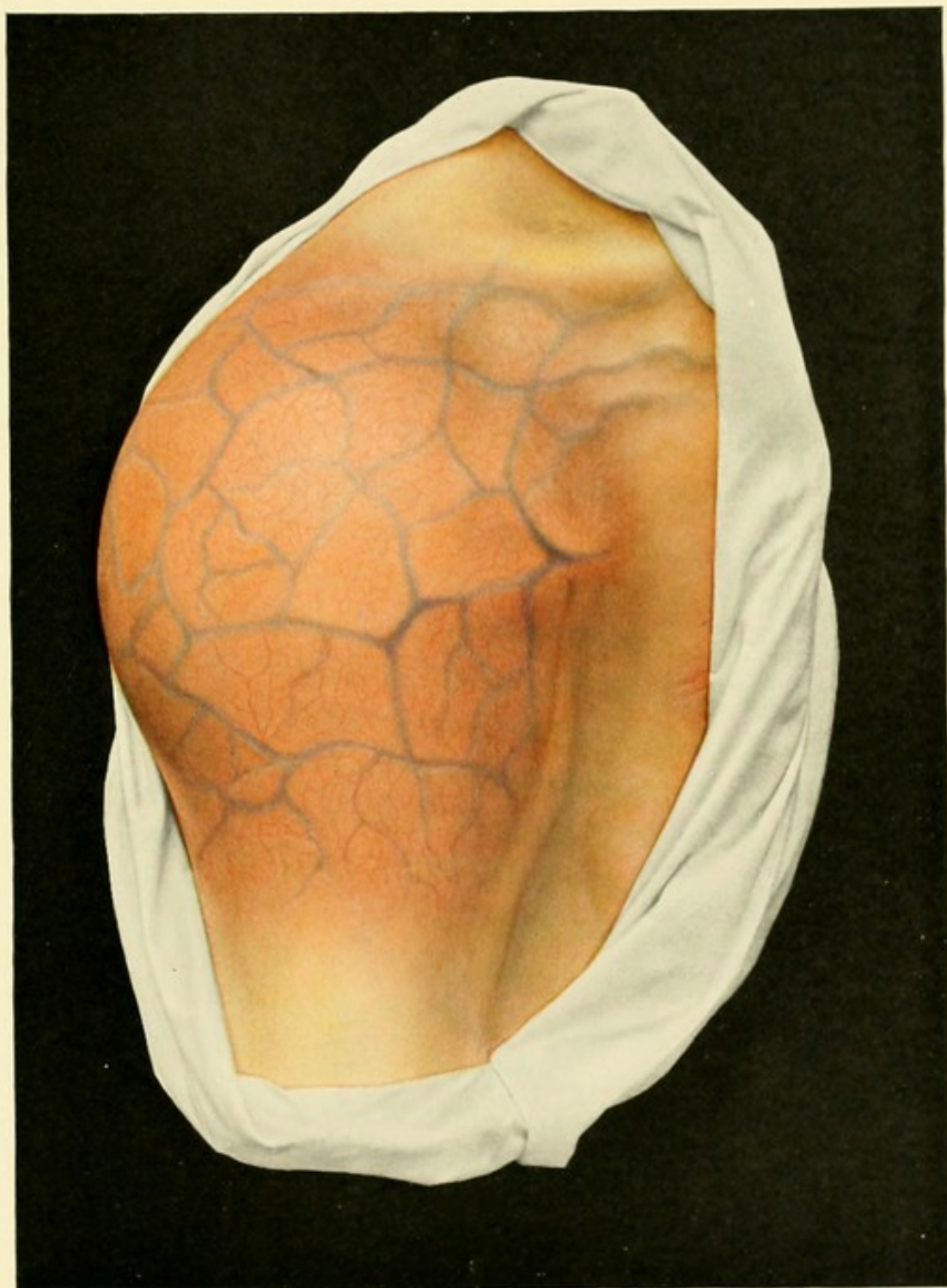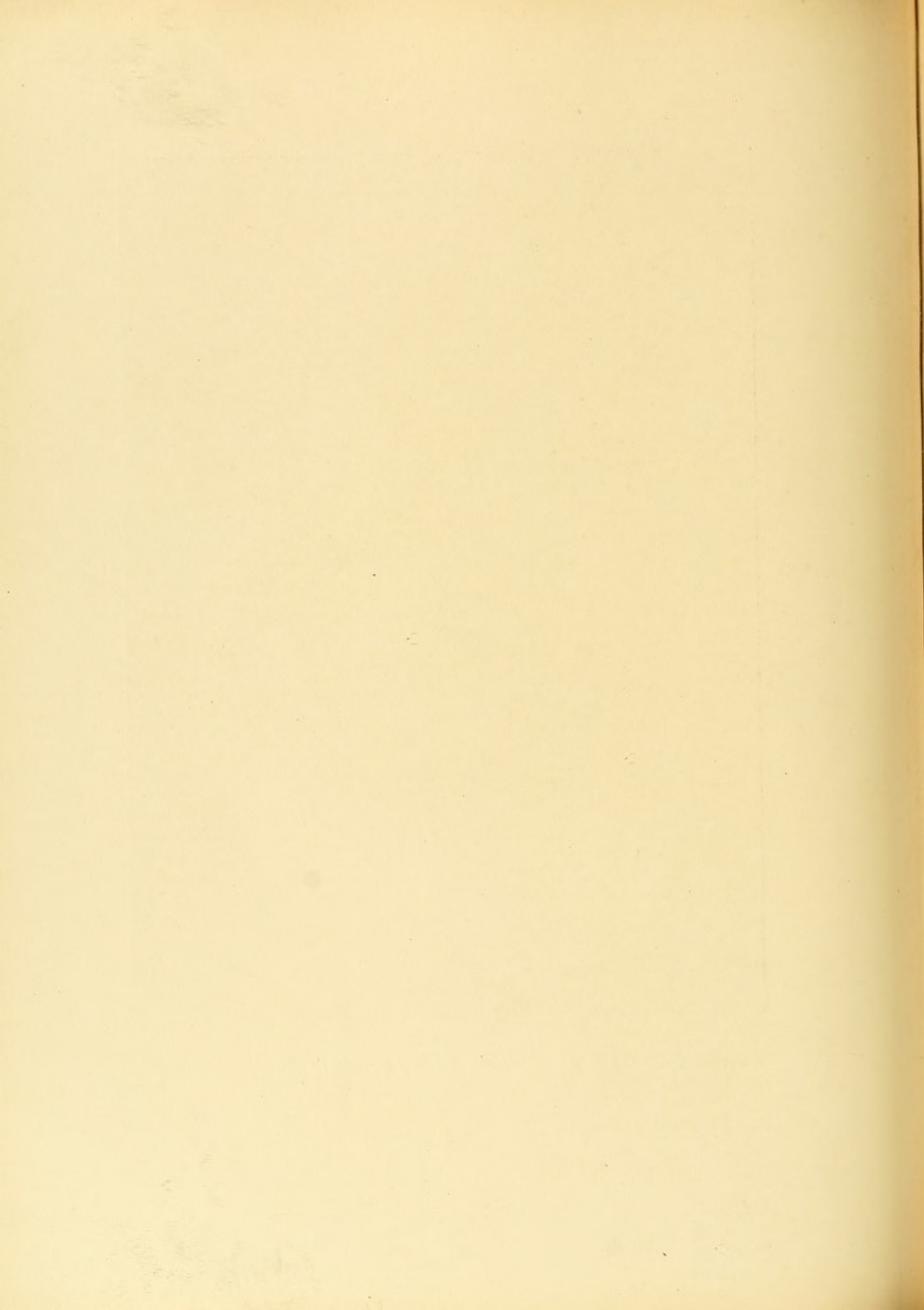
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Fig. 32. Sarcoma humeri periphericum.

the tumor has extended to the scapula region. The X-rays show complete destruction of the upper part of the humerus.

Bone sarcoma is one of the most important, and frequent varieties of sarcoma.

Osteo-sarcomata are best divided into peripheral and central; the latter may arise from the cortical, spongy or medullary portions. Division into periosteal and myelogenous tumors is clinically impossible, and the word myelogenous may be replaced by osteal. Tumors which appear clinically to be periosteal often arise from the superficial layers of the cortex. The X-rays enable easily to divide them into peripheral and central tumors; this leaves open the possible origin of the sarcoma from any part of the bone, and this can only be conclusively settled by section of the bone after removal. This classification is all the more rational because sections of specimens which were clinically regarded as periosteal sarcomata show that these arose from small foci in the medullary cavity. Periosteal tumors may extend into the medullary cavity and so simulate osteal tumors. In extensive tumors the origin of the tumor from any definite part of the bone cannot as a rule be established.

Both forms have special seats of predilection: in the long bones, the neighborhood of the epiphyses *e.g.* the upper end of the humerus (**Fig. 32**), the lower end of the femur, especially the internal condyle, the head of the tibia, the lower end of the radius; the flat bones, especially the scapula and bones of the skull. Both forms also grow in a globular type involving the whole circumference of the bone and finally its whole thickness. They appear at puberty and during the whole period of growth, generally in young and robust individuals.

Both kinds soon break through their own capsule and that of the bones and then extend sometimes into the neighboring joints, often into the muscles, especially the muscular insertions into the bones, and into the veins, eventually forming enormous tumors which break through the skin and protrude as fungoid masses. The superficially situated tumors have a tendency to frequent hemorrhage and destructive inflammation.

Microscopically, *spindle cells* are often found in *peripheral sarcoma,* and *giant cells* in *central sarcoma.* The other forms of sarcoma cells are also present.

The X-rays, in peripheral sarcoma, show little change in the cortex. In central tumors, especially those arising from the medullary cavity, they often show spherical transparent spaces in the interior,

while the cortex is very thin and excavated—forming a shell—in the same way as in bone cysts, osteomyelitic abscesses, isolated tuberculosis and gumma.

In the early stages the diagnosis of osteo-sarcoma is difficult. The peripheral tumors are naturally easier to recognize, as they present a rapidly growing mass, firmly attached to the bone, with irregular boundaries toward the muscles. Rheumatic pains and effusion into the joints frequently occur when the tumors are situated near the joints. The nearer the sarcoma comes to the skin the easier it is to palpate the superficial tumor masses, which infiltrate the soft tissues, and consist of cells only without bony infiltration. *Swelling of the cutaneous veins* occurs early from pressure of the tumor on the vessels, while the skin becomes reddish brown, thin and almost transparent, especially when the tumor is adherent to it. These two features are clearly visible in **Fig. 32.**

Slow-growing central sarcomata can at first be diagnosed only by the X-rays, later on they present themselves as hard spheroidal swellings like billiard balls. The more they extend and approach the skin, the thinner becomes their bony shell, which finally gives the sensation of parchment crepitation, first described by *Dupuytren*. Central tumors are often first diagnosed by the occurrence of spontaneous fracture. Extensive forms, which assume a more spindle-celled formation are easy to recognize. Through growth of the tumor into the joints and muscles, typical functional derangements are produced, and separation of the epiphyses. Metastases in the lungs develop early. Disintegration of the tumor cells gives rise to *fever,* especially in rapidly growing, small, round-celled sarcomas.

Parosteal sarcoma is easily mistaken for peripheral sarcoma, and is often impossible to distinguish by the X-rays. It is often of very soft consistence, and was formerly called encephaloid.

Chondrosarcoma only occurs in the neighborhood of the joints and forms irregular nodular tumors (**Fig. 34**).

Sarcoma situated near the large vessels and pulsating with them may be mistaken for *aneurism,* but the X-rays will assist the diagnosis. Central sarcomata have been wrongly considered as aneurism of the bone, owing to their vascularity and their reddish-brown color on section, which is due to frequent hemorrhages.

Myelomata are multiple, occur chiefly in the vertebræ, and albumose is found in the urine.

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Metastatic carcinoma, which occurs especially in the neck of the femur after mammary carcinoma in women, and in the head of the humerus after carcinoma of the thyroid gland (*v. Eiselsberg*), must be diagnosed by the primary growth.

Osteo-sarcoma may possibly be mistaken for *arthritis, rheumatism, osteomyelitis, syphilitic and tuberculous processes;* but in most cases the diagnosis can be made by careful clinical analysis, by the history of the case, by the X-rays, by anti-syphilitic treatment, and in osteomyelitis by search for hemolysin (*Bruck, Michaelis, Schultze*). Uninterrupted increase of a diffuse growth should always raise the suspicion of malignant tumor. In doubtful cases an exploratory incision must be made.

In all cases the prognosis is very bad. The harder forms of sarcoma (spindle-celled and giant-celled) sometimes have a better prognosis, particularly the giant-celled, which *Bloodgood* considers as non-malignant. Soft, round-celled sarcomata are the most malignant on account of their rapid growth and early metastases.

Treatment

The earlier operative treatment is undertaken, the more likely is a radical cure.

Small, central sarcomata can be removed by the chisel, and the medullary cavity scraped, particularly in the giant-celled variety. Larger circumscribed tumors still confined to the bone can be removed by free resection of bone, very wide of the limits of the tumor. The defect can be repaired by bone grafting (auto- or hetero-plastic). But, except for giant-celled sarcoma, conservative operations are not advisable.

If the sarcoma has already invaded the muscles, high amputation or, preferably, exarticulation, must be performed.

Inoperable sarcoma is to be treated according to the rules for inoperable tumors. (See page 4.)

In the case represented in **Fig. 32,** as there was no evidence of organic metastases, the arm and shoulder girdle (scapula and outer half of the clavicle) were removed after section through the middle third of the clavicle and ligation of the subclavian artery and vein. (*Interscapulothoracic amputation.*) The axillary and supra-clavicular lymph glands were also removed.

On section, the whole of the upper portion of the humerus was found to be transformed into a large tumor, the central parts of which were hard from bony infiltration, while the periphery was

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soft and fungoid. The tumor was a *round-celled sarcoma,* but it was too extensive to decide from which part of the bone it originated.

Fig. 33 shows a rapidly growing, recurrent, ulcerated **sarcoma of the fascia of the arm.** The younger nodules are covered by livid skin, which is intact in some parts and thin in others. In other parts there are white cicatrices left by former operations. The X-rays show that the sarcoma has extended to the bone. Owing to the growth having broken into the elbow joint, this is immobilized in a right-angle position. There are some small, soft, enlarged glands in the axilla.

Fascia and sheaths of blood-vessels are often the starting point of sarcoma; not only of pure round-celled and spindle-celled sarcoma, but more often of mixed forms—myxosarcoma and fibrosarcoma. Fibrosarcoma is characterized by its firm consistence and slow growth; it is frequently circumscribed and partly encapsulated. Myxosarcomata are characterized by their softness and rapid growth without encapsulation. Pure sarcomata appear as soft, many-celled, rapidly growing tumors, or in a harder form which is of slower growth and not so malignant.

In the early stages of fascial sarcoma (fascia of the arm, thigh, abdomen) we find small tumors fixed to the fascia, but movable over subjacent tissues and under the skin. The skin is soon involved and becomes tightly stretched over the tumor and pigmented, and finally the tumor breaks through it. At the same time the muscles, and eventually the whole region, are infiltrated with tumor substance (bones, joints, peritoneal cavity). The chief growth, however, takes place on the external surface in the form of nodular fungoid vegetations, which exhibit all the characteristics of sarcoma. They are of soft consistency, both in the center and at the periphery; the surface is much smoother than in carcinoma, bleeds easily on account of its vascularity, and is covered with sanious discharge. Nodules succeed one another till an enormous cauliflower growth is formed (**Fig. 33**).

Ulceration of the tumor is followed by regional glandular metastases, organic metastases, fever and severe anemia.

Differential Diagnosis

These rapidly growing malignant tumors are so typical in their situation and development that it is only on the scalp that they can be mistaken for ulcerating carcinoma. Sarcoma of the scalp often has hard borders with deep fissures as in carcinoma, and also gives rise to early glandular enlargement.

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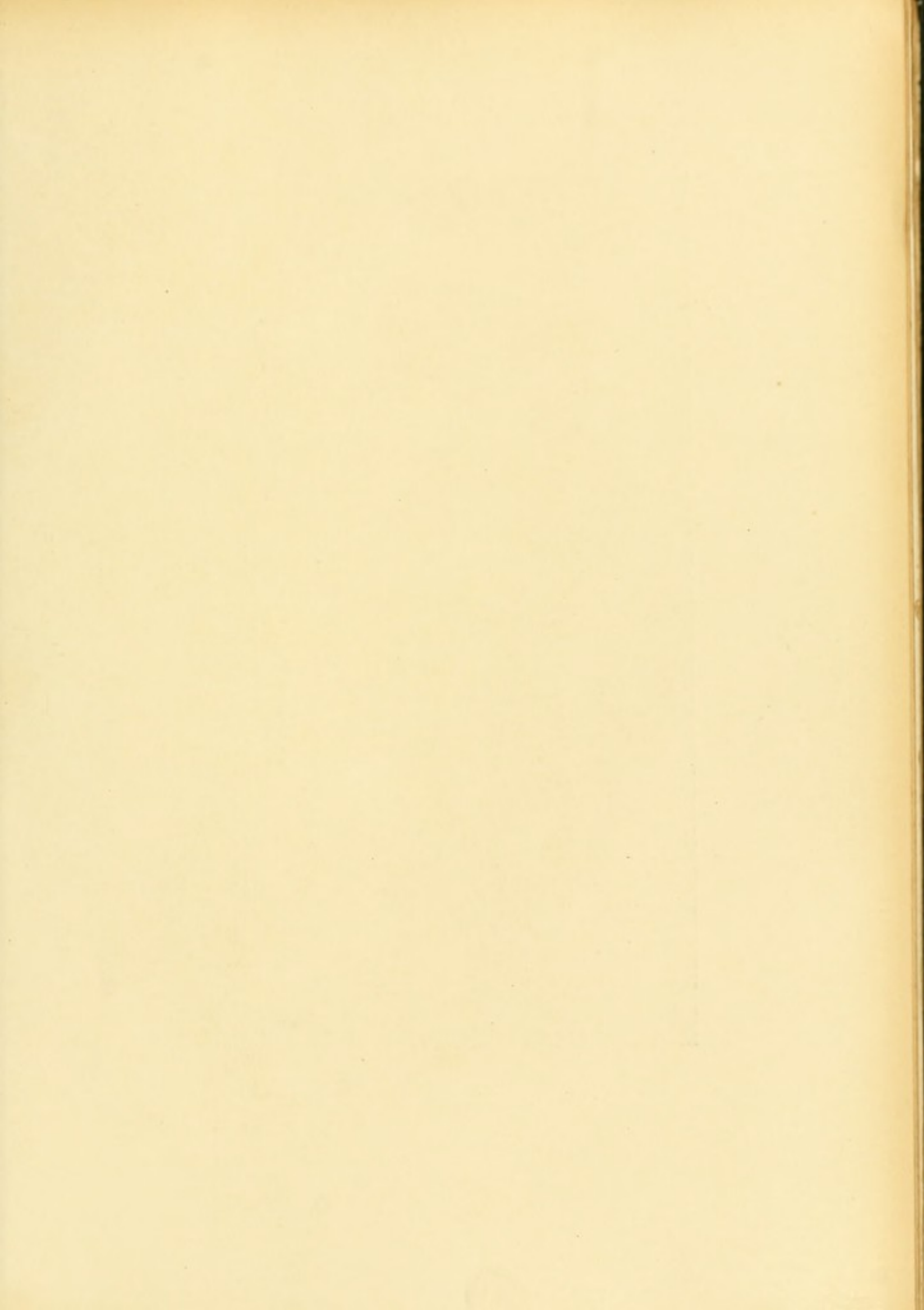Fig. 33. Sarcoma fasciae brachii exulceratum.

Tab. XXVI.
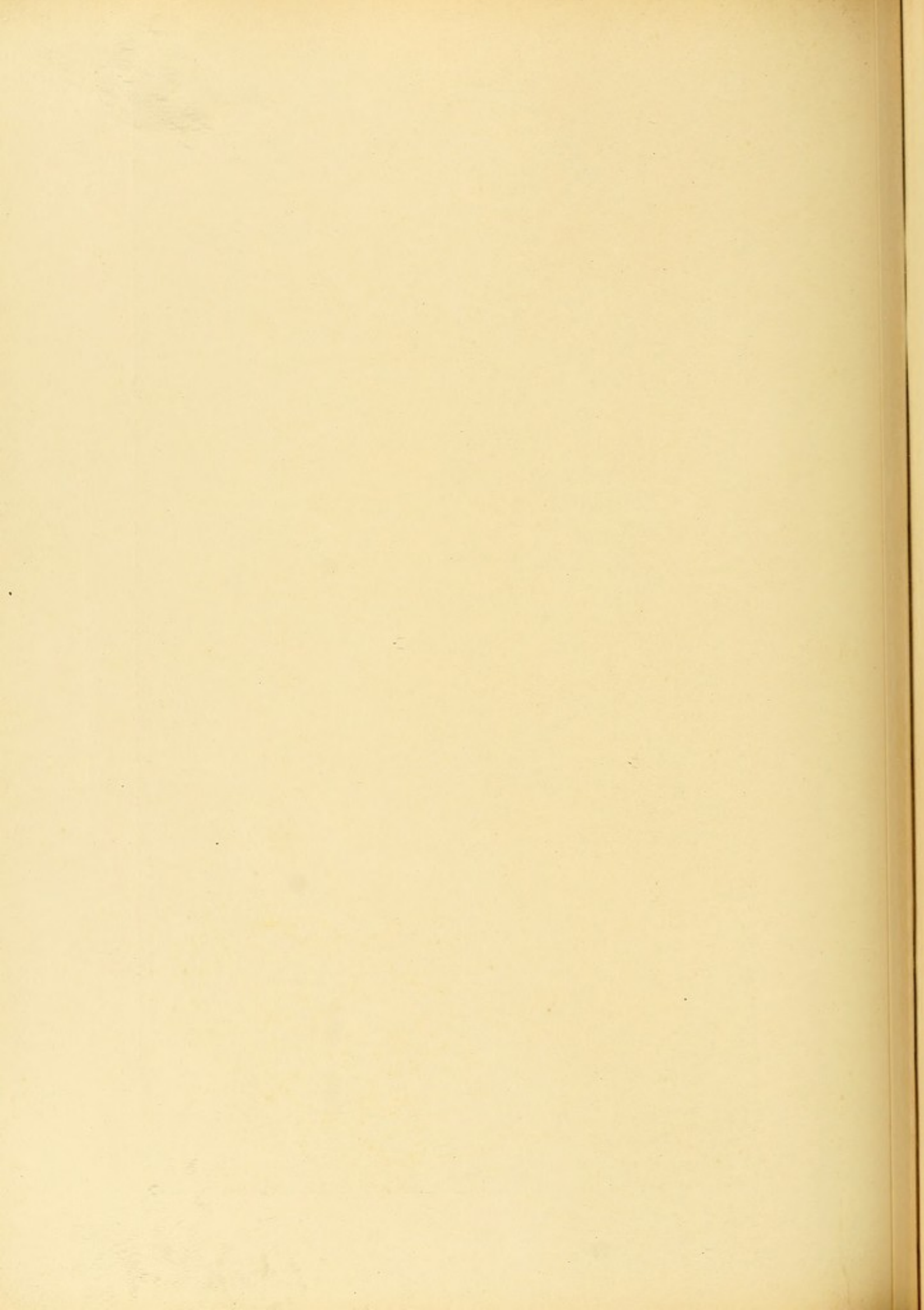
Fig. 34. Chondromyxosarcoma — Exostoses malignae.

Small, slow-growing sarcomata can be removed by free excision, but local recurrence is frequent. In extensive, and especially in ulcerated, tumors of the extremities amputation is indicated. Tumors which arise in the abdominal fascia often become inoperable owing to extension to the peritoneal cavity. In the case shown in **Fig. 32,** the arm was amputated and the axilla cleaned of lymphatic glands.

Fig. 34 shows a nodular tumor of almost bony hardness arising from the tibia. Some portions of the tumor, however, are soft. The tumor has pushed forward under the skin, which has become thin and livid, and is ulcerated in some places through which the tumor is beginning to discharge. The movements of the knee joint are very limited. No glandular or organic metastases were found. This tumor, when removed, was found to be a **chondromyxosarcoma.**

Chondrosarcomata are situated on or near joints. Most frequently they arise from the head of the tibia or the upper end of the humerus, also from the lower end of the radius. They may also originate from previous chondromata of the phalanges, metacarpal and metatarsal bones. They generally form large, nodular, *hard* tumors consisting of hyaline cartilage, osseous, mucoid and sarcomatous tissue and contain cystic cavities due to softening and hemorrhage. They then resemble in appearance benign, cystic chondrofibroma.

They often form rapidly growing tumors which destroy the bones and joints and give rise to sarcomatous metastases containing no cartilage. Their prognosis is, therefore, very bad. In young individuals they cause disturbance in growth (shortening, etc.). Spontaneous fractures are frequent in the forms which show an abundant development of sarcomatous tissue and much cystic degeneration. Chondrosarcoma may also develop in chondroma arising from cartilaginous exostoses, which are due to arrested development of the skeleton and disturbances in growth.

These tumors are so typical that they cannot be mistaken for other growths when they have attained a certain size. On the contrary, when small, their nature may be suspected, but rarely decisively affirmed, as they then much resemble *exostoses.*

All exostoses and chondromata which show signs of rapid growth must be removed. In sarcomatous tumors, removal of the growth may be attempted, if the neighboring parts are not too involved. In large tumors, amputation, and chiefly exarticulation, are necessary.

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Fig. 35 shows a soft tumor, the size of a cherry, arising from the alveolar border of the first right bicuspid tooth, in a young woman, and which has grown rapidly during pregnancy. On the surface is a pin point ulceration from which frequent hemorrhage has occurred.

This tumor is an **epulis,** which name has been given to sessile or pedunculated fibrosarcomata with numerous spindle and giant cells, arising from the periosteum or alveolar connective tissue of the upper and lower jaw. They are hard or soft according to their histological constitution, with a smooth surface covered by mucous membrane, of rounded form and the size of a walnut. In women they grow rapidly during pregnancy. They seldom ulcerate. In children and young people they occur equally in both sexes. They often arise in the spaces between the teeth, and then bear the imprint of the neighboring teeth on their surface. They sometimes develop from the lateral surface of the alveolus and then grow over the teeth, usually the molars, which they may loosen. They are very vascular and bleed easily, but cause no other trouble.

These tumors, although sarcomatous, have usually a good prognosis, for their growth remains circumscribed, rarely involves the bone and gives rise to no glandular or organic metastases. They only assume a malignant character by their frequent recurrence after incomplete operations.

Differ   Diagnosis and Treatment

Polypi of the gums (*gum boils*) arising from alveolar fistula and bad teeth do not attain the size of epulis. The flaccid fibromata of the gum seen in leontiasis ossea do not form globular tumors, and are only slightly vascular.

Carcinoma occurs at a later age, seldom arises from the alveolar border, and can easily be recognized by its hard borders, fissures, and metastases.

Epulis should never be simply snipped off with scissors. The adjoining part of alveolar border should always be chiselled away. This was done in the case represented in **Fig. 35.** After such a removal, recurrence is rare: on the contrary, it is frequent after simple excision of the growth without bone removal.
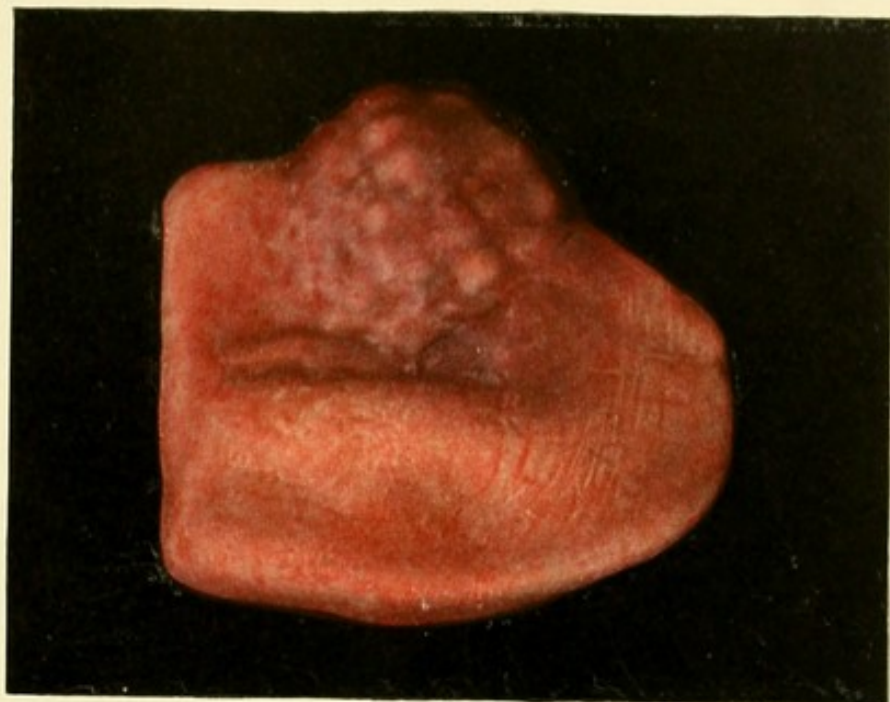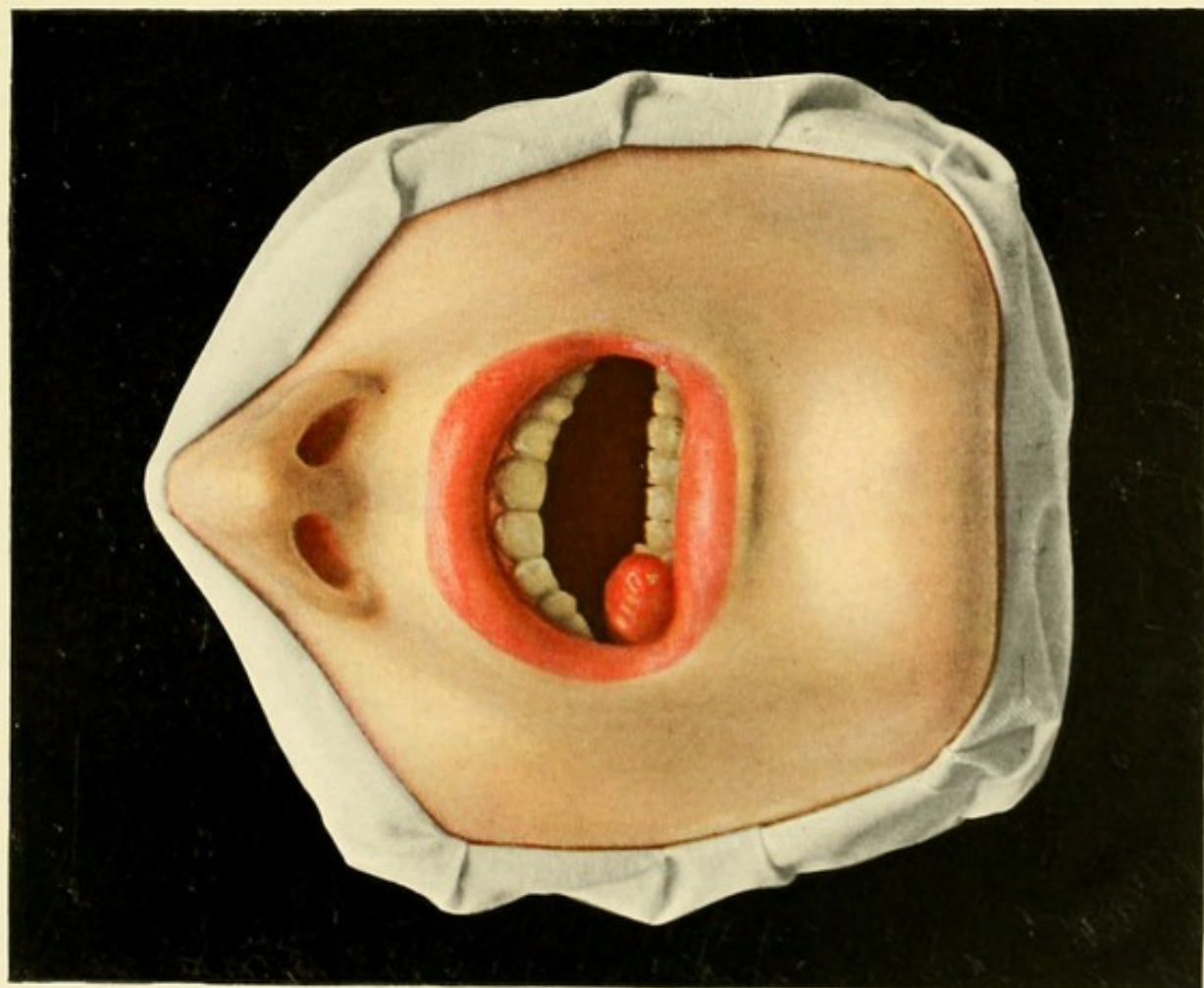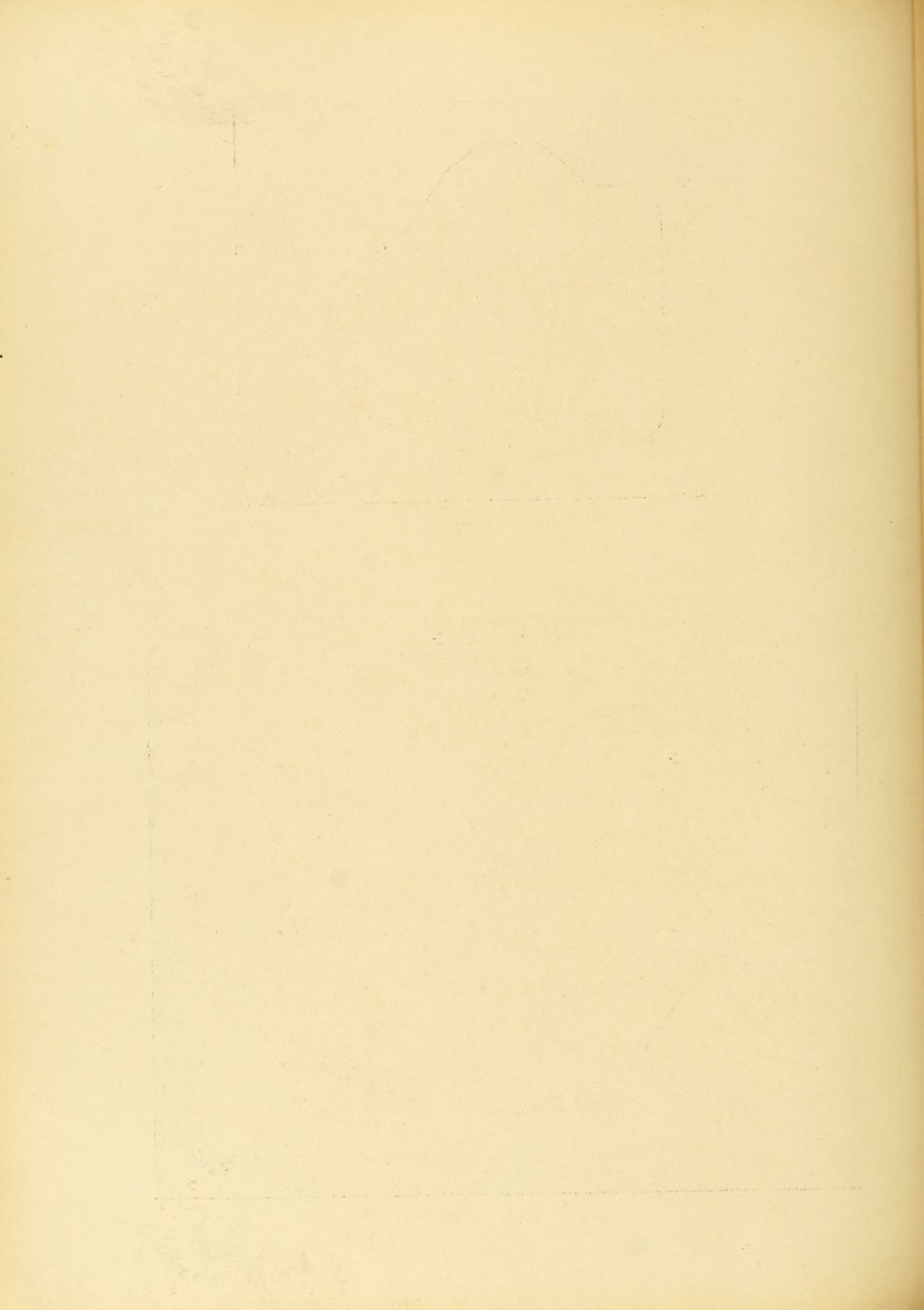
Fig. 36. Cavernoma linguae.

Fig. 35. Sarcoma gigantocellulare — Epulis.

MIXED AND BENIGN TUMORS

Fig. 36[1] shows an encapsulated *cavernous hemangioma of the tongue* developed, after puberty, from a previous congenital simple hemangioma, a slightly raised red spot which often remains unnoticed. This is the most common sequence. Such a cavernous angioma may also occur as a congenital tumor which becomes fully developed in adolescence or sometimes later, and extends more deeply than simple hemangioma into the mucous membrane and sub-mucous tissue. The tumor consists of new blood-vessels, especially capillaries, and cavities lined by endothelium and filled with blood.

Cavernomata, as a rule, present themselves as bluish, glistening tumors with several small nodular projections on the surface. The mucous membrane in the region of the tumor is so thin that a dark fluid mass appears to be seen through it. Apart from this characteristic appearance, the softness of the tumor, and the fact that it can be emptied by pressure and made tense by bending the head are noteworthy features. The growth consists of cavernous tissue, such as is found normally in the corpora cavernosa penis, and on this account the name erectile tumor has been applied to it. Besides the superficial growth there is also a deeper one into the mucous membrane, so that the whole tongue, the floor of the mouth, the soft palate, the lips and the cheeks may be involved. Eventually the tumor may implicate the whole side of the face and extend through the orbit to the brain. In other cases the tumors are encapsulated. Sometimes there are multiple encapsulated cavernomata lying close together, but without any direct connection. Tumors which, starting from the buccal mucous membrane, come to bulge under the skin of the face, give rise to thinning and a bluish glistening coloration of the latter. Apart from the deformity large cavernomata are dangerous, as they may rupture and cause profuse and sometimes fatal hemorrhage (as often occurs in cavernomata of internal organs, alimentary canal and liver). Sometimes ulceration occurs at the points of rupture, which may cause general septic infection, and in the tongue acute glossitis and edema of the glottis.

[1]For other lesions of the tongue, see **Figs. 6, 7, 8, 9, 118, 119, 120.**

Cavernous lymphangioma (see **Fig. 145**) is composed of larger protuberances and has a greenish surface. Moreover, lymphangioma, though *diminished by pressure,* remains independent of the circulation and is not increased by bending the head, stooping or coughing. As the result of inflammatory changes, hard nodules form in these tumors, which are disseminated in the soft parts. *Sarcoma of the tongue* is rare and can generally be recognized by its smooth surface and rapid growth. *Retention cysts* of the mucous membrane of the tongue are smaller, circumscribed, and have a uniform surface. On the other hand, they are also covered by thin, bluish, glistening mucous membrane.

Treatment

Cavernous hemangiomata can be extirpated if they are encapsulated. Diffuse forms may be incised and scraped, after which the big vessels are ligated, and the inside of the cavity treated by boiling water or the cautery and packed. If there is a recurrence, the procedure must be repeated.

Inoperable tumors are best treated with injections of alcohol, or with *Payr's* magnesium. Both methods aim at thrombosis, after which shrinking of the tumor takes place. Injections must be made deeply under the mucous membrane to avoid necrosis, and are not absolutely devoid of danger. For the treatment of simple hemangioma, see p. 104.

Fig. 37[1] shows the right breast of a woman (at the menopause) much more projecting than the left. The upper half of the right breast is involved in a tumor, the irregular surface of which can be recognized by the bulging of the skin. The skin is thin and reddened. The tumor, which was at first remote from the nipple in the inner and upper quadrant of the breast, has grown toward the nipple without causing retraction. It is completely encapsulated, freely movable, and of moderately hard consistency. It was removed through a radial incision, together with the adjacent mammary tissue, and microscopical examination confirmed the clinical diagnosis of **cystic fibro-adenoma** already established by the above-mentioned signs.

Real adenoma and *pure fibroma* are rare in the breast. Myxoma, angioma, chondroma and mixed tumors are very rare. **Fibro-ade-**

[1]Compare with **Figs. 10-16** (carcinoma of the breast), **29** and **30** (sarcoma), **85-86** (inflammations).
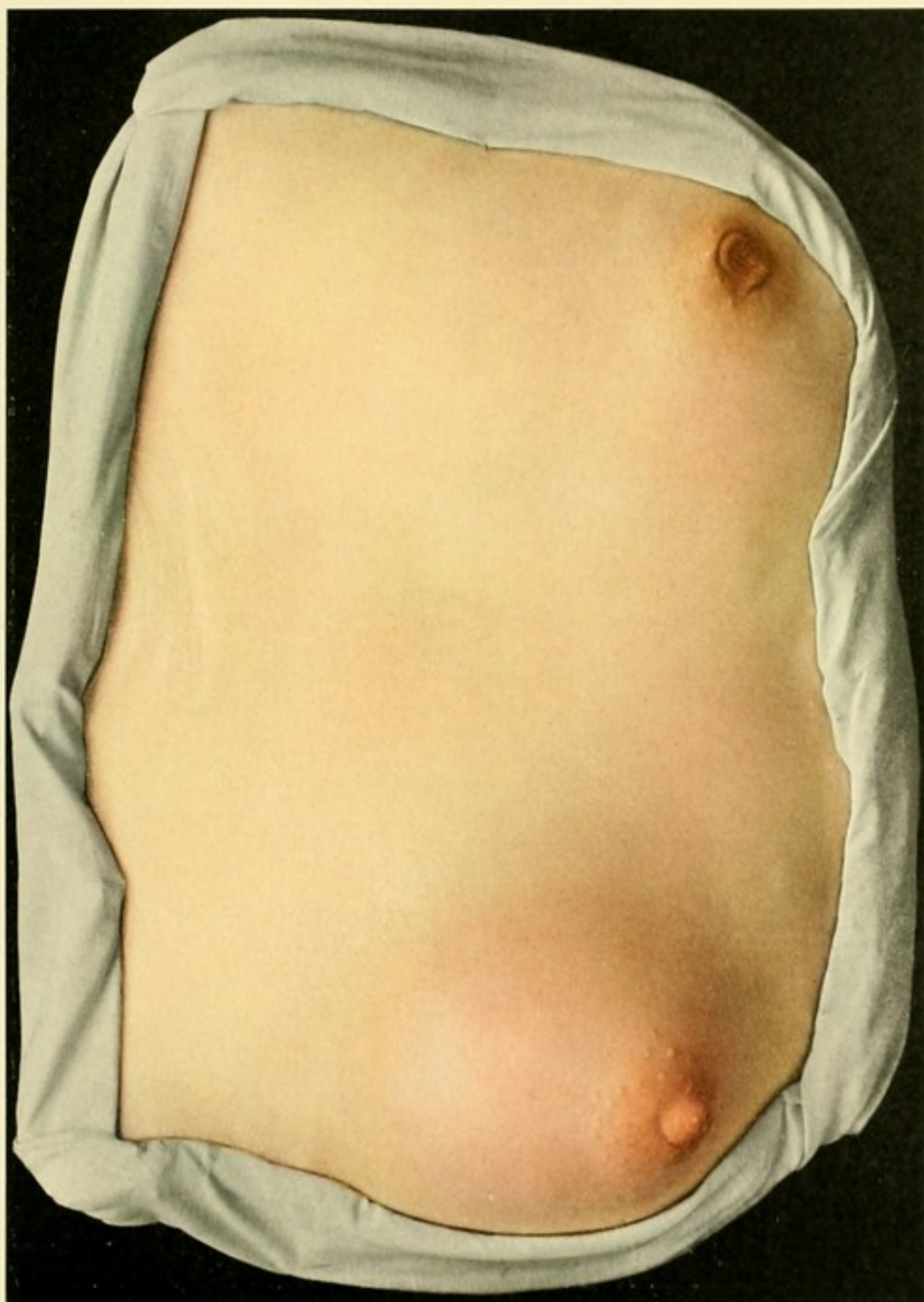
Tab. XXVIII.

Fig. 37. Fibroadenoma mammae cysticum.

noma is the only benign tumor of the breast deserving consideration, on account of its comparative frequency.

Fibro-adenoma usually develops in the peripheral portions of the mammary gland in young women, as a slow-growing, nodular tumor, so well encapsulated that it is freely movable within the breast. Tumors of this type are rarely multiple and seldom affect both breasts. When there is an abundant development of connective tissue the tumors are firm; when cystic cavities develop they are soft and fluctuating (*cystic fibro-adenoma*).

The tumor described as cystadenoma papilliferum, or intracanalicular fibroma, which is formed by connective tissue processes covered by epithelium projecting into the cavity of the cyst, belongs to the group of benign mammary tumors. In older women, especially at the menopause, small multiple cystadenomata occur chiefly in the region of the nipple, without causing retraction; sometimes in both breasts. These feel like solid tumors owing to their thickened walls. The name of chronic cystic interstitial mastitis has been given to these tumors by *König*, and that of "*cystic disease of the breast*" by many authors.

The benign nature of these tumors is shown by the fact that they ordinarily cause neither glandular nor organic metastases. On the other hand, these tumors, especially cystic fibroadenoma, after slow increase in size may become enormous growths, as large as a man's head, and then cause much inconvenience by their weight, and also radiating pains in the arm. Moreover, there is a possibility of a transformation into carcinoma or sarcoma, so that here again (see p. 14) we must be doubly certain before we affirm the non-malignancy of any given case, and it is better to err on the side of radical interference than on that of too much expectancy.

Differential Diagnosis

Chronic interstitial mastitis may give rise to a nodular infiltration of the mammary gland, but this disappears under treatment by cleansing the nipple, injection of alcohol into the nodules, and suspension of the breast; in distinction to the steady growth of tumors. But there are many doubtful cases, and this diagnosis is very difficult. **Cysts** occur chiefly in the neighborhood of the nipple, from which a brownish fluid can be expressed. When they appear under the skin they can be recognized by their bluish, glistening surface.

Galactocele begins developing during a lactation period and has, when large, a special doughy consistency. Incision discloses masses of cheesy material.

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Carcinoma of the breast is characterized, as already said (page 13), by its *hardness*, its *infiltration* into the tissues, retraction of the nipple and dimpling of the skin. However, a few cases of metastatic carcinoma in the breast (*e.g.*, a metastasis from a chorioepithelioma observed by the writer) are encapsulated, movable and of slow growth as a benign tumor. Such rare occurrences could be suspected only by the knowledge of the primary growth, and correctly diagnosed only by microscopical examination.

The latter, made by a competent man, is the last and final resort for the diagnosis of breast tumors, and must be performed in all uncertain cases. If the immediate examination of frozen sections at the time of operation is conclusive (which unfortunately is not always the case) the surgeon has a safe criterion to guide his further course.

Treatment

Early removal of all chronic nodular formations in the breast is advisable. They should be exposed by an incision radiating from the nipple (but avoiding it) and extirpated with the adjacent mammary tissue. Large tumors can be removed subcutaneously by raising the breast through a curved incision at its lower border so that, after healing, the scar is hidden under the breast which overhangs it.

In very extensive growths, especially cystic fibroadenoma and multiple cystic disease of the breast, the whole gland should be removed. But it is not necessary to remove the pectoral muscles, or thoroughly clean the axilla, so that the unpleasant after effects of radical amputation of the breast (edema of the arm, interference with the function of the latter) will not be so marked.

Fig. 38 shows a slightly curved **cutaneous horn** about three-fifths of an inch long, in an old countrywoman, in the zygomatic region, with all the characteristic features. The skin at the base of the growth is scaly and somewhat reddened. The same figure also shows multiple pin point **adenomata** of the sebaceous glands.

Cutaneous horns occur more frequently in old people (senile keratoma), and in those subject to exposure (sailors, etc.). They develop on preëxisting sebaceous and dermoid cysts and warts, and are observed on the eyelids, nose, lips, cheeks and ears, also on the scalp and genital organs. They are seldom multiple. They generally form sessile, freely movable, curved or spiral structures which have an irregular, grooved, yellowish-brown surface and a horny consistency.

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Fig. 39. Endothelioma cutis.

Fig. 38. Cornu cutaneum — Adenomata sebacea.

These benign formations, which may attain a length of two inches or more, are formed by a proliferation of the horny layer of the epidermis. The papillæ are also lengthened, which accounts for the soft consistency of the inner core.

Differential Diagnosis and Treatment

In young people multiple nævi with cornification occur, but these have a wider base, and a flatter and more prickly surface.

As about 10 per cent. of cutaneous horns degenerate into carcinoma (see page 1), excision by the knife with a ring of healthy skin is indicated. Recurrence takes place after removal by simple ligature.

Adenoma of the skin develops from the sebaceous glands or from the sweat glands (*adenoma sebaceum, adenoma sudoriparum*). Both conditions are rare: adenoma sebaceum seems fairly frequent in England.

Adenoma sebaceum consists in small translucent tumors, imbedded in the skin: of pin point to small pea size, round, movable, encapsulated and circumscribed, which usually occur on the flush area of the face, often in women, young or old, and is very often associated with telangiectases. The consistency of the tumors is quite firm. This lesion is benign, does not involve the lymphatics and does not recur after removal. It is usually congenital.

Adenoma sudoriparum (multiple benign cystic epithelioma, *Pusey*) is still rarer; the tumors are much like those of adenoma sebaceum, but generally slightly larger. The surface is quite smooth and glistening: the tumors look a little like vesicles. It is also a congenital lesion. Any large adenoma of the skin should be extirpated. Smaller, pin-point size lesions may be treated by the X-rays. This was done in the case represented in **Fig. 38,** and the small tumors disappeared to a great extent. Electrolysis is also a suitable method.

Fig. 39 shows a horseshoe-shaped **endothelioma** of the zygomatic region, in an old woman. The tumor is situated in the skin and has grown out of it. It is movable over the subjacent tissues. The borders are regular on all sides. The skin over the tumor is reddish brown like sarcoma, very thin, and cannot be raised from the tumor. It shows numerous fine ramifying vessels. In the middle of the horseshoe is an ulcer which resembles a rodent ulcer. There are thus points of resemblance to both carcinoma and sarcoma. The

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soft borders and consistency, the circumscribed form, and the absence of glandular involvement, show the benign nature of the tumor. In endothelioma of the face the occurrence of small multiple cysts in the cutaneous covering is more common than ulceration.

Excision of the tumor was performed and the defect was repaired by a plastic operation. Microscopic examination showed the growth to be a plexiform hemangio-endothelioma.

The group of tumors linked together under the name of **endothelioma** is far from being homogeneous; its histology is exceedingly complex and unsettled, and the very existence of a group of tumors to which the name endothelioma would properly belong has recently been questioned.

The opinions heretofore most generally admitted may be summarized as follows:

Endotheliomata (*Golgi*) arise from the endothelium of the blood-vessels and lymphatics, which, according to *Borst,* consists of specially modified connective-tissue cells. Owing to the double nature of the endothelium, it is not surprising that those who regard endothelial cells as epithelial cells give the name of endothelial cancer to the tumors arising from them, while others, who regard endothelial cells as connective-tissue cells, call these tumors endothelial sarcoma, plexiform angiosarcoma (*Waldeyer*) and angiosarcoma (*Kollaczek*).

If we hold with *Borst* that endothelium cells are but connective-tissue cells, which may assume all kinds of modifications, it follows that tumors of varied structure may arise from the different varieties of endothelium. According to cases, these tumors bear a resemblance to fibroma, sarcoma or carcinoma (but without cornification). Thus we avoid the endless number of names given to these tumors, and clinically have only the term endothelioma, to be further analyzed microscopically as hemangio-endothelioma and lymphangio-endothelioma. In these two great groups we can still divide cases into alveolar, plexiform or vascular, according to their microscopic structure.

Clinically, endothelioma may appear in the most varied forms and be mistaken for *fibroma, adenoma, sarcoma* and *carcinoma* (more particularly the first two), from which it may be distinguished only by microscopical examination.

Endothelioma may arise from all kinds of endothelium and is most frequently observed in the skin of the face, the mucous membrane of the mouth and pharynx, the bones of the face and skull, the peritoneum, the pia mater of the brain and spinal cord, and the parotid gland. (See **Fig. 40.**)
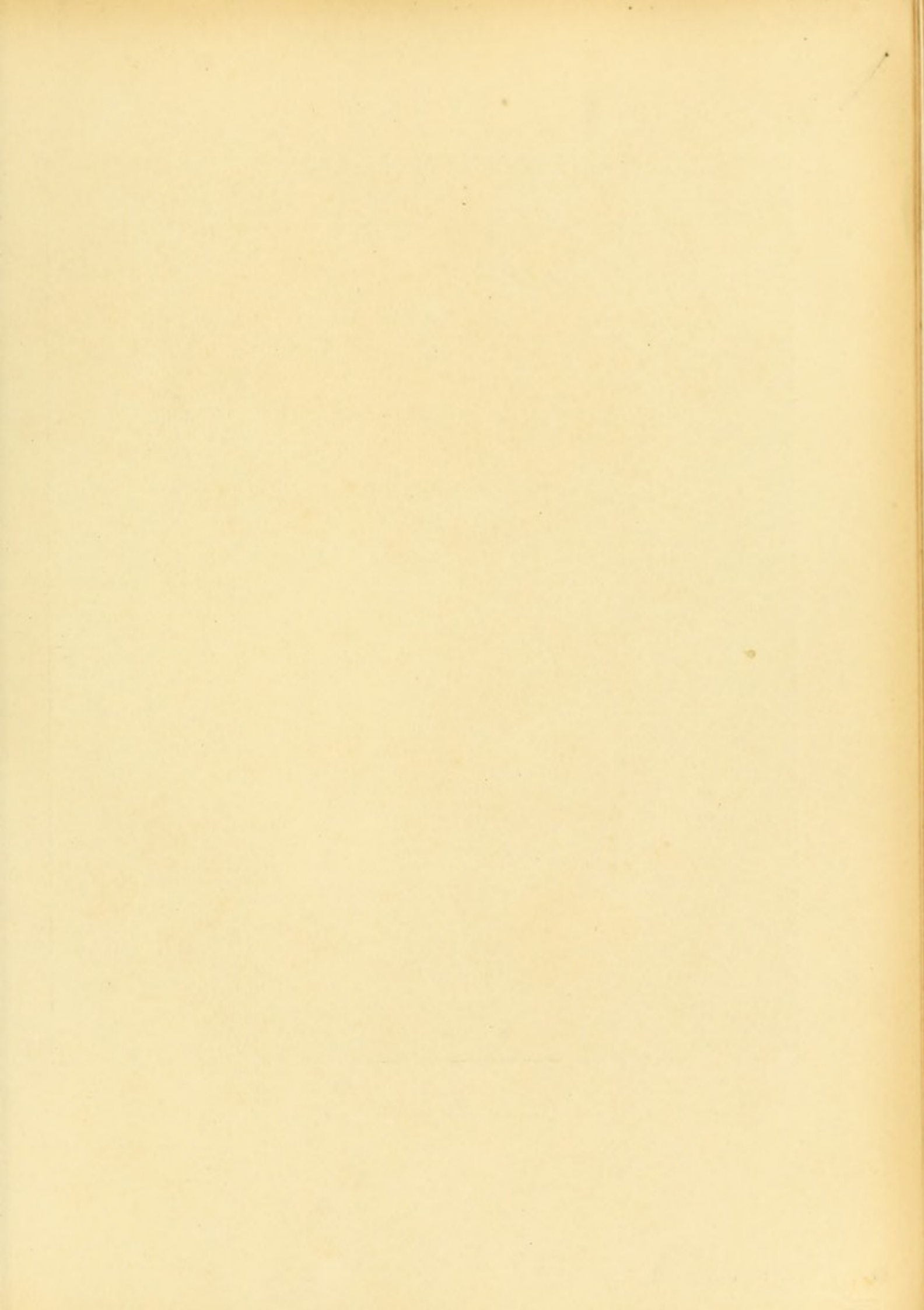
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Fig. 40. Endothelioma parotidis — Tumor mixtus.

Occurring at any age, it forms encapsulated, generally slow-growing, comparatively benign tumors which seldom cause glandular or organic metastases, but have a tendency to local recurrence.

As the shape, surface and consistency of the tumor may assume all possible varieties, the clinical signs of endothelioma are very indefinite. The shape is often irregular, especially in endothelioma of the face (**Fig. 39,** horseshoe shape). The surface may be smooth, uneven or ulcerated. The consistency may be hard, soft or cystic. Sometimes the tumors are very vascular and the epidermis takes the reddish-brown coloration which is seen in sarcoma (**Fig. 40**) at other times they are poor in vessels. Although they are at first encapsulated they may later on give rise to a diffuse infiltration of the tissue along the endothelial clefts, and then have irregular boundaries.

Treatment

Early excision is indicated, as transformation in rapidly growing tumors is possible. In the diffuse forms, which represent malignant tumors like carcinoma and sarcoma, extensive operations are necessary. When multiple nodules develop in the extremities amputation is sometimes necessary. Metastases in the lymphatic glands, which appear in the form of soft nodules, should also be removed.

Fig. 40 shows a ***mixed tumor of the parotid*** which slowly developed during three years in a woman aged 30. Profuse salivation, and latterly rapid growth of the tumor, led the patient to seek advice. The skin is freely movable over the tumor and shows a fine network of vessels. The tumor lies under the fascia and has spread to the anterior and lower region of the ear. The surface is irregular; the consistency of the posterior portion, where the surface is uneven, is hard; that of the anterior portion, where the surface is smooth, is soft and fluctuating. The tumor does not project into the buccal cavity; it is freely movable over the subjacent parts, and there is no glandular enlargement.

The tumor was extirpated with its capsule, and the facial nerve avoided. Part of the parotid gland was left behind. On section, cartilage, cysts, calcification, and fibrous and sarcomatous tissue were found.

Mixed tumors occur frequently in the parotid, less often in the other salivary glands. These mixed tumors, which also occur in the breast, kidneys and testicles, are regarded as endotheliomata (see

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page 49) by some authors, while others hold that they arise from epithelial and connective-tissue cells.

On section, they show a very variegated structure, in which are found parts resembling carcinoma and sarcoma, mucoid and calcified tissue, cartilage bone, cysts. The presence of cartilage, which, to the feel, is the most characteristic element, coupled with the often slow growth, is responsible for the name *enchondroma* often given these tumors.

Parotid tumor occurs more often in young individuals, and appears as an encapsulated, smooth or nodular tumor, movable over subjacent parts, lying under the fascia, and covered by intact non-adherent skin. The rare tumors which lie above the parotid fascia probably originate in aberrant parotid rudiments. The consistency of parotid tumors may be stone hard, hard, soft or cystic, according to their composition, and may differ in different parts of the same tumor. At first they increase slowly, but may suddenly take on rapid growth, break through their capsule, infiltrate the surrounding parts like malignant tumors, and finally perforate the skin and ulcerate. In such cases there are glandular and organic metastases.

Tumors arising from the anterior part of the parotid cause swelling of the cheek; those arising from the posterior part of the gland raise up the external ear. Large tumors may extend toward the chin, the nape of the neck and the clavicle. In a few cases, the growth bulged exclusively toward the faucial region (*Mixter*) and could be extirpated through the buccal cavity.

Small tumors cause hardly any pain, but sometimes salivation. Extensive tumors may give rise to pain in the ear, deafness and facial paralysis.

Differential Diagnosis

The more common *cartilaginous tumors* with uneven surface are easy to distinguish from other growths, but the soft tumors with smooth surface may be mistaken for *salivary cysts, cavernous angioma, lymphangioma, lymphadenoma, lipoma, fibroma, myxoma, sarcoma* and *carcinoma*. As all of these tumors call for extirpation, an exploratory incision which will become the first step of a radical interference, is justified in all doubtful cases.

Treatment

Mixed tumors should be *extirpated as early as possible* on account of the possibility of their taking on malignant character. Both benign and malignant recurrence may take place from the remains of
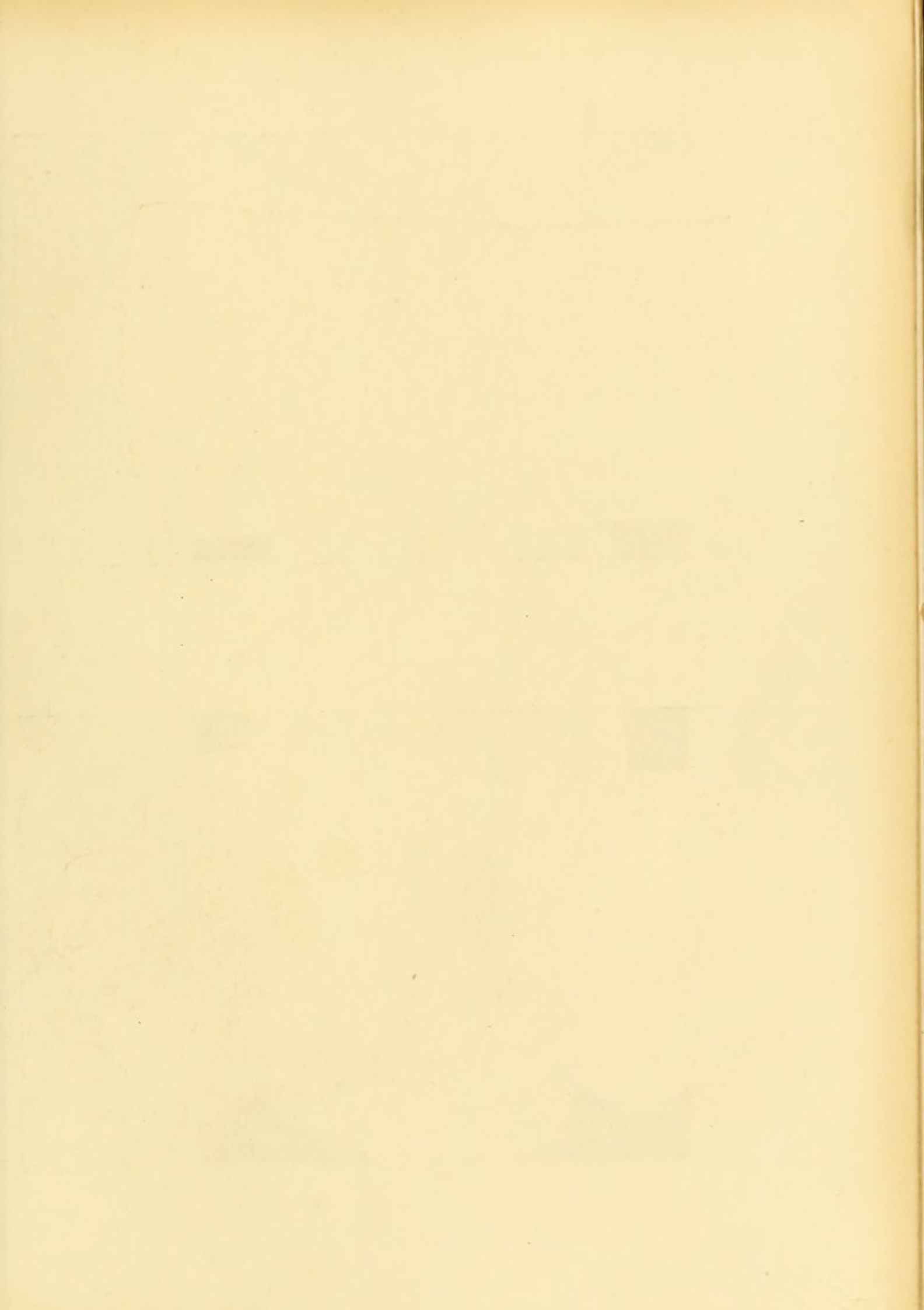
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Fig. 42. Bursitis praepatellaris acuta.

Fig. 41. Ganglion carpale dorsale.

the capsule after removal of tumor. The capsule must, therefore, be completely removed during extirpation, which, however, is always a very difficult and delicate procedure owing to the more than intimate anatomical relations between the parotid tissue and the **_facial nerve._**

The latter must not be sacrificed except when the major interest of radical removal makes it imperative (malignant tumors). Even when care is taken to avoid large branches of the nerve, when part of the gland is unaffected and can be left behind, facial paralysis is frequent on account of stretching during operation, or of infiltration of the sheath of the nerve by blood. But if the nerve has not been divided, paralysis is only temporary.

In tumors of the submaxillary gland, very similar in nature to those of the parotid, the whole gland should always be removed. This procedure presents no special difficulty as to technique.

Fig. 41 shows a so-called **_"ganglion,"_** that is, a periarticular cyst, developed in a hernial protrusion of the synovial membrane of a joint (more particularly of the wrist and hand) through an interstice between adjoining bundles of fibres of the capsule. The case shown in **Fig. 41** is in a typical situation. It was observed in a young girl, and was a recurrence of a previous forcibly broken cyst. Extirpation of the ganglion resulted in cure. The unilocular cyst contained colloid matter. The presence of septa gave evidence of an earlier multilocular structure.

Ganglions most often occur on the dorsal surface between the extensor carpi radialis and extensor indicis, less commonly on the palmar side near the flexor carpi radialis (especially in pianists); also on the dorsum of the foot at the joints of the cuboid bone and in the neighborhood of the knee joint.

According to the theory most commonly accepted, ganglions are only retention cysts in a protrusion of the synovial cavity which has secondarily become isolated, or is connected with the articular cavity only by a more or less slender pedicle. According to another theory colloid degeneration of the joint capsule and the periarticular connective tissue gives rise first to multilocular, then unilocular cysts. Ganglions of the tendon sheaths develop in the bursæ normally existing between two tendons where they cross each other. They occur chiefly in the sheaths of the flexor tendons over the metacarpo-phalangeal joints, and cause neuralgic pain by pressure on the digital

nerves. They often occur after rowing and fencing, *i.e.,* from traumatic causes.

Spherical ganglions occur most commonly on the dorsal aspect of the hand in young women, and resemble exostoses on account of their hardness. They often cause neuralgic pains and slight trouble in the movements of the joints.

Ganglions are of slow growth, the skin is unaltered and movable over them; the surface is smooth or slightly wrinkled. The consistency is hard in small ganglions, soft and fluctuating in larger ones. In pedunculated ganglions there is slight mobility over the joint.

Differential Diagnosis

In the knee joint they may be mistaken for *affections of bursæ;* in the foot, for ganglions of the tendon-sheaths. *Tuberculous tenosynovitis* is distinguished by its nodular surface, its fusiform shape following the direction of the tendon, and sometimes by the fine grating sound it gives on motion or pressure.

Treatment

The only treatment to be recommended is *extirpation* of the cyst and its pedicle under strict observance of all rules of asepsis, for ganglions often communicate with the joint, or are only separated from the latter by a thin membrane.

The time-honored method of bursting the ganglion by violent pressure of both thumbs or a blow with a wooden hammer, and then compressing with a bandage often leads to recurrence. So do subcutaneous discision, puncture, injection of alcohol or simple incision. All these methods are nowadays obsolete.

Fig. 42 shows an **acute purulent inflammation of the prepatellar bursa. Fig. 43** shows a **chronic inflammation** of the same bursa, attended by the development of the cystic formation commonly called **hygroma.** The lower half of the *pretibial bursa* is also involved.

Bursitis is *acute* or *chronic, purulent* or *non-suppurative.*

Acute bursitis supervenes especially after injuries of the region, or after neighboring inflammations (furuncles, lymphangitis, erysipelas).

In *serous bursitis* (rheumatism) the skin is unchanged. In purulent bursitis, it is red and edematous far beyond the limits of the in-
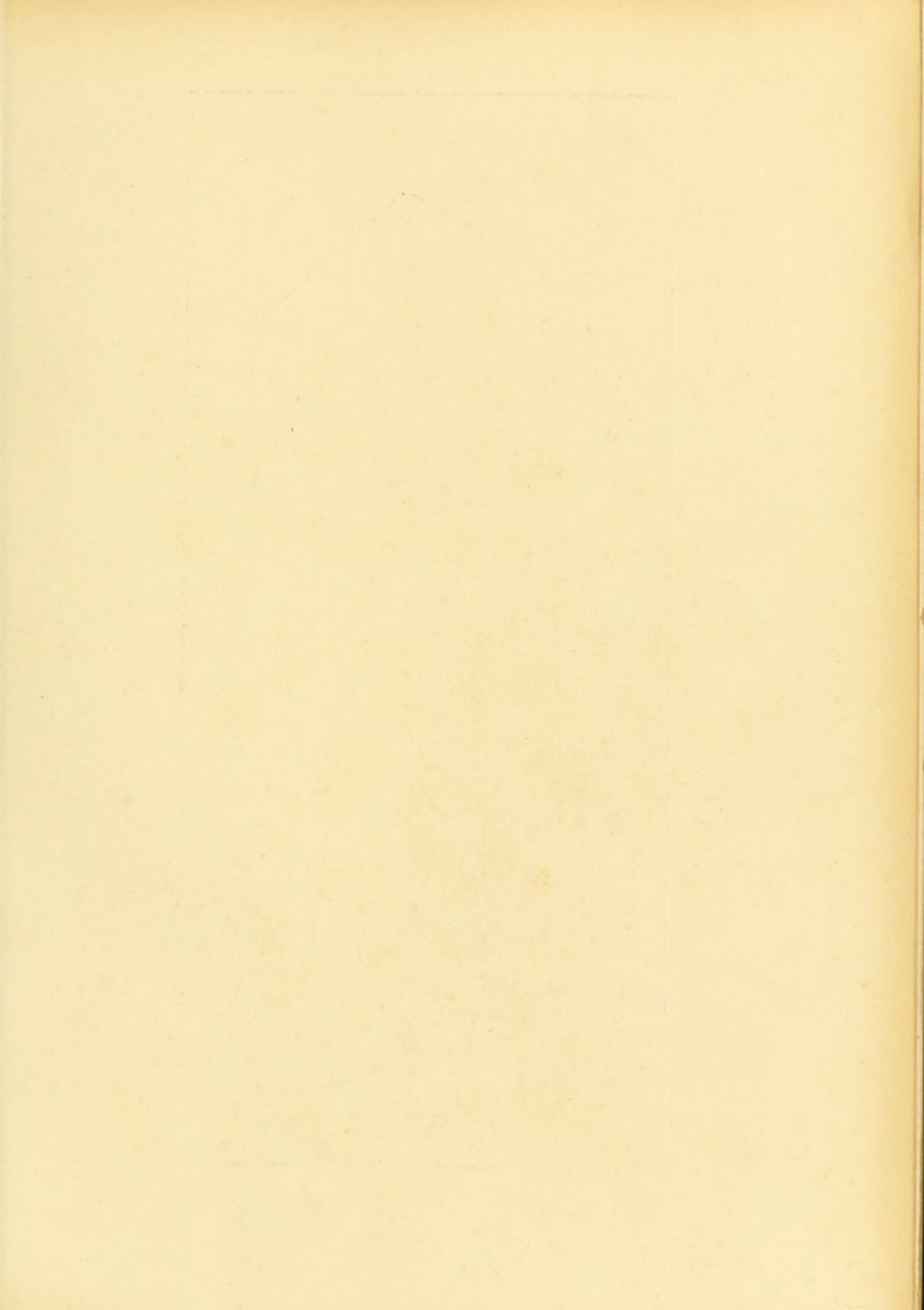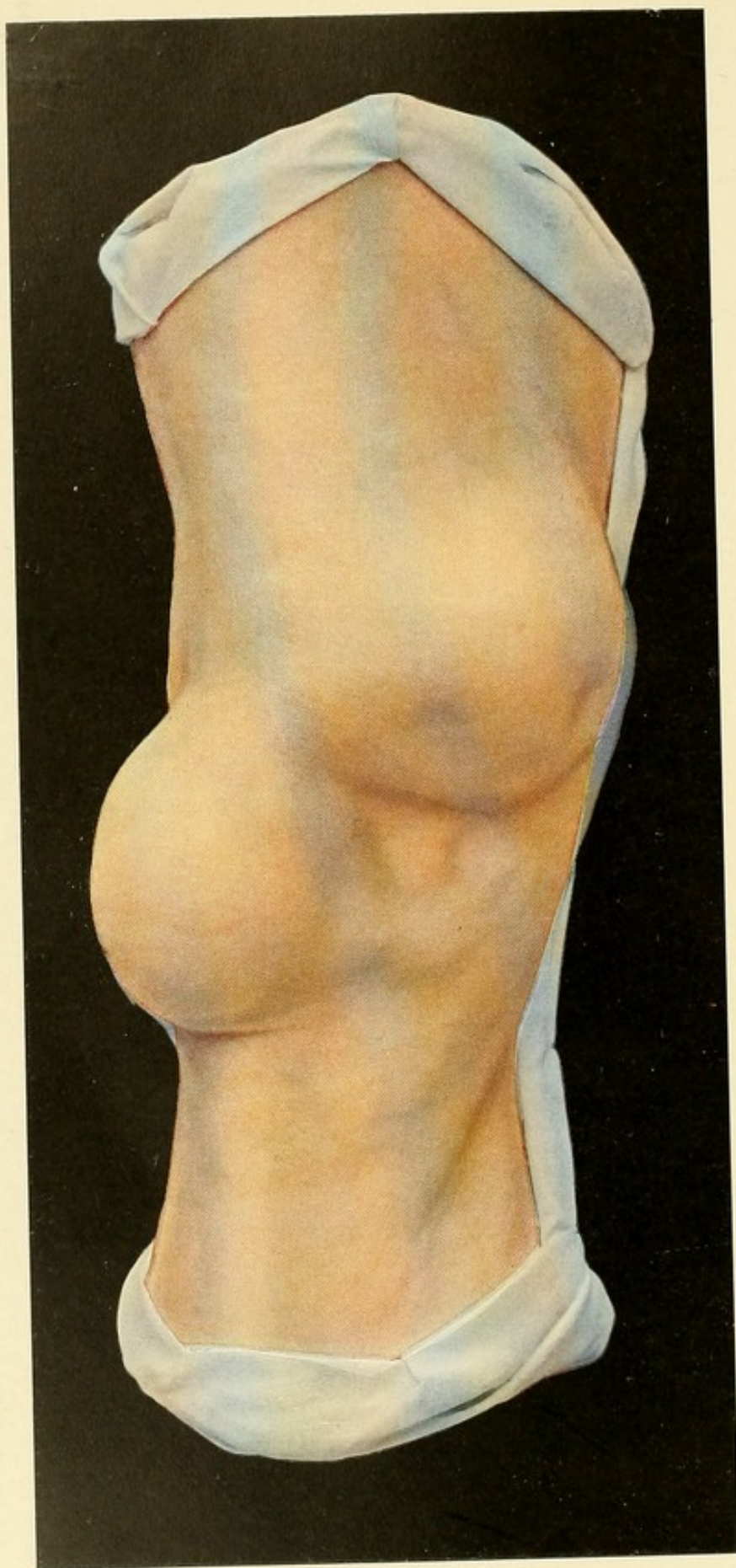
Fig. 43. Hygroma genus multiloculare.

flamed bursa. Suppuration also may extend beyond these limits and cause a diffuse phlegmon. The movements of the neighboring joint are painful and limited and there is high fever. Under the movable skin, in the case of superficial bursæ (*e.g.*, the prepatellar), a hemispherical, tense, sometimes fluctuating, slightly movable swelling with a smooth surface can be felt, limited to the anatomical position of the bursa (**Fig. 42**).

Hygroma is very generally an *occupational disease*, because repeated contusions and chronic irritation are the most important etiological factors. *Hygroma of the prepatellar bursa* is well known among persons who have to work in the kneeling position (as was the case with the man whose knee is shown in **Fig. 43**): hence the name of "*housemaid's knee.*" The "*miner's elbow,*" hygroma of the olecranon bursa, and the *rider's hygroma,* on the internal aspect of the internal condyle of the femur, are other frequent proofs of the same causal relation. It would be easy to multiply examples: there is not a trade requiring constant pressure and rubbing over a special point of the body that does not supply instances of the development of bursæ in this same traumatized point and of subsequent hygroma.

Hygroma may also develop in adventitious bursæ developed without occupational trauma, for instance, on the toes over a corn or bunion, or in any other point where the skin passes over a prominence of bone subjected to pressure.

Villous proliferations and rice-like bodies are often observed in the walls of chronic hygroma. When rice-like bodies are found, the case is generally considered as tuberculous.

The skin covering a hygroma is movable over the tumor, but rough and thickened, owing to the repeated irritation. The hygroma forms a *tumor* of varying size (some as large as a child's head) and *hardness* according to the thickness of its walls, but always *too tense to give real fluctuation.* The hygroma is spherical when developed in a unilocular, regular bursa; it is multilocular and irregular in shape (**Fig. 43**) when in a large bursa.

It causes no pain, and no functional disturbance except by its volume; when large, it hinders movement. In the case of "miner's elbow," there may be disability or neuralgic pain from pressure on the ulnar nerve.

Differential Diagnosis

The different forms of bursitis may be mistaken for arthritis of the adjacent joint, owing to limitation of movement, *e.g.*, subdeltoid

and sub-trochanteric bursitis. The strict localization of the affection to the anatomical position of the bursæ should make the diagnosis easy. Multiple bursitis is chiefly observed in tuberculosis, syphilis, gonorrhea and gout, and inquiries must be pushed in the direction suggested.

