

Wright's work on opsonins suggested that the reason that the light treatment is of such great value is that the inflammatory reaction floods the affected tissues with opsonins. If that were the explanation it would follow that other measures which determine the flow of blood to the part should be equally successful. Some years ago I was given the opportunity, by the courtesy of the inventor, of trying Gould's apparatus. The instrument consists essentially of an air pump to which was attached a modified cupping glass. Mr. H. Rischbieth, who was then my clinical assistant, and I made many trials with this apparatus in comparison with the Finsen lamp. We never obtained a reaction which was in any way comparable with the latter. The effect appeared to be too transitory. Since then I have used cupping-glasses for the same purpose but without obtaining any satisfactory results. Prolonged fomentation has also been tried. Dr. W. Bulloch suggested dry heat and I had several patients treated for weeks with small bags filled with hot sand. In no single instance was there any appreciable result. One more point has been tested. It was suggested that the pressure used to the affected part in the Finsen treatment was more important than the light. To determine the value of this I had several patients subjected to compression of a lupus spot with the usual compressors with no benefit or, indeed, any sign of alteration in the character of the lupus tissue.

In conclusion, I must thank my clinical assistants for their valuable coöperation and especially the sisters and nurses in the light department, to whose constant attention to the difficult details of the work the success of the treatment is due.

Manchester-square, W.

A CASE OF EGG POISONING.

BY ALFRED T. SCHOFIELD, M.D. BRUX., M.R.C.S. ENG.,
L.R.C.P. LOND.

THIS case is of interest because, although egg poisoning is not extremely rare, I have been unable to find any record of cure. Jonathan Hutchinson in his "Pedigree of Disease" (1884), p. 35, speaks of eating eggs producing violent vomiting, a sense of sinking, and abdominal distress quite inexplicable, and temporary defect of sight. An artist could not see to paint after eating an egg, there being temporary suspension of the power of accommodation in both eyes, with heat of the stomach and abdominal discomfort one or two hours after breakfast. A little egg in a pudding or sauce would be sufficient to cause these symptoms, which were quite cured by abstinence from eggs. Other cases are recorded but I can find no real attempt at cure. It is possible the following account may call forth several others.

In June, 1906, I saw a boy, aged 13 years, whose parents complained that he could not eat egg in any form. He could not eat meringues nor any cake with white of egg. He had had an attack after eating bacon cooked with eggs, and the smallest piece of bread or bun with white of egg upset him. In the attack there was first of all free secretion of saliva, the lips burned, the patient felt sick, itched, and an urticarial rash shortly broke out; he swelled all over, with puffy eyelids and lips, tight, red, swollen skin, and could hardly breathe from a sort of asthmatic attack. He was found gasping in an attack after eating a small apple fritter. Never in his life had he eaten an egg. As a baby he spat out any food that contained the least trace of egg. As a child he could eat any cake free from egg, but not the smallest piece of sponge cake. He swelled up almost instantly after eating a small bun free from egg but which had been brushed over with the white of egg. Raw egg blistered his skin. He has also had attacks after eating soup cleared with white of egg, and with the smallest bit in apple sauce when not in the least suspected; so that "suggestion" played no part. He may have had some 150 attacks. He is a healthy boy generally, but has had enlarged glands, cured without operation, is of lymphatic temperament, and has some gouty eczema; the lungs and organs generally are sound and healthy.

In December, 1906, treatment was begun on the lines of establishing tolerance to this especial poison. Six weeks before, being at school he had eaten at lunch about six mouthfuls of a ginger pudding which was found to have egg in it. He felt a pricking in the tongue and throat which got better; and then in half an hour an urticarial rash came out

and lasted two or three hours. There were swelling, shortness of breath and wheezing, and swollen eyes and joints. The theory of the poisoning was that the egg albumin acted in some way on the serum albumin so as to cause transudation.

