most apparent in the two upper interspaces near the sternum, and has been termed the dimpling of the hilum.⁴ At the same time the scalmi and sterno-mastoid of the same side—incidentally of both sides—may be seen to contract. Simultaneously, with care, a unilateral hardening of the rectus and obliqui may be felt. This sign may be suggested as a test for the determination of the presence of incomplete expansion of the lung. A sudden rigidity of the intercostals may also occur (Pottenger's sign of early phthisis). In conditions of insufficient pulmonary dilatation there appears to be a struggle between opposing muscular groups—the sterno-mastoid, scalene, external intercostals and diaphragm on the one hand, and the rectus, obliqui, and internal intercostals on the other.

9. In hilum phthisis some impairment of expansion exists at least in the terminal areas supplied by the three chief upper bronchi. These should correspond to the inner and outer ends of the superior interspaces anteriorly and to the apex of the lower lobe behind at the fourth rib, near the spine of the scapula. In ordinary and in stereoscopic radiograms we have been able to trace conspicuous linear shadows to these three areas.⁵ In addition to venous and lymphatic stasis induced by the pressure of enlarged glands (often the right tracheo-bronchial group) on vessels at the root of the lung, there may be a condition of partial atelectasis in these situations produced by pressure on the bronchial wall. Before the absolute termination of a deep inspiratory movement, when the limit of expansion is prematurely reached, the auxiliary inspiratory muscles are called into activity, but their contraction will be readily overcome by the auxiliary expiratory muscles, since the inspiratory impulse is now on the wane and rapidly approaching its normal conclusion. In other words, if mechanical conditions forbid further dilatation, the expiratory muscles contract forcibly. The resistance of the chest to further expansion probably excites end organs within the tendons of the inspiratory muscles, the effect of which leads to the reflex contraction of the recti, obliqui, and others. Moreover, in a similar manner, a very small difference of pressure within and without the lungs renders inspiration impossible.⁶ Dr. Steuart, late chief assistant in the x-ray department at St. Bartholomew's Hospital, found in some cases, which exhibit the sign, if the breath be held for a few seconds after a deep expiration, the movement of the diaphragm in the succeeding inspiration is apparently normal.⁷ The explanation appears to be that during the longer pause the blood within the centre itself becomes increasingly loaded with carbon dioxide, so that the next inspiratory discharge becomes excessive. Reinforced by the inspiratory reflex from the pulmonary vagus itself this is able, under suitable circumstances, to overcome any expiratory muscles which might otherwise prove efficacious in curtailing the diaphragmatic movement. If the experiment fails, a real mechanical hindrance to full dilatation is present.

10. It has been stated that when the locus of disease is situated posteriorly, the diminished excursion is more evident in the dorsal half of the arch.⁸ In other words, the spinal part is affected more than the sterno-costal. The muscles which oppose the crural fibres may be the deeper muscles of the back and possibly the quadratus. In any case, the effect of this reflex must be a reduction in duration and amplitude of the ordinary inspiratory wave, and its object the attainment of a definite measure of rest and protection for the diseased area of the lung.

NOTE.—In this communication the occurrence of an early expiratory reflex (proceeding from nerve-endings of the vagus in alveoli or in unstriated muscle in the manner described) is not sufficiently emphasized. The necessary condition is the presence of diseased and hypersensitive areas within the lung. Actual proof might be afforded by the determination of the time relations of the antagonistic muscles by means of tambours and a revolving drum. When the loci become quiescent, the second and later expiratory reflex, under suitable conditions, would still persist.

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BETA-NAPHTHOL POISONING OCCURRING DURING THE TREATMENT OF ANKYLOSTOMIASIS.

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AN obvious instance of this complication has recently been the subject of inquiry at Sandakan, British North Borneo. As it is only one of a series coming under the writer's notice, the time is apparently ripe for a word of warning.