Treatment

Acute bursitis requires *early incision* to stop the progress of the condition and its extension to the adjacent structures. Acute serous bursitis is apt to undergo spontaneous resorption, which may be hastened by *compression. Tapping* and *injection of a few drops of carbolic acid* or alcohol will, at times, prove useful.

For **chronic hygroma,** the only treatment to be recommended is *total extirpation,* when feasible (sometimes the cyst is too large, or has too intimate connections with a joint, and part has to be left behind); or, when total excision is not possible, *incision, scraping and swabbing with carbolic acid, alcohol or tincture of iodine.* When treating a hygroma, always remember the possibility of communication with a joint.

The bursitis shown in **Fig. 42** was incised. All three bursæ—subcutaneous, subfascial and subaponeurotic were full of pus and in communication with each other.

The hygroma shown in **Fig. 43** was extirpated. The two bursæ were in communication.

Fig. 44 shows a tumor the size of a walnut, in an old woman. Its situation in the isthmus of the thyroid gland is evidenced by its ascension during swallowing. Its rounded form and regular outline and consistency show that it is a *cyst.* The lesion is therefore a **cystic goiter.**

Goiter is endemic in some countries (Switzerland), but sporadic cases are fairly frequent everywhere. Its real cause is unknown, though many are the hypotheses that have been made on this point.

Pathologically, the goiter may be follicular, colloid, vascular, cystic or fibrous. These different varieties may all be found together in the same tumor. The characteristic feature of all thyroid tumors is **ascension with the larynx during deglutition.**

The simplest form of goiter is *follicular hypertrophy.* The gland is slightly enlarged and studded with small, hard nodules which may persist indefinitely, undergo resorption, or more frequently, increase in size and lead to colloid or cystic degeneration.

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Tab. XXXIII.
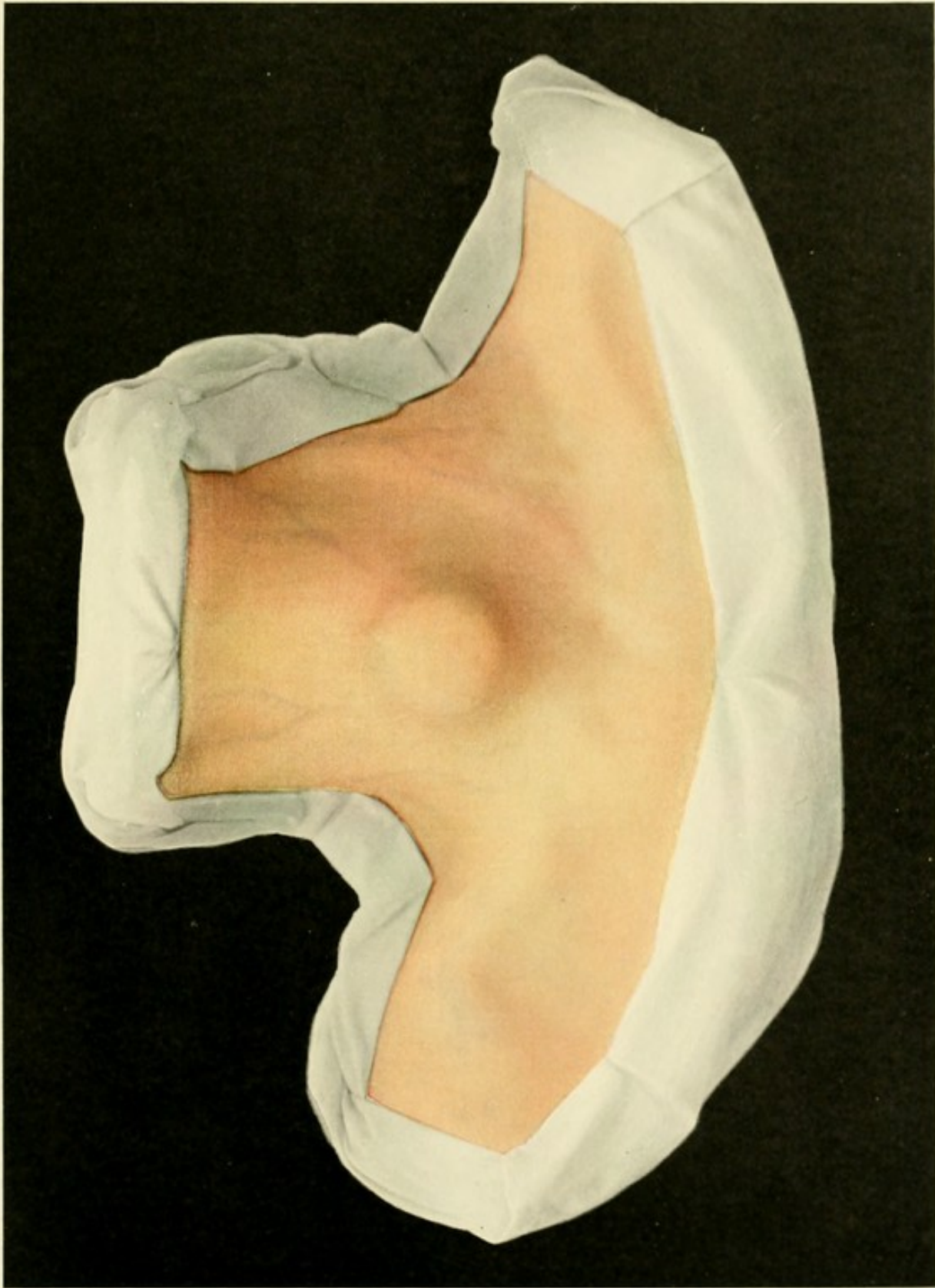
Fig. 44. Struma cystica.

Colloid degeneration is accompanied by a larger hypertrophy of the gland with formation of a horseshoe-shaped tumor containing several large nodules of gelatinous consistency. If the vascular element predominates, there is in addition pulsation and compressibility. If several smaller colloid nodules coalesce to form a cyst, there results a hemispherical tumor as shown in **Fig. 44,** with distinct fluctuation, which, however, may be lacking if the walls of the cyst are sclerotic, thickened or calcified. If fibrosis, the ultimate evolution in some cases, is marked, there is atrophy and hardening of the thyroid gland.

The **symptoms** caused by a goiter depend on the *location* of the tumor and on its *size,* which may vary within very wide limits. Even small goiters cause marked and early deformity. Further increase is accompanied by *pressure on the veins of the neck* (cyanosis of the face, development of the subcutaneous network of veins).

Pressure on the trachea causes displacement, stricture and flattening of the cartilaginous rings, causing the tube to assume the shape of a saber sheath. Sometimes, but not nearly as often as assumed by *Rose,* there may be softening of the tracheal wall to such an extent that the latter gives way after a violent movement of the head. Changes in the tracheal wall, important to know before operation, may be detected by the X-rays. Pressure on the trachea causes dyspnea, especially of the inspiratory type, stridor, sudden asthmatic attacks. Sudden death has been sometimes observed.

Pressure on one recurrent nerve is of little consequence, and often unnoticed because unilateral recurrential paralysis is compensated and can be detected only by laryngoscopic examination, as hoarseness is not present. *Bilateral recurrential paralysis* is, on the contrary, exceedingly grave (asphyxia, pneumonia).

Small fibrous tumors arising in the midline from the isthmus of the thyroid and those lying behind the sternum (*retrosternal* or *"plunging" goiter*) are those that cause the most troublesome pressure symptoms.

Diagnosis

Diagnosis of goiter is not particularly difficult.

Carcinoma of the thyroid is a disease of elderly and old people. It is a nodular, very hard and rapidly growing tumor which soon infiltrates all the tissues of the neck and promptly leads to paralysis of the vocal cords, to glandular metastases and cachexia. A sudden, rapid growth, in old people, in an old standing goiter is always suggestive of malignancy.

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Sarcoma of the thyroid is a rare affection occurring in young people. The infiltration is diffuse, but the consistency is soft. Sarcoma may break through the capsule and give rise to severe hemorrhage.

Syphilitic gumma of the thyroid gland is probably not as rare as the scarcity of observations would lead us to believe, but the clinical history is little known, and barring anamnesis, a positive *Wassermann* reaction and the influence of specific treatment, there are no diagnostic elements.

Tuberculosis of the thyroid gland may also assume a nodular form, not unlike follicular goiter, but the cases well studied are not numerous enough to enable one to give a clear description.

Aberrant goiters, when connected with the thyroid by a palpable pedicle, are easily recognized. But when free, they may be mistaken for lymphoma, adenitis, sebaceous or dermoid cysts or malignant tumors.

Retrosternal goiter is a mediastinal tumor, and as such may be mistaken for any of the other kinds, particularly aortic aneurysm.

Exophthalmic goiter (*Graves' disease*) is characterized by *symptoms of hyperthyroidism* (tremor, palpitations, highly nervous condition, etc.) associated with bulging of the eyes and hypertrophy (always moderate in pure cases) of the thyroid gland. The train of symptoms is sufficiently typical to allow the diagnosis to be made: but symptoms of hyperthyroidism may sometimes appear in cases of long-standing goiters.

Treatment

Incipient follicular hypertrophy can be happily influenced by iodine preparations or thyroid extract (administered with caution, lest we produce symptoms of hyperthyroidism).

In localized lesions, cysts or nodes, the remainder of the gland being sound, *enucleation* is indicated.

In more diffuse forms, in colloid degeneration particularly, **partial thyroidectomy** by the *Mayo subcapsular* technique is the best operation. Total thyroidectomy is not physiologically permissible, as it is followed by post-operative **myxoedema,** akin to congenital myxœdema, sometimes associated with cretinism, observed in countries where goiter is endemic. An entire lobe is generally left, that is, the more affected lobe and the isthmus are taken away.

The subcapsular technique wards off the danger of **post-operative tetany** due to the removal of all **parathyroid** glandules situated

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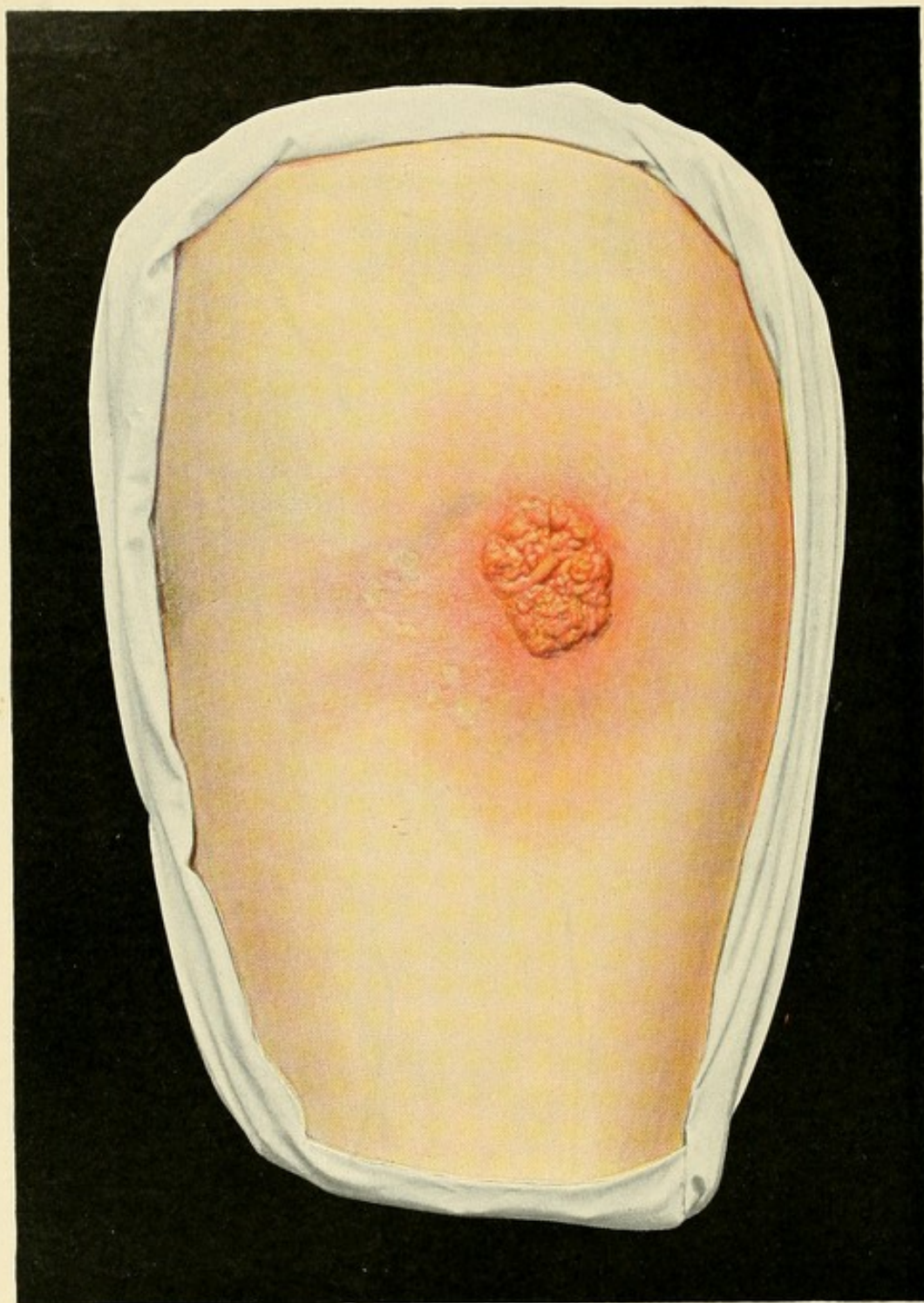
Fig. 45.   Papilloma cutis inflammatorium.

behind the thyroid lobes, and the anatomical disposition of which is variable. Should all parathyroids be inadvertently removed, tetany may be prevented by immediate *implantation* into the abdominal wall of one or two of the excised parathyroids; or it may be cured by subcutaneous injection of an extract of parathyroids or by the use of calcium lactate (*Beebe, McCallum*).

Transplantation of thyroid fragments has also been attempted for the cure of myxœdema.

In *Graves' disease*, when the hygienic treatment fails, operative measures are indicated. In not too severe cases ligation of the thyroid arteries (up to three) often brings about marked relief and lessens the hyperthyroidism. It can be done quickly under local anesthesia and in severe cases may be a preliminary step, preceding by a few days *partial thyroidectomy*, which is then indicated. There are no cases in which post-operative toxemia can be so hyperacute. Therefore the operation must be done under local anesthesia, as quickly and as gently as possible so as not to squeeze thyroid secretion into the circulation. The results are excellent when the dangerous post-operative period can safely be tided over, and no class of patients are more grateful to the surgeon who has cured them (*Crile*). Therefore it is important not to delay surgical interference too long, and not to dally with ineffectual internal remedies until the patient is in a hopeless condition.

Fig. 45 shows a so-called *"papilloma"* of the skin. It is a small tumor freely movable over the underlying parts, of rather soft consistency, and covered with warty projections. It has been frequently cauterized: hence the abrasion of the surface, and the inflammation of the surrounding skin. The surface is covered with a yellowish, fetid secretion, and between the villous projections are deep depressions caused by ulceration, so that the appearance in some places is almost that of carcinoma, but the borders are not hard.

In the past, the name "papilloma" has been used to designate various growths of the skin, consisting of hypertrophied papillæ covered with epithelium (warts, nævi, condylomata). It was even claimed that true papilloma, as distinguished from papillomatous formations, was a special type of fibro-epithelial tumor.

Nowadays the term papilloma of the skin is obsolete; at least in so far as the skin and external mucous membranes (see papilloma of the tongue, **Figs. 6** and **7**) are concerned, it is admitted that the

so-called "papillomata" are not tumors in the usual sense of the word, but simply papillary and epithelial hypertrophies developed under the influence of repeated irritations, chiefly of an infectious nature, in regions that are warm and moist; hence the frequency of these lesions around the mucocutaneous junctures of the genitals (venereal warts).

Similar growths may be observed on internal mucous membranes (larynx, intestines, bladder). On account of their vascularity, they bleed very easily, and hemorrhage is one of their chief symptoms. In the bladder, villous tumors sometimes degenerate into carcinoma or recur as carcinoma after excision.

Treatment

The best treatment of papilloma is *excision*. Papillomatous tumors of internal organs, especially the bladder, are very well removed by sparking with high frequency currents (*Beer, Keyes Jr.*). External "papillomata" may be destroyed with the galvanocautery or strong caustics. Mild cauterizations only irritate the lesion.

Figs. 46, 47 and **48** show three cases of ***dermoid cysts.***

Fig. 46 shows a ***dermoid of the forehead,*** where it is often observed, either above the root of the nose, in the inner angle of the eye, or laterally near the glabella (fissural dermoid cyst). The skin is movable over the tumor, which was observed in early youth, and shows a small white scar left by a former insufficient operation. The surface of the tumor is smooth and hemispherical. At the periphery there are raised bony walls. The tumor slowly attained its present size after the former operation and then remained stationary. There is no diminution on pressure over the tumor. It is of doughy consistency and but slightly movable over the subjacent bone.

Fig. 47 shows a ***dermoid of the prepuce,*** situated symmetrically on both sides of the raphe, and present since birth. The skin is so thin that the contents can be seen through it. The tumor has caused phimosis and balanitis.

Fig. 48 shows a ***dermoid of the neck*** in the position of the second branchial arch. Symmetrical dermoids in the middle line may occur above or below the larynx. Dermoids of the floor of the mouth may cause bulging of the submental region. The tumor has the size of a hen's egg, a smooth surface, a doughy, semi-fluctuating consistency.

Fig. 46.   Dermoid — Recidiv.
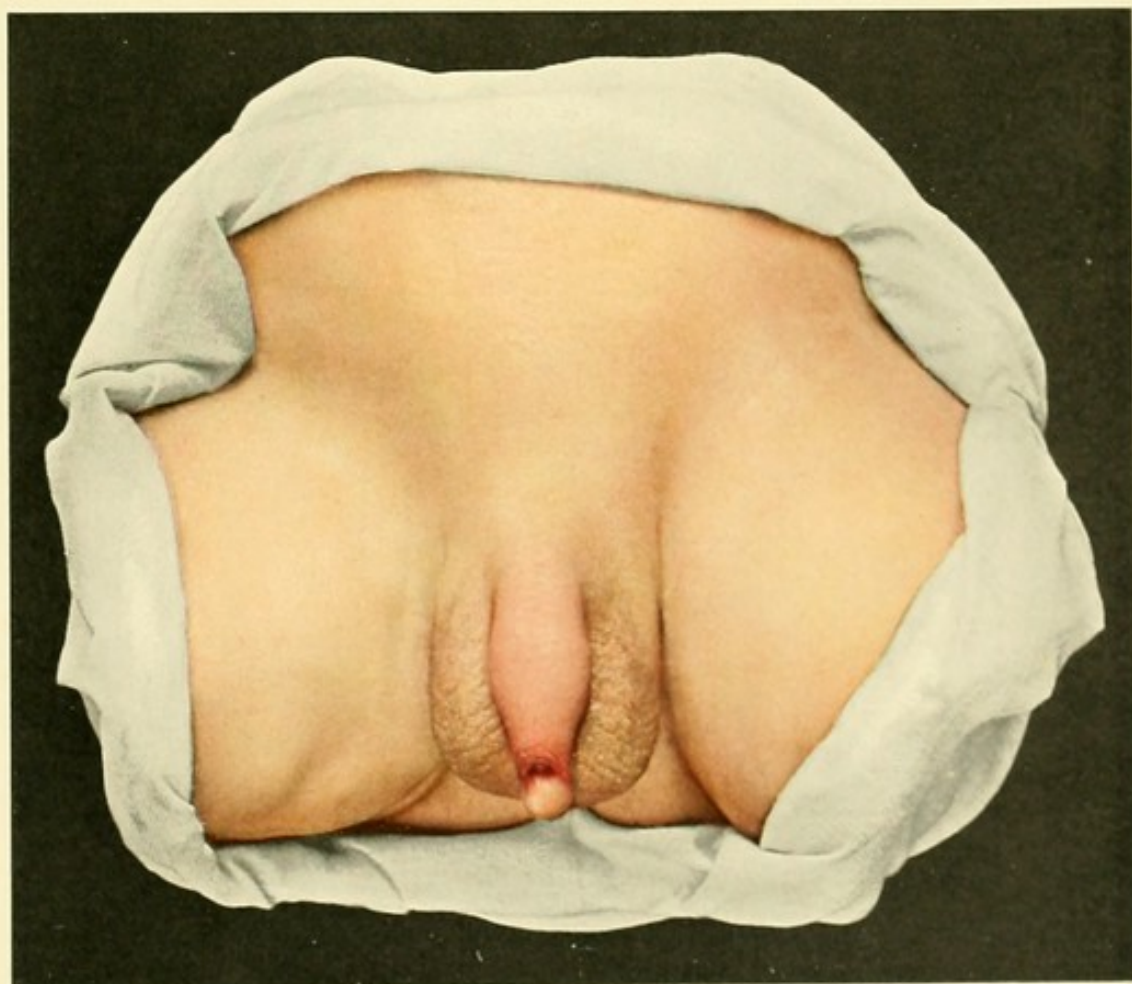
Fig. 47.   Dermoid — Phimosis.

Fig. 48. Dermoid — Cystis.

It is not adherent to subjacent parts nor to the unaltered skin. It dated back to infancy: it first grew slowly, later became stationary, and caused no inconvenience apart from the disfigurement.

True dermoid cysts are formed by invagination of the epiblast only, while compound dermoid cysts (*Teratoma*, **Fig. 146**) include all three embryonic layers.

Dermoid cysts occur only where, in embryonic life, there were folds, furrows or recesses, or in places where organs are developed by invagination of the epiblast. The latter mode explains dermoids in the vertebral canal, cranial, thoracic and abdominal cavities, retroperitoneal tissue and kidneys. The former mode of development accounts for the fissural dermoid cysts in the regions of the head, of the face (**Fig. 46**), in the neck, at the umbilicus and in the coccygeal region.

Dermoid cysts of the testicles and ovaries, on account of their complicated structure, are not pure dermoids.

Pure dermoids are unilocular or multilocular cysts, the external walls of which consist of connective tissue, and are connected with the surrounding tissues while the internal surface resembles skin (hence the term dermoid), and presents papillæ, squamous epithelium and hair. Those dermoids which contain bone, cartilage and teeth are formed at a very early embryonic period, before differentiation has taken place.

The contents of the cyst consist of a yellowish-white, caseous, odorless, fatty mass, mixed with numerous hairs, the appearance of which varies according to the situation of the dermoid (in the region of the eye, eyelashes, etc.). The contents are rarely serous or hemorrhagic. In the cutaneous or subcutaneous tissue the cysts form spherical or hemispherical tumors with a smooth surface and tallowy consistency. They are covered by intact skin, and are often attached to the bones. The superficial dermoids usually occur in youth. They are slow-growing and painless, and about the size of a walnut. Sometimes fistulæ form, from which hairs protrude.

Diagnosis

The diagnosis of superficial dermoids is easy to establish by the above signs.

But, according to the region of the body in which they are situated, even superficial dermoids may be mistaken for other conditions, such as *sebaceous cysts*, for instance, but the contents of the latter are yellow and foul smelling.

Lipomata are not congenital and are generally lobulated.

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A cyst in the location of that shown in **Fig. 46** might be diagnosed *encephalocele;* but the latter attains a much larger size and diminishes on pressure (see **Fig. 142** and page 231).

Also in the same case, owing to the scar of the previous operation, an *epidermic inclusion cyst* might be thought of; however, the latter is not congenital, develops only after a trauma, and, on microscopical examination, its walls contain only squamous epithelium without any sebaceous or sweat glands.

A *dermoid cyst of the neck* (**Fig. 48**) may be mistaken for a *tuberculous adenitis,* a *branchiogenous cyst,* a *thyro-glossal cyst* (see **Fig. 57,** *median fistula of the neck*). None of these conditions, however, has the doughy consistency of a dermoid cyst.

Dermoids of the umbilicus, on account of their special hardness, may be mistaken for malignant tumors, but they are of slow growth and circumscribed. Dermoids of the abdominal walls are often mistaken for sarcoma and fibroma, but the latter increase in size while dermoids remain stationary.

Deeply situated dermoids of the various cavities and organs, which often are noticed only by accident, cannot as a rule be distinguished from other tumors.

Treatment

Extirpation of the whole cyst is necessary, as recurrence takes place if any part is left behind. Commencing carcinoma has been observed in the inner surface of the cyst wall (*Wolff*).

Extirpation was carried out in the three cases represented.

Fig. 49 shows a **fibroma of the sheath of the flexor tendon** of the finger, the yellowish-white surface of which shows through the skin. The skin is slightly movable over the hard nodular tumor. The tumor itself is movable over the subjacent structures, and remains unaltered in position on moving the finger, which fact shows its independence from the tendon itself. Fibromata of tendon sheaths are rare on the whole, and are due to traumatic causes.

After injuries and stretching of tendons similar growths occur, sometimes multiple; they are due to proliferation of the cellular tissue. In *Dupuytren's contraction* (**Fig. 60**) nodules also develop in the palmar aponeurosis, which somewhat resemble fibromata.

Thickenings which occur in tendons and tendon sheaths, and lock the movements of the fingers in certain positions, are not true fibromata.

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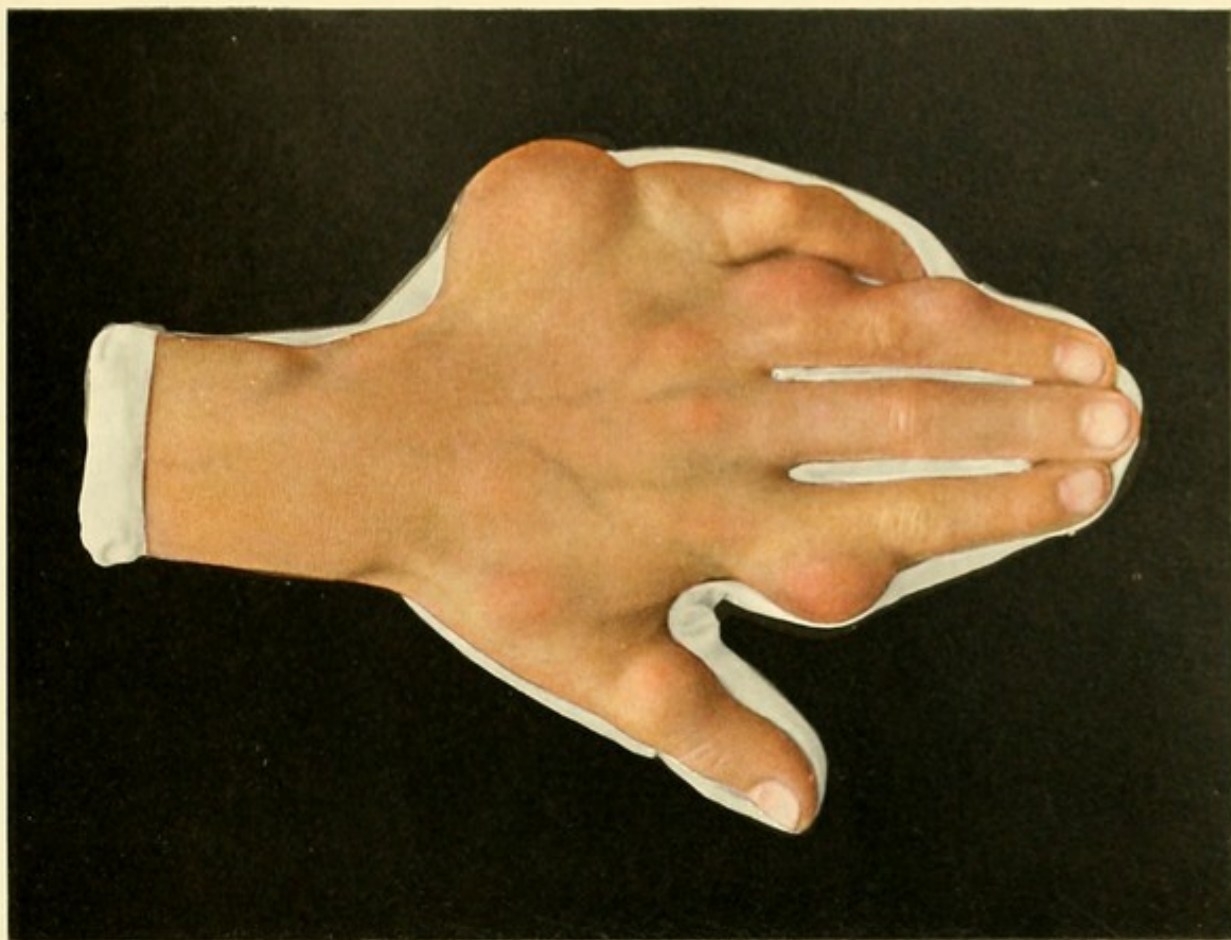
Fig. 50. Enchondromata manus.

Fig. 49. Fibroma vaginae tendinis.

Fibroma is a benign connective-tissue tumor, consisting of connective-tissue cells, fibrillar, inter-cellular substance and a variable amount of blood-vessels and lymphatics. When the matrix is hard and abundant, with slight development of spindle-cells, the fibroma is hard, while soft fibroma is formed by spongy tissue with numerous blood-vessels.

Those fibromata which consist of fibrous connective tissue with few nuclei are also termed *desmoids*, especially when they occur in the fascia of the abdominal walls, while the term fibrosarcoma is applied to tumors which consist of irregularly arranged spindle cells with little intercellular substance, and show degenerative changes and an absence of mature tissue.

Transitional forms from fibroma to fibrosarcoma and sarcoma are especially observed in the tumors occurring in fascia. Mixed forms are often found, such as fibro-lipoma, fibro-myoma, fibro-adenoma and fibro-myxoma. Cystic formation is also seen in fibromata.

Fibromata occur in all situations where fibrillar connective tissue is present—in the cutaneous and subcutaneous tissue (back and thigh), in intermuscular, intertendinous (**Fig. 49**), submucous and subserous tissue (alimentary canal, uterus, larynx). They may also develop in fasciæ and aponeuroses, nerve sheaths and periosteum (*naso-pharyngeal tumors,* **Fig. 25,** and *epulis,* **Fig. 35**), and also within the internal organs. Fibroids of the uterus are the most frequent of all.

Fibromata form circumscribed tumors of firm consistency and smooth surface, often encapsulated, slow-growing, sessile or pedunculated (fibrolipoma pendulum, **Fig. 52**). Pedunculated submucous fibromata often occur in the larynx in singers. Fibroid tumors may occur at any age; they are seldom congenital. After metaplastic changes (ossification) they may become hard.

In the skin and subcutaneous tissue they have a yellowish-white surface (**Fig. 49**). On section they show stratification and a glistening appearance like tendon tissue.

Differential diagnosis and treatment

Superficial hard fibromata of the skin and subcutaneous tissue are easily recognized by their form, consistence, clear demarcation and solitary appearance. It is only transitional forms between fibrosarcoma and sarcoma that present any difficulty. Deep fibromata which often attain a large size (*e.g.,* in the abdominal cavity) are recognized by their nodular surface, hardness and encapsulation.

Treatment is *excision* of the tumor with its capsule. For the removal of deep fibromata extensive operations are necessary. Sometimes they are so firmly attached to the neighboring tissues or organs that a portion of the latter must be removed with them. In other cases, they can be shelled out without difficulty (enucleation of uterine fibroids).

Fig. 50 shows a case, observed in a young man, of **multiple chondroma of the fingers,** which had been present since childhood. The nodular tumors are situated in the phalanges and metacarpal bones, and have caused thinning and reddening of the skin by pressure. The X-rays showed the origin to be in the medullary cavity. The tumors on the first, second and fourth fingers were incised and scraped. The little finger was removed with its metacarpal bone, on account of the multiplicity of the tumors.

Although cartilaginous tumors are pathologically divided into two groups: (1) **ecchondroma,** or hyperplastic proliferation from pre-existing cartilage, which only occurs in places where cartilage is usually present; (2) heteroplastic cartilaginous growths, or **enchondroma,** which occur in places where cartilage is not normally present, these two forms are often impossible to distinguish clinically. We therefore include both forms under the name of **chondroma.**

The tumors either consist of the different forms of cartilage, or else they are **mixed** (*chondro-myxoma, chondro-lipoma,* or *chondro-sarcoma*). Cystic degeneration may also occur in chondroma, and by liquefaction of cartilaginous tumors large *cysts* may form in the long bones. True chondroma may occur in the soft parts from aberrant pieces of cartilage in the neighborhood salivary glands, neck, ear, lungs, trachea, mammary gland.

The mixed tumors occurring in the testicles and salivary glands, which develop cartilaginous tissue through metaplasia, are not true chondroma.

Congenital chondroma and chondroma developing in infancy, according to *Virchow,* are due to disturbances in the development of bone during the period of growth, and arise from islands of cartilage left in the diaphysis. Rickets appear to play a certain rôle in this connection owing to the irregular ossification of the epiphyseal cartilages. In some cases there appears to be a hereditary tendency to the formation of chondroma.

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Fig. 51. Hämorrhoides et Fibromata ani.

True chondroma, or enchondroma, develops from the periosteum or medulla, most commonly in the phalanges and metacarpal or metatarsal bones; it is usually multiple. Cases of isolated chondroma also occur in the upper end of the humerus, the lower end of the radius, the head of the tibia, the pelvic bones and the scapula, often combined with cartilaginous exostoses (ossified ecchondromata with a cartilaginous covering).

Chondroma forms slow-growing, *hard*, nodular, circumscribed tumors, which may cause pressure atrophy of neighboring parts (**Fig. 50**). Multiple tumors, especially in the hands, cause considerable deformity by disturbance of growth (shortening and twisting). Spontaneous fractures may occur from destruction of the cortex, in tumors growing from the medullary cavity.

The *softer* forms of chondroma, less common than the hard, must be regarded as malignant, because they take on an infiltrating growth, extend to the veins and give rise to metastases. (*Chondrosarcoma.*)

Differential diagnosis

Central medullary chondroma has to be diagnosed from *osteomyelitic abscesses* and from *central sarcoma*. The former, on X-ray examination, show thickening of the periosteum; the latter can often only be distinguished by operation, as the X-ray appearances are very similar in chondroma and sarcoma (when the chondroma is single). Large chondromata of the head of the tibia or upper end of the humerus, and generally speaking peripheral chondromata are easily recognized by their nodular surface and typical hard consistency. Multiple hard tumors are always suggestive of chondroma.

Treatment

Any isolated chondroma should always be extirpated, as it may develop into sarcoma. Multiple tumors (case of **Fig. 50**) may be incised and scraped. If rapidly growing recurrence takes place, resection or amputation must be performed.

Fig. 51 shows around the anus yellowish **nodular hemorrhoids,** which, owing to the concomitant moist eczema and repeated ulceration and inflammatory changes, have undergone fibrous changes and somewhat resemble fibromata. In one place there is a typical bluish, glistening hemorrhoidal nodule.

Hemorrhoids, the most common surgical condition of the anus, are *external* or *internal,* according to their location.

External hemorrhoids are those developed from the inferior hemorrhoidal plexus. Constipation and pelvic congestion are two favoring factors in their development. They form bluish, compressible, nodular, sessile or pedunculated growths covered by thin skin and situated around the anal orifice. When turgid and inflamed they cause much itching, pain and tenesmus, while the nodules bleed easily and thrombophlebitis is frequent. The latter and the moist eczema bring about the changes shown in **Fig. 51.**

Multiple internal hemorrhoids of the lower part of the rectum bleed easily without being inflamed and are often accompanied by a slight mucous prolapse.

Diagnosis

When situated high up in the rectum, a *proctoscopic examination* is necessary. For ordinary cases, *digital examination* is generally sufficient for the diagnosis.

Condylomata acuminata, frequent in the anal region, might be mistaken for hemorrhoids only on superficial examination. The cockscomb-like vegetations are too characteristic. The same may be said of *condylomata lata.*

Genuine anal *fibroma* is rare, solitary and pedunculated.

Carcinoma of the papillomatous type is recognized by its early ulceration with hard borders and irregular outline, and surface bleeding to the slightest contact. In all cases of hemorrhoids, a digital exploration should be made for carcinoma.

The ***treatment*** of *hemorrhoids* consists in cleanliness, antiphlogistic measures during the periods of inflammation. and *removal* of the nodules if they are too troublesome, either by the *clamp and cautery method* (applied in the case of **Fig. 51**) or by a bloody operation if there is a complete ring of sessile tumors. Anal dilatation is the first step in all operations for hemorrhoids.

Figs. 52 to **54,** inclusive, show three types of ***lipoma.***
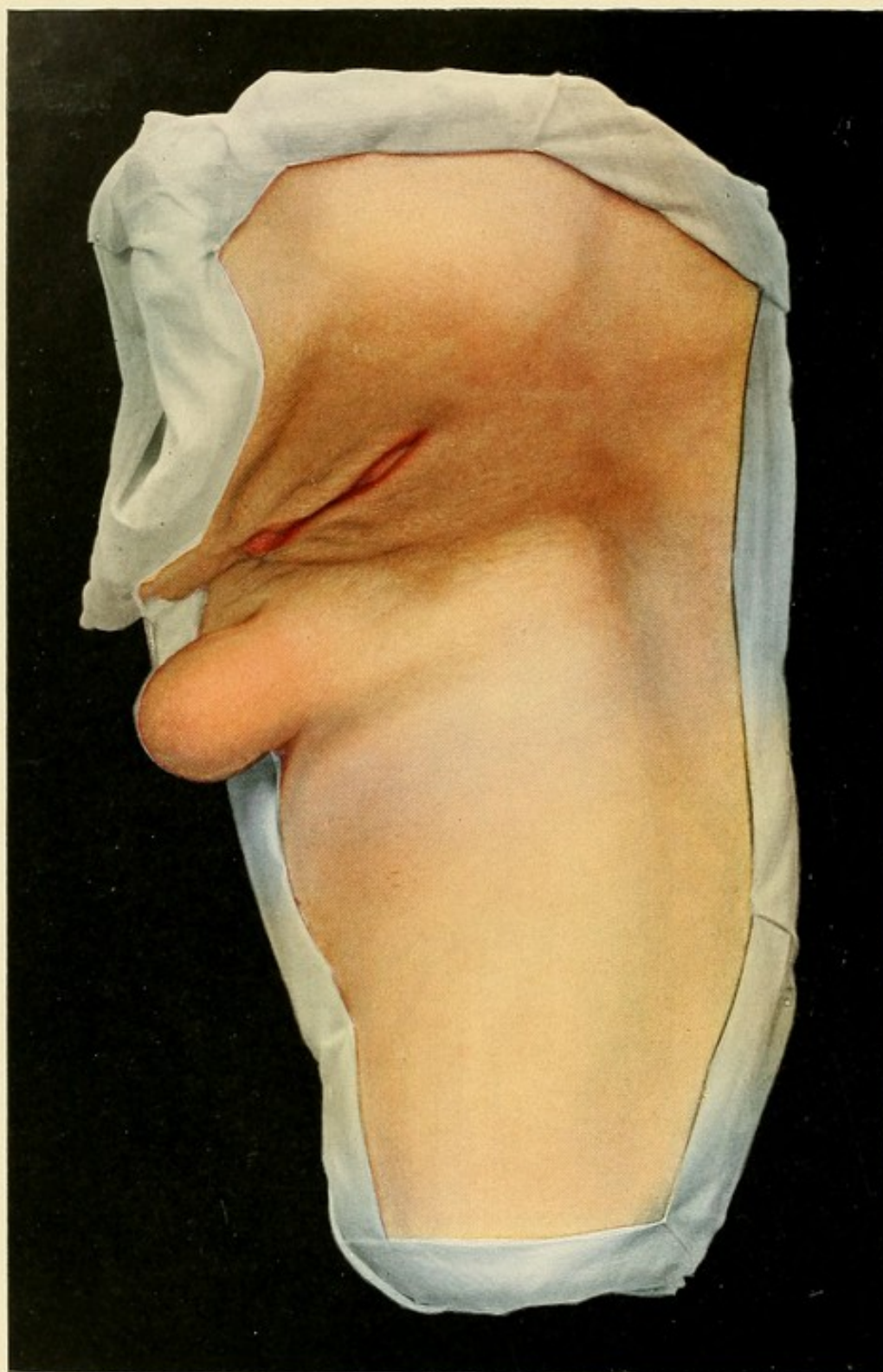
Fig. 52.   Fibrolipoma subcutaneum pendulum.

Fig. 52 shows a pendulous *fibro-lipoma* in a middle-aged woman. The skin is somewhat reddened, but non-adherent. The tumor is smooth, moderately hard in consistency and movable over the fascia. Its base is broad, on account of its small size.

Fig. 53 shows a *sub-cutaneous lipoma* the size of the fist in a common situation in a middle-aged woman. The puckering of the skin is clearly seen. These puckerings (white spots in the figure) are also found in the breast, and are due to processes of the lipoma extending into the latter.

Fig. 54 shows *symmetrical lipomata* in the region of both parotids, in the upper eyelids, and in various parts of the neck (both sides of submaxillary region and sublingual region) in an old man. The painless tumors had not increased in size for some years. Their lobular surface and their consistency distinguish these solid tumors from symmetrical cystic formations in the salivary glands, which cause similar swellings in the face and neck. (See about lymphangioma and *Mikulicz'* disease, page 236.) The disease is distinguished from simple adiposis by consisting of multiple, separate, encapsulated tumors. There were no other lipomata in other parts (in distinction to cases in which lipomata occur over the whole body).

Lipomata are tumors formed of fatty tissue, and have, therefore, the yellowish-white color, *soft consistency*, and *lobular structure* of fatty tissue. The individual fat lobules are separated by more or less strongly developed connective-tissue septa, and the whole tumor is demarcated from the surrounding tissues by a thin capsule. Lipomata are of soft, often pseudo-fluctuating consistency; in rare cases they are harder, because they contain more connective tissue.

They are slow-growing, globular tumors, which sometimes attain an enormous size, and are usually supplied by a single vessel at their base. They are generally sessile. In large tumors the skin is often drawn so as to form a broad pedicle. Narrow pedunculated lipomata (**Fig. 52**) are rare.

Lipomata are *essentially benign* tumors, which neither recur, nor give metastases, nor undergo malignant changes.

Besides the fat, which differs from ordinary fatty tissue only in that the globules are slightly larger, there may be other constituents, hence the varieties *fibro-lipoma, myxo-lipoma, angio-lipoma, chondro-*

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lipoma. Cystic degeneration may give rise to so-called oil-cysts in the interior of lipomata.

The etiology of lipoma is unknown. That it is a true tumor is shown by its persisting in severe emaciation. Thus are multiple lipomata distinguished as a nosological entity from obesity, though, clinically, it may be difficult in some cases to know which of the two conditions we are dealing with.

That repeated irritations may act as a predisposing cause is demonstrated by the development of lipomata on the back of carriers and on the forehead of persons who wear hard hats. Pregnancy may give a sudden impetus to the growth of stationary lipomata.

Developmental and trophic disturbances undoubtedly play an important part in the production of *multiple lipomata,* which are usually *symmetrical* (**Fig. 54**).

Symmetrical lipomatosis has been described as a separate morbid process. Multiple lipomata may be connected with nerves or with lymphatic glands, which sometimes have been found within them. The connections with nerves explain why those multiple lipomata are often painful (*Dercum* has described a variety under the name of *adiposis dolorosa*).

Congenital lipoma is found especially in spina bifida of the myelocystocele variety (usually myxolipoma, **Fig. 144**).

Lipomata are most often found in the subcutaneous tissue (**Figs. 52, 53** and **54**), where they appear as soft, encapsulated tumors with a lobulated surface, covered by non-adherent skin. The skin over the tumor becomes dimpled when pinched up, owing to its connection with the tumor by connective tissue (**Fig. 53**). The seats of predilection for subcutaneous lipomata are the back, nape of the neck (fatty neck), axilla, shoulder, upper arm, thigh, buttocks and scrotum. In the limbs lipomata become less and less frequent as the region becomes more distant from the attachment to the trunk.

Subfascial lipomata are much less common. They may occur under the fascia of the forehead (where they may be mistaken for dermoids, see page 63) and under the palmar fascia. Intermuscular lipomata occur behind the pectoralis major and in the tongue. In the knee joint arborescent lipoma occurs, which has the typical structure of fatty tissue, and is connected by some authors with tuberculosis of the knee, healed or of very low virulence. Lipomata may also arise from the submucous and subserous tissue (gut and larynx); subperitoneal lipomata may give rise to hernia through the linea alba. Subserous lipomata also sometimes appear in the inguinal
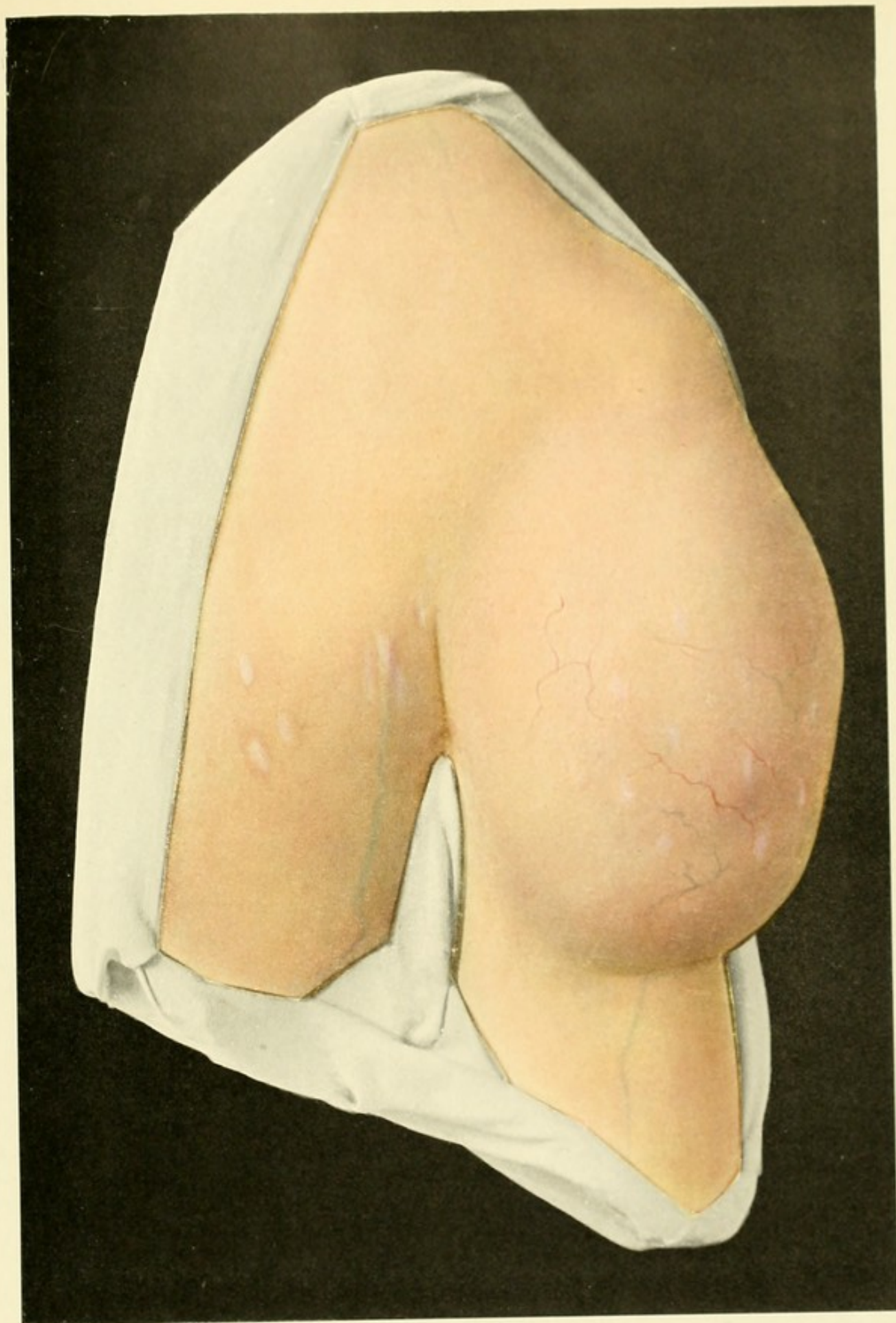
Fig. 53. Lipoma diffusum subcutaneum.
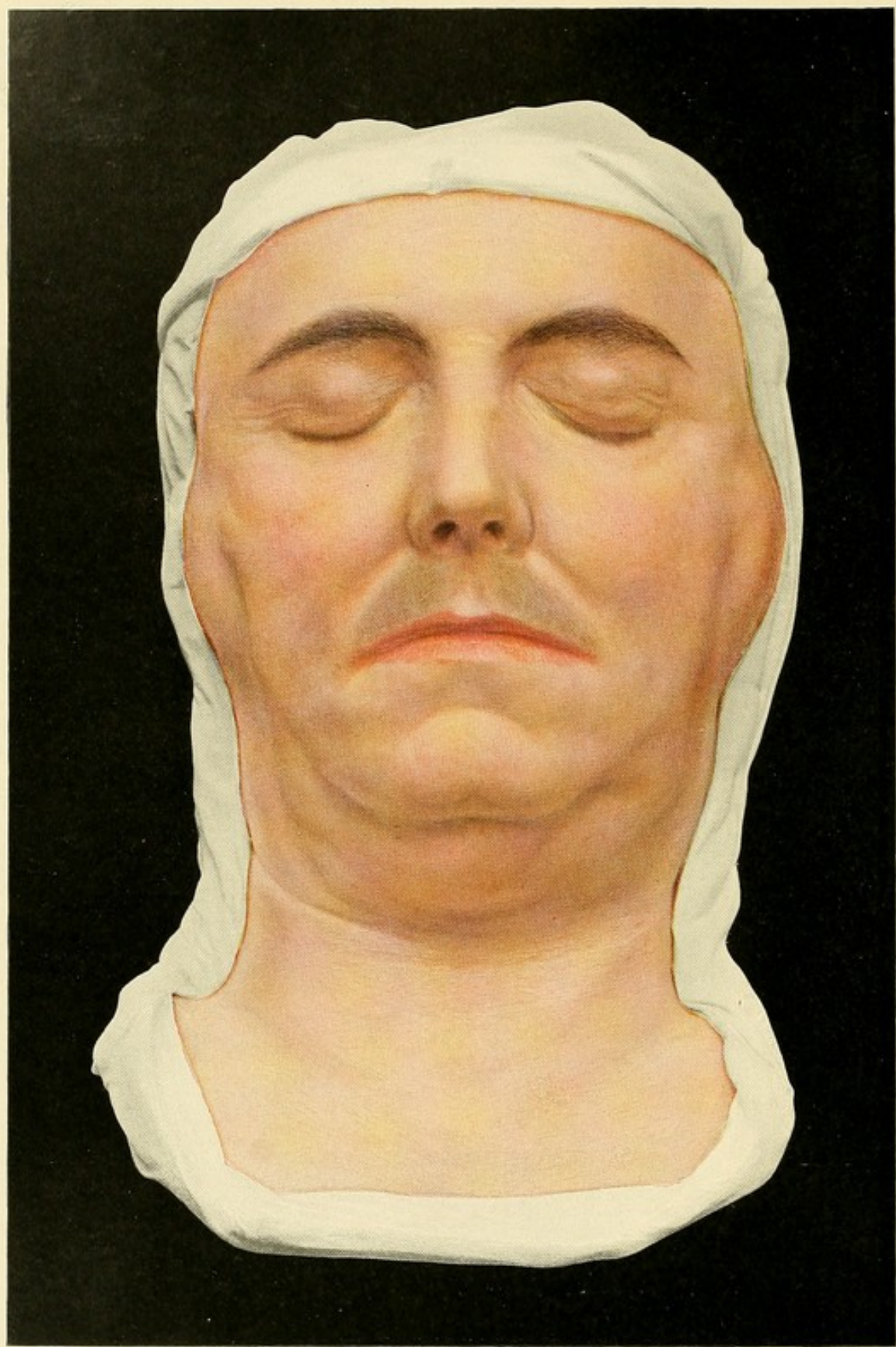
Fig. 54. Lipomata subcutanea symmetrica.

and femoral canals; in the omentum and mesentery; in the retro-peritoneal tissue, and in the glandular organs (breast and kidney).

All lipomata, especially subcutaneous, subfascial and inter-muscular, have a tendency to send processes into the surrounding parts.

Diagnosis

The characteristic features of lipoma, from the standpoint of diagnosis, are the *soft, pseudo-fluctuating consistency* and the *lobular surface* with puckering of the skin.

Fibromata are harder; *sebaceous cysts* are round, smooth and more tense; *cysts* and *hygromata are perfectly* smooth; *dermoids* have a special doughy consistency. *Adenitis* has small, hard nodes, unless already suppurative and does not much resemble lipoma.

The fatty accumulations seen after long suppurations, for instance in the perirenal capsule (lipomatous perinephritis) are not true lipomata. Nor is diffuse lipomatosis (obesity).

Treatment

The treatment of lipoma is *extirpation* of the tumor and of all its processes. This was applied to the three cases represented in the illustrations: several sittings were necessary for the case represented in **Fig. 54.**

In cases of lipoma of the limbs (such as that of **Fig. 53**) care must be taken, because, despite its encapsulation, the tumor may have sent processes in all directions, ensheathing the big blood vessels and nerves, which it is important not to wound.

Adiposity of the abdominal walls has recently been treated surgically. *Kelly* advocates removal of large masses of fat by wedge-shaped excisions before laparotomies. This facilitates the work of the surgeon, insures better repair of the abdominal wall and lightens the patient.

MISCELLANEOUS LESIONS

Figs. 55-83

A.—Scars—Fistulae—Figs. 55-59

B.—Deformities Due to Contractions of Muscles or
to Fractures—Figs. 60-66

C.—Naevi—Figs. 67-69

D.—Lesions of the Lymphatic and Vascular Systems—Figs. 70-83
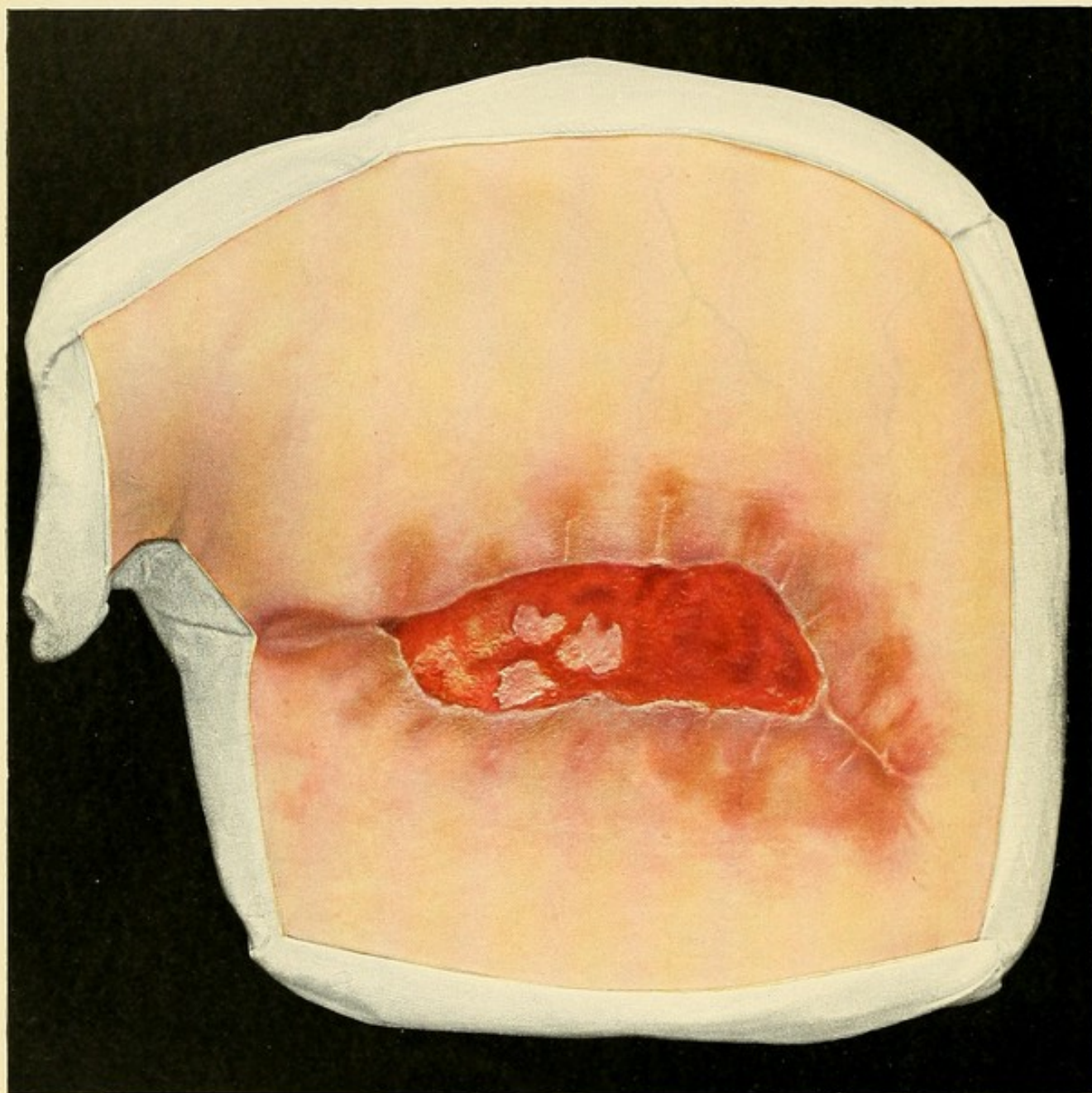
Fig. 55. Granulationes et Transplantationes.

SCARS—FISTULAE

Fig. 55 shows a *granulating wound* left by the extirpation of the right breast, and *three epidermic grafts* that have been transplanted thereon. After extirpation of the breast, an attempt should always be made to close the wound by sutures, but these should not be tied too tightly, especially in the center of the wound, where there is much tension, as they are liable to tear through the tissues and cause sloughing. **Fig. 55** shows the reddish-brown holes of the sutures, which have led to partial closure of the wound in the middle. The remainder of the wound can be left to heal by granulation, and *Thiersch's* grafts may be applied.

A wound is ready for epidermic grafts when it is covered with *red, vigorously sprouting* granulations. When the granulations are still yellowish (as on the axillary side in **Fig. 55**), it is still too early. Balsam of Peru, either pure or mixed with oil, is a very good dressing to promote granulation.

When the whole surface of the wound is covered with red, exuberant granulations, these are scraped off with a sharp spoon, and the bleeding surface compressed with hot compresses soaked in saline solution until all blood oozing has been absolutely checked: this is essential to success, as oozing would raise the grafts from the surface of the granulations; the largest possible epidermic grafts are then applied, each one overlapping the preceding, and covered with sterile rubber tissue soaked in saline solution (*no antiseptics* in the whole process of skin grafting).

Scarlet red salve (5-8%) is very efficient to hasten the epidermization of granulating surfaces of all kinds (ulcers, partially successful grafting operations).

Fig. 56 shows a *fistula* due to *insufficient drainage* of a kidney. As a result of incision of a paranephritic abscess, a fistula has remained, which, in spite of drainage, packing and repeated scrapings, has not healed. The surrounding skin is inflamed and edematous. The granulations at the opening of the fistula are unhealthy, dirty-brown and purulent. Shreds of tissue with a fetid odor are discharged from the fistula.

Such an appearance of the fistula and its surroundings is typical of all cases where the external opening is too small, so that an abscess in connection with it is not sufficiently drained, or where necrosed pieces of tissue in the deeper parts are cast off and act as foreign bodies (*e.g.* bony sequestra in coxitis, etc. **Figs. 95** and **96**). Similar fistulæ, with an offensive sanious discharge, sometimes result from tampons, drains, or instruments being left behind after operations. Hence very simple and important rules of caution.

In pyogenic lesions which have been insufficiently incised, the presence of unhealthy, purulent granulations shows that the pus has not a free outlet, or that the lesion is extending. When a local pyogenic lesion gives rise to general pyæmia the wound shows similar changes, but the granulations, besides having a dirty yellow appearance, are quite dry.

Treatment

Treatment must be directed to the cause of the fistula. The latter should be laid open freely, and foreign bodies or pieces of necrosed bone removed, after which healing will take place.

Such cases are not suitable for the *Beck* bismuth paste method (see page 192).

In the case represented in **Fig. 56,** the kidney was found to be almost completely destroyed by suppuration. Healing quickly took place after removal of the kidney.

Fig. 57 shows a ***median fistula of the neck*** in a girl aged 19. The fistula first appeared at the age of 15, and was treated by injection and incision, without any result. A drop of secretion is seen at the orifice of the fistula. Radiating cicatrices are also visible. The fistulous track could be felt as a cord as far as the hyoid bone, but its further course could not be made out by injections of fluids. The foramen cæcum was deep. After an incision around the opening of the fistula together with the scar tissue, the track was dissected out. The center of the hyoid bone, through which the track penetrated, was removed, so as to push the extirpation up to the base of the tongue. Microscopic examination showed squamous epithelium in the lower part of the fistula and ciliated, cylindrical epithelium in the upper part.

Fistulæ of the neck are *median* or *lateral.* They may be *complete, blind internal* or *blind external.*

Those we are considering now all result from an arrest in the

Fig. 56. Fistula ex corpore alieno.
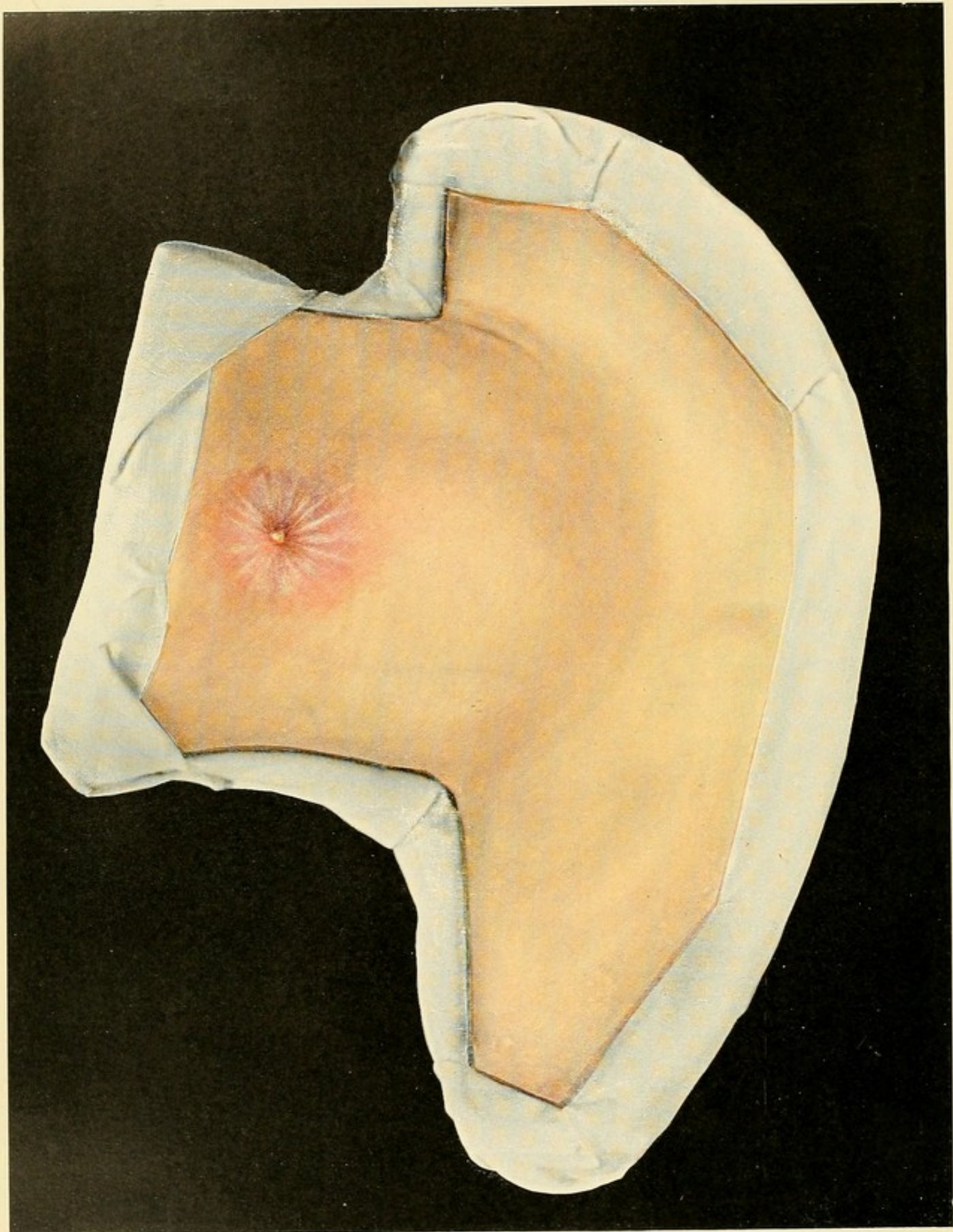
Fig. 57. Fistula colli mediana.

development of the cervical region, and, therefore, are *all congenital;* but some are *complete at the time of birth,* while others (as was the case in the patient of **Fig. 57**) become *complete only by the secondary opening* to the skin of a blind internal fistula. The outer opening of fistulæ of the first variety *always corresponds in position to a point where during development there was a transitory orifice:* the outer opening of fistulæ that have become secondarily complete does not necessarily, and in fact, in most cases, does not, correspond to such a point.

Median fistula of the neck is due to the persistency of the *thyroglossal duct,* which in embryonic life leads from the foramen cæcum at the back of the tongue to the middle lobe of the thyroid gland. In most cases, when complete, it belongs to the class of fistulæ that have become secondarily complete, which fact explains why they are not noticed before a certain age.

Lateral fistulæ of the neck were formerly attributed to imperfect closure of the second branchial cleft, when the existence of genuine clefts was admitted; now that we know that there are no real clefts, but simply thinnings between the thickened branchial arches, lateral fistulæ are ascribed to anomalies in the evolution of the *sinus præcervicalis.*

Heredity is an important factor, found in 25% of the cases.

Median fistulæ open in the midline between the hyoid bone and the sternum: those opening low have been considered as tracheal fistulæ, but there is not a single well-authenticated case to prove the existence of a tracheal communication. There are no median supra-hyoid fistulæ, and embryology shows that there can be none.

Lateral fistulæ generally open along the inner border of the sterno-cleido-mastoid muscle, usually about an inch above the sterno-clavicular joint, and more frequently on the right side. Bilaterality is fairly frequent (22%).

The *outer orifice* is generally button shaped, partly cutaneous, partly mucous (sometimes purely cutaneous and difficult to see). Sometimes its lips are glued together by secretion; sometimes a free drop of secretion exudes from it (**Fig. 57**). If there is much secretion the skin very likely is eczematous.

The *tract* itself may be felt by palpation as a hard, round cord, as thick as a quill pen, directed upward and inward toward the greater cornu of the hyoid bone in case of lateral fistula; straight upward toward the body of the hyoid bone in case of median fistula. In the latter case, the tract passes behind or through the hyoid bone and

ends at the *foramen cæcum of the tongue.* The tract of lateral fistulæ passes below the *facial* nerve, before the *glosso-pharyngeal* nerve and stylo-hyoid ligament, between the *external* and *internal* carotid arteries (that is, between the vessels and nerves of the second and third branchial arches). The upper part of the tract is sometimes, if not always, innervated by the glosso-pharyngeal nerve.

The *internal orifice* of lateral fistulæ is found in a constant position in the tonsillar region.

The *direction* of the tract may be further ascertained by probing: this, however, is generally disagreeable and painful to the patient, and the probe can hardly ever be passed above the level of the hyoid bone; which fact does not prove that we are dealing with a blind external fistula, but that there are kinks in that portion of the tract, because if we inject milk or hydrogen peroxide stained with methylene blue (*Lynch's* method) or sapid solutions, we can often demonstrate the existence of the internal orifice.

Narrow fistulæ cause little trouble to the patient, but in wide, lateral fistulæ accumulation of food may cause inflammation and abscess. Carcinoma may arise from fistulæ and cysts of the neck; it is called *branchiogenous,* as it is derived from the epithelium of the branchial clefts.

In most cases, the history of the case, the appearance of the opening and the anatomical relations of the tract are so striking that the **diagnosis** is made without any hesitation whatever. Fistulæ arising from tuberculous or inflammatory processes differ both in their external appearance and in the course of the fistulous track. In doubtful cases microscopic examination may be made, which will show the epithelial lining absolutely characteristic of a congenital tract.

Treatment

Injections with the object of causing obliteration of the fistula are absolutely useless, and so is incision and scraping. The only rational treatment is *total extirpation of the fistulous tract* through a long incision, bearing in mind the anatomy of the region and the very important connections of the upper part. In lateral fistula it is best to remove the internal orifice together with the tonsil. In median fistula, it is sometimes necessary to remove the middle part of the hyoid bone, in order to follow the track to the foramen cæcum. Recurrence is unavoidable if the smallest part of the fistulous track is left behind. Microscopic examination of both median and lateral fistulæ shows squamous epithelium in distal sections, cylindrical

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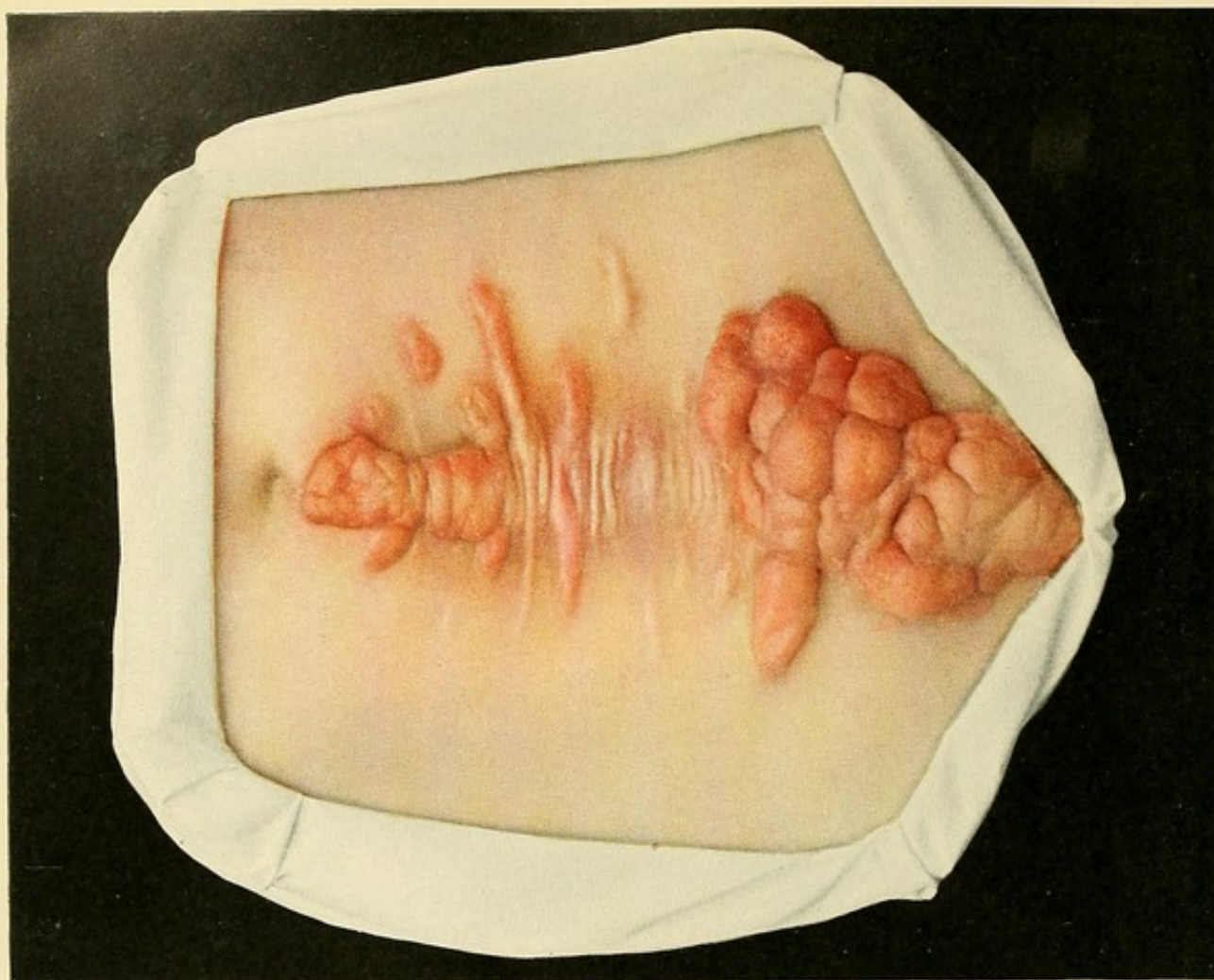
Fig. 59. Keloide post laparotomiam.

Fig. 58. Keloide post vaccinationem.

epithelium in proximal sections. The presence of lymphoid tissue in the wall of the fistula is characteristic.

Figs. 58 and **59** represent two cases of the hypertrophic lesion of scars known as *keloid*.

Fig. 58 shows a keloid which arose on a vaccination scar, in a young girl, and recurred extensively after extirpation. It appears as a large flat growth with radiating processes; smaller nodules are scattered in the neighborhood.

Fig. 59 shows a big nodular keloid developed, in a woman of twenty, in the scar of a laparotomy. Each suture hole has become the seat of a nodule. In the lower part are seen hard, cauliflower-like nodules, freely movable and covered with epidermis.

Keloids, the etiology of which is still little known, are characterized by the formation of homogeneous, fibrous nodes in cicatrices; which nodes consist of hypertrophic scar tissue with thickened blood-vessels. The chief part of the growth consists of dense, hyaline, often interlacing bundles of connective tissue, while cells and elastic fibers are few in number. The papillary bodies are unchanged, but lying under them are nodules or lamellæ, more or less rich in cells. In the lamellar form (**Fig. 58**) there are radial processes at the periphery which are often prolonged as fine processes into the skin.

A *keloid* is a painless tumor of hard consistency, with a smooth, glistening surface, of reddish (**Fig. 58**) or yellowish white color (**Fig. 59**). It is situated in the skin, at the site of a former scar, and movable over the underlying structures. After it has reached a certain size, it remains stationary.

Barring their unsightly appearance, keloids cause no inconvenience. They are absolutely benign lesions; cancerous degeneration is seen only after ulceration. Only if the keloid is very large, and from its situation exposed to repeated pressure and irritation, does pain sometimes occur.

Keloids are more common in young women. Scars of *burns,* ulcers and vaccination are more liable to undergo keloidal evolution. The rôle of infection is not definitely established. There seems to be a local or general predisposition in the individual affected. Some races (negroes and other dark races) have a special tendency to keloid formation. Certain parts of the body are more affected than others: shoulders, face, abdomen, ear.

Diagnosis

The appearance is typical and the diagnosis simple. Merely hypertrophied scars, such as are seen after large infected wounds, and wounds that have been drained for a long time, are not real keloids: they are usually very tender and nearly always flatten out in the course of a few years. But sometimes it is difficult—and maybe the question has but an academic interest—to say where scar hypertrophy stops and where keloid formation begins.

Treatment

It is best to avoid operations, as cauterization and scraping simply increase the keloid, and extirpation, with or without plastic repair of the defect, is almost uniformly followed by a recurrence often larger than the first lesion. (This happened in the case of **Fig. 59**.) Long continued compression, especially of young scars exhibiting a tendency to keloidal hypertrophy, has a certain prophylactic value. Electrolysis or injections of a 10% solution of fibrolysin (thiosinamin) sometimes cause improvement. This latter method was applied to the case represented in **Fig. 59** and a partial disintegration was obtained; but later on recurrence took place.

Bier claims good results from passive hyperemia, and *Kromayer* excellent results from the use of the quartz lamp in keloids.

X-rays, particularly with the single dose method (*Mackee*) are very efficient.

Fig. 60. Contractura aponeurosis palmaris (Dupuytren).

DEFORMITIES DUE TO CONTRACTIONS OF MUSCLES OR TO FRACTURES

Figs. 60 to **63**, inclusive, show *acquired deformities of the hand,* due to *contraction* (or better *retraction*) of various anatomical organs (**Figs. 60, 61, 63**) or to *paralysis* of muscles (**Fig. 62**) due to nerve injury.

Fig. 60 shows a case of *Dupuytren's* **contraction of the palmar aponeurosis** in a man of 50. As is usually the case, the fourth and fifth fingers are more particularly affected. The little finger is markedly flexed and only the last phalanx can be freely extended. The first phalanx of the fourth finger is immobilized in flexion, and the second begins also to feel the effects of the fibrous retraction. There is a very slight incipient involvement of the third finger. Nevertheless, the condition, which had existed for several years, caused so little trouble that operation was refused.

Dupuytren's **contraction** (or, better, *retraction*) is a chronic shrinking of the palmar aponeurosis, i.e., the triangular fibrous structure that continues the palmaris longus muscle, spreads over the palm and sends processes to the proximal phalanges of all the fingers, and is also connected with the skin:

At first, *nodules* develop in the aponeurosis and skin. Later these nodules become **cord-like thickenings,** which are found not only in the palm, but even more commonly on the second, third, fourth and fifth fingers. Contraction of the cords gives rise to an abnormal position of the fingers, immobilization in flexed position of the first and second phalanges, while the terminal phalanx remains movable.

