not appear to hear. There was also a weak infrequent cough.

On the following day (the 26th) he had perfect movement in the respiratory muscles, though the breathing was slow, and there was a little wheezing on the left side. Temperature subnormal.

Nov. 27th.—Respiration 16; pulse 52; temperature 96°. Abdominal respiration nearly absent; tactile vibrations present on the abdomen, and to a less extent on the chest; knee-jerks weak; ankle-clonus present; posterior muscles of the legs rigid.

28th.—The patient tells me he was last week doing lead casting in the course of his employment, and the day before his attack was mixing red and white lead. At tea on the 29th he had shrimps, and it is probable that the action of the finger-tips in picking the shells off worked the lead from under the nails on to the fish, and, thus seasoned, he swallowed them, as he subsequently found lead under the nails. The gums showed the lead line well marked, and there was apparently no other source but the lead under the nails for the poison, as the lead was not touched in the process of casting or afterwards, nor was any other person in the house affected.

The question remains, what was the immediate cause of the paralysis and of its rapid disappearance? Shock alone would not account for it, because the pupils were not dilated, as is always the case, I believe; nor was the cutaneous circulation altered, nor would the small dose of morphia have had such widespread and permanent effect on the sympathetic. The course the pain took was that of the symptoms of lead poisoning and of lead poisoning. The symptoms and the result of the treatment confirm this view. To take the last first. The improvement in respiration and pulse can be accounted for if, looking upon the case as one of inhibition of the respiratory centre by the vagus, we remember that atropine injected in even the smallest doses has the power of preventing this inhibitory action of the vagus. Indeed, this was what first gave me a clue to a rational explanation of the phenomena. The inhibition was no doubt set up by excessive stimulation of the gastric terminal branches transmitted to the central ending of the nerve by its main trunks, and through the respiratory centre could have caused the slowing of the heart; this was very considerable, if the rate of the breathing is compared with it, the usual proportions being reversed. The temporary paralysis of the palate might be accounted for by regarding it as part of the respiratory system. The known unexcitability of the branches of the vagus might account for the absence of change in the voice. The continuance of action of the phrenic nerve on the right side can only be accounted for by supposing that the respiratory centre was unequally affected. I forgot to say that soon after the pain ceased the urine was passed; a later sample showed merely excess of phosphates and urea. The amount was slightly in excess of the normal quantity. The bowels were confined on the day following the attack.

My remarks are based on the third edition of Landois and Stirling and on Foster's text-books.

CLINICAL NOTES.

MEDICAL, SURGICAL, OBSTETRICAL, AND THERAPEUTICAL.

NOTE ON A POINT OF DIFFERENCE IN THE PATHOLOGY OF GOUTY AND RHEUMATOID ARTHRITIS.

By E. T. Wynne, M.B. Cantab., House Physician, St. Bartholomew's Hospital.

That both these diseases produce deformities of joints is well known, and the deformities are described as due in the one case to deposition of urates, in the other to changes in the hard tissues of the joint. But there is a change in the bony structures of joints met with in many cases of articular gout which at first sight would seem to bring these two diseases (formerly confounded, now clearly separated) into closer connexion with each other, but which I hope by my description to show really marks a new, and, if my interpretation of the condition noted a rare condition between them. This change has been recognised and described before, but its microscopic characters have, I believe, never been carefully investigated. Cornil and Ranvier mention it roughly in their work on Pathology, but their description does not correspond entirely with what I have found. The condition is best seen in the knee. On examining such a knee through the skin, the margins of the articular surfaces are felt to be more protuberant than usual, and opening the joint the articular cartilage appears to have been softened by pressure, so as to produce a "lip" or ridge around the margin. This lipping has a superficial resemblance to the condition seen in early rhematoid arthritis, often described as a heaping up of the cartilage at the margin. A closer examination shows radical differences. In rheumatoid arthritis such an outgrowth is found microscopically to consist of cartilage in a state of fibrillation, which may in parts be calcified, but which rarely shows ossification. I have examined microscopic specimens of extreme cases showing ossification; but the outgrowth is found even in the most advanced cases to have a complete investment of cartilage. The outgrowth is therefore a chondroma produced by overgrowth of the cancellous tissue of the epiphysis, carried by the cervical spine of the capsule of the joint. In gouty arthritis, the opalescent cartilage is seen to stop short at the summit of the outgrowth, and microscopic sections show the same. Such a section taken at the edge of the joint shows the cartilage healthy, except for minute changes, being 96°. To take the last first. The improvement in respiration and pulse can be accounted for if, looking upon the case as one of inhibition of the respiratory centre by the vagus, we remember that atropine injected in even the smallest doses has the power of preventing this inhibitory action of the vagus. Indeed, this was what first gave me a clue to a rational explanation of the phenomena. The inhibition was no doubt set up by excessive stimulation of the gastric terminal branches transmitted to the central ending of the nerve by its main trunks, and through the respiratory centre could have caused the slowing of the heart; this was very considerable, if the rate of the breathing is compared with it, the usual proportions being reversed. The temporary paralysis of the palate might be accounted for by regarding it as part of the respiratory system. The known unexcitability of the branches of the vagus might account for the absence of change in the voice. The continuance of action of the phrenic nerve on the right side can only be accounted for by supposing that the respiratory centre was unequally affected. I forgot to say that soon after the pain ceased the urine was passed; a later sample showed merely excess of phosphates and urea. The amount was slightly in excess of the normal quantity. The bowels were confined on the day following the attack.

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