and its duration being left out of account. The modifications and improvements that it has undergone since it was first put forward diminish, but do not remove, the defect. The wide adoption of this anatomical classification, whether modified or not, seems to me to be responsible for a somewhat mechanical view of the disease, to the comparative disregard of its constitutional aspect.

Trudeau divides his cases into: (1) Incipient, (2) Advanced, (3) Far Advanced. In this classification the combination of local conditions and constitutional disturbance is recognized.

Nearly all the great clinicians—British, American, French, and German—of the past as well as of the present generation have classified cases as Acute and Chronic, with subdivisions based on the pathological condition, the pathological condition corresponding fairly well to the intensity.

Thus in the chief classifications of recent years the extent of the disease, its intensity, and its duration have served as principles of division. Alone, each principle of division can yield only distorted or inadequate results. While, as a joint principle of division, extent and intensity, not running parallel courses in pulmonary tuberculosis, must be forced somewhat in order to yield the rather rigid combination at present in vogue.

In the following classification I believe I have succeeded in allowing full value to each of the three aspects in question—Extent, Intensity, Duration—at the same time preserving their independence. This classification, while fuller and more exact, is also, I think, easier to apply than any previous scheme. In addition, I may say that it embodies a large amount of clinical experience.

The plan is to estimate numerically, according to the definitions presently to be given, the Extent, the Intensity, and the Duration of the disease, from 1, the lowest grade, to 5, the highest, except that the highest grade of Intensity must be taken to range from 5 to 10, so as to include excessively acute or fulminating cases. The sum of these figures will indicate the class in which the case is to be placed.

EXTENT.

Amount of Tubercle in Lung.

- E^1 = Not more than $\frac{1}{8}$ of the volume of one lung, whether in one spot (massive), or distributed throughout both lungs (scattered).
 E^2 = Not more than $\frac{1}{4}$ of the volume of one lung, whether in one spot (massive), or distributed throughout both lungs (scattered).
 E^3 = Not more than $\frac{1}{2}$ of the volume of one lung, whether in one spot (massive), or distributed throughout both lungs (scattered).
 E^4 = Not more than $\frac{3}{4}$ of the volume of one lung, whether in one spot (massive), or distributed throughout both lungs (scattered).
 E^5 = Not more than $\frac{7}{8}$ of the volume of one lung, whether in one spot (massive), or distributed throughout both lungs (scattered).

INTENSITY.

All the conditions named under one heading need not be present at the same time.

- I^1 = Sluggish, with little or no rise of temperature or other general symptoms. Only slight, if any, hæmoptysis. Physical signs of active disease slight, just detectable.
 I^2 = Slight pyrexia and night sweats; moderate loss of appetite and of strength. Physical signs of moderate activity. Bronchial catarrh, if present, slight in amount and restricted to the area of tubercle.
 I^3 = Moderate fever and night sweats; seriously impaired health; loss of strength and of weight. Hæmoptysis, if present, followed by continued pyrexia. Physical signs of softening or of cavity formation. Bronchitis, if present, more marked or more diffused than in I^2 . Recent tubercle, if elsewhere than in lungs, very slight.
 I^4 = Grave constitutional disturbance—more pronounced than in I^3 . Physical signs of greater activity. Bronchitis, if present, more marked or more diffused. Tubercle, if present elsewhere, more active than in I^3 .
 I^{5-10} = Very serious illness. Physical signs more active still. Bronchial catarrh more pronounced or more diffused. Acute or fulminating phthisis.

DURATION.

- D^1 = not over 3 months. D^4 = not over 3 years.
 D^2 = " 6 " D^5 = over 3 years.
 D^3 = " 1 year.

CLASSES.

- I = 3 IV = 10-12
 II = 4-6 V = 13-15
 III = 7-9

EXAMPLES OF CLASSES.

$$\begin{array}{l|l} E^1 + I^1 + D^1 = 3 = \text{I} & E^3 + I^5 + D^4 = 12 = \text{IV} \\ E^2 + I^3 + D^1 = 6 = \text{II} & E^4 + I^4 + D^5 = 13 = \text{V} \\ E^2 + I^3 + D^3 = 8 = \text{III} & E^2 + I^{10} + D^1 = 13 = \text{V} \end{array}$$

Extent, Intensity, and Duration are to be understood as indicating the extent, intensity, and duration at the time of examination. The disease may begin with I^5 and end with I^1 or arrest. Similarly in a case that is undergoing arrest the extent may have been E^5 ; but by the removal or transformation of the diseased tissue, the volume of actual tubercle may have diminished to E^1 . Likewise in regard to duration there may have been a previous attack in which complete healing took place, only fibroid tissue or a cavity remaining as evidence of the former tubercle. In such a case a fresh attack would be marked D^1 for the first three months, and so on.