The thumb generally is unaffected; the contraction is often symmetrical. It progresses slowly, so that after some years the finger is completely doubled on itself into the palm and cannot be extended. There is generally some power of extension left in the middle and terminal phalanges, but as motion is painful, it is avoided by the patient. The nature of *Dupuytren's* contraction is unknown. It occurs almost exclusively in men, and hence a traumatic influence was admitted by *Dupuytren.* At any rate it is often found in men in whom the palm of the hand is exposed to continued pressure (post-office

clerks, from stamping, hunters and gun-bearers, carpenters, etc.). The influence of trauma is not accepted by all: nor is that of gout. A trophic nervous origin is the hypothesis most in favor now, because the contraction is often symmetrical and equally developed on both sides, and it sometimes coexists with conditions manifestly of nervous origin (e.g., *Recklinghausen's* disease, for which see **Figs. 67, 68, 69**).

Fig. 61 shows a *hard, slightly movable* **scar,** extending from the palmar aspect of the last joint of the middle finger to the center of the palm: this scar results from an incision made for **suppuration of the tendon sheath** (see **Fig. 93**). The flexor tendon is destroyed, the finger is half flexed and stiff, without any power of motion. Both the *retraction of the scar* and the *destruction of the tendon* contribute to the faulty position of the finger.

Fig. 62 shows the **"claw hand"** attitude observed in injuries of the ulnar nerve. In this case, the cause was a blow on the ulnar side of the wrist-joint, which, perhaps, directly contused the ulnar nerve, and, in addition, caused a hemarthrosis of the joint which pressed on the same nerve.

When a nerve supplying part of the muscles of a given anatomical region is injured, these muscles are paralyzed, and their antagonists immediately become preponderant, and produce in the region affected (this is particularly plain in the limbs) a *deformity, always the same for the same nerve;* thus the typical deformity of ulnar *"claw hand"* (**Fig. 62**) is due to the paralysis of the interossei muscles, whose function is to flex the first phalanges: consequently in ulnar "claw hand," we find the first phalanges of the fourth and fifth fingers *hyperextended,* while the second and third are flexed. The other fingers are not in claw position, although their interossei are paralyzed, because their lumbricales (supplied by the median nerve) are still active.

In the case of **Fig. 62,** there was slight swelling on the back of the wrist joint, chiefly on the ulnar side. Fluctuation was present. The sign of *"snowball crunching"* indicated the presence of hemarthrosis. Motion in the joint was limited and painful; it was in slight flexion (the natural position of the wrist joint in case of intra-articular effusion), but could easily be extended.

Fig. 63 shows a *very important type of* **retraction of the flexor muscles of the forearm,** that consecutive to too prolonged **ischemia**

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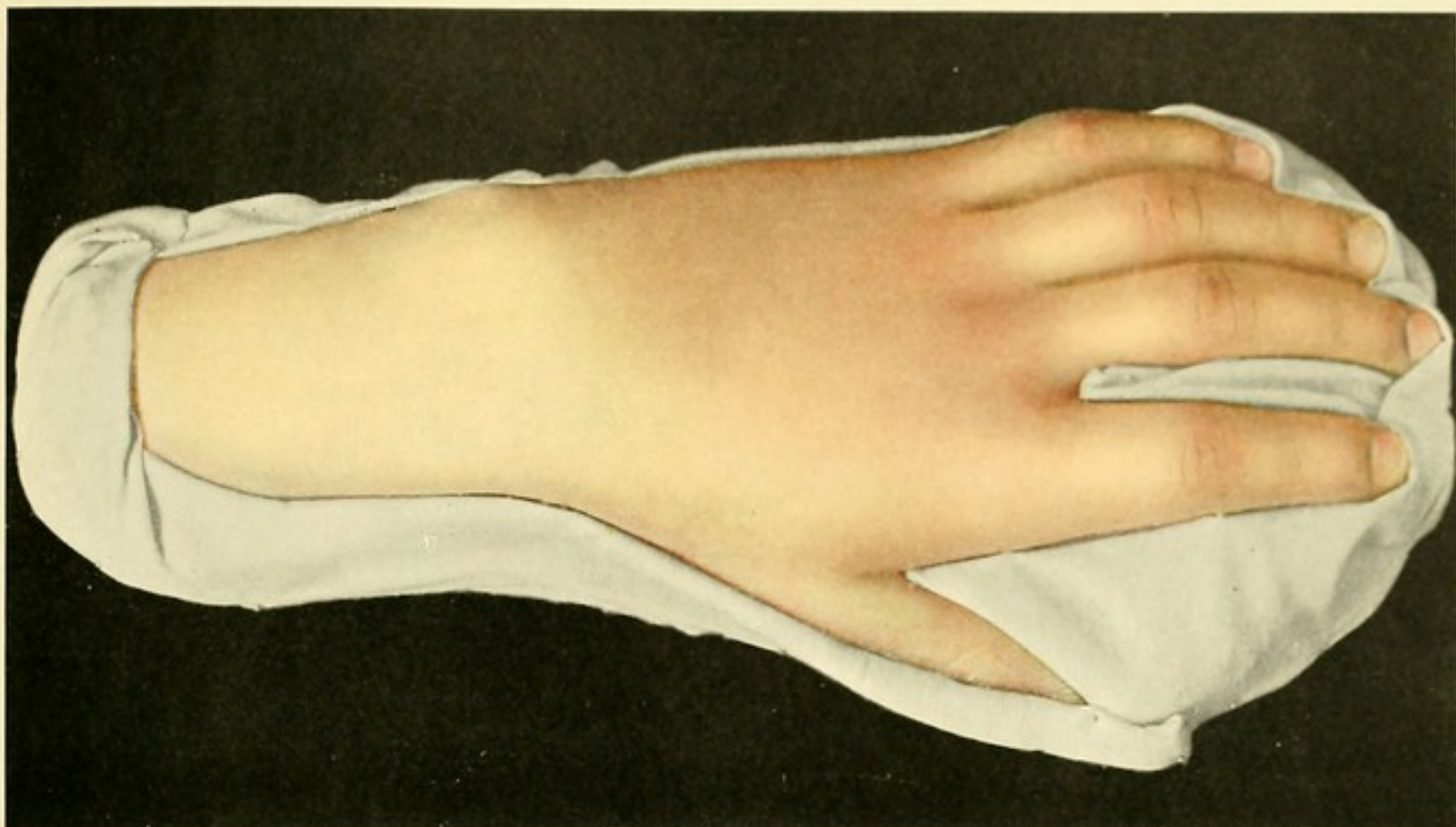
Fig. 62. Haemarthros — Compressio N. ulnaris
Neurogene Kontraktur
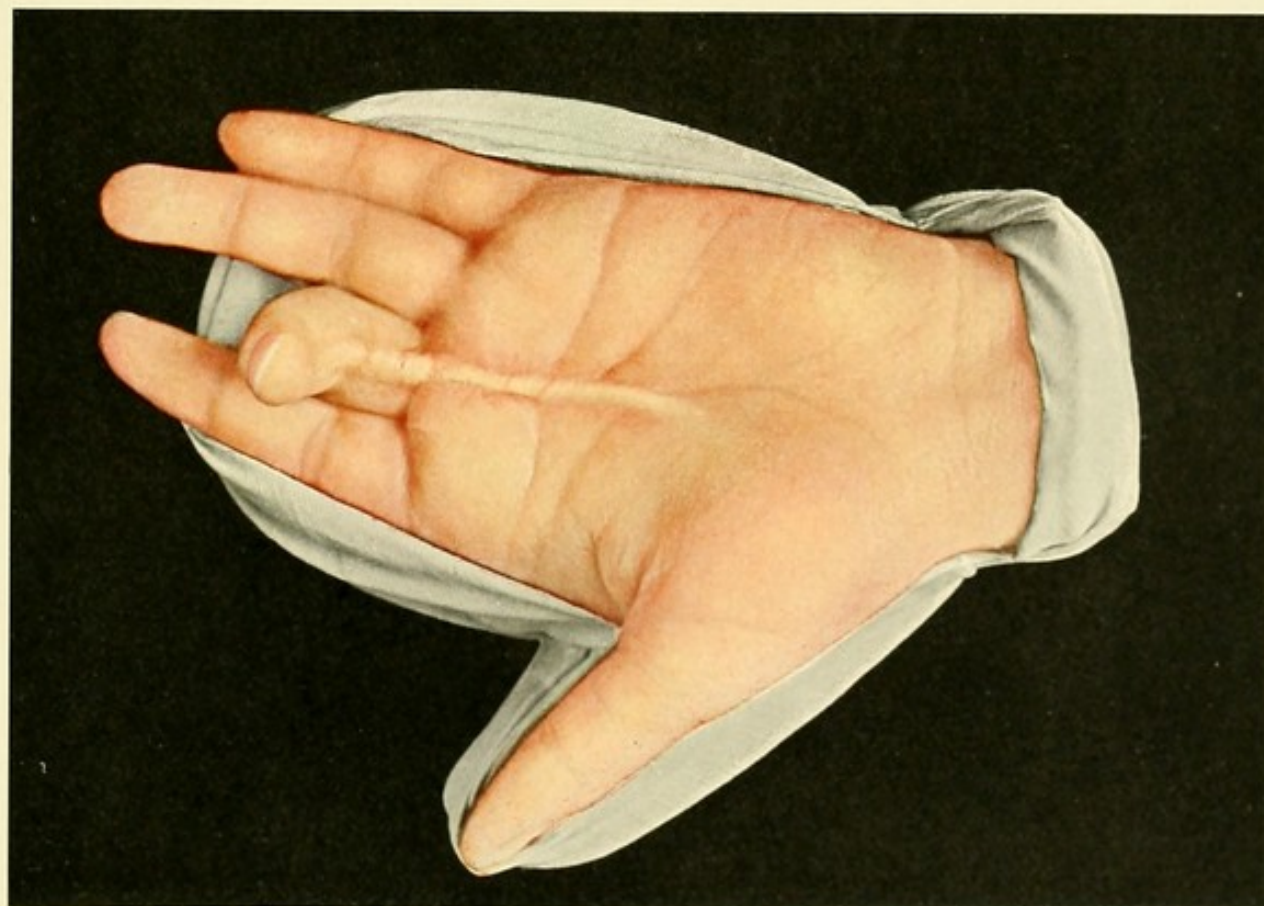
Fig. 61. Contractura post panaritium tendinosum.

Fig. 63. Contractura ischaemica.

of the same muscles. The practical importance of this condition lies in the fact that it may be foreseen and foretold in some cases where it is hardly avoidable, and is avoidable, in a great majority of instances, through the observance of simple rules of caution; while, once it is established, it is hardly curable. It can only be partly improved by surgical interference and patient after-treatment.

This post-ischemic retraction was first described by *Volkmann* under the name of *ischemic paralysis;* it was later studied by *Lexer* and other German authors. Of late years, American cases have become quite frequent. *Thomas* has well studied the implication of nerves in this condition, and American surgeons (*Freeman, Huntington, Powers*) have been conspicuous in the development of surgical methods of treatment.

These ischemic contractures are *myogenous* and appear when the blood supply of the muscle has been cut off for too long a time: this induces a special degeneration of the muscle fibers, which is followed by sclerosis and retraction. Muscular tissue stands ischemia less well than the skin, because the arteries of muscles are terminal.

The causes of muscular ischemia are manifold, but are met only in traumata of the limbs. It may be an *injury to the main artery,* a complete laceration with hematoma, or simply an abrasion of the intima in one point followed by thrombosis and obliteration of the artery, or *too tight a constriction by a tourniquet,* or *long exposure to cold;* but by far the most frequent cause, the one that has most practical importance, and the only one known to the authors who first described ischemic contracture, is the **application of too tight a bandage around a fractured limb.**

Fractures of the upper limb, and particularly of the lower end of the humerus, are those after which ischemic retraction has been most often seen. Next come fractures of the bones of the forearm. Out of about 200 published cases only one pertains to the lower limb; and strange to say it is the original case from which *Volkmann* individualized the type of ischemic "paralysis." The predominance in the upper limb is explained by the lesser bulk of the muscles in that region, and the frequency with which the flexors of the fingers are affected is quite naturally explained by the flat surface of the forearm and the rigid frame constituted by the two bones of the forearm on which the flexors are directly applied.

The more common occurrence of ischemic contracture in younger individuals is due to the greater compressibility of their muscles and vessels. In order to produce contracture, the ischemia must be

marked enough to cause irretrievable lesions, but not sufficient to cause gangrene. The latter condition is what happens in older persons (whose blood vessels are sclerotic), either as a result of strong pressure, or of obliterative thrombosis after slight pressure. Unfortunately, it seems much easier to obtain the necessary degree for ischemic contracture unwillingly under a bandage applied for a fracture than to gauge it accurately in experimental work. Up to the present time, ischemic retraction, fairly frequent clinically, has not been reproduced experimentally.

A few hours after the application of a bandage around a fractured limb, the patient begins to feel great pains and numbness of the fingers. These become blue, swollen, incapable of active motion, while passive motion is painful. The fingers are flexed. If the bandage is then removed, the skin appears white, while the muscles feel as hard as board and are incapable of motion (hence the appellation "paralysis" given at first). If the lesions are not yet too marked, recovery may take place. But if the bandage has been left too long, the muscles become the seat of a very painful swelling, rapidly developing soon after it is removed; the fibers are dead and later are replaced by fibrous tissue, which forms hard lumps in the muscular body, and progressive retraction sets in. The skin of the fingers gradually becomes yellowish-white like parchment. The swelling of the fingers is followed by shrinking. First of all the fingers, then the metacarpal bones, and finally the wrist become fixed in a position of flexion. The fingers are eventually so strongly flexed that the hand becomes useless. The movements of the wrist are also very limited, and the muscles of the forearm become atrophied and are covered by pale skin. Sensory disorders may occur from pressure of the shrunken muscles on the nerves, and the implication of nerve trunks in the sclerotic masses is always marked in severe cases. But this nerve involvement, although important and calling for a particular treatment in many cases, is always *secondary* in *Volkmann's* contracture.

The disease has no tendency whatever to spontaneous improvement. Only an energetic and patient treatment can give hopes of a partial cure.

Diagnosis of deformities due to muscular action in the upper limbs

Each one of the types shown is sufficiently characteristic to avoid mistakes.

Dupuytren's **contraction** is seen in *middle aged* or *elderly* people, *develops slowly* and *gradually,* and there are palpable *nodes* and

thickenings in the palmar aponeurosis. *Fibromata* or *cysts* of the tendon sheaths (see **Fig. 49**) are easily differentiated from these thickenings, because they are generally single, well limited, and round in shape, while the process in *Dupuytren's* contraction is diffuse.

Cicatricial contractions are always easily diagnosed, owing to the history of the disease and the presence of an always very visible scar. The only point is to determine how much the scar is at fault, and how much the tendon.

Ankylosis of the finger joints, such as is observed in *gouty* individuals (see **Fig. 141**) are recognized by the antecedent history. Moreover, the joints alone are involved.

Volkmann's **ischemic contraction** is seen in *young individuals*, after a *trauma*, and after the application of *a tight bandage*. The clinical succession of phenomena is so typical that the diagnosis can be made at once, if only one thinks of it. A very important point, however, and one absolutely essential to a judicious treatment, is determining exactly the respective shares of the muscle degeneration and of the secondary implication of nerves.

Deformities of neurogenous origin are usually typical enough to be easily recognized. The difficult point is to ascertain the cause. The condition may be due to a *central lesion* (hemiplegia, Little's disease, poliomyelitis), and an examination of the whole nervous system is often necessary to settle the diagnosis. However, in a general way, we may say that a *myogenous lesion* is *fixed* and *unchangeable* while *central contracture slightly varies from day to day* and *disappears* under general anesthesia.

Again, the condition may be due to *local lesions* along the course of the nerve. Among the latter, we shall only mention a few: (1) **callus of fractures of the clavicle** pressing on the brachial plexus; (2) **supernumerary cervical ribs,** well studied in America (*Keen, Sherman*), and the possibility of which must always be borne in mind in ulnar "claw hand," and in writer's cramp; (3) *old and recurrent* **dislocations of the shoulder joint** with radicular lesions of the brachial pleuxus; (4) **habitual forward dislocation of the ulnar nerve** *over the tip of the epitrochlea* (*Cobb*). X-ray examination in these cases is invaluable; it ought to be performed in all cases of pressure symptoms on the nerves of limbs.

Treatment of muscular and fibrous retractions

There is no effective mechanical and massage treatment susceptible of stopping the progress of *Dupuytren's* **contraction.** In severe

cases, *Kocher* advises excising all the affected parts of the palmar aponeurosis and of the neighboring skin when it is involved; the loss of substance is repaired with skin flaps. Massage must be begun shortly after the operation. Injections of fibrolysin give only doubtful results.

The conservative treatment of **cicatricial contractions** such as that represented in **Fig. 61,** where both the skin and the tendon are involved, is not very successful, because of the impossibility of making a new serous sheath around the tendon. After excision of the scar, contracture occurs in the new cicatrix, in spite of extension of the finger, lengthening of the tendon, transplantation of tendon or catgut, or plastic operations. If the patient is incapacitated from work by the contraction, exarticulation of the finger is the best policy.

In cases of contractions limited to the skin, such as those after cuts and burns, keloids, superficial suppuration, etc., the prognosis is much better. Excision of the scar, extension of the finger, in some cases lengthening of the tendon, and repair of the wound by skin flaps, can restore the function of the finger. In young persons good results are obtained by orthopedic treatment, when the scar is not very extensive, nor hypertrophic, nor of too long standing.

In **contractures of central origin,** especially in the paralytic contractures due to anterior poliomyelitis, nerve transplantation, and shortening or transplantation of tendons may be performed. Treatment by massage, electricity and orthopedic apparatus is also useful.

In **contractures of peripheral origin,** operative interference, *free exposure* and *removal of the compressing agent* is successful if the condition is not so old that the nerve is hopelessly destroyed: and even then it may regenerate in time.

By far the best treatment of **ischemic contraction** is prophylaxis. *No apparatus that causes swelling, cyanosis of the fingers, and pain should be left in situ for any length of time.* The patient's complaints must be heeded.

In every fracture, a *careful examination must be made to detect injury to the blood-vessels or nerves,* lest they later should be unjustly charged to neglect or incompetency of the attending surgeon.

Mild cases of ischemic retraction may be improved, almost cured, by long-continued orthopedic treatment (*Jones, Sayre*).

The other forms call for surgical interference: *Tendon lengthening* is preferred in England, *resection* of about an inch and a half of the ulna and radius (which has the same effect as tendon lengthening, but is less complicated technically) is preferred in Germany. In

Tab. XLIX.

Fig. 64. Hallux valgus — Hammerzehe — Arthrogene Kontraktur.

America, surgeons lean to *bone resection*, and justly devote considerable attention to the *condition of nerves:* neurolysis, freeing of the nervous trunks from the sclerosed masses of tissue and transposition above the fascia are necessary when there is nerve implication.

After-treatment, massage, electricity, is very important and must be kept up for a long time.

Fig. 64 shows the *outward deviation* of the big toe called **hallux valgus.** The second toe is affected with the deformity known as **hammer-toe.** Besides, there are the usual sequelæ of these conditions, namely, *bunions* and *corns;* and also dry eczema of the foot. All of which, coupled with a considerable degree of flat-foot, made walking very difficult and troublesome.

The deviation of hallux valgus is generally attributed to *too pointed shoes.* However true in most cases, this explanation does not hold for those cases of hallux valgus seen in young children or in peasants and workingmen who never wore tight pointed shoes. Heredity may here play a rôle. *Contraction of the extensor hallucis* maintains the deformity, once it is produced, and so do *changes in the metatarso-phalangeal joint* (atrophy, inflammation, arthritis deformans), so that in advanced cases reduction of the deformity is no longer possible without operation.

The deviation of the big toe may be as much as 50°, so that the toe crosses the second, generally *over* (**Fig. 64**), sometimes under the latter. Over the projecting metatarso-phalangeal joint, a *bunion* (inflamed bursa) develops, also *corns* (**Fig. 64**), while the head of the metatarsal bone is the seat of a *marked hyperostosis* on the inner side, so that the articular surface instead of being terminal and symmetrical, occupies only the outer half of the head of the bone. The bunions may suppurate and open externally (**Fig. 64**). An *ingrowing toe-nail* (compare **Fig. 99**) generally develops on the outer side of the great toe (**Fig. 64**).

Hammer-toe is characterized by *hyperextension of the first phalanx* of the toe, while the two last are flexed; the result is that the whole pulp of the toe presses directly on the ground; a corn always develops there (**Fig. 64**), and, on account of pain, walking becomes difficult. Hammer-toe has been attributed to contraction of the extensor hallucis; but it more likely is a condition analogous to *Dupuytren's* contraction (**Fig. 60**) in the hand. Owing to its innocuousness, and the ease with which it can be simulated, it has been

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a favorite deformity for malingerers who want to avoid military service. However, an artificial hammer-toe is only a forced incurvation of the two last phalanges, without the hyperextension of the first, always present in genuine hammer-toe. The latter usually affects the second toe (**Fig. 64**).

Subungual exostoses (**Fig. 140**) also occur in these cases.

Prophylactic treatment of all such deformities consists in attention to the feet, baths, cutting the toe-nails straight instead of curved, wearing properly fitting boots, etc.

When recent, hallux valgus may be straightened by the long-continued use of splints. But, if old, if changes have taken place in the joint, cuneiform osteotomy of the metatarsal head gives the best results.

Hammer-toe is very refractory to treatment. Splints, tenotomy, resection may be attempted; but exarticulation will be needed in most of the marked cases.

Exostoses are chiselled off; subungual exostoses require preliminary removal of the nail.

(For the treatment of corns, see page 141.)

In the case represented in **Fig. 64,** cuneiform osteotomy of the metatarsal head was performed; the bunions and corns were excised, the second toe exarticulated, and the eczema treated with *Hebra's* ointment. The functional result was excellent.

Fig. 65 shows a case of **rachitis,** affecting the whole skeleton in a girl, aged 4, with marked incurvation of both legs.

Rachitis, or *rickets,* is a disturbance of growth affecting the whole skeleton. Owing to faulty nutrition (the exact causes of which are not as yet fully known, but in which fat deficiency and carbohydrate excess in the food seem to play an important part), and, maybe, owing also to some racial predisposition (negroes, Italians), the lime salts necessary for the normal growth of the bone are not properly assimilated and the bones soften. In the epiphyses, there is abnormal proliferation of cartilage and at the same time imperfect ossification of the latter, leading to the formation of *osteoid* tissue. This results in the development of the epiphyseal "*beads,*" pathognomonic of rickets, and considerable deformity in all parts of the body.

In the *skull* the disease affects chiefly the frontal and parietal bones. In extreme cases the bones, particularly the occipital, are soft and flattened, and yield to pressure (*craniotabes*). In other places,

Fig. 65. Rhachitis. — Infractiones cruris utriusque.

especially the frontal and parietal eminences, the bones are thickened and prominent, giving the so-called "*Olympic forehead.*" The cranial sutures and fontanelles remain open far longer than normal; the upper and lower jaws are irregularly developed and flattened and the implantation of the teeth is abnormal and irregular.

The weight of the body causes bending of the softened bones; the spine becomes *kyphotic* or *scoliotic;* the antero-posterior diameter of the thorax increases, while the transverse diameter decreases; the sternum is projected forward (*chicken breast*) or, on the contrary, sunken. There may be a constriction (*rickety girdle*) in one point of the thorax, the upper part of which seems narrow, because the costal margin is spread out and widened. The junctions of the cartilage and bone of the ribs become thickened (*rachitic rosary*).

The pelvic bones remain small, and *rachitic pelvis* in women is a frequent cause of dystocia. Lastly, in severe cases, or if the child is allowed to walk during the period of softening of the bones, the lower extremities become bent, and the bones are liable to greenstick fractures. Both conditions existed in the little patient of **Fig. 65.** *Genu valgum* is the most frequent deformity: *genu varum* is sometimes observed. *Flat foot* is also a result of rickets, and so are most cases of *coxa vara.* In severe cases, the subjects may be permanently dwarfs.

The disease begins with anemia, digestive disturbance and diarrhea, *sweating of the head,* and marked bulging of the abdomen; it tends to recovery after a time, when normal bone replaces osteoid tissue. In many cases, no trace is left; in others, some of the above-mentioned deformities persist for life.

The *prognosis* of rickets is favorable *per se;* but rickets nevertheless remain an important factor of infantile mortality, owing to the physical and mental backwardness of the subjects affected, of their lesser resistance to intercurrent infections, and to the greater gravity of pulmonary and cardiac affections in those whose thorax is deformed.

Diagnosis and Treatment

There is usually little trouble in recognizing rachitic children, with their characteristic facies, pot belly, and epiphyseal heads.

Hereditary syphilis (which *Parrot* thought the main cause of rickets, a view untenable nowadays) affects fewer bones, particularly the tibia, and bone-lesions are almost always associated with other signs of congenital syphilis (interstitial keratitis, *Hutchinson* teeth, positive *Wassermann* reaction).

Pott's **disease** differs from rickety scoliosis by the presence of a sharp angular bend, instead of the long curve of the rachitic deformity; besides there are points painful on pressure and a marked rigidity of the diseased segment, and no other bony deformities.

Osteomalacia is a softening of the bones, seen particularly in women, in adult life, never in young children.

Treatment

The treatment of rickets at first is purely *dietetic* and *hygienic;* correct feeding, fresh air, salt baths, and, maybe, phosphorus.

The surgical treatment of rachitic deformities must never be undertaken before the process has stopped and spontaneous recovery and straightening of bone has progressed as far as it will go. To ascertain this, X-ray examinations are useful; in active rickets the epiphyseal lines appear wide and irregular, sometimes with incomplete fractures; while, when the disease has come to a standstill, the epiphyseal lines have become regular, and the cortex appears as thick as the deeper parts.

Rachitic deformities, being quite varied, may call for a number of operative procedures, on which we do not intend to dwell in detail here. Genu valgum and too marked incurvation of the tibia, as shown in **Fig. 64,** indicate *cuneiform osteotomy.*

Fig. 66 shows a **pseudarthrosis,** an *ununited oblique fracture of the tibia above the malleolus,* in a man 60 years of age. Although the injury dated back two years, the distal part of the tibia was freely movable with the foot. The X-rays showed an overlapping of the fragments and a united fracture of the distal end of the fibula a few centimetres above the external malleolus. The nature of the injury had not been diagnosed.

The causes of delayed union of fractures may be *general* (rachitis, syphilis, tuberculosis, pregnancy, old age) or *local* (infection in compound fractures, interposition of soft parts, periosteum or muscle).

Pseudarthrosis occurs in the leg, chiefly after oblique fractures with dislocation or comminuted fractures; in the thigh (neck of the femur) and upper arm (humerus) after transverse fractures also. In the arm, interposition of soft parts is the chief cause. In fractures of the femoral neck, particularly those of the intracapsular variety, non-union was especially frequent, and it was very generally blamed in former days on the old age of the patients, but *Whitman* has shown
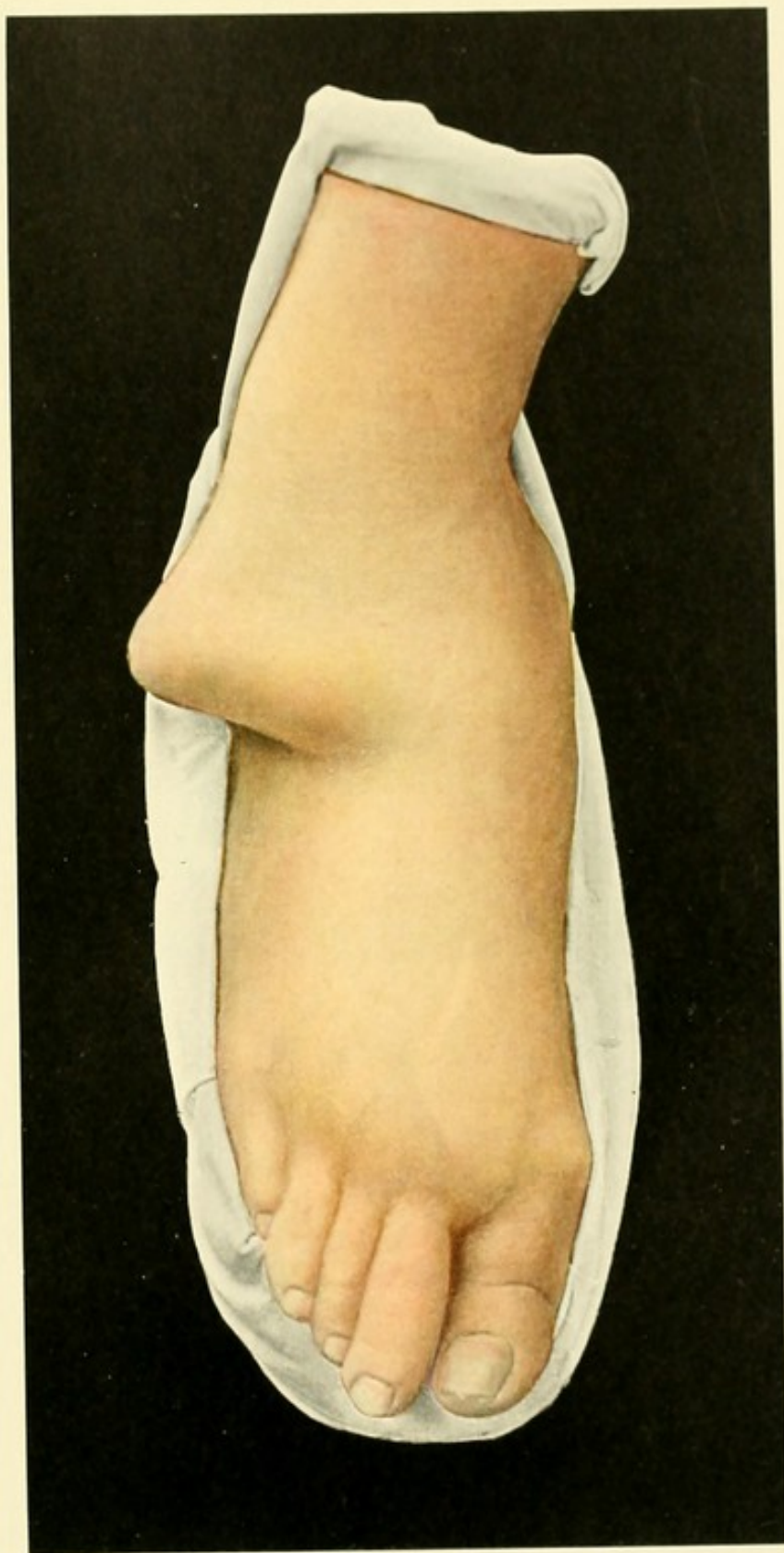
Fig. 66. Luxatio cum fractura cruris — Pseudarthrosis.

conclusively that the rôle of senile rarefaction of the bone has been considerably exaggerated; that most of the pseudarthroses were due to the fact that the accepted treatment *did not bring the two fragments* in exact apposition; that, if the extreme abduction treatment is applied, coaptation is obtained, and bony union follows, even in old people.

This emphasizes the necessity, in cases of non-united fractures, of carefully analyzing all data, and of ascertaining all local conditions, before we have a right to attribute non-union to a general cause. In this respect X-ray examinations are invaluable.

Treatment

Simple delayed union may be accelerated by passive hyperemia, *Bier's* blood injection between the fragments (*Kiliani, Lyle*), and, perhaps, by the internal administration of calcium salts.

Carrel's recent work shows that bone reparation may be considerably hastened by the direct application on the line of fracture of pulp of glandular parenchyma, chiefly of the thyroid gland.

Non-union depending on local conditions calls for a correction of the latter: interposed soft parts must be removed, bone ends must be freshened and brought in correct position, and maintained there, either by a plaster of Paris cast, or by *plating*.

The operative treatment of fractures has been much advanced in late years; *Lane's* plates (or better, the modified vanadium steel plates of *Sherman*), when properly used, give excellent results in ununited fractures, or in those in which the displacement cannot be corrected by the other methods of treatment. A strict asepsis is necessary; hence the operative treatment of fractures is not applicable in cases of non-union in infected compound fractures of long standing.

In old fractures, and in those near joints, resection may become necessary to insure reduction.

When syphilis is suspected, mixed treatment or salvarsan should be administered.

NAEVI

Figs. 67, 68 and **69** show *naevi* and a condition of cutaneous nerves sometimes found in conjunction with them.

Nævi are developmental defects of the skin characterized by an excessive growth of *pigment* (*pigmentary nævi*, **Figs. 67** and **68**) or of *vascular tissue* (*vascular nævi*, **Figs. 75** and **76**).

Fig. 67 shows a large pigmentary hairy nævus, which was present at birth and increased in size till the age of puberty. The borders are smooth, but the central parts are warty (*nævus verrucosus*). The color is blackish brown in the center and brown at the periphery.

Fig. 68 shows a slightly pigmented nævus extending over most of the forearm, with a bluish-red, irregular elevation in the center. This nævus was present at birth. Small pigment spots were scattered over the whole body. The bluish-red elevation in the centre was formed by a *fibroma of the sheath of a large subcutaneous nerve*. In addition, there were fibromata along the course of the principal nerves of the arm (*neuro-fibromata*) (on the upper arm and the axilla, of the size of a walnut) and multiple cord-like formations (*plexiform neuroma*) could be felt under the nævus. There were also small soft nodules in the skin (*fibromata mollusca*). All these formations had appeared later than the nævus, but had been present many years.

Fig. 69 shows a similar condition in a girl aged 20.

The whole of the right half of the scalp, the right side of the forehead and the ear are the seat of a lobulated growth (*elephantiasis nervorum*) fixed on the head like a cap. The growth was congenital, and on its surface are numerous pigment spots and soft, small, painless tumors (*fibromata mollusca*). Numerous cord-like formations were found in it by palpation (*plexiform neuroma*). This is a typical location for the disease; the nape of the neck and the back are also affected.

Fig. 67. Naevus pigmentosus pilosus.

Fig. 68. Naevus neuromatosus — Neurofibroma cutis.

As seen from the preceding description, various names have been bestowed upon this condition about the etiology of which, barring *congenitality, hereditary predisposition,* and a possible influence of trauma and repeated irritations, nothing is known: *elephantiasis nervorum, fibroma molluscum, plexiform neuroma, generalized neuro-fibromatosis.*[1] None of these names is absolutely adequate for the whole of the disease, but each is partly justified by some of the anatomical alterations found, as we shall presently explain.

Neuro-fibromatosis is, perhaps, the best name, because the chief lesions are multiple small fibromata along the course of superficial nerves. However, these are fibromata *of the nerve-sheaths,* for which the term *neuro-fibroma* is not altogether proper, because they consist of fibrous tissue only, without any proliferation of nerve-fibers. These multiple, small fibromata are generally disseminated over the whole body, forming small, soft, subcutaneous tumors when they affect the fine cutaneous nerves, and are associated with numerous pigment spots. The small tumors may lie so closely together that the skin assumes a finely lobulated appearance (temples (see **Fig. 69**), neck and back). This condition has been termed *elephantiasis nervorum,* because with the fibrous tissue formation there are numerous lymphatic vessels.

Besides, in distinction to these small, soft, multiple fibromata, there may be fibromata of the larger nerve trunks, which appear as hard fusiform tumors of the sheaths of the cutaneous (**Fig. 68**) or subcutaneous nerves. They are very painful on pressure and may cause functional disorders (paresthesia, for instance).

In addition to these two forms of fibroma, there may be in other points true neuromata, which resemble cirsoid aneurism, hence the name of cirsoid or plexiform neuroma. These are formed of twisted cords, which may form an inextricable network of nerve cords and contain nerve fibers, while the above-mentioned types of lesions do not, despite the name bestowed upon them.

Diagnosis

The diagnosis of nævi is a very simple matter.

Von Recklinghausen's disease, with its multiple elements, is so characteristic that no hesitation is possible.

Treatment

Nævi on exposed parts of the body should be *excised,* for cosmetic

[1] Or, simply, *von Recklinghausen's disease,* because this writer was the first to draw the attention to the relation between disseminated pigment spots, certain lesions of peripheral nerves and mental backwardness.

reasons. Removal of all nævi showing inflammation or a tendency to degeneration is also indicated (about melanotic tumor evolution, see **Figs. 17, 23, 28,** and pages 20, 25, 36).

Electrolysis may be used in small nævi. But we must guard against methods that do not destroy thoroughly, because they simply irritate and promote malignant changes. X-rays are sometimes beneficial. *Pusey* has found freezing with carbon dioxide snow (see farther, about vascular nævi, page 102) a very good method.

Nævus neuromatosus should only be excised when it shows papillomatous proliferations or when fibromata or a plexiform neuroma are situated beneath it.

Isolated fibromata of the nerve sheaths can generally be excised without injuring the nerve; but in large fibromata the nerve may have to be resected, with subsequent suture. Recurrence is rare.

Multiple fibromata are apt to recur after operative interference. They should be removed only when rapidly growing, as they may then undergo sarcomatous or myxosarcomatous proliferation.

Plexiform neuromata must be completely extirpated, as recurrence takes place if any part is left behind. At the same time, the thickened skin should be removed if it shows elephantiasic changes. In extensive cases the operation may be done at several sittings. This was done in the case represented in **Fig. 69.** In that of **Fig. 68** a very painful fibroma in the axilla was first removed, and the nævus neuromatosus was excised later.

Fig. 70. Rhinophyma — Akne rosacea.

Fig. 69. Elephantiasis nervorum — Fibromata mollusca.

LESIONS OF THE LYMPHATIC AND VASCULAR SYSTEMS

Fig. 70 shows, in an old man, the chronic congestion of the flush area of the face with acne lesions and dilatation of skin capillaries, which constitutes **acne rosacea;** and the irregular, lobular thickening of the nose, which is called **rhinophyma** and is the result, in some cases, of long-standing rosacea.

Rosacea is an *angioneurotic disturbance,* generally depending on faulty diet or metabolism (alcoholism, dyspepsia). When the pink coloration of the nose and adjacent area has existed for some time, *acne lesions* develop, and permanent telangiectases. Finally, the hypertrophic thickening of the skin, at first regular, is modified by the *development of large sebaceous follicles,* with dilated pores, from which yellow secretion can be expressed, which finally cause *rhinophyma*. Once they are developed, rosacea and rhinophyma have no tendency to regression: they cause no subjective symptoms. This condition is more frequent in old people.

Diagnosis

Rosacea is easy to recognize from *simple acne* by its limitation to a special area of the face.

Rhinophyma is a typical lesion with its dilated orifices of sebaceous follicles, and the greasy appearance of the skin due to exaggerated secretion of these follicles.

Rhinoscleroma causes softer tumors, which soon ulcerate, and may destroy the whole face.

Lupus is distinguished by its apple-jelly nodules, ulceration and scarring.

Tubercular syphilides of the nose (**Fig. 120**) are less symmetrical and ulcerate.

A *pachydermatous condition* may result from repeated attacks of erysipelas (see page 129), but differs from rhinophyma in not affecting the nose any more than the rest of the face.

Treatment

The correction of all dietetic errors is important in rosacea. Washing the face with lukewarm water and soap, and applying lotio alba

is useful. Telangiectases are treated as will be said later (page 102). X-rays favorably influence the hypertrophy of the skin and sebaceous follicles. However, when rhinophyma has attained the development shown in **Fig. 70** *decortication* of the nose with the knife is the only treatment. Owing to the very abundant blood supply of the region, and the hypertrophy of the epidermis, the wound heals very promptly, the scarring is trifling and results are excellent.

Fig. 71 represents a case of *elephantiasis of the penis,* in a man of 40.

Elephantiasis (or *pachydermia*) is a chronic, diffuse hyperplasia of the skin and subcutaneous tissue due to persistent obstruction of the lymph channels. In the case shown in **Fig. 71,** the obstruction had been caused by bilateral extirpation of the inguinal glands. The swelling had begun soon after the operation and had progressed slowly, the chronic course being interrupted several times by acute exacerbations, which subsided after a few days' rest in bed.

The thickening of the skin was not uniform, but lobulated. It felt soft and spongy. In such cases there is a marked fibrosis of the subcutaneous tissue and dilatation of the blood and lymph-vessels, the latter being the primary factor.

The skin was pigmented and the scrotum covered with crusts, and there were numerous depressions, as in rhinophyma. There was no pain.

In tropical countries, elephantiasis results from a specific cause, namely, the blocking of lymphatics by the *filaria sanguinis hominis* and kindred parasites. In our climate, elephantiasis is *not a specific disease,* but only a symptom, which depends on manifold causes; chronic edema, or eczema, recurrent erysipelas, syphilitic and tuberculous lesions, varicose ulcers, phlebitis and thrombosis of veins, and pyogenic infections of the skin. We have already mentioned double extirpation of the inguinal glands (**Fig. 71**). A double radical operation for hernia has sometimes had the same result.

The legs are the parts most frequently affected; next come the male and female genitalia. In prostitutes, the labia, clitoris and perineum sometimes become affected with elephantiasis from gonorrheal discharges and syphilis.

The tissues feel at first soft, but afterward become firm and elastic. Parts having undergone pachydermic alterations are more liable than healthy tissue to intercurrent infections. Repeated attacks of

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Fig. 71. Elephantiasis penis lymphangiectatica.

Fig. 72. Ulcus cruris varicosum — Elephantiasis, Pachydermia acquisita.

lymphangitis are common, after each of which there is an increase of the elephantiasis. Eczema, bullæ, pigmented spots, scabs and crusts, condylomatous or papillomatous proliferations, or, finally, ulceration, may occur on the surface. Ulceration causes intolerable suffering: otherwise, the condition is painless and causes inconvenience only by its weight or when it prevents walking on account of its size.

Diagnosis

It is hardly necessary to dwell on the differential features existing between ordinary elephantiasis and the so-called "*elephantiasis nervorum*" of *von Recklinghausen's* disease (**Figs. 68** and **69**). The other symptoms of the latter condition, fibromata, neuromata, disseminated pigment spots, do not leave room for any hesitancy.

In *partial giantism* there is an overgrowth of all the tissues, including the bones, dating back to early infancy.

The recognition of the cause is important. The diagnosis of endemic elephantiasis is settled by the detection of the parasite in the blood taken at night.

Treatment

All lesions of the inguinal glands, especially if bilateral, must receive prompt attention in order to avoid lymphatic obstruction. All conditions enumerated above, susceptible of leading to pachydermia, must be carefully treated.

Recent and light cases may be improved by elevation, massage and compression. More extensive cases have to be treated by cuneiform excision. This was performed in the case shown in **Fig. 71.**

Fig. 72 shows an ***elephantiasic thickening of the toes*** developed in connection with a ***varicose ulcer*** of the leg. The toes are enormously thickened and constricted in places; the whole foot is also enlarged and the arch of the foot is obliterated. The thickening of the foot continually increased and extended to the ankle. Frequent attacks of erysipelas aggravated the affection.

The varicose ulcer is situated on the inner side of the leg, at its lower third, and extends nearly over the whole circumference.

Varicose ulcers occur generally in old people of the poorer classes, who cannot take proper care of their varices and have to do much standing; uncleanliness makes matters worse. All diseases of the nervous system and arteriosclerosis may cause the development

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of trophic ulcers (see **Fig. 138**), which are not unlike varicose ulcers, but are still more refractory to treatment than the latter.

Varicose ulcer of the leg is characterized by its irregular, slightly raised edges, while the surrounding parts may be covered with scattered, flabby granulations, crusts and blood-scabs (**Fig. 72**). Around the base of the ulcer are fine, bluish, dilated veins, from which frequent bleeding takes place. The ulcer is often connected with a ruptured varicose vein.

In small ulcers *temporary healing* may take place, but the scar is very thin, generally pigmented, and gives way again on the slightest cause; after which no further healing usually takes place, but the ulcer continues to extend. The whole neighborhood of the ankle joint, and even the whole leg, may be involved in ulceration, which often has a sanious discharge. In extensive ulcers there is generally severe pain and the leg becomes more or less useless owing to the extent of the ulcer and the elephantiasis.

Differential diagnosis

Large ulcers with sanious discharge may suggest **carcinoma,** owing to their hard borders, but in carcinoma there are always irregular, hard-tumor masses in the whole extent of the ulcer. The possibility of transition of an ulcer of the leg into carcinoma must be borne in mind (see **Fig. 20**). In doubtful cases, always excise a piece of the indurated edge for microscopical examination.

Gummatous ulcer, frequent on the leg, is more regular, often circular, and has a punched-out appearance. The base of the ulcer is smooth and covered with a tenacious, yellowish, fatty core. The ulcer is generally less extensive and there is no bleeding. Anamnesis and the *Wassermann* reaction may clear up the diagnosis. The tibia may show some of the characteristic features of gummatous osteitis.

Tubercular ulcers are rare on the leg. However, their thin, undermined edges, and particularly anemic appearance are sufficient to enable one to recognize them without difficulty.

Treatment

Inveterate varicose ulcers are difficult to heal, owing to the generally very poor condition of the circulation in the limbs affected. *Rest in bed,* with the foot elevated, is absolutely necessary. An enormous number of substances have been extolled as dressings. The best are styrax, balsam of Peru and especially *scarlet red salve* (see page 73).

Fig. 73. Detachment of the Skin.

Skin grafting (see **Fig. 55** and page 73) may be resorted to. In obstinate cases, *Moreschi* has advocated a circumferential incision of the skin and subcutaneous tissue of the leg above the ulcer. Excision of the whole ulcer with repair of the defect by means of the skin flap has also been done.

Operative treatment of the varices (for which see **Fig. 83** and page 113) is often necessary to bring about the cicatrization of an ulcer.

Fig 73 shows a **detachment of the skin,** that is, a subcutaneous separation of the skin from the subjacent structures, with the special variety of effusion described by *Morel Lavallée* under the name of **traumatic effusion of serosity.** It is the result of a blow on the left elbow, which acted *tangentially*, so that the skin was not injured, but slid on the resistant underlying fascia; the connective tracts uniting the skin to the fascia were torn and with them innumerable lymph vessels, from which exuded the clear, yellowish fluid, which was evacuated by tapping a few days after. The absence of blood showed that no important blood vessels had been torn. If any blood vessels have been torn in such an injury, the result is an ordinary hematoma, which promptly undergoes spontaneous resorption, while those lymph effusions persist indefinitely with slight variations from day to day.

The above-described mechanism explains why such a condition is more frequent in the thigh on account of the tough fascia lata, and on the abdomen. A carriage-wheel passing over the thigh gives the ideal condition for its production. The effusion causes a fluctuating bulging of the skin, which never becomes very tense. There is *no discoloration of the skin*. This, with the *indefinite persistency and the fact that the effusion develops slowly, and not immediately*, differentiates lymph effusions from hematomata.

The treatment is repeated aseptic tapping followed by compression. Incision ought to be resorted to only in case of suppuration.

Fig. 74 shows the *mixed blood and lymph effusion* in the auricle known as **othematoma.** In the auricle (as sometimes in the nasal septum) we find the condition already stated as essential (see above) for the production of such collections, namely, a tense structure (here, the cartilage), over which the skin can slide when struck tangentially.

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Othematoma occurs especially in the upper half of the auricle, and is found in the mentally affected as the result of ill-treatment by blows on the ear, etc.; in workmen who carry on the shoulder loads which graze the ear; in carpenters through carrying planks; in butchers through carrying troughs, etc. It is also a common injury in boxers (*cauliflower ear* results from repeated othematomata) and in acrobats.

It generally causes little trouble. As already stated about **Fig. 73** (page 97) blood effusion is indicated by the *rapid development* of a *tense, dark-blue swelling,* which, after a time, *subsides.* Lymph effusion is indicated by a swelling which *does not develop till some time after the injury* and *has little tendency to subside;* the *skin is not discolored.* Lymph effusion in the ear is nearly always slightly mixed with blood, and always forms a tense swelling, in distinction to lymph effusions in other parts of the body (page 97).

Blood and lymph effusions in the auricle may undergo chronic inflammation, which first causes thickening, later on atrophy and necrosis of the auricle, with considerable mutilation. If the skin is much abraded, the effusion may become septic, with consequent destruction of the cartilage.

Differential diagnosis

Cavernous angioma, which often occurs in the upper part of the auricle, somewhat resembles hematoma. However, angioma is congenital; it can be reduced by pressure and has a special bluish coloration (see **Figs. 36, 80** and **81**). Other vascular anomalies (such as *nævi,* see **Fig. 76**) are usually present in the neighborhood of the tumor.

Treatment

Prophylactic treatment consists in the wearing of ear caps. The hematoma must be protected from injuries which may cause septic infection of the effusion. It undergoes spontaneous resolution, but more slowly than in other places. Lymph effusions recur after repeated puncture; *immediate compression* by strips of adhesive plaster and *massage* are useful in most cases. Massage brought down the othematoma shown in **Fig. 74,** after it had recurred after tapping.

Figs. 75, 76, 80 and **81** (and also **Fig. 36**) show different types of hyperplastic localized lesions of the *vascular system, nævi and angiomata.*

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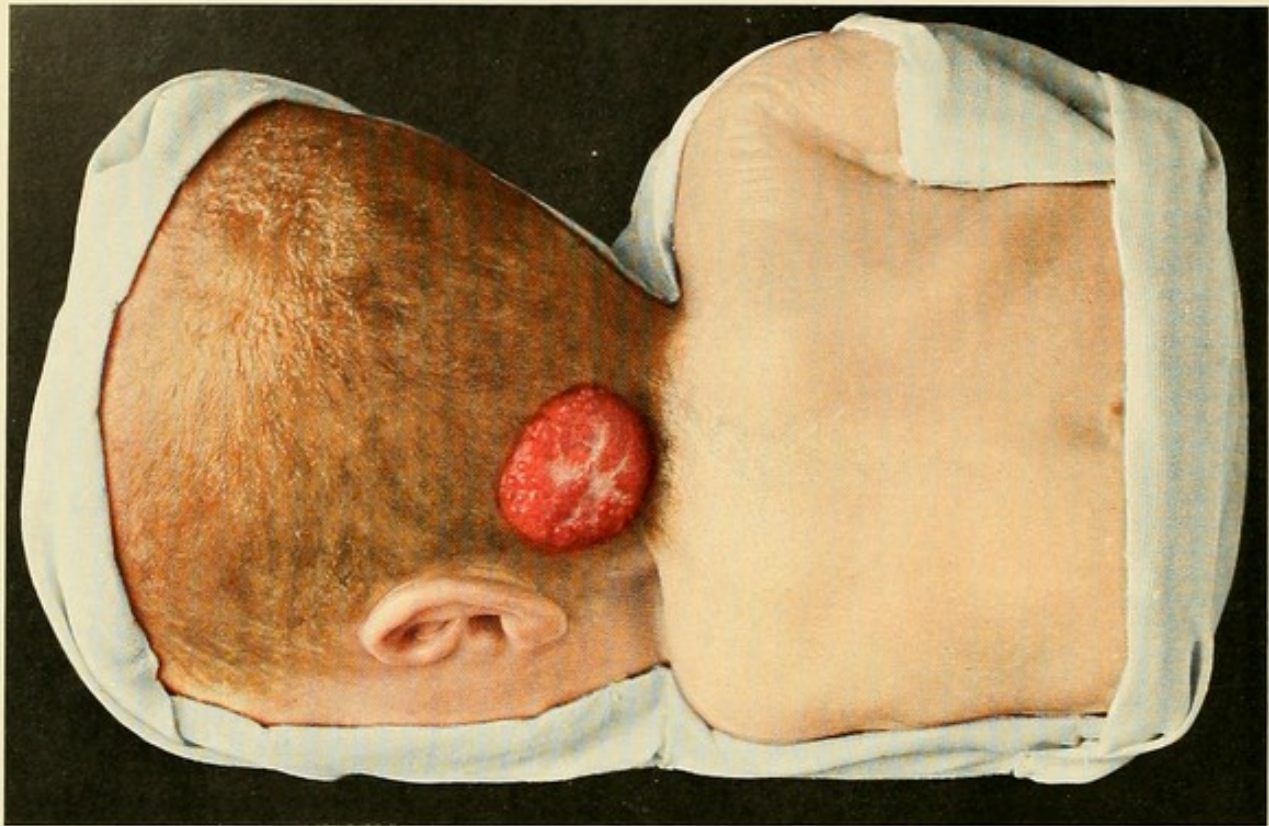
Fig. 75. Haemangioma simplex.
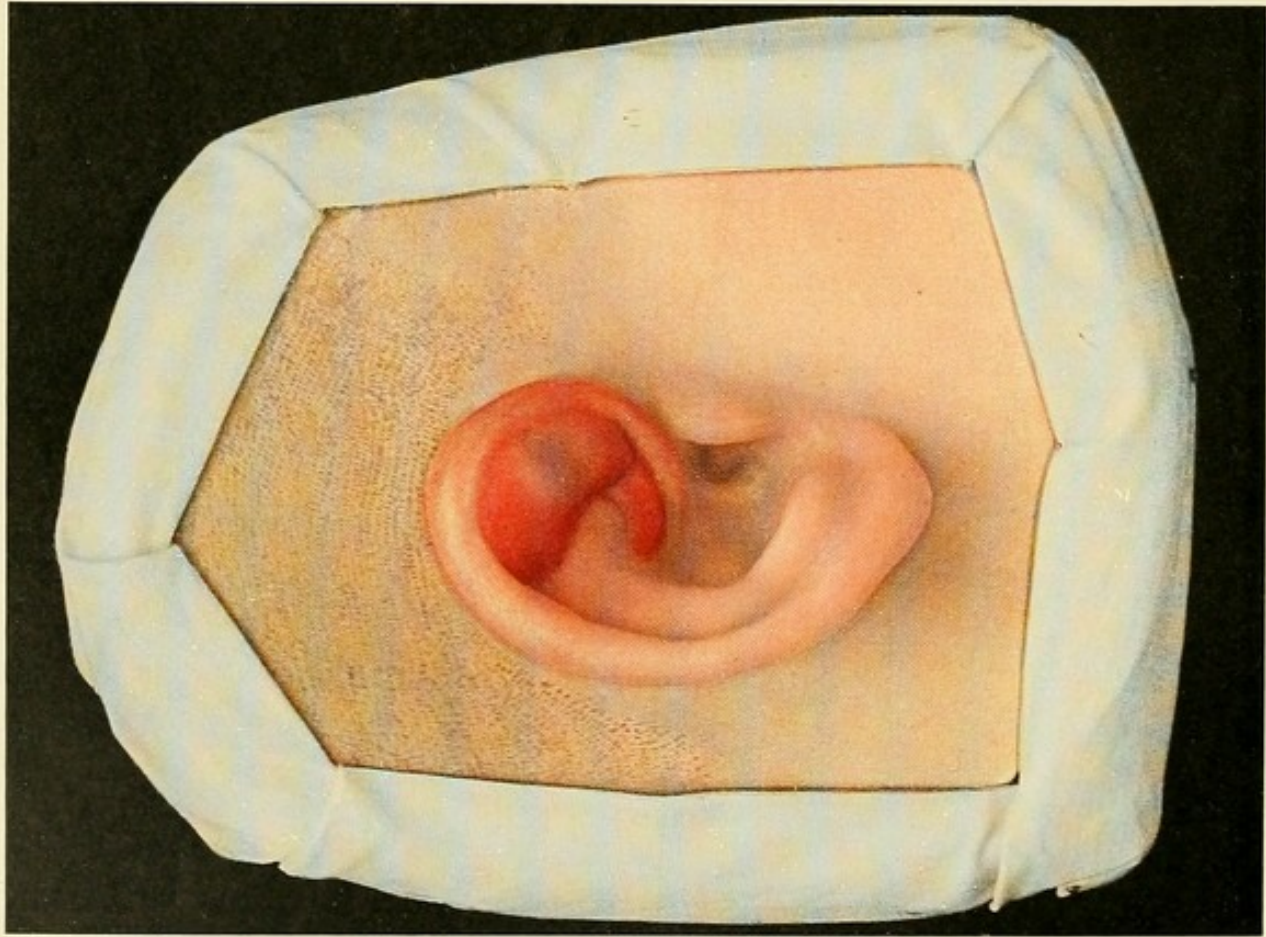
Fig. 74. Othaematoma.

Fig. 75 shows a typical simple **cutaneous angioma of the nape** of the neck, which appeared as a red spot soon after birth and ceased growing after the second year. The borders of the growth are red and show small, ramifying blood-vessels. The center is bluish-red and partly covered by normal skin. The tumor was soft, freely movable over subjacent parts and sharply defined.

Fig. 76 shows a **vascular naevus** (so-called "*wine spot*") covering almost the whole of the left side of the face of a child.

Fig. 80 shows a **subcutaneous cavernous angioma,** which often occurs in the region of the rectus abdominis muscle, sometimes in the muscle itself. The skin is already destroyed over the blue parts of the tumor, and is of a livid color at the periphery. The growth is encapsulated and freely movable over the abdominal fascia (in distinction to infiltrating cavernoma). In some parts the cavernous spaces can be seen through the surface. In the center of the angioma the skin is yellow in some parts and brown in others. The growth was soft, elastic and compressible; in some places thrombosis with consequent shrinking had taken place. The growth had remained stationary for a year.

Fig. 81 shows a combination of **cutaneous and subcutaneous angiomata** with **telangiectases,** affecting the leg. The telangiectases are seen as red spots, in some places arranged in the form of a wreath. There is also an extensive subcutaneous angioma, of a bluish-red color, with more or less normally colored skin in the central parts, and, at the lower part of the subcutaneous angioma, there are cutaneous hemangiomata, appearing as more elevated, round formations in the skin, resembling the simple cutaneous angioma represented in **Fig. 75.** In the whole region of the subcutaneous angioma fine ramifying blood-vessels can be seen.

These hyperplastic conditions of the vascular system may be divided into:

Telangiectases, simple dilatations of pre-existing vessels.

Angiomata, in which there is formation of new blood-vessels. Both may coexist as seen in **Fig. 81.**

Telangiectases can hardly be classed as tumors despite their relations with the other kinds of angioma as they consist in dilatation, lengthening and tortuosity, rather than new formation, of vessels.

For vascular nævi, the point is much more debatable, as those are unquestionably new formations and not a simple dilatation of the

skin capillaries, though many authors have considered them as the greatest degree of telangiectases. This fact is demonstrated by the injection of nævi from their supplying blood-vessels, without at the same time injecting the capillaries of the surrounding skin.

For angiomata of the cavernous type there can be no question that we have to deal with a real vascular tumor (not only in the clinical sense of the word) with continuous tendency to encroach upon neighboring organs. Now, cavernous angioma is simply the highest degree of the lesions found in nævus; it is, in fact, a cavernous nævus having large blood spaces instead of capillaries, and often develops from a simple flat nævus (see page 75).

The form known as racemose or plexiform angioma also almost always consists in a dilatation of a vascular region, not a true, new formation of vessels. It is, therefore, better to give the name cirsoid aneurism to these formations, which are usually congenital and due to fetal remains, but sometimes traumatic. Lastly, neither aneurisms nor varices belong to true vascular tumors.

A form described by *Ziegler* under the name of hypertrophic angioma is best named hemangio-endothelioma, as, in addition to new formation of vessels, there is extensive proliferation of the endothelium.

Nævi and angiomata are congenital conditions due to a defect of development, the nature of which is not yet known. The popular idea of maternal impression has no basis except fancy. They have distinct relations with the lines of embryonic clefts.

The simplest form of nævus is a dilatation of the capillaries of the corium. When it extends, it often reaches the subcutaneous tissue. An association of cutaneous and subcutaneous angioma is, therefore, not rare (**Fig. 81**). Conversely, the cutaneous angioma sometimes develops when the deeper growth appears under the skin.

Clinically, the superficial flat nævus appears as a bright red or dark patch, situated on the lips, cheeks, face, neck, in the region of the fetal clefts. It is present at birth or appears soon after. The margin of the nævus is sharp, but sometimes modified by secondarily developed smaller nævi. In area, it goes from a pin head to half the face (**Fig. 76**) or even more. In such large nævi the edges are jagged and show fine ramifying vessels. The coloration of the skin varies in points, and there are usually different tints in the same nævus. It is often dark purple in the center and bright red at the periphery. It is often broken up by normal skin, which gives a variegated appearance.

In another type (**Fig. 75**) we find a raised growth with a well-defined border. The overlying skin is thin and adherent and of a reddish-blue color. The edges are dark-red and often bordered by an areola of fine ramifying vessels. The tumor is soft, spongy, somewhat compressible, and freely movable over the underlying structures. This is the form that most resembles the so-called "plexiform angioma," already referred to.

More extensive growths may form large, nodular, lobulated tumors, which, when situated in the orbit, may be dangerous from extension to the brain; but this is purely mechanical, as are all the destructive effects of growing cavernous angiomata, and not due to any malignancy of the growth.

Involution of flat and hypertrophic angiomata has been observed as the result of inflammation, generally accompanied by ulceration.

Nævi cause no trouble apart from the disfigurement, when situated on an exposed part of the body.

Cavernous angiomata occur most frequently in the skin and subcutaneous tissue, where their purple color and lobulated surface make them somewhat resemble a mulberry. They are often combined with simple angioma or with telangiectases. They also occur in muscles and bones, and in the brain, breast, liver and tongue (see **Fig. 36**).

In cutaneous angioma the skin is much thinned and appears lobulated and of a bluish-black color. In subcutaneous angioma the skin may be unaltered at first, or slightly irregular and marked by telangiectases. Afterward the skin becomes thinned or destroyed by pressure of the subcutaneous growth, and assumes various colors. (**Fig. 80**).

In the face, combinations of cutaneous and subcutaneous angiomata sometimes form a characteristic appearance, the subcutaneous growth giving a blue color to the skin, while the cutaneous angioma appears in the form of lobulated growths or of bluish-red nodules projecting from the surface. In **Fig. 81** the difference in color between the cutaneous and subcutaneous angioma is very marked, the former being red, the latter bluish in color.

Subcutaneous cavernoma of the scalp requires special mention, as it may communicate with a dural sinus through the emissary vessels, without the scalp showing much change.

Compression with a pad, or by painting the surface with collodion, may sometimes favor spontaneous involution of small nævi. *Excision* is feasible in nævi if not too extensive. It was performed in the case of **Fig. 75.**

Electrolysis is a good method. The nævus is replaced by a superficial scar. Many sittings are necessary, as only a small surface can be treated at a time. In raised nævi, electrolysis may cause thrombosis and embolism.

Cautious cauterization with nitric or carbolic acid achieves the same result. Great care is needed when working near the eyelids.

Freezing with *carbon dioxide snow* (*Pusey*) is the method now most in vogue with American dermatologists. It gives the best results and is even superior to *radium* or *X-rays*.

Telangiectases are very well treated by electrolysis or cauterizations with carbolic acid after scarification.

For the treatment of cavernous angioma see **Fig. 36** and page 48.

Fig. 77 shows a **subconjunctival and subcutaneous ecchymosis** and a large **hematoma** of the left side of the forehead in a child, aged 6. The interest of the case lies in the fact that those were *spontaneous hemorrhages* in a **hemophiliac** individual. The effusion on the forehead occurred intermittently for a time and gradually subsided. There were no other hemorrhagic foci in the body.

Hemophilia is a congenital tendency to hemorrhages, either spontaneous, or under the slightest traumatic provocation. It presents a very *striking example of hereditary transmission*. There are definite families of bleeders. As a rule only the *male* descendants are *bleeders,* but the *hereditary tendency* is transmitted solely through the *female line.*

The real cause of hemophilia is unknown. The only demonstrable alteration of the blood is a *markedly decreased coagulability,* which has been explained by an anomaly of the vascular cells, blood cells and vascular endothelium (*Sahli*), by a deficiency in thrombokinase, by an excess of antithrombin (*Weil*), by an inherited anomaly of the construction of the prothrombin of the body, evidenced by an undue slowness in its activation (*Addis*); which latter explanation seems, up to date, the one giving the best account of the symptoms of the disease. Maybe, in some cases, there is an abnormal friability of capillaries.

Fig. 77. Haematoma diffusum — Haemophilia.

Fig. 76. Naevus vasculosus.

But, whatever the cause, there occur, either spontaneously or after trifling injuries (contusion, cuts, tooth extraction), uncontrollable, repeated hemorrhages, either in the skin or subcutaneous tissue, or externally, or internally.

Of the external hemorrhages, *epistaxis* is by far the most frequent. In the few female bleeders known, menstruation did not seem to cause as much hemorrhage as might have been feared, but often there was considerable anemia.

Needle pricks through the skin (preferably with a round needle, or venous puncture) do not give rise to much hemorrhage in bleeders, because the elasticity of the tissues obliterates the puncture. So that we can take blood specimens for examination without fear. Intravenous injection is preferable to subcutaneous, which almost invariably is followed by a hematoma.

Subcutaneous hemorrhages give the appearance shown in **Fig. 77.**

An interesting condition in hemophilia is *spontaneous hemarthrosis*, which is recognized by the ''snowball crunching'' of the clots and hemorrhagic infiltration of the skin. The effusions at first increase intermittently, and later on become stationary. From the deposit of fibrin on the articular ends of the bone, the cartilages may be extensively destroyed, with resulting anchylosis in a flexed position, or subluxation. However, sometimes, *restitutio ad integrum* has been seen. The knee is the joint chiefly affected.

Renal hemophilia is rare, but exists. Formerly many of the cases of hematuria without discoverable cause were called essential or hemophiliac. Progress of kidney exploration has considerably restricted the number of those cases. The hematuria is profuse, comes on irregularly, and stop also without apparent reason. It causes marked anemia.

Treatment

The **anamnesis** and **knowledge of the bleeding diathesis in the family** are of capital importance in all cases of hemophilia. Generally the patients volunteer the information, even before it is asked for.

Scurvy occurs only under special conditions, is accompanied by fever and ulcerations of the gums; no hemorrhages occur under the skin, in the joints and internal organs.

Purpura may be difficult to diagnose except by the antecedent history.

Barlow's disease occurs only in children, and is associated with subperiosteal hemorrhages, which can be felt as thickenings or tumors near the epiphyseal regions.

Leukæmic hemorrhages are recognized by the changes in the blood and spleen.

Hemophiliac hemarthrosis is very characteristic. If any doubt should arise between this condition and a *myeloid sarcoma* involving the joint, X-ray examination would clear up the doubt.

Treatment

Besides local measures, there is only one treatment of hemophiliac hemorrhages: the *injection,* either subcutaneously or intravenously, of 10 to 20 c.c. of *fresh human serum,* gathered aseptically (*Weil, Welch*). If no fresh serum can be had, horse or rabbit serum, or diphtheria antitoxin may be used. The injection of 10 c.c. of a 5% solution of peptone of *Witte* in saline solution is also good.

Locally, dusting the wound with thrombokinase is frequently efficient.

Fig. 78 shows a **subcutaneous hematoma** with large **ecchymoses,** the result of a **gunshot injury** of the arm. There are present all the well-known discolorations of the skin in such cases: purple, brownish-red, green and yellow.

As is the rule with modern projectiles of great penetrating power, the *aperture of entry,* which is surrounded by *radiating fissures,* is *smaller* than that of *exit,* which has irregular *everted borders.* Moreover, the skin is black and is studded with *inlaid powder grains,* owing to the shot having been fired at close range (a fact of great medico-legal importance).

Treatment

The treatment of gunshot wounds is nowadays *as conservative as can be.* Immediate probing of the wound to locate and extract at once the bullet is no longer the rule. A bullet in the body has little importance in itself: the damage it has caused on its passage is what counts.

There may be an injury to a highly vascular organ (spleen, liver) or to a big blood-vessel: **hemorrhage** then commands immediate interference. There may be perforation of one of the hollow viscera of the abdomen: the ignorance of what injury has been produced and the fear of **hemorrhage** and **peritonitis** make immediate laparotomy imperative in all cases. Of course, wounds of the heart call for operation without the slightest delay. Wounds of the lungs give

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Tab. LX.

Fig. 78. Sugillationes et Suffusiones — Haematoma subcutaneum.
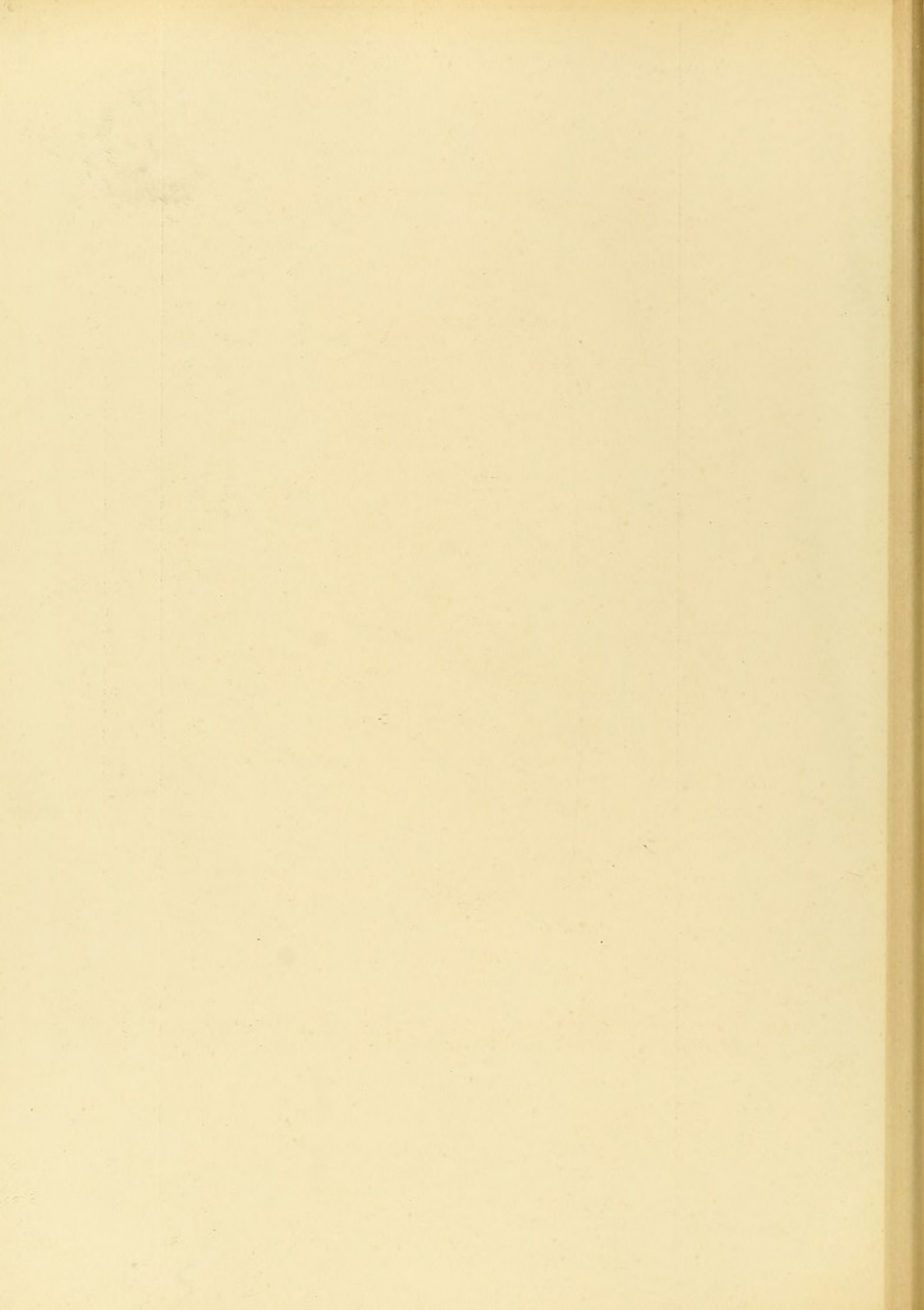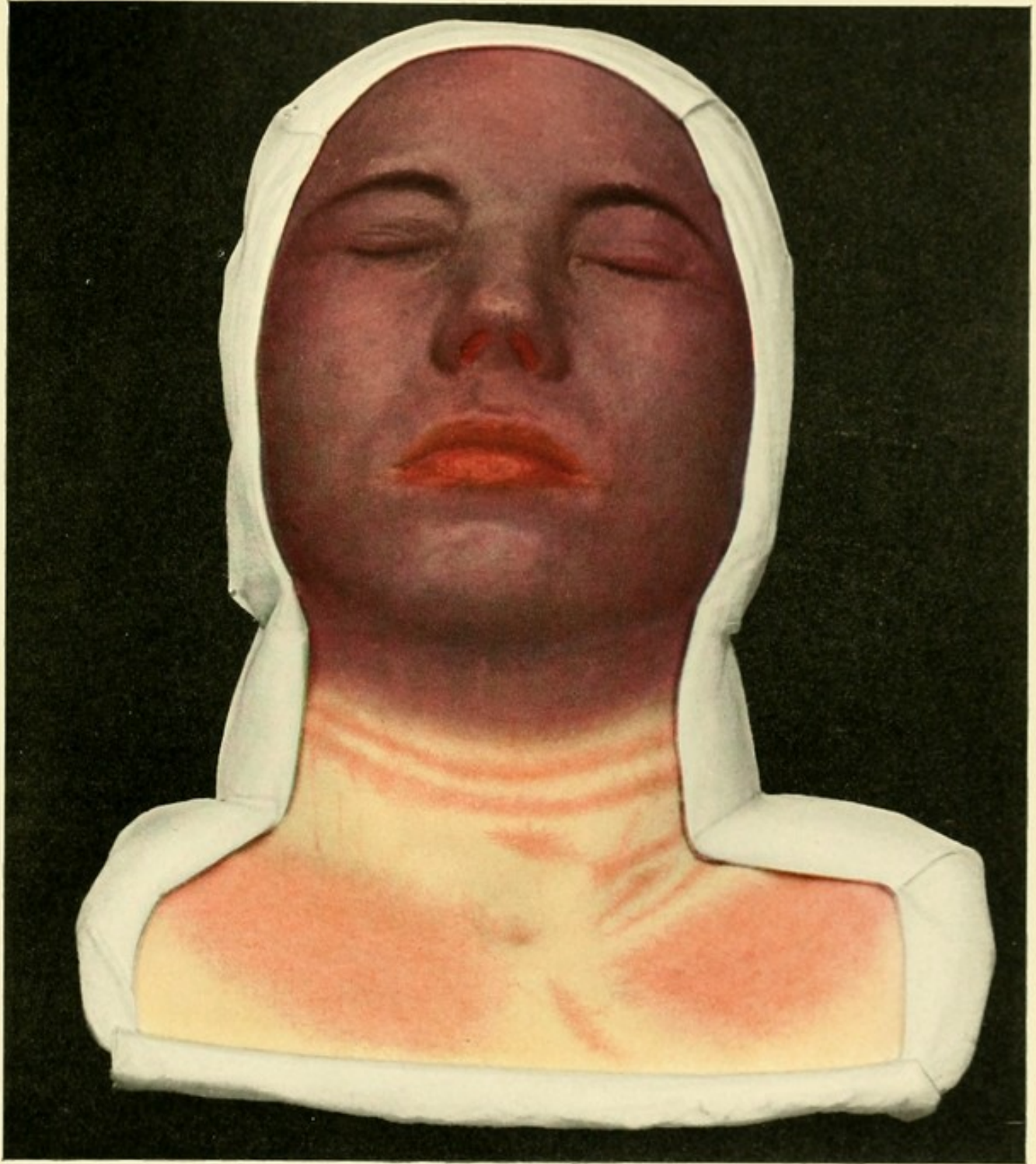
Fig. 79. Petechiae et Haemorrhagiae per compressionem.

Fig. 80. Haemangioma cavernosum subcutaneum.

less formal indications; many heal without trouble. **Hemorrhage** is still here the chief indication for operation.

Gunshot injuries of the skull and brain call for immediate operation when there are *pressure* symptoms due to **hemorrhage** or *fracture*).

But gunshot wounds of soft parts, with or without bone shattering, and not affecting any vital organ, are best left alone, after thorough disinfection of the orifices of entry and exit. Especially is this true of battlefield surgery, where the emergency treatment will solely be a generous swabbing of the surrounding skin with tincture of iodine, and the application of an appropriate dressing. If later infection sets up in the wound, it will be treated according to accepted principles. The bullet will be located by the X-rays; if in a position where it will cause no trouble, it is best to leave it alone, unless it be quite superficial and easy to extract. The bullet itself rarely causes infection, and generally becomes encysted in the tissues. Shreds of cloth carried into the tissues by the bullet are more liable to cause septic complications. Bone and nerve injuries give special indications, but none that would require immediate interference at the time of the accident.

Tetanus is a possible contingency of all gunshot wounds and of those due to explosion of blank cartridges and fireworks. A thorough disinfection of the wound with hydrogen peroxide and a preventive injection of tetanus antitoxin are elementary rules of caution.

Fig. 79 shows a case of *congestive hemorrhage due to compression* of the thorax in a rolling mill, a case of so-called **traumatic asphyxia.**

Hemorrhage from compression of the lower parts of the body generally occurs in the thorax: compression of the thorax (crushing by wheels, machinery accidents) or of the abdomen causes a reflux of blood in the valveless veins of the neck, and the result is the very striking appearance shown in **Fig. 79.**

The whole of the face is colored dark purple, the mucosæ of the lips and nostrils are swollen, and there is also a subconjunctival ecchymosis. (Such ecchymoses sometimes occur after death by abdominal compression without visceral lesions, hence their medico-legal value.) In the neck there are petechiæ and ecchymoses, forming stripes: there were also some ecchymoses in the auditory canal and

the drum membrane. As usual in those cases, there was no intra-cranial hemorrhage and the ocular fundus was normal.

All these symptoms disappeared in a few days under the influence of rest in bed, the only treatment required in such cases, unless the causal compression has brought about lesions of internal organs.