Beta-naphthol was introduced at a much later date than thymol and eucalyptus, Nicol probably being one of the first to use it on a large scale in Natal (*Transvaal Medical Journal*, June, 1910). Beta-naphthol was particularly welcomed as by reputation it was safe, and in 1911 it was common to see it exhibited in the Federated Malay States in three doses of 20 grains each, preceded and followed by Epsom salts. Among thousands of cases treated with these doses, in the writer's memory only one fatal case occurred. Of late, however, it has become the fashion to use it in enhanced doses, three portions of 30 grains each being quite usual, and even larger being exhibited by some. We are inclined to deprecate such large doses as a routine administered by dressers in native hospitals. In the fatal case about to be recorded the medical officer, had he been at hand and able to see the patient two or three times daily, would have reduced the dose. I may add that two other fatal cases have come under my notice, though, unfortunately, notes were not recorded; both resembled the present case very closely.

L. T., a Chinaman, 28 years of age, was admitted on August 26th, 1914, into the Gaol Hospital, Sandakan, for a trivial abscess on the knee, and being somewhat anaemic his faeces were examined for ova, the result being recorded as follows: Ova of *Ankylostoma*, of *Clonorchis*, and of *Trichuris trichiura*. On the same evening 5 grains of calomel were given. Half an ounce of Epsom salts was administered two days later, and on August 29th 30 grains of beta-naphthol were taken at 7 a.m. and at 9 a.m., and again at 11 a.m. At 1 p.m. the patient took Epsom salts, and the resulting stool contained seven ankylostomata.

The treatment was repeated on August 30th. When the dresser visited the hospital at 7 a.m. on August 31st the patient was given his first dose of the beta-naphthol mixture, but as he began to vomit shortly afterwards the second and third doses were not administered. Whenever the gaol dresser has finished his morning duty he leaves, in order to take up the non-working hours at the Sandakan Civil Hospital—namely, 11 a.m. to 2 p.m. and 4 p.m. to 7 p.m. This being the case, second and third doses are left in charge of an attendant, who in this instance did quite correctly in not giving the second and third doses, but unhappily did not mention this fact to the medical officer nor call his attention in any way to the patient.

At 6 p.m. another dresser was at the Gaol Hospital and recorded the patient's temperature as 102.8° F. On the morning of September 1st the fever had fallen to 99° F. Vomiting continued, the urine became extremely dark in colour, and slight jaundice showed itself. In the morning the medical officer found the patient in so collapsed a condition that $\frac{1}{10}$ grain of strychnine was ordered at 11 a.m., 4 p.m., and 9 p.m. The temperature in the evening, however, again rose to 101° F., and in the early hours on September 2nd the patient died.

On the bed head ticket being examined, it was found that, as usual, before such treatment the urine had been tested, the record being: Specific gravity 1010, acid, cloud of albumin.

It is felt that the presence of albumin is of paramount significance, as it was also found in the other fatal cases mentioned above.

At the post-mortem examination it was found that the liver weighed 3 lb. 5 oz., was deep yellow in colour, and contained numerous *Clonorchis sinensis*. The right and left kidneys weighed respectively 4 and 5 oz. The capsule peeled off without tearing the tissue beneath. The most characteristic feature was the colour of the cortex on section. It had much of the aspect of the liver itself, though it was less deeply stained and was rather narrow. The cut surface was quite dry, and appeared somewhat fatty. A sample of urine which was removed after death

was highly albuminous, very dark, partly owing to bile pigments, but partly no doubt to the beta-naphthol. Microscopic examination showed a quantity of debris and a few scattered red blood cells. No spectroscopic examination was made.

Conclusion.

These cases teach us that in individuals suffering from diseases of the kidneys beta-naphthol should be used with the utmost caution, if at all. Probably it would also be wise to go back to the old Egyptian rule of leaving an interval of a full week between any two treatments.

Dr. J. E. A. Ferguson of British Guiana administers to patients with ankylostomiasis 10 grains of thymol in a cachet every night for many weeks, and finds that it cures the disease completely without any ill effects on the kidneys. Perhaps other physicians who have had experience in this thymol treatment may publish their results in the JOURNAL, especially noting its effect on kidneys.

One more observation, which may be interesting to those in charge of hospitals in the tropics, is that, in the writer's experience, whereas in *Necator* infection one treatment is usually sufficient to dislodge all the worms, in infection with *A. duodenale* three are generally necessary. Can it be that the more formidable mouth parts of the latter are responsible for this difference?