The treatment consisted in the constant administration of egg with a little calcium lactate added to stop the transudation. Pills were made containing $\frac{1}{10000}$ th part of a raw egg and two grains of calcium lactate each. The boy continued his usual school and home life all the time but was closely guarded against any egg in the food. He never knew there was egg in the pills. The first month (December) he took $\frac{1}{10000}$ th egg daily. The next (January) this was gradually increased every four days till $\frac{1}{1000}$ th of raw egg was taken daily with no symptoms. (Far less than this had previously caused symptoms.) In the month of February pills with cooked and raw egg were taken alternately, and the quantity in each pill was increased to $\frac{1}{500}$ th of an egg daily, the calcium lactate being continued. In March the amount was steadily increased till $\frac{1}{250}$ th was taken daily with no symptoms. In April it was raised to $\frac{1}{150}$ th of an egg and in May $\frac{1}{75}$ th of an egg was reached, still without symptoms, it being now quite clear that real tolerance was being established. In June it was raised to $\frac{1}{35}$ rd. The patient by this time had consumed a whole egg in the six months for the first time in his life. In July the pills were dropped and the boy was given as a test some puddings and cake in which he thought there was egg but which had none and no symptoms ensued. He then had egg in his food constantly, till by the end of July he was taking $\frac{1}{5}$ th of an egg daily. In July alone he ate nearly four eggs in his food. All this time he was quite well, and the amount was rapidly increased till he ate an egg a day and since has had egg in some form in food every day, and can now eat anything.

Some may think a great deal of trouble was taken to cure this idiosyncrasy, but when we remember that it was not connected with some rare food such as pineapple, which could easily be avoided, but with an article that enters into nearly all a schoolboy eats, and that his life had been more than once in danger from such food, it will be seen that the trouble taken was amply justified. The difference to the boy is, of course, enormous, and there was no sign whatever that he "would grow out of it." It would seem that with sufficient care and patience tolerance may be established in the case of most poisonous foods, and, of course, there is abundant general evidence as to this. I have ventured to record the present case because, as far as I know, it is unique, though, as I have said, I daresay this may call forth letters to prove that it is not.

Harley-street, W.

Clinical Notes:

MEDICAL, SURGICAL, OBSTETRICAL, AND THERAPEUTICAL.

WARNING AGAINST THE INDISCRIMINATE USE OF THE OPHTHALMO-REACTION (CALMETTE) IN THE DIAGNOSIS OF TUBERCLE.

BY A. MAITLAND RAMSAY, M.D. GLASG.,

SURGEON, OPHTHALMIC INSTITUTION, AND CONSULTING OPHTHALMIC SURGEON, ROYAL INFIRMARY, GLASGOW.

THE case, that of a school-girl, 12 years of age, is reported to demonstrate that the ophthalmic-reaction (Calmette) must be used with caution and discrimination. The patient was, on Oct. 8th, 1907, admitted to the Ophthalmic Institution suffering from superficial vascular ulceration of the right cornea. There was a history of a similar attack in the left eye two years before and though careful examination revealed no sign of tubercle in the lungs or the abdomen both cervical and submaxillary glands were much enlarged. On the 10th one drop of a 1 per cent. solution of tuberculin was instilled into the left eye which was at that time perfectly free from inflammation, although there was a faint nebula on the cornea the result of the previous attack of ulceration. Within 24 hours there was violent muco-purulent reaction, the discharge being very abundant and accompanied by marked swelling of the lids and thickening of the palpebral conjunctiva. The inflammation could not be

influenced by treatment and progressed till the cornea quickly became vascular and abraded over the central area. On the 20th 0.25 cubic centimetre of a 1 in 1000 solution of Koch's old tuberculin was injected subcutaneously. This was followed by a rise in temperature and resulted in great improvement in the condition of the right eye, though the left remained unchanged. Subsequently the old tuberculin was again injected twice, the first time to the amount of 0.25 cubic centimetre and the second time to the amount of 0.5 cubic centimetre, and by the end of November the right eye was almost quite recovered, while in the left the discharge had begun to lessen. After that date the improvement in the latter was steady but there remains as a result of the Calmette instillation a considerable opacity of the centre of the cornea, and in consequence the vision is seriously impaired.