Fig. 82 shows a case in which there was a visibly pulsating swelling in the region of the right sterno-clavicular joint in a middle-aged man, with a probable history of syphilis. From the pulsation, diminution on pressure, systolic bruit and buzzing over the swelling, the diagnosis of **arterial aneurysm** was made; and X-rays showed the tumor—which increased in size slowly, but continually—to be an aneurysm of the aortic arch. There was a pressure paralysis of the recurrent nerve, pressure symptoms on the brachial plexus, and on the veins: while dysphagia (pressure on the esophagus) and dyspnea (pressure on the bronchi and lung) were absent.

Arterial aneurysms are partial dilatations of arteries. *True aneurysms* are those formed by all arterial coats. *False aneurysms* are only pulsating hematomata due to an extravasation of blood outside of an injured artery; they are fairly frequent complications of gunshot and stab wounds of arteries, or develop after rupture of a true aneurysm.

True aneurysms are caused by *disease of the arterial wall, chiefly of syphilitic origin.* In generalized arteritis, they may be multiple. More usually they are single and their location is governed by anatomical conditions: in the aorta where the rebound of systolic impulse is more directly felt than anywhere else; in places where the arteries are submitted to traction or pressure (popliteal artery, femoral artery in case of "rider's bone," an osteoma developed in the adductor muscles near *Hunter's* canal, etc.).

Anatomically aneurysms are *circumscribed* or *diffuse:* when circumscribed they are *cylindrical, fusiform,* or *sacciform.* The anatomical variety is important for the operative treatment.

From the clinical standpoint, aneurysm may be defined as a **tumor belonging to the arterial system;** that is, because it belongs to the arterial system, it gives certain characteristic **physical signs;** because it is (*clinically only*) a tumor, it gives **pressure symptoms.**

The former are: 1st. *pulsation,* synchronous with the heart beat, increased by compression of the artery between the tumor and the peripheral parts, and stopped by compression of the artery on the

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Fig. 81. Hämangioma cutaneum et subcutaneum — Teleangiektasiae.

Fig. 82. Aneurysma arteriale.

side next the heart, this latter character being of course demonstrable only in case of aneurysms of the neck and limbs; 2d. a *bruit* on auscultation, or a thrill, also synchronous with the systole of the heart.

Pressure symptoms are particularly marked in intrathoracic aneurysm, that is, aneurysm of the aorta, arch and descending portion, and aneurysms of the base of the neck. Pressure on the *big venous trunks* causes *cyanosis* and *edema* of the face, neck and upper limb; pressure on the *esophagus, dysphagia* (for which bougies are sometimes passed with fatal results); pressure on the *brachial plexus* produces *paresthesia* in the arm; pressure on the *recurrent nerve* (particularly the left, on account of its anatomical position), *paralysis* of said nerve, with characteristic hoarseness, unless there be compensation, in which case it can only be detected by laryngoscopic examination. An early symptom of aortic aneurysm is *tracheal tug,* i.e., a sensation of traction from below when the larynx is pulled upward.

When the attention is drawn to the mediastinum by one or several of those pressure symptoms, which develop only slowly and gradually as the sac itself, an X-ray examination is necessary. Fluoroscopy will show a pulsating swelling otherwise indetectable, and a *Wassermann* reaction must be taken to find out whether there is still an active syphilitic process.

In aneurysms of the limbs, the pressure symptoms are evidenced by *edema, trophic* and *sensory disturbances, decrease of the muscular power, ulcers,* etc. A large aneurysm may cause pressure atrophy of the neighboring bones (sternum, vertebræ).

Diagnosis

Abscesses, or **benign** and **malignant tumors,** especially sarcoma, when they receive pulsation from an underlying vessel, may be mistaken for aneurysm.

Conversely, aneurysms in which there is no pulsation or bruit, owing to thickening of their walls from thrombosis, and which have caused inflammatory changes in the skin by pressure, may be mistaken for abscesses and incised.

In **cavernoma** there is dilatation of the vessels, but no pulsation. *Racemose aneurysm* presents itself as an irregular serpentine arterial swelling caused by the tortuous dilatation of a vascular area.

Aneurysm of the aorta is frequently difficult to distinguish from *gumma* or *syphilitic sclerosis* of the mediastinum, all the more because close causal relations exist between those three conditions.

X-rays are very helpful in the diagnosis of internal aneurysm because they show the outline of the pulsating tumor.

Treatment

As a rule, aneurysms have a slow, but persistent growth and eventually tend to rupture. Therefore, the prognosis, generally speaking, is unfavorable, but *much depends on the condition of the arterial wall.* If only the dilated segment is diseased, a cure may be obtained if this segment can be buttressed by good and solid clot (thoracic aneurysms) or an operation may be performed with good success; but if all of the arterial system be diseased, reinforcing one point will be of little or no avail, since neighboring points are ready to yield before the arterial pressure.

In aneurysms of the limbs, all depends on the *efficiency of the collateral circulation.* Several tests have been devised for this purpose and must be performed before a final decision is taken as to the treatment.

The treatment of aneurysm has been considerably improved of late owing to the tremendous impetus given in late years to vascular surgery, and a better understanding of the underlying causes of aneurysm.

The growing, and seemingly well founded, belief that most aneurysms are traceable to antecedent syphilis, and in some cases, due to an active direct syphilitic process, indicates the use of *antisyphilitic treatment,* particularly in aneurysms of the chest. However, this treatment cannot include salvarsan, for which well-developed aneurysm is a formal contraindication.

The other methods of general treatment, formerly much in vogue, are practically discarded. Injections of gelatinized serum alone might sometimes prove serviceable in some early cases of thoracic aneurysm.

Surgery claims the treatment of all aneurysms of the limbs. It has not yet succeeded in curing those of the aorta, but the experimental attempts of *Carrel* and *Matas'* work seem to justify the hope that it is only a question of time. Aneurysms of the abdominal aorta are certainly amenable to endoaneurysmorrhaphy (*Gibbon*).

Simple ligation of the artery on the side of the aneurysm next the heart is no longer practiced. Ligation distal to the aneurysm is still the only operation feasible in aneurysms of the innominate artery or of the first portion of the carotid and subclavian arteries. *Ligation* on both sides and *extirpation* of the sac is the method still
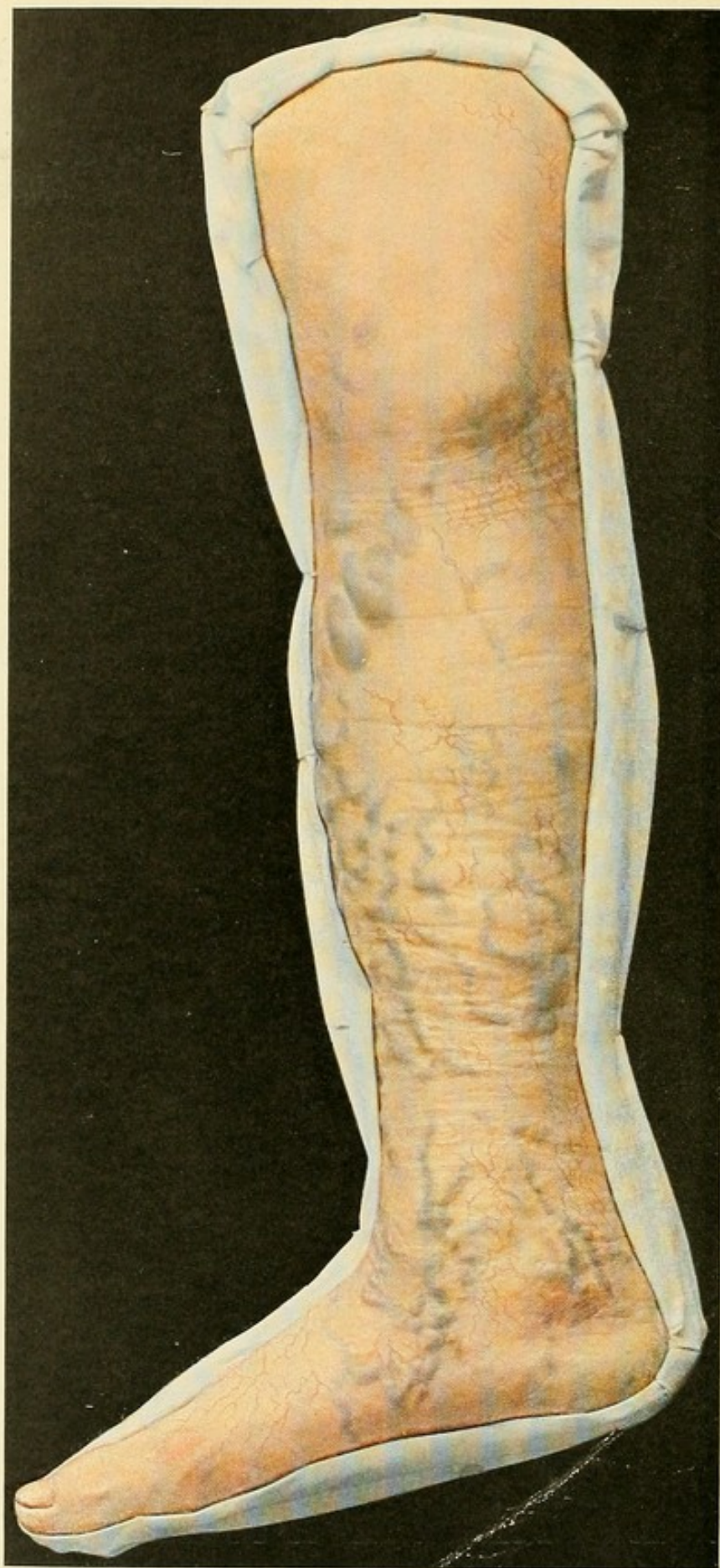
Fig. 83. Varix cirsoides — Pes valgus.

most in favor on the Continent, and its results are satisfactory. *Extirpation followed by restoration of the blood stream by suture* of the vessel or *transplantation* of a piece of another vessel is certainly the ideal method theoretically, but practically it is too difficult, and the same results may be obtained with much less trouble with **endoaneurysmorrhaphy,** which is the method of choice: *exposure* of the sac, *incision, closure by suture of the collaterals;* and then *restoration.* (sacciform aneurysm) *reconstruction* or *obliteration* (fusiform aneurysm) of the arterial channel. The *Matas* operation disturbs the collateral circulation much less than any other technique, and gives a minimal risk of gangrene: it is not a particularly difficult operation. The obliterative type, the one most frequently indicated, is "ridiculously" simple and easy (*Binnie*).

For aneurysms which still remain inoperable, such as those of the aorta, **wiring** (*Finney, Hare, Lusk*) with electropuncture relieves the pain and may prolong life. It is not a dangerous procedure.

For those arteries in which obliteration would be technically practicable, but would endanger the supplied territory (common carotid, abdominal aorta) *Halsted's* and *Matas'* method of progressive occlusion by metallic bands offers a good way of testing, and promoting the development of collateral circulation.

Fig. 83 shows well-developed **varices** of the leg in a woman of 40, who had had many pregnancies. As usual, the territory of the great saphenous vein is most affected. The dilated veins are seen as tortuous, ramifying blue cords under the thinned skin. Where the veins have valves, nodular swellings are visible. The skin has a reddish-brown appearance, due to a network of very fine dilated veins between the larger trunks.

Varices are most common in the leg, in tall individuals and in those who are compelled by their profession to stand long on their feet. Any pelvic tumor obstructing circulation increases the dilatation. Thus is explained the influence of pregnancy. Varices also occur in the hemorrhoidal (hemorrhoids, **Fig. 51**) and spermatic (varicocele) plexuses. Also at the lower end of the esophagus. They are very rare on the upper limbs, where they develop only in conjunction with tumors blocking the circulation.

Varicose veins are not only dilated, but also *markedly altered and sclerotic,* which explains why they remain gaping when cut; hence the copious, sometimes fatal, hemorrhages that occur from apparently

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unimportant venous twigs. (Hematemesis in case of esophageal varices, death after rupture of a varix of the leg.)

Varices particularly appear in individuals having a poor muscular development. Though causing no immediate danger, they are responsible for a number of unpleasant and painful symptoms.

The patients suffer more when standing than when walking. The chief symptoms are a sensation of heaviness of the limb, tingling and numbness, cramps in the calves, especially when the deeper veins are affected; swelling of the feet after walking, disappearing after rest in bed. All this results in more or less disability.

Frequent complications are *eczema*, **ulcer** and elephantiasis (see **Fig. 71**). Besides, varices may be *dangerous* from *rupture* and *hemorrhage*. As a rule the small, thin, ramifying peripheral vessels rupture, sometimes the larger trunks. The blood being under considerable pressure, spurts out in a jet. Fatal hemorrhage may take place unless the limb is elevated and the bleeding stopped by pressure. Death may occur in rupture of subcutaneous varices in the leg and in the internal organs (e.g. brain and liver).

Another danger is *thrombo-phlebitis,* an example of which is represented in **Fig. 84.**

Aseptic thrombosis is a frequent eventuality in varices. Hard lumps are then felt in the veins under the skin. Sometimes the thrombi are calcified and are then known as phleboliths.

Diagnosis

Varices are easily recognized. The only possible mistake would be to take for varices collateral venous circulation developed over a tumor (see **Fig. 32,** sarcoma) or over a deep inflammation. But this cannot withstand a serious examination.

An important point, however, is to recognize the insufficiency of the valves of the great saphenous vein. To demonstrate this, raise the limb till the varices have emptied themselves of blood; then compress the saphenous vein at its opening into the femoral vein in the thigh, lower the limb and suddenly remove pressure on the saphenous vein; the varices then become again immediately filled from above downward with blood from the femoral vein.

Treatment

Prophylactic treatment consists in avoiding long standing, in cleanliness and massage. If the varix is caused by pressure of a tumor, this must be removed when possible.

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Tab. LXVI.

Fig. 84. Thrombophlebitis purulenta acuta.
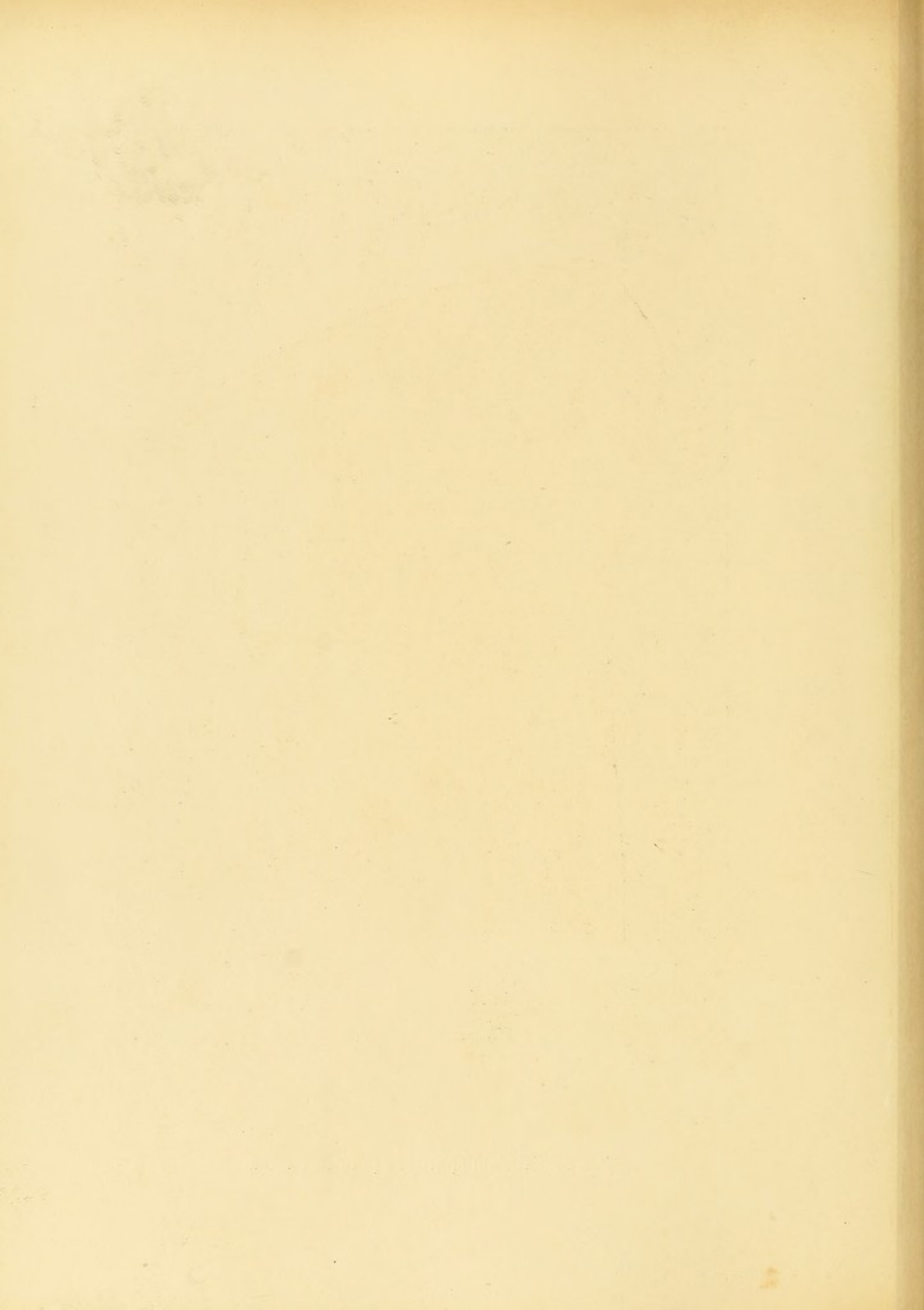
In slight cases the circulation of the limb can be improved and the patient made quite comfortable by the application of flannel bandages from the toes upward (*Martin's* rubber bandage is liable to cause eczema) or by the wearing (when not in bed) of well-made elastic stockings.

Surgical treatment is called for when the varices keep growing, circulatory disturbances are very marked, valvules insufficient and the nutrition of the limb seriously impaired (ulcers, etc.).

Simple ligation of the saphenous vein is useless; the most radical operation is *removal of the whole saphenous vein* through a number of incisions, each segment of the vein being pulled out through the incision immediately above. *Partial resection* of the vein is sometimes sufficient. Elastic bandages should be worn for some time after the operation. Extirpation of secondary varices due to thrombosis of the deeper veins is useless.

Fig. 83 also shows a **flat foot,** a condition frequently associated with varices, either because both depend on a congenital dystrophy of the tissues, or because the falling of the arch of the foot entails poor circulatory condition and venous stasis in the lower limb.

INFECTIONS

Figs. 84-131

PYOGENIC INFECTIONS

The entrance of bacteria in the body immediately starts a wonderful defensive process, the chief agents of which are the leucocytes of the blood. *Metchnikoff* has demonstrated the importance of *phagocytosis* and *Ehrlich* has explained the complicated processes that lead to antibody formation and immunization, by his *side-chain theory*.

Clinically, when bacteria begin to develop in a given point, the *inflammatory reaction* which is the outward manifestation of the defensive process is evidenced by four cardinal symptoms: *Redness, heat, swelling* and *pain*. The *first two* are due to the *active hyperemia*, the blood being called in greater abundance by chemotaxis to the part. The *swelling* is due to the transmigration (*diapedesis*) of leucocytes through the walls of the capillaries, favored by the slowing of the blood stream. The *pain*, pulsating in character in many instances, is due to the *increased tension* in the tissues. It is directly proportional to the rigidity of the tissues. This is why the pain is marked in dense tissues, while, where the connective tissue is lax, there may be enormous edema with but little pain.

The exudation is at first serous and formed exclusively by leucocytes and clear serum; later, when the fight between leucocytes and invading micro-organisms has resulted in the death of many of both, it becomes cloudy owing to the formation of *pus* corpuscles. A pus corpuscle is a leucocyte that has engulfed one or several microbes and has died from its victory. It then becomes a foreign body and has to be cast off. When there are but few microbes and but little pus is formed, the latter may be absorbed by other phagocytes of the body, and no collection ensues. This termination by *resolution* is the most favorable. It is frequent in normal, healthy subjects, in case of low virulence infections.

The pus formation is a fairly accurate gauge of the manner in which the defensive process is successful. Numerous pus cells give a creamy, thick pus and indicate a successful resistance of the anatomical elements against infection; the latter will become circumscribed, and the only result will be an *abscess*. When few pus cells are present after the septic process has lasted a little time, it means that the resistance is unsuccessful, either because the attacking organisms are very virulent or the defending leucocytes are weak. When

sero-purulent fluid or only turbid serosity is found on incision, it generally foretells a grave infection, a *diffuse phlegmon*. If the latter is not stopped in its progress, the result is the invasion of the blood stream: *generalized infection, septicemia* or *pyemia*.[1] The lymphatic channels play also a very important part in the extension of infection.

The strength of the resistance depends on the general health of the body: old, feeble and diseased individuals (e.g. diabetics) are less capable of combating bacterial invasion. In these cases, infections that would be trifling in a normal body may become severe or even fatal.

The virulence of the invading microbe depends to a certain extent on its nature: there is no pathogenic microbe that cannot form pus: the pneumococcus, gonococcus, colon-bacillus, *Eberth* bacillus, pyocyaneus, tubercle bacillus, are frequent pus producers; but the most important pyogenic organisms, from the surgical standpoint, are the *staphylococci* and *streptococci*.

Staphylococcic infections are very common (furuncles, carbuncles, osteomyelitis, etc.) and generally lead to circumscribed purulent inflammations. Streptococcic inflammations are generally more severe in type, more diffuse, and may lead to general infection. Mixed infections are not rare, but there always is a *predominant* microbe.

This first stage of inflammation—pus formation—is *destructive* in character, not only of leucocytes and microbes, but also of surrounding connective tissue elements, which undergo *necrosis*. Necrosis is not much marked in circumscribed abscesses; it is considerable in diffuse phlegmons, where most of the tissues bathed in the turbid serosity may become necrotic, so that these diffuse processes are accompanied by much sloughing.

When the destructive stage has come to an end, the *repair* process begins. The fixed connective tissue cells proliferate and form granulations (in abscesses, the so-called *"pyogenic" membrane*), the inflammatory area becomes isolated and demarcated; the necrosed tissue is cast off with the pus, and the wounds eventually heal by scar tissue, which is but a later evolution of the vascular granulations. During this stage of reparation, the clinical symptoms gradually subside; all this is much hastened if the pus has been evacuated outside the body. The elimination of pus is effected by gradual involvement of tissues and rupture in cases left to spontaneous evolution; but it may be brought about much more quickly by a free *incision*, which saves all the time that would be required for the

[1]General infection, see page 157.

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spontaneous ulceration from within outward of the structures overlying the pus, and, finally, the skin.

Aside from the **local symptoms** of inflammation, enumerated above, there is a **general reaction** showed by a more or less considerable *rise in temperature*. The fever is due to the action of toxalbumins (*toxins*) secreted by the bacteria, liberated by them into the blood stream, and influencing the thermic centers of the brain. Besides these *exotoxins*, there are insoluble *endotoxins*, which remain fixed to the body of the bacteria themselves.

Another general reaction common to all bacterial infections is the increase in the number of leucocytes in the circulating blood, or *leucocytosis*. This is the response of hematopoietic organs, spleen and bone marrow, to meet the demand for leucocytes to destroy bacteria. So constant is this leucocytosis in surgical infections that a blood count is now a routine procedure in the examination of such cases. The leucocytosis is also, to a certain extent, a gauge of the intensity of the defensive process.

Treatment

Every pyogenic infection, however slight it may seem, requires watchful treatment, because unexpected and unpleasant surprises are *always possible*. Cases are frequent where an apparently trifling septic wound has resulted in a fatal septicemia. Under ordinary conditions, and with appropriate treatment, the *prognosis is favorable in most pyogenic infections:* but, nevertheless, these remain one of the big factors of post-operative mortality, perhaps the biggest, despite the progress of surgical technique.

To *limit the infection* and to **favor the natural defensive process** is the *alpha* and *omega* of surgical treatment of suppurative conditions.

General treatment consists in a substantial diet and tonics. Sera and vaccines, which are useful in chronic conditions and medical septicemiæ, are not—barring a few exceptions—of much value in surgical acute infections, nor are, probably, colloidal metals (collargol, electrargol, etc.). Nucleinic acid (20 c.c. in 200 c.c. of saline solution) has found favor in Germany.

Local measures[1] are *rest, dressings* and **hyperemia.** It was *Bier* who, running against all principles formerly admitted, drew the attention to the fact that hyperemia and its consequences, far from being

[1] Inunctions with ichthyol or mercurial ointment, and iodine preparations may hasten the resolution of a non-suppurative infection, particularly adenitis. This is but one kind of hyperemia treatment.

harmful and having to be restricted and kept under control, was indeed the very effort of the organism to bring about a cure: hence his method of *passive hyperemia*, which has been enthusiastically received by some, wrongly applied to all sorts of cases, among which, of course, some where it was not indicated, and subsequently condemned as worthless, as a "double-edged" sword, as impairing the nutrition and delaying the absorption of bacteria and bacterial toxins.

Certainly *passive hyperemia*, improperly applied, may do harm, but this is no fault of the method. Nor is it just to decry it, because it has not fulfilled the expectations of those who asked of it what it could not give. Passive hyperemia is not supposed to take the place of *all* other methods of treatment of pyogenic infections; it is only *one* of several means, and one which, when employed correctly, in proper cases, is undoubtedly beneficial. However, we shall add that, generally speaking, we have seen much better results in subacute and chronic than in acute inflammatory processes.

But the subject of hyperemia in the treatment of acute infections goes far beyond the question of *passive* hyperemia, which is only a particular (and not the *natural*) mode. **Active hyperemia is the natural defensive process, and many of our methods are only means of inducing it.** Thus the swabbing of an infected surface with tincture of iodine is nothing but hyperemia; so is the touching of a small boil with carbolic acid; so is also the use of *moist, warm, antiseptic dressings or of hot antiseptic baths*, one of the most efficient treatments at our disposal. Ice bags are injurious, though they may relieve pain, because they make the blood-vessels contract and retard hyperemia. *Rest* favors hyperemia by slackening the circulation. *Elevation of the limb* is useful only to *relieve the pain* and has no curative value.

It goes without saying that all sources of irritation (foreign bodies, stones, etc.) must be removed.

When pus has formed and collected, it must be evacuated by an *incision sufficient to insure* **free drainage** and **no retention.** Formerly long incisions were the only ones considered as worthy of the name of "*surgical.*" A judicious application of *Bier's* method enables us to shorten the incision in many cases. A notable instance is given by finger suppurations. The scar retraction shown in **Fig. 64** is due in part to the long incision, formerly so much in honor. An infinitely better result is obtained nowadays by multiple small incisions alongside the tendon, and hyperemia (*Klapp's* method).

The time for incision in circumscribed abscesses is when the pus

has collected. In diffuse processes, it must be made without delay, as it is the only means of checking further progress.

Early incision is also indicated when the inflamed tissue is encased and, so to speak, strangulated in a tough inextensible sheath, because the pain is then very severe and incision affords a sure and prompt relief (e.g., treatment of epididymitis by *Hagner's* method).

When the incision cannot be done rapidly without anesthesia, or under superficial anesthesia by freezing, it is best to resort to general narcosis, unless the abscess is situated in a part where regional (conduction) anesthesia is possible; because local infiltration anesthesia is unsatisfactory in inflamed and edematous tissues. All connective tissue septa within the abscess cavity must be broken. Retention of pus increases the virulence of the bacteria and the danger of absorption (see general infection, page 157). Insufficient drainage leads to fistulæ such as that of **Fig. 55.**

The drainage of abscesses is done best with rubber tubes, if the discharge is profuse; by a cigarette drain if the same is scanty.

Various dressings can be applied on infected wounds or those resulting from the incision of abscesses. *A hot wet dressing covered with impervious material,* such as oiled silk, is only a way of inducing hyperemia, as already stated, and belongs to the early, *pre-incision,* stage of treatment. *A wet dressing covered with absorbent cotton, but without impervious fabric,* dries up soon and powerfully draws pus by capillarity; it is the best dressing to apply just after the incision when there is much pus.

A *dry dressing* is the best to promote cicatrization: it is the kind to apply when the discharge becomes scanty. If it adheres to the surface of the wound, it must not be stripped off violently, but first soaked with hydrogen peroxide or a solution of sodium perborate, which makes its removal easy.

When no more discharge exudes, the wound may be packed with gauze to allow it to close *from the bottom upward.* Superficial closure over an imperfectly drained cavity leads to the formation of *sinuses* such as that represented in **Fig. 55.** Granulation tissue should be controlled in its growth by frequent applications of silver nitrate.

Immobilization of the part must be continued till all signs of inflammation have subsided. After healing, massage and electricity are indicated, according to the situation and nature of the affection.

Fig. 84 shows an *acute purulent thrombo-phlebitis* affecting a varicosity of the saphenous vein, which developed after pregnancy. There is a patch of diffuse redness with yellowish nodules indicating the development of abscesses in the infiltrated and thrombosed vein.

This is a not infrequent complication of the condition shown in **Fig. 83** (see page 112).

Inflammation of veins, or *phlebitis,* may be seen after many pyogenic affections (lymphangitis, furuncle, carbuncle, erysipelas, varicose ulcer of the leg). Antecedent pathologic changes of the venous walls (varices most commonly) make them more liable to infection.

In some cases the contamination comes by *contiguity* from a neighboring suppurative process. In every pyogenic infection microscopical purulent thrombi are found in the small venous radicles. In larger veins, periphlebitis develops first and then the vein itself is involved: e.g. involvement of the lateral sinus in otitis media, of the facial vein and cavernous sinus in furuncle of the face.

But, more frequently, the phlebitis is a result of a **blood infection.**

Anatomically, phlebitis is invariably accompanied by the formation of a thrombus, which, in the cases now under consideration, is **always septic.** (Aseptic thrombosis is possible after surgical operations, particularly on the abdomen.) If the virulence of the microbes is not too great, and the resistance power of the tissues good, the thrombus does not go to pus formation. The result is the plastic form of phlebitis, seen particularly in the femoral vein of women as the result of puerperal parametritis, and known under the name of *phlegmasia alba dolens* (painful white leg), which describes the cardinal symptoms of the condition.

If the virulence is great, purulent degeneration of the thrombus sets in: the thrombus disintegrates and by repeated embolisms causes a general, usually fatal, pyemia, with formation of multiple abscesses. Thus infection of the portal vein gives rise to pylephlebitis and multiple abscesses of the liver.

Non-suppurative phlebitis is heralded in by a *local* thermic ascension, in the part affected, as well as by fever. The part (leg generally) becomes *swollen, white, painful,* so that motion is impossible. The *edema* is *hard.* The thrombosis may be so extensive as to cause gangrene of the extremities. The veins are felt as thick, hard cords (*not to be manipulated roughly*).

Purulent thrombo-phlebitis, if superficial, gives the ordinary symp-
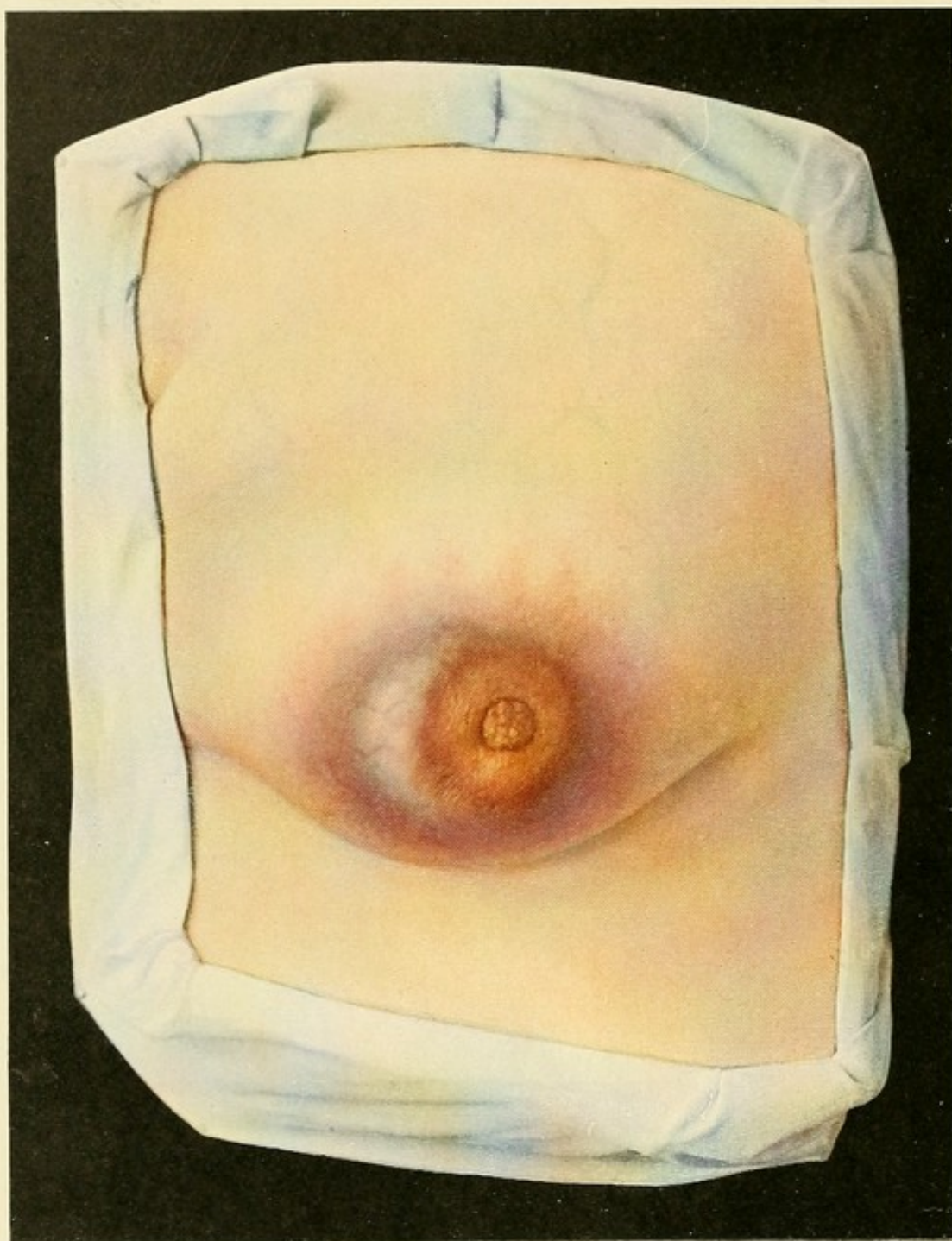
Fig. 85.   Abscessus subcutaneus.

toms of inflammation, redness, swelling and edema of the skin and subcutaneous tissue, pain, fever and chills. These last symptoms are much more marked, and may be the only symptoms, in deep-seated purulent phlebitis. A chill after an operation for a septic condition (otitis media, mastoid, for instance) is always an ominous symptom, claiming immediate attention.

In non-fatal cases of thrombo-phlebitis, resorption of the thrombus takes place, the cells of the endothelium of the thrombosed spot proliferating and invading the clot. This explains why there always remains, in those cases, a *thickening* of the vein, which often goes to *complete occlusion*. Hence the post-phlebitic chronic congestion, which, in the lower extremities, leads to deficient nutrition (ulcer, eczema, elephantiasis, equinus club-foot). Thrombi may become transformed into hard, painful phleboliths, by deposit of calcareous salts.

The thrombosed veins, that are felt as thick, hard cords, along the anatomical course of veins, differentiate thrombo-phlebitis from lymphangitis. When those cords cannot be felt, in deep situated lesions, the diagnosis of thrombo-phlebitis from other pyogenic infections becomes often very obscure.

The two dangers of phlebitis are **embolism** (hence very important and obvious rules of caution) and **generalized infection.** Embolism is the mechanism of generalized infection, but it has also special dangers of its own.

Treatment

Complete *immobility* and *rest* are **essential,** and practically constitute all the treatment of non-suppurative phlebitis.

Suppurative phlebitis calls for *incision:* there need be no fear of hemorrhage: thrombosed vessels do not bleed. *Ligation* proximal to the diseased segment, or *resection* between ligatures, is indicated to ward off an impending generalization (jugular vein in otitis media, facial vein in furuncle of the lip, pelvic veins in puerperal septicemia).

After subsidence of *all* symptoms, gentle massage is indicated to improve the circulatory conditions in limbs that have been the seat of thrombo-phlebitis.

Fig. 85 shows a **subcutaneous abscess** surrounding the nipple, shortly after childbirth. The portal of entry of bacteria was a crack of the nipple. The skin around the latter is bluish-red and swollen.

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Fluctuation indicates the presence of fluid in the subcutaneous tissue. Despite the apparently slight extent of the abscess, there were well-marked general symptoms. The abscess healed quickly after incision.

Acute abscesses must be distinguished from *cold abscesses,* that is, from all purulent collections resulting from the liquefaction of infiltration deposits in chronic infections (tuberculosis, syphilis, sporotrichosis, etc.). The rapid evolution and the cardinal symptoms of inflammation make this distinction easy.

Superficial abscesses are easily detected by *fluctuation.* The purulent nature of deeper collections is established by *exploratory puncture. Incision* and *drainage* is the only treatment.

Fig. 86 shows another suppurative condition of the breast, due also, as are the most frequent inflammations of that gland, to the puerperal state. This condition is of more moment than the one shown in **Fig. 85.** It is an intraglandular infection, a **mastitis,** situated in the outer and lower quadrant of the organ. (This location is the most frequent, owing to the dependent position of this segment and consequent congestion.) The inflammatory symptoms are very marked. The skin is reddened, tense and infiltrated; the whole of the outer and lower part of the mamma is hard and painful. No fluctuation anywhere. There were the usual general symptoms, fever and *malaise,* and radiating pains in the arm.

In some cases, purulent mastitis causes an acute suppurative lymphangitis with involvement of the axillary glands.

Differential diagnosis

Mastitis, that is, abscess of the breast itself, must be distinguished from **superficial abscesses** in the region of the nipple (**Fig. 85**): the latter are well circumscribed and do not cause deep infiltration. Also from the deep **retro-mammary abscesses;** there may be some uncertainty in the diagnosis when inflammation of the mamma exists at the same time. A phlegmon of the retro-mammary bursa raises the whole gland from the thorax; the skin is usually intact; palpation of the breast causes no pain, while pressing the breast against the thorax is very painful. The pus bulges in the fold under the breast. There is generally an acute axillary adenitis and pain on moving the arm is more marked than in ordinary mastitis.

Chronic mastitides, either simple, or tuberculous, or actinomycotic, or syphilitic, do not have the acute course of puerperal

Tab. LXVIII.

Fig. 86. Mastitis puerperalis purulenta.

Fig. 88. Furunculosis.
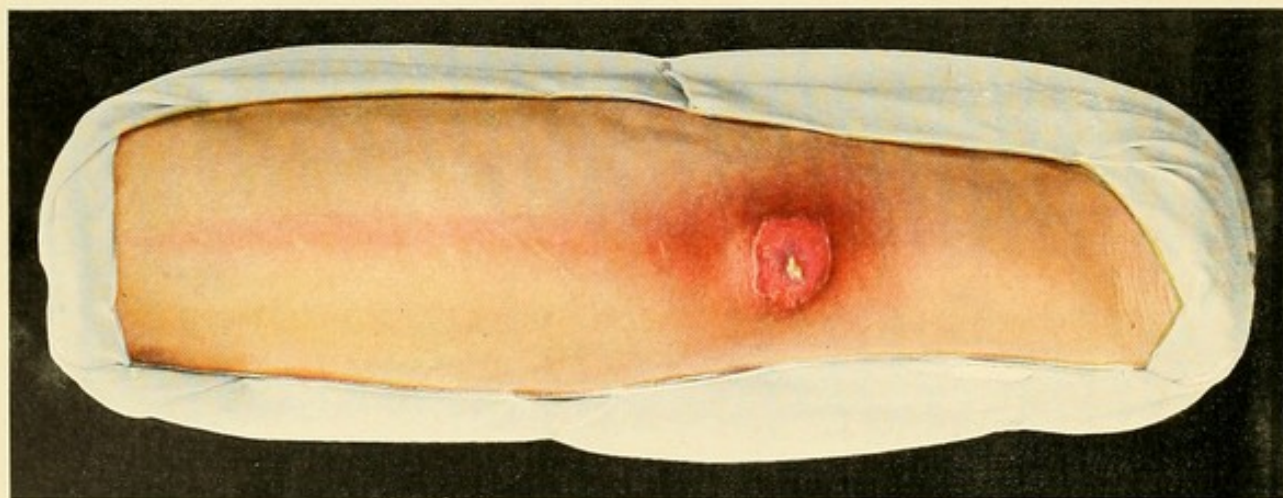
Fig. 87. Furunculus — Lymphangitis.

mastitis and the anamnesis is different. It is much harder to differentiate these various types of chronic lesions between themselves than from acute pyogenic inflammations. Nor does *mastitis neonatorum* or the similar congestive condition observed at puberty give cause to any confusion. However, it may sometimes be attended by superficial abscess formation.

One form of **acute cancer of the breast** (**Fig. 15**), the *mastitis carcinomatosa* of *Volkmann,* or *carcinoma mastitoides,* somewhat resembles mastitis, inasmuch as it also develops during the lactation period. However, the malignancy is so evident that the diagnosis is easy if only one thinks of that form of cancer, as one always should do.

Galactocele and milk abscess have a special doughy consistency and disappear after removal of the milk by a breast pump.

Treatment

Treatment of puerperal mastitis is incision in a radiating direction. All recesses should be opened and connective tissue septa broken: free drainage should be insured. Naturally, the breast is of no further use for nursing at the present time.

Bier's suction cups are very useful in the treatment of suppurative mastitis. Aspiration of the pus, with the application of passive hyperemia, often spares the necessity of making a large incision, which will leave an unsightly scar.

Fig. 87 shows a **furuncle** with **lymphangitis** in one of the most common locations, the nape of the neck; **Fig. 88** a case of **furunculosis** in a young child; **Fig. 89** a **carbuncle** of the nape of the neck in a man of 40.

All these lesions are staphylococcic infections of the *pilosebaceous system.*

The bacterial invasion occurs through the ducts of the sebaceous glands. Even slight friction is sufficient to cause staphylococci, which are always present on the skin, to enter the sebaceous glands, where they find more favorable conditions for their growth than on the surface of the skin. In uncleanly persons pustules often occur on the skin, each one pierced by a hair. This purulent inflammation of the sebaceous glands is called *folliculitis.* (In the eyelids folliculitis of the eyelashes forms *hordeolum,* or stye.) Folliculitis is cured by epilation of the hairs, and may be avoided by cleanliness.

The inflammation may extend beyond the sebaceous gland and

cause inflammatory infiltration of the skin and a *furuncle* (boil) develops. The pathological process consists in hyperemia and exudation, with redness and hard swelling of the skin, followed by necrosis of the tissue in the center of the infiltration; afterward regeneration by the formation of granulation tissue.

Furuncles occur especially in parts which are exposed to irritation—the nape of the neck, the wrist joint, the buttocks, the thigh and the face. They often develop secondarily to cracked conditions of the skin caused by eczema, excoriations, etc. In diabetics, furuncles are very common owing to the dry condition of the skin and the scratching produced by pruritus, also to the body being especially vulnerable to bacterial invasion. For this latter reason, carbuncles in diabetics are still more common than furuncles. Furuncles may also appear in all cases where the bodily resistance is impaired—in children, old people, and the tuberculous. Then, either they are few in number or there is an outbreak of furuncles over the whole body, *furunculosis* (**Fig. 88**), in which most of the furuncles are not as well developed as when there are but a few of them (**Fig. 87**). In furunculosis of children, and in many aborted boils of full-grown people, the process consists in the formation of multiple, small nodular infiltrations in the skin, in which there is no central necrotic core, but a small abscess. This is seen in a few of the larger boils in **Fig. 88.**

The clinical appearance of furuncle is typical. From a small punctiform redness develops a hard, redder, painful nodule imbedded in the skin, and which extends at its periphery and also deeply toward the fascia. The epidermis is at first intact, but afterward ruptures at the apex of the projecting furuncle, exposing a yellowish centre, which becomes more and more demarcated from the hard, red infiltration. In this way a round, crateriform ulcer is produced with a central yellowish core (**Fig. 87**). Sometimes a hair is situated in the centre of the furuncle.

Large furuncles are extremely painful owing to the inextensibility of the inflamed tissues and the resulting high tension in the central parts. Motion exaggerates the pain, so that patients instinctively immobilize the region (stiff neck in furuncles of the nape).

General symptoms are marked in large furuncles. They subside when the central core becomes loosened by suppuration. The cavity is then quickly filled by granulation tissue, which may form a cicatrix in a few days. The hard infiltration remains for a long time and generally causes unpleasant itching of the skin. The scar, which is

Fig. 89.   Carbunculus.

always hypertrophic in all inflammatory processes, may also cause trouble.

Carbuncle is but an agglomeration of furuncles, resulting from the infection of several sebaceous glands and differing only by its greater extent, both in surface and depth. It also differs in being frequently a streptococcic condition.

The skin gives way in several places and there are several yellow cores. Commencing as a small, red nodule, it quickly develops into a hard infiltration, extending to the fascia, and may eventually attain the size of a hand, and cause more or less diffuse inflammatory infiltration of the neighboring parts. Lymphangitis and adenitis are generally present. The affection is accompanied by severe pain, high fever and rigors. Furuncle can develop into carbuncle, especially when the core has been forcibly expressed. Carbuncle of the nape of the neck may attain an enormous size and spread from ear to ear. In diabetics, carbuncle causes extensive necrosis and is a serious affection.

Complications may increase the severity of furuncle. There is always lymphangitis, especially in the extremities, and often lymphadenitis. Early implication of the lymphatics signifies extensive inflammation and virulent bacteria.

Several furuncles are sometimes found close together, either from simultaneous infection of several sebaceous glands or from secondary infection from the primary furuncle. This often occurs after the application of plaster or other measures with the object of "drawing out" the furuncle. Such cases must be distinguished from primary carbuncle (**Fig. 88**), which, as already said, is also an agglomeration of furuncles, but of a distinct type.

Extensive furunculosis may be fatal from exhaustion. It may also lead to purulent thrombo-phlebitis and general sepsis.

Furuncles of the lip or carbuncle of the face may cause meningitis by thrombo-phlebitis of the facial vein and cavernous sinus and general pyemia may be caused by thrombo-phlebitis of the jugular vein. Renal abscess and osteomyelitis (see page 147) are other possible complications.

Lymphangitis, as shown in **Fig. 87,** appears as red, diffuse patches, which soon develop into irregular red cords extending from the periphery to the root of the limb. The number of those red cords, which are smaller than the cords of thrombo-phlebitis, diminishes in

the upper part of the limb, till finally there is only one reaching the regional glands, which are swollen and tender. The cords themselves are slightly raised above the level of the skin, tender on pressure, and abscesses frequently develop within and around them.

Diagnosis

The acuminated, localized swelling of an ordinary furuncle can hardly be mistaken for any other affection.

Furuncles originating in the *sweat glands* are often described as a separate condition (*hydradenitis* or *hidrosadenitis*). The distinction has practically no importance. The sweat gland furuncles are particularly frequent in the axilla.

Metastatic furuncles are associated with other pyogenic infections.

Acne is sometimes associated with furuncular lesions, and might somewhat resemble furunculosis in its attenuated form. But the presence of comedones and the painlessness of lesions help settle the diagnosis.

Anthrax (malignant pustule) differs from **carbuncle** in the presence of small vesicles filled with turbid fluid and early central necrosis of the skin, and in the absence of cores (see **Figs. 112** and **113**). In doubtful cases a bacteriological examination must be made.

Treatment

All constitutional disturbances (diabetes, anemia) should receive proper treatment, the diet should be invigorating, and the skin kept very clean.

Old-fashioned poultices are to be rejected, as they favor auto-inoculations in the neighboring skin. On the contrary, hot antiseptic dressings are beneficial (hyperemia).

Small furuncles may sometimes be aborted by pulling out the central hair and swabbing the resulting hole and the apex of the furuncle with pure carbolic acid.

Incision does not materially shorten the duration of a boil, but markedly decreases the pain. When, therefore, it becomes likely that a furuncle will suppurate, there is no reason to delay free incision, so as to give complete relief of tension. This is done after careful disinfection of the skin, under local anesthesia for simple furuncles, under general anesthesia for carbuncles, in which the crucial incision must be deep. The core of a furuncle should never be forcibly expressed. The best way to hasten healing in a carbuncle is to remove all the inflammatory mass. Protecting the surrounding skin with fatty

Fig. 90. Erysipelas erythematosum.

antiseptic ointments is good. The thermocautery was formerly much used in the treatment of carbuncles. It has no special advantages.

Furunculosis has been treated internally with yeast preparations, and more recently with staphylococcic vaccines (stock or autogenous), which give good results in acne.

Fig. 90 shows a typical case of **erysipelas of the face,** which originated from a fissure on the nose.

Erysipelas is the streptococcus infection of the skin. The streptococcus of erysipelas, formerly thought a distinct species, is very likely the *Streptococcus pyogenes.*

While in lymphangitis the deeper and larger lymphatics are infected, in erysipelas the smaller lymphatic spaces of the skin and subcutaneous tissue are plugged with streptococci. A similar condition may occur in the superficial layers of the mucous membranes.

Erysipelas may occur wherever there is a solution of continuity in the skin—after scratches and excoriations, after all injuries and operation wounds. Spontaneous or "medical" erysipelas is most often seen on the face, and the portal of entry is an excoriation of the skin or of the nasal mucosa. It generally begins in the inner canthus. It may also be combined with various pyogenic affections—whitlow and phlegmon. Conditions which give rise to constant irritation of the skin, such as lupus, tuberculous fistula, ulcer of the leg, foreign bodies, etc., may also give rise to erysipelas, which is then often relapsing. Relapsing erysipelas of the face and leg may cause elephantiasis, flattening of the nose and considerable deformity. Lastly, erysipelas may arise in general streptococcic infection, and is then always combined with other pyogenic conditions—abscess, phlegmon, etc.

Some subjects seem to have a particular predisposition to erysipelas, so that they will contract the disease whenever exposed to the infection.

The common form of erysipelas, which consists in a red elevation of the skin, is called erythematous erysipelas (**Fig. 90**). In bullous erysipelas the skin is covered with vesicles (**Fig. 91**). In hemorrhagic erysipelas there is hemorrhage in the skin (**Fig. 91**). In the great majority of cases erysipelas ends by resolution, but sometimes it may cause cutaneous abscesses, and in the form of gangrenous, phlegmonous erysipelas may give rise to ulceration and extensive destruction of the skin.

The clinical symptoms of erysipelas are characteristic. The disease usually commences by a *chill, high temperature* (104°-106°) and *redness* of the skin. There is itching and tension in the skin, and tenderness on pressure. There is considerable constitutional disturbance, owing to high fever, headache and vomiting, which continue while the disease progresses. The affected skin is *red, tense,* somewhat *glistening* and *slightly raised* above the level of the rest of the skin. The *borders are well defined, distinctly raised* and zigzag (seen in **Fig. 90,** especially toward the scalp and the neck), so that the extension of erysipelas, especially on the face, has been compared to lambent flames. When the disease spreads over the whole body, it is spoken of as migratory erysipelas. In places where the skin is loosely attached (eyelids, scrotum), there may be considerable swelling and edema, as may be seen in **Fig. 90.** The eyelids were so swollen that the patient could hardly open them.

Then the temperature falls suddenly, the redness ceases to extend, and the skin, after slight desquamation, resumes its normal condition in about a week from the onset of the disease. In relapsing erysipelas the whole process is considerably shorter and may not take more than one or two days.

Erysipelas of the mucous membranes is generally difficult to recognize, except when it is an extension from erysipelas of the skin. The mucous membrane is swollen, edematous, sodden and of a deep-red color. Constitutional disturbance is generally severe. Erysipelas of the buccal mucous membrane may occur after tooth extraction with dirty instruments. It may cause death by meningitis or edema of the glottis.

Diagnosis

Erythematous erysipelas is so characteristic that it can hardly be mistaken for other affections. The *advancing, irregular, raised edge* and *shiny surface* are enough to differentiate it from other inflammatory conditions.

Eczema itches and burns much more than erysipelas.

Lymphangitis is a diffuse redness without raised border, or hard cords going directly to lymph glands.

Fulminating gangrene (see **Fig. 109**) is sometimes called by Continental writers "bronzed erysipelas," but there is no similarity whatever between this condition and ordinary erysipelas. Bullous erysipelas might be more confusing.

Fig. 91. Erysipelas bullosum hämorrhagicum.

Diffuse suppurative cellulitis quickly leads to infiltration and pus formation. If there are any abscesses in erysipelas, they always remain very small.

Anthrax can be confused with bullous erysipelas (see **Fig. 91** and page 132) much more than with the common erythematous form.

Treatment

Erysipelas is a contagious disease until after the period of desquamation.

The patient should be isolated and the room disinfected after recovery.

The best local applications are 0.5% carbolic oil or 10% ichthyol. Drawing a circle with tincture of iodine beyond the raised border is but one way of inducing active hyperemia and establishing a ring of leucocytic infiltration, which acts as a barrier against the peripheral spreading.

Serum therapy, extolled by some, gives uncertain results; in theory, it does not seem rational, since repeated attacks of erysipelas, though lessening the symptoms, do not confer any immunity against infection.

An intercurrent erysipelas often has a beneficial influence on a pre-existing disease of the skin. It also sometimes brings about the regression of malignant tumors, chiefly sarcoma. Hence the use of toxins made by *Coley* in the treatment of sarcoma, to which reference has already been made (see page 30).

Inunctions of colloidal silver ointment have been found beneficial in streptococcic infections.

Fig. 91 shows a case of ***hemorrhagic bullous erysipelas*** of the arm consecutive to a horse bite. Around the three superficial abrasions due to the teeth the skin is dark red, and there are annular extravasations of blood. There are also several vesicles filled with turbid fluid, in which streptococci were found.

There is extensive diffuse reddening, especially on the forearm, and a brownish coloration due to numerous extravasations of blood from the smaller blood-vessels situated around the lymphatic vessels. In the upper arm there is a macular and cord-like reddening due to lymphangitis. The axillary glands are much swollen and painful; the swelling of the forearm was so extensive that a deep phlegmon was suspected, which suspicion was all the more justified because

wounds caused by bites from animals or men tend to become severely infected, but the symptoms quickly subsided after suspension of the arm and the constitutional disturbance always remained mild. In the place where the erysipelas was hemorrhagic and bullous, there occurred a superficial phlegmonous inflammation, which led to gangrene of the skin.

Diagnosis

Anthrax also commences with redness of the skin and the formation of vesicles (**Fig. 112**), fever and rigors, and may, in its early stage, be confounded with this form of erysipelas. But the redness is not so extensive in anthrax, nor so rapidly developed. Anthrax always causes early gangrene of the skin. In doubtful cases anthrax bacilli must be looked for in the contents of the vesicles.

In the case shown in **Fig. 91,** which resulted from a horse bite, there was a suspicion of **glanders.** But, in the latter the redness is punctiform or macular; the vesicles are larger and purulent, and soon rupture, giving rise to gangrenous ulcers, and the *bacillus mallei* is found.

Subcutaneous phlegmons, caused by virulent streptococci, may exhibit an erysipelatous redness of the skin, but this only occurs in the region of the phlegmon, and does not extend so rapidly as erysipelas. Vesicles may also form on the skin in virulent streptococcic infection.

Phlegmons due to gas-forming bacteria (e.g. *malignant edema,* **Fig. 109**) cause rapid redness and swelling of a whole limb. Increase of pressure in the tissues from the formation of gas also leads to the development of vesicles, but these are very large and often raise the epidermis over the whole part affected (**Fig. 109**). In these severe forms of phlegmon there are signs of general infection from the beginning—chills, delirium, diarrhea, dry tongue, and bacteria in the blood.

In all the above-mentioned cases the clinical pictures may be very similar, and the diagnosis should always be established without delay by bacteriological examination. A correct diagnosis is all the more important because the treatment is not the same in the different affections. In erysipelas, anthrax and glanders conservative measures are indicated, while streptococcic phlegmon requires early incision to prevent general infection, and in gas-phlegmon very extensive incisions, or even early amputation of the limb, are necessary to save the patient's life.

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Fig. 92. Erysipeloid.
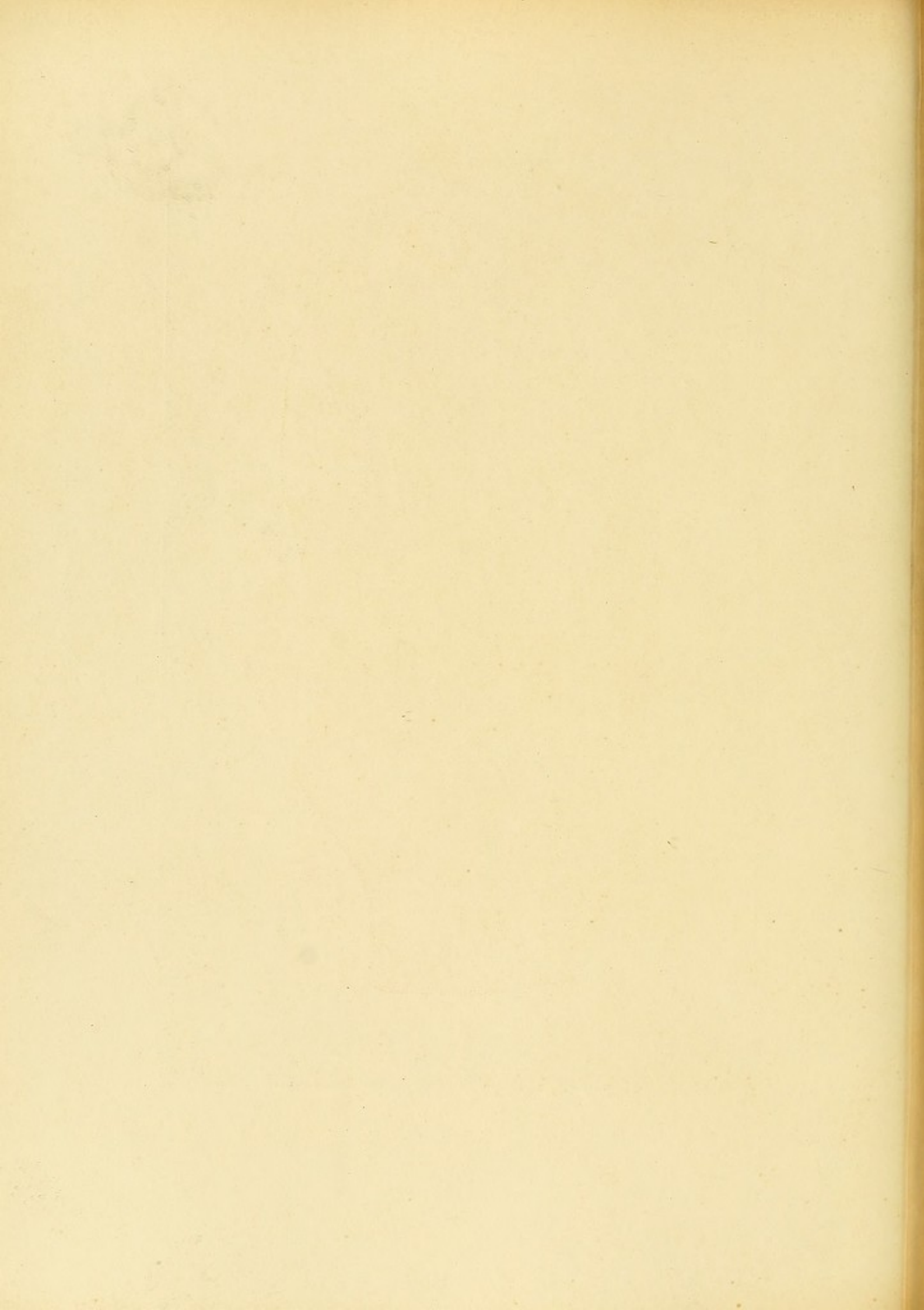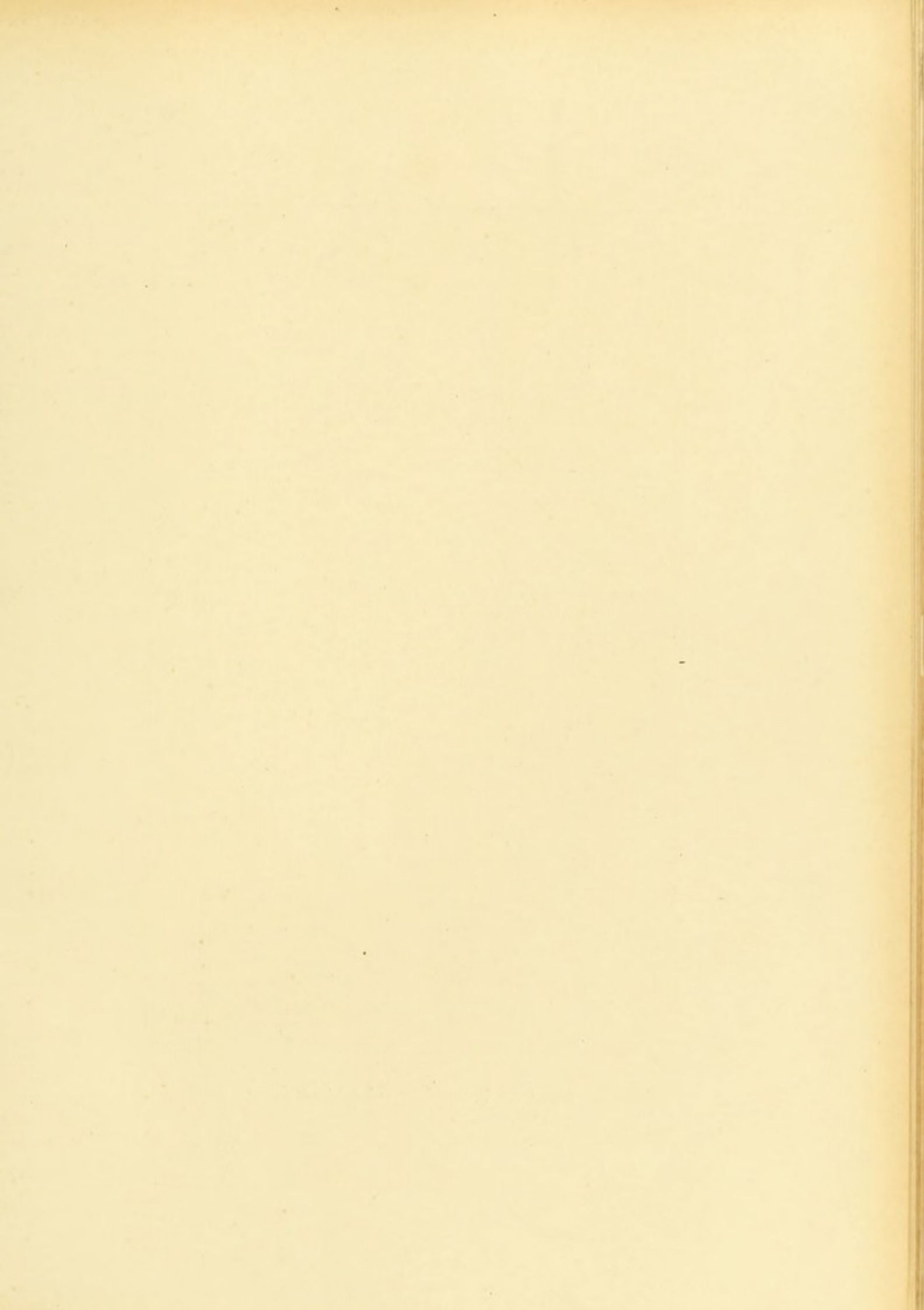
Fig. 93. Panaritium subepidermoidale.

Fig. 92 shows a case of an affection very similar to erysipelas, and called chronic erysipelas or **erysipeloid.** It is a bacterial infection of the skin, of a very harmless nature, maybe caused by the *staphylococcus albus,* which has sometimes been found in it.

The case was observed in a cook, a few days after handling game. This is the common etiology, as erysipeloid generally occurs after injuries to the fingers, especially by fish and game. It is, therefore, more common in venders of fish and game, cooks, butchers, curriers, etc. Sometimes the injured spot is invisible, as the redness and swelling generally appear a few days after the injury. In other cases foreign bodies are found in the skin. The affection has been observed in surgeons after operating upon infected persons; it is more common in the autumn.

It begins with redness and swelling of the fingers. Like erysipelas, the redness has sharp, irregular borders. The redness spreads slowly but continuously over the whole finger, and may extend to the next finger and as far as the wrist. At this point the inflammation stops. There are no constitutional symptoms; no fever nor chills. The patients only complain of itching and a feeling of tension in the skin. In some cases there is lymphangitis, generally on the extensor surface, as far as the axilla. In rare cases adenitis with high temperature has been noted. The symptoms generally subside in a week, but relapses are common. Suppuration has never been observed. Erysipeloid differs from erysipelas in its chronic, apyretic course, paler color and demarcation at the wrist.

Treatment consists of ointments, rest and support on splints.

Figs. 93 to **98,** inclusive, show the principal types of the suppurative conditions of the different tissues of the finger grouped together under the name of **panaris, felon** or **whitlow.**

Whitlows are most frequent in workingmen owing to the numerous cracks and fissures of their skin. It often occurs after punctured wounds, which directly inoculate bacteria (staphylococci or, more rarely, streptococci) in the finger. It is far from rare in surgeons.

The most superficial whitlow is the *subepidermic* (**Fig. 93**). But the most common, and the most important to know, is the *subcutaneous* variety (**Fig. 94**), because from this, if improperly treated, derive all the other, deeper, and more severe types.

The anatomical disposition of the subcutaneous tissue of the

fingers is peculiar: vertical connective-tissue septa separate the fatty tissue into a number of distinct compartments. If bacteria gain an entry into one of these, the inflammation naturally is at first circumscribed: there is hyperemia, exudation and tissue necrosis: the latter occurs rapidly owing to the impairment of nutrition from the great pressure in the inflamed area enclosed within inextensible walls. In this way a necrotic core is formed, as in furuncle. If properly drained at that time, no further damage ensues; but if not, the pus burrows in the direction of least resistance. The tendon sheath is bathed in pus, finally involved and perforated. The result is a *tendon-sheath suppuration* (**Fig. 96**).

Going still deeper, the pus may involve the periosteum of the phalanges, the bone, and the finger joints (*osteal and articular whitlow,* **Fig. 95**).

The clinical symptoms vary in severity in proportion to the depth of the pathological changes, and the virulence of the bacteria. In *subepidermic whitlow* (**Fig. 93**), a purulent vesicle develops, generally on the dorsal surface of the finger, with slight redness of the surrounding skin. The raised epidermis sometimes shows several yellow spots, where the pus breaks through. Pain and functional disturbance are slight, the inflammation remaining local. Lymphangitis is rare, there is no tendency to spread, and but little or no constitutional disturbance.

In *subcutaneous whitlow* it is quite otherwise (**Fig. 94**). The whole finger is red, swollen, flexed and extremely painful, especially at one spot. This greatest pain on pressure in one spot is often the only symptom pointing to the primary seat of infection, as in horny-handed workmen, the latter is often very difficult to see. In a few cases only, the skin gives way and a yellow core becomes loose and cast off, after which healing takes place by granulation tissue. More usually, as the hard skin of the palmar surface of the fingers prevents escape of pus, the latter takes paths of less resistance. The vertical connective tissue septa, mentioned above, guide it toward the peritendinous zone, where it may spread along the whole length of the tendon. It may also reach the loose connective tissue on the dorsal surface, and give rise to marked redness, swelling and edema, while inflammatory signs may be slight or absent at the primary focus of infection on the flexor surface. There is some lymphangitis of the hand and forearm, a moderate fever (102°) and constitutional disturbance.

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Tab. LXXV.

Fig. 94. Panaritium subcutaneum — Lymphangitis acuta.

Tab. LXXVI.
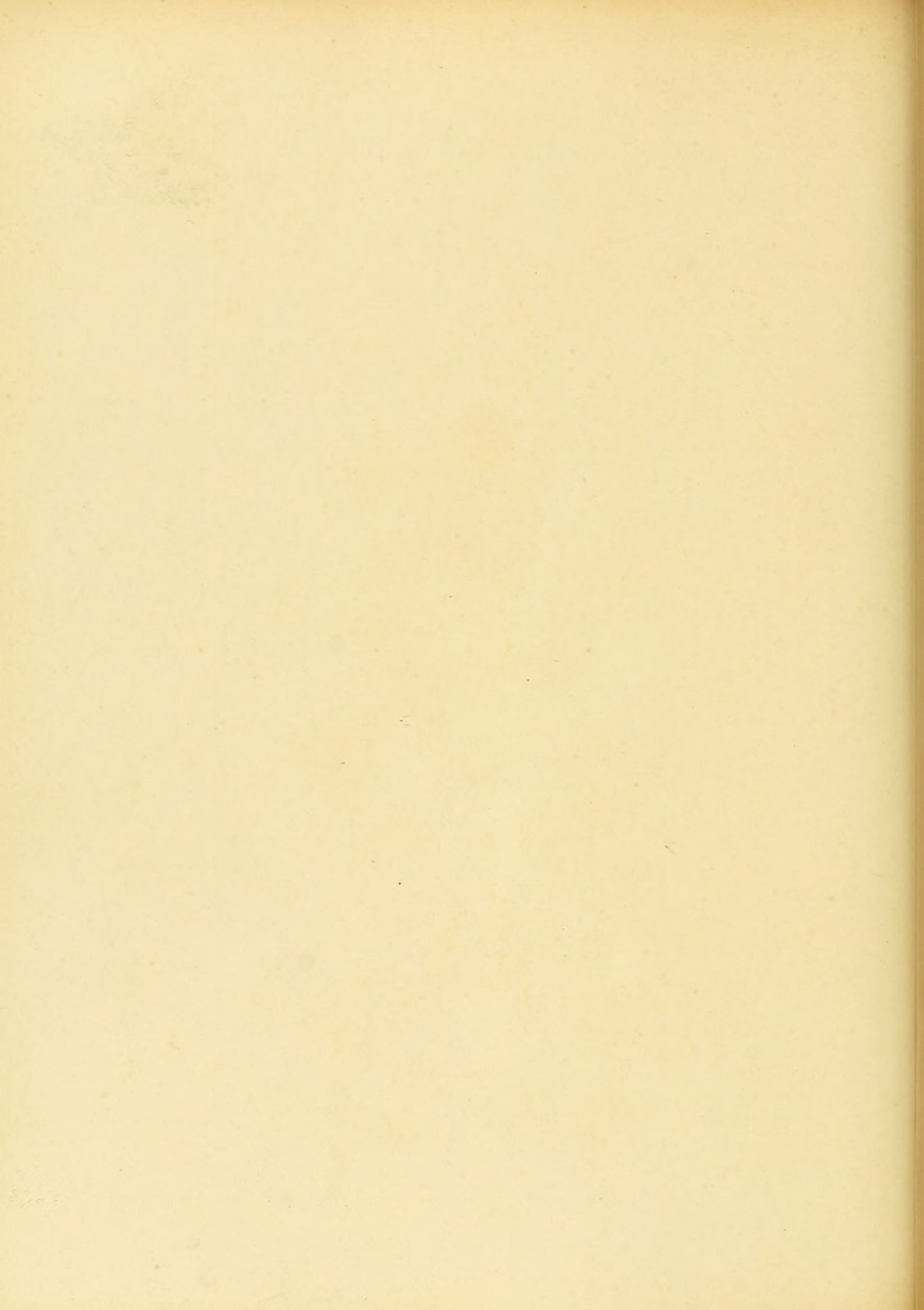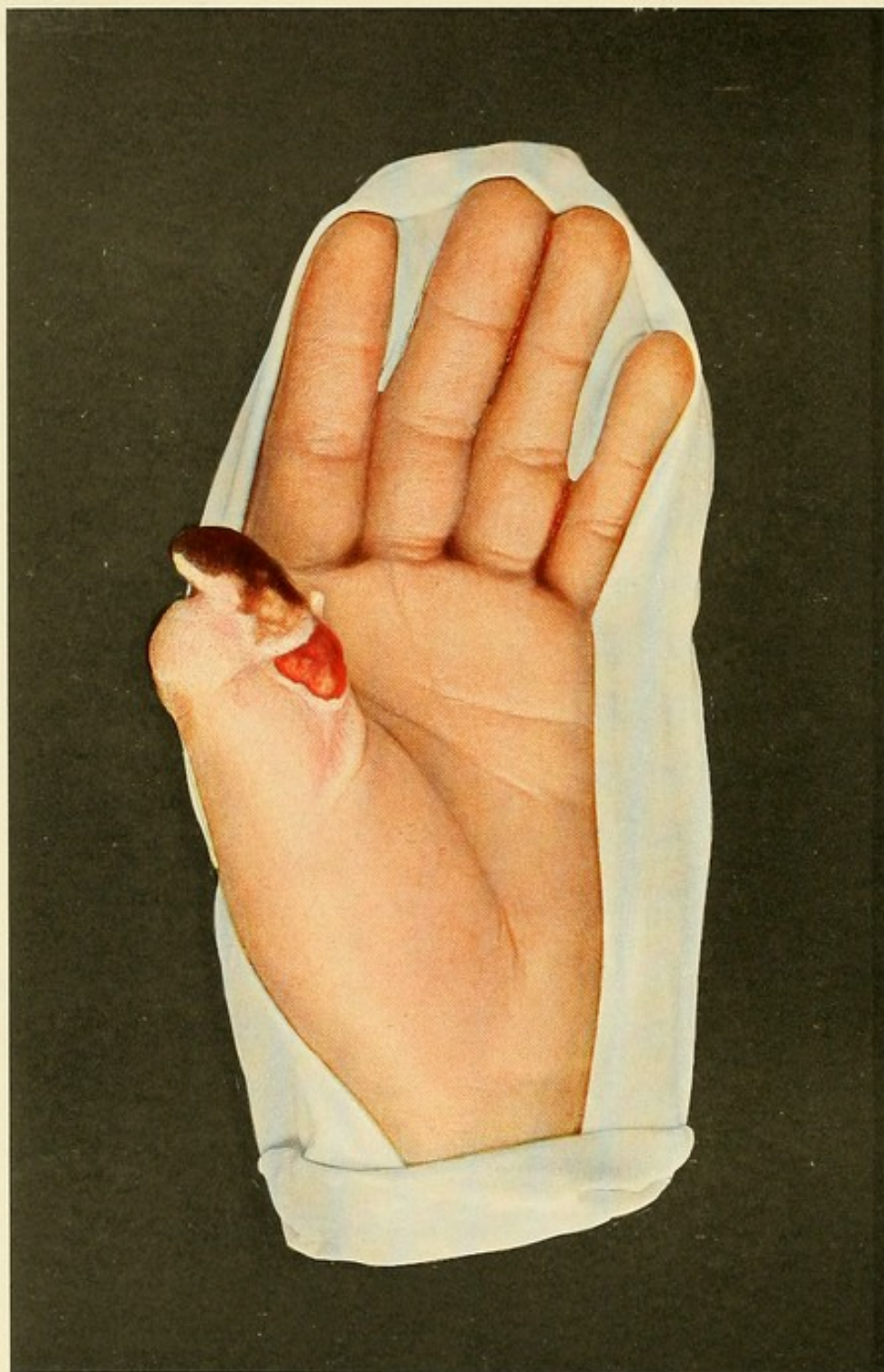
Fig. 95. Panaritium ossale et articulare.

An interesting variety of subcutaneous whitlow is **interdigital whitlow,** shown in **Fig. 97.** It is a subcutaneous suppuration between the metacarpal bones; in the actual case, between the thumb and indicator finger. Redness and edema are marked on the dorsal surface, and, as pus is present in more considerable quantity than in ordinary subcutaneous felon, fluctuation, which is rare in the latter, is present.

The symptoms are most severe in **tendinous whitlow** (**Fig. 96**). There is more swelling of the finger, and the participation of the tendon sheath is evidenced by the flexion of the finger. There is pain on pressure along the whole tendon sheath, and usually over the whole palm. Movement of the tendon causes great pain, and extension is almost impossible. Lymphangitis and erysipelatous reddening often extend far beyond the seat of infection. There are chills and fever (104°), sleeplessness, and considerable *malaise.*

If the tendon sheath of the thumb or little finger is infected, the pus may extend along the course of these sheaths as far as the wrist; whereas, suppuration in the tendon sheaths of the second, third and fourth fingers does not extend beyond the metacarpo-phalangeal joints, where these tendon-sheaths end.

In the wrist the tendon-sheaths widen and lie so close together that suppuration may extend from one to the other. In this way, infection of the tendon-sheaths of the thumb may result from that of the tendon of the little finger; and inversely, infection of the little finger may come from the thumb. This has been called V-shaped whitlow. It is obvious that infection of both tendon-sheaths causes more severe symptoms—high fever and much constitutional disturbance. The thumb and little finger are flexed, swollen and very painful on pressure. The pus often breaks through the sheaths and spreads between the muscles of the forearm up to the elbow joint. In other cases the wrist-joint is infected. Such cases may give rise to general sepsis.

V-shaped whitlow is recognized by its severe clinical symptoms and typical appearance. In the early stages there is often pain, redness and swelling in the palm, or on the flexor surface of the wrist. When suppuration has existed for some time and become extensive it seeks a way to the surface. Thus fistulæ are formed along the tendon-sheaths, discharging much pus, and often exposing the greenish-yellow remains of the necrosed tendon (**Fig. 96**). The orifices of these fistulæ are surrounded by flabby, unhealthy granu-

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lations, which, as mentioned before (**Fig. 56**), indicate necrosis in the deeper parts.

