stretched and smooth and often paler than normal. No fluctuation nor redness till very late. Examination will often show a cause for such an abscess within the mouth. The best incision for opening a submaxillary abscess is one parallel to the lower margin of the jaw and over the maximum part of the swelling. There is no need to cut deep, for Hilton's method can be employed, and allows an adequate opening of the abscess. With a submental abscess the incision should be one along the middle line from the point of the chin downwards.

One of the largest and most important of the areolar spaces in the body is that around the kidney so that it is not surprising that large abscesses form in this situation. They are usually consequent on an abscess or whitlow elsewhere, and the causative organism is the *Staphylococcus aureus*. Lying deeply under cover of the lower ribs these abscesses often go for weeks before the correct diagnosis is made, and it is exceptional for the condition to be recognised before a fortnight has elapsed from the commencement of symptoms. Since the treatment of these cases is a very satisfactory affair it is well to emphasise some of the points in diagnosis. There is first a stage in which the only guide is the symptoms. Slight fever, malaise, possibly a rigor, and locally only slight pain when the fingers are pressed in under the ribs posteriorly. As the abscess grows bigger, signs appear at the base of the corresponding side of the chest—a little dullness on percussion with other indications either of compressed lung or slight collection of fluid. Lastly, a swelling may appear in the loin, but by the time this obvious swelling appears the patient may be in the last stage of weakness and anæmia. There may be no alteration in the urine. Any continued fever of a moderate elevation, accompanied by no other signs than some dullness at one base and some deep tenderness posteriorly at the erector-costal point, is very suggestive of a perinephric abscess in any patient who has previously had a whitlow or boil and in whom no trace of renal disease is obtainable by examination of the urine. Such an abscess is, as a rule, easily opened by a small incision at the outer border of the erector spinæ; the middle lamella of the lumbar fascia is penetrated, and the finger opens up the retrorenal space and evacuates the pus.

As a contrast to the large slowly-forming and often overlooked perirenal abscess I should like to mention a small inflammatory condition which is very painful, but may be misdiagnosed for a short while. I refer to the rather common boil which sometimes forms in the external auditory meatus. Though a boil is strictly speaking a focus of infective gangrene, yet for our purpose it may in this instance be regarded as an acute abscess, for the slough is not a noticeable feature. A furuncle in the meatus may cause such pain as to keep the patient awake for several nights running. The exudation is under tension. Some of the œdema and tenderness

betray themselves in front of the auricle whilst a certain amount of redness appears over the mastoid process. Hence the common mistake in diagnosis, for unless a very careful examination of the external meatus is made it is possible—I have known it happen several times—that an inflammation of the mastoid process may be diagnosed and an incision may be made over the mastoid under the supposition that there was an abscess there. A boil, as a rule, causes much more pain than a mastoiditis. When such a boil is seen in the meatus an anæsthetic must be given and an incision made by a narrow knife into the furuncle.

THE DIAGNOSIS OF SOME ACUTE ERUPTIONS.

BY

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(Concluded from p. 148.)

BULLOUS ERUPTIONS OF THE SKIN.

I NOW pass to the acute bullous eruptions of the skin. It may appear surprising that in a disease with so characteristic a lesion as that of pemphigus the question of differential diagnosis should arise, but to two eruptions the name of pemphigus was formerly given, though they are both in reality probably forms of contagious impetigo. I refer to the so-called *Pemphigus tropicus*, which I have not seen, and *Pemphigus neonatorum*, of which I have seen many cases. I do not think that true pemphigus is a disease which is ever found in the newborn. There are two main bullous eruptions of this age—namely, *Pemphigus neonatorum* and the bullous congenital syphilide, to which the rather unsuitable name of *Pemphigus syphiliticus* was formerly given.

Put shortly, the diagnostic distinctions are the following: In *P. neonatorum* the general health of the child is not usually disturbed at first, and may not be at any time; in the bullous syphilide the condition of the baby is very bad, and, as a matter of fact, I have never known a patient with the bullous congenital syphilitic eruption survive. Secondly, the distribution of the eruption of *P. neonatorum* is general, without sites of predilection, unless possibly the skin under the binder and in the flexures of joints is more apt to be affected; in the bullous syphilide the palms and soles are almost if not quite invariably affected from the beginning and form very definite sites of predilection. Thirdly, the bulla of *P. neonatorum* arises on apparently healthy skin, whereas that of syphilis arises on a flat infiltrated area. Lastly, there is often a history of contagion in *P. neonatorum*.