Glasgow.

NOTE ON A CASE OF POISONING BY GELSEMIUM.

By JOHN BELL, L.R.C.P. LOND., M.R.C.S. ENG.,

SUPERINTENDENT OF THE GOVERNMENT CIVIL HOSPITAL, HONG-KONG.

As "Taylor's Medical Jurisprudence" states that this vegetable poison has never been the cause of a criminal charge the following case may be of interest to the readers of THE LANCET, more especially as the defendant's barrister, with "Taylor" in his hand, made much of this fact. The case was tried at the Criminal Sessions on Jan. 20th, 1908, before Sir Francis Piggott, C.J. The woman was found guilty and sentenced to death. The brief facts were that in consequence of a love affair having led to a disturbance she boiled some herbs and gave them to her husband as "cooling medicine" and he died shortly afterwards. The man had his food about 5.15 P.M.; the poison was administered about 6 or 6.15 P.M. The first symptoms appeared about half an hour afterwards and he died about three and a quarter hours after taking the "cooling medicine."

Mr. F. Browne, the Government analyst, identified the herbs as "gelsemium elegans" and extracted one-sixth of a grain of gelseminine from the contents of the man's stomach. The woman attempted to commit suicide but recovered. When sent to this hospital at 11.50 P.M., four hours after she had taken the poison according to her own story, she was semi-conscious, with dilated pupils insensible to light, and with slight contractions of the muscles, more especially of the upper limb. She was quite well on the next day. I inclose the police report.

[INCLOSURE.]

Western District Police Office, Jan. 21st, 1908.

Poisoning Case at No. 59, High-street.

SIR.—On the evening of Nov. 17th, 1907, Cheung Fuk was poisoned by his concubine, Lam Kin, alias Mo Ho, at the above address. Cheung Fuk was a foreman stonecutter and contractor and was a Hakka, as probably about 90 per cent. of the stonecutters in this colony are. The concubine had a paramour and both her and the paramour also were Hakkas. At about 1 P.M. on Nov. 15th, 1907, Cheung Fuk returned home, and being about three hours earlier than usual found the paramour, Ng Nin, leaving his house after paying a visit to his concubine in his absence. He did not interfere with Ng Nin, but on entering his house he demanded an explanation from his concubine and as no explanation was forthcoming he assaulted her with an umbrella. On Nov. 17th Cheung Fuk is said to have returned home at about 4 P.M. and to have gone out again until about 5 or 5.10 P.M., when he had his rice, &c., and some little time afterwards the concubine boiled some herbs as a cooling medicine for him and boiled the poisonous stems and leaves and put into the cooling medicine and gave it to him to drink. We cannot ascertain the exact time he took it, but most probably about 6 P.M. to 6.15 P.M. The concubine in her statement before the court gave the time as 7.30 P.M. when she gave him the medicine, and stated that he died at 8 P.M. A witness named Cheung Chung (who is deceased's nephew) stated "that when he visited Cheung Fuk at about 7.30 P.M. he found him rolling about on his bed in great pain and that he rolled off on to the floor, the muscles appeared to be all on the work, twitching and contracting, and the face distorted," and that he died at about 9.30 P.M. The concubine told us "that she took of the poison at about 7.50 P.M.," and when Sergeant Lenaghan entered the house at 11 P.M. "he found her rolling on the floor, she was in a dazed condition, almost unconscious, was perspiring very freely, muscles twitching, and face distorted, the eyes very full and pupils dilated."

1. Cheung Fuk's age was about 50 years.
2. The poison was given with herb tea, not with the food; he, however, had a feed of rice and congee at about 5.15 P.M.
3. Probably about half an hour after taking the poison the symptoms began.

4. As near as I can ascertain he died about three and a quarter hours from the time he took it, or two and three-quarter hours from the time the symptoms began to show.

A. CONNS, Inspector.

The Superintendent, Govt. Civil Hospital.