By this notation the fluctuations in Extent and in Intensity are readily and exactly shown; but a seeming difficulty is presented by the Duration, which would continue adding to the gravity of the case. Within certain limits this corresponds with the clinical facts; but in regressive or healing cases some correction may be needed. The correction is required only when the higher grades, 4 or 5, of Duration coexist with rapidly vanishing Extent and Intensity. The simplest way of making the correction is, I think, by deducting instead of adding a point for the Extent or for the Intensity when the grade of either has fallen to 1 from some higher grade within the previous three months. In the case of D^5 , where both E and I had fallen to E^1 and I^1 within the previous three months, two points would be deducted. This could be expressed as follows: $E^{-1} + I^{-1} + D^5 = 3 = \text{I}$.

A CASE OF ACUTE LUPUS ERYTHEMATOSUS.

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AND

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Not alone the rarity of the following case—Mr. (now Sir) Jonathan Hutchinson, with whom we were able to discuss it, having declared it to be unique in his experience—but also its apparent purity and freedom from complications, and the presence of symptoms not previously recorded, justify the publication of these notes.

A young lady, aged 15, had been out of sorts for some days before medical advice was sought. She complained at that time (the beginning of April, 1908) of headache, loss of appetite, and a little nausea. In a little time these symptoms abated sufficiently to permit her to return to school; but after a few days they reasserted themselves, and she took to her bed. At that time, too, slight evening rises in temperature were noted—from 99° to 99.5° F. Generally a bright, high-spirited girl, she now lay in bed, pale, listless, with eyes closed, but keenly alive to all that was going on around her. The pulse-rate was not increased. She rather shunned the light, and was unusually sensitive to cold. The conjunctivæ were slightly injected. The tongue was moist, furred, broad, indented at the edges, and, what attracted particular attention, was very tremulous. There were no sordes; the lips and gums were pale. The breath was somewhat offensive, and on further examination of the mouth we were surprised to find two ulcers on the hard palate, one on each side of the raphe, each nearly 1 in. in length and $\frac{1}{2}$ to $\frac{3}{4}$ in. wide, dusky red, with irregular, flat edges, and bleeding readily on touch, but not tender. There was a similar but smaller ulcer on the posterior wall of the pharynx. The patient was quite unconscious of their existence, and had made no complaints respecting her mouth. No glands could be felt.

A remarkable appearance was presented by the hands and feet. The skin over the back of the second and third phalanges of each finger and toe, and over the terminal phalanx of each thumb, was thin, as though tightly stretched over the bones, yellowish-white and waxy in appearance. Around each nail this colour changed to a reddish-purple mottling, and the skin was shrivelled. Around the nail of each little finger the skin was very superficially ulcerated, and as though worm-eaten. The palmar surface of each finger-tip was similarly discoloured and shrivelled. On the hypothenar eminence were splashes and dots of purplish-red, not raised above the level of the skin, and a few delicate patches were also seen on the ball of each thumb. The finger-tips were a little tender, the toes distinctly so. The lobes of the ears were slightly swollen, dusky-red, and scaly. These changes were symmetrical, but rather more pronounced on the left side. They had been noted by the mother some four months before, but no special notice was taken of them, the

child having in previous winters suffered from chilblains, for which these patches were, very naturally, mistaken; it was noticed, however, that they were painless on this occasion.

The knee-jerks were faint and very difficult to elicit. No other changes were found indicative of visceral disease. The blood presented the characters of secondary anaemia: red cells, 2,500,000; white cells, 7,000; haemoglobin (Sahli), 35 per cent.; slight poikilocytosis. The Widal test, performed by one of us (F. W. E.), was negative.

During the following week the symptoms persisted with little change. The temperature rose a little higher in the evenings (100° or over), and often failed to reach the normal in the morning. There was at the end of that time a fresh development in the form of a purplish tender swelling over the left malar region, of the size of half a crown, doughy, and the epidermis over it felt thickened and was dry and scaly. The mottling on the finger-tips and toes was more deeply coloured and more extensive; the skin over the backs of the fingers looked thinner and more glossy, and was difficult to pinch up; so also the skin over the front of the tibiae.

Up to this point we made no diagnosis, though we inclined towards lupus erythematosus. Within a couple of days a patch similar to that on the left cheek developed over a corresponding area on the right side, and a connecting streak of purplish discoloration traversed the bridge of the nose, thus completing the bat's-wing arrangement of the eruption. By this time the eyelids had become swollen as well as the nose and cheeks. We now concluded that the case was one of acute lupus erythematosus, and the bad prognosis which attaches to this form of the disease came true only too soon. From that day onward, that is, from the end of the third week, the disease progressed rapidly. The temperature gradually mounted higher, and the rash spread, appearing on the loins in the shape of small, purplish, slightly elevated patches; then on the buttocks, on the back and sides, always arranged more or less symmetrically. The spots on the sides were grouped as though following the lines of the intercostal nerves. The spots on the lateral aspects of the hands began to spread upwards, but the backs of the hands and the centre of the palms remained comparatively free. In a few days the spots became confluent. The patch on the left cheek developed one or two small herpetiform blebs, with clear serous contents. Then, three days before death, that is, on May 9th, the skin began to break superficially, and soon a diffuse haemorrhagic exudation issued, from minute points, from the skin of the back, buttocks, and face. One would have thought that it would be exquisitely painful to be handled while in such a condition; but it appears not to have been so. She complained far more of the least exposure to cold—even of the touch of a cool finger at the wrist—than of any discomfort caused by necessary manipulations. The face finally became an unsightly mass of boggy swellings from which blood-stained fluid trickled; crusts formed in the nostrils and on the lips; blood oozed from the ulcers on the roof of the mouth; and during the last two days of life there was trismus, rendering an efficient cleansing of the mouth impossible. During these last three days the temperature ranged from 105° to 106°, and death ensued—preceded a few hours by a convulsion—on May 12th, one month from the first day of observation. With the exception of a little albuminuria during the last week and of some dilatation of the heart towards the end, there were at no time any symptoms referable to any internal organ. A week before death a further examination of the blood was made, both bacteriologically and histologically, with negative results.