In *periosteal and osteal whitlows,* which generally occur at the ends of the fingers, the periosteum and bone are surrounded by pus and destroyed. In the terminal phalanx total necrosis may occur. A fistula forms and discharges the fetid, slimy pus characteristic of necrosed bone. Frequently dead bone is eliminated (**Fig. 95**). Parts of the skin may become necrosed, so that, eventually, the whole finger-joint may be lost. Beginning with sharp pain, the acute stage gradually becomes more chronic, and in this stage bone involvement may be overlooked.

In the first and second phalanges there is often infection of the joints, either secondarily to periostitis, or directly from the surface. Articular whitlow generally manifests itself by chills. The joint is fixed in a position of flexion and is very painful on movement. The capsule and ligaments are soon destroyed, and erosion of the cartilage causes grating on motion. Articular whitlow may also give rise to general sepsis.

Diagnosis

Tuberculous and **syphilitic inflammations** are more chronic and cause less pain and fever. They do not heal after incision, but require specific treatment.

It is not always easy to diagnose the **stage** of the whitlow. Patients of the working class generally come so late for treatment that there is often infection of the tendon-sheath, periosteum and joint. In other cases the pain is so severe as to suggest tendinous whitlow, while it is only subcutaneous. A correct diagnosis can often only be made after incision.

Treatment

All whitlows require **early incision.** In subepidermic whitlow the purulent bulla must be opened, its edges pared off, an antiseptic dressing applied, and the arm suspended in a sling. Subepidermic whitlow may cause infection of the deeper tissues, and there is also the danger of erysipelas. Hence, do not consider it too lightly.

Subcutaneous whitlows should be incised as soon as possible, under an anesthetic. *Schleich's* infiltration anesthesia does not work well in inflamed tissues, but regional anesthesia is very practical on the fingers. However, general anesthesia should be resorted

Fig. 97. Phlegmone interdigitalis.
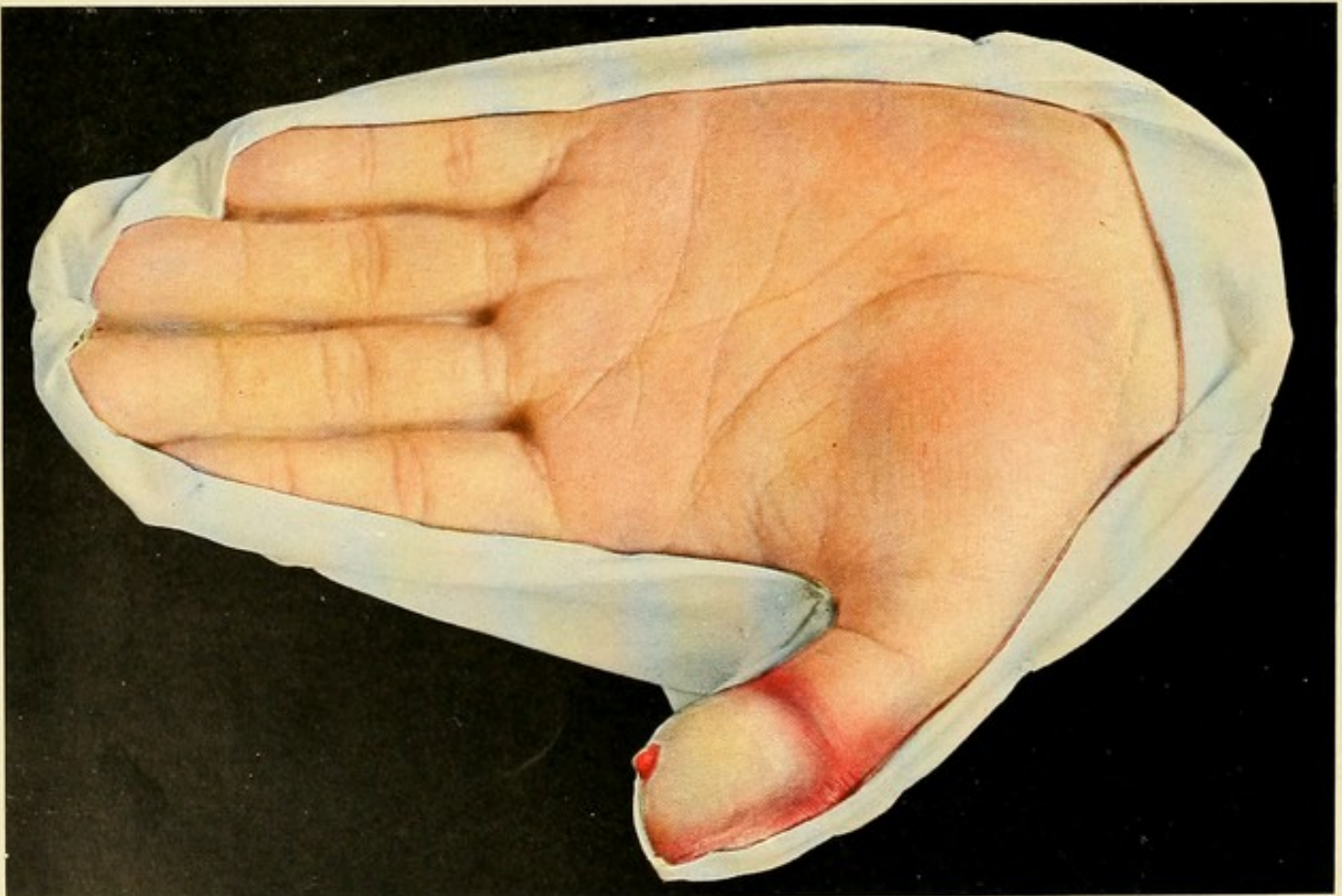
Fig. 96. Panaritium tendinosum — Phlegmone subcutanea.

to in all cases where the extent of the suppuration is not clear. Do not let yourself be influenced by the sometimes enormous swelling of the dorsal surface of the hand. The primary focus is in the palm. Therefore make an incision over the primary focus, away from the midline and the tendon. The incision must be large enough to allow of a free examination of the wound. Not ascertaining the extent of the suppuration at the time of incision is gross carelessness, which may lead to serious damage to the finger. But the incision need not be as extensive as was urged before hyperemia treatment came in favor.

There is often, in subcutaneous felon, a *"collar button"* abscess, that is, two pus collections, a superficial and a deeper one connected by a narrow perpendicular tract. Particular search must be made for this condition, so as not to neglect the deeper collection.

Tendon-sheath whitlow requires very careful treatment, in order to preserve the tendon and the function of the finger. *Bier's* passive hyperemia renders here great services, because it enables us to replace the long incisions formerly used by several smaller incisions along the edges of the tendon. Thus we do not have to fear scar retraction (**Fig. 64**) and we save the finger and its function. When carefully applied and watched, there is no danger of letting the infection spread, as has been repeatedly charged. But, unfortunately, many cases come too late for effective treatment. Saving the tendon function is out of the question: preventing general sepsis by hasty and large incisions is all that can be done.

In osteal whitlow necrosed bone must be removed if present. In the terminal phalanx it is often sufficient to remove the peripheral end. If the joint is much destroyed resection of the bone, or even amputation, may be required.

The *after-treatment* is very important in all finger suppurations, but especially in tendinous whitlow. Hyperemia methods have done away with the drains and packing formerly so much in honor. Packing in tendon sheath suppuration brings about desiccation of the tendon. The pus is gently squeezed out every day; newly formed abscesses, if any, are incised; an irrigation with warm saline solution may be given, and a large loose dressing applied. Passive motion is begun from the first day, of course with the utmost gentleness. This alone can save the tendon function by preventing the development of adhesions between the tendon and its sheath.

Patients should be kept in the hospital. Severe forms of finger suppuration are not fit for ambulatory treatment.

Fig. 98 shows a type of subepidermic whitlow, having special features on account of its location; it is the *peri-ungual whitlow* or **paronychia.** The skin is bluish-red and tender to the touch. The nail bed is red, infiltrated, and painful on pressure.

Paronychia may result from punctured wounds, tearing of the nail, foreign bodies or manicuring with dirty instruments. There is often suppuration around the nail, which is raised from its bed, may become quite loose, and generally falls. In severe cases there is much pain, fever and lymphangitis.

Syphilitic chancre of the finger often resembles paronychia. It begins with redness and hard infiltration, which develops into an unhealthy ulcer with flabby granulations. This is followed by painful infiltration of the lymph vessels and glands. This form of chancre is very chronic and painful (thus differing from most other chancres). Syphilitic chancre should be borne in mind in every case of chronic paronychia which is refractory to treatment. It is especially common in medical men and midwives.

Tuberculous infection of the nail bed may also occur among physicians and nurses. This begins as a dark-red infiltration of the skin. Nodules then develop and break down into an ulcer with flat, irregular borders. The tuberculous granulations are grayish-red and bleed easily. This affection is very chronic. The nail may be lost and replaced by thickened tissue in both tuberculous and syphilitic paronychia. In some cases the whole finger may be destroyed. The diagnosis of tuberculous paronychia can sometimes only be settled by microscopic examination, or by inoculation to the guinea pig. The diagnosis of syphilitic chancre is confirmed by finding the *spirochæta pallida* in scrapings.

If the inflammation is not around the nail bed, but under it, we are dealing with the condition called *sub-ungual whitlow*. Owing to pressure of the nail, the virulence of the infecting bacteria is increased, so that the inflammation rapidly spreads and soon leads to necrosis of the tissues. Sub-ungual whitlow causes severe pain and lymphangitis. It is often overlooked, as the changes under the nail are not at first visible, and the first sign is usually a yellow coloring seen under the nail. The diagnosis is suggested by the severe pain elicited by pressure on the nail. As the pus cannot break through the nail, it extends deeply and may cause necrosis of the terminal phalanx by infection of the periosteum. Corns and exostoses may also develop under the nail and cause inflammation with severe pain.

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Tab. LXXVIII.

Fig. 99. Unguis incarnatus.
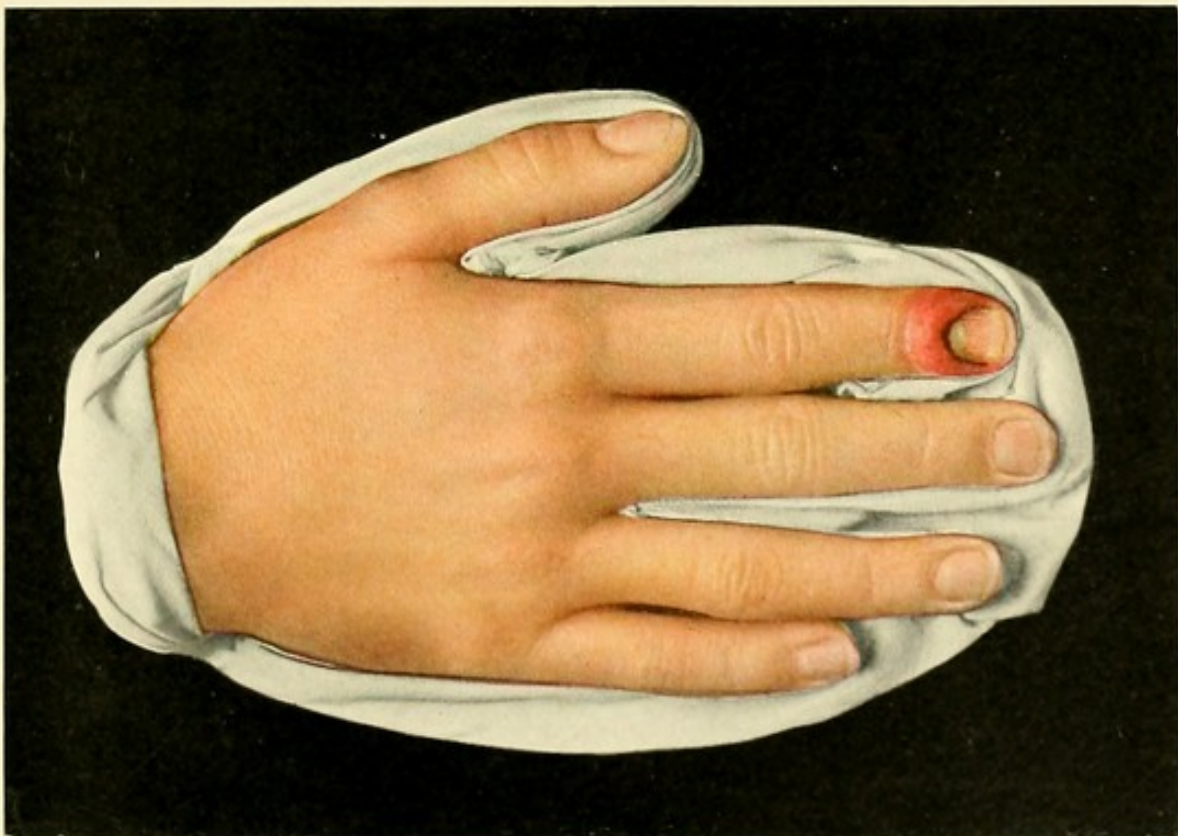
Fig. 98. Paronychia.

Paronychia requires early incision before the pus has loosened the nail; this is the only chance of saving the latter, and the growth of a new nail requires quite some time. It is best to make a horse-shoe incision through the soft parts some distance from the nail, to avoid interfering with its nutrition. The hand should be immobilized for a few days. If the nail is extensively separated it must be removed.

Tuberculous paronychia requires treatment by the sharp spoon or *Paquelin's* cautery. Syphilitic chancre calls for immediate anti-syphilitic treatment (see page 186).

In sub-ungual whitlow the nail may be pared down with a knife, under local anesthesia, so that the inflammatory area can be incised. If suppuration is extensive the nail must be removed.

Fig. 99 shows an **ingrowing toenail** on the outer side of the right great toe. The thickened soft parts have grown over the border of the nail. There is a purulent discharge from unhealthy granulations. The nail is so imbedded in the swollen soft parts that it is only partly visible.

Ingrowing toenail affects almost exclusively the nail of the great toe; generally the outer side, less often the inner side, occasionally both.

It often occurs in connection with hallux valgus (**Fig. 64**); it may also be caused by anomalies of the nails or toes, by wearing too short boots, or by cutting the nails too much at the sides..

Ingrowing toenail gives rise to severe inflammation of the soft parts next the border of the nail; first redness and swelling, afterward ulceration and granulation tissue. The inflammation is usually limited to a small area, but may sometimes spread over the whole nail-bed. The affection causes considerable pain and often prevents the patient from walking. There may be lymphangitis. If both sides of the nail are affected the symptoms are naturally more severe.

Differential diagnosis

Sub-ungual exostoses may cause inflammation around the nail, but in these cases the latter is always raised in front and is very tender to pressure. *Syphilitic chancre* has also been known to occur on the great toe, after sucking the toe (*Bockenheimer*).

Ingrowing toenail may be avoided by prophylactic treatment. The toenails should be cut straight and not too short, so that the free border extends beyond the soft parts, especially at the sides. Attention should be paid to cleanliness and to the wearing of properly made boots. In slight cases the edge of the nail may be raised from the inflamed soft parts by a small gauze tampon, or partial excision of the nail may be performed.

But in severe cases these methods are useless. Avulsion of the nail, which was formerly the procedure most frequently employed, is also ineffectual, as the condition recurs after.

The most rational method is *excision of the whole lateral border of the nail together with the inflamed soft parts,* down to the bone; taking care to include the posterior part of the matrix, so that recurrence cannot take place. The wound is dressed with aristol and sterilized gauze and immobilized for a week, after which the wound is usually healed. This was done with good result in the case shown in **Fig. 99.**

In ingrowing toenail affecting both sides the same operation is performed on each side, leaving the middle part of the nail in place.

Fig. 100 shows an inflammatory condition affecting the whole of the second toe and extending to the dorsum of the foot. The skin on the dorsal surface of the toe was at first raised by purulent blisters. After these had broken, the necrosed epidermis came away, exposing a considerable area of the corium. The redness and swelling are most marked over the first interphalangeal joint, which was very painful on movement. On the dorsal side of the joint fluctuation was present. The remains of a corn are seen on the great toe, in the form of a yellowish-white projection, together with a fistula leading to the deeper parts, where there was an infected bursa communicating with the joint. The corn on the second toe was due to its being exposed to pressure from its crooked position.

Corns are *circumscribed* thickenings of the horny layer of the epidermis. They generally occur on the great and little toes; sometimes between the toes, especially when these are crooked owing to bad boots. They also develop in connection with hallux valgus, hammer-toe (see **Fig. 64**), club-foot, etc. The more they project above the level of the skin the more painful they are to pressure. They differ from the *diffuse,* horny thickenings which are observed

Fig. 101. Phlegmone progrediens putrida.

Fig. 100. Clavus inflammatorius — Arthritis purulenta.
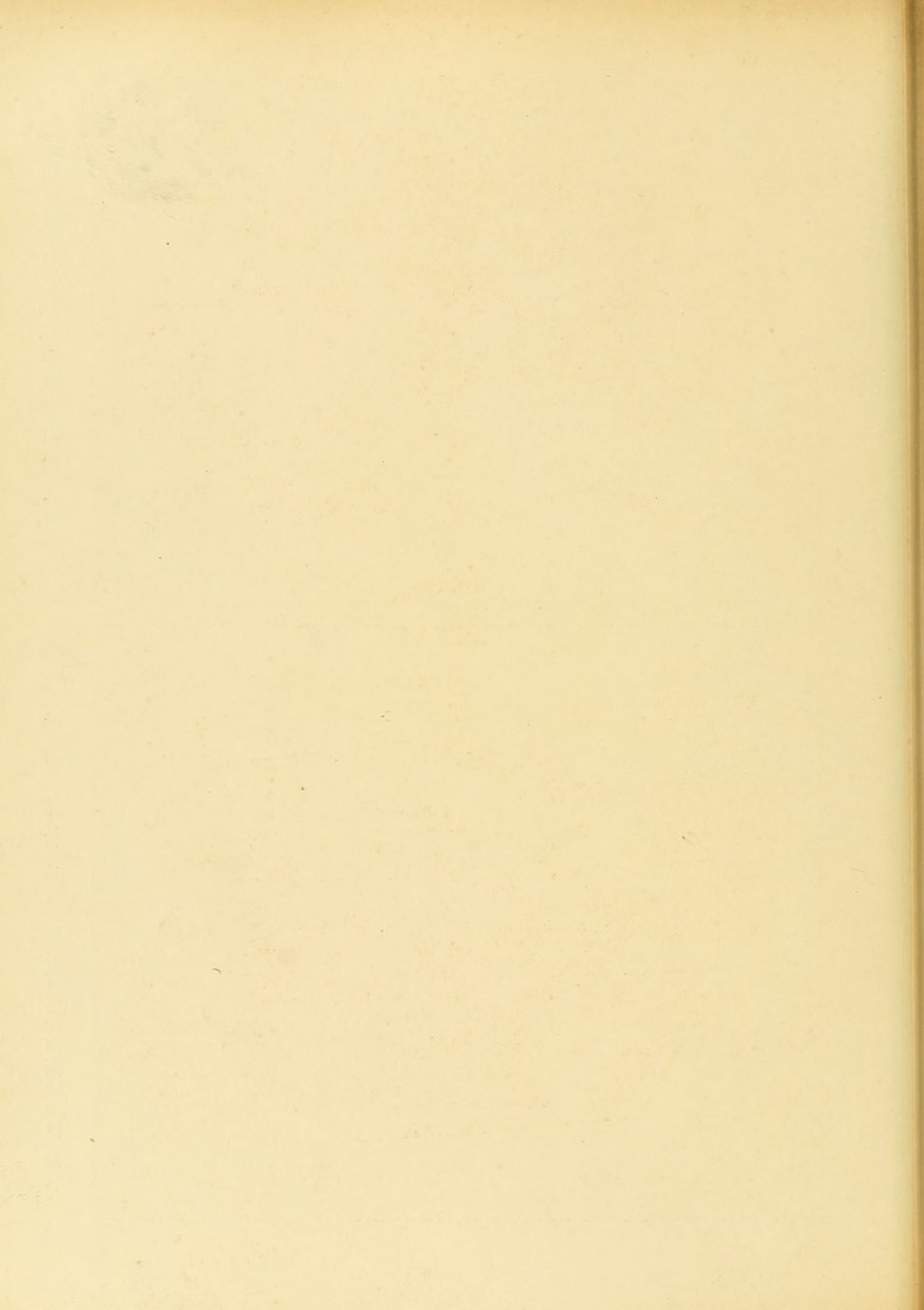
on the hands, and consist in a circumscribed horny formation, which grows from a soft, conical core situated in the depth of the cutis. When the horny layer is removed the soft yellowish-white core is seen in the centre. Lacerations caused by unskillful cutting of corns may easily give rise to subcutaneous abscess. Underneath large corns there usually is a bursa, which is liable to become inflamed (bunion) from external pressure. The inflammatory exudation from the bursa generally discharges by a fistula near the corn (**Figs. 64** and **100**). Septic infection of the bursa may arise through the fistula, and extend to the neighboring tendon-sheath or joint. Joint infection is especially frequent when the bursa communicates with the joint; and is manifested by severe local inflammation, fever, chills and constitutional disturbance. All these, and lymphangitis of the foot and leg were present in the case represented in **Fig. 100.**

Purulent arthritis may even give rise to fatal general sepsis, which has also been seen as a consequence of removal of corns with unclean instruments.

Treatment

Prophylaxis of corns consists in cleanliness and the wearing of proper boots. If one forms it should be removed with a *sterile* knife. It is not sufficient to pare off the horny layer; the deeply situated core must also be removed, otherwise recurrence takes place. Other methods, such as the application of salicylic collodion (10%) only loosen the horny layer and do not prevent recurrence.

If a bursa forms under the corn, it must either be incised and packed, or, better, excised. If suppuration spreads to the joint this must be opened; in some cases resection or exarticulation may be necessary.

In the case shown in **Fig. 100** the joint was opened on the dorsal surface by a transverse incision, and the superficial suppuration by another incision on the dorsum of the foot. The corn and bursa were excised subsequently.

Fig. 101 shows a **progressive gangrenous phlegmon,** due to a combination of pyogenic and putrefactive bacteria, in a diabetic patient in whom, owing to the general debilitation of the body, such infections are always severe. But this is more than the ordinary progressive suppuration common in diabetics. Following a slight wound of the great toe, a subcutaneous suppuration rapidly spread

to the tendon-sheath and the joint, necessitating amputation of the toe. Although this operation was performed in sound tissue, further suppuration occurred on the sole of the foot and gained rapidly, so that the soft parts, sinews, muscle and fascia, were destroyed and the metacarpus was involved, as shown by the great swelling around it. The periosteum was already loosened and the cortex and medullary cavity infected. A high temperature, chills, a dry tongue and drowsiness were suggestive of impending general sepsis, but after a while the infection assumed a more chronic course. Under lumbar anesthesia, amputation was performed, above the knee, because of the thrombo-phlebitis and lymphangitis in the leg, and of the advanced arteriosclerosis. The patient recovered. (Compare with **Fig. 139, *diabetic gangrene*)**.

The appearance of the wound in this form of inflammation is characteristic. Owing to the fibrinous exudation, the wound is coated with a diphtheroid membrane. This condition has been called "wound diphtheria"; but this is a misnomer, and it is better to use the term diphtheroid (true infection of wounds by the diphtheria bacillus is rare). In putrefactive phlegmon dry, unhealthy granulations are present along with the diphtheroid membrane. There is also a sanious, fetid, dirty discharge from the wound, containing numerous pieces of necrosed tissue. Similar conditions are found in wounds in general infection.

If an incision is made in these cases, all the tissues are seen to be bathed in a turbid, green fluid and in a state of necrosis, often consisting only of yellowish-green necrotic shreds. The skin, fascia, muscles and tendons are the first to be destroyed, while the bones resist longer, but are finally involved.

Treatment

In those cases free incisions must be made in the diseased tissues as early as possible, as general sepsis develops rapidly and toxemia is extremely marked. If the process continues its onward march in spite of the incisions, amputation through healthy tissues must not be delayed too long; otherwise the patient will succumb in spite of amputation.

In the phlegmonous inflammations occurring in diabetes, which often begin in the toes and spread destruction over the whole foot in a few hours, the conditions are especially serious. If, after extensive incisions, the temperature does not immediately fall, amputation must be performed; otherwise general infection will supervene rapidly. In

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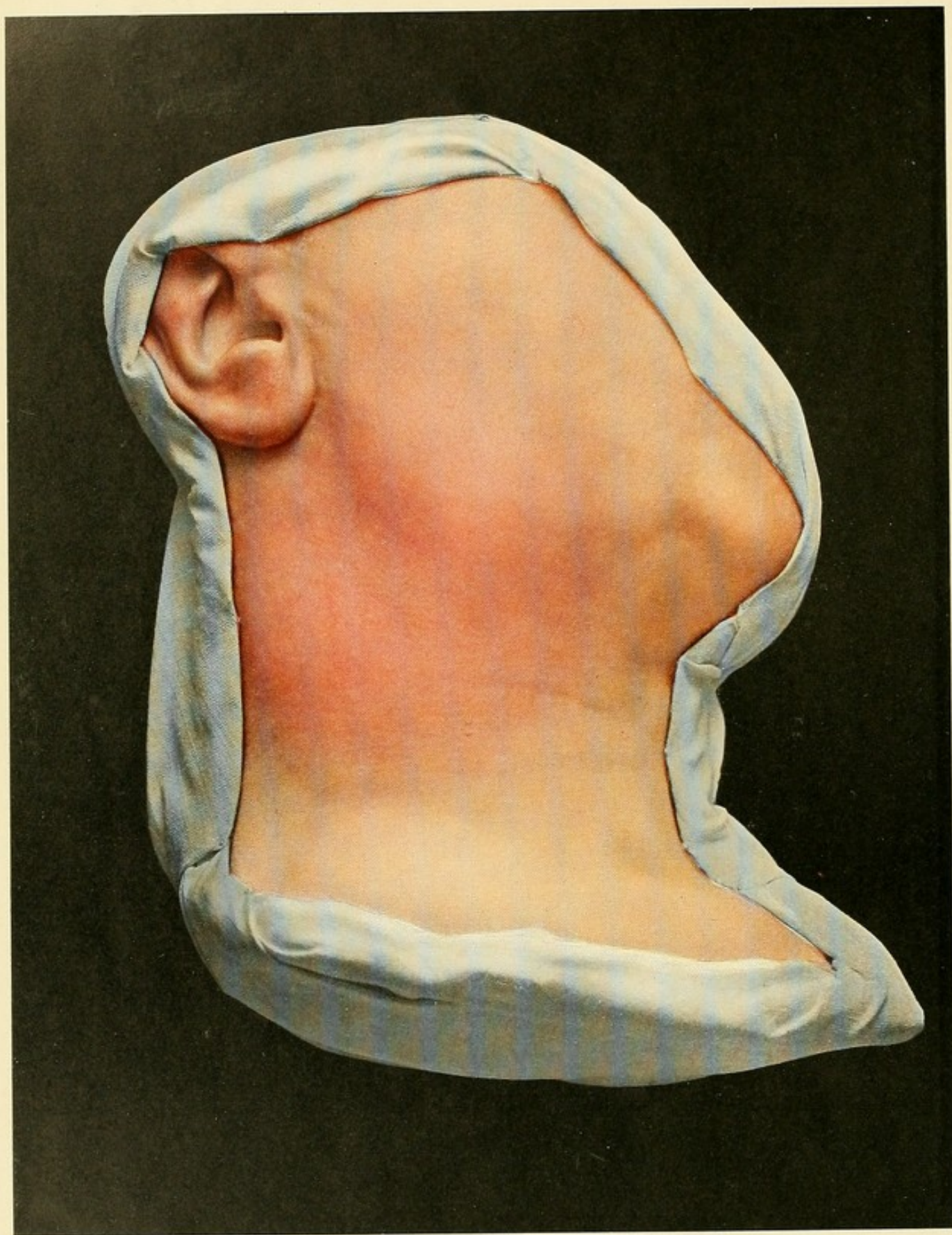
Fig. 102. Phlegmone colli — Phlegmon ligneux.

any case of phlegmonous inflammation in a diabetic patient death may result from coma or heart failure.

Fig. 102 shows an *acute inflammation of the submaxillary lymph glands* with formation of an *abscess* under the skin.

Subcutaneous and subfascial adenophlegmons of the neck are common, owing to the numerous groups of lymph glands in this region, and the multiple places from which absorption may take place. All septic conditions of the mouth and pharynx may be the starting point of an adenophlegmon. Eczema and other affections of the head and face may play the same rôle.

Staphylococci are the usual agents, sometimes streptococci, and then the infection is more severe and may assume the highly virulent character of the *diffuse cellulitis of the neck,* known as *Ludwig's angina.*[1] Occasionally other bacteria are present. Putrefactive bacteria may reach the abscess cavity from the mouth; hence the peculiarly offensive smell of the pus in many of these perebuccal abscesses.

Subcutaneous phlegmon in the neck manifests itself by redness of the skin, inflammatory infiltration and fever; later on fluctuation can be made out. In nearly all cases a circumscribed abscess forms on one side of the neck. Large abscesses may cause dyspnea by pressure on the larynx, and dysphagia by pressure on the esophagus.

Lateral abscesses are due to suppuration of the submaxillary group of glands; median collections are developed in the submental group and are due to lesions of the lower lip.

Subfascial suppurations in the neck arise from the deep lymphatic glands. They develop after lesions in the pharynx, esophagus and larynx, also after tonsillitis and scarlet fever, and are more dangerous on account of their deep situation. They develop with fever, chills and diffuse inflammatory infiltration in the neck, while fluctuation is often absent. This deep suppuration manifests itself by cyanosis of the face, oblique position of the head, trismus of the jaw, attacks of asphyxia and difficulty in swallowing. The pus may burrow down to the supraclavicular fossa or in the axilla.

[1] The meaning of the term "Ludwig's angina" is not always altogether clear, because it has been applied to a variety of conditions. Some consider "Ludwig's angina" as the phlegmon of the submaxillary *salivary* gland; others use the name for all cases of diffuse cellulitis originating in the upper part of the neck. The correct definition is: a diffuse cellulitis of the neck beginning in the floor of the mouth as a sublingual phlegmon (*Thomas*).

It must not be forgotten that there are submaxillary *lymph* glands within the fascial compartment that encloses the submaxillary *salivary* gland; which lymph-glands often become the seat of septic processes of high virulence because of the inextensibility of the submaxillary fascia which encloses them.

In *Ludwig's* angina there is no pus, but a dirty, fetid, greenish fluid, which infiltrates all the tissues. Dysphagia and dyspnea are extreme, and the toxemia is very marked. Such cases are generally fatal if not operated on promptly (sepsis, asphyxia). Diffuse inflammation may also occur after operations on the neck, larynx and esophagus, and cause death by extension to the mediastinum.

A special type of cervical phlegmon is interesting, namely, the so-called **"woody phlegmon"** (*phlegmon ligneux*). It is a chronic inflammation of the neck, consecutive to lesions of the mouth and pharynx, probably caused by bacteria of slight virulence, which give rise to an infiltration of wood-like hardness, often extending over the whole neck, with but mild inflammatory symptoms. The skin is slightly blue, edematous, and pits on pressure. There is no fever nor pus formation. The infiltration may cause dyspnea by pressure on the larynx. When incised, a dirty, greenish-yellow fluid is seen in the subcutaneous, subfascial and intermuscular tissues, extending through the whole region of the neck.

Differential diagnosis

This has to be made from **alveolar periostitis** (**Fig. 104**), **osteomyelitis of the lower jaw** (**Fig. 105**), **tuberculous adenitis** (**Fig. 124**), and *cystic tumors in the neck* (blood, dermoid, sebaceous, branchial cysts). Changes in the bone are revealed by an incision in the case of periostitis and osteomyelitis. Acute symptoms and fever are absent in the other formations, but suppuration of a cystic tumor may resemble glandular suppuration. In cases of deep suppuration in the neck, **retro-pharyngeal abscess** must be borne in mind, especially in infants.

Woody phlegmon of the neck may be mistaken for incipient **actinomycosis,** but the latter soon gives rise to a fistula, which discharges pus mixed with the characteristic yellow bodies (**Fig. 115**).

Treatment

The treatment is **incision** in all cases. In subcutaneous phlegmons with a tendency to become circumscribed, incision should be deferred until an abscess forms. Under local anesthesia an incision is made through the skin at the lowest part of the abscess, and the pus evacuated by means of blunt dressing forceps. In the submaxillary region the facial nerve and vessels must be avoided.

But in all deep suppurations of the neck we must not wait for the appearance of a superficial abscess, nor for fluctuation. A free

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Fig. 103. Periostitis alveolaris purulenta — Parulis.

incision must be made along the inner border of the sterno-mastoid muscle. Extensive cases require counter incisions. In *Ludwig's angina*, the thermocautery is useful. No rubber drains must be left in contact with the big blood-vessels of the neck, for fear of pressure ulceration. Large incisions in the neck heal very well and often leave only surprisingly small scars.

Woody phlegmon sometimes requires multiple incisions.

In all cases of cervical cellulitis in which there is much infiltration of the floor of the mouth, with difficulty in breathing and swallowing, preliminary tracheotomy is advisable, as death might occur from sudden edema of the glottis during anesthesia.

Fig. 103 shows a ***purulent alveolar periostitis*** of the lower jaw, with formation of a subcutaneous abscess, which is the usual termination (*Parulis*).

This common condition is caused by lesions of the gum (tooth extraction with unclean instruments), fractures of the jaw, *dental caries* or *fistulæ*.

Infection of the periosteum of the alveolar portion of the lower jaw gives rise to a circumscribed subperiosteal accumulation of pus, which descends to the submaxillary region and lies over the fascia, covering the submaxillary gland. The signs of purulent inflammation are most apparent in this region, while symptoms at the seat of infection are often slight.

The symptoms commence with fetor of the breath, fever and chills, and inflammatory infiltration in the submaxillary region. Soon afterward the presence of fluctuation indicates abscess formation, after which the symptoms somewhat abate. In most cases the suppuration is circumscribed, but sometimes it is diffuse and causes considerable infiltration of the soft parts, swelling, and redness of the side of the face. There is often in those cases trismus and edema of the buccal mucosa, with difficulty in mastication and often dyspnea. In these diffuse forms there are severe constitutional symptoms—chills, high fever, headache, etc.

Although the circumscribed form is not dangerous, the diffuse form may be quite serious, especially when improperly treated. If incision is too long delayed, as was the case in the patient shown in **Fig. 103,** the bone may be denuded of periosteum for a considerable length, or there may develop osteomyelitis of the jaw (**Fig. 104**).

In the upper jaw, infection of the periosteum may also cause

subperiosteal suppuration, which here does not meet with such favorable anatomical conditions for propagation to the subcutaneous tissue as in the lower jaw. Small abscesses caused by morbid conditions of the teeth may burst into the mouth and cause no trouble, but more virulent infections may cause osteomyelitis of the upper maxilla, which rapidly spreads to all the bones of the face, and often causes death by general pyemia. In these cases there is infiltration of the upper part of the face, edema of the eyelids, high temperature, chills, headache, etc.

In abscesses due to alveolar periostitis, staphylococci are generally associated with some of the putrefactive bacteria of the buccal cavity. Hence the fetid, dirty, reddish-brown pus, mixed with broken-down tissue that is generally found on incision.

Diagnosis

Subcutaneous abscesses due to alveolar periostitis of the lower jaw are distinguished from *cervical lymph gland suppuration* by the history, the finding of a purulent peridental focus in the mouth, and the fact that the swelling of cervical phlegmons is *all below* the lower border of the jaw, while that of periosteal abscess is situated higher and *encroaches upon the face*. (Compare **Figs. 102** and **103**).

Parulis of the upper jaw may be mistaken for *antral suppuration*, but transillumination of the antrum will show that the latter is intact.

Acute osteomyelitis of the lower jaw begins with more marked symptoms. *Actinomycosis* is, on the contrary, chronic and painless from the outset, and first infiltrates the floor of the mouth.

Treatment

Mild cases of periostitis can be treated by the hyperemia method (hot fomentations, figs boiled in milk applied on the gum), but too much time must not be wasted. Many cases can be *incised through the gum*. Only when things have gone very far, and there is distinct subcutaneous abscess must an *external incision* be made, because with the latter there is sometimes a risk of permanent external fistula. Of course, the treatment of the tooth, either conservative if it is worth while trying, or avulsion, is of prime importance. However, it is best not to do anything to the tooth during the acute stage of the periostitis.

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Figs. 104 to 107, inclusive, represent various types of **osteo-myelitis** of different bones; either **acute** (Fig. 105) or **chronic** (Figs. 104, 106, 107).

The term osteomyelitis is applied to pyogenic affections of bone in general, while in the stricter sense these are divided into purulent periostitis, osteitis and osteomyelitis. Since all three parts of the bone are generally the seat of suppuration and clinically the process can only be localized to the bones as a whole, and as the majority of cases begin with infection of the bone-marrow, the name osteo-myelitis is rational.

Infection of the bones may result, by continuity or contiguity, from lesions of the soft parts, *compound fractures, operations* (this was common after amputations in the pre-antiseptic days); after *pyogenic affections of the neighboring parts* (subcutaneous abscess, whitlow, otitis media). In the latter cases the periosteum is first infected, the cocci then invade the Haversian canals in the cortex and contaminate the medullary cavity.

Infection may also, and in fact does more frequently take place through the blood; the medulla is then first infected, and the suppuration spreads to the cortex and periosteum, finally appearing as a sub-cutaneous abscess.

As in all pyogenic infections, the great majority of cases are caused by the *staphylococcus pyogenes aureus;* while the staphylo-coccus albus, the pneumococcus, the streptococcus and the *Eberth* bacillus are less frequently found in the pus.

In all pyogenic affections in which microbes circulate in the blood—and this does not only include such septicemic diseases as typhoid fever, pneumonia, or scarlet fever, but many, if not all, so-called "local" infections (see page 157), furunculosis, whitlow, tonsillitis, otitis media—the bone marrow is infected by cocci, but the power of resistance of the body is generally sufficient to withstand the actions of the latter, which remain harmless till the defensive process is weakened for some reason, such as a fracture, overexertion, ex-posure to cold, etc. Osteomyelitis may thus occur after injury to a bone, even after a slight contusion. In this case the resulting effusion of blood favors further growth of the cocci and leads to infection. Therefore, according to circumstances, suppuration of the bones may develop sometimes directly after, and sometimes a long time after septic conditions in other organs of the body; again, according to the number and virulence of the bacteria, it may take an acute or chronic form, with correspondingly violent or mild symptoms. As usual, the-

process begins at the seat of infection with *hyperemia and exudation;* then occur **suppuration, degeneration** and **regeneration;** these processes assuming a special type corresponding to the structure of the bone. Thrombo-phlebitis may develop and give rise to metastatic infection by embolism in other parts of the body (bones, endocardium, meninges, etc.).

As the great majority of cases arise from blood infection, it is clear that the bones most liable to infection are those which are most richly supplied with blood-vessels, especially during their *period of growth,* when they are most vascular. The *diaphyses* of the long bones are thus most often affected at their *junction with the epiphyses.* The lower ends of the femur and radius and tibia, and the upper ends of the humerus and tibia, are the places of predilection. Osteomyelitis is less common in the short and in the flat bones. It is also rare after the thirtieth year. According to the statistics of *Garré,* in 20% of the cases several bones are affected simultaneously.

The *symptoms* of acute osteomyelitis are more severe than those of any other pyogenic affection. The deeper the infection, the greater is the virulence of the bacteria. Bacteria in the bone-marrow are under greater pressure than in any other tissue, and this increases their virulence. In young individuals osteomyelitis often begins suddenly after an injury, with high fever, chills, pains in the joints and severe constitutional disturbance. Pain on pressure on a localized point of the bone, or on movement, and loss of function are suggestive of an affection of the bones. Serous effusion soon takes place in the nearest joint. Changes first appear under the skin when pus collects under the periosteum. The subperiosteal abscess appears as a sharply defined fluctuating swelling with hard borders, and the skin over it is tense and reddish-blue. If the subperiosteal abscess bursts, it gives rise to intermuscular and subcutaneous infiltration, with redness and swelling of the skin, and edema of the soft parts; the regional lymphatic glands are swollen and painful.

Although operation often only reveals a subperiosteal abscess, especially in children, in cases of hematogenous origin the cortex and medulla of the bone are also affected. Involvement of the cortex is shown by the presence of yellow spots on the surface, which correspond to small holes discharging pus. After removal of the cortex, the infected medulla shows reddish-brown or yellowish spots, which may lead to the formation of a circumscribed abscess, or to diffuse suppuration in the medullary cavity. If the condition is not recognized early and the spread of infection checked by operation, separa-

tion of the epiphyses or infection of the joint may occur, or general sepsis with death in a few days. In extensive disease the whole bone is whitish-yellow; white from bloodlessness due to thrombo-phlebitis, and yellow from pus formation. Numerous pits are seen from which pus has been discharged under the periosteum.

The amount of **necrosis** corresponds to the degree and extent of infection. In subperiosteal necrosis the destroyed cortex and medulla may regenerate without loss of substance, especially when the pus has been given an early outlet. If the cortex has been for some time the seat of extensive purulent inflammation, necrosis must result with the formation of a *sequestrum*. According to the extent of the inflammation, this necrosis will be limited to part of the bone or involve the whole thickness and length of the bone (**Fig. 107**). In disease of the cortex the sequestrum is generally lamelliform, slightly corroded and pitted; in disease of the medullary cavity the sequestrum is, to a certain extent, a cast of the cavity, and trough-shaped.

The sequestrum in osteomyelitis is large and continuous, thus differing from the sequestra in tuberculous bone disease, which are generally multiple, small and much corroded. Complete necrosis of the diaphysis occurs in acute cases which have been operated upon too late and in chronic cases.

The sequestrum becomes separated from the healthy bone by a zone of inflammatory demarcation, more or less rapidly according to its size. In extensive necrosis the demarcation process may last for months, so that patients who escape death from general infection may succumb from exhaustion, albuminuria or amyloid degeneration of the kidneys. Spontaneous expulsion of the dead bone should be assisted by operation (sequestrotomy).

The **regenerative** or osteoplastic process goes hand in hand with the degenerative. The suppuration not only causes necrosis, but also irritation, which stimulates the osteogenic activity of the periosteum. This results in thickening of the cortex at the seat of necrosis; and in cases of total necrosis, complete repair of the destroyed bone (at least in young subjects). This irregular formation of new bone is sometimes called the "sequestral capsule." There are numerous holes in this capsule where the periosteum has been destroyed. From these holes pus is discharged from the zone of inflammatory demarcation; and eventually the sequestrum, after passing through one of these apertures, is eliminated through a fistula in the skin

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(**Fig. 107**). The X-rays are useful in showing the extent of necrosis, and also separation of the epiphyses.

The whole process of degeneration and regeneration takes much longer than in suppurative diseases of the soft parts, and the acute stage is followed by a chronic stage after the pus has been evacuated spontaneously or by operation. However, an acute relapse may occur at any time during the chronic stage, especially after improper treatment, or after a trauma.

Besides acute osteomyelitis, there exists a form which is chronic from the outset. In these cases there is often a history of previous acute inflammation of the bone, and the condition is really a mild recurrence, often at the age of puberty, or later in life; hence bones which have been previously affected with osteomyelitis must be deemed as *loci minoris resistentiæ* and protected against injury and overexertion.

The clinical symptoms in these cases often resemble those of chronic rheumatism, but the pain is localized to one bone, or sometimes a definite part of a bone. There is often a history of pyogenic disease in youth, and scars and fistulæ may be found in the bone concerned or in others. The affected bone is often very tender to pressure at certain points. In the course of time it becomes thickened, and the diaphysis lengthened. The growth in thickness may be enormous at the seat of disease, both the periosteum and cortex sharing in the hyperplasia.

The changes in chronic osteomyelitis are as follows: Sometimes there is a small sequestrum in the interior of the bone, showing in an X-ray picture as a clear spot surrounded by bony proliferation, sometimes a circumscribed abscess in the medullary cavity, shown by the X-rays as a round space surrounded by bone. If bony proliferation is absent the X-ray pictures resemble those given by tumors or cysts in the bone. The diagnosis of chronic osteomyelitis, therefore, may be difficult when there is no history or evidence of former osteomyelitis. Pain on pressure suggests the infectious nature of the disease.

If large portions of the cortex and medulla are affected by chronic osteomyelitis the result is large sequestra, which seek a way to the surface in spite of the considerable formation of new bone. In these cases we find numerous holes in the bony capsule, subcutaneous abscesses and fistulæ (**Fig. 106**); while the whole bone is thickened, and the X-rays show changes in the periosteum, cortex and medulla.

A third form of chronic osteomyelitis is limited to the periosteum, under which a hyaline sero-mucoid fluid develops, forming a sharply

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defined, fluctuating swelling with hard borders. This has been called *albuminous periostitis,* but is a form of osteomyelitis. Staphylococci are present in the fluid.

All these chronic forms are due to infection by staphylococci of slight virulence. However, every chronic osteomyelitis may become acute, especially when the bones are exposed to the effects of over-exertion, injury or massage (wrong diagnosis or osteopathic treatment).

In the long bones, both acute and chronic osteomyelitis may cause disturbance in growth, shortening or lengthening of the limb, spontaneous fractures and pseudarthrosis. Chronic osteomyelitic fistulæ may give rise to carcinoma (see page 23).

Although the great majority of cases of acute and chronic osteomyelitis affect the long bones, both forms may occur in the short and flat bones; in the skull, after compound fractures, incised and punctured wounds; in the scapula, pelvic bones and vertebræ; in the bones of the face, after tooth extraction. As the cortex is thin in these bones, there is greater destruction. Osteomyelitis of the cranial bones may spread through the diploë to half the skull, form large sequestra of the inner table, and epidural abscess. In the scapula the whole bone may be destroyed by multiple abscesses and sequestra, necessitating complete removal of the bone (**Fig. 105**). In osteomyelitis of a facial bone, infection may involve all the bones of the face, causing extensive destruction and consequent deformity. Osteomyelitis of the cranial and facial bones may give rise to meningitis.

In streptococcic osteomyelitis the pus is thinner and very abundant, and the disease is more severe, like all streptococcic infections. In these cases the skin usually shows erysipelatous reddening.

Osteomyelitis due to typhoid bacilli or pneumococci can be distinguished from the other forms only by the history and bacteriological examination.

Diagnosis

Acute osteomyelitis, at the onset, may be mistaken for any general infectious disease causing high fever. The only diagnostic sign is then the *finding of tender points in the juxta-epiphyseal region of bones.* Search must always be made for these points in obscure febrile conditions of children.

A **deep diffuse phlegmon** resembles osteomyelitic abscess. Incision is the best way to clear up the diagnosis. Propulsion of

bones from below upward is painful in osteomyelitis; and not in abscess independent from the bone.

The chronic forms may be confused with **tuberculous** or **syphilitic osteitis.**

Tuberculous bone disease *generally affects the epiphyses,* while osteomyelitis *attacks the diaphyses.* Osteomyelitic fistula has hard borders and bright red granulations, and passes directly to the bone, while tuberculous fistula has *yellow,* slimy *granulations,* irregular borders and an irregular course through the deep parts (**Figs. 125** and **130**). In osteomyelitis the pus is reddish brown, in tuberculosis it is *thin and greenish yellow.* In doubtful cases an incision will decide the diagnosis; in osteomyelitis the *periosteum and cortex will be found thickened* and the *sequestrum large and continuous;* in tubercular bone disease there are *multiple, small corroded sequestra.* In other words, osteomyelitis tends to **hyperostosis;** tuberculosis only to **destruction of bone** without regeneration. Besides, osteomyelitis generally has had an *acute onset;* and, if dating back to childhood, causes much more marked disturbances in the skeletal growth.

Syphilitic osteitis is recognized by the anamnesis, the *Wassermann* or luetin reaction, the *pains more intense during the night,* and the X-ray findings, showing a diffuse thickening of all layers of the bone, and a uniform dark shadow with irregular borders, corresponding to the periosteum; while, in osteomyelitis, dark shadows together with clear spaces are shown, corresponding to sequestra and abscesses respectively. If fistulæ form in syphilitic bone disease they present the characteristic sharp borders and prolific granulation tissue around them (**Fig. 122**).

Osteitis deformans (*Paget's* disease) is characterized by affecting the whole extent of both tibias, and by the early appearance of marked curvature.

Sarcoma and bone-cysts may also in some cases be difficult to distinguish from chronic osteomyelitic abscess, even by the X-rays. However, the rapid increase in case of sarcoma soon dispels any doubts, and an exploratory incision is always justified.

Treatment

Acute osteomyelitis calls for early incision and opening of the bone-marrow canal, where lies the primary focus of infection. This is done with a concave chisel; the bone marrow is scraped out: this

Tab. LXXXII.
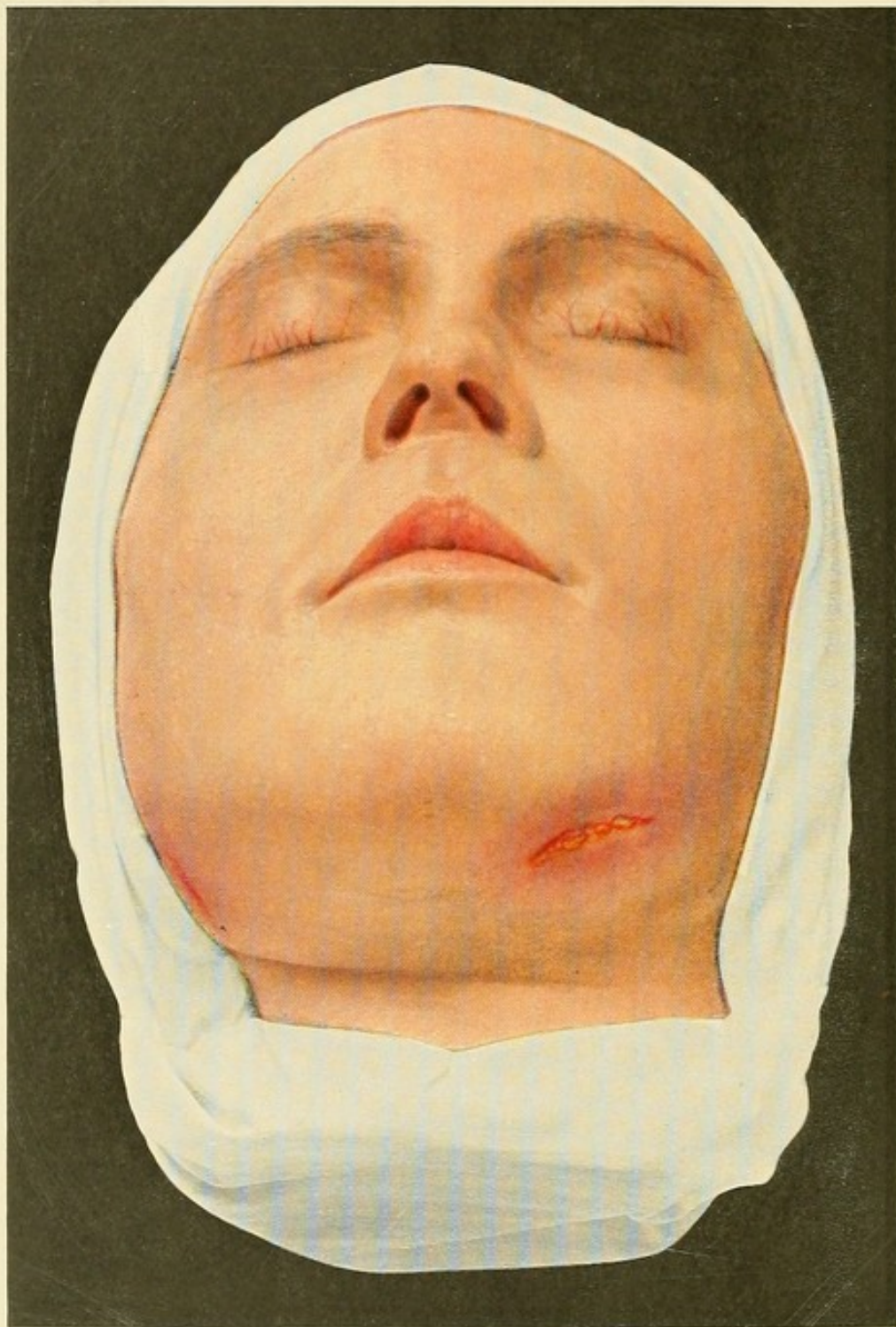
Fig. 104. Osteomyelitis maxillae inferioris.

does not impair the nutrition of the bone. The wound is drained and loosely packed. Complete immobilization is necessary.

In chronic osteomyelitis it is best to wait till the sequestrum is completely loosened and new bone has begun to form around it (X-ray examination) before performing sequestrotomy. If there are subcutaneous abscesses these must be opened. As small sequestra and abscesses often cause considerable pain, in some cases the indication is to chisel open the bone, even if the X-rays show no changes. The operation is then troublesome, as the small sequestrum or abscess is often situated deep in the middle of hardened sclerotic bone. Fistulæ in chronic osteomyelitis must be freely opened up and the exuberant bone removed. The cavity remaining in the bone after chiselling must be left open and drained till healing takes place from the bottom. Immediate plugging of the bone cavity with *Mosetig's* iodoform-wax mixture renders good service when all sequestra have been removed and the cavity is clean. Even when the filling mass is subsequently expelled, it has the advantage of making the dressings easy and painless (*Homans*), while removal of a packing from a granulating trough in a bone causes much bleeding and suffering. Therefore, as soon as the cavity is well granulated, it is scraped, disinfected with peroxide lotion, dried with *Hollander's* hot-air apparatus till all oozing is absolutely checked, and filled with a mixture of iodoform, glycerin and spermaceti. Whenever possible, the periosteum should be united over the plug and a covering of skin made over the cavity. Strict asepsis is necessary.

When the whole diaphysis of a bone has been destroyed, bone transplantation may be indicated. Thus the fibula may be used to replace a missing tibia (*McEwen*), or a segment of rib to replace the lower jaw, or splinters of bone laid in the empty periostic sheath of a humerus. In the first place, the fibula hypertrophies and assumes the function of the tibia; in the latter cases, the transplanted fragments serve only as a temporary support and a stimulant to new bone formation, and slowly undergo resorption.

Frequent recurrences in chronic osteomyelitis, with emaciation, albuminuria, amyloid degeneration, etc., necessitate amputation. Contractures must be treated by extension on a splint, or when they cannot be extended, by resection.

In flat bones subperiosteal removal of the whole bone is often necessary (e.g. scapula, see page 155). This may be followed by complete regeneration and restoration of function. In osteomyelitis of the cranium sequestra and epidural abscesses must be evacuated

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through a large trephine hole, which can afterward be repaired by bone grafting.

Fig. 104 shows *chronic osteomyelitis* of the lower jaw in a girl of 19. It developed after a tooth extraction. It may also result from neglected cases of suppurative alveolar periostitis (**Fig. 103**). A painless, diffuse swelling of the lower jaw slowly developed. The skin gradually became tense, red and edematous: a fistula was produced; it was opened up; the discharge decreased, but the fistula did not close, because necrosis had occurred. Radiographs showed a diffuse swelling of the bone.

A special occupational necrosis of the lower jaw is of very particular interest, namely, *phosphorous necrosis* (phossy jaw). It occurs in workers in white phosphorus, the vapor of which causes ulceration of the gums, from which buccal germs invade the periosteum and bone. The whole of the lower jaw becomes greatly swollen. The teeth progressively loosen and fall, while the gums are ulcerated and fetid; many patients succumb to pneumonia or general sepsis. The bone becomes both sclerosed and brittle. After some years (if the patient survives, the mortality is as high as 60%) total necrosis occurs with a row of fistulæ along the border of the jaw. As phossy jaw cannot be prevented, even with the best care of the mouth, most countries (the United States recently) have prohibited the use of white phosphorus in the manufacture of matches.

A similar condition observed in workers in mother of pearl is much less severe; it undergoes spontaneous resolution, if the patients change their occupation.

Chronic osteomyelitis of the lower jaw, which is much more frequent than the acute form, must be diagnosed from *actinomycosis* (**Fig. 116**). In the latter the swelling is situated in the floor of the mouth and in the muscles and only later on extends to the bones.

In the stage of painless swelling, chronic osteomyelitis may resemble *cystic adenoma* of the jaw. *Malignant tumors* are easily excluded by their rapid growth.

The treatment is early incision; later on, extraction of sequestra. In phossy jaw partial resection is useless, and owing to the total character of the necrosis, subperiosteal resection of one or both sides of the jaw should be performed. After this, regeneration of the jaw may take place if the periosteum has been preserved, and relapses

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Fig. 105. Osteomyelitis scapulae acuta.
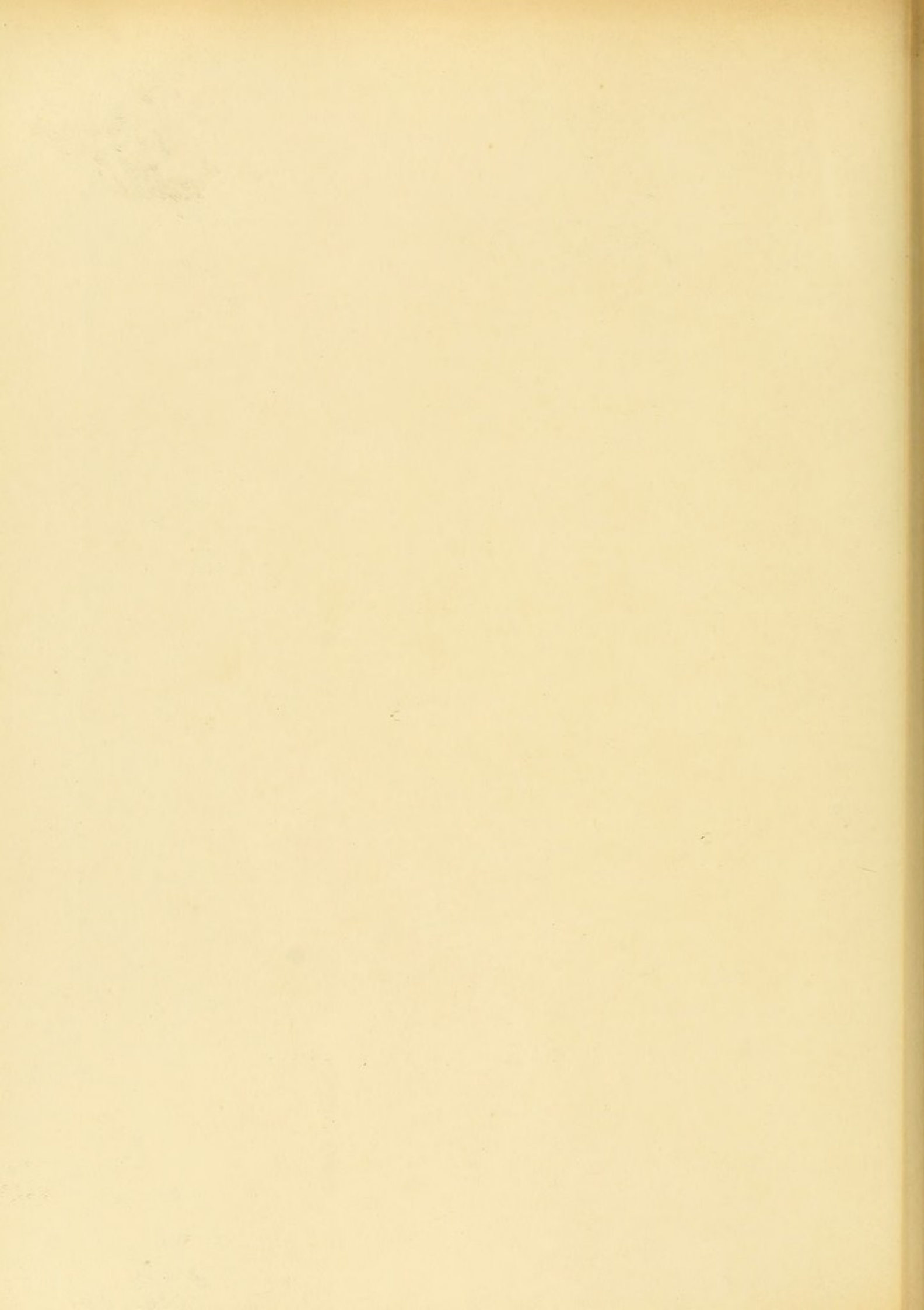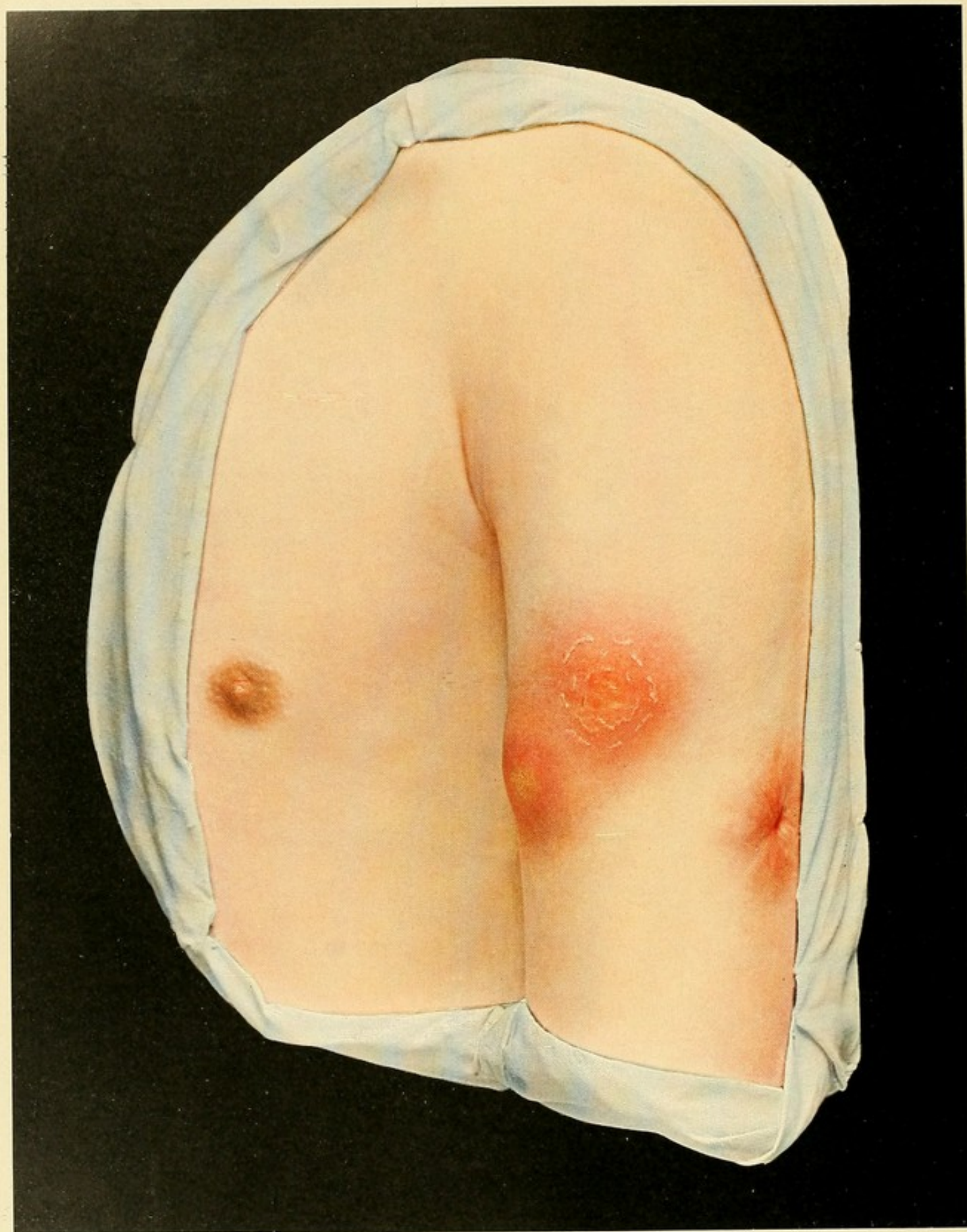
Fig. 106. Osteomyelitis humeri chronica.

are avoided. In all cases of total resection of the jaw bone grafting (see page 153) should be resorted to.

Fig. 105 shows a case of *acute osteomyelitis of the scapula* developed a few days after an injury. A swelling appeared over the whole scapular region as far as the supra-clavicular fossa, accompanied by fever and chills. The skin became red and mottled, and a large fluctuating subcutaneous abscess developed. The function of the shoulder-joint was abolished. An incision was made and pus evacuated; the bone at the seat of injury was infiltrated with pus. Healing took place without any necrosis.

In osteomyelitis of the scapula, especially when due to blood infection, an abscess usually forms at the anterior border of the scapula, as the osteomyelitic focus in this mode of infection is situated in the body of the bone. The pus is at first limited by the subscapularis muscle; on the other hand, the pressure of the muscle causes rapid extension of suppuration in the medulla of the bone. The abscess may thus not be recognized till it breaks through into the axilla. An early symptom of osteomyelitis of the scapula is painful effusion into the shoulder joint; on this account it may be mistaken for an affection of that joint, the true seat of disease only being revealed after incision. In doubtful cases the anterior surface of the scapula should be exposed by an incision in the axilla. In most cases of osteomyelitis of the scapula, the wound does not heal after incision of the abscess; the occurrence of multiple abscesses and necrosis is unavoidable, owing to the extension of suppuration through the medulla of the bone. For this reason the disease may last for years. In these cases, and also in acute cases where incision shows extensive destruction of the bone, subperiosteal total extirpation of the scapula is indicated, taking care to preserve the muscular attachments and the important nerves. (This is generally indicated in acute osteomyelitis of the flat bones, which often gives rise to early general infection.) After total extirpation of the scapula relapses are avoided, and complete regeneration of bone with normal function is possible (*Bockenheimer*).

Fig. 106 shows a painful club-shaped swelling of the left humerus, which gradually developed at the age of puberty, in a patient who had frequently suffered from tonsillitis in childhood. The patient

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attributed it to over-exertion at his work as a blacksmith. A year after the onset, a fistula opened at the posterior and external side of the arm, with hard borders and red granulations at its orifice. A probe passed down the fistula discovered rough bone, denuded of periosteum. Subcutaneous abscesses formed at the front of the arm, where the skin was thin and reddened. Examination by the X-rays showed a sequestrum, along with new bone formation. **Chronic osteomyelitis of the diaphysis of the humerus** was diagnosed. An incision was made down to the bone in the lower third of the outer side of the arm, avoiding the radial nerve. The periosteum was destroyed at one place and a hole was found leading to a sequestrum, which was removed by carefully chiselling the bone; the cavity was scraped and plugged, and the fistulous track with its hardened walls excised. The subcutaneous abscesses were opened and scraped. The arm was immobilized for a long time. Healing took place after some months, and the patient was told to choose a lighter occupation in order to avoid recurrence of the disease.

Fig. 107 shows **acute osteomyelitis of the tibia** in a child, aged 9 years, which began with severe pain in the leg and knee joint, accompanied by high fever and chills. There was no history of a previous attack. A few days before, the child had received a blow on the tibia. In spite of the severe clinical symptoms and the marked swelling of the knee-joint, operative treatment had been neglected, and an incision was made only when a subcutaneous abscess developed. Although the acute symptoms subsided after this, the swelling in the leg persisted and the wound discharged fetid pus. In a few months almost the whole shaft of the tibia became necrosed. In **Fig. 107** the yellow, dead bone and the open medullary cavity with slimy granulations are clearly seen. Between the necrosed and the healthy bone are granulation tissue and pus. As the leg had not been properly fixed, a fracture occurred at the lower part of the tibia. The general condition was poor. The X-rays showed that the sequestrum extended further down and that a thick, bony capsule had already formed behind and at the side.

The wound was enlarged downward, the necrosed bone removed, the cavity scraped and packed, and the leg immobilized in correct position on a splint.

Such extensive necrosis could have been avoided by early chiselling of the bone and proper after-treatment.

Fig. 107. Osteomyelitis tibiae — Necrosis totalis.
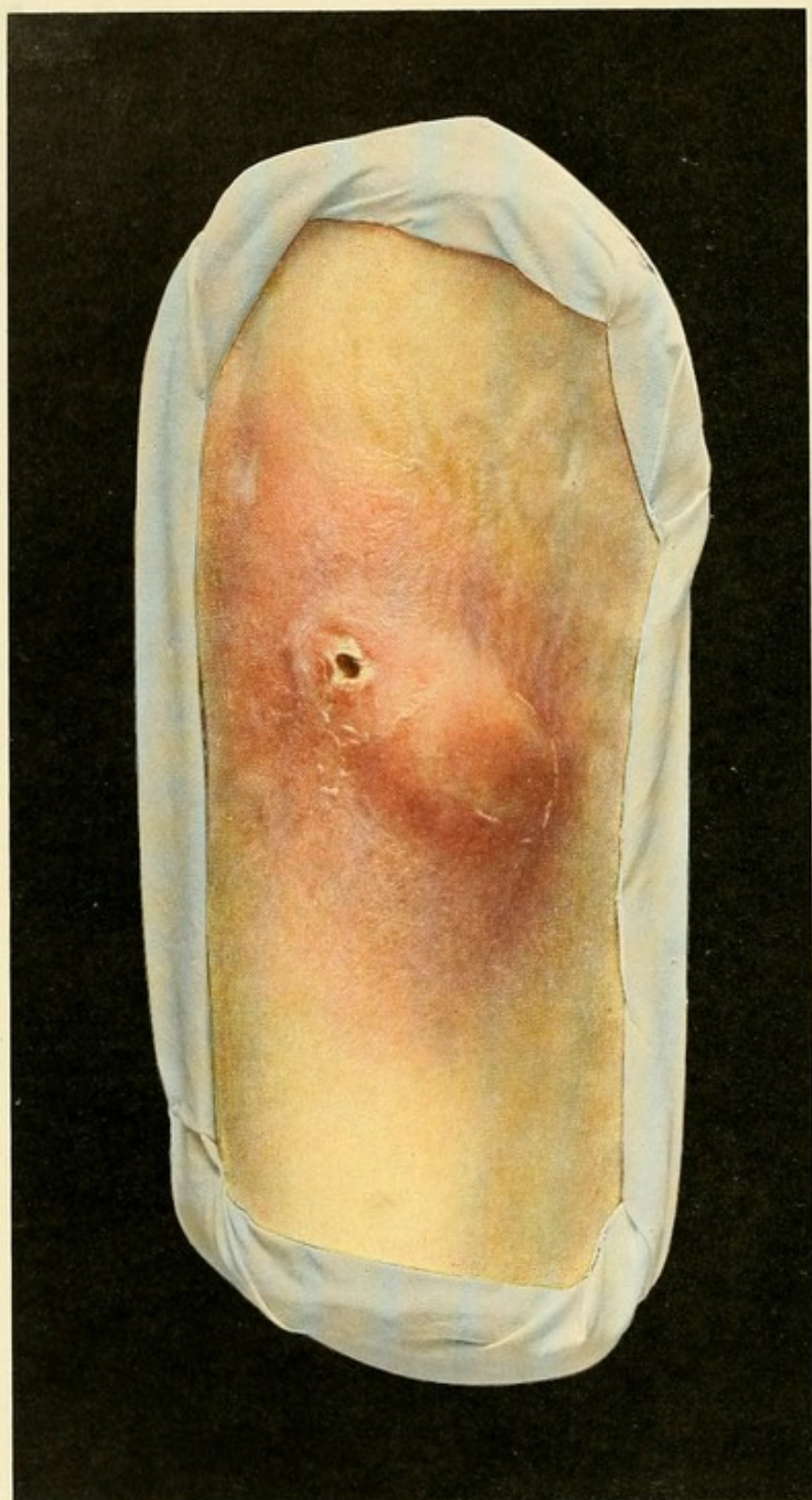
Fig. 108.   Infectio generalisata.

Fig. 108 shows a *metastatic abscess of the thigh,* one of several developed in the course of an *acute generalized infection* arising from a subcutaneous whitlow, which was insufficiently incised and drained, and which spread to the adjoining tendon-sheath and joint. The temperature rose to 106° F., with chills; it remained high for a few days and then became remittent during the formation of several metastatic abscesses, which required incision and contained thin pus with but few staphylococci.

There also were other general symptoms: dry tongue, jaundice, subdelirium and diarrhea. The wound in the finger was dry and unhealthy: the finger was exarticulated, and it is noteworthy that bacteria which had been found in the blood prior to this operation were after it no longer detected therein: a direct proof of the origin of the virulent microörganisms, confirmed, besides, by the improvement in the general condition and the checking of further local inflammation. Under the influence of stimulating treatment and repeated saline injections recovery took place in a few months: but for a long time afterward the pulse remained rapid.

In every pyogenic condition there is a slight degree of general infection: even in apparently benign and localized lesions such as furuncle, staphylococci may be found in the blood. This explains the occasional occurrence of osteomyelitis after such affections, and the often existing disproportion between the local inflammation and the general impairment of health. So that, *strictly speaking,* there is no really localized infection: but the general involvement is usually not sufficient to be recognized clinically.

All pyogenic microörganisms may be the source of a general infection: pneumococcus, *Eberth* bacillus, colon bacillus, *staphylococcus* and *streptococcus.* From the surgical standpoint the last two are by far the most important. Among putrefactive bacteria, the proteus vulgaris is the one most frequently at fault: it causes a special type of sepsis, but it is seldom alone: it generally is associated with the staphylococcus or streptococcus.

It would be very interesting to be able to distinguish in general infection what is due to the bacteria themselves and what is due to their toxins. There is always simultaneous bacteriemia and toxinemia; but toxins are not easy to detect in the blood owing to the tendency they have to become combined with organic protective substances.

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The part played by the blood in general infection is preponderant; dissemination takes place by small bacterial emboli (this must be distinguished from propagation by purulent thrombi in suppurative phlebitis); but we now begin to appreciate that the lymph stream also often has an important rôle, and that many metastatic deposits, formerly unhesitatingly labelled *hematogenous* are really *lymphogenous* in origin.

The more rapidly virulent bacteria invade the blood, the more severe the symptoms. In very grave cases, the temperature rises to 104° or 107°; such cases generally cause death in a few days without *clinically appreciable* metastatic abscesses. But this does not necessarily mean that there are no *anatomical* metastases; indeed, small foci are frequently found post mortem, especially in the kidney. There is even a form of general infection, well studied by *Brewer, Cotton, Cunningham,* in which small miliary abscesses in a kidney are the only anatomical lesions of a general sepsis that usually proves fatal unless nephrectomy is hastily performed. In less severe forms, the thermic ascension becomes intermittent after the initial high ascension; maybe because microbes enter the blood only intermittently, or because there are fewer of them. When the body defenders conquer the bacteria and their toxins, and sufficient antibodies have been formed, the temperature falls. When the invaders gain the upper hand, the temperature rises. If the outcome is going to be fatal, the longer the process goes on the more frequent are the chills and the shorter are the intermissions, so that finally a stage of continuous high fever is reached, as in those cases, already referred to, where no remissions occur from the outset.

But, if the organism is victorious, the infection expands its energy in the formation of metastatic abscesses in those parts of the body which are specially adapted to absorb bacteria, render them harmless, and finally destroy them (subcutaneous tissue, serous cavities, peritoneum, pleura, joints).

Staphylococcic general infection has a marked tendency to cause metastatic abscesses (95% of cases), while the initial focus is circumscribed. Streptococcic infections, on the contrary, as a rule have no localized initial focus, and in their spread keep the same character of diffuse processes, as they hardly ever cause metastatic abscesses. Streptococcic infections are more regularly fatal than staphylococcic, precisely on account of that same character. The rare cases of streptococcic general infection that end in recovery are those which lead to the formation of pus collections.

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Clinically, general infection is ordinarily *acute,* rarely chronic. Acute general infection may be primary or secondary, mild or severe. The severity depends on the number and virulence of the invading microbes, and on the defensive power of the body. The severest forms develop so quickly after the local infection that the latter remains in the background; the portal of entry may even be unrecognized and the general infection seem spontaneous; this must occur most frequently after lesions of the internal mucous membranes. Severe forms are frequently seen in physicians who prick their fingers during an operation or an autopsy (streptococcus): they also result from infection by putrefactive bacteria (proteus) or a symbiosis; both these kinds of microörganisms.

In the great majority of cases, however, general sepsis is of gradual onset, and arises from a local primary focus; but it has often reached an advanced stage before it is recognized. It may occur after a progressive extension of the suppuration in the primary focus, but *also may arise without further extension of the latter,* a fact of prime importance, always to be borne in mind.

In the hyperacute forms, the **symptoms** appear suddenly, while, in milder cases, there is a premonitory stage with general disturbances (insomnia, loss of appetite, headache, pain at the seat of infection). A frequent small pulse points to the onset of general infection, even before the *great thermic ascension.* This occurs suddenly (102° to 106°) with chills. As already stated (see page 158), the fever may remain *continuous* (hyperacute cases) or become *intermittent* or *remittent,* to become again *continuous* in cases tending toward a fatal outcome, or to subside in those terminating in recovery. All varieties in the temperature chart may be seen. *Every fresh infection of the blood is heralded in by a rise of temperature.* For example, after an extremity has been amputated for progressive suppuration, the temperature falls; but it may rise again after a time, showing that the organism was already saturated with bacteria and their toxins and that the operation came too late to save life. However, under those restrictions, a fall in temperature after extensive surgical interference is always a hopeful sign. Hypothermia, sometimes observed in very severe cases, is a very grave omen, as it indicates the utter collapse of the organism and complete toxemia. It is noteworthy that the pulse in remittent fever remains small and rapid during and after the fall of temperature, and during convalescence. This shows how much the heart is affected in general sepsis, even in curable forms.

