expelled from the liver, for the blood corpuscles in the vessels are not destroyed, while they usually are dissolved by the bile.

This subject is one of the greatest possible practical interest, because its investigation may explain the causation of those very common and very troublesome symptoms to which the term

Fig. 4.—Action of pilocarpin on a fasting liver. Rabbit 6 weeks old. Last food, 17½ hours before death. Injection of ½ gr. pilocarpin 1½ hour before death.

“biliousness” is commonly given, and which are usually ascribed to a “torpid liver.” Experiments on the effects of nitrogenous and non-nitrogenous foods on the liver cells are required in order to give us a firm physiological basis; and then a study of the

Fig. 5.—Action of toluene on fasting liver. Rabbit 6 weeks old. Last food, 20 hours before death. Toluene, 1 minim, 4½ hours before death.

action of various substances belonging to the aromatic and ammoniacal groups may enable us to discover what products of albuminous decomposition in the intestine lead to hepatic congestion, “torpid liver,” and “biliousness.” At the same time, we may

hope that such an investigation will also enable us to find efficient remedies for these conditions. At all events, I think it is evident that the whole subject of hepatic stimulants must be studied anew, not by the older method of trying to ascertain their action on the organ as a whole, but rather on the cells which compose it. As I mentioned in my first lecture, the latest development of pharmacology is the “pharmacology of the cell;” and to it we must look for the fullest information regarding the action of remedies, whatever be the class to which they belong.

Fig. 6.—Action of toluene on active liver. Rabbit 6 weeks old. Last food, 6.30 hours before death. Toluene, 1 minim, 5 hours before death.

Time will not allow me to enter upon the relations between the chemical structure of remedies and their action upon other glands than the liver, and especially upon the kidneys. Nor can I even flatter myself that I have treated any part of my subject fully. All I have done is to attempt to give a brief outline of it, not discussing it with a view to completeness, nor always bringing prominently forward those points which would be most interesting to a chemist, but rather endeavouring to bear in mind the practical intention of these lectures, and to show as much as possible the relationship of my subject to the prevention, control, and cure of disease. In trying to do this, I have thought it advisable to look forward as well as backward, and to indicate lines of research on which work is desirable, even at the expense of time which might have been given to a fuller exposition of results already obtained. In conclusion, I must again return you my most cordial thanks, not only for the honour you have done me in selecting me to give these lectures, but for the patient and kindly hearing you have given them.

NOTES OF THREE CASES OF XERODERMA PIGMENTOSUM, OR DERMATOSIS KAPOSÍ.

By W. BROWN HUNTER, M.D.,
Londonderry.

In the number of the JOURNAL for June 8th there is a typical case of this disease well recorded by Dr. McCall Anderson. In a note to that report it is said that there are only forty-one cases of this disease recorded. I write this report chiefly for the purpose of adding three more cases to that record, two of which are certainly typical, and the third I have not seen, but for reasons given below its identity is undoubted. I enclose photographs of the two I have seen, and the third case was a brother of these, who is now dead. The family in which these cases occurred consists of eight children altogether, two of whom are dead, one a girl at the age of 2½ years, who was unaffected with the disease, and a boy aged 18 when he died, who was the eldest of the family

and was affected. The other six children are alive—three boys and three girls. The girls' ages are 19, 7, and 5 respectively, and are all quite free of the disease. The boy who is unaffected is 12 years and 6 months. I mention the ages of these so that it may be seen that they have got past the age safely at which the disease generally, though not invariably, begins to be noticed. It will also be observed that in this family it has as yet obeyed the general law of keeping to the sex first attacked. The family, too, are agricultural, and much exposed to all weathers. The parents are well, and have always been healthy, and no members of the family on either side have ever been known to be affected by any such disease as this. The mother also mentions that the parts affected seem to get worse in the summer and harvest, and clear up a little again during the winter.

I shall now give a short description of each case:—

CASE I.—H. J. C., aged 18 when he died. He was the eldest in this family, and is said to have died of "a wasting away"—most probably phthisis, as he had a cough, and, near his end, diarrhoea. He is said, by parents, relatives, photographer who also took his likeness as well as those I now send, and by everyone who saw him, that he was affected in the same way as his two brothers now living are, that at an early age the freckles were noticed, and the disease assumed all the appearances which we see in these living cases, so that there can be no doubt of the identity of the affection.