PS.—This poison is well known to the Hakkas ("O Mūn Ting" or "Tai Cha Yeuk") and is given to pigs and goats as a medicine by them. Other sections of the Chinese seldom, if ever, use it.—A.C.

Medical Societies.

ROYAL SOCIETY OF MEDICINE.

MEDICAL SECTION.

Acute Suffocative Catarrh.—A Case of Acute Ulcerative Colitis with some Unusual Symptoms.

A MEETING of this section was held on Feb. 25th, Dr. S. J. GEE, the President, being in the chair.

Dr. SAMUEL WEST read a paper on Acute Suffocative Catarrh. He said that the acute suffocative catarrh of Laennec was still but little recognised—an acute catarrh of sudden onset, with suffocative dyspnoea, lasting from 24 to 48 hours. If not fatal it ran the course of an acute bronchitis. He cited a case which occurred in a young man, aged 23 years. In 24 hours from a condition of perfect health the patient passed into a state of urgent dyspnoea with considerable cyanosis and looked likely to die. Rhonchus and sibilus were the only physical signs and the temperature was raised to 101° F. In 24 hours more the urgent dyspnoea passed off. A little expectoration was then brought up and was found to contain a few pneumococci but a large number of diphtheroid bacilli of uncertain nature. The case ran a slow and tedious course towards recovery. Two other bacteriological examinations, made at intervals, showed the same bacilli. It was possible that the attack depended upon the bacilli and their wide dissemination through the bronchial tubes. The following conditions were such as were either liable to be confused with true suffocative catarrh or presented interesting relations with it: 1. Acute suffocative pulmonary oedema, or as it had been called acute non-inflammatory congestion of the lungs. Of this two instances were given with antecedent morbus cordis, one in the course of mitral disease and the other of aortic disease. It also occurred without antecedent morbus cordis, as in the course of hyperpyrexia or of malignant or septic fevers. 2. Acute inflammatory congestions. Capillary bronchitis and secondary broncho-pneumonia gave rise to little confusion. Primary broncho-pneumonia, or acute disseminated pneumococcal pneumonia, was very closely allied to suffocative catarrh. There was a form of acute pneumococcal bronchitis, and indeed suffocative catarrh might be, as the case described suggested, of pneumococcal or other bacterial origin. In the early congestive stage of acute pneumonia the dyspnoea might be urgent and the whole lung be affected. The general congestion passed off as the local lesion developed. That might be attended even with considerable hæmoptysis. 3. Collateral fluxion or pulmonary failure where one or both lungs were much taxed. The extra work might easily and rapidly pass into overwork and the failure was marked by the signs of congestion or bronchitis—a condition of gravity which if it could not be relieved at once was rapidly fatal. Pulmonary failure might arise on the opposite side in a case of pleuritic effusion or pneumothorax in both lungs in a case of abdominal distension—e.g., ascites, acute peritonitis, or tympanites, and in the other parts of the lungs in a case of acute pneumonia. Laennec's acute suffocative catarrh was a peculiar and characteristic affection and might, as the case recited suggested, be due to a widespread bacterial infection.—The PRESIDENT described how impressed he had been with the first case he had met with in his practice of acute suffocative catarrh described by Laennec, and added that it had made him very careful in expressing an opinion of the future course of any case of bronchitis at the beginning of the attack.—Dr. F. J. WETHERED narrated a case of a man who died 48 hours after the administration of ether for an operation with symptoms similar to those described by Dr. West. The question was, Was there any association between the two? In regard to Laennec's acute suffocative catarrh being due to bacterial infection, Dr. Wethered asked Dr. West to express an opinion on the case of three men who had cleaned out a filthy cesspool and who within a few hours were seized with symptoms of urgent dyspnoea and died within 24 hours.—Dr. W. EWART suggested artificial respiration in the prone position as an early treatment in cases of acute suffocative catarrh.—Dr. WEST, in reply, suggested that the explanation of the death of the patient mentioned by Dr. Wethered with acute pulmonary symptoms after the administration of