On post-mortem examination nothing was found which could throw the faintest ray of light upon the disease. The spleen, as might be expected, was a little enlarged and pulpy; the liver a little fatty; and there was cloudy swelling of the heart muscle, liver, and kidneys. Though every organ was subjected to a microscopic examination, nothing else was found except a single small calcified mesenteric gland. It is particularly to be noted that there were no other signs of tuberculosis, old or recent. Films were made from every organ, and examined for bacteria and protozoa, but nothing suspicious was found. The epithelial layer of the skin, particularly the rete Malpighii, was markedly thickened in places, and there was a diffuse cellular infiltration of the corium; in other places the rete was unusually thin. The more minute histological changes of the skin could not, however, be studied, as the rest of the specimen was unfortunately lost during a removal.

The diagnosis of acute disseminated lupus erythematosus (Kaposi) seems a justifiable one. The initial discoloration round the finger-tips, so like that of chilblains; the affection of the lobes of the ears; and, later, the bat's-wing arrangement on the face, are highly suggestive, if not characteristic. The principal objection that could be raised against the diagnosis is the development of minute blebs on the malar regions. Bullae are said not to be a feature of the disease. However, Kaposi in his earlier publications refers to the occasional formation of minute bullae and serous exudation, principally over the cheek bones, as in this case; and subsequent observers have now and again noted the same occurrence. The bullae were certainly not associated with erysipelas; the "erysipelas perstans" which Kaposi mentions as being of frequent occurrence in these cases was absent. A very unusual

feature was the ulceration on the hard palate, the rest of the buccal mucous membrane escaping. Trismus has not previously been noted, so far as we know. Very remarkable was the contrast between the objective symptoms and the patient's actual complaints; on the one hand, an acute and diffuse affection of the skin, with serous and haemorrhagic effusions and exfoliation of the horny layer, and ulceration of the hard palate; on the other, only headache, general malaise, photophobia, and acute hyperaesthesia to cold. To this very marked hyperaesthesia we have found no reference elsewhere; it was her constant complaint, not only when the temperature was high, but in the earliest stages. It is worthy of note that the general symptoms were already those of a severe toxæmia at a time when the cutaneous changes were still slight and confined to the fingers, toes, and ears. These toxic symptoms steadily increased in severity, though the patient never lapsed into the typhoid state.

The pathology of the disease is still unknown. Tuberculosis is still considered by some to be an important factor. Our case, however, does not support this view; the family history showed no tendency to tuberculous infection, and there was no sign of tuberculosis of any organ; the one calcified mesenteric gland may safely be discarded. That the process must be an acute infective one no one can doubt who has watched such a case, especially when neither erysipelas nor pneumonia (the most frequent complications of the disease) have blurred the picture.

As for treatment—everything conceivable was tried in turn—beta-naphthol, quinine, mercury by the mouth, atoxyl injections in the later stages, ointment and plasters, prolonged warm baths, and lotions. The only conviction to which we came was that ointments were badly borne, and that lead lotion alone gave the slightest relief.

A CASE OF HENOCH'S PURPURA.

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OWING to the rarity of Henoch's purpura, the following case appears to be worthy of record. It was characterized by the following symptoms: (1) Swelling and oedema around joints; (2) purpura; (3) abdominal colic; (4) vomiting; (5) oedema of lax tissues; (6) ? subperiosteal haemorrhages; (7) haemorrhage into the lungs; (8) albuminuria; (9) haemorrhages from the bowel; (10) slight haematemesis, with large quantities of phosphates in the urine.

N. J., male, aged 5 years, had an attack of epigastric pain on November 1st, 1908. He was given some calomel, and the pain was relieved. He then developed a slight cold.

On November 4th there was some swelling of the right ankle and elbow and left hand. There was oedema over the joints; but no effusion into them, and no pain on movement. There were a few scratches and papules around the swollen areas, but none of these were purpuric. The boy seemed well. There was no rise in temperature and no increase in the pulse-rate. The condition seemed unusual, but not significant of any definite disease, and I ascribed it to an irregular urticarial

state, with some swelling of the subcutaneous tissues. In the evening there was definite swelling of the joints of the hands; of both knees and of the left ankle-joint with pain on movement. There were definite purpuric spots on the legs. There was a heavy deposit of phosphates on boiling the urine.

On November 5th abdominal pain, referred to the umbilicus, occurred. This gradually became worse, until it was so severe that the child sat doubled up and screamed with pain.