In the bullous eruptions of older patients one may be trapped by that produced by iodoform. The idiosyncrasy to this drug is so great that unless one is aware of it one is very liable to be deceived. I was on one occasion asked to see a patient in the wards by a surgical colleague for what was suspected to be acute pemphigus. A boy of about 18 had suddenly developed a rigor, a temperature of 104° F., and a widespread bullous eruption. As a matter of fact he had been taken in for a tuberculous abscess, which had been emptied, scraped, and injected with iodoform, and this was the cause of his eruption, which subsided shortly after the cavity was syringed out and no more iodoform used.

Another case was sent to me with the diagnosis of pemphigus, and this turned out to be a widespread impetigo from which I cultivated a streptococcus in pure culture, and found to have been excited by friction with a strong turpentine liniment.

A third to which I draw special attention was also sent to me as pemphigus of the lower extremities only. This patient was an elderly man with rheumatoid arthritis of the knees, who had developed an acute eruption of large bullæ scattered on the fronts of his legs from the middle of the thigh to the ankle. These arose in the classical way from skin that was not reddened and was apparently healthy. In this case I was able to make the diagnosis by sheer luck, because I had seen a similar case on one leg only in a boy who handled kerosene and had gone about with one leg of his trousers saturated with the oil. On inquiry we found that the old man had been treating his knees by rather widespread rubbing of kerosene. An amusing thing is that when I asked him if he had rubbed in kerosene he denied it, but subsequently admitted that he had used paraffin.

Genuine acute pemphigus is a rapidly fatal disease, and the points to which I would draw attention are, first, the occupation of the patient, who is nearly always engaged in handling something connected with animal flesh, such as meat or raw hides. There is usually also a history of some rather insignificant punctured wound or rather deep cut which has in many cases healed before the outbreak of the eruption. The bullæ first appear in the vicinity of the injury, and become rapidly generalised. The condition of the patient, even early in the disease, is one of severe toxæmia, and the fatal termination of the disease is not long delayed. The nature of the infection is not, in my opinion, established firmly, but a diplococcus described by Demme, Bulloch and others has some claims to causal relationship.

I am not certain whether *Pemphigus vulgaris* and *Dermatitis herpetiformis* should be included under the heading of acute eruptions or not. They are certainly rather chronic in their course, but as they not infrequently come out acutely, I think they should, since when confronted with an erup-

tion which has appeared acutely it is not always easy to prophesy whether the course will be acute or chronic.

If we include them, then there will be three eruptions to distinguish from one another—namely, *Erythema multiforme bullosum*, *Dermatitis herpetiformis*, and *Pemphigus vulgaris*. The differential diagnosis is, in my opinion, important, since *E. bullosum* is a comparatively harmless disease, of limited duration, though liable to become of seasonal recurrence; *Dermatitis herpetiformis* is a torturing disease, but does not often produce grave constitutional effects; and *Pemphigus vulgaris* is, in my experience, an almost invariably fatal disease.

In the early stage we have to make the diagnosis by the lesion and its arrangement alone. In *E. bullosum* the early lesion is a dark red, cedematous papule, which develops more or less quickly into a vesicle, and this vesicle lies between the base of the epidermis and the true skin. It is, therefore, always thick-walled and, at first, pearly in colour. Since slight hæmorrhage frequently follows the first serous exudation, the vesicle tends to become bluish even to a dark plum colour. The area of the primitive lesion extends and then shows a central vesicle or small bulla surrounded by a red, inflammatory circle, and outside this a zone of pallor. With its further development the centre dies down to a dark red, almost black scab, and around this is a bluish circular bulla. It is important to note that this bulla is single and not a rim of discrete vesicles arranged in a circle. The fully developed lesion finally consists of a central dark scab, a bluish pearly rim of bulla round this, a purplish zone of inflammatory thickening further outside, and, lastly, the above-mentioned areola of pallor. This concentric arrangement is, then, a characteristic of *E. bullosum*, whereas it is only rarely found in *Dermatitis herpetiformis*.

In this latter disease the first discernible lesion may be and often is a red papule, but this almost invariably spreads out into an inflammatory circle of much less solidity than is the case with *E. bullosum*, and on the edge of this red area a ring of small vesicles develops on account of which the adjective herpetiformis is applied. It is true that in this disease occasionally large bullæ are formed, arising from apparently healthy skin, but the first form is generally present as well and when it fails the diagnosis is not to be made from *Pemphigus vulgaris*. In pemphigus the lesions are by definition clear bullæ, arising from apparently healthy skin.