A FURTHER CONTRIBUTION TO THE DIAGNOSIS OF GOUT.

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A PAINFUL inflammatory swelling of the big toe, apparently of constitutional origin, or pain only, which soon after the consumption of a glass of generous wine is felt in a small bone of the hand or the foot, is commonly regarded as pathognomonic of gout. If that term is meant to denote a specific pathological process, of which a deposition of urates is supposed to be the exciting cause of its clinical manifestation, then its indefinite application to all cases presenting such symptoms is not only misleading, but, for more reasons than one, mischievous also in its consequences. For there are numerous cases called gout in which, when they happen to come to *post-mortem* examination, not a trace of a uratic deposit can be found. If, nevertheless, the clinical diagnosis of them is upheld, as is frequently done, it is on the alleged ground that the urates have been absorbed. This explanation would have some validity if it were conclusively established that the urates had been previously deposited. As it is, there is nothing to support it but the tacit yet unfounded assumption that the region above referred to has a singular immunity from other pathological processes except the one connected with uric acid. There is proof, however, of its inadequacy; for whether a deposit of urates acts as a mechanical irritant or as a toxic agent, invariably it leaves behind it indelible changes in the articular cartilage in which it has taken place; whereas, in the unanimous opinion of those who advanced it, there are numerous instances in which the articular cartilage appears on *post-mortem* examination to be perfectly normal.

The fact is that there is a pathological process of frequent occurrence which gives rise to clinical phenomena closely similar to those commonly called a gouty paroxysm, but which is not due to uric acid. The habit of looking for the cause of the painful swelling to the surface of the joint has diverted attention from its other constituents. Yet if the corresponding bone be carefully examined, it will be found that very often take place within it, silently and almost imperceptibly, changes which in their last stages fully account for those symptoms. Such examination discloses in the first metatarsals, and occasionally also in the phalanges, a cystoid degeneration, which starts in the epiphysis, extends to the articular cartilage, and after perforating it allows the discharge of the cystic contents into the joint, thus producing an acute "perforative synovitis." The resulting fistulous openings are generally minute (Figs. 1, *a*; 3, *a*), and readily escape detection, as do the cysts themselves so long as

they are of small size and concealed by the fat marrow; but when the bone is fully prepared for microscopic examination they are easily recognized, and are found to be either isolated in their earlier stages (Fig. 1, *b*) or

Fig. 1.—Section through the subchondral portion of a first metatarsal bone. *a*, Fistulous opening through the articular cartilage; *b*, softening of connective tissue and formation of cysts; *c*, *c*, *c*, areas of fibroid tissue; *d*, transverse section of an artery.

multiple (Fig. 2, *a*), and gradually coalesce so as to form a macroscopic excavation (Fig. 3, *b*).

Of the contents of those cysts little is definitely known, as they rarely, except in accidents or surgical operations, come under anatomical observation. They appear to consist, when of comparatively recent origin, of a coagulable substance (Fig. 2, *b*), which later on softens and becomes serous or haemorrhagic. So long as the canal thus formed remains pervious, or when its calibre becomes enlarged, the necrotic matter periodically passes into the joint and gives rise to periodic attacks of synovitis; only in the cases which are not here under consideration, where the cysts are situated near or within the diaphysis without reaching to and perforating the cartilage, it accumulates within the bone, which then is gradually expanded by its pressure.

Fig. 2.—Section through the epiphysis of a first metatarsal bone, of which the articular cartilage was worn off. *a*, *a*, *a*, Cysts; *b*, *b*, coagulated substance; *c*, *c*, fibrous tissue.

From the histological details of the affected epiphyses it may with safety be inferred that this cystoid degeneration originates from an anomaly of the vascular and osseous systems. Conspicuous amongst them is the abundance of dilated and thin-walled veins which are here and there studded with emigrated blood corpuscles.

There is consequently evidence of a state of chronic congestion under the influence of which the trabeculae are decalcified and the adjoining fat marrow becomes fibrous. From both these sources are formed patches of fibrous