The *respiration* is rapid and shallow: it may become stertorous when coma sets in, near the end, in severe cases.

The *tongue* in general infection shows characteristic changes; at first smooth, dry, salmon colored, it later becomes rough, fissured and brownish black. In severe cases the teeth also are dry and coated with sordes. The *conjunctivæ* are yellow, and in severe cases the *jaundice* (hematogenous icterus) may be general. There is profuse sweating and unquenchable thirst.

These symptoms are the only ones in case of general sepsis of internal origin (e.g. after pylephlebitis), but when the portal of entry is a preëxisting suppurating wound, the latter exhibits *local changes,* the appearance of which is fairly characteristic: the wound becomes *painful and edematous;* the granulations look *unhealthy and flabby;* the discharge of pus is much lessened and replaced by a scanty, dirty, often fetid secretion; the surface of the wound becomes dry and often covered by a diphtheroid membrane (**Fig. 101**). Pus retention, necrosis, extension of suppuration, lymphangitis and adenitis are often concomitant signs. In infection by putrefactive bacteria (**Fig. 109**) there are bullæ in the infiltrated skin and crepitation due to the formation of gas, and bubbles of gas in the secretion. In order not to overlook these signs, suspicious wounds must be frequently dressed. Another frequent sign, more common in advanced stages, is *septic secondary hemorrhage* in the wound, one of the most dreaded scourges of preantiseptic surgery, and which is due to vascular degeneration.

The gastro-intestinal canal is severely affected: there may be hematemesis, bilious vomiting and uncontrollable diarrhea. The skin is pale and cold and may present various types of erythema, scarlatiniform, morbilliform, erysipelatous, or vesicular eruptions and purpura. Almost all the internal organs are saturated with bacteria and their toxins. Hence the *nephritis* (evidenced by albuminuria and casts), the meningitis, pleuritis, pericarditis and endocarditis that are seen so frequently. Nephritis is even constant. The spleen is enlarged, and so may be the thyroid gland.

In the advanced stages, the patients become subdelirious, then delirious, finally, and generally, unconscious. Just before the end, if not comatose, the patient may have maniacal excitation followed by collapse.

In streptococcic general infection, there is nearly always suppuration in the joints: in staphylococcic, in the bones. Bacterial emboli carried to the capillaries can and do disseminate the infection to all

organs of the body (particularly the lungs, liver, heart and kidneys). Embolus of the central artery of the retina causes panophthalmia. Even if the involvement of the central organs escapes clinical detection at first, overshadowed as it is by the more noisy and threatening signs of generalized infection, it may become prominent afterward, after an apparent cure, and residual foci of old septicemiæ, long quiescent, are frequently the cause of grave trouble subsequently (suppurative nephritis, endocarditis, pleurisy, or pneumonia).

Multiple metastatic abscesses, as already said, are much more frequent in staphylococcic infections: they may remain cold and painless and contain but few bacteria, as was the case in the abscess shown in **Fig. 108.**

The chronic forms of sepsis, which occur after long-standing fistulæ, suppuration and necrosis (particularly osteomyelitis) are characterized by their gradual development and mild symptoms. Many cases, however, are fatal from heart failure or albuminuria (*amyloid degeneration*), or a chronic form may become acute. In chronic cases, the remission periods are long, and metastatic abscesses are the rule. Recovery may take place after removal of the primary cause, but convalescence is very slow.

Prognosis

It depends on the *general condition of the subject*. Young and robust individuals may survive acute forms; older, weakened, diabetic subjects do not, barring exceptions.

It depends also on the *nature of the infecting host*. Streptococcic infections are the most redoubtable of all: staphylococcic infections are more likely to end in recovery. Pneumococcic infections are the mildest of all.

It clinically depends on the type of the disease: hyperacute cases, with continued fever, are always fatal; subacute remittent cases, with abscess formation, are less serious; chronic cases recover, unless so much time is wasted before the necessary surgical interference that irretrievable damage is done to the internal viscera.

Abscess formation is always a hopeful sign; so is generally a decrease of the number of bacteria circulating in the blood, with this qualification, that in some cases such a decrease is followed by an increase in the clinical severity of the toxic symptoms.

Always remember, after recovery, the possible existence of encapsulated metastatic foci or incompletely healed septic infarcts.

The clinical picture of general infection, though many-sided, does not generally leave room for doubt, especially if there is a known primary focus.

Direct demonstration of the bacteria in the blood, by blood culture, clinches the diagnosis. Streptococci are more easily demonstrated in the blood than staphylococci.

Blood cultures, and the different **agglutination reactions,** establish the nature of the infection, and differentiate *typhoid fever, pneumonia, miliary tuberculosis,* all diseases which closely resemble general infection, because, in fact, they *are* general infections, but caused by other microbes than those at fault in the two great types of surgical sepsis.

Acute rheumatism is soon recognized by its exclusively articular involvement, and its changeable localizations.

Severe erysipelas may somewhat resemble general infection: sometimes the diagnosis is only a distinction without much difference, because there is often general infection in those severe cases.

Treatment

Everything that promotes the strength of the body is indicated (nourishing diet, tonics), as is the stimulation of faltering organs (caffeine, strychnia). Saline infusions restore tone to the circulatory system and effectively help in the elimination of toxins. No antipyretics should be given, as they are useless, and simply depress the heart. The high temperature is best reduced by tepid or cool sponging.

Locally, if there be a wound, *frequent dressings* are necessary: *no antiseptics* should be used. Hot wet dressings induce active hyperemia of the part.

Alcohol or ether dressings have been sometimes very efficient in grave infections of the limbs. *All retention of pus should be suppressed.* In progressive infection of the limbs amputation should not be too long delayed.

In impending general infection from thrombo-phlebitis, *ligation,* or even *excision,* of the veins should be resorted to (see page 123). Metastatic abscesses must be opened early, according to the rules governing incisions of abscesses in the region where they are situated. Joint resection is often necessary.

The serum treatment of general sepsis has been considerably improved during recent years. However, the results are not always

Tab. LXXXVII.
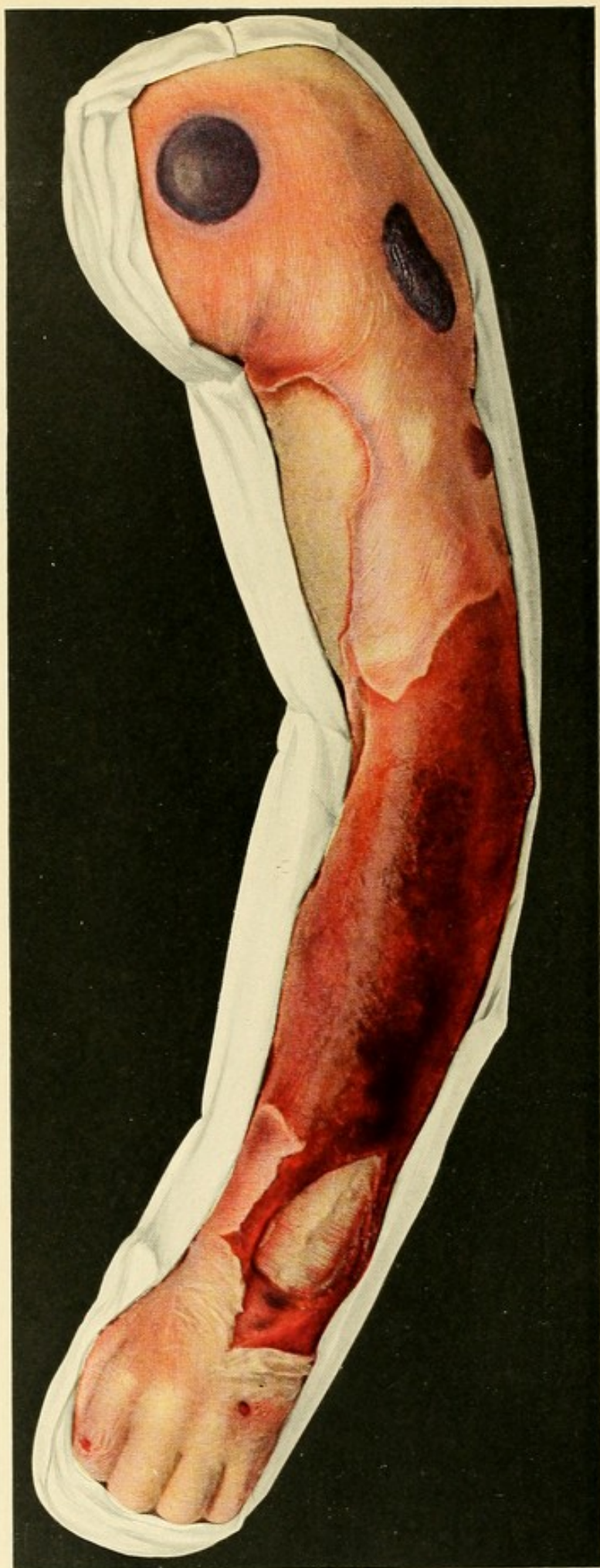
Fig. 109. Oedema malignum. Phlegmone emphysematosa-gangraenosa.

as satisfactory as could be desired. Nevertheless, as an injection of antistreptococcic polyvalent serum can do much good, and cannot harm, it is always indicated to use it early in streptococcic septicemia. Antistaphylococcic vaccines, very useful in chronic staphylococcic infections, are uncertain in their results in general infection. The injection of collargol is not much used now. Other metallic colloidal (gold, selenium) salts are reported by some authors as having a favorable action.

As the development of an abscess generally is a hopeful occurrence followed by marked improvement, it has been attempted, particularly in puerperal infection, to determine the formation of an abscess by a subcutaneous injection of turpentine (*Fochier*). This has sometimes proved successful in apparently absolutely hopeless cases.

Strengthening the body resistance during convalescence of recovered cases is always essential. The tonic treatment must be kept up for months.

Fig. 109 shows a very striking example of a hyperacute septic condition, which, in preantiseptic days, was a not infrequent complication of crushed wounds and amputations, and which is possessed of more names than any other surgical affection; a few being **gaseous phlegmon** or **gangrene,** *fulminating gangrene, bronzed erysipelas, malignant edema, emphysematous gangrene, acute purulent edema,* etc. It was one of the first conditions to disappear when antisepsis began; to-day it is a rarity, almost a ''laboratory'' disease.

The characteristic feature is rapid putrefaction under the influence of gas-forming bacteria. Which are the latter is not known with certainty, but they belong to the anaërobic group. *Pasteur's septic vibrio,* the *bacillus emphysematosus* and the *proteus vulgaris* are the bacteria found, generally associated with pyogenic microbes, staphylococci and chiefly streptococci. It is even thought that such a symbiosis is necessary for the development of gaseous gangrene, one kind being responsible for the gangrene, the other for the septicemia.

Typical gaseous gangrene is sometimes seen after trifling lesions of the skin (in **Fig. 109,** two small abrasions of the fingers with a meat knife); but it usually follows (or, rather, followed) crushing injuries and compound fractures, especially when soiled with earth and ground dirt. It occurs chiefly on the extremities or on the back in connection with bedsores.

A similar, but less acute and fatal, condition is seen in operative wounds on the rectum, when infected by feces; in the penis, scrotum and perineum, from lesions of the urethra with so-called "extravasation of urine" (which is not at all an infiltration of urine, as was thought formerly, but a diffuse gangrenous phlegmon of urinary origin; the serosity is that of inflammatory edema, and does *never* contain urea): in the neck, after operations on the esophagus and pharynx. All these wounds are deep and anfractuous, thus giving anaërobic bacteria a favorable ground to grow on.

The wound becomes dry, coated and fetid, and extensive swelling rapidly spreads from it on all sides. The wound discharge is brownish or greenish, fetid, and mixed with necrotic shreds of tissue. Severe pain, extreme anxiety and agitation, later on delirium and frequent pulse indicate the onset of general infection, which, from the beginning, is of the severest type and progresses very rapidly without any fever, or at least without the high fever we are accustomed to find in general sepsis. The advance of the gaseous infiltration has sometimes been visible to the naked eye: in a few hours large portions of the body are affected by the rapid formation of gas.

The circulation is obstructed by the great pressure of gas in the tissues. The skin of the extremities becomes pale and cold and presents brown and green spots, bluish livid streaks of lymphangitis and punctiform hemorrhages. Small vesicles filled with dark fluid then appear, which later on become larger: finally the whole epidermis of the affected part is raised and underneath it is offensive, dirty fluid. In other places, the skin is reddish-brown (bronze color), hard and infiltrated. There is no formation of circumscribed fluctuating collections of fluid, but the tissues are saturated with fetid, sanious, gas-filled serosity.

On pressure tissues give the *characteristic crepitation of cutaneous emphysema*. The infiltration is best seen after incision: the tissues cannot be distinguished from each other; muscles, fascia and periosteum are transformed into sodden, homogeneous, greenish shreds. If the medullary cavity of a bone is opened, it is filled with sanious fluid. Sometimes circumscribed cavities containing fluid and gas are found under the skin, and such accumulations may give rise to enormous bulgings near the root of the limbs and where the subcutaneous tissue is loose. Pressure of gas may cause gangrene of the peripheral parts of the extremities, and the appearance is similar to that given by the putrefaction of a corpse (**Fig. 109**). The lymphatic glands are swollen and painful; the veins are thrombosed;

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finally, the arterial walls are destroyed: hence severe hemorrhage. A sanious fluid fills the neighboring joints.

The emphysema may cover very large areas: for instance, the whole back or the abdomen, or a whole limb. Death occurs from general infection or edema of the glottis or mediastinitis. Bacteria are not usually found in the blood.

Despite the most heroic measures, the prognosis is fatal in true gaseous gangrene in more than 95% of cases: within thirty-six hours, three or four days at the utmost. A few cases, in young, strong individuals, have a slightly slower evolution: to those belong the *very small* minority of cases ending in recovery. In post-operative diffuse gaseous phlegmon the prognosis is not so bad; it is decidedly better in extravasation of urine, provided it be properly treated.

Diagnosis

The ultrarapid evolution of the disease, the necrosis of all tissues, the **crepitation** distinguish gaseous gangrene from *hemorrhagic bullous erysipelas* (**Fig. 91**) and *anthrax* (**Figs. 112** and **113**). A bacteriological examination is always useful.

Treatment

A particularly careful disinfection of all crushing injuries and compound fractures by tincture of iodine is the best prophylaxis.

When gaseous gangrene is detected, it is always too late to save the part. In a limb, amputation must be performed forthwith, and high above the limit of crepitation. Numerous free incisions with the thermocautery in infiltrated tissues, when amputation is not possible, and moist dressings with hydrogen peroxide deprive the anaërobic bacteria of the medium necessary for their existence.

This treatment, applied to urinary infiltration, often leads to recovery, and it is often surprising how enormous defects of sloughed skin on the penis and the scrotum heal up quickly and nicely.

In gaseous phlegmon of the neck, a preliminary tracheotomy is necessary before operation.

In the case represented in **Fig. 109,** despite free incisions and exarticulation of the shoulder, on the third day the patient died.

Fig. 110 shows an *acute lymphadenitis of the left inguinal region,* consecutive to an excoriation of the skin of the thigh, which has already scabbed over. The lymph glands act as barriers between initial lesions (pyogenic or cancerous) and the rest of the body. In case of pyogenic bacteria coming from a primary focus, the glands stop them and destroy them, unless they be too virulent or numerous, in which case the glands become themselves affected. Lymph gland inflammation is non-suppurative or suppurative. Besides the cervical glands (see **Figs. 102** and **114**), the axillary and the inguinal groups are the most frequently involved. Suppurative adenitis of the groin is called *bubo.*

To the superficial inguinal lymph glands go the cutaneous lymph-vessels of a very wide area, lower limb, perineum, scrotum, penis, anus and abdominal wall below the navel. To the deeper group go the deep lymphatics of the thigh and the lymphatics of the glans penis in the male, and of the clitoris in the female.

It was formerly thought that each subdivision of this territory of the superficial inguinal glands corresponded to a special group of glands, always the same. Recent studies have disproved this idea; there is no rigid systematization. However, but only as giving a relative indication, and no absolute certainty, it may be stated that the lymphatics of the lower limbs go to the *lower group* of inguinal glands; those of the scrotum and genitals to the *internal* glands of both the lower and upper groups: those of the perineum in the *upper* glands of both same groups; those of the anus in the *upper and external* group; those of the umbilicus and abdominal wall to the *upper* group. This may give a *general* indication as to where to look for the origin of any given suppuration.

Any portal of entry for bacteria within this territory may cause bubo; hence the frequency of the latter. Excoriations of all kinds, ulcers, ingrowing toenails, lesions of the genitals (particularly *soft chancre* and gonorrhea) are the most frequent causes. Chancroidal bubo may contain the *Ducrey* bacillus, but more commonly is the result of a secondary infection; so is the bubo of gonorrhea generally due to balanitis and balano-posthitis.

The acute forms are very painful and prevent motion of the limb. The skin becomes red and is at first movable over the inflamed and tender glands (pure adenitis); but it gradually becomes infiltrated and bluish in color while the glands become matted together (peri-adenitis); finally diffuse suppuration may set in (**Fig. 110** and also **Fig. 102**). Then there are chills, fever, and constitutional disturbance. In other cases, the inflammation is better localized (see **Fig.**

Fig. 110. Lymphadenitis inguinalis diffusa (Bubo).
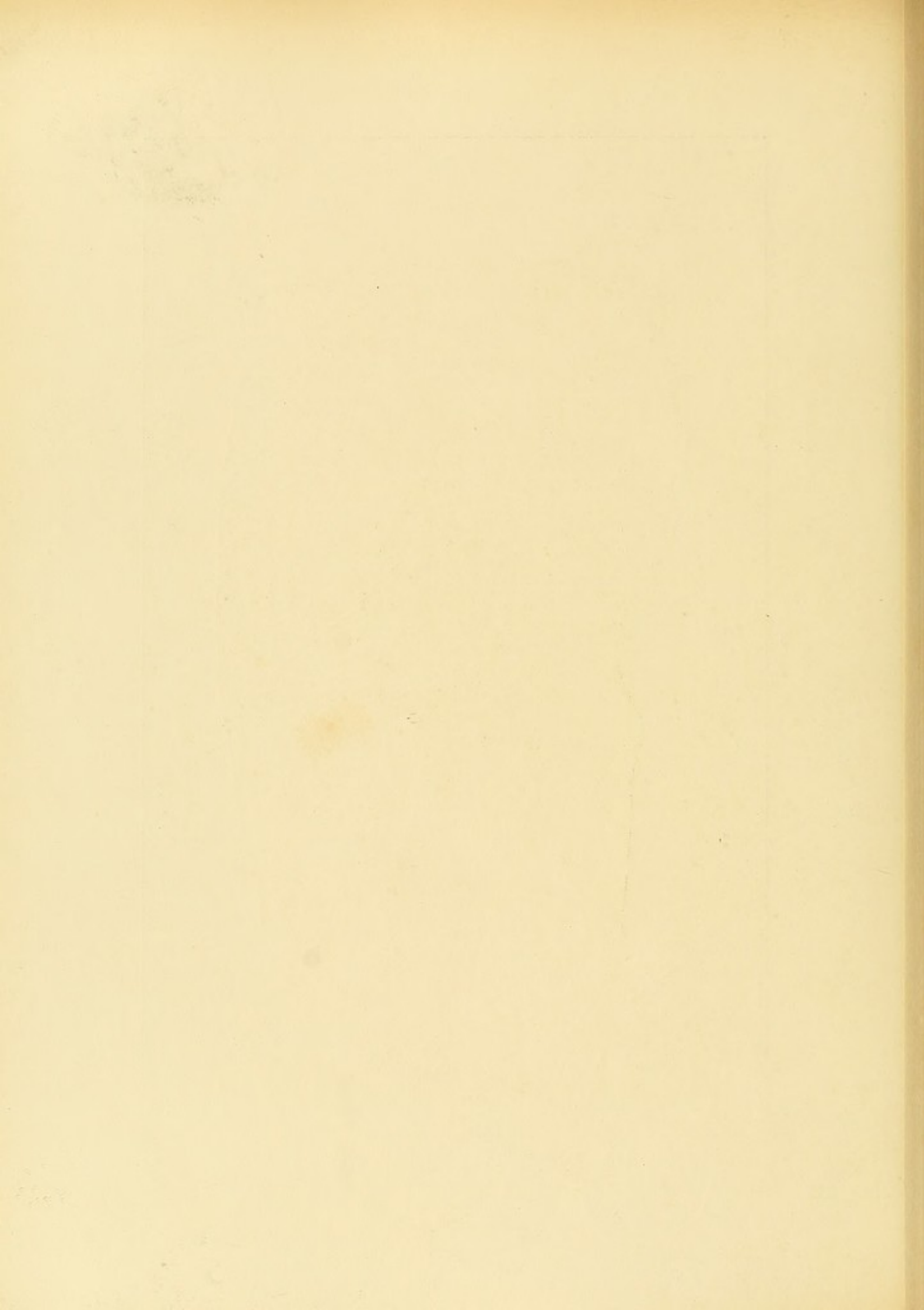
114). The skin becomes thin, and, if not incised previously, ulcerates and the pus is discharged, after which the pain subsides, but healing is often very slow, especially in chancroidal bubo. This is probably due to autoinoculation of the wound by the *Ducrey* streptobacillus. When such an infection does not take place, inguinal adenitis heals just about as quickly as any other adenitis.

Diffuse suppurative adenitis causes necrosis not only of the glands themselves, but also of the periglandular tissue and even of the subcutaneous tissue. Moreover, burrowing abscesses may develop in remote places (in the pelvis after bubo, in the retropharyngeal glands after cervical abscesses). Again general infection is possible, though rare.

In chronic forms, inflammatory symptoms are absent. There only is a slightly painful enlargement of the glands which subsides after some time, leaving, however, a fibrous hyperplasia of the nodes. Chains of small, chronically inflamed lymph glands in the groin are exceedingly common.

Diagnosis

Acute lymphadenitis is easily diagnosed from its situation. Other conditions have already been discussed (see **Fig. 102** and page 144).

A femoral hernia may be mistaken for a bubo, especially if inflamed. This must always be remembered, as incision in hernia might be unpleasant to patient and physician. However, the inflammatory symptoms of the skin are absent in hernia, and in bubo the nodes are generally multiple.

Tuberculosis of the inguinal glands and cold abscess is distinguished by the torpid evolution, without pain or fever, the thin, greenish pus, and the undermined edges of the fistula, if present.

Adenitis of the axilla may be mistaken for hidrosadenitis (see page 128), but the latter are multiple small tumors inlaid *in* the skin, and not subcutaneous.

Chronic adenitis must be differentiated from syphilitic adenitis (see page 179), tuberculosis and cancerous involvement. The inguinal glands are involved in cancer of the anus.

Treatment

Cleanliness, the early treatment of the causal disease, and the *avoidance of cauterization in too old chancroids* (which only dams back the virulent secretion and decidedly favors the development of bubo, besides doing absolutely no good to the chancroid itself) constitute the prophylaxis of venereal bubo.

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Suppurative adenitis must be **incised** as early as the presence of pus is ascertained; in the case of chancroids, great care must be taken to prevent, if possible, the inoculation of the *Ducrey* bacillus, but often the latter is in the pus itself: injections of a 10% emulsion of iodoform in glycerin are useful. Frequent dressings are needed. Patients are best kept in bed. Silver nitrate, Peru balsam are employed to stimulate the always slow granulation process.

In case of large glands causing pain, or of multiple fistulæ, the best is to excise all diseased glands, taking care, however, to insure a good hemostasis and not to be too radical in the removal of connective tissue; as bilateral extirpation of the inguinal glands is sometimes (**Fig. 71**) followed by lymphatic stasis and elephantiasis.

Inunctions with iodine preparations, ichthyol or mercurial ointment may prove useful to bring about the resolution of chronic non-suppurative inguinal adenitis.

Fig. 111 shows a case of **gonorrheal arthritis of the wrist** in a woman.

Gonorrheal arthritis is the commonest systemic manifestation of gonorrhea, its frequency being estimated to about 1% of the treated cases of urethral gonorrhea. It is much more frequent in men than in women, and particularly frequent in children, if the smaller number of cases observed in the latter be taken into consideration.

The focus of absorption in the male is the *posterior urethra*. Gonorrheal rheumatism does not occur in simple anterior urethritis. It is particularly frequent in posterior urethritis complicated by prostatitis and vesiculitis. Indeed, seminal vesiculitis has been blamed for the most obstinate cases (*Fuller*). This influence of the prostate and seminal vesicles as a safe harbor for gonococci in gonorrheal rheumatism is confirmed by the relative infrequency of the affection in women.

There are no parallel variations between the intensity of the local urethral process and the systemic manifestations of gonorrhea. The worst case of gonorrheal tenosynovitis (akin to arthritis) I ever saw was in a young man with a very mild anterior urethritis (first attack) treated from the outset, and apparently almost cured in three weeks. Frequency denoting the posterior involvement, and fever and the tendon-sheath metastasis appeared suddenly and *simultaneously*. But, even if such unpleasant surprises are possible, it goes without saying that every fresh infection, every relapse of an incompletely cured gleet, increases the chances of gonorrheal rheumatism, espe-

Fig. 111.   Arthritis gonorrhoica phlegmonosa.

cially in those who have already suffered from the latter. Recurrent attacks are not infrequent, and are *always possible as long as the individual harbors gonococci.*

Gonorrheal rheumatism, as a rule, is not as polyarticular as ordinary rheumatism; it ordinarily is *monoarticular* and strikes *large joints.* The *knee* is by far the most frequently affected, next come the ankle, foot and wrist. But no joint is immune, and several may be involved.

Anatomically, the lesions are *limited to the joint,* or affect *both the joint and the bone.* Of course, the latter cases have a much more unfavorable prognosis in so far as ultimate damage to the structures and function is concerned.

The fluid in the joint, in case of arthritis, may be serous, *fibrinous* or purulent. The fibrinous type is the one met with oftenest, suppurative arthritis being rare and generally caused by a mixed infection. The tendency to periarticular infiltration is more marked than in other types of arthritis. The inflammation is destructive in the severe cases, commonly it is *hyperplastic,* hence the tendency to *stiffening, limitation of function and anchylosis,* which is the most serious danger of gonorrheal arthritis, even in its mild forms.

This danger is intensified when primary bone lesions exist, as then there is destruction of bone, and irregular proliferations of the irritated and thickened periosteum. Prognosis as to function is then very doubtful.

Acute gonorrheal arthritis is very sudden in its onset, and attended by severe pain, preventing any movement of the affected joint. In a few hours the soft parts become infiltrated and edematous, the infiltration is more or less limited to the region of the joint, or, frequently, spreads to the neighboring muscles and tendons. The skin is red and tense (**Fig. 111**). In severe cases there is high fever and complete loss of function. In chronic cases there usually are aching pains in the joint before the arthritis becomes evident.

When the effusion is merely serous or sero-fibrinous and limited to the joint, resorption generally takes place in one or two weeks and *restitutio ad integrum* is complete. But in the more common form of *fibrinous arthritis,* with marked periarticular infiltration, the outcome is not so favorable. In the joint are produced fibrous bands which limit motion. When there is a bone involvement (which is *primary,* and *not* due to the extension of the articular inflammation, as heretofore believed; this is clearly established by early X-ray examination), cartilaginous and even bony anchylosis is the most

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frequent termination. Destruction of the capsule may cause sub-luxation or dislocation, and prolonged immobility leads to muscular atrophy.

In the rarer forms of purulent arthritis, fever is very high, general symptoms are marked, the skin is red and the swelling tense.

Chronic forms are less noisy in their clinical expression, but multiple relapsing arthritis may reduce the patients to a deplorable condition, as they often cannot walk or use their arms.

Diagnosis

Gonorrheal rheumatism supervening during an acute attack of gonorrhea, or relapsing cases, are generally easily diagnosed, because the causal relation is evident.

But it may be exceedingly doubtful when occurring a long time after the acute stage, when the uncured chronic condition has been overlooked or forgotten. In every case of arthritis, *the possibility of gonorrheal infection must be borne in mind,* and investigations made to find whether or not the patient harbors gonococci. The **complement-fixation test** (*Schwartz* and *O'Neil*) will render signal services in this respect. A complete examination of the genital tract is indispensable (two or three glass tests, rectal digital examination of the prostate and seminal vesicles, smears and cultures). X-ray examination also must be performed. It is particularly interesting to find from the outset whether there is a zone of rarefaction in the bone, as this has a direct bearing on the prognosis (see above).

Acute rheumatism is more polyarticular, wanders more from joint to joint, involves the smaller joints, and yields promptly to salicylic treatment, while the latter hardly influences gonorrheal rheumatism.

Purulent gonorrheal arthritis must be diagnosed from other joint suppurations by the anamnesis and bacteriologic examination of the fluid withdrawn by tapping. However, this proves often sterile in all types of gonorrheal arthritis.

Syphilitic arthritis may be very hard to distinguish from chronic gonorrheal cases; but the *nocturnal character* of the pain, which is decreased by exercise, the *Wassermann* or luetin reactions, the previous history, and X-ray examination may solve the problem.

Tuberculous arthritis would be considerably easier to differentiate, at least in its ordinary forms (see **Figs. 125, 126, 127**).

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The best *treatment* of gonorrheal arthritis is *prophylaxis* by the correct treatment of *all* cases of gonorrhea, however trifling they may look, especially to patients.

The milder serous forms recover after two weeks of *rest* in bed and immobilization of the joint in a splint apparatus or a light plaster of Paris cast. *Bier's passive hyperemia* greatly aids to resorption. Active hot-air hyperemia is contraindicated in this stage as it increases pain. Rest must be complete and active treatment of the gonorrhea must be kept up as long as there are any inflammatory symptoms, but as soon as complete subsidence of the latter has been obtained, gentle massage and passive and active motion must be started. This treatment brought about complete restoration of function in four weeks, in the case depicted in **Fig. 111.**

Besides *rest* and *passive hyperemia, serum or vaccine treatment* is the only thing really efficacious against gonorrheal rheumatism. Serum seems to act better in the acute and subacute stages, while vaccines are far superior in the chronic forms (*Schmidt*).

Purely arthritic forms require a more prolonged immobilization than those of primary bone involvement, in which passive motion must be begun early in order to avoid complete loss of function and anchylosis.

Chronic forms are often very obstinate. *Vaccine* (not serum) treatment does not give as regular an improvement as in the acute, recent cases. It benefits (particularly as regards *pain*) 40 or 50% of the cases. *Active hyperemia* (hot-air baking, 280°-300°) has given me better results than passive congestion. Massage and passive motion are indicated when there is no acute exacerbation.

Arthrotomy and washing of the joint are resorted to only in the suppurative cases; that is, comparatively rarely, especially if the serum and rest treatment has been properly applied from the outset.

Starting from the idea that the seminal vesicles are the "thorn" that keeps chronic cases relapsing, *Fuller* in inveterate gonorrheal arthritis performs vesiculotomy and drainage. He claims excellent results.

Fig. 112 shows a case of *malignant pustule* of the face, which developed in a tanner after a slight abrasion of the skin. **Fig. 113** shows the same case a few weeks after infection.

Malignant pustule (external anthrax or malignant edema) is the inoculation lesion of a general infectious disease caused by the *anthrax bacillus,* that is, the germ of cattle fever. The bacilli and spores are found in the alimentary canal of animals (horses and cattle); also in damp soil on which these animals graze, and in the skin, fur and excrements of the infected animals. They are transmitted to man either directly from diseased cattle, pasture or soil (farmers), or by the skins and furs of infected animals (butchers, tanners, wool sorters, furriers, workers in horsehair, etc.). Anthrax in cities is *exclusively an occupational disease,* the reporting of which is compulsory in many States (New York, for instance).

The disease may also be transmitted by flies feeding on the corpses of dead animals, and by earthworms. Besides external anthrax, there is also an internal anthrax, resulting from the inoculation of the virus in some point of the alimentary canal, by ingestion, or from the inhalation of spore-containing dust (pulmonary anthrax; this form is less common).

The bacilli may remain localized at the seat of infection or may enter the blood-stream and give rise to metastatic foci in other places, while their toxins play only a subordinate part.

External anthrax is observed particularly on the face and neck, small abrasions of the skin being the portals of entry. The infection may be conveyed to the mouth by the finger and the spores may thus be inhaled or swallowed, and give rise to internal anthrax.

Anthrax of the skin has a very characteristic appearance. A small, red spot first appears, with fever, and often chills; this develops into a small blister with a yellowish or turbid liquid content where the specific bacilli are found. This is the *malignant pustule,* which ruptures and is replaced by a scab. At the same time, the surrounding skin becomes green—a sign of commencing necrosis. *Early necrosis* of the skin (**Fig. 112**) is one of the salient features of anthrax. Around the central eschar, and forming a *ring* around it, develop a number of vesicles absolutely similar to the original vesicle; outside of this ring the tissues become infiltrated as in carbuncle, and still more peripherally there is a zone of edema, which can assume huge dimensions in regions where the subcutaneous tissue is loose. The redness of the skin extends rapidly and irregularly, resembling erysipelas. The newer vesicles rupture; others, still more peripheral, appear, and also rupture; after each rupture the necrosis

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Tab. XC.

Fig. 112. Anthrax — Pustula maligna.
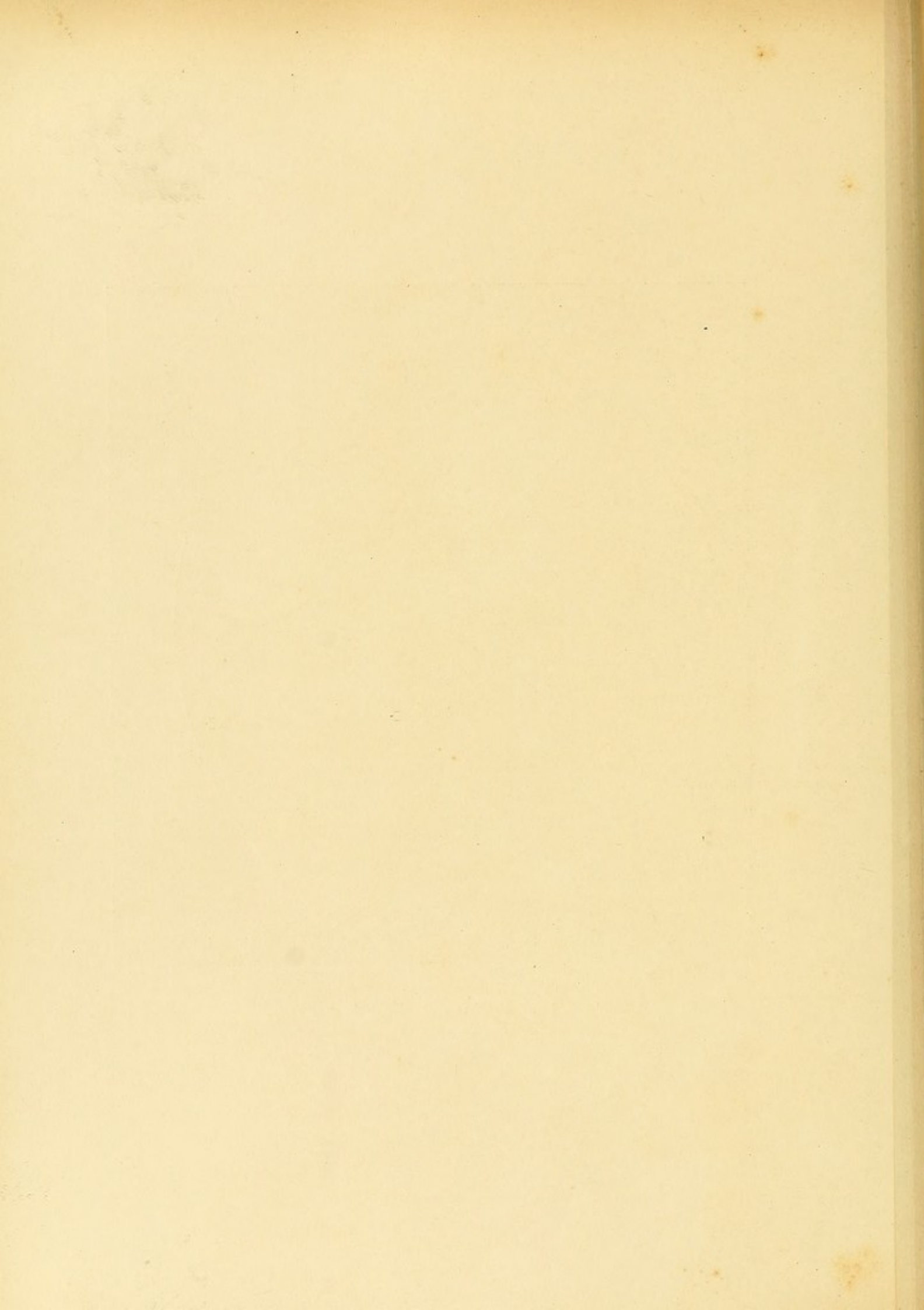
Fig. 113. Anthrax — Necrosis.

of the skin extends more and more. Thus, always presenting its three concentrical zones—central necrosis, middle vesicular ring, external infiltration—the malignant pustule spreads until it has reached quite a large size (**Fig 113**).

Besides the above-mentioned symptoms, there are, if the pustule is located on the limbs, lymphangitis, adenitis and mixed infection abscesses. General symptoms are always marked, fever, chills, headache, rapid pulse. General infection with dry tongue, jaundice, diarrhea, enlargement of the spleen occurs in about 25% of the cases. Death may result from collapse in a few days.

External anthrax has a less unfavorable prognosis than internal, except when located on the face, where it has all the dangers of facial septic conditions, and besides may spread to the mouth and the digestive tract. In the milder forms of anthrax septicemia, metastases are caused by emboli in the skin, lungs, liver, brain, etc.; hence pleurisy, pneumonia, ulcers, peritonitis, meningitis, which are generally fatal.

Internal anthrax usually affects the intestines (buccal infection); it causes hemorrhagic ulcerations of the small intestine with tendency to gangrene. The mortality is 80%.

Pulmonary anthrax clinically resembles pneumonia. The mortality is 90%, from pulmonary edema and pleurisy. Internal anthrax may cause secondary metastatic foci in the skin. Internal and external anthrax may coexist.

Diagnosis

A typical malignant pustule, with its central eschar, surrounded by a ring of vesicles and a zone of hard infiltration, is not difficult to diagnose, especially when the occupation of the patient is one of those exposing to anthrax infection.

However, **pyogenic or putrefactive infections** of a virulent type (**Fig. 109**) and **hemorrhagic bullous erysipelas** (**Fig. 91**) may cause the formation of vesicles on the skin and, therefore, be mistaken for anthrax; but the course is different, there is no necrosis of the skin and the three zones do not exist.

Glanders also cause blister formation and gangrenous ulceration, but the characteristic infiltration of anthrax is absent. In all cases where there is the least suspicion, a bacterial examination of the fluid of the bullæ must be made forthwith. The anthrax bacillus has pathognomonic appearance: it shows as immobile rods with sharp, angular corners, often arranged in a row of long chains. In the centre of the rods are clear spaces corresponding to spores, which are very resistant to desiccation and heat.

Finding the bacillus in the stools of suspected cases of intestinal anthrax is practically the only way of making the diagnosis, just as finding it in the blood-stained sputum of a pulmonary case is the only means of differentiating it from ordinary pneumonia or pleurisy.

Treatment

Prophylaxis consists in strict supervision of trades in which there is a danger of anthrax infection. Skins should be disinfected and workingmen taught hygiene. Preventive vaccination of cattle against anthrax was the first ever applied (*Pasteur*).

A rather large percentage of the not too severe cases of malignant pustule recover spontaneously, under expectant treatment consisting simply in isolation, complete rest, local applications of ointments to prevent auto-inoculation, and tonics to raise the defensive power of the body. Such a treatment was successfully applied in the case represented in **Figs. 112** and **113.** But systematic abstention cannot be made a rule.

When the malignant pustule is still small and situated on the limbs, it may be destroyed with the thermocautery. When it has reached a large size and spreads rapidly, its progress may be checked by subcutaneous injection in the infiltrated peripheral zone of iodine solutions so as to form a ring around the pustule. Such injections are repeated several times, as needed. Tincture of iodine is extremely powerful against the anthrax bacillus.

When the infection does not extend any more, but has left a large eschar (as in **Fig. 113,** where the leathery, blackened, necrosed skin, firmly adherent to the underlying tissues, is demarcated by a zone of pus and slimy granulations from the surrounding skin, which is still red and infiltrated), no attempt should be made to remove said eschar by sharp spoon or knife, lest we start a fresh outbreak of infection. It is therefore allowed to separate gradually, and the defect is repaired by a plastic operation, if need be.

Sclavo's serum may sometimes prove useful in anthrax.

Fig. 114 shows an ***abscess*** of the lymphatic glands behind the ear. The cause is *pediculosis of the scalp* (note the gluing of the hair and the punctiform deposits (nits) on them). Pediculosis, through scratching, is frequently the origin of eczema and adenitis, suppurative or non-suppurative. In the case shown, the submaxillary lymph nodes were enlarged and tender on pressure. The abscess was incised.

(See **Figs. 102** and **110**).

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Fig. 115. Aktinomykosis incipiens.

Fig. 114. Lymphadenitis circumscripta abscedens.
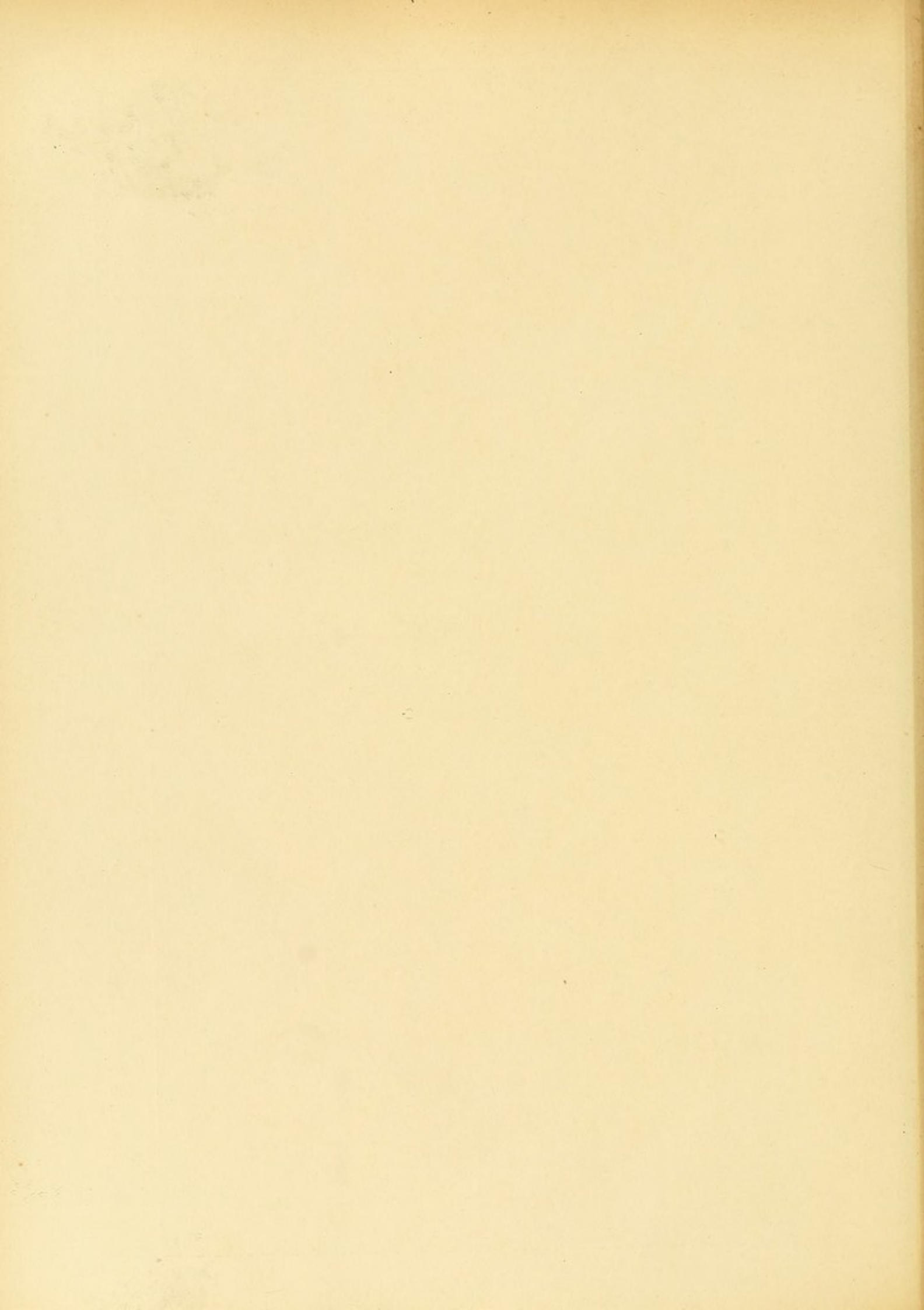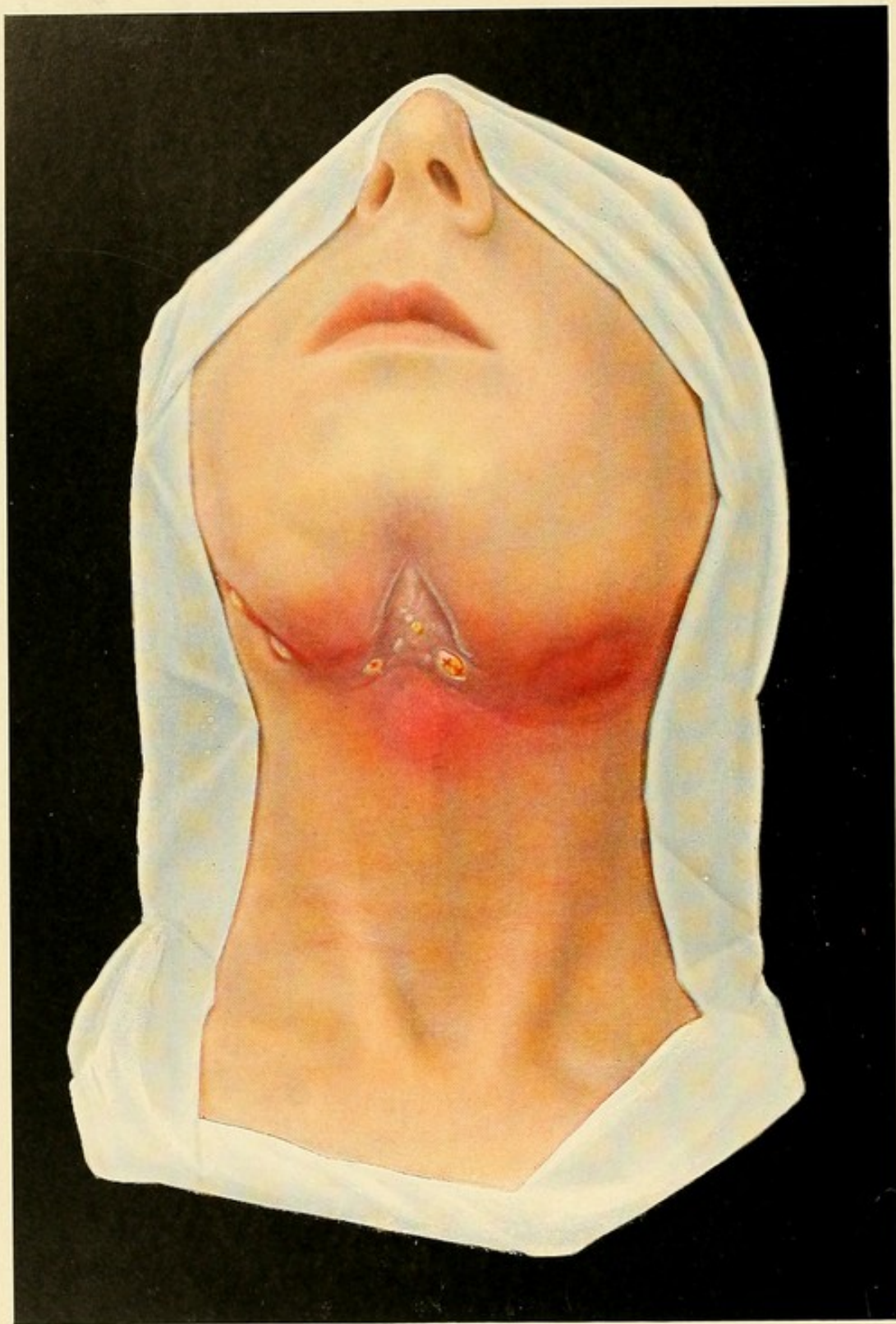
Fig. 116. Aktinomykosis progressiva.

CHRONIC INFECTIONS

Fig. 115 shows a case of *incipient actinomycosis* of the cheek in an old countrywoman. **Fig. 116** shows a case of *extensive actinomycosis* of the neck in a young countryman.

Actinomycosis is a comparatively rare disease, due to infection by the *ray-fungus,* which is found in corn, straw and flour. Hence the greater frequency of actinomycosis in country people, farmers and millers. In countrymen who have the habit of chewing corn or straw, infection takes place through a carious tooth (case of **Fig. 115**) or through the parotid duct. Actinomycosis of the buccal cavity, mucosa, tongue, lower jaw (*lumpy jaw*), pharynx and neck constitute a great majority (56%), while lung actinomycosis claims 13%, and abdominal actinomycosis 21% of all cases.

Actinomycosis of the buccal walls presents itself as a stringy, nodular infiltration, which, by becoming confluent, produces a swelling of woody hardness. Acute inflammatory symptoms are absent. The skin becomes bluish-red when the infiltration extends through the cheek or into the neck (**Figs. 115** and **116**). The infiltration spreads gradually into the neighboring tissues and its progress is unlimited. Simultaneously the centre of the mass softens, ulcerates and a complex fistula is formed, which discharges through several openings pus containing characteristic, pin-head size, *yellow bodies* (in which the microscope readily detects the fungus).

There is much induration around the fistulæ; this often causes retention of pus, and, in the case shown in **Fig. 115,** the pressure of the induration on the malar bone had been sufficient to cause gangrene of a patch of skin. Granulation tissue is scanty, yellowish-red in color, and rapidly disintegrated. Large abscesses may result from mixed infection: the yellow bodies are then often absent, the fungus being destroyed by the pus cocci.

In actinomycosis of the cheek, there always is an external fistula. If the infiltration involves the masticatory muscles, there is some trismus (case of **Fig. 115**). The fungus may invade the bones and give rise to enormous tumors. If the upper jaw is involved, the fungus may reach the base of the skull and lead to meningitis or cerebral abscess. If the tongue is infiltrated, its motion is hindered. If the process implicates the base of the tongue or the fauces, dys-

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phagia, and later dyspnea, develop. In these cases abscesses form, which generally discharge through cervical fistulæ and give rise to *secondary* actinomycosis of the skin, more common than *primary* skin actinomycosis.

Lung actinomycosis may result from direct inhalation or secondary buccal involvement. The symptoms are very much like those of tuberculosis. However, pleural and thoracic involvement lead to abscess formation and external fistula. The disease may spread to the pericardium, the vertebræ, the diaphragm and the abdominal cavity. The patient becomes exhausted from empyema and multiple burrowing abscesses. Adequate drainage is next to impossible to insure, so that relapses are very common.

Intestinal actinomycosis chiefly affects the ileo-cecal valve, where it causes a hard, tumor-like infiltration, which, if markedly developed, may produce intestinal obstruction. Thence the disease may spread in all directions, and bring about the formation of an external fistula.

Diagnosis and prognosis

When actinomycosis is visible externally, the diagnosis usually is not difficult: the wood-like infiltration, multiple fistulæ, yellow granulations and yellow bodies in the pus are pathognomonic. Of course, finding the ray fungus under the microscope, a very simple thing, absolutely clinches the diagnosis. *It must be done in all doubtful cases.*

As microscopical examination is so easy and so conclusive, it will suffice to mention the conditions which at first sight might resemble actinomycosis.

Lupus, tumors of the cheek and tongue, cold abscesses, gummata, woody phlegmon of the neck might be mistaken for cervico-facial actinomycosis, or *vice versa;* tuberculosis of the lung for lung actinomycosis.

Infiltration of woody hardness between the ribs is always suggestive of actinomycosis. The latter must always be borne in mind in presence of a hard, diffuse tumor of the ileo-cecal region, the other possibilities being tuberculosis, cancer, or dense old adhesions.

The prognosis is fairly good in cervico-facial lesions; two-thirds of the patients recover under appropriate treatment. It is very bad in lung actinomycosis: recoveries are exceptional. It is slightly better in abdominal actinomycosis.

Fig. 119. Gumma linguae – Lingua bifida.

Fig. 118. Sklerosis syphilitica linguae.

Fig. 117. Lingua geographica.

When surgical removal is feasible, it is the best treatment (resection of the gut in ileo-cecal actinomycosis). When not, incision and scraping of the abscesses and sinuses, and frequent dressings should be resorted to, with intermittent courses of treatment with *potassium iodide,* 40 to 60 grains a day. X-rays have proved useful in *Stelwagon's* hands. General treatment, tonics, arsenic, sustaining diet, are very important, as exhaustion is the chief danger in chronic cases.

Fig. 117 shows a case of **marginal glossitis,** also called **geographical tongue.**[1] This is a rare affection, chiefly seen in infants, but also, occasionally, in adults, and characterized by gyrate, gray, figures involving the borders and dorsum of the tongue. The chief lesion is a hyperkeratosis of the lingual papillæ. The patch begins as a scaly macule and spreads peripherally, while the centre again becomes normal.

The nature of marginate glossitis is unknown. *Kaposi* and *Parrot* considered it as syphilitic, and *Fournier* as parasyphilitic; but the actual consensus of opinion is against any such relationship. It may, perhaps, result from gastro-intestinal disturbances (*Stelwagon*). The course is chronic, and the lesions may continue indefinitely.

Diagnosis and treatment

Marginate glossitis is chiefly of interest on account of the possibility of its being mistaken for other lingual conditions. **Leucoplakia** (**Figs. 8** and **9**) gives an altogether different picture. So does **tertiary syphilitic glossitis** (**Fig. 119**).

The treatment consists simply in bland mouth washes, swabbing with tincture of myrrh and attention to the gastro-intestinal tract.

[1]Compare **Figs. 117, 118, 119,** with **Figs. 6, 7, 8, 9.**

SYPHILIS

Figs. 118 to **123,** inclusive, represent various **syphilitic lesions.**
Fig. 118 shows an ***initial sclerosis*** on the tongue; the others show
gummatous conditions, that is, they belong to a much later period
of the evolution of the disease.

Syphilis is a specific, chronic, infectious disease due to the in-
vasion of all the body tissues by the ***Spirocheta pallida,*** a spirillum
described by *Schaudinn* and *Hoffmann* in 1905, recently cultivated by
Noguchi, and which has now been found in all syphilitic products and
also in the blood in both acquired and hereditary syphilis.

Infection takes place through slight abrasions of the skin or
mucosæ. In most cases, the origin is *direct* and *genital,* but *extra-
genital direct contamination* is possible in any point of the body, and
is very far from uncommon. *Indirect* contagion through towels,
drinking glasses, cigars, pipes, shaving brushes, razors, is also fairly
frequent. Congenital syphilis is the result of infection of one or both
of the progenitors.

The *spirocheta pallida* has a **marked predilection for the
intima of blood vessels.** *Endarteritis and the perivascular arrange-
ment of round-cell infiltration are constant characteristics of all
syphilitic lesions, from the earliest to the latest.*

Acquired syphilis begins by an incubation period of three to five
weeks, followed by the development at the inoculated point of an
initial lesion (*hard chancre*), that is, a *circumscribed, hard, painless*
infiltration of the skin or mucosa. The chancre forms a hard nodule
movable over the underlying structures. It is a *flat erosion* with an
even, dark-red surface, regular smooth borders (**Fig. 118)** and a
characteristically indurated base. The chancre can be situated in
any point of the body: the genital zone (prepuce, glans penis, labia)
is naturally most commonly affected; but the tongue (**Fig. 118**),
lips, tonsils, fingers, and nipple are other frequent locations.

Every case of acquired syphilis begins by a chancre, except very
rare instances of direct blood inoculation (physicians pricking a
finger during an operation). But in a good proportion of cases (10%
according to some authors) the chancre is overlooked.

Without treatment, the chancre *heals* in a few weeks (much quicker
under the influence of treatment), leaving *no scar* if no secondary

infection has taken place, because the erosion of the chancre is *purely epidermic* and does not involve the corium. Suppuration only takes place when the chancre is infected by pus cocci; sometimes the chancre becomes *phagedenic*.

Mixed chancre is due to simultaneous infection by the *spirocheta pallida* and the *Ducrey* bacillus: owing to the shorter incubation period of the latter, the chancroid develops first, and induration appears later, sometimes when the soft chancre has already healed up.

From the chancre, its initial focus, the spirocheta pallida invades the whole body. First, about two weeks after the appearance of the chancre, the *regional lymph glands* become enlarged and form hard, painless, movable swellings, which never suppurate, except in mixed infections; later, the *adenopathy becomes general,* and what has been called the *"secondary explosion"* takes place.

It is characterized by general constitutional disturbances similar to those of constitutional infectious diseases, foremost among which are anemia, fever, and especially *pains in the muscles, bones and joints,* and *headache;* all these painful symptoms are *more marked at night,* are not influenced by analgetic drugs, but yield to specific treatment. Headache is due to arteritis of the cerebral arteries: it is an evidence of early involvement of the nervous system; but there are many cases without headache in which the nervous involvement is proved by the lymphocytosis of the cerebro-spinal fluid, and the increased proportion of albumin in the latter. Nervous involvement exists in 67% of the cases of syphilis during the secondary period (*Ravaut*).

Externally, the secondary period is heralded in by an **eruption.** A rose-red *macular rash* (roseola) develops on the abdomen and thorax. Later on various eruptions develop (secondary syphilides), the most common of which is an eruption of flat, rounded, reddish-brown or ham-colored *papules* on the trunk, face and limbs. On the forehead these papules form the so-called "corona veneris." On the genital organs and around the anus these papules become sodden and white, and are known as *condylomata lata,* which are liable to ulcerate. In some cases pustular eruptions form, and in severe or neglected cases the pustules become ulcers covered with limpet-shaped crusts (syphilitic rupia). Acneiform eruptions are common on the scalp, and scaly or psoriasiform syphilides on the palms and soles. *Most secondary eruptions disappear without leaving any trace,* but the ulcerative forms (rupia) leave pigmented scars, which later on become white in the centre.

The macular rash is *toxic* in origin; all the other syphilides are *round-celled infiltrations,* similar to the round-celled infiltration of primary sclerosis, similar also to the infiltration of tertiary gummata. These infiltrations never tend to permanent connective tissue organization, but to involution and resorption. They always *spread peripherally,* while the centre heals up.

Clinically syphilitic eruptions are characterized by their reddish-brown or ham color, their tendency to polymorphism, to occur in groups, and to be arranged in circinate figures (more marked in the later stages of the disease and in the negro race [*Fox*]), the absence of subjective symptoms, itching or pain.

The mucous membranes, especially of the mouth, are affected by papular, erosive or ulcerative syphilides, which are known as *mucous patches.* These develop on the tonsils, fauces, tongue, and inside the lips and cheeks, in the form of grayish-white patches or streaks, with a red border. Later on they may become eroded or ulcerated in their central parts, and then appear as red erosions with a gray border. In early secondary syphilis the tonsils and fauces may be acutely swollen (syphilitic angina), but more often there is a dark-red coloration of the tonsils, fauces and soft palate.

In secondary syphilis there often is a special *alopecia areata.* The loss of hair is sometimes due to acneiform syphilides of the scalp, but more often appears without any apparent lesion. The nails are sometimes affected with onychia or paronychia.

The secondary period lasts until the symptoms of an active general infection disappear, that is, from a few months in most cases, to one or two years. Then outward signs may keep lacking forever: perhaps in a few cases there is a complete cure, but of the latter there is no proof, and we have no criterion to judge of it. In a rather large number of cases the disease is simply latent, as evidenced by the positive result of the *Wassermann* test, and by the subsequent development of parasyphilitic conditions, chiefly of the nervous system. In some other cases there occur circumscribed or diffuse infiltrations, *not differing in any essentials from the round-celled infiltration of the other periods of syphilis* and called **gummata.** The latter are considered (somewhat arbitrarily) as the characteristic feature of the stage of syphilis designated as **tertiary.** Gummata may appear in all stages of the disease, but they are rare during the secondary period, and the older the infection becomes, the less chances of seeing gummata develop. With the ordinary mercurial treatment of but few years ago, a great majority

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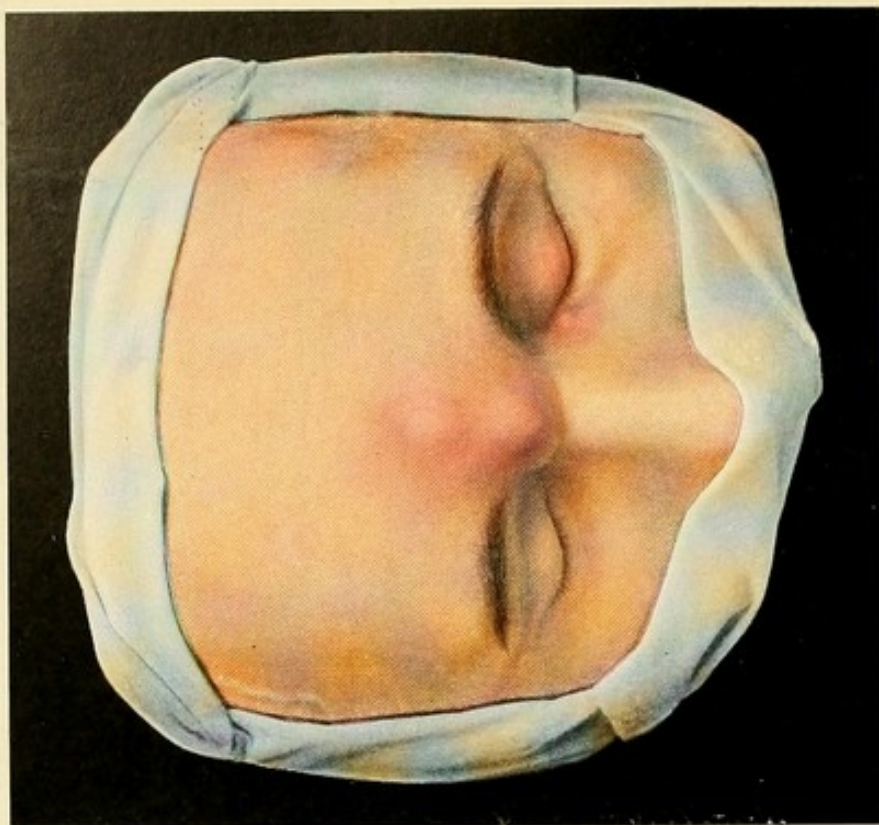
Fig. 121. Abscessus gummosi.
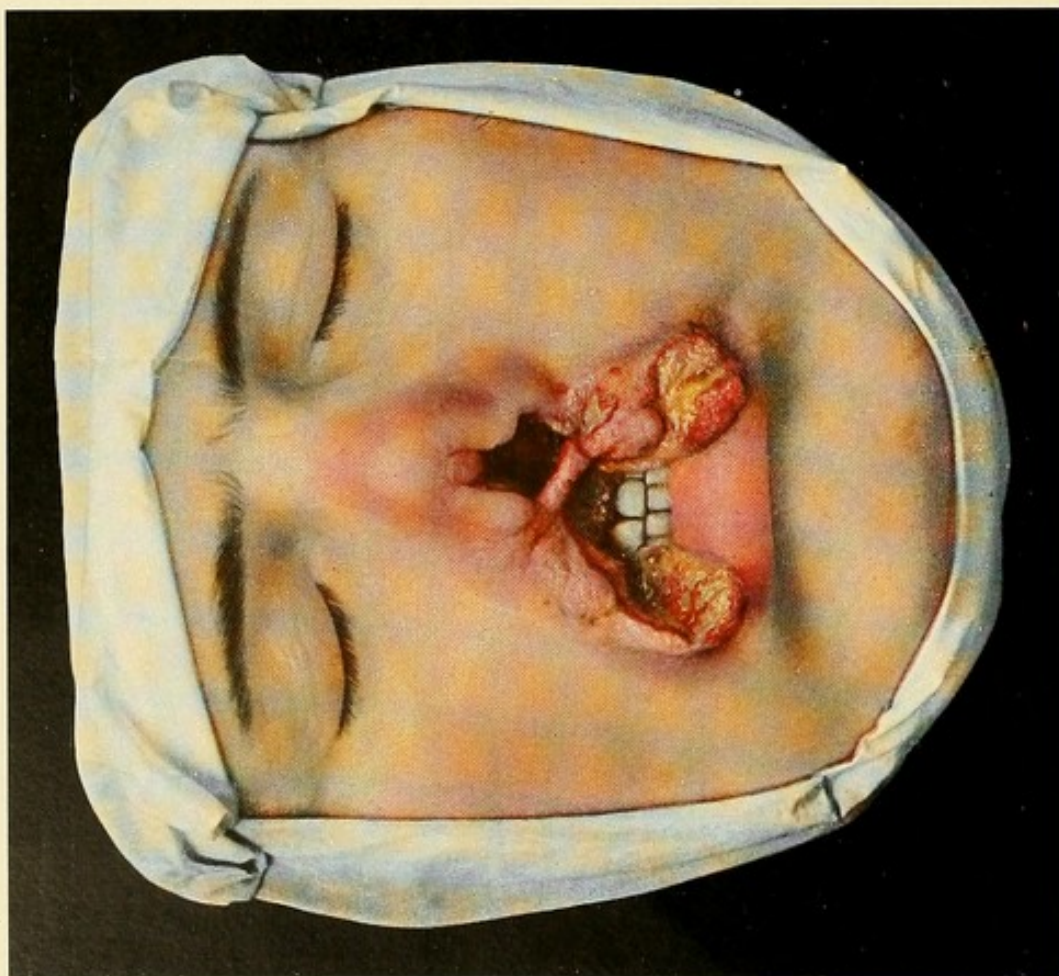
Fig. 120. Gumma labii superioris et nasi.

of the treated cases remained free from gummatous lesions. With the newer methods, still better results may be confidently expected.

In gummata, the tendency to simultaneous peripheral extension and central regression exists as in all syphilitic processes; but, owing to the ever-present endarteritis and consequent interference with the nutrition of the syphiloma, the inability to organize into permanent connective tissue reaches a maximum, and the central part undergoes *fatty degeneration* and *caseation*. A gumma naturally tends to formation of a cold abscess; if the latter is superficial, it breaks through the skin and thus gives rise to a **gummatous ulcer** (**Figs. 120, 122** and **123**). **Fig. 121** shows a non-ulcerated abscess.

For a long time, tertiary lesions were considered non-contagious. Spirochetæ in such lesions are few in number and less virulent than in the earlier stages, but the inoculability of gummata has been demonstrated beyond doubt.

Gummata occur in all tissues of the body. In the skin and subcutaneous tissue they first appear as circumscribed nodules. After a while, the skin becomes red (**Fig. 121**), fluctuation is felt, and, finally, a characteristic gummatous ulcer (see more particularly **Fig. 123**) is produced. The borders are hard, smooth, not undermined, but circular and sharply cut, as if punched out; the surface is covered by a tough, tenacious, yellowish deposit, or core. If the latter be forcibly removed, no bleeding occurs (endarteritis and blocking of the small arteries).

In the skin several gummata usually occur close together (**Fig. 121**); these break down in some places and heal in others, thus giving rise to an irregular or serpiginous appearance, which is characteristic of tertiary syphilitic ulceration (**Figs. 120** and **123**).

Gummata of the skin or mucous membranes may extend to the deeper tissues, and *vice versâ*. Diffuse gummatous infiltration of the skin and subcutaneous tissue gives rise to multiple fistulæ, which discharge a scanty secretion.

Gummata may cause extensive deformity by destruction of tissue, especially in the face (**Fig. 120**). In these cases the soft parts and bones are generally affected together, whichever may have been primarily affected. This leads to necrosis of the bone. One of the most frequent localizations of this gummatous infiltration, suppuration and necrosis process, is the nose and hard palate; it is also one of the most disfiguring, and one of the most painful and disagreeable for the patient (dysphagia and offensive smell).

Primary gummata of the bones may develop in the periosteum,

cortex or medulla, as circumscribed growths or a diffuse infiltration. Generally, all three parts are affected with simultaneous destruction and proliferation, causing an irregular, corroded appearance of the bone. Gummatous osteitis may undergo fibrous transformation, or may suppurate, cause necrosis and lead to ulcer formation (**Fig. 122**). Necrosis of the cranial bones often leaves circular cavities, to which the smooth, glistening skin is firmly adherent.

Syphilitic disease of the *tibia* is the most frequently seen in the long bones. The weight of the body may produce a curvature of the bone. Abnormal brittleness of the latter, possibly leading to spontaneous fracture, is not very rare.

Examination by X-rays shows irregular shadows in the periosteal region, while the cortex and medulla cannot be distinguished from one another. The whole bone is thickened and irregular.

Patients often complain of pain, *chiefly nocturnal*, in the bones (osteocopic pains) before any changes are visible. Palpation of the anterior surface of the tibia often reveals an irregular, uneven surface. The ulna and radius (**Fig. 122**), fibula, sternum and clavicle may also be the seat of syphilitic osteitis.

Articulations are also involved in tertiary syphilis, either by gummatous infiltration of the perisynovial tissue, or specific osteitis of the articular ends. The knee is the joint most frequently affected.

Gummata are also seen in the muscles (gastrocnemii, sterno-cleido-mastoid, tongue). Gumma of the tongue (**Fig. 119**) is usually situated in the centre, and associated with sclerous glossitis, and may make the organ assume a bifid shape, as shown in the figure.

The brain, the liver, the heart, the testicles, the lungs, the thyroid gland, the pancreas, the adrenals can all be the seat of gummatous degeneration. Each localization has its special symptoms. Particularly interesting are the gummata of glands having an internal secretion, and their possible, but not yet elucidated, relationship with symptom complexes depending on alterations of those glands (pancreas and diabetes, adrenals and *Addison's* disease).

In hollow viscera, pharynx, larynx, rectum, the cicatrization of gummatous ulcerations entails marked deformity, on account of *cicatricial stenosis.*

The constancy of vascular lesions in syphilis explains the frequency of post-syphilitic arterial degenerations (see about *aneurysm*, page 106, and *arteriosclerosis*, page 205).

Congenital syphilis may be congenital in the strict sense of the

Tab. XCVI.

Fig. 122. Ostitis gummosa.

word, that is, exist at the time of birth, or be delayed in its appearance several years.

Among the characteristic signs of early hereditary syphilis are bullous syphilides of the palms and soles (*syphilitic pemphigus*) and *epiphysitis*. The latter is a form of osteochondritis affecting the epiphyses of the long bones, and causing thickening. It is more common in the arm and gives rise to impotency of the limb (*Parrot's* syphilitic pseudo-paralysis). Epiphysitis may interfere with the growth of the limb.

In late hereditary syphilis the bones are frequently affected with gummatous processes identical with those of acquired syphilis. The tibias are often curved forward and outward owing to osteoplastic periostitis. This condition is known as "saber blade tibia," and is a characteristic sign of late hereditary syphilis. The skin over the bones is often ulcerated.

Syphilitic dactylitis may occur in both early and late hereditary syphilis. It causes thickening of the phalanges, usually the basal ones. It is generally multiple, sometimes bilateral, and tends to spontaneous resolution without suppuration.

The bones in hereditary syphilis are often very brittle. Other signs of hereditary syphilis are *interstitial keratitis, deafness* (due to disease of the internal ear), *notching of the incisor teeth* (*Hutchinson's teeth*). These three signs have been called the **"Triad of Hutchinson."** Radiating scars around the mouth left by former ulcerations are also characteristic.

Acquired syphilis may also occur in infants and children, but it then in no wise differs in its evolution from the acquired syphilis of full-grown subjects. It, however, markedly stunts the growth of the body.

Diagnosis

Syphilis is so widespread among **all** *classes of society that it must always be borne in mind in cases of doubtful diagnosis.*

No circumstantial evidence, no consideration of social standing or of personal habits may ever be made sufficient ground for an *a priori* rejection of the diagnosis of syphilis. Syphilis is ubiquitous and polymorphous to the extreme.

The diagnosis of syphilis **must be made as early as possible.** Not many years ago, the only evidence we had at our disposal to decide whether a **chancre** was syphilitic or not was purely clinical. The features well shown in **Fig. 118,** particularly the *cartilaginous,*

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elastic induration of the base, the appearance of the *regional adenitis,* and finally, the secondaries allowed of an easy recognition in a great majority of cases. But the diagnosis could be made with certainty only if *several* signs were present; often, *it was necessary to wait to observe the evolution,* and this is no longer considered as desirable, but, on the contrary, as **very detrimental to real therapeutic success.** The discovery of the *Spirocheta pallida* has enabled us to depend less exclusively on clinical evidence (which, however, remains of the *utmost importance,* but sometimes is deceptive, owing to the frequent existence of mixed infection), and, nevertheless, to make an *earlier,* more *certain* and more *accurate* diagnosis. *In every suspicious sore,* wherever located, *search must be made for the spirochetæ,* in the serosity, either directly with the *dark ground illuminator* or after staining. The India ink stain is technically the most rapid and the simplest: the identification of the spirochetæ may be a little more difficult, but with some practice the method is fairly safe and satisfactory; it however remains inferior to the dark field illuminator, which observes the *living* features of the organism.

When *spirochetæ pallidæ* are found, it at once clears the diagnosis, and treatment must be started without delay.

(Compare the objective aspect of *chancre* (**Fig. 118**) and *carcinoma* (**Figs. 1, 7, 8, 9**).

Secondary syphilides of the skin and mucous membranes may be mistaken for various affections, though, when a number of symptoms are present and they are all taken in consideration together, hesitancy is hardly possible. Here also *laboratory methods* bring an extremely important adjunct to clinical investigation, namely, the **complement fixation,** or *Wassermann* **reaction.**

The latter is not of much usefulness in the diagnosis of chancres, because it is not present from the beginning, and it sometimes develops rather late in the primary stage, but in the secondary period and, generally speaking, while the disease is active, it remains *positive.* The diagnostic value of the test is greatly enhanced by the fact that it exists only in a few other diseases, which cannot be mistaken for syphilis (scarlet fever, noma, leprosy). *Therefore, a positive reaction means syphilis;* but a negative reaction has much less value, because it does not mean necessarily that there are *no* spirochetæ in the body, but simply that if any are present they are either too few in number to induce antibody formation, or encapsulated so that their toxins do not enter the circulation. Encapsulation

being frequent in tertiary lesions, it is readily understood why many manifestly luetic conditions are accompanied by a negative reaction.

Negative *Wassermann* tests acquire value only when a series in the same patient covering a long period of time (a year) gives uniformly negative results (and all possibilities of defective technique have been eliminated) and there are no active clinical manifestations. However, valuable as it is, the *Wassermann* reaction is but *one* element of diagnosis, and it cannot be made the sole criterion of diagnosis, prognosis and treatment. There has, perhaps, been a little exaggeration in this respect.

For the diagnosis of **tertiary lesions** laboratory methods do not bring as much help as in the earlier stages, as spirochetæ are not to be found in the discharge of gummatous ulcers, and the *Wassermann* reaction is negative in a considerable proportion of cases. The *luetin* reaction (*Noguchi*) has not yet been sufficiently studied. Consequently, the clinical diagnosis still retains all of its former importance.

Tuberculosis and **tumors** are the two great causes of error in the diagnosis of gummata.

Tuberculous ulcers have an anemic appearance, undermined edges, no yellow core (see **Fig. 130**); tuberculous pus is thin. In the testicle gumma begins in the testicle itself, while tuberculosis begins in the epididymis (see page 203).

In muscles, in the brain, liver, spleen and other internal organs, the symptoms of gumma are at first those of *tumor*, and it is only by the anamnesis, the *Wassermann* reaction and a process of exclusion that the real diagnosis may be reached.

We have already spoken of the diagnosis between **gumma** and **cancer** about carcinoma of the tongue (see **Figs. 7, 8** and **9**, and page 10; compare with **Fig. 119**), and carcinoma of the lip (see page 6; compare **Figs. 3** and **5** with **Fig. 120**).

Diffuse gummatous infiltration of the skin with fungoid proliferation may suggest sarcoma (cf. **Figs. 24** and **26**), but differs in the absence of any tendency to bleeding, in the presence of circular scars and brown pigmentation of the surrounding skin, and the existence of other signs of syphilis.

Central gumma of bone may resemble central sarcoma or bone cyst, and may give the same appearance on X-ray examination, but gummatous changes in bone are characterized by implication of the periosteum. In doubtful cases, a test course of antisyphilitic

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(Salvarsan) treatment may be given, but too much time must not be wasted (see page 11).

A gummatous ulceration such as that shown in **Fig. 123** somewhat resembles objectively a furuncle (see **Fig. 87**), but the evolution is so different that no confusion is possible.