The situation of the bulla as regards depth is sometimes of help. In *E. bullosum*, as already stated, it is invariably under the whole epidermis, whereas in both *D. herpetiformis* and *Pemphigus vulgaris* it varies, being sometimes beneath the whole epidermis, but on the whole more generally between the stratum mucosum and the horny layer. The sites of predilection are also sometimes of assistance, though, as I have said before, I hold that generally

in dermatology site of predilection is an unreliable criterion.

All three diseases may attack the mucous membranes of the mouth and eyes, but in *E. bullosum* the chief distribution of the eruption falls on the backs and palms of the hands and similar situations on the feet and the extensor surfaces of the knees and elbows. In *Dermatitis herpetiformis* the eruption may come anywhere, with perhaps a special liability to attack the axillæ and groins, and *Pemphigus vulgaris* has, as far as my experience goes, no favourite sites. Subjectively, *E. bullosum* is usually associated with burning and tingling, and *D. herpetiformis* nearly always with maddening pruritus. Pemphigus may either give rise to a sense of soreness or of itching, though it is fair to admit that some observers classify all pruriginous pemphigoid eruptions with *D. herpetiformis*.

Finally, I should like to refer to two other forms of generalised acute eruption. Dermatologists have generally taken the suffix "ide" as a convenient term to denote that the eruption is of hæmatogenous spread—e.g., syphilide, tuberculide. Of recent years it has been observed on the continent, where the occurrence is fairly common, and also in this country, where it is, I think, rare, that in certain cases of ringworm of the scalp a generalised, non-progressive eruption of minute scaling papules, occurring either singly or in ringed groups, may appear on the trunk. Without going into controversial detail, I may say that the doctrine brought forward is that this eruption is due to the entry of particles of the fungus into the blood stream, and the subsequent deposition therefrom into the skin. Hence it has been named "Trichophytide." It somewhat resembles the eruption commonly called dry seborrhœic eczema of the trunk.

The second form is analogous to this, and was described by me. In certain cases of impetigo of the scalp in children one finds also a similar eruption of minute follicular scaly papules occurring singly or in groups and coming out acutely on the trunk. It is not bullous or even vesicular, and just as the "Trichophytide" does not resemble the original, local inoculation of ringworm of the scalp, in like manner this eruption, secondary to the impetigo, does not resemble the true bullous lesion of inoculated impetigo. By analogy with the "Trichophytide" I have called this the "Streptococcide." It is far from uncommon in England, and the only thing that surprises me is that it had not been described before I called attention to it. The diagnosis in both cases is, of course, simple, provided that due attention is paid to the general examination of the patient's skin.

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STREPTOCOCCI: THEIR TOXINS AND ANTITOXINS.*

BY

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OUR time will be spent mainly in considering the streptococci, and chiefly the pathogenic ones, under the headings of classification and pathology—i.e., what the streptococci are and what they do. It is probably fair to say that the attention of bacteriologists has recently, and more fruitfully, become more centred on what these organisms do than on what they are.

CLASSIFICATION.

In all scientific work accurate classification is of fundamental importance, and many attempts have been made in the past to classify these streptococci by the careful investigations of eminent workers in bacteriology; light has gradually dawned, and we are to-day almost in sight of a rational view of the group.

We may for the sake of clearness take some historical liberties and consider each of these attempts in sequence.

The first attempts were naturally based on morphology from the time when Pasteur drew his historical chain of cocci on the blackboard at a meeting in Paris, and soon such names as *longus* and *brevis* arose. Throughout all this work attempts were constantly being made to link these various groupings with the various diseases produced by the cocci. Thus, *Streptococcus longus* was thought by von Lingelsheim, 1899, to be pathogenic, and the short-chained variety, *brevis*, much less so.

Next, cultural characters were carefully explored and groupings were arranged on the basis of fermentation of carbohydrates, &c; now arose the names *pyogenes*, *faecalis*, &c. This work is largely due to English bacteriologists—Gordon, Andrewes, and Horder (1902–1906).

Blood plates were next (Schottmüller, 1903, Th. Smith and Brown, 1915) extensively used and a division was made into: (1) the non-hæmolytic group; (2) those giving a green ring, the *viridans* group; and finally (3) the hæmolytic group, which produce a clear ring around the colony, a so-called "hæmolysis," though the appearance suggests that there has been also decolorisation. This latter group gives hæmolysis of red blood cells in suspension in a test-tube. It was clearly pointed out by these workers that there was a transition from cocci giving rapid and complete hæmolysis to those with feeble hæmolytic power, but the

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