### A New Use of Atropine.

Sir,—We often find it necessary to advise patients not to read, or write, or do any work requiring close attention. In many cases, the patients are equally ready to promise obedience, and to break the promise under slight temptation. Under these circumstances, we may fairly make the necessity for careful ophthalmoscopic examination (always desirable in these cases) an excuse for crippling the power of accommodation by a tolerably strong solution of atropine; the instillation to be repeated as often as necessary. I generally use homatropine to dilate the pupil for merely diagnostic purposes, because the effect of it passes off in a few hours. But in such cases as I have alluded to, atropine is preferable. Of course, this suggestion is obvious enough; but it may be none the less useful for that.—I am, etc., Frederic C. Coley, M.D.

21, Eldon Square, Newcastle-on-Tyne.

### Death After Injury: Fat Embolism (?)

Sir,—I shall feel obliged if any of your readers can tell me about the following case.

A carter, aged 50, of intemperate habits, was knocked down and run over by his cart, which, with the load at the time, weighed about a ton and a quarter. His right femur was fractured comminutely, the tibia on the same side obliquely (his leg being bent on the thigh at the time of the accident). The fracture of the femur was simple, that of the tibia compound. Chloroform was given, and the limb fixed up with short and long splints, the wound over the tibia being carefully washed out with carbolic oil (1 in 8). For the next thirty-six hours, he did well, but had no sleep, being afraid, he said, of moving the leg or splints. About this time, he fell into a sort of stupor; and when I saw him six hours later (forty-two hours after the accident), he was in a state of profound coma. His urine, which was drawn off, was free from albumen and otherwise normal, except that it was rather scanty. His bowels had not moved. Croton-oil and pilocarpin were tried with little effect, and he died, ninety-six hours after coma set in (or 132 hours after the accident). I suspect "fat-embolism" as the cause of coma and death, and will be glad of any information on the subject.—I am, etc., C. J. R. M.

### Swollen Mouth after Eating Pine-Apple.

Sir,—"Pelobates" will find, I think, that his patient, while "carefully peeling the pine-apple," cut the fruit with the same knife, thereby leaving some of the rind's juice on the parts he eat. In Queensland and the West Indies, the rind-juice is well known to cause sore lips, the only cure for which is salt.—I am, etc., Francis O. Hodson, Fellow of the Royal Colonial Institute.

The Chantry, Bishop Stortford.

### Question of Treatment.

Sir,—I shall feel obliged if any of your readers will give me some suggestions as to the best way to treat a case of uric acid diathesis, medicinally and dietetically. Their is no history of either gout or rheumatism in the case. The patient's age is 40 years. The attack has lasted about five months, and there is a copious deposit of uric acid on the urine cooling. Medicines, alkalies, and lithia seem to do very little good.—Faithfully yours, L.R.C.P.

### Bacteriotherapy.

Sir,—Since, in a recent number of the Journal, reference has again been made to Cantani's ingenious method of treatment, let me mention some conclusions arrived at as regards the occurrence of tubercular bacilli, after examining a large number of cases of chest-disease during the last twelve months, and, in a certain proportion of cases, comparing the clinical with the *post mortem* results. Gibbes's double stain was used in all cases.

1. The absence of tubercular bacilli from any sputum, when tested on several different occasions, is no proof that such bacilli do not exist in the lung. For, unless there be considerable disintegration of pulmonary tissue and free communication with a bronchus, the tuberculous *débris* may not be expectorated. Even a comparatively large cavity may be formed in the extreme apex, and it may, *post mortem*, furnish tubercular bacilli in great abundance, yet during life the cavity may never have been evacuated; clinically, the only signs may have been dulness and a few large *râles;* and the bacilli may never have been present in the sputa. This is, doubtless, due to the situation, to the loss of elasticity in the pulmonary tissue, and to the frequent cicatricial contractions. A similar retention of tuberculous matter in the lower portions of the lung (where the expulsive efforts of coughing take greater effect) may be met with in the insane, in cases of advanced phthisis unaccompanied by cough.

2. In some cases of phthisis, in which there is a large hollow cavity (for example, in the intraclavicular region) and profuse purulent expectoration, no microphytes of any kind may be present. So is it also in some typical cases of catarrhal pneumonia, and as yet I have not found microphytes in cases of chronic inflammatory congestion with fibrinous sputa, or in cases of chronic bronchitis with excessive mucopurulent expectoration.

3. In many cases of catarrhal pneumonia in adults, and of capillary bronchitis in children, the sputa contain microphytes in great numbers. These may be nontubercular bacilli, of various sizes, or micrococci, solitary, or arranged in chains or sarcinoid groups; and these take up the blue colour only in double staining.

4. In some cases of phthisis, in which there are signs of lobular condensation of the lower parts of the lung with excavation of the apex, the sputa may be free from tubercular bacilli, but crammed with the bacilli and micrococci that take up the blue stain. Yet *post mortem* tubercular bacilli may be found distributed abundantly throughout the lung. In such cases the presence of the nontubercular microphytes with the bronchi and alveoli(?) does not seem to retard the course of the disease.

5. When tubercular bacilli are certainly present in the sputa, I have never found any other commensal microphyte. Whether that can be taken as an indication that tuberculous matter is inimical to the vitality of putrefactive microphytes requires to be proved. Tubercular bacilli do not appear to find a suitable nidus in the secretions of the bronchi, for while blue staining microphytes are often to be seen within, or upon (for example) the flat cells from the lower layer of the bronchial mucous membrane, or large catarrhal cells derived from the alveolar lining, the tubercular bacilli usually float free in the liquor sputi, or are embedded in little irregular masses of a granular material that also takes up the magenta stain. I have never observed such bacilli within cells recently shed, that is, free from granular fatty degeneration. Their occurrence in the sputa seems to be, in a manner, accidental, due to an admixture of pulmonary *debris* with the true bronchial secretion. Accordingly the number expectorated fluctuates, and a numerical diminution cannot be taken, *per se*, as evidence of diminished reproduction. Since a pause in the course of true tubercular phthisis may occur in cases treated with, or without, systematic antiseptic inhalations, I cannot attribute any temporary arrestment in the one set of cases to such intrapulmonary medications. Undoubtedly symptoms may be palliated, and putrefactive microphytes (if present) prevented from multiplying; but I fear the ultimate result is not effected. At least, I have never heard of the absolute cure of any case of phthisis in which tubercular bacilli were expectorated. If antiseptic methods be futile, Cantani's septic system does not seem more hopeful. The *rationale* of bacteriotherapy appears to depend on a mere hypothesis, that tubercular bacilli may be starved out through the more active bacteria appropriating and rapidly decomposing the dead caseous material in which the bacilli flourish. If, however, such an antagonism really existed between these microphytes, it would be natural to expect that the hardy and omnipresent bacterium should have by this time exterminated the tubercular bacillus in the natural struggle for existence.—I am, yours, etc.,

W.

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## BOOKS, etc., RECEIVED.

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Select Methods in Chemical Analysis. By W. Crookes, F.R.S. Second Edition. London: Longmans and Co. 1885.