CASE II.—C. C., aged 17, health good in every respect. At the

CASE II.

age of 4 freckles numerous and dark-coloured were noticed coming out on his face, and since that time to the present, his face, neck, hands, forearms up to elbows, have been affected. On examining the legs and feet they are also found to be affected, though not so observable, and were said not to be affected. I first saw him

about three years ago, observing the peculiar discoloration of his face. About two years ago he came to me complaining of a sore on his right cheek. This sore was almost as large as a shilling, lupus-like in appearance, with large flabby granulations on surface, edges slightly raised and quite round. His mother says it arose out of a pimple being opened by a knife and grew rapidly. I burned it with strong nitric acid, and afterwards treated it with a powder of boracic acid and iodoform. It healed completely, leaving a cicatrix with a black spot in its centre. This is the only ulcer which has as yet formed. His face presents now—first, the freckles, some larger than others, and measuring in longest diameter 4 millimètres; secondly, the small hard nodular bodies; thirdly, a few red small vascular spots (telangiectasis); and, fourthly, the white cicatricial-like spots, which vary in size. These are more marked over the bridge of the nose and below the eyes; in fact, nearly all the surface in these localities is thus affected. In consequence, I presume, he suffers also from ectropion and some conjunctivitis. His lips are also swollen and prominent, especially the upper, rough on the surface, and a hard scab is formed on the upper at one spot. There is a small black spot the size of a split-pea on the forehead and one or two on the cheeks, but smaller.

CASE III.—J. C., aged 10, in good health. Disease first noticed at the age of 5. The freckles in this case are very abundant and the pigmentation very noticeable. Same parts affected as those of brother. Feet and legs still less marked. It may be remarked here that these children went to school during the summer months without shoes or stockings and the trousers often rolled up. The latter is done so as not to interfere with running or jumping, and in wet weather to keep them (the trousers) clean. This is a com-

CASE III.

mon habit in the country districts, and thus, in these cases, the feet and legs which are affected were also exposed. His face also presents the small nodules, a good number of the telangiectases, and some of them raised. There is only one cicatricial-like spot, and this is on the middle of the nose, where the skin looks whitish and thinned. He has also some conjunctivitis, but no ectropion.

On the lower left eyelid there is a hard warty growth, the size of a pea, chalazion-like, but wants the characteristics of the latter. The boy who died had a similar growth, the mother now tells me.

ON LUMBAR HERNIA.

By J. HUTCHINSON, JUN., F.R.C.S.

It will be admitted that any contribution to our knowledge of this comparatively rare form of hernia is worthy of record, since there are several points about it which are still doubtful. It is generally assumed that the small triangular space bounded by the external oblique and latissimus dorsi muscles, and the crest of the ilium (Petit's triangle), constitutes a relatively weak spot in the abdominal wall, that hernial protrusion may here occur, but that strangulation of the contained intestine is very unlikely to develop. Indeed, almost the only record of an operation for strangulated hernia in this region which we possess dates from as far back as 1738 (Ravaton's case), since in Mr. Edmund Owen's case¹ the rupture was neither strangulated nor obstructed. To Mr. Owen, so far as I can ascertain, belongs the credit of having performed the first operation for radical cure of a lumbar hernia. There is singularly little evidence as to the anatomy of this form, and what there is hardly confirms the current view that the presence of Petit's triangle accounts for the development of the hernial protrusion. No specimen had hitherto been shown at the Pathological Society; and I believe the one which I was able to exhibit there a few weeks ago, through the kindness of Dr. Stephen Mackenzie, is the only dissection of a lumbar hernia in existence in London. It was obtained from an elderly and rather emaciated man, who died (from other causes) in the London Hospital; the hernia was about the size of one's fist, was situated in the left lumbar region lying over Petit's triangle, and extending nearly from the last rib to the iliac crest, and it had existed for some years. It could be made to markedly diminish on pressure, was resonant to percussion, and had an impulse when the patient coughed. Gurgling could be detected when reduction was made, the swelling returning when the pressure was taken off. It caused the patient no pain, and hardly any inconvenience. I was naturally inclined to think that the hernia protruded through the orthodox triangle, but found, on dissection, that this was not the case, as the aperture, which would admit two fingers and was circular in shape, was really situated above and to the inner side of the triangle. The protrusion was, in fact, just outside the quadratus lumborum, through the transversalis aponeurosis and the latissimus dorsi, where the latter arises from the strong fascia covering the erector spinæ. Another interesting feature at the *post-mortem* examina-

a, latissimus dorsi muscle; b, external oblique; c, Petit's triangle. The dotted circle in the hernia indicates the relative size of the aperture and its position with regard to Petit's triangle.