Gummata must also be distinguished from the lesions of **sporotrichosis,** an infectious granuloma, chiefly of the skin and sub-cutaneous tissue, due to the sporotrichum fungus, first described by *Schenck*, later by *Hektoen* and *Perkins*, and well studied by French writers, foremost among whom *de Beurmann* and *Gougerot*. The nodular formations and indolent abscesses of sporotrichosis, which particularly affect the arm and forearm, can be clinically suspected, but truly identified only by culture of the fungus; these are characteristic, with their radiating fringe of mycelia extending deep into the medium as a white mass; microscopical examination shows the typical ovoid spores. Histologically the lesions of sporotrichosis cannot be differentiated from those of tuberculosis, and it is very likely that, in former times, many cases have been diagnosed and treated either as syphilis or tuberculosis.

In **congenital syphilis** the lesions are generally typical and the *Wassermann* is always strongly positive.

In **parasyphilitic affections,** the frequent occurrence of a strongly positive *Wassermann* indicates that there is still an active syphilitic process, and gives a hopeful therapeutic indication. In many cases of parasyphilitic conditions of the nervous system, the blood *Wassermann* may be negative, while the test made with the cerebro-spinal fluid is unmistakably positive. Examination of the same cerebro-spinal fluid for lymphocytosis and albumin contents also gives positive results.

Treatment

The treatment of syphilis must be **begun as soon** *as the diagnosis is made.*

It must be **kept up** *until all clinical manifestations have disappeared, and the Wassermann reaction has become permanently negative.*

If, for a year, the reaction remains steadily negative in a patient who, meanwhile, has not received any treatment, the presumption of a **cure** is justified; but, up to now, we have no decisive proof of a radical cure of syphilis, except when genuine reinfection occurs, and

Tab. XCVII.
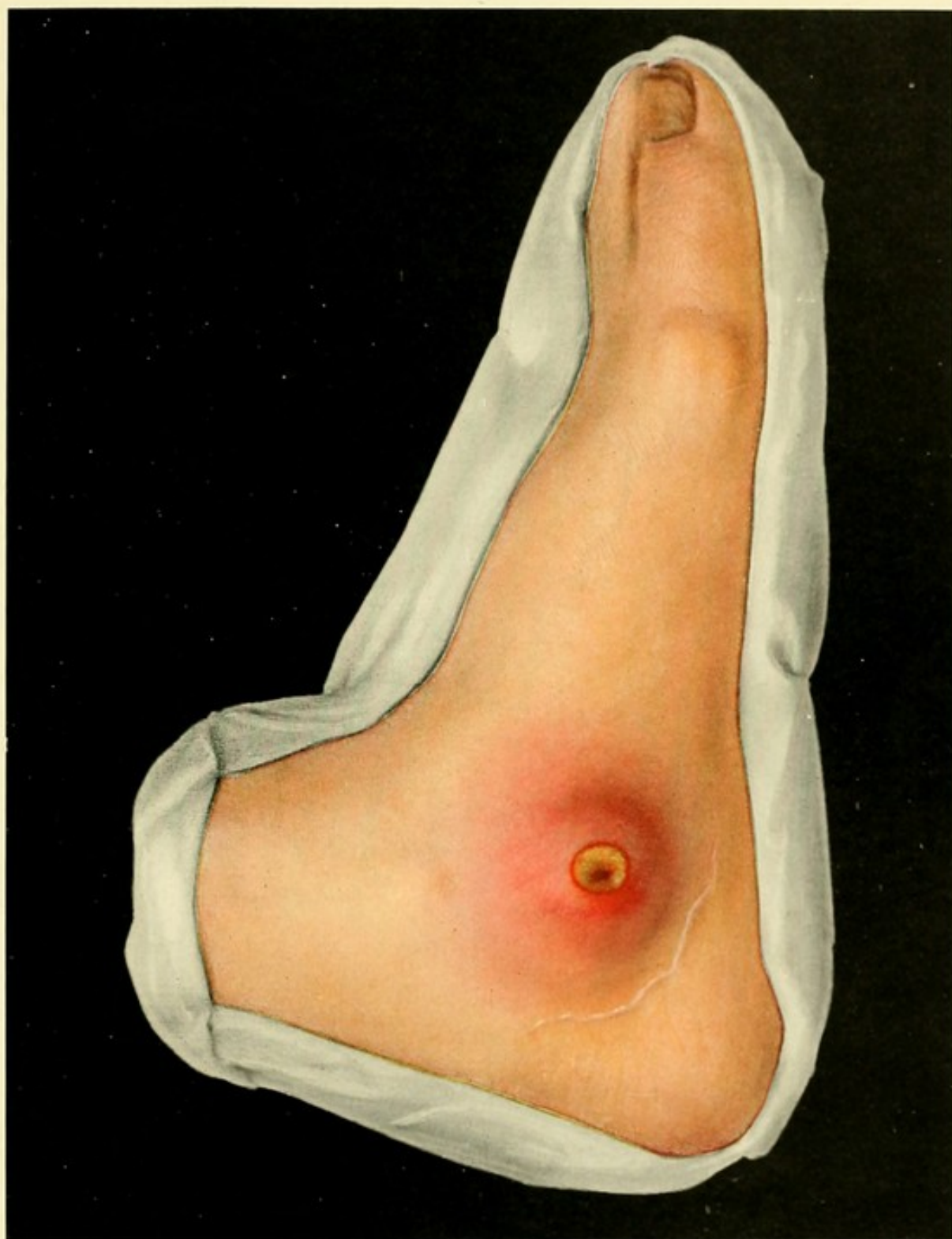
Fig. 123. Ulcus gummosum.

this seems to become much more frequent with the newer methods of treatment (*Fordyce*).

All direct syphilitic processes are influenced by antisyphilitic treatment. Parasyphilitic conditions are not so favorably, and it has long been claimed that they were not at all, influenced; but this is no longer strictly true for some of them, though remaining the general rule.

The treatment of syphilis, formerly restricted to mercury and iodine derivatives, chiefly iodides, and already satisfactory, has been considerably strengthened in the past three years by the discovery of salvarsan, and quite recently, by that of neosalvarsan. Whatever may be argued about the *curative* value of these drugs in the general treatment of syphilis (a question which time alone can answer), it cannot be gainsaid that they have very remarkable *healing* properties on all syphilitic lesions, primary, secondary and tertiary, even on those types that formerly proved refractory to mercury and iodides.

Neosalvarsan is somewhat *slower* in its action than salvarsan; but it is also *less toxic,*[1] *much easier to administer,* and can be used in *much higher doses,* at *short intervals*—an invaluable advantage in the attempted abortive treatment of early cases.

If the diagnosis of chancre is made early (within two weeks of the onset), a series of four or five intravenous injections of neosalvarsan every other day, so that the total dose is at least 4.5 grams (corresponding to 3 grams of salvarsan), or more in strong individuals, is well tolerated, and **may** abort the disease; that is, the *Wassermann* reaction never becomes positive, and no secondaries develop. Of course, the *Wassermann* must be taken at frequent intervals and the treatment resumed at the slightest indication of the reaction tending to become positive again. It is even wiser systematically to start a course of mercurial treatment (inunctions and injections) shortly after the first series of neosalvarsan injections, and three months later, to give two more intravenous injections.

When the diagnosis is made later, when the *Wassermann* is already positive, and *a fortiori* when secondaries have developed, an abortive treatment is no longer possible; but the treatment must remain *intensive* and *prolonged.* Insufficient treatment is the source of all neuro-recurrences. This does not necessarily mean that high doses must be given; *smaller doses frequently repeated,* and mercurial treatment between the salvarsan courses, seem to be better adapted to

[1] Though, probably, slightly more *neurotropic.*

the nature of syphilis, a chronic intermittently relapsing disease, calling for a chronic intermittent treatment. The *Wassermann* reaction is the best guide for the treatment; latent cases with a positive *Wassermann* must be deemed active cases, in silent relapse, and treated accordingly.

Salvarsan, and also mercurial treatment, have a decided influence on the *Wassermann* reaction. However, this action becomes less marked with the age of the disease, and a positive *Wassermann* in an old case is generally very hard, if not impossible, to change into a negative.

In tertiary lesions, salvarsan treatment works wonders in most cases. Several intravenous infusions of 0.6 gm. (on the corresponding amount of neosalvarsan) will make a gummatous infiltration melt very quickly without leaving any traces, or will cause an ulcer to heal in much less time than was formerly needed with mercury and iodides. Of course, if further treatment is desired for general vascular lesions, after the gumma has healed, iodides must be resorted to. When a lip or the nose have been destroyed by a gumma, plastic repair of the defect may be required. But it henceforth will be very exceptional that a syphilitic lesion will require more than antisyphilitic treatment (for instance, scraping in ulcers, extraction of sequestra in osteitis). We now can make a diagnosis early enough, and have a treatment powerful enough, to be able to prevent any syphilitic lesion from causing irretrievable destruction of tissue.

In congenital syphilis, salvarsan treatment is also efficient, but the *Wassermann* reaction is never changed.

In parasyphilitic diseases of the nervous system, salvarsan treatment sometimes seems to give favorable symptomatic results. Its real value is not yet fully established. It is contraindicated in advanced lesions of the nervous system and in marked cardiovascular disease.

Fig. 124. Lymphomata colli tuberculosa.

TUBERCULOSIS

Figs. 124 to **131,** inclusive, show a number of conditions all caused by the **tuberculosis bacillus.** They all belong to the types termed "surgical" tuberculosis; those cases are nowadays generally attributed to the bovine bacillus, while the "medical" forms, tuberculosis of the lungs, internal viscera and acute miliary tuberculosis, are due to the humane bacillus.

Whether tuberculosis is chiefly an *inhalation* or an *ingestion* disease is still a much debated question. From a practical standpoint it matters little which mode is the more frequent: both are possible, and both must be guarded against.

"Surgical" tuberculosis lesions, such as those here figured, are *seldom primary; almost always,* if not always, they are **secondary** to a primary focus situated elsewhere in the body (most often lungs and bronchial lymph glands). The propagation is generally admitted to take place by the *blood;* but the *lymphogenous* origin of many a case is now established.

The tuberculosis bacillus may settle in all tissues of the body: it, however, has a preference for some of them. The muscular tissue is the least frequently affected. Among surgical cases, the **lymph glands** (**Fig. 124**), the **bones** and **articulations** (**Figs 125** to **128,** inclusive, **130** and **131**) are the most frequent sites. All glandular organs are often involved. **Fig. 129** shows a very common condition, tuberculosis of the **testicle.**

When the bacillus invades an organ, it sets up an inflammatory reaction in the surrounding tissues. After the first stage of leucocytic infiltration, common to all infections (see page 117), the reaction of the tissues to the tuberculosis bacillus assumes a special type, which leads to the formation of a **tubercle.** In the latter, there always are a large number of round cells, and in fresh tubercles epithelioid cells; in older tubercles, giant cells, which are agglomerations of leucocytes engaged in the destruction of bacilli.

The tubercle exhibits a double tendency in its evolution. Owing to the absence of blood-vessels in its centre, the latter undergoes **caseation;** but, at the same time, there is, at the periphery, a natural tendency to limit and surround the focus by hyperproduction of **fibrous tissue.** Central caseation and purulent disintegration, on the one hand, and peripheral sclerosis, on the other hand, always

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progress simultaneously, but not necessarily equally. According to whether one or the other process is more marked, or both are equal, the tubercle becomes *caseous,* or *fibro-caseous,* or *fibrous,* and the evolution is toward aggravation, a stationary or slowly progressive condition, or spontaneous recovery.

Recovery may occur spontaneously by fibrous encapsulation of the focus, which then becomes simply quiescent or undergoes calcification. Judging from the results of autopsies of old people, about 70% of all subjects have, at one time or another, harbored in their body tuberculous foci which have spontaneously healed. But a simply quiescent focus is always a menace to health. Under the influence of trauma or of decreased resistance of the body, it may become active again, give rise to new lesions or even to generalized miliary tuberculosis.

Recovery may, and in fact does often, take place after caseation, and formation of a **cold abscess:** such abscesses generally open outside, and become fistulous. Tuberculous *fistulæ* are surrounded by pale, anemic-looking, vitreous granulations. They run an irregular course and sometimes open at distant points of the skin (especially in bone tuberculosis); the walls of the fistula are soft and bleed easily. The *pus* is thin and mixed with fibrin, caseous masses and shreds of tissue. The *tuberculous ulcer* is characterized by thin, soft, ragged, undermined borders, and a base covered with yellow, caseous masses, or pale-red or gray granulations. Tuberculous granulations may destroy all the surrounding tissues (bones, cartilage and muscles). In these cases, the extension is almost indefinite, as there is but little fibrous tissue formation. Besides, external fistulization of a tuberculous lesion almost unavoidably entails the risk of *secondary pyogenic infection, a decidedly unfavorable element.* The pus of tuberculous fistulæ is not very virulent, and the danger of tuberculous contamination by open foci of "surgical" tuberculosis is negligible, if not absolutely *nil.*

Diagnosis and prognosis

The diagnosis in case of open lesions is often not difficult. It has to be examined separately for each special localization of the disease (see below).

A doubtful diagnosis of tuberculosis may be confirmed by *intraperitoneal inoculation to the guinea-pig, microscopical examination,* or a *tuberculin test,* subcutaneous (*von Pirquet*), intradermic (*Mantoux*) or percutaneous (*Moro*). The inoculation is the most accurate and reliable method; unfortunately, it entails too long a delay.

It is futile to look for the tubercle bacillus in smears of tuberculous pus of ulcerated lesions; it is never found. If no associated pyogenic infection is present, the pus seems to contain only pus cells and no micro-organisms at all. *Such a pus without microbes is always very suggestive of tuberculosis.*

The prognosis depends on the extension of the disease, the power of resistance of the patient, and the tendency to fibrous tissue formation. The extension of the disease is directly influenced by an early diagnosis and appropriate treatment.

Treatment

It must be *prophylactic, general* and *local.*

We cannot discuss here at full length the prophylaxis and general treatment of tuberculosis. Suffice it to say that in surgical conditions the latter is always indicated and that, particularly in children, *sunshine* (heliotherapy) and *seaside treatment* will work wonders, and, with conservative measures, often bring about a cure; while in full-grown subjects, operative measures are more frequently called for.

Tuberculin treatment (TR or BE) has been much discussed in recent years. If applied in such a way as not to cause general febrile reactions, it has a certain usefulness, chiefly against toxic symptoms, and is not harmful. Consequently, as an adjuvant to other treatment, it is welcome; but it *cannot alone take the place of other treatment,* except, perhaps, in very early cases; even then, the evidence at hand is still to meager to allow definite conclusions.

Surgery does not cure tuberculosis, but it considerably helps the natural defense of the body, and may turn the tide of battle in favor of the latter, while before operation the tuberculous process had the better of it. A striking example is afforded by reno-vesical tuberculosis. As long as the tuberculous kidney remains in place, all treatments are unavailing to cure the bladder ulcerations; as soon as the kidney is removed, those same ulcerations heal up, often without special treatment. It must be remembered that surgical cases of tuberculosis are *secondary* to a primary focus, which generally cannot be reached; that, therefore, our operations for tuberculous conditions can *never claim to be radical,* but are only *partial.*

Each type of surgical tuberculosis gives rise to special therapeutic indications, to be considered later on. We shall here, however, to avoid repetitions, set forth briefly the general methods of treatment.

If the focus is *apparently* well localized (tuberculosis of the skin, of the kidney, of the lymph glands, for instance), **extirpation** is the method of choice. We know that there is no *really* localized tubercu-

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losis; but, in many cases, even a *very incomplete removal* of the diseased tissue will considerably help; as the natural defensive power of the body, *which, after all, is the* **one curative agent** *in tuberculosis,* will be able to do the rest. This explains why **scraping** of tuberculous lesions (some forms of lupus, and chiefly open *fistulous* foci) aften does so much good.

Hyperemia in tuberculous lesions is not as useful as in acute pyogenic infections: active hyperemia is absolutely contraindicated; passive hyperemia must be very carefully handled and its results are uncertain.

Whenever a focus undergoes caseation and causes an abscess, our aim at first must always be to bring about a cure **without external opening,** because of the danger of secondary infection. Therefore, primary incision of a cold abscess is not advisable: if a tuberculous pus collection threatens to ulcerate the skin and become fistulous, it is better to ward off this eventuality by **tapping** (not directly over the swelling, which would immediately lead precisely to what we are seeking to avoid, namely, fistulization, but *very obliquely,* so as to make a long subcutaneous track, which closes readily) and **injecting** within the abscess cavity some fluid capable of happily influencing the evolution of its walls; camphor naphthol, iodoform dissolved in ether (5%) or suspended in glycerin (10%). Iodoform has long been credited with a special influence on tuberculous fungosities; it certainly works well, better than any other substance. These tappings may be repeated; they are often sufficient, when combined with general treatment, to bring about a cure without open operation; but if, despite the injections, a fistula develops, exposure of the focus and scraping is generally indicated.

For the treatment of tuberculous fistulæ, either developed spontaneously, or persisting after an operation, *Beck* has strongly urged the employment of *vaselin-bismuth* (33%) paste as a filling mass. When no foreign bodies are behind the fistula (sequestra, for instance), *Beck's* method is successful in a great number of cases and undoubtedly is very valuable. The danger of bismuth poisoning exists, but is very small, and is *nil* when dealing with not too large a cavity. The method is applicable, no matter what organ (bone, lymph and gland, etc.) is the origin of the fistula.

Fig. 124 represents a case of **tuberculosis of the submaxillary and cervical glands,** in a man who since youth had suffered from eczema of the face and inflammation of the eyelids.

While tuberculous lymphangitis is rare and occurs only, in the

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form of nodular cords, in connection with tuberculosis of the skin or lymph glands, tuberculous adenitis is extremely frequent and surgically important.

All lymph glands of the body may be affected, but not with equal frequency. Tuberculosis of the visceral glands chiefly concerns the internist. Tuberculous adenitis is not common in the axillary and inguinal glands, but it is very frequent in the glands of the neck in children. Tuberculous adenitis of the neck is one of the chief elements of what was formerly called "*scrofula*"; that is, a seemingly not very virulent form of tuberculosis, in which repeated attacks, as evidenced by chronic blepharitis, eczema, torpid ulcers, otitic discharge, have apparently induced some general immunity. The portal of entry is generally found in the mouth or pharynx. Acute infectious diseases with bucco-pharyngeal lesions (scarlet fever, measles, etc.) are frequent predisposing causes.

In the glands, the bacilli first induce the formation of miliary tubercles. Several of the latter become confluent and form larger nodules. Later the process extends to the periglandular tissue and skin. Then the node is no longer covered by intact skin under which it is freely movable, but becomes adherent. The skin finally ulcerates and a cold abscess discharges typical thin greenish pus, often in places remote from the primary node.

The differential diagnosis of cervical adenitis has already been considered on page 31, about **Fig. 24**. Some cases are typical and the diagnosis is easy: this is particularly true of those cases in children which are accompanied by the *scrofula mask* (thick, prominent upper lip, blepharitis, skin eruptions, etc.), or in those cases in which there is a characteristic tuberculous fistula. Early in the evolution, when there are only round, movable, circumscribed lumps, the diagnosis is much more difficult. Excision of one, microscopical examination and a tuberculin test may be resorted to.

The treatment must include *that of the cause,* and be *general* and *local* (see above, General treatment of tuberculosis). Circumscribed lymph gland abscesses may be tapped with a fine needle, and the cavity injected with a 10% emulsion of iodoform in glycerin. Large ulcerated abscesses may be scraped and packed with iodoform gauze. Fistulæ, especially when persistent, are successfully treated by injection with *Beck's* bismuth vaselin paste.

Good results have been reported by several writers from the use of X-rays. But the general tendency nowadays is toward *surgical ablation* when the lymph glands have not yet reached the caseation and suppuration stage, and when general treatment alone is insuffi-

cient. Tuberculous lymph-glands of the neck are usually removed by a free longitudinal incision along the *anterior* (or, less often, the posterior) border of the sterno-cleido-mastoid muscle. These glands are always in close connection with the big vessels and nerves of the neck, and not unfrequently they form a continuous chain from the base of the skull down to the supraclavicular fossa.

Transverse incisions (*Kocher, Dowd*) are preferred by many, because they give a much less visible scar, almost completely hidden in the normal folds of the skin.

Gentleness of manipulation and avoidance of undue squeezing of the mass are required in extirpation of tuberculous glands, so as not to send any tuberculous matter in the general circulation. In removing glands with suppuration in their interior, care must be taken not to break into them and thus infect the wound.

Figs. 125 to **128,** inclusive, show different types of *joint tuberculosis;* **Figs. 130** and **131** two cases of *bone tuberculosis.*

Joint and *bone tuberculosis* (which must be considered together, on account of their close relations) are surgically the two most important forms of tuberculosis.

Tuberculosis invades *bones* by *way of the blood.* It may invade articulations in the same manner, but this primary hematogenous mode is comparatively rare, and tuberculosis of joints is ordinarily *secondary* to a juxta-epiphyseal focus of bone tuberculosis which has extended to the articular cavity. The bones are generally affected in certain places according to the distribution of their blood-vessels. The nutrient artery of the long bones terminates in the epiphyses; this explains the frequency of tuberculous deposits in these same *epiphyses,* whence the line of least resistance for propagation naturally leads through the cancellous tissue to the joint. In short bones the nutrient artery ends soon after its entrance in the middle of the bone: hence the localization of tuberculosis to the middle.

Among the epiphyses most frequently attacked are those of the tibia and femur: hence the predominance of tuberculosis in the *hip, knee* and ankle joints. Next come the wrist and elbow.

The evolution of the tubercle bacillus in *bones* is the same as in any other tissue of the body. The central necrosis leads to the formation of a sequestrum. Small sequestra often give rise to large cold abscesses, which become visible under the skin (**Fig. 125**), often at some distance. In tuberculous bone disease *there is little tendency to the formation of new bone* or to *peripheral sclerosis,* therefore, little tendency to *limitation* or *spontaneous recovery.*

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In some rare cases the focus of disease may become encapsulated in the bone, but is always liable to recrudescence, especially after an injury. More commonly the sequestrum is discharged piecemeal through a fistula, thus differing from the large sequestrum of pyogenic osteomyelitis. Multiple foci of disease often occur in one or more bones. When the bone is exposed by incision, irregular, corroded, caseous fragments are seen, together with pus. When the disease occurs in the epiphyses of the long bones it may break into the joint; this, as already stated, is the common causative mechanism of joint tuberculosis.

In the ribs, the lesions are usually confined to the periosteum and to the formation of a large subperiosteal abscess. In the phalanges tuberculosis gives rise to a special type of osteitis (*spina ventosa,* **Fig. 131**), to be spoken of later.

In **joints** not only the articular ends are involved, but also the synovial membrane and periarticular soft parts. Here the anatomical evolution is not as one-sided as in pure bone tuberculosis; central necrosis and peripheral sclerosis are better balanced, and instead of the merely destructive type of lesions found in bones, we find mixed types due to the predominance of one or the other process.

First, in the rare types of primary hematogenous infection of the synovial membrane, we find granulation tissue and effusion in the joint. In the mildest forms the effusion may be only serous (**articular hydrops**), but more commonly it is sero-fibrinous. The fibrin forms villous deposits on the synovial membrane and cartilage, and so-called "rice bodies," which are lumps of loose fibrin in the joint, rolled by the motion of the latter.

A second form is known as **fungoid arthritis,** owing to the formation of fungoid or spongy granulation tissue, which gives rise to globular swelling of the joint. In these cases the whole joint is filled with grayish-red or yellowish-white granulations, and there is only slight exudation. The fungous granulations tend toward *caseous degeneration,* and after a time to suppuration. This form does not remain limited to the joint, but soon extends to the ligaments and periarticular structures, and eventually to the subcutaneous tissue and skin (**Figs. 125** and **126**).

A third form is **fibrous arthritis** (**Figs. 127** and **128**), in which there is formation of hard fibrous tissue in the joint. A type of this is the *caries sicca* of *Volkmann,* which is common in the shoulder and hip joints, and is characterized by a great tendency toward atrophy of the articular end of the bone, giving rise to dislocations

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and to muscular wasting. In distinction to the above atrophic form, there is another, and more frequent, type of fibrous arthritis, which causes globular swelling of the joint owing to the abundant formation of fibrous tissue. This is especially common in the knee joint and may be mistaken for bone tumor. It is known as *"white swelling"* or *tumor albus,* on account of the white anemic appearance caused by pressure of the fibrous tissue on the skin (**Fig. 128**).

A fourth, and much less common, form of tuberculous joint disease is *purulent arthritis.* This is often due to mixed infection of one of the above-mentioned forms by staphylococci—for example, through a fistula in the skin. However, purulent arthritis sometimes occurs seemingly primarily, especially in children (**Fig. 126**).

In all these forms of tuberculous arthritis the cartilage is much damaged by the fibrinous exudation. In fibrinous hydrops and caries sicca, the corrosive action is generally limited to the cartilage; but in fungoid and purulent arthritis the whole epiphysis may be destroyed, and the infection may spread to the diaphysis.

Tuberculous arthritis generally begins with **pain,** which is often remote from the affected joint; e.g. in disease of the hip joint pain is referred to the inner side of the knee. This is followed by slight rises of temperature and pain in the region of the affected joint. Motion of the joint is avoided, the whole region becomes swollen, and characteristic positions are taken by the different joints. Each assumes the position in which its capsule reaches its greatest capacity (flexion for the knee and elbow, abduction and flexion for the hip, external rotation for the shoulder), and is immobilized in that position at first by *reflex contracture,* later on by *fibrous retraction* of the periarticular muscles. The greater the destruction of the articular tissues, the more abnormal the positions of the affected limb.

There are also some **local signs.** In hydrops there is fluctuation. In fungoid arthritis the whole joint is filled with soft, spongy tissue, causing balloon-like swelling of the joint; this spongy tissue extends to the periarticular tissue and reaches the skin, which becomes reddish-blue, and later on breaks down into tuberculous ulcers and fistulæ (**Fig. 125**). Besides this, multiple abscesses often develop at some distance from the joint (see in **Fig. 125** the scar in the middle of the thigh).

Recovery but seldom takes place with *restitutio ad integrum* of the joint function. Usually, in those cases that recover, there is **ankylosis,** either *fibrous* (**Fig. 125**) or even *bony* (**Fig. 127**). Anky-

losis must be regarded as a favorable outcome, and as a process to be promoted, not hindered. When there is no tendency to fibrosis, the disease keeps on progressing until general exhaustion, miliary tuberculosis, or amyloid degeneration kills the patient.

The prognosis is more favorable in young individuals than in old people.

The diagnosis of either bone or joint tuberculosis is generally easy when there is a fistula discharging characteristic thin tuberculous pus mixed with caseous débris and fragments of sequestrum, or when there is evidence of tuberculosis in the lungs or other organs. The differential characters of chronic osteomyelitis and syphilitic osteitis have already been set forth (see page 152). *Caries sicca* is characterized by the marked atrophy of the joint, the abnormal positions, the muscular wasting and complete loss of function. *White swelling* is recognized by the extensive tumor-like swelling covered by white skin (**Fig. 128**) and does not much resemble bone tumors (see **Fig. 32**).

Tuberculous hydrops may be mistaken for traumatic effusion, gonorrheal or syphilitic arthritis, hydarthrosis. The diagnosis depends on the history of the case and thorough examination of the whole body. In doubtful cases the joint may be tapped, or inoculation to the guinea pig may be resorted to.

Acute forms of fungoid tuberculous arthritis can hardly be distinguished clinically from pyogenic affections of the joint.

In cases where complete healing of the joint has taken place, with bony ankylosis, it is sometimes impossible to distinguish tuberculous cases from joint disease secondary to pyogenic osteomyelitis of the diaphysis. In old people healed tuberculous joints may be mistaken for arthritis deformans or chronic rheumatism. In younger subjects the same condition is often very hard to differentiate clinically from gonorrheal osteo-arthritis (see page 170).

Purulent tuberculous arthritis is generally diagnosed correctly only after incision.

Treatment

Bone tuberculosis calls for operative interference sooner than does joint tuberculosis. The focus must be exposed and scraped, and the cavity packed with iodoform gauze. Abscesses are treated by tapping and injection (see above) or incision and scraping.

In the extremities, immobilization; in tuberculous disease of the spine, operative interference was formerly limited to the treatment of abscesses. But recent operative methods (*Hibbs*) allow of a

quicker cure with much better result and no angular sinking of the spine, as was formerly essential to recovery.

In its early stages tuberculous arthritis may be cured by immobilization by means of extension splints or plaster of Paris casings. *Conservative treatment should always be adopted in the early stages.*

Hydrops may be treated by repeated puncture, injection of iodoform-glycerin emulsion and immobilization of the joint. Recurrence is common, and complete restoration of function seldom occurs. The joints should, therefore, be allowed to ankylose in the most useful position; that is, extension for the knee and flexion for the elbow. When abscesses and fistulæ form, and when an extensive focus of bone disease is shown by the X-rays, conservative treatment must be abandoned, at least in adult subjects. Children may still recover under conservative treatment after abscess formation, and in them resection is more serious, from the standpoint of functional results, as it may markedly impair the growth of the limb.

In fibrous arthritis, caries sicca and white swelling, which are more frequent in adults, *resection* of the joint should be performed as early as possible, to prevent muscular atrophy. In the shoulder joint resection gives good results; but in the knee joint, bony ankylosis in straight position is the only possible result.

In fungous arthritis, especially in young patients, operation may be limited to opening the joint and carefully removing all tuberculous disease (*arthrectomy*). The capsule of the joint must be excised wherever it is diseased, and tuberculous foci in the cartilage and bone removed with the rongeur and curette. In young subjects *typical resection of the joint is to be avoided*, owing to interference with the growth of the limb by extensive removal of the epiphyses.

In adults, on the other hand, the joint may be resected and all diseased parts carefully removed. If, after resection of the epiphysis, the medullary cavity is found to be diseased, it must be scraped out. Abscesses and fistulæ require incision and scraping. In purulent arthritis the joint must be freely opened; in advanced cases resection is necessary. In extensive tuberculous arthritis with tuberculous disease of the neighboring bones and soft parts, amputation may be necessary, especially in old people (**Fig. 130**).

After atypical resection, the joint must be packed with iodoform or sterile gauze. After a typical resection, only a drain is left. In both cases, it is immobilized in a plaster cast. Immobilization, despite the muscular atrophy it provokes, must be kept up until complete recovery.

Joints which have become healed in abnormal positions may be

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Fig. 125. Arthritis tuberculosa fungosa — Ankylosis genus fibrosa — Abscessus frigidus.

forcibly corrected under an anesthetic when the ankylosis is fibrous; but there is danger of rupture of the vessels and consequent gangrene, especially in the knee joint, where the head of the tibia is always in a position of posterior subluxation (**Fig. 127**) and threatens the popliteal vessels in case of sudden straightening, and also when an artery, shortened by retraction, is suddenly elongated (**Fig. 132**). It is better to treat fibrous ankylosis by gradual extension; while bony ankylosis in a bad position may require resection.

In addition to what has already been referred to in the text, a few special points of clinical interest can be gleaned from the examinations of the appended illustrations.

Fig. 125 shows a case of *multiple tuberculosis* of the joints, bones, and soft parts, associated with pulmonary tuberculosis, in a young individual. There is a fungoid arthritis of the ankle joint, with typical ulcers, and thin, greenish discharge. The X-rays showed a primary focus in the astragalus. The foot is in a faulty equinus position. The knee joint was the seat of an old fibrous arthritis and is ankylosed in a right angle position. There was also an old healed hip disease, which rendered the limb useless. The thigh was flexed and X-ray examination showed destruction of the upper margin of the acetabulum and displacement of the head of the femur onto the ilium. In the middle of the flexor surface of the thigh is a healed fistula due to a burrowing abscess. In the middle of the extensor surface of the thigh is a clearly visible swelling due to another burrowing abscess, (common in this situation in tuberculosis of the hip joint or of the vertebræ; in the latter case the abscess travels along the psoas muscle). Fluctuation was present, but the skin was intact.

The ankle joint was treated by free scraping, the faulty position of the knee corrected under anesthesia, and the limb put in a plaster of Paris cast.

The abscess due to the tuberculous coxitis was evacuated by puncture and injected with iodoform-glycerin. Resection of the hip joint was postponed till the general condition of the patient was improved.

Fig. 126 shows a case of *purulent tuberculous arthritis* of the ankle-joint. Owing to the inflammatory symptoms, this type may be mistaken for acute pyogenic arthritis; and, in fact, it is often due to a mixed infection.

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Two incisions were made on the outer and inner sides of the joint, and characteristic thin pus mixed with fibrin was evacuated. The joint was then put up in plaster of Paris. Purulent tuberculous arthritis in children often recovers after early incision; but there is generally some stiffness in the joints, so that these must be put up in the most suitable position for future use.

Fig. 127 shows a case of old-standing *fibrous tuberculous arthritis* of the knee joint with *bony ankylosis,* as shown by the X-rays. Owing to neglect of prolonged fixation of the point in the straight position, *flexion contracture with backward displacement of the tibia* (a very common, almost constant, occurrence in tuberculous arthritis of the knee; already referred to on page 199, and which creates considerable difficulty and danger to the popliteal vessels if simple, forcible straightening is attempted) has taken place. Therefore cuneiform osteotomy was performed.

Fig. 128 shows a *white swelling,* a common form of tuberculous arthritis of the knee in adults. It belongs to the fibrous variety (see page 196). The disease was of several months' duration, and was associated with tuberculosis of the lungs. The patient attributed the affection of the knee to an injury. (In tuberculous arthritis trauma often has a localizing influence in a subject already harboring a tuberculous focus.) The X-rays showed lesions of the bones, as well as of the synovial membrane—the usual combination in tuberculosis of the knee joint. Resection and arthrectomy were performed.

A swelling somewhat similar in shape to that shown in **Fig. 128,** but more distinctly fluctuating, occurs in the rarer cases of tuberculous hydrops, the simplex form of tuberculous joint disease. Effusion into the joint often precedes the arthritis and is recognized by *ballottement* of the patella, which is raised from the femoral condyles by the fluid in the joint. The fluid is generally sero-fibrinous, with numerous free "rice bodies."

Still more common than the fibrous form is fungoid arthritis, which may go on to suppuration and cause much destruction in and around the joint.

In all forms of tuberculous arthritis of the knee, the joint is in a position of valgus and flexion, which makes the internal condyle of the tibia seem hypertrophied and very prominent. There is much atrophy of the muscles of the leg and the growth of the latter is considerably retarded.
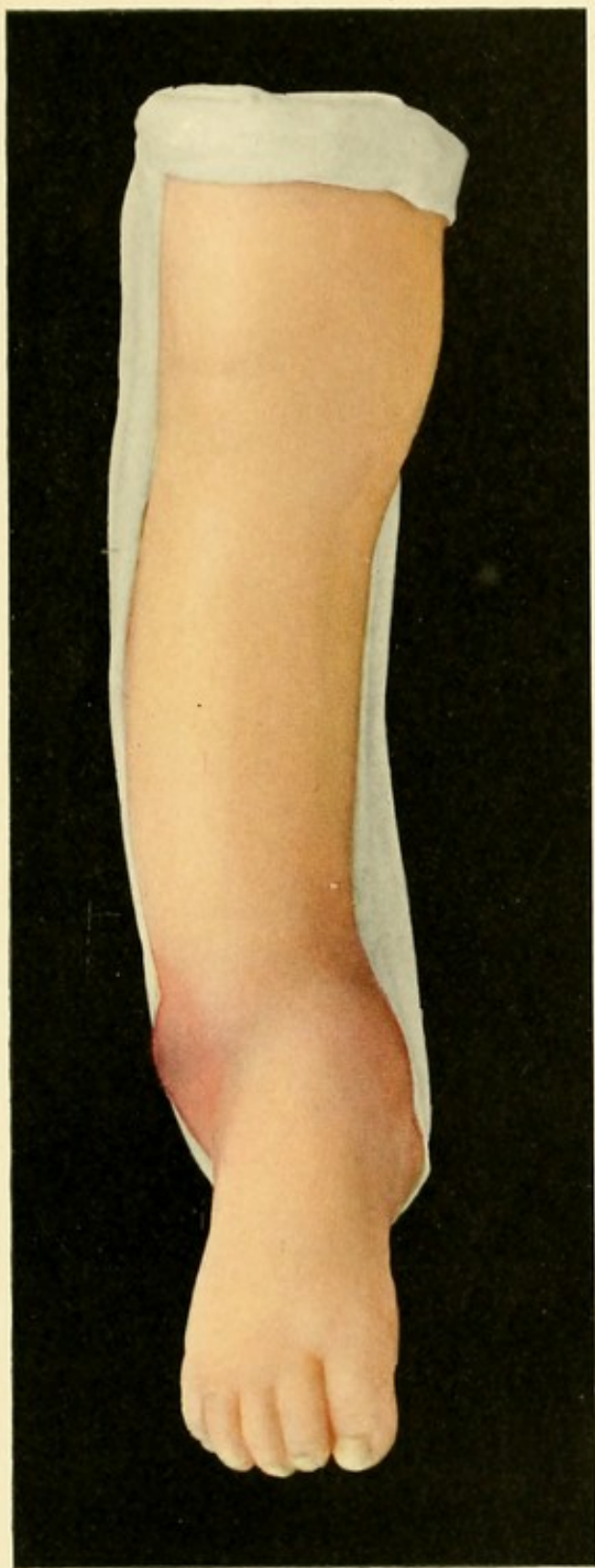
Fig. 126. Arthritis tuberculosa-purulenta.
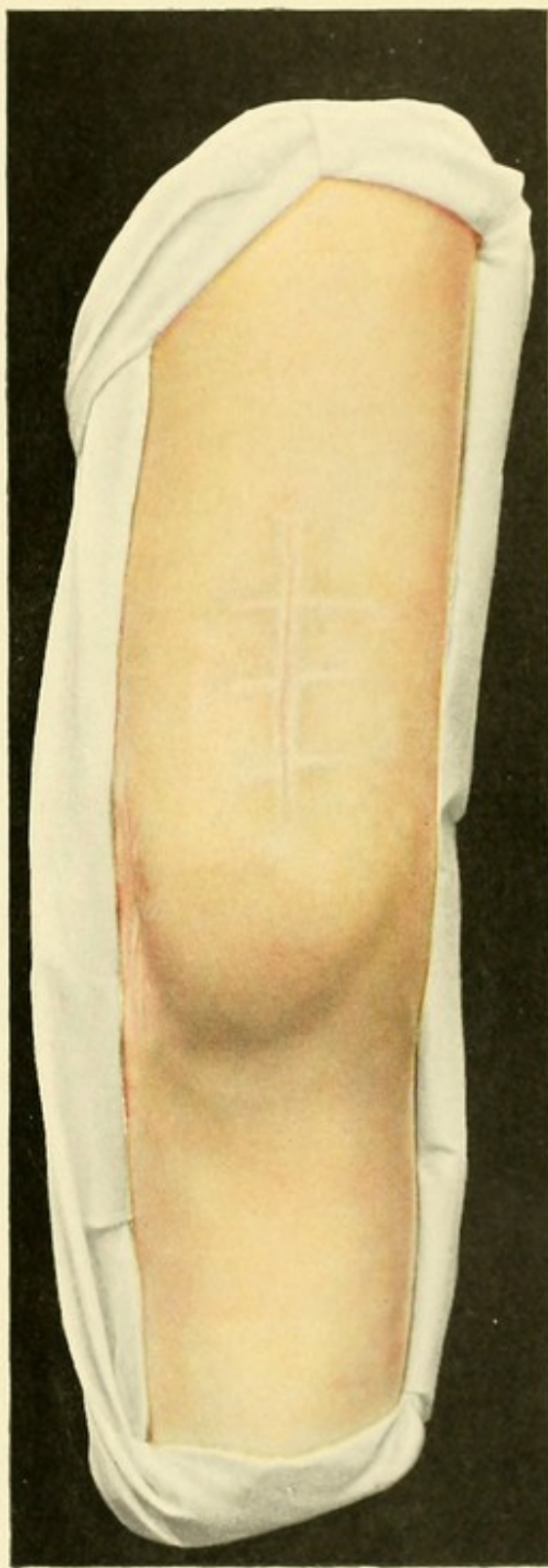
Fig. 127. Arthritis tuberculosa fibrosa —
Ankylosis ossea — Subluxatio.
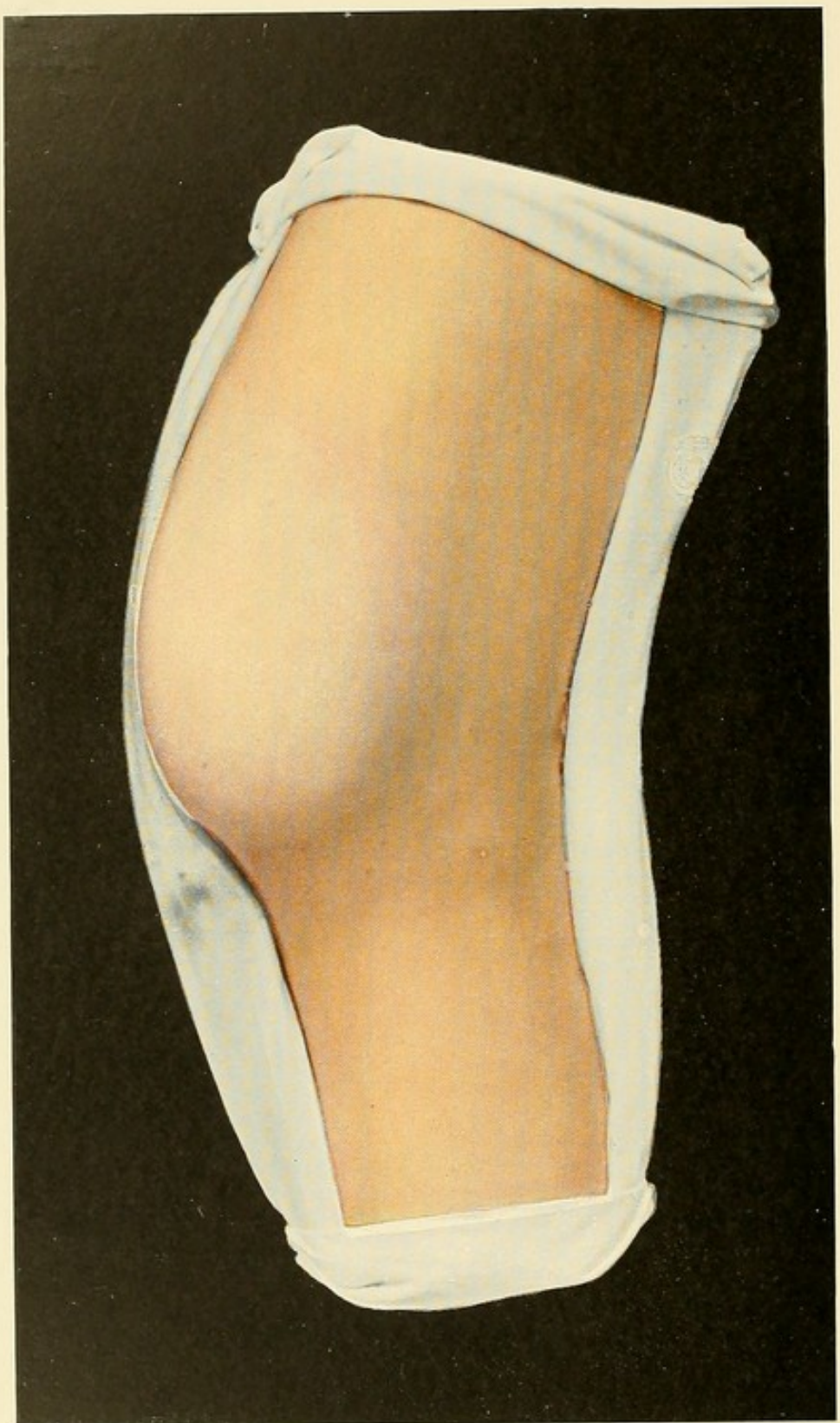
Fig. 128. Arthritis tuberculosa — Tumor albus.

Fig. 131 shows the special type of tuberculosis seen in the phalanges and called *spina ventosa.* ,

Tuberculosis of the phalanges begins in the medulla and extends to the cortex and periosteum. The whole diaphysis may be destroyed by suppuration and caseation, while the periosteum produces a thin shell of new bone. The bone then appears swollen, as if inflated, hence the name.

The disease generally affects several phalanges of several fingers on both hands, and is often found in the children of tuberculous parents. The destructive process is more severe than in any other form of tuberculous osteitis, several phalanges being often completely destroyed. Fistulæ open in the edematous skin and discharge caseous matter. Growth of the fingers is interfered with, so that there often only remains deformed stumps after the disease has healed.

Despite the very characteristic picture, the disease is often overlooked, as it is at first painless; but early diagnosis can be made by the X-rays, which show the changes in the bone.

Syphilitic dactylitis differs in causing less destruction of bone, and in the usual absence of suppuration and necrosis; but the diagnosis often depends on other signs and an antecedent history of syphilis or tuberculosis.

The treatment is early incision, scraping, and bone transplantation, to ward off subsequent deformity.

Fig. 130 shows a case of **tuberculosis of several structures of the back of the hand,** in an old woman suffering from advanced pulmonary tuberculosis. A swelling gradually developed and extended on the back of the hand, limiting finger motion. Two typical tuberculous ulcers discharging thin pus and caseous matter developed on the back of the hand. Passive movement at the wrist joint was very limited and caused crepitation. The X-rays showed **tuberculous disease of the carpal and metacarpal bones.**

At operation, **tuberculosis of the tendon-sheaths** was also found. This condition is more frequent in the upper extremity (as are all tendon-sheath diseases) and may exist independently of a bone focus. It appears either as *tuberculous hygroma*, with serofibrinous fluid and rice bodies causing crepitation on movement, or under the *fungoid form*, with spongy granulations in which the sinews are imbedded.

Tuberculosis of the wrist joint in old people is often so extensive as to require amputation. In the present case, it was treated by **resection, iodoform-glycerin** injections and a plaster cast. Tubercu-

lous tendovaginitis is treated by careful scraping, so as not to injure the tendons.

Fig. 129 shows an extensive case of **tuberculosis of the testicle and epididymis,** in a patient having advanced tuberculosis of the lungs. The skin is thin in several places and ulcerated in one place. The testicle was removed, and on section showed miliary nodules in some parts, caseous foci and abscesses in others.

Tuberculosis of the testicle is *usually consecutive to similar conditions of the prostate and seminal vesicles,* but it is *always* consecutive to another pre-existing tuberculous focus in the body. (In the case of **Fig. 129** there was pulmonary tuberculosis, but no prostatic or vesicular lesions.)

In the early stages hard nodules are felt in the epididymis. Later on the testicle is involved; the nodules become soft and adherent to the skin, which breaks down and forms a typical ulcer (**Fig. 129**). In advanced cases there may be several scrotal ulcers and fistulæ. The spermatic cord is usually thickened and irregular; the seminal vesicles are found nodular by rectal examination.

Diagnosis

The diagnosis is sometimes easy, sometimes very difficult. In its early stage, tuberculosis may be mistaken for **syphilis;** but the latter primarily affects the testicle, not the epididymis; there is a characteristic *loss of the special sensitiveness of the testicle to pressure;* the antecedent history and the *Wassermann* reaction may give confirmatory evidence. **Malignant growths** increase more rapidly; the tuberculin test may here be useful. **Acute epididymitis** may be confused only with the rare *acute forms* of testicular tuberculosis. **Chronic epididymitis** is recognized by the antecedent history and the progress toward regression, not caseation or fistulization.

Treatment

The treatment of tuberculosis of the testicle is *general* and **local.** The general treatment includes the usual hygienic and dietetic prescriptions (very important) and the use of tuberculin, which is beneficial in these cases (*Walker, Belfield, Young*). The best local treatment in recent cases is **epididymectomy** (*Keyes, Jr., Barney*). In older cases, castration may be required, if scraping and injections are ineffectual.
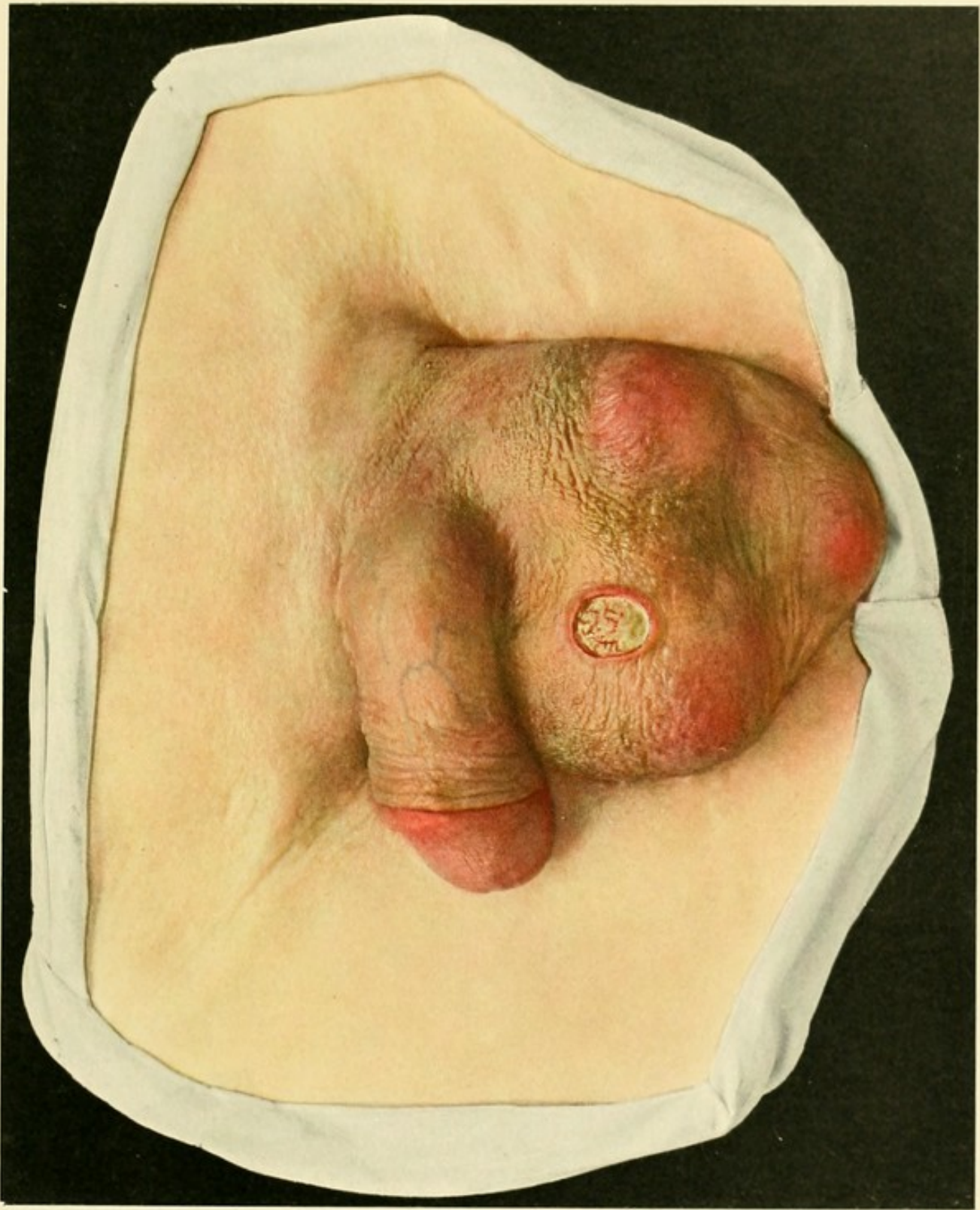
Fig. 129. Tuberculosis testis.

Fig. 130. Tuberculosis manus.
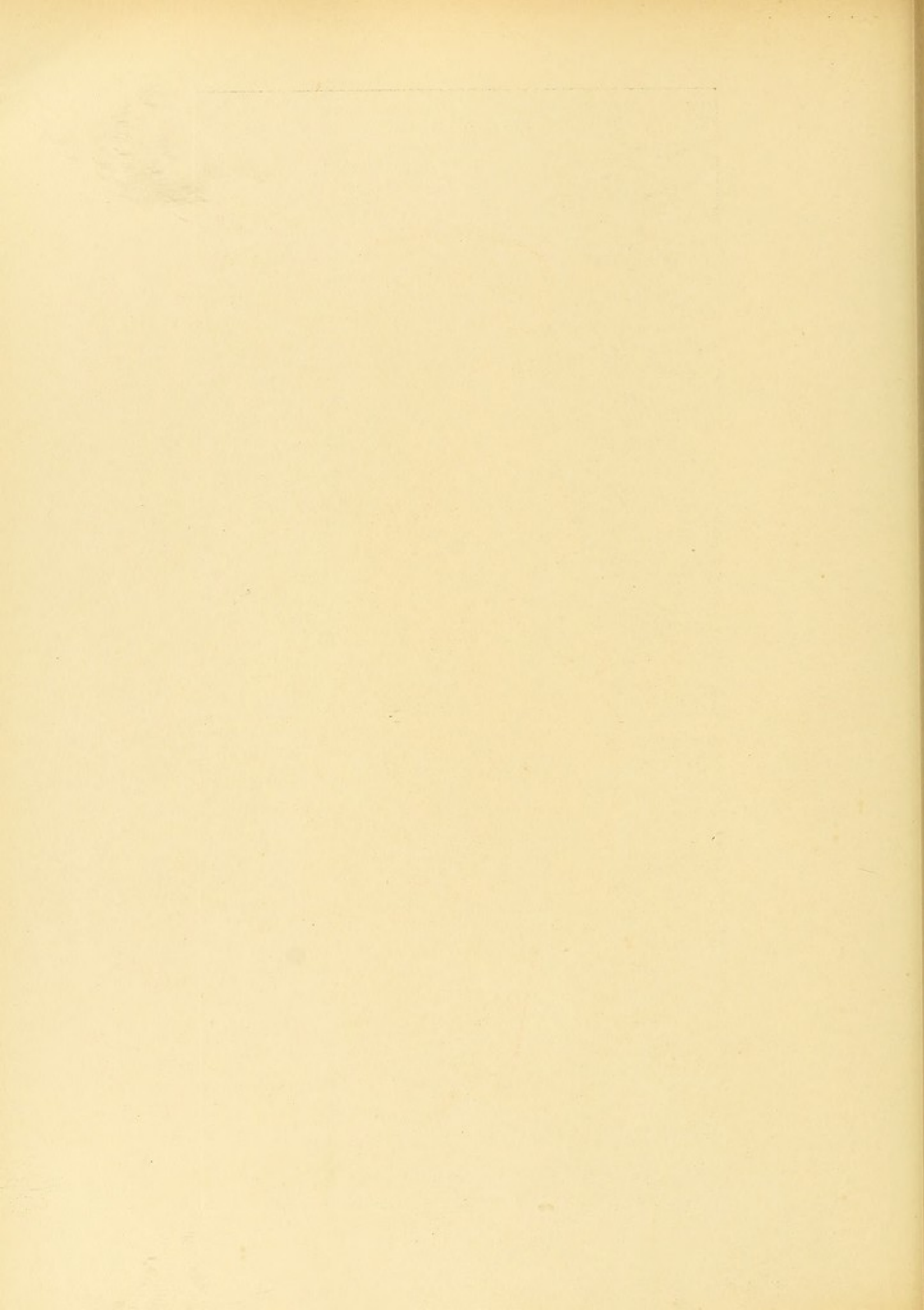
GANGRENES

Figs. 132-141

Burns

Congelation

Fig. 132. Gangraena humida pedis.

Fig. 131. Ostitis tuberculosa — Spina ventosa.

GANGRENES

Figs. 132 to **140**, inclusive, show conditions caused by **gangrene** due to various causes.

Gangrene is the progressive death of the superficial tissues of the body; while the word necrosis is more particularly applied to the death of the deeper structures (fascia, muscle and bone). But this distinction, which dates back to preantiseptic days, when external gangrene was always septic and the deep tissues alone could die aseptically, is now obsolete. The essential process of gangrene and that of necrosis are identical; both are **primarily aseptic,** but external gangrene is *very often modified by infection;* this is why the limits of the nosological group of gangrenes are not always very clear. But **gangrene is not putrefaction,** though both conditions are often associated: putrefaction is an *added process, a complication* of gangrene.

Primarily aseptic gangrene is due to **direct** or **indirect** causes. *Direct* causes are *burns, congelation* or *traumata.* Among the latter, *crushing injuries* and *contusions* are the most dangerous as to gangrene. *Long continued pressure* causing ischemia has already been spoken of under *ischemic retraction* (**Fig. 63**); it is a powerful cause of gangrene of the skin (see page 210) and of the internal mucous membranes.

Indirect causes are **vascular lesions,** *nervous disturbances* and *blood alterations.* Any vascular lesion that results in the shutting off of the blood supply of a region leads to gangrene. *Ligation* of the main artery of a limb was a frequent cause of gangrene in preantiseptic days, when the thrombus was infected and extended much farther than it does in aseptic operations. For this reason the danger of gangrene from this cause is much smaller nowadays, but it still exists, particularly when the arterial walls are diseased (see page 110 about the efficiency of collateral circulation in aneurysm). *Subcutaneous rupture of arteries* (**Figs. 132** and **133**) remains particularly liable to cause gangrene.

Embolism of an artery (e.g. from heart disease) is attended by sudden and extensive gangrene of the territory supplied. **Thrombosis** in diseased arteries is, perhaps, to-day, the most important etiological factor. *Senile gangrene* is due to **arteriosclerosis:** the

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loosened intima in the small terminal vessels (and also in some of the larger) provokes thrombosis, and as the collateral circulation is insured only by altered and sclerosed channels, gangrene follows. Arteriosclerosis, the deficient power of resistance of the anatomical structures, and maybe also blood alterations, explain *diabetic* gangrene (**Fig. 140**). *Carbolic gangrene* (**Fig. 135**) is probably due to thrombosis of small vessels.

In younger people, *obliterating endarteritis* is chiefly due to syphilis: it gives rise to a slowly progressing type of gangrene (angiosclerotic gangrene).

Vaso-motor constriction of the blood-vessels is a cause of gangrene in certain nervous diseases (e.g. *Raynaud's* disease, **Fig. 139**). Gangrene consecutive to subcutaneous injection of adrenalin is probably due to too prolonged ischemia by vaso-constriction. The same explanation was formerly unreservedly admitted for gangrene following the administration of ergotin. Now, it seems that blood alterations may play a part in this case.

About gangrene due to blood alterations little definite is known.

When the anatomical process consists in desiccation of the tissues it is called **dry gangrene** (**Fig. 134**); when it ends in liquefaction from the invasion of putrefactive bacteria, it is called **moist gangrene** (**Figs. 132** and **134**). Dry gangrene may change to moist, and both processes may occur simultaneously in different parts of the same limb, when one part becomes septic and the other does not.

Gangrene due to arteriosclerosis is preceded by *pain* in the affected part. That caused by syphilitic endarteritis is often heralded in by *severe intermittent pain*, which, in the lower limbs, causes the patient to limp (*intermittent claudication*). The gangrene of *Raynaud's* disease is generally preceded by paresthesia and disturbances of the thermic sensibility.

The *extent* of the gangrene varies according to the cause; it may be circumscribed (after local applications, such as carbolic acid), or progressive (after embolism). In both forms the dead tissue becomes separated from the living by a zone of demarcation. The zone of demarcation forms a groove filled with granulation tissue (**Fig. 132**). It may be circular (**Fig. 135**) or irregular (**Figs. 133** and **134**).

In the early stage of dry gangrene the condition resembles that of ischemic muscular contracture (**Fig. 63**), especially when the condition is due to plugging of the blood-vessels. The skin, at first cold and pale, next dotted with bluish patches arrayed irregularly, finally

becomes dry, shrunken and parchment-like. In the extremities the peripheral parts are flexed and immovable. The skin becomes gradually yellowish-brown and finally *black* (**Fig. 133**). All the subjacent structures may undergo dry atrophy. The dead tissue is gradually separated by the zone of demarcation, and the whole of an extremity may thus undergo spontaneous separation.

While in dry gangrene there is *diminution* in volume and charring of the affected part, in moist gangrene there is *increase in volume,* due to preceding edema. In moist gangrene there is always more or less liquefaction and putrefaction, due to bacteria. The skin is cold and moist, and the epidermis becomes raised in bullæ containing blood-stained fluid. After rupture of the bullæ the skin is reddish-brown (**Fig. 109**). Finally the tissues become disintegrated and the odor emitted is horribly foul; lymphangitis, lymphadenitis and general infection then follow.

A deep groove of demarcation, separating the dead from the living tissue, also forms in moist gangrene, and spontaneous elimination may occur if the patient does not succumb to general infection. In less extensive cases of moist gangrene we can wait for the establishment of this line of demarcation; but the gangrenous part must be removed if there are chills and high temperature, else the patient would die of acute toxemia caused by the resorption of poisonous products from the gangrenous part.

Diagnosis and prognosis

The appearance of gangrene, when fully developed, is so characteristic that it can hardly be mistaken for any other condition. The two forms of gangrene are also sharply defined from each other. Dry gangrene might be mistaken for burns of the third or fourth degrees, if signs of the first and second degree of burn were not always present in the neighborhood, and if the anamnesis was not always so clear.

Moist gangrene might be mistaken for putrefactive phlegmon, especially progressive gaseous phlegmon (**Fig. 109**), if, in the latter, the signs of general infection were not present at a very early stage. The history and a thorough examination will not only establish the diagnosis, but in most cases will decide the cause of the gangrene.

The prognosis naturally depends on the cause and on the extent of the gangrene. Angiosclerotic (endarteritic) gangrene spreads very slowly; it may remain stationary; or parts which appeared to be affected may recover. Plugging of a large vessel causes extensive

gangrene of the part supplied by the vessel, and the prognosis is not favorable. Diabetic gangrene and senile gangrene are characterized by their progressive course. Gangrene is more extensive when there is much edema.

Treatment

In threatening arteriosclerotic gangrene, the limb should be raised and wrapped in wool: hot-air treatment is useful for the pains: alcohol should be avoided. If a syphilitic origin is suspected, mercury and iodides should be freely given; salvarsan is contraindicated in marked cardiovascular degeneration.

The gangrenous part should be covered with aseptic dressings. If surgical interference is not urgent, it is better to wait for the zone of demarcation to appear and then to perform amputation in the most conservative way possible. But, sometimes, in moist gangrene of an extremity, early removal is indicated in order to forestall general sepsis. In diabetic gangrene (see page 213) high amputation is often necessary. The same was true, not long ago, of most cases of embolic gangrene. Now *arteriotomy* has been performed for the condition (*Stewart*). While the clot almost invariably reforms in the point operated on, the operation may give the collateral circulation a little more time to develop and lessen the area of mortification.

In amputation for arteriosclerotic gangrene the tourniquet is not to be used, as it may cause thrombosis at the point of application and further gangrene of the stump. If the vessels in the stump only bleed slightly, this shows that they also are already diseased and that the gangrene will probably extend further. The veins in the amputation stump bleed freely, owing to the absence of the sustaining arterial *vis a tergo*. After amputation any pressure of the dressings is to be avoided.

Arterio-venous anastomosis of the femoral artery and vein has been performed about forty-five times in case of arteriosclerotic gangrene (*Hubbard* and others) in the hope of reversing the circulation in the limb. Unfortunately, this reversal is not obtained, and, barring exceptional cases (*Wieting, Ballance, Davies*), the operation has not yielded any permanent benefit. However, it is but fair to add that it was employed only as a last resort in otherwise hopeless cases, generally by men not sufficiently trained in vascular surgery, and that it holds better promises in cases where gangrene is simply impending, instead of actually existing. (See about *Raynaud's* disease, page 216.)

Fig. 133. Gangraena sicca brachii — Mumificatio.

Fig. 132 shows a case of *moist gangrene of the foot* due to a special and interesting origin, as it developed after forcible correction of flexion contracture due to tuberculosis of the hip-joint, a contingency already alluded to on page 199.

Soon after this operation the toes became cold, blue and flexed, and finally black. As the gangrene was limited to the anterior portion of the foot, it is probable that the injury implicated the intima only and was not a complete rupture of the femoral artery, and that gangrene was due to thrombosis of the vessel. In the figure, necrotic bone is seen to protrude from fistulæ (first and fifth toes). In the sole of the foot is seen a wide, granulation covered zone of demarcation separating the gangrenous part from the healthy tissues behind.

Fig. 133 shows a typical case of *dry gangrene* or *mummification of the arm,* affecting all the tissues. The fingers are contracted and blackish-brown in color. The skin is hard. In the forearm commencing gangrene is seen in the yellow, leathery skin. The line of demarcation is seen as a red zone of granulation tissue, separating the dead from the healthy parts. After the line of demarcation had extended all around the limb, amputation through the arm was performed.

The cause of gangrene in this case was of a nature similar to that of the case of **Fig. 132,** namely, an arterial tear during an operation for an articular condition accompanied by periarticular sclerosis and retraction. It was due to rupture of the axillary artery during an operation for reduction of an old dislocation of the shoulder. In such cases bloodless reduction is generally impossible and may cause rupture of the artery. But this disadvantage also applies to reposition by open operation, for the displaced vessels are liable to become damaged by pressure of the dislocated head of the humerus and are easily torn during reduction of the dislocation. This accident may be avoided by resection of the head of the humerus, after carefully dissecting free the artery, which is generally united to it.

Fig. 134 shows a case of *moist gangrene of the skin with necrosis of the abdominal fascia.* The necrotic part of the skin is separated by a zone of demarcation from the healthy, somewhat reddened and inflamed skin around it. It is still firmly attached to the subjacent structures. In some places the skin has separated, expos-

ing the abdominal fascia, the yellowish color of which shows that it already has undergone necrosis. The borders of the ulcer were undermined, and it discharged fetid pus.

In this case the gangrene was caused by a subcutaneous injection of salt solution, given to a patient in a state of collapse. Gangrene of the skin may occur after injection of large quantities of salt solution when the injection is made *intracutaneously* instead of *subcutaneously,* or when the fluid is too hot.

The ulcer became clean under dressings of peroxide lotion; the gangrenous skin and necrotic fascia were shed spontaneously; the edges of the fascia and the skin were sutured separately, and primary union took place.

The skin, being the most superficial part of the body, is exceedingly liable to injuries which may cause gangrene of external origin. *Long-continued pressure,* especially in places situated over the bones, may cause gangrene of the skin. In this way gangrene may be caused by the *pressure of tight bandages or splints* (see ischemic paralysis, **Fig. 63** and page 83); also by a displaced piece of bone in fractures; by pressure on the outer side of the foot in pes varus; by tight sutures, e.g. after amputation of the breast which leaves a wide space to be closed (**Fig. 55** and page 75).

Uncleanliness, loss of consciousness, nervous diseases (trophoneuroses, syringomyelia, hemiplegia, paraplegia, tabes), cachexia, diabetes, typhoid fever, osteomyelitis, phlegmonous inflammation, general infection and comatose conditions, all predispose to gangrene, which, in emaciated persons, may become very extensive. Gangrene of the skin caused by the pressure of edema and gaseous formation in the tissues has already been mentioned (**Figs. 91** and **109**). After operations, gangrene of the skin (bed-sores) may occur over the heels, buttocks, spinous processes, shoulder blades and back of the head, if care is not taken to change the position of the patient and apply soft, smooth, protective coverings.

Gangrene of the skin begins with *pain* and *redness;* then slight swelling and blue discoloration; finally, raising of the epidermis in bullæ. The epidermis then separates, leaving exposed the corium, which is at first greenish-yellow, afterward blackish-brown and leathery. At the edge of the gangrenous part the skin becomes inflamed, and by the formation of pus and granulation tissue a gutter-shaped, often circular zone of demarcation is gradually formed. The more severe the injury, the deeper is the gangrene, so that subcutaneous

Tab. CVI.

Fig. 134. Gangraena humida cutis — Necrosis fasciae — Ulcus decubitale.

Fig. 135. Gangraena carbolica.

tissue, fascia (**Fig. 134**), muscles and bone may become necrosed and cast off.

After separation of the gangrenous part there is left a *decubital ulcer* covered with slimy, greenish-yellow connective tissue shreds and fetid pus. A neglected decubital ulcer may give rise to extensive putrid inflammation or gaseous phlegmon, as the pus always contains putrefactive bacteria, especially when situated over the sacrum, as it then is always infected from the feces. Erysipelas may also occur.

Gangrene of the skin may, in many cases, be prevented, or at any rate limited, by prophylactic treatment. Decubital ulcers (bed-sores) may be prevented by applications of spirit of camphor and dusting powder to the skin of the parts exposed to pressure, by air cushions and frequently changing the patient's position. If the skin is discolored an ointment dressing should be applied, and this should be changed if the patient complains of pain. As pain also subsides in a few days under a dressing, removal of the latter is often neglected, and when it is done there may be gangrene down to the bone. In emaciated patients, therefore, the bony prominences should be well padded, the skin disinfected before applying the dressing, and the latter changed frequently.

If gangrene has developed, the skin must be protected against infection by a dressing. Separation of the gangrenous part may be hastened by moist dressings with 2% boric acid or 3% peroxide solution, applied several times daily. Forcible removal of the gangrenous parts while they still are firmly adherent is not advisable; they should be trimmed off with scissors when almost completely loose. The ulcer may be treated with moist dressings or ointments, and with caustics when granulations have sprung up (see **Fig. 55** for the treatment of granulating wounds with balsam of Peru, red salve and skin-grafting, page 75). After extensive gangrene of the skin, the space may be closed by undermining the surrounding skin and suturing; or, if this is impossible, by a plastic operation by means of pedunculated flaps.

Fig. 135 shows a case of **carbolic gangrene** caused by dressings applied to a wound in the finger. The end of the latter became white and the epidermis was destroyed as far as the carbolic acid dressing extended, exposing the corium. The patient suffered severe pain and had no feeling in the tip of the finger, which gradually became black and shrunken. As shown in the figure, there was a total slough-

ing of the terminal phalanx, while the greenish-yellow color at the junction of the terminal with the middle phalanx indicates commencing gangrene. In the middle of the second phalanx there is a wide zone of granulation tissue corresponding to the line of demarcation. Severe pain in the finger was due to thrombosis of the terminal arteries caused by carbolic acid. Later on there was anesthesia in the finger due to alterations of the sensory nerves.

Moist dressings were applied, and in a few weeks a groove of demarcation extended down to the bone. In the peripheral part gangrene spread to the fascia, muscles, tendons and bone. Healing took place after exarticulation at the interphalangeal joint.

It must be borne in mind that even 1% carbolic solution, after a few hours' application only, may cause sloughing of the skin and deep necrosis by thrombosis of the vessels. Certain individuals appear to be predisposed to gangrene after fomentations with carbolic acid (and sometimes lysol), especially when gutta percha tissue is placed over the dressing, preventing evaporation. After a short application the skin may recover.

Contused wounds must *never* be disinfected with carbolic dressings.

Fig. 140 shows incipient *gangrene of the right foot* in a man of 56, suffering from *diabetes* and *arteriosclerosis* for some years. The toes are bluish-red in some parts, grayish-black in others, while the dorsum of the foot is red. The skin was pale and cold. The discoloration appeared in the course of a few hours, and in a few days extended to the ankle joint. Moist gangrene spread rapidly from the toes, and lymphangitis extended up the leg.

The X-rays showed numerous calcareous deposits in the interior and posterior tibial arteries. Amputation was performed above the knee joint, after the sugar had been reduced from 5 to 2% by three days' treatment of the diabetes. After operation the sugar diminished still further, and the temperature fell—two favorable signs. Secondary suture of the stump was performed on the fifth day and the wound healed in four weeks. After general treatment the sugar disappeared from the urine.

The figure also shows other changes. On the inner side of the foot over the metatarsophalangeal joint is a large corn, and another on the fifth toe. The nail of the great toe is affected with *onychogryposis,* a common condition in old people who neglect their feet.

We already several times had occasion to lay stress on the importance of diabetes as regards septic conditions.

Gangrene is frequent in diabetics. Dry gangrene may develop suddenly in the lower extremities when there is concomitant arteriosclerosis. The whole leg may be affected owing to thrombosis of the popliteal artery. The first symptoms are those of arteriosclerosis: high blood pressure, hardness of arteries (radial, temporal), numbness and tingling sensations in the toes: later the appearance is that shown in **Fig. 140.**

In this stage there are often severe neuralgic pains, while the general condition of the patient is impaired by increase of sugar in the urine, sleeplessness, headache and exhaustion. In old diabetics with dry gangrene of the toes demarcation may take several months to develop. Dry gangrene may always change to moist, the latter progressing more rapidly.

Diagnosis and Prognosis

Diagnosis of diabetic gangrene is made by the search for sugar in the urine, which must be undertaken in *all* cases of pyogenic infection. Diagnosis of arteriosclerosis is made by the blood pressure test and palpation of the hardened arteries; sometimes by the X-rays in case of marked calcification.

In diabetic gangrene, the prognosis is bad; *as to life,* it depends chiefly on the amount of sugar and the response to antidiabetic treatment. *As to the limitation of the damage* caused by gangrene, it also depends on the extent of the arteriosclerosis and the sufficiency of the collateral circulation.

Treatment

Prophylaxis consists in early diagnosis, antidiabetic treatment, and measures that can check the progress of arteriosclerosis.

Once gangrene has set in, the general principles of treatment of gangrene (see page 208) are applicable.

Active (hot air or bath) *hyperemia* (*Stetten*) so improves circulation that conservative treatment becomes sufficient in most cases and yields far better results than early or high amputation. Passive hyperemia is, of course, absolutely contraindicated.

In dry gangrene it is best to wait for demarcation, unless extensive arteriosclerosis is present. If, however, the popliteal artery is pulseless, amputation of the leg is the only remedy. If there is no arteriosclerosis, the gangrene may slowly extend for months. When

demarcation is complete amputation may be performed directly above the line of separation. Before demarcation the parts should be treated with dry aseptic dressings (moist dressings cause putrefaction), and be suspended. In slowly extending moist gangrene demarcation may be waited for if the temperature does not remain high. In rapidly extending moist gangrene with high temperature early amputation is indicated some distance above the gangrene. In gangrene of the lower extremity with arteriosclerosis it is better to amputate through the thigh; for the flaps after amputation through the leg are badly nourished even in healthy individuals, and in diabetics they are liable to slough off. Amputation through the thigh is best performed above the condyles or through the epiphyseal line. Epiphyseal stumps have considerable supporting power. As a rule, amputation may be conservative in slowly progressing cases which are not complicated by phlegmonous inflammation, arteriosclerosis or high temperature. On the other hand, rapidly extending gangrene complicated by arteriosclerosis and phlegmon always requires high amputation. (About arterio-venous anastomosis, see page 208).

Diabetics with gangrene of the lower limbs are always poor surgical risks. Not over 50% recover after operation, and diabetic coma is a frequent complication after anesthesia. Lumbar anesthesia is useful when it works well: infiltration anesthesia is contraindicated, as it causes inflammation of the weakened tissues. Gas-oxygen anesthesia is probably the best.

When the vessels are sclerosed, hemostasis during the operation should be secured by digital pressure only, as the application of the tourniquet may cause thrombosis. The wound should be dressed with sterile gauze; iodoform and in general all antiseptics are contraindicated on account of the danger of toxic phenomena. Primary suture of the flaps should not be attempted, and these, therefore, should be made larger than usual. Secondary suture may be performed after a few days if the progress of the case is satisfactory. Ligatures must not be applied too tightly to vessels affected with arteriosclerosis, lest the coats of the vessel give way and secondary hemorrhage should result. The operation must be performed under the strictest aseptic precautions, as diabetic tissues are easily infected, and osteomyelitis may occur in the bone stump or phlegmonous inflammation in the soft parts. Ulceration of the stump is common after healing. Antidiabetic treatment must be vigorously pushed during all the time. Incipient diabetic coma may be cured by intravenous infusions of 5% sodium bicarbonate solution.

Fig. 139 shows a *perforating ulcer* of the foot over the head of the third metatarsal bone; the epidermis is fissured and thickened around the small ulcer, which is covered with granulations and from which a necrosed piece of fascia is seen to protrude.

The peripheral part of the foot showed the diffuse bluish-red discoloration, characteristic of **Raynaud's disease,** which existed also symmetrically on the other foot.

Perforating ulcer of the foot is of trophoneurotic origin and due to disease of the nervous system. It occurs in tabes, syringomyelia, certain forms of spina bifida (**Figs. 143** and **144**), and also in diseases where sensation is lost in the lower extremities. Owing to the loss of sensation, the patient does not notice the injury to the sole of the foot caused by pressure, and in this way a trophoneurotic ulcer develops, characterized by hard borders due to the horny epidermis which is normally present in the sole of the foot. These ulcers may also develop on the outer border of the foot in cases of paralytic pes varus (**Fig. 143**). Some authors attribute the condition to disease of the blood-vessels (*arteriosclerosis, endarteritis obliterans*) as well as to trophoneurotic disorder, and in many cases both conditions are probably present. That the blood-vessels play a part in the pathogeny of perforating ulcer is supported by the fact that this condition is often met with in syphilitics and alcoholics with vascular degeneration.