tion was the absence of any peritoneal sac. The looseness of the attachments of the peritoneum in this region is well known, and

is taken advantage of in certain operations, such as removing the kidney through a lateral incision. It was quite easy to make the peritoneum, by gentle pressure, protrude into the centre of the hernial tumour, and, as it was certain that it had habitually during life contained intestine, it is obvious that every time the latter was reduced the peritoneal sac must have returned with it into the abdominal cavity. Whether this feature has been present in other cases of lumbar hernia it is impossible to decide, but it is one rarely met with in connection with the more common varieties of rupture.

What part of the intestine was present during life in the hernia I was unable to decide; the descending colon lay just to the inner side of the aperture, but it is quite probable that some coils of small intestine really protruded. The fact that this can occur is proved by Ravaton's case (No. 3 in the appended list), and that omentum may form part of the contents was also demonstrated in Baron Larrey's case (No. 17). But it is no doubt true, as suggested by several writers on the subject, that the colon (ascending when the hernia is on the right side, descending when on the left) is the most usual part of the abdominal contents to protrude in this region.

With regard to the coverings of a lumbar hernia it might be expected that the outer part of the sac would contain some part of the internal oblique and the transversalis aponeurosis, for the muscular fibres of the latter cease externally to the usual site of the hernia. But such little evidence as we possess on the point does not confirm this view, for in the cases in which an incision has been made there is no mention of any muscular fibres being cut through. In my case the sac was really formed by a local hypertrophy of the subperitoneal fat which had insinuated itself through the aponeurosis and muscles, and had thus come to form a tumour the size of an orange in the subcutaneous tissue. I have demonstrated the mode of development of fatty herniæ in the femoral, inguinal, and umbilical regions by a series of preparations shown at the Pathological Society.² It is certain that this mode of origin of hernia (the protrusion of subperitoneal fat followed by the descent of a peritoneal sac, and sometimes of intestine) is more common than one would be led to suppose from the ordinary textbook accounts, and it was especially interesting to me to find the same thing occurring in the lumbar region. It is probable that the cases reported by Marmisse, Auzias-Turenne, Gosselin, and Marquez (Nos. 11, 15, 18, and 20) were of the same nature. Out of the twenty-nine cases of which I have abbreviated the records, sixteen (over 50 per cent.) developed spontaneously, or were attributed to strain, and all of them were in adults or elderly subjects. Males and females appear to be equally liable to lumbar hernia; seven cases of this class (of apparently spontaneous origin) were on the left side, four on the right.

In six cases (about 20 per cent.) the hernia followed in the track of a previous abscess or sinus, four in males and two in females. An ingenious suggestion has been made that the path which the protrusion takes may be determined by the course of a nerve, especially perhaps the lowest posterior lumbar branch, and this may apply to the original abscess, which leads later on to the hernia.

In five cases the hernia was due to wound or other severe traumatism of the loin, and two were said to be congenital. About the origin of the last form of lumbar hernia we are absolutely in the dark, for there is nothing in the development of the abdominal wall that explains the formation of a gap in the lumbar region.

For practical purposes we may say that the spontaneous form of lumbar hernia is confined to the period of adult life or old age, whilst that due to abscess or traumatism may occur much earlier.

It is quite erroneous to attribute to M. Petit the first account of lumbar hernia. It was mentioned by Barbet so long ago as 1650, and as seen in the list appended, several cases were reported before M. Petit's very imperfect record was published in 1783; nor, as already mentioned, is there strong evidence that in the majority of cases the protrusion occurs through the triangle named after the French surgeon. Mr. Edmund Owen states that in the child on whom he successfully performed a radical cure the protrusion was at this site; but in the two other cases, which one can feel certain on the point (Braun's, No. 27, and my own), the triangle was not the seat of the hernia; in the former case it did not exist. As is well known, the latissimus dorsi and external oblique may be in close apposition at their attachments to the iliac crest; and Lesshaft, by examination of a considerable

¹ Reported in the JOURNAL for May 5th, 1888.

² Transactions for 1886 and 1887.