The ulcer begins as a hard, horny thickening of the epidermis over the heads of the third or fifth metacarpal bones, somewhat resembling a corn, but more extensive. The epidermis becomes fissured and finally ulcerated in the centre. The ulcer is characterized by its tendency to extend deeply, and by its persistency in spite of all kinds of treatment. The disease is essentially chronic and leads to destruction of muscles, tendon-sheaths, bones and joints, by continuous crateriform extension of the ulcer into the deeper tissues. The epidermis always remains thickened at the border of the ulcer, and is sometimes undermined. The visible surface of the ulcer is small and is covered with flabby granulation tissue. Necrotic shreds often protrude, indicating extensive destruction of the fascia and tendons. There is often loss of sensation in the skin for some distance around the ulcer. As a rule there is little pain, but sometimes paresthesia. The general health may suffer from prolonged suppuration, or the condition may be aggravated by acute progressive phlegmonous inflammation.

Raynaud's **disease**—which is sometimes called gangrene, but better **local asphyxia,** as it only consists in the first stage of gangrene—is usually **symmetrical,** and affects the feet more often than the hands. After a short premonitory stage during which the digits become cold and white (vaso-motor constriction), the tips of the fingers or toes become dark-purple and the proximal parts red (vaso-motor paralysis). The disease is due to vaso-motor disturbance depending on disease of the peripheral or central nervous systems. The symptoms consist in paresthesias and disturbance in the temperature sense, and pain on changes of temperature.

Diagnosis and treatment

A beginning perforating ulcer may be mistaken for a corn complicated by a mucous bursa and central fistula (**Figs. 64** and **99**); but the latter does not extend so deeply.

Syphilitic and tuberculous ulcers are recognized by their usual characters, already mentioned (pages 180 and 189); besides they are rare in the sole of the foot; the location of perforating ulcer is characteristic.

Raynaud's disease may be confounded with the early stages of other forms of gangrene (**Figs. 132, 133** and **140**), or frostbite (**Fig. 137**); but the changes in *Raynaud's* disease are *diffuse* and *symmetrical.*

Even in the early stage of perforating ulcer, removal of the callosity and necrosed tissue gives little result. In the later stages no treatment is very efficient. The wound must be protected from infection. *Partial amputation of the foot* (tarso-metatarsal exarticulation in the case of **Fig. 139**) is sometimes the only resource, but the ulcer may recur on the stump. The internal administration of iodides is useful, and so, perhaps, would be salvarsan treatment in those cases in which a syphilitic or parasyphilitic etiology is likely. Several successes have been claimed (especially by French surgeons) after *elongation* of the plantar nerves.

Raynaud's disease is perhaps sometimes of syphilitic origin and would be benefited by antisyphilitic treatment. Avoidance of exposure to cold is necessary: hot-air hyperemia and massage are useful.

Arterio-venous anastomosis has been resorted to several times for *Raynaud's* disease. *Bernheim* recently published a very remarkable case in which he successively performed the operation on all four limbs, with perfect success each time. As, in most cases, *Raynaud's* disease is, as set forth above, a menace of gangrene rather than

Fig. 136. Combustio erythematosa — bullosa — escharotica.

actual gangrene, good results might be expected from this operation (see page 208).

Fig. 136 shows all four degrees of **burns** caused by red-hot metal, which remained longer in contact with some parts than others.

The **first degree** is characterized by active hyperemia, *reddening* and swelling of the skin; the **second,** by the raising of the epidermis in *blisters* by exudation of lymph between said epidermis and the corium; the blisters contain yellow, clear fluid and develop within 24 hours. The **third degree** is accompanied by destruction of the epidermis and corium; gangrene of the skin results from dehydration of the tissues, coagulation of albumin, and acute thrombosis of the blood-vessels. A black *eschar* forms, which becomes slowly separated from the subjacent tissues, after which healing takes place by granulation. The **fourth degree,** sometimes subdivided into three groups, so that the total number of degrees is six, contains all cases where the lesions are more than simple destruction of the skin, up to the complete charring of a limb or the body.

Pain is generally slight and temporary in first degree burns; it is severe in those of the second degree, especially if the epidermic cover of blisters has been ruptured and the cutaneous nerve endings of the skin are bared. In the third and fourth degrees there is little or no pain, because the nerve endings, which are irritated in first and second degree burns, are here actually destroyed. But around third and fourth degree burns there is always a zone of second and first degree lesions, which cause intense pain. In case of extensive second degree burns, the suffering may be so agonizing that the patients truly *die of nervous exhaustion caused by pain.*

In extensive burns, there are severe general symptoms due to resorption of toxins from the charred parts. In some cases the temperature is subnormal, the skin becomes pale and cold, the pulse is small and rapid and the patient dies in a few days in a state of collapse. In other cases there is high temperature, delirium, diarrhea and coma. At autopsy, intestinal ulcerations are found, particularly in the duodenum, also ecchymoses and thromboses in all the organs, parenchymatous nephritis, etc.

If the patient survives, burns of the first and second degree heal simply, without leaving any scar, *provided no infection has supervened.* In the third degree, healing takes place by granulation after shedding of the eschar; the process of cicatrization is slow, and the

scars are hypertrophic, unsightly, adherent to the deeper tissues, and have a marked tendency to become retracted. This latter character often proves very troublesome, as a joint may be immobilized in faulty position, and a limb made useless, not to speak of the resulting deformity.

But burns have an unfortunate tendency to **become septic,** and when infection occurs, it greatly complicates matters. Suppuration in extensive burns may cause progressive exhaustion, and death from amyloid degeneration; at best, it markedly retards recovery and makes the scar still uglier.

This gives us the general indications for treatment: **allay the pain,** and **prevent infection.** As pain is due to a great extent to exposure of nerve endings to air contact, therapeutic deductions are obvious: *disinfect the burned part,* under general anesthesia, if necessary, and *apply a sterile dressing* (no antiseptics, for fear of possible absorption). The same effect is obtained by keeping the patient, or at least the burned limb, in a *sterile bath* evenly maintained at body temperature: this is the handiest way of treating extensive burns, and the one that most relieves the patient. In second degree burns care must be taken not to break the epidermic cover of the blister; the latter, if large, is evacuated by a puncture made in a point of the periphery with a fine sterilized needle. In burns of the first degree, where the skin is unbroken, we need not be so particular about the means employed: flour, talcum powder, bismuth subnitrate, oil, are all good. A mixture of equal parts of lime water and olive oil (Carron oil) has a somewhat surfeited repute in burns.

When repair has progressed to the point when the dead has been demarcated from the quick, various operations may be needed to regularize the parts. Probably no condition requires **skin grafting** as frequently and extensively as burns. Charred and necrosed bones (second and fifth fingers in **Fig. 136**) must be exarticulated; likewise must be parts so mutilated that they are no longer capable of function.

Of course, during all the period of repair, the general strength of the body must be promoted by diet and tonics. In the beginning, caffeine, digitalis, camphor oil may be called to sustain a faltering heart, and saline infusions to restore tone to a weakened vascular system. Morphia must be used only when absolutely necessary, as it inhibits the working of the kidney, which is essential to recovery and, on the contrary, must be stimulated by diuretics.

(See again the treatment of **granulating wounds, Fig. 55** and

Fig. 137. Congelatio erythematosa — bullosa.
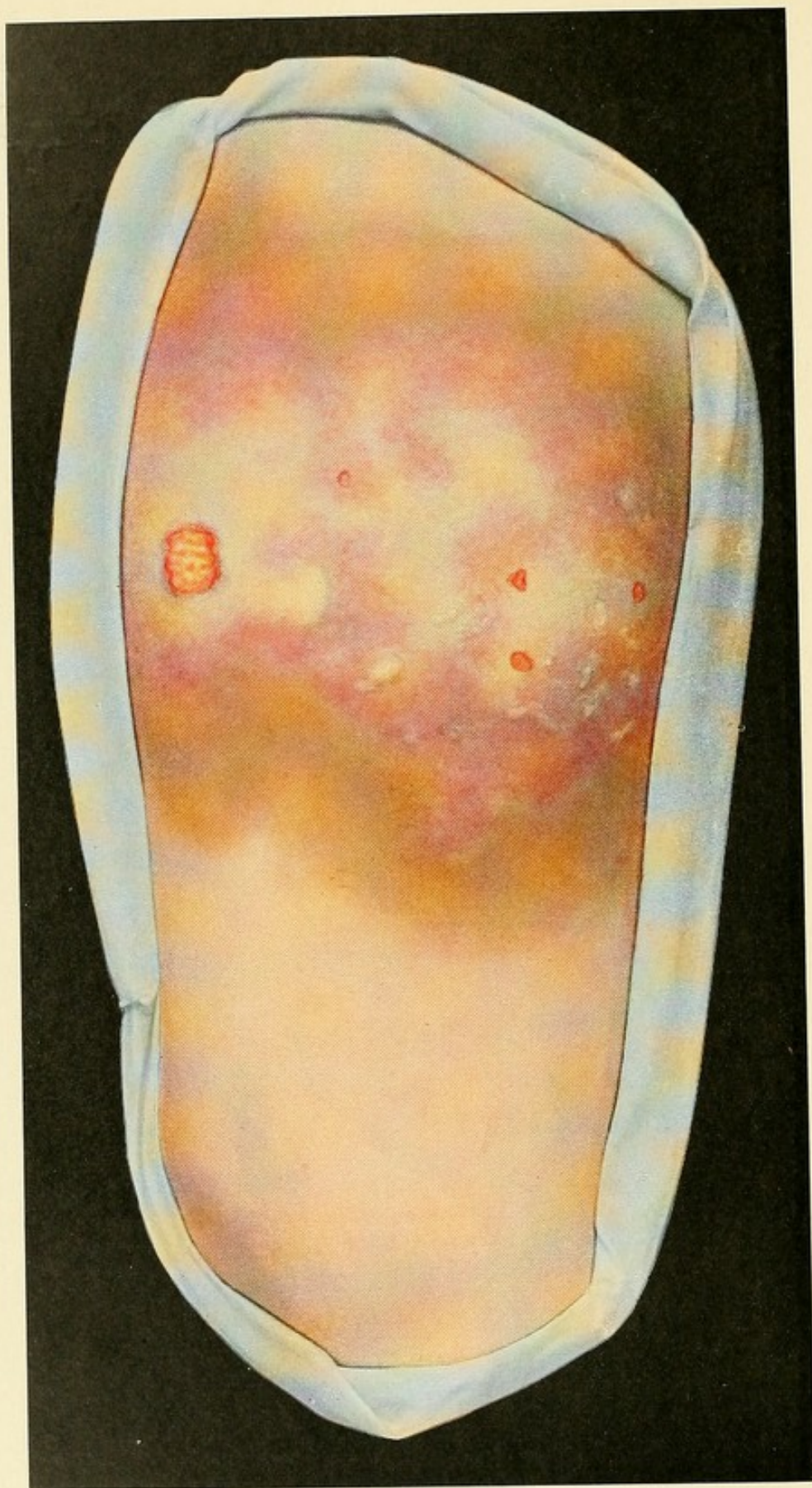
Fig. 138.   Combustio (X-Rays).

page 75; see also **Figs. 58** and **59, *keloids,*** as scars of burns have a marked tendency to keloidal hypertrophy.)

Chemical burns often require chemical neutralization: acids (vinegar, lemon juice) for alkalies; alkalies (baking soda, soap) for acids; but this neutralization is of no value unless applied a very short time after the caustic substance.

Heatstroke, sunstroke, lightning stroke and electrocution are other effects of heat, but cannot be considered here, as their treatment is purely non-surgical, and they raise too complicated physiological questions.

Fig. 138 shows an ***X-ray burn*** which followed a long exposure made for a swelling of the thigh. The skin became red, then white, and finally ulcerated in several places. The brown coloration indicates healing of the less affected parts. The ulcers healed after the application of simple dusting powder.

X-ray dermatitis was frequent and severe in the early days of radiology, when the potentiality for harm of the rays had not been recognized and workers did not protect themselves. Several enthusiastic pioneers, after suffering for years from a chronic dermatitis, with dry, cracked and fissured skin, brittleness and falling of the nails, sometimes necrosis, have seen *X-ray carcinoma* develop in their hands, and despite multiple and mutilating operations, have lost their upper limbs and finally succumbed after years of untold suffering.

To-day we know how to limit the action of the X-rays to the part we want to treat: the use of lead glass in X-ray tubes is a sufficient protection for the workers, and we know how to administer X-rays in graduated doses. There are several means of measuring the quantity given, and to each disease seems to correspond a determined dose, sufficient and necessary for a cure. This is the principle of the ***single dose method*** (*Mackee*), which in some conditions (favus, keloids and particularly rodent ulcer, see page 3) seems to give better results than the older, empirical, fractional method.

There is no individual susceptibility to the X-ray; that is, a given dose, measured in *Holzknecht* units, will produce the same therapeutic effect, or the same burn, in different individuals, if applied to the same region. The only variations are those due to age. A slight reaction is often therapeutically sought in many dermatoses.

The case shown in **Fig. 138** is interesting because the X-rays, which were applied to a peripheral sarcoma of the femur, not only

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caused no improvement, but aggravated the tumor. This demonstrates the inefficiency of treatment of some malignant tumors by the X-rays, and, as operative interference is postponed, more extensive removal becomes necessary later on (see page 30). In this case X-ray examination showed the presence of sarcomatous masses in the soft parts, necessitating high amputation through the thigh.

Fig. 137 shows a case of **frostbite** of the first and second degrees in a workman who had had repeated milder attacks in the winter, after exposure of his hands to cold water during his work. The hands were permanently blue, and in the winter painful chilblains developed on the fingers, especially on the extensor surface. He finally developed frostbite of the second degree, which is shown by the whiteness of the ends of the fingers, and other changes in the fourth finger. The skin over the first joint of the fourth finger is blue, and a large blister containing yellow lymph has developed on the extensor surface of the last joint.

Cold as well as heat may destroy tissues. Here again the effects depend on the degree of cold, the duration of its action and the condition of the patient. Dry cold is better borne than moist cold. Certain individuals are especially liable to the effects of cold—persons in a state of alcoholic intoxication, anemic individuals, children and old people, cooks and others who are exposed to rapid changes of temperature. Frostbite may be caused by the action of snow, ice, liquid air or carbon dioxide snow. The latter, which in the past few years has become a valuable therapeutic agent in dermatological practice (see treatment of nævi, page 104), must be handled carefully.

Chilblain (or *pernio*) may be regarded as a *chronic form of frostbite,* affecting the fingers, toes and ears. It is especially common in chlorotic subjects and causes swelling and cyanosis of the skin with numerous bluish-red nodules. These often cause unbearable itching and burning sensations, and, when scratched, give rise to intractable ulcers.

There are several degrees of **acute congelation of tissues,** just as there are degrees in burns. Those parts of the body that are most exposed and where the circulation is slackest are most frequently affected: namely, the ears, nose and toes.

In the *first degree* of frostbite there is redness of the skin from hyperemia (erythematous congelation). This is usually followed in a short time by the development of a blister. The redness increases

Fig. 139. Mal perforant du pied — Gangraena Raynaud.

Tab. CXII.

Fig. 140. Gangraena diabetica — Arteriosklerosis.

when the patient comes into a warm room, or takes alcoholic drinks. It is accompanied by burning and itching pains, which may continue for a long time. But recovery is the rule.

A longer exposure, or exposure to more severe cold, causes venous stasis, edema, and blister formation. In this *second degree*, as in the second degree of burns, pain is more severe. The skin becomes blue or white, cold and insensitive to the touch, and is often covered with numerous blisters, with bluish-black contents; after rupture of these blisters the exposed corium is dark in color and very painful. Infection is liable to occur, causing extensive ulceration with little tendency to heal, and leading to cicatricial contraction.

In frostbite of the *third degree*, in the same way as in third degree burns, there is sloughing of the skin and necrosis of the deeper tissues, due to thrombosis of the vessels. The skin is at first bluish-black, cold and insensitive, later on quite black. Separation of the frozen tissues may take place either by dry or moist gangrene. The zone of demarcation has often a putrid character. Progressive phlegmonous inflammation may spread from the borders of the frozen area, and may lead to general infection. Along with frostbite of the third degree the neighboring parts are affected in the first and second degrees, and other parts are ulcerated; so that the clinical picture is variegated. The dead parts, after some months, are cast off spontaneously. The nails soon fall off in frostbite of the hand. In frostbite of the third degree, parts which at first showed signs of the second degree only, may afterward become gangrenous.

Healing eventually takes place by production of very unsightly hypertrophic scars, which may cause retractions. Contractures may also be caused by paralysis of nerves, or by waxy degeneration of muscle fibres. Frostbite is said to cause changes in the blood-vessels which may lead to secondary gangrene.

The general condition of the patient is little impaired in acute local frostbite of circumscribed regions. The period of healing varies according to the degree of the frostbite, but is usually longer than in burns and after effects are more severe.

General frostbite is observed in very cold winters, in persons lost in the snow, and in Arctic explorers. An invincible somnolence is a premonitory symptom. If the sufferer is allowed to stop and sleep, he certainly will freeze to death. Only early attempts at resuscitation have any chance of success. Patients must be *very gradually* warmed; placed at first in a cold room and rubbed with snow, then with ice water, then, in the course of several hours, the temperature

is very slowly raised. If breathing has stopped, artificial respiration must be resorted to.

Chilblains may be treated by hot air apparatus or sandbaths, together with the general treatment of anemia with iron and arsenic. The irritation may be relieved by painting with tincture of iodine, balsam of Peru. Ulcers are best treated with mild antiseptic dressings and bland ointments. Recurrence can be limited by prophylactic measures, avoidance of exposure to cold and wearing woollen garments.

In acute local frostbite the parts must be warmed gradually—by rubbing with snow or cold applications. Early treatment in this way may restore the frozen skin. In frostbite of the second degree large blisters should be opened and broken epidermis pared off. Ulcers should be treated with strict asepsis, and dressed with sterile gauze or ointment. The extremities should be suspended on splints, all pressure being carefully avoided.

In cases with moist gangrene and putrefactive phlegmonous inflammation, early amputation is often necessary to prevent general infection. In dry gangrene, amputation may be deferred till a zone of demarcation has formed. Plastic operations are often required after spontaneous separation of gangrenous parts of the fingers or toes. Morphia may be necessary for the severe pain in the early stages of frostbite and has not the drawbacks it has in burns.

Fig. 141 shows a case of gouty arthritis of the metacarpophalangeal joint of the second finger, in a predisposed subject, who had already had several previous attacks. The whole joint is swollen and very painful to touch and on movement. Tophi are present on the other metacarpo-phalangeal joints and on the interphalangeal joints of the second to the fifth fingers. The skin over the tophi is white from pressure.

Gout is a disorder of metabolism occurring in middle-aged men, often with a hereditary predisposition, who indulge in high living and take too little exercise.

Sodium urate is imperfectly eliminated and deposits are formed in various places, especially the articular cartilages, but also in the synovial membranes, tendons, subcutaneous and periarticular tissue, bursæ, bronchi, intestinal mucosa and kidneys.

An **acute attack of gout** is caused by deposits of sodium urate in a joint, usually the metatarso-phalangeal joint of the great toe

Fig. 141. Arthritis urica.

(*Podagra*). The symptoms are great pain in the affected joint, slight rise of temperature and a certain amount of constitutional disturbance (gastric pain, nervous phenomena, rheumatic pains, etc.). The first attack is sometimes excited by an injury to the foot. The region of the joint is swollen and edematous, and the skin shows erysipelatous reddening and phlegmonous infiltration. The slightest touch or movement causes intense pain. There is a slight effusion in the joint. After some hours the pain subsides, but it generally recurs on the second night; and so on for about two weeks, till the attacks gradually become less painful and finally disappear. A slight swelling of the affected joint remains. Later on fresh attacks may occur, often after many years. During the attacks there is always a heavy sediment in the urine. Repeated attacks may give rise to a permanent nodular swelling of the joint, and slight trauma may bring on another acute attack (e.g. vigorous shake-hand on gouty fingers, stubbing of the toes).

Chronic gout, which is rarely primary and generally results from the acute form, is observed also among the poorer classes. It often affects articulations, but is less painful. The frequency with which the metatarso-phalangeal joint is attacked is perhaps due to bad circulation of the blood, owing to its peripheral position. (This joint is also affected by arthritis deformans in old people). Large deposits of sodium urate give rise to gouty nodules or **tophi,** which occur in the joints of the fingers, hand, foot and elbow. They also occur in the cartilages of the ear, nose and eyelids in the form of small, yellowish nodules, which become hard and painful. In advanced cases these nodules may be found in all articular and periarticular structures, tendon-sheaths, cartilages of the ribs, and in other tissues.

Chronic gout is very liable to acute flare-ups, especially after indiscretions in diet. Tomatoes are believed to have a very bad influence and *Johnson* has described what he calls the *tomato joint*. There may be some slight exaggeration in this.

Microscopical examination of gouty deposits shows the presence of sodium urate crystals. These act as foreign bodies and cause pain and pressure necrosis. The cartilages are eroded; subluxation and ankylosis are frequent. Suppurative arthritis is always due to a mixed infection and a serious complication.

Although joints are chiefly affected, gouty deposits in other organs may give rise to the most diverse symptoms: pain in the heel, sciatica, lumbago, asthma, bronchitis, nephritis, iritis, emphysema, etc.

Chronic interstitial **nephritis** and **calculus** are the two main complications in gout of long standing.

Gouty arthritis is most often confounded with **chronic rheumatism,** but in the latter the skin over the joints is unchanged. In **purulent arthritis** there is high temperature and rigors, while the temperature in gout does not exceed 100° provided no suppuration is present. The localization of gouty arthritis to small joints of the hands and feet is a good diagnostic sign; but this is not constant. Enchondroma of the fingers (**Fig. 50**) differs from gouty deposits by the absence of pain and its size.

Gout of other organs must be diagnosed by the history of the case. Large deposits of sodium urate can be seen by X-ray examination; e.g. in bursæ.

Treatment

Treatment of uncomplicated gout is purely internal. Prophylaxis consists mainly in hygienic and dietetic prescriptions, care being particularly paid to the purin contents of food. During acute attacks, colchicum and rest; in the interval, hot air hyperemia and mineral water "cures" are the main therapeutic elements.

Surgery intervenes in gout only in case of complications, such as suppurative arthritis, or of sequelæ, such as stone of the kidney.

MALFORMATIONS

Figs. 142-150

Fig. 142. Encephalocele occipitalis — Rachischisis.

MALFORMATIONS

Figs. 142 to **149,** inclusive, represent *congenital malformations.* These are accounted for by embryology.

Slight developmental disturbances are called *anomalies:* greater deformities, *malformations..*

There are *primary* malformations which affect the embryo early in its development, and *secondary* malformations, or *arrests of development,* in which an influence acting later in intra-uterine life modifies the growth of a part already formed. The earlier the cause begins to act, the greater the malformation. The causes which lead to malformation may be *intrinsic,* that is, lie in the embryo itself, or of *external origin.* Experimental observations on animals have shown that malformations may be caused by injury. In the lower extremities malformations may be caused by pressure or by abnormal positions of the fetus in the uterus (various forms of talipes—pes varus, pes valgus, pes calcaneus). Pressure on the fetus may be caused by a uterine tumor or by deficiency in the liquor amnii, and signs of such pressure can often be seen after birth of the child. Many malformations are due to anomalies in the membranes; e.g. amniotic adhesions, which may prevent the union of parts which should normally become united (branchial clefts) or may cause duplication of parts, or partial or complete separation (amniotic amputations, **Fig. 149,** aberrant glands).

Figs. 142, 143, and **144** represent malformations due to *imperfect closure of the cerebro-spinal canal.* In the spine, there result different degrees of **spina bifida,** from *meningocele* and *spina bifida occulta* to *rachischisis* (**Fig. 142**). In the skull, there is formed an *encephalocele* (**Fig. 142**), which is nothing but a *cranial spina bifida.*

Encephalocele occurs in the *nasal* region (syncipital encephalocele, subdivided into naso-ethmoidal, naso-frontal and naso-orbital varieties) and in the *occipital* region (occipital encephalocele, distinguished as superior and inferior according to its situation above or below the occipital protuberance). Encephalocele is rare (about 1 case in 5000 births).

In extensive cases there may be acrania or anencephalus, while

in slighter degrees there is only a defect in the bone and dura mater. Owing to the defect in the dura mater there may be prolapse of the brain through the bone (*encephalocele proper*); generally there is a hernial protrusion of one of the ventricles (*hydroencephalocele*). The existence of a true congenital meningocele in which the dura is intact, and there is only protrusion of the membranes through the gap in the bone, must be regarded as doubtful. *Bockenheimer's* observations on myelocele (**Fig. 144**) have also shown that the inner covering of the protrusion, which is said to be dura, often consists of connective tissue only, and that the inner wall is often formed of ciliated columnar epithelium, and, therefore, represents the degenerated ventricle of the brain. Hence the so-called meningocele is a true encephalocele (or myelocystocele). Again, so-called encephalomeningocele has been shown to be not a true meningocele, but a cystic formation which has become gradually cut off from a primary hernia cerebri or encephalocele.

As the subdivision of the different forms into meningoceles, encephaloceles, encephalomeningoceles, encephalocystoceles and encephalocysto-meningoceles depends on pathological anatomy, and cannot be made clinically, it is sufficient for all practical purposes to use the term encephalocele for all hernial protrusions through the skull.

Syncipital encephaloceles generally have a wide base while occipital encephaloceles are pedunculated (**Fig. 142**) and may attain a large size (as large as the child's head); the skin at the base of the tumor is thickened and covered with radially arranged hair. The tumor may be covered with normal skin, but more commonly most of the surface resembles fresh scar tissue; or, when ulceration is present, it resembles the mucous membrane of the intestine. Vascular anomalies, telangiectases and angiomata, are often present.

The tumor is *diminished by pressure,* and can be completely emptied in cases when it apparently contains only fluid. After the tumor has been emptied by pressure the hole in the skull can be felt, situated symmetrically in the middle line. It is generally small and circular, and can sometimes be shown by X-ray examination. As the tumor can be decreased by external pressure, so is it increased by internal pressure; *e.g.* when the child cries.

In some cases there is but little diminution on pressure. Irregular partitions can then be felt in the interior of the sac. Firm pressure then usually causes bulging of the fontanelle, or sometimes convulsions. Cystic encephaloceles may be *translucent.* In other cases there may be *pulsation.* Then the skull is usually very small and flat-

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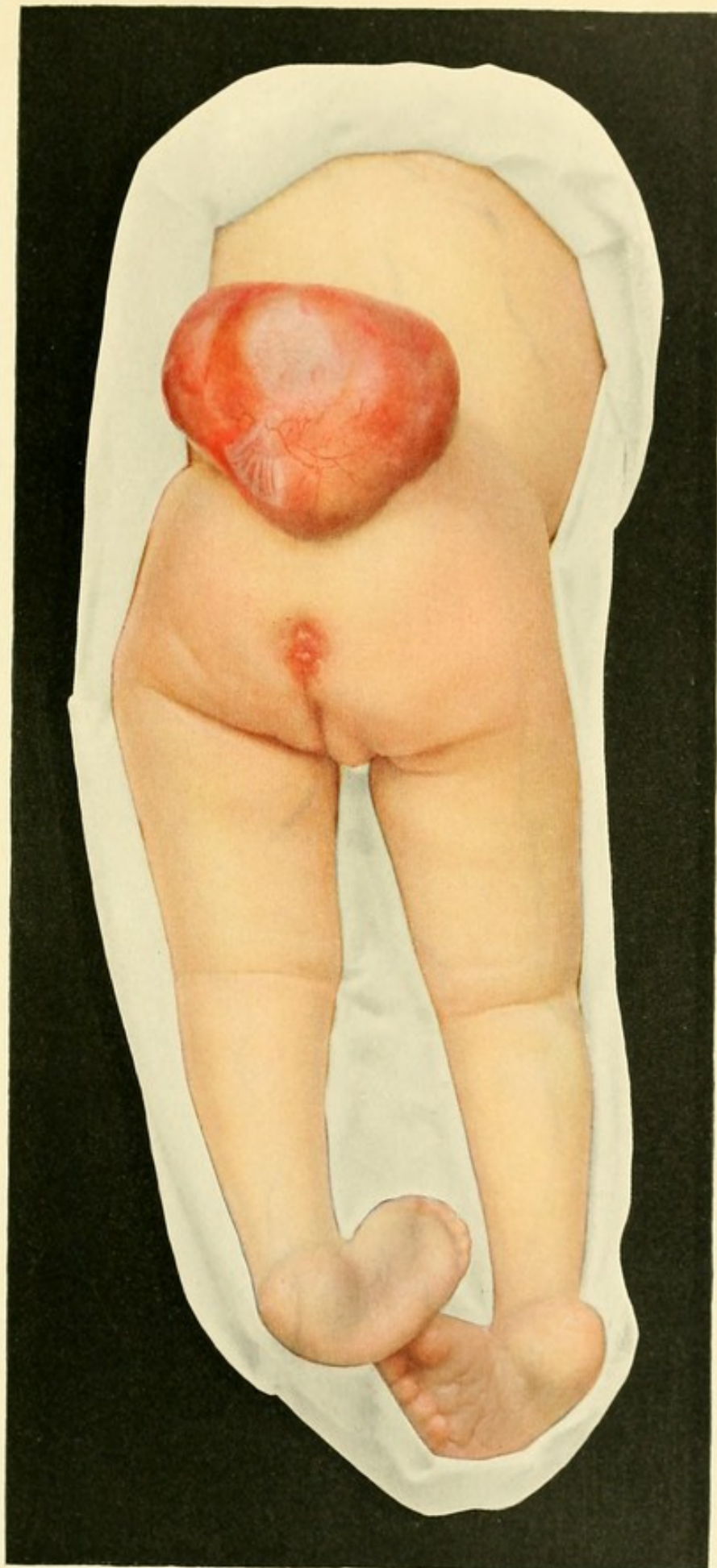
Fig. 143. Myelocele — Pedes vari.
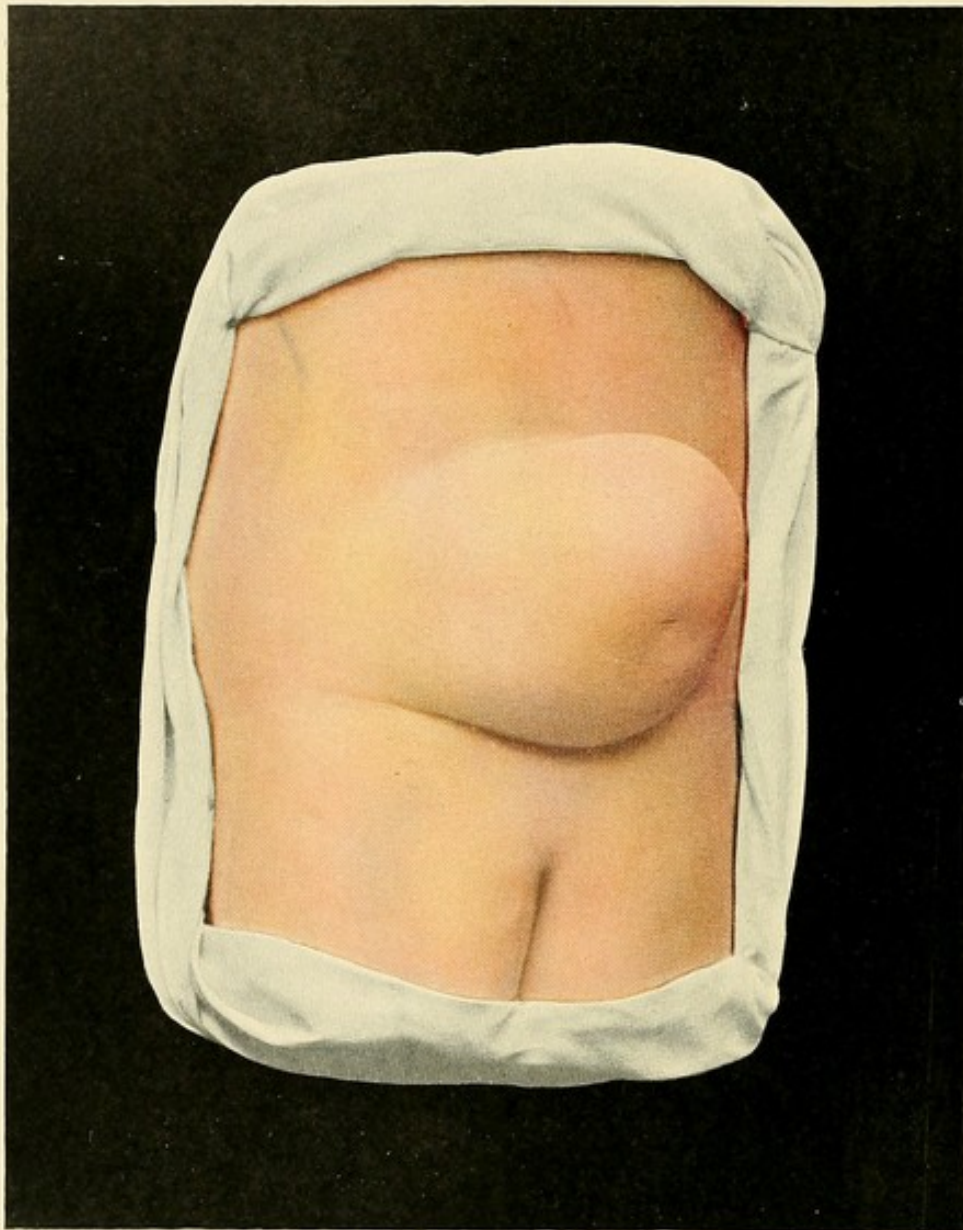
Fig. 144. Myelocystocele — Myxolipoma.

tened, and other malformations are present; the infants are weakly and have a subnormal temperature.

Spina bifida is slightly more frequent than encephalocele (15 in 10000 infants) and exists in several degrees. The most extreme is *rachischisis* (**Fig. 142**), in which there is a lack of closure of the bones, soft parts, spinal cord and membranes. It may extend for the whole length of the spine (total posterior rachischisis) or be limited to a shorter segment. In **Fig. 142** it extends from the twelfth dorsal to the third lumbar vertebra. It is most common in the lumbo-sacral region, because in this region the medullary groove closes last to form the neural canal. Rachischisis is usually associated with other extensive malformations, such as anencephalus, acrania, absence of vertebral bodies, etc. Three typical zones can be distinguished symmetrically on each side of the vertebral column: (1) a circular, peripheral zone of *thickened skin,* often covered with abundant hair; (2) a middle zone which resembles fresh cutaneous scar tissue, or the serous coat of the intestine, and hence has been called the *epithelio-serous* zone; a central zone of *flabby granulations* with a depression at the upper and lower ends, which represents the open and exposed spinal cord. The depressions at each end of the central zone lead to the ependymal canal of the spinal cord. Rachischisis is caused by a very early arrest of development in embryonic life. The second form (*myelocele*) results from a later disturbance and is limited to a smaller extent of the spine, although it may include the soft parts, bones and spinal cord; this forms a tumor-like swelling. The third form (*myelocystocele*) occurs still later in embryonic life, at a time when the spinal cord and the skin have already closed on the dorsal surface of the embryo, but the dura mater and bone have not yet united. The fourth form (*meningocele*) only occurs in the lumbo-sacral region where the spinal cord has become the filum terminale. *Spina bifida occulta,* which also occurs at the lower extremity of the vertebral column, is not to be regarded as a special form, but as a meningocele.

By far the most common variety is **myelocele** (**Fig. 143**), which is usually situated in the lumbo-sacral region, sometimes in the cervical or thoracic. It forms a characteristic swelling with a broad base, symmetrical as to the midline, with the three zones already described for rachischisis, viz.: *external* of hairy thickened skin; *middle,* epithelio-serous; *central* on the apex of the tumor, red, very vascular and covered with pus a few days after birth. This third zone is called vasculo-medullary and represents the remains of the cleft spinal cord.

At its upper and lower ends are depressions through which a probe can be passed into the ependymal canal.

In myelocele there is no *reductibility on pressure,* owing to the absence of communication with the subarachnoid space.

In myelocele the spinal nerves become dragged upon by the increasing growth and motor paralysis of the lower extremities, bladder and rectum (of the upper extremities in high myelocele) results. The common occurrence of pes varus in these cases (**Fig. 143**) is due to the myelocele being usually situated at the junction of the lumbar vertebræ with the sacrum where arise the nerves supplying the anterior and posterior tibial muscles; viz. fourth and fifth lumbar, first and second sacral nerves. Sensory disorders are rare in myelocele, but trophoneurotic disorders occur in the form of extensive eczema and decubital ulcers, especially on the feet; in pes varus on the outer border of the foot. Other malformations, umbilical hernia, etc., are frequently associated with myelocele.

Myelocystocele (**Fig. 144**) is caused by arrest of development of the vertebral arches and the dura mater only. It appears in the third week of embryonic life, at a time when the medullary groove has closed to form the neural tube, and the epiblast has grown over it. Hydrops of the central canal causes bulging of the posterior part of the spinal cord through the gap in the vertebral arches, giving rise to a tumor-like swelling of the spinal cord covered by the soft parts. The substance of the spinal cord soon undergoes degeneration and can only be identified by the presence of ciliated cylindrical epithelium on the inner surface of the cavity (the remains of the ciliated epithelium of the central canal of the spinal cord). In the external coverings of myelocystocele there is often lipoma, myxoma, lipomyxoma (**Fig. 144**), lymphangioma or teratoma. The tumor has a wide base and is covered with *normal skin,* which is thickened at the base of the tumor. Sometimes small depressions caused by the remains of amniotic bands are present in the skin (**Fig. 144**). The tumor is of soft consistency, and fluctuation is always elicited. The fluid contents of the tumor *can be completely reduced by pressure,* as there is direct communication with the central canal, and also with the subarachnoid space. By pressing on the tumor the transmission of fluid pressure can be felt at the fontanelle.

Myelocystocele is often combined with hydrocephalus. Paralyses are rare, as the motor nerves are not displaced by the malformation; at the most there may be pes varus or valgus on one side, due to the tumor being situated unsymmetrically more to one side of the midline

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and thus pulling on a motor nerve. However, extensive myelocysto-cele of the lumbo-sacral region may cause paralysis of the bladder and rectum. Trophoneurotic disorders are common. Sometimes paraly-sis occurs at a later age, the tumor gradually increasing in size and dragging on the spinal cord and nerves. Defective bone formation is often associated with myelocystocele—absence of vertebral bodies, unilateral defects in the vertebral laminæ, absence of ribs or patella, scoliosis, etc.

Meningocele can only occur in places where the spinal cord is absent. In this condition there is a defect in the formation of the vertebræ and dura mater, so that the pia mater protrudes posteriorly, inclosing the filum terminale. In this way a pedunculated swelling is formed, *covered by normal skin,* which may attain the size of a child's head as the amount of cerebro-spinal fluid in the sac increases. Pa-ralysis only occurs when the meningocele is large, and generally is of limited extent. There is sometimes an abundant growth of hair on the prominent part of the swelling. Fluctuation is always present, but there is only slight diminution on pressure. The space in the bone is generally smaller than in myelocele. Meningocele occurs most often in the sacral region.

Spina Bifida Occulta is a form of meningocele which becomes ruptured and undergoes spontaneous healing under the skin. The pressure of the cicatrix may cause disturbances which are not noticed till the child grows older.

Diagnosis and prognosis

Syncipital encephalocele may be mistaken for *dermoid* (see **Fig. 48**) or *lipoma*. The diagnosis depends on the presence of a gap in the bone, diminution of the tumor on pressure and the presence of other deformities. (See page 64).

Occipital encephalocele may be mistaken for *cephalhematoma,* which sometimes occurs on the occipital bone, especially as the base of a cephalhematoma may be surrounded by a hard ring due to the raised periosteum. Cephalhematoma is not diminished by pressure. However, diminution by pressure may be absent in encephalocele if the gap in the bone is occluded. In doubtful cases an operation will settle the diagnosis.

Encephaloceles must be distinguished from acquired protrusions through a loss of substance of the skull. The prognosis is generally unfavorable, but is better in cases where the tumor can be completely emptied of fluid by pressure, and when no brain substance can be felt

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in the sac after evacuation of the fluid. Cases of occipital encephalocele with a large gap in the bone, often extending to the vertebræ of the neck, and protrusion of both occipital lobes and the whole of the cerebellum, are soon fatal. Other cases have a surgical interest because of possible operative interference.

That a tumor implanted in the midline in the lumbar region (or more rarely in other parts of the spine) is a spina bifida, is generally not difficult to recognize: the **nervous disturbances,** *motor, sensory* and *trophic,* the *paralysis of the bladder and rectum,* the *varus clubfoot,* all point to a spinal cord origin. Communication with the cerebro-spinal canal, as evidenced by reductibility and increase of pressure noticeable at the fontanelles is pathognomonic, but does not exist in all forms. *Lipomata, lymphangiomata, teratomata, dermoids* are not modified by pressure. The possible coexistence of spina bifida and lipoma (**Fig. 144**) must be borne in mind. Some cases of very vascular myelocele might be mistaken for *cavernoma* (compare the objective aspect in **Figs. 75, 142** and **143**), but the latter is not accompanied by any nervous disturbances.

To differentiate clinically from one another the several varieties of spina bifida, we have at our disposal two great signs which also have a prognostic significance. The *absence of normal skin* on the tumor *and of diminution on pressure* characterizes the latter as a *myelocele* or *rachischisis* (there being between these two types only a difference in extent); that is, we have to deal with a very early trouble in the evolution of the cerebro-spinal axis itself; *the cord is malformed; no operative cure is possible* and death will result in a few days from septic meningitis, because the meninges are widely open.

On the other hand a tumor *covered with normal skin* and *reducible on pressure,* announces a *myelocystocele* or *meningocele,* that is, a malformation due to a much later trouble in the evolution, with a spinal cord well formed except for the functionless, degenerated part contained in the sac, in case of myelocystocele, or even altogether normal, in case of meningocele. No meningitis is to be feared, and operative correction is possible.

To sum up, the prognosis is very bad in myelocele, when the lesions are inoperable, and the associated malformations often of the gravest character; it is not unfavorable in myelocystocele, provided other malformations are absent and the infant has a strong constitution; it is generally good in meningocele.

The possibility of spina bifida occulta must always be borne in

mind in cases of trophoneurotic disorders in the lower extremities, the presence of which is hard to account for. An X-ray examination of the spine will then solve the problem. Such an examination is always useful, even in the other varieties, to show the extent of the bone malformations.

Treatment

Tapping and injection are useless, and dangerous in all these malformations of the nervous system. However, *tapping* is the only palliative measure applicable in myelocele where a radical operation is impossible, as removing the cystic sac would necessitate dividing the spinal cord, which would unite with the scar. On the other hand, reduction of the vasculo-medullary zone would simply hasten death by septic meningitis.

But in encephalocele, myelocystocele and meningocele, a *radical operation* is possible; it is somewhat similar in principle to that for hernia. The sac is exposed by a free incision, dissected down to the bone and opened; the contents are reduced or resected according to their nature and importance, the sac ligated and excised, and the defect in the posterior wall repaired. The gap in the bone may be bridged over by suturing the periosteum over it, by a pedunculated bone flap, a bone transplantation, or a celluloid or silver plate.

In case of encephalocele, where brain substance is found in the sac, radical operation is feasible only when said brain substance can be reduced through the gap in the bone without producing cerebral pressure symptoms. Removal of portions of brain still possessed of function may cause dangerous disturbances, but a degenerated dropsical protrusion may be removed without danger. Cases in which there is a large defect in the skull and cervical vertebræ, or cases combined with other extensive malformations, are inoperable. The after-treatment is complicated by the escape of cerebro-spinal fluid, which is always abundant, even after the most careful closure of the bone defect. The dressings, therefore, require changing several times daily to prevent ascending infection of the wound.

In myelocystocele the sac is often covered by a fatty tumor which also requires removal. Removal of the sac after ligation is not dangerous in these cases, as it consists only of functionless degenerated spinal cord. Meningitis sometimes follows these operations, but most cases recover and may grow up.

In meningocele, conditions are best and the operation is simple.
In spina bifida occulta with disturbances due to pressure of the

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cicatrix, the latter may be excised and the gap in the bone repaired.

The development of hydrocephalus, which may occur after operation on all forms, is an unfavorable sign.

In the case represented in **Fig. 142,** the *encephalocele* could have been operated on, if the *rachischisis* had not made the condition of the child hopeless.

In **Fig. 143,** the *myelocele* was accompanied by *double club-foot* (*varus*). Death occurred soon after birth.

In the case shown in **Fig. 144,** there was a superficial mass of fatty tissue, while underneath was a cystic tumor which could almost be emptied by pressure. There were no motor or sensory disorders present, and no other malformations. The X-rays showed a small cleft in one of the vertebral arches a little lateral to the midline. The superficial fatty tumor was removed and found to be a *myxolipoma.* The myelocystocele was then dissected free down to the bone, ligated and removed. The gap in the vertebra was closed by transplantation of a piece of the iliac crest. Microscopic examination showed the presence of cylindrical epithelium on the inner wall of the cyst, thus confirming the diagnosis.

A few words must be added about the *varus clubfoot,* shown in **Fig. 143.** Varus clubfoot may be *congenital* or *acquired.* The congenital form may be caused by arrested development, or may be secondary to pressure caused by amniotic adhesions, etc. Congenital varus is common in connection with myelocele, and is due to paralysis of the nerves, as already explained.

Acquired varus occurs in rickets, and as the result of poliomyelitis which causes paralysis of the pronators and dorsal flexors of the foot. The chief effect takes place at the midtarsal joint and consists in supination, plantar flexion, internal rotation and adduction. Changes also occur in the astragalus and os calcis, especially in long-standing cases. These changes can be seen by the X-rays. There is also retraction of the muscles, tendons, fascia and ligaments, especially shortening of the tendo Achillis (talipes equino-varus). Pressure ulcers may develop on the outer border of the foot.

In congenital clubfoot treatment should be begun as early as possible, by repeated manual correction (if reduction is easy without straining the arch of the foot), followed by fixation in an over-corrected position in a plaster of Paris cast. In sucklings, strapping in over-corrected position for six months, by means of strips of adhesive plaster may give good results.

Fig. 146. Teratoma monogerminale.

Fig. 145. Lymphangioma congenitum multiplex.

After the ninth month **preliminary tenotomy of the tendo Achillis** *is necessary,* before the foot can be brought into the proper position, and the same operation is indicated whenever correction cannot be obtained without using violence. To prevent relapse boots should be worn with the sole raised on the outer side, but care must be taken to avoid producing flat foot. In varus clubfoot due to poliomyelitis, tendon transplantation may be performed. Old-standing cases of clubfoot in adults require osteotomy or sometimes even more extensive operations, or exarticulation.

Fig. 145 shows a **congenital tumor** involving the lower part of the right cheek, the whole right side and the greater part of the left side of the neck. The skin was unchanged and movable over the tumor which, on examination, was found to be multilocular and cystic. There was no diminution on pressure. The tumor also extended to the floor of the mouth, so that the tongue, which was considerably enlarged (*macroglossia*), owing to the presence therein of a similar cystic formation, was displaced upward. The greenish surface of the cyst was visible under the mucous membrane of the mouth, so that the diagnosis of **congenital multiple cystic lymphangioma** was made.

The term *lymphangioma* should be restricted to those tumors in which there is neoformation of lymphatic vessels, and not be made to include simple dilations (lymphangiectases) without new formation. (This distinction is identical with that between angiomata and angiectases, see page 101). Clinically, we divide lymphangiomata into single and multiple; anatomically into simple, cavernous and cystic. All three forms are often present in the same patient.

Lymphangioma in most cases is *congenital* or appears soon after birth. Its site of predilection is the subcutaneous tissue; but is also observed in the skin, muscular interstices and subserous tissue.

Simple lymphangioma is common in the tongue or lips where it forms a circumscribed tumor always somewhat adherent to the slightly thickened skin. Sometimes the name simple lymphangioma is wrongly applied to lymphangiectases, which form lobulated growths in the head, trunk and limbs.

Cavernous lymphangioma is always a painless, diffuse, slow-growing tumor, of soft consistency and with a smooth surface and irregular outline, adherent to the overlying skin or mucosa. It *gradually decreases under pressure,* because its endothelium-lined cavi-

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ties communicate with the neighboring lymph vessels. When visible under the skin or mucosa, it imparts to the latter a pale green hue, very different from the reddish blue color of cavernous hemangioma (**Figs. 36, 80** and **81**). In the cheeks, tongue, and lips they give rise to enlargement of these parts (macromelia, macroglossia, **Fig. 145,** and macrocheilia). In the neck it causes a dimpled swelling of the skin, owing to the numerous processes which it sends in all directions. (**Fig. 145.**) As already mentioned, lymphangiomata may be situated over encephaloceles or myelocystoceles. Gradual atrophy of the bones may be caused by the pressure of extensively progressing lymphangioma.

Cystic lymphangioma occurs in the subcutaneous or intermuscular tissue, most often in the lateral cervical region (**Fig. 145**). It is composed of large, cystic endothelium-lined cavities containing a whitish or brownish fluid. It is also almost always congenital and characterized by its slow growth, which may cease after some years. The skin is unchanged and can be raised from the tumor. Fluctuation is present, but there is no diminution of the tumor on pressure. Extensive lymphangioma of the neck may be dangerous from pressure on the trachea. Cystic lymphangioma may also occur in the axilla, the popliteal space, the bend of the elbow, the groin and the sacral region. Infants with congenital lymphangioma sometimes show other malformations, and are often incapable of surviving.

Diagnosis

Simple lymphangioma may be mistaken for *fibroma, lipoma* or *hemangioma.* The presence of transitional stages to cavernous lymphangioma in some cases helps the diagnosis.

Cavernous lymphangioma can only be mistaken for hemangioma, as no other tumor diminishes on pressure. It differs by its greenish color (compare **Figs. 36, 80** and **81** with **Fig. 145**) and the nature of its contents.

Cystic lymphangioma, when occurring as a single multilocular cyst, may be mistaken for various tumors, according to its location: *blood cyst, branchial cyst, lipoma* or *dermoid.*

In a case like that shown in **Fig. 145,** on account of the situation of the tumor on both sides of the neck in the submaxillary, submental and parotid regions, the case might be mistaken for an affection first described by *Mikulicz,* in which there is symmetrical enlargement of all the salivary glands and glands of similar structure in the head and neck. In this case, however, there was no change in the lacrymal

glands, which are usually affected in *Mikulicz's* disease; also there was a characteristic lymphangioma in the tongue, which is absent in *Mikulicz's* disease. The swelling of the floor of the mouth on each side of the frenum of the tongue also resembles a *ranula,* i.e., a retention cyst of the sublingual gland.

The prognosis of limited forms of lymphangioma is not bad; it occasionally undergoes spontaneous resolution.

Treatment

Circumscribed lymphangioma is best excised. In diffuse cavernous lymphangioma (macrocheilia, macroglossia, macromelia) cuneiform excision may be performed. The introduction of magnesium in the cavity to cause thrombosis and shrinking of the tumor may be tried. It makes extirpation easier and avoids infection through a lymph fistula, which so often occurs after the usual operation. Radical operations should not be performed unless the child is in good condition. Puncture and injection of tincture of iodine are unsafe measures, while lymph fistulæ often remain after incision and packing. Lymph fistulæ must always be removed by a radical operation, on account of the danger of infection through them. Lymph fistulæ which occur from injury to the thoracic duct after extensive extirpation of tuberculous glands of the neck can be cured by prolonged packing.

Fig. 146 shows a ***teratoma of the left side of the face,*** nearly as large as the fist, involving the left orbit and almost the whole of the buccal cavity, and covered by livid, movable skin. It was surrounded by a connective-tissue capsule. Further examination showed that it arose from the base of the skull, but did not communicate with the cranial cavity. The tumor was soft and fluctuating in some places, hard in others. Investigation by the X-rays showed the presence of a piece of bone, which was afterward identified as part of the upper jaw. On microscopical examination the tumor was found to consist of neuroglia, neuroepithelium and cysts lined with epithelium. There were no other malformations present except mutilation of the right ear. Death occurred soon after birth.

Teratomata may be *bigerminal* or *monogerminal.* In bigerminal teratoma there is a true double formation—a fetus within a fetus. In monogerminal teratoma all the tissues are derived from one embryo only. The latter includes all kinds of mixed tumors, which are

constituted by all three embryonic layers (epiblast, mesoblast and hypoblast). Those dermoid cysts which are formed by all three embryonic layers belong to teratomata. A distinction between monogerminal and bigerminal teratomata is not always possible, and is of little clinical importance. In the case figured in **Fig. 146,** as the mass consisted of epiblastic products only, it must be regarded as a monogerminal tumor which originated from a separated portion of the epiblast. This view is supported by the fact that the tumor developed in a region (base of the skull) where epiblastic inclusion is possible. On the other hand, it appears somewhat arbitrary to consider the tumor as a bigerminal teratoma simply because of its large size at birth.

Teratomata are rare on the whole, and always congenital. They are most often found in the buccal cavity, where they may be mistaken for naso-pharyngeal polypi (**Fig. 25**). They also occur in the face, neck and coccygeal region, and have been observed in the mediastinum and abdominal cavity. They may attain a huge size and have then an irregular, uneven surface. The *consistency* also varies, some parts being cystic, others soft and others hard. Teratomata often form encapsulated tumors. They may cause extensive destruction by pressure on the neighboring parts. A distinction between teratomata and teratoid mixed tumors is clinically impossible. The exact pathological diagnosis in many cases is only made after examination of the extirpated tumor.

Diagnosis

Teratomata which appear as large, congenital tumors can generally be recognized by the above-mentioned characteristics, especially by their situation in the embryonic fissures. The diagnosis is assisted by the X-rays, which frequently reveal the presence of bones and teeth. Teratomata occurring in the thorax, abdomen and pelvis, especially when they do not assume a tumor growth till later years, can often only be diagnosed by operation. Dermoid cysts of the ovary are one of the most frequent and surgically interesting kinds of teratoma.

Treatment

Teratomata have been successfully removed both in children and in adults, especially those of the ovary or testicle.

Extensive teratomata of some regions (**Fig. 146**) cannot be removed by operation. Moreover, the presence of other deformities,
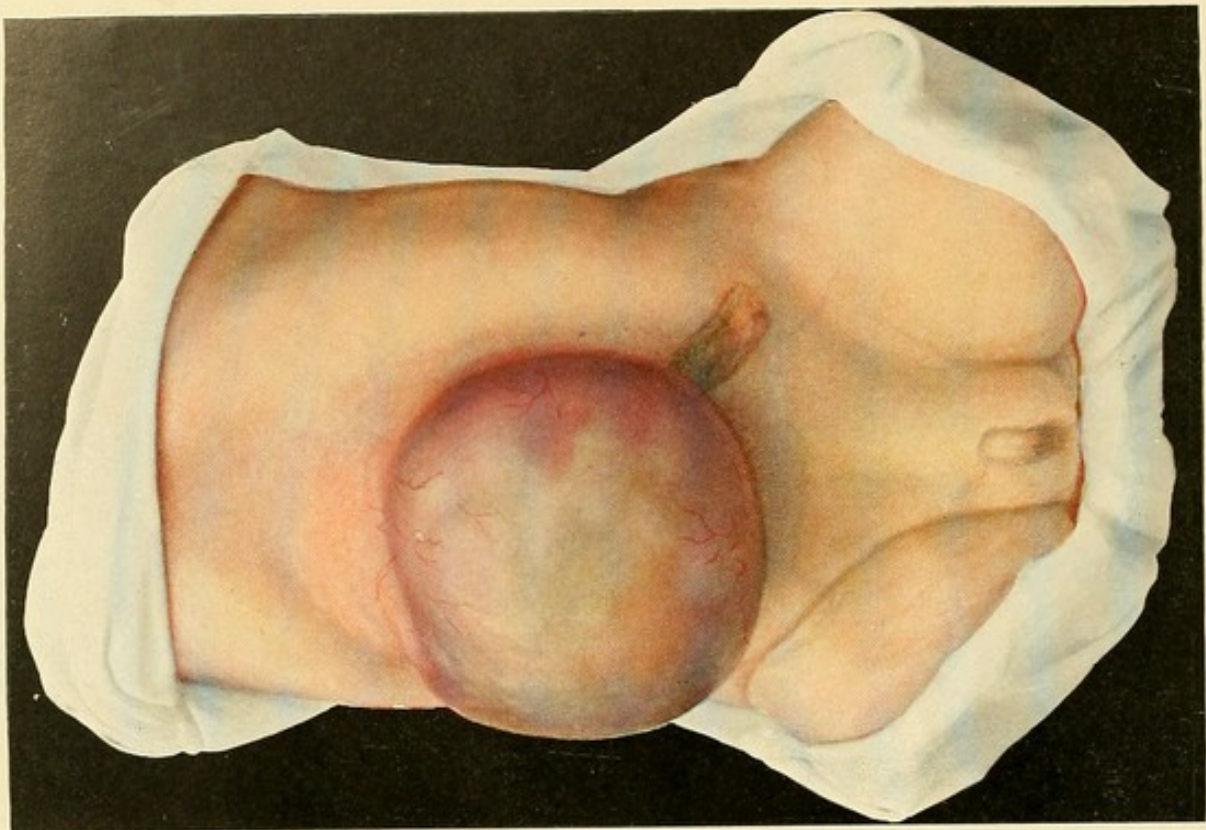
Fig. 148. Hernia funiculi umbilicalis congenita.
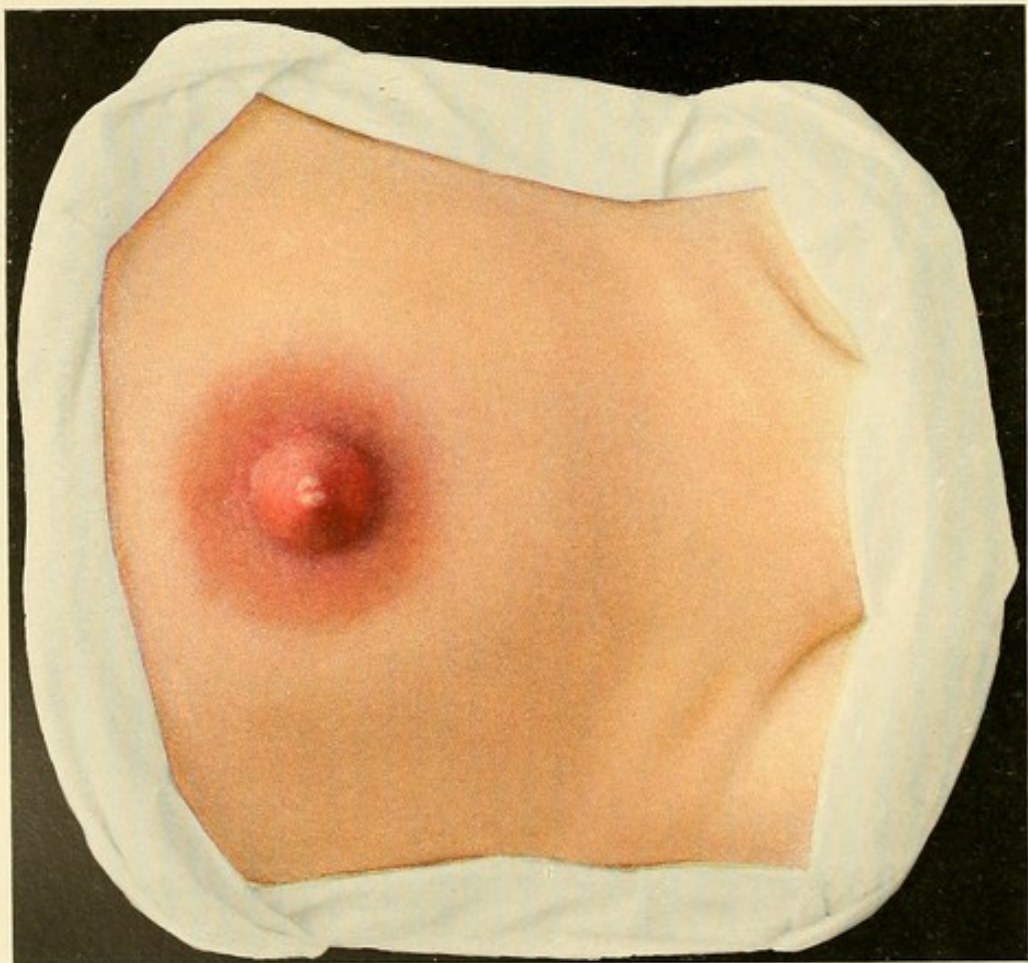
Fig. 147. Ductus omphalo-mesentericus persistens.

such as spina bifida, and the feeble condition of the infants often renders operative treatment impossible.

Fig. 147 shows a case of **fistula due to the persistency of the omphalo-mesenteric (or vitelline) duct;** that is, of the communication between the alimentary canal and the umbilical vesicle, or yolk-sac, which usually disappears about the eighth week of fetal life.

When it persists, it is known as **Meckel's diverticulum** and is implanted anywhere on the small intestine, but most frequently about 30 inches above the ileocecal valve. Its frequency is estimated to 2% of autopsies. It may become attached to the umbilicus or even extend for a short length into the umbilical cord. When the latter is tied close to the abdominal wall, the diverticulum is pinched, and when the cord falls, a small fistula is formed, which discharges *feces* if the diverticulum is patent throughout, or *mucoid secretion* if only the external end is pervious. Cystic dilatations may develop if both ends are closed and the middle pervious.

Later in life *Meckel's* diverticulum plays a not unimportant part in abdominal pathology (*Porter, Balfour* of *Mayo's* clinic). It is more dangerous than the vermiform appendix. Diverticulitis therein is not rare: about 2% of the cases of intestinal invagination originate in *Meckel's* diverticulum; again, it may become entangled with other loops of gut and cause obstruction. The band connecting the diverticulum with the navel is more dangerous in this respect than the diverticulum itself.

In umbilical fistula due to *Meckel's* diverticulum a red globular swelling is seen at the navel, with a small depression at its apex. The surface of the swelling is formed by mucous membrane. A probe can be passed through the depression as far as the small intestine, if the whole duct is open. If such be the case, the greater part of the feces are discharged through the fistula, causing inflammation of the skin surrounding the navel. Death often occurs from prolapse of the small intestine. In small, incomplete fistulæ there is no such danger and the only inconvenience is that of slight discharge and skin irritation.

Diagnosis

Septic conditions of the navel attended by granulation tissue formation somewhat resemble the above-described condition. Umbilical fecal fistulæ consecutive to ligation of the cord and without symptoms of intestinal obstruction are all due to *Meckel's* diverticulum.

There are also *umbilical fistulæ due to other causes,* e.g. to **persistency of the urachus,** which represents the remains of the communication between the bladder and the allantois in fetal life, and normally becomes obliterated and constitutes the median ligament of the bladder. Complete patency of the urachus causes a *urinary fistula;* incomplete patency a cyst. The fistula also appears after separation of the umbilical cord. In full-grown subjects the diagnosis of urachal fistula is not very difficult, as it may be helped by cystoscopy, and injection of colored fluids; but in the newborn, when the diagnosis cannot be made by chemical and microscopical examination of the secretion, powdered charcoal should be given by mouth: in case of vitelline fistula it appears at the navel; in case of urachal fistula, it does not.

In adults, *tuberculosis* of the intestine or peritoneum, actinomycosis, purulent peritonitis, empyema of the gall bladder and dermoids may cause umbilical fistulæ. The antecedent history often makes the diagnosis clear; but we must remember that fistulæ due to persistency of the urachus or *Meckel's* diverticulum do not always appear at birth, but often only late in life, and that tuberculosis of a partially patent urachus has sometimes been seen (*Pearse* and *Miller*).

Treatment

Fistula of the vitelline duct can sometimes be prevented by discovering the condition before tying the umbilical cord. The cord is then thicker than usual at its base. The end of the duct can then be reduced and the cord tied further away from the navel.

In cases of complete fistula leading to the intestine laparotomy is necessary, with resection of the diverticulum and suture of the intestine. This was done in the case represented in **Fig. 147,** but the child was in bad condition owing to the prolapse of the gut, the evacuation of feces from the navel and the excoriations of the surrounding skin, and the operation was unsuccessful.

Fistula of the urachus must be dissected down to the bladder and excised, after that the bladder is sutured.

Fig. 148 shows a large **congenital umbilical hernia** containing, as is often the case, the liver and intestines. Such herniæ are frequently associated with various forms of spina bifida or exstrophy of the bladder. Extensive umbilical hernia occurring at birth are

due to arrested development and incomplete closure of the abdominal walls.

There are also in infants umbilical herniæ resulting from a lesser disturbance of evolution; the abdominal walls are closed, the umbilical ring is not much widened, the hernia is cylindrical in shape, not large, and never contains anything but small intestine. These herniæ are frequent, but may be so small as to be overlooked at birth, and then be included in the ligature of the umbilical cord. The base of the latter should, therefore, always be examined to see if it contains intestine.

Congenital umbilical hernia forms a large *globular swelling* in the region of the navel (**Fig. 148**). The surface is *destitute of cutaneous covering* and shows the greenish-yellow remains of the amnion. The remains of the umbilical cord are generally seen at one side of the swelling. In rare cases epidermization takes place at the borders; more commonly the swelling ruptures from pressure, with consequent prolapse of the viscera and death from peritonitis.

Diagnosis and treatment

Both types of congenital umbilical herniæ are so characteristic that they cannot be mistaken for any other condition.

The occurrence of symptoms of intestinal obstruction, or threatening perforation of the sac, indicate *immediate laparotomy*, with excision of the sac, reduction of its contents and closure of the abdominal walls. In some cases the viscera are adherent to the sac and must be freed by dissection. Reduction of the visceral contents is sometimes difficult or even impossible, especially when the liver is contained in the sac. If operation is not urgent, it may be postponed till the child is stronger, the sac being supported by bandaging in the meantime. The results after these operations for large congenital herniæ are not very favorable; they are difficult; there is not enough abdominal wall to allow suture without great tension and pressure on the viscera; and death generally occurs soon after the operation, as happened in the case shown in **Fig. 148.**

Most of the herniæ of the smaller type become cured without any treatment. The placing of a pad of cotton over the umbilical ring is a bad practice, because it only tends to enlarge the ring. In older children a persistent umbilical hernia must be operated on as in adults; that is, by dissection and excision of the sac followed by careful repair of the abdominal wall.

Fig. 149 shows *constrictions of the fingers due to amniotic adhesions.* This is a mild type of a condition that may go as far as complete absence of the part affected (so-called *amniotic amputations*). There is a deep circular groove extending down to the bone; but, despite this, the circulation and the function of the fingers remained normal. In other cases there is often elephantiasic thickening from lymphatic congestion. In some cases the bones are constricted, as shown by the X-rays. The remains of the amniotic bands are often present in the constricted places.

Other malformations, also due to tightness of the embryonic membranes, are syndactylia (webbed fingers), harelip, cleft palate, transverse fissure of the cheek, macrostomia, and fissure of the tongue. In the case represented in **Fig. 149** there was also harelip and cleft palate.

More extensive varieties of malformation of the extremities include *amelus* and *phocomelus.* In amelus the extremities are absent or only represented by stumps. This condition may affect all four extremities, both arms or legs, or one arm or leg. In phocomelus there is arrested development of the proximal segments of the arms or legs, or of all four extremities. The hands or feet are then situated directly on the trunk. Some of these mutilated individuals attain adult age, and one has been known to live to sixty-two; several were among *Barnum's* freaks.

The treatment of amniotic constrictions or amputations is *nil,* except exarticulation of the useless stumps when needed.

Fig. 150 depicts a case of *acromegaly,* a condition in which there is enlargement of the terminal portions of the body—hands, feet, nose, cheeks, tongue and ears. The enlargement affects all the tissues and does not appear till after the termination of the period of growth, thus differing from congenital giantism. In some cases there is increased growth of hair, and curvature of the vertebral column. The disease causes considerable disfigurement of the face. It generally appears between the twentieth and fortieth years and may remain stationary, but generally increases slowly. In many cases there is, first of all, hypertrophy of the bones of the hands, feet and face.

There are usually headache, visual disturbances and more or less deterioration of the mental power.

Acromegaly has been traced to *tumors of the pituitary body,*
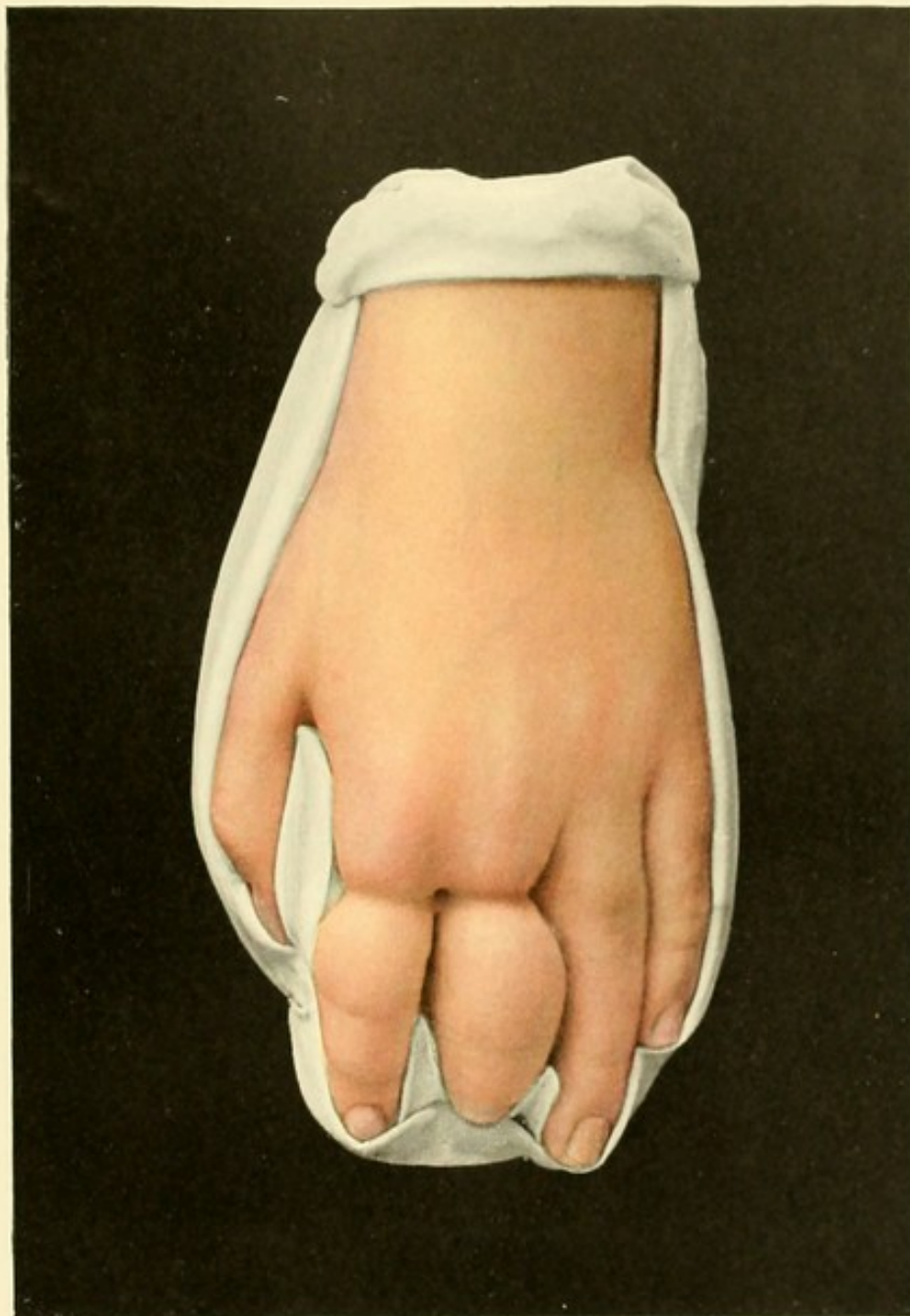
Fig. 149. Amputationes amnioticae.
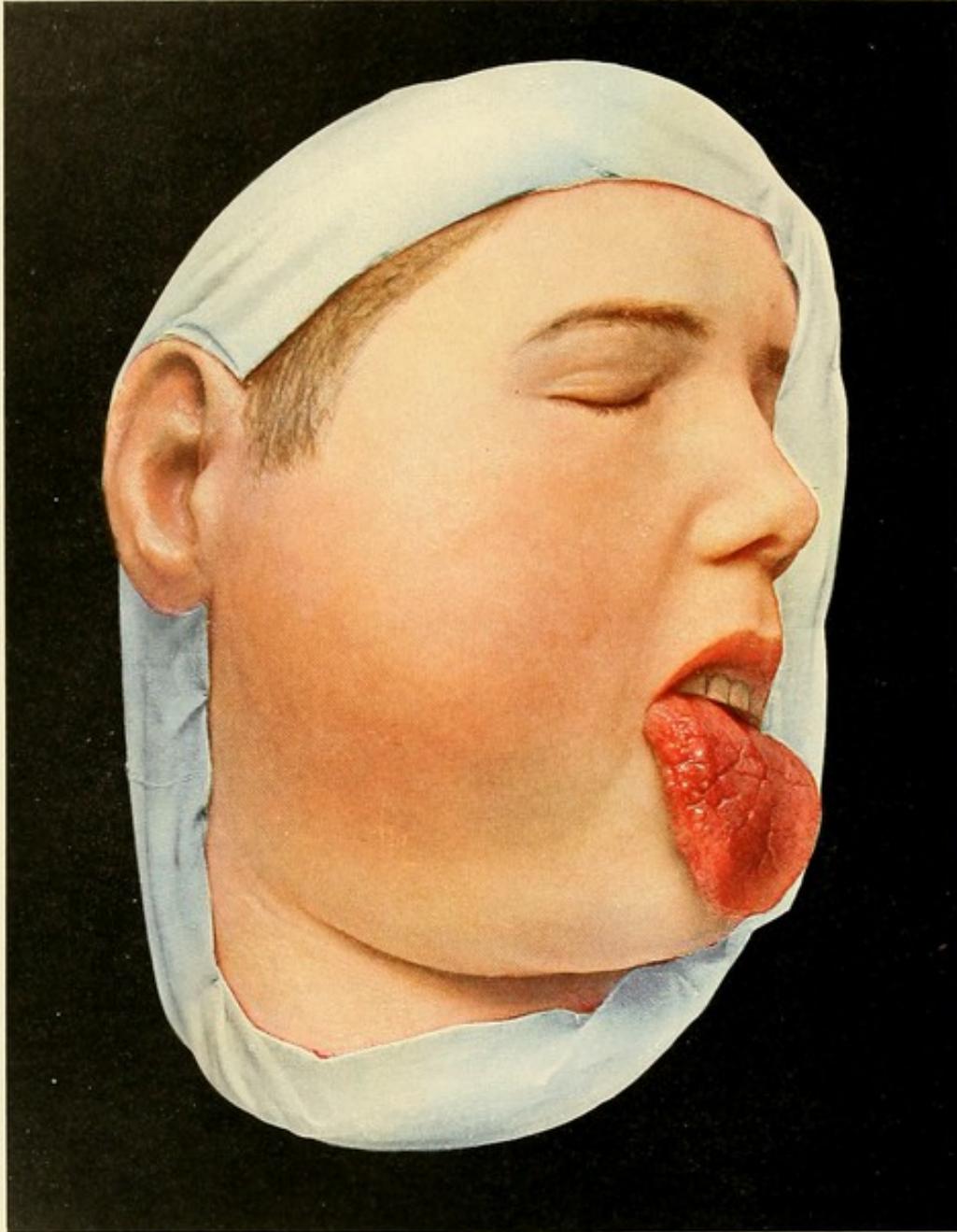
Fig. 150. Akromegalia — Makromelia — Makroglossia.

particularly to *adenoma of the anterior lobe*. It also has been seen associated with persistency of the cranio-pharyngeal canal, so that a congenital maldevelopment of the hypophysis may be a factor in some cases. Acromegaly is a condition due to **hyperpituitarism,** while **hypopituitarism** brings about *Fröhlich's adiposo-genital syndrom (Cushing).*

Tumors of the pituitary body can be demonstrated by the X-rays, which show a widening of the sella turcica. Large tumors of the pituitary body may press on the optic and ocular nerves.

Diagnosis

Partial giantism, which also begins in the hands and feet, differs from acromegaly by being congenital. In *leontiasis ossea* there is enlargement of the bones, while the soft parts are more often atrophied, and the enlargement is predominant in the bones of the face, not in the lower jaw.

Acromegaly affecting one extremity only might be mistaken for *osteitis* or *arthritis deformans* or for *chronic osteomyelitis,* as there may be lengthening of the bone in both these diseases.

Acromegaly differs from *elephantiasis* in the presence of enlargement of the bones, which can be shown by the X-rays. Acromegaly commencing in the face might possibly be mistaken for *tumor of the upper maxilla,* but there is usually early hypertrophy of the cheeks (macromelia), lips and tongue (macroglossia), and of the hands and feet.

Acromegaly is sometimes impossible to distinguish from the bone hypertrophy that occurs in a few cases of *syringomyelia,* and it is not unlikely that some of the cases reported as acromegaly without pituitary tumor were indeed syringomyelia.

Treatment

As acromegaly often markedly impairs the mental efficiency, an active treatment is indicated, even if the progress of the disease is slow.

The condition being, as it is, due to pituitary hypersecretion, opotherapy by pituitary extract is not rational. *Partial hypophysectomy (Cushing, v. Eiselsberg, Halstead, Kanavel)* has given notable subjective improvement, and the outlook in this direction is hopeful.

No treatment was applied in the case represented in **Fig. 150.**

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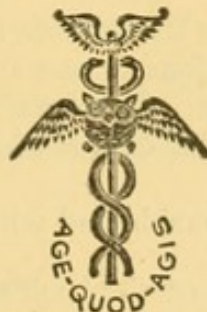
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