THE INFLUENZA EPIDEMIC OF 1918:

COLOUR OF THE BLOOD IN FATAL CASES.

BY

J. S. HALDANE, C.H., M.D., F.R.S.

ONE of the most striking facts about the world-wide and extraordinarily fatal epidemic of "influenza" in 1918 was that in the bad cases one of the symptoms was a peculiar blue or violet form of cyanosis. At the time this cyanosis was attributed to broncho-pneumonia; but against this explanation was the fact that administration of oxygen failed to abolish or diminish the cyanosis, whereas in the cyanosis which sometimes accompanies ordinary lobar pneumonia, or the lung inflammation of poisoning by irritant gas, the cyanosis can usually be abolished easily by adding some oxygen to the inspired air.

In a paper published in the *British Medical Journal* of August 1st, 1925 (p. 187), on the presence post mortem of nitric-oxide-haemoglobin, by Dr. Banham, Dr. Savage, and myself, a fatal case of broncho-pneumonia (mistaken at first for carbon monoxide poisoning) was described, in which the blood was found to be everywhere red post mortem, and to give certain of the usual tests for CO-haemoglobin, although the patient was cyanosed just before death, and had not been exposed to carbon monoxide at all. The red colour was evidently due to NO-haemoglobin, which appears post mortem in poisoning by nitrite,¹ while the cyanosis during life was due to the fact that in poisoning by nitrite methaemoglobin is formed, producing practically the same colour in the blood as ordinary cyanosis due to reduced haemoglobin.

In this paper we mentioned the fact that Dr. Banham, when in command of the medical section of the military

hospital at Cannock Chase during the war, had repeatedly observed that in the bodies of those who died in the 1918 influenza epidemic the blood was red on exposure post mortem. He had also made a special report on the subject to the War Office.

Since our paper was written I have received information from the doctors and nurses who saw the bodies in fatal cases, both in this country and in France; and they confirm Dr. Banham's experience that the blood was red post mortem, though cyanosis was present during life.

It thus appears to be at least extremely probable that the organism responsible for the very fatal character of the 1918 epidemic was a nitrite-forming organism, and that the main immediate cause of death was anoxaemia due to conversion of oxyhaemoglobin into methaemoglobin. In fatal nitrite poisoning in animals the immediate cause of death is anoxaemia.

Although nitrite-forming organisms are familiar to soil microbiologists, very little, if any, attention has hitherto been given to pathogenic organisms of a similar character. Apart from the 1918 influenza epidemic I have heard of several fatal cases of broncho-pneumonia in which deep cyanosis, probably due to methaemoglobin, was present. It is probable, therefore, that such cases are not very rare, even in the absence of an epidemic of them.

I have written this paper in the hope that some case of this kind may be diagnosed clinically or post mortem, and a proper bacteriological investigation made. It is easy to detect methaemoglobin clinically in a drop of blood, and equally easy to recognize nitric-oxide-haemoglobin post mortem. It seems, however, that unless there is any suspicion of carbon monoxide poisoning not much attention is usually paid at post-mortem examinations to the colour of the blood.

REFERENCE.

¹ Haldane, Makgill, and Mavrogordato: *Journ. of Physiol.*, xxi, 1897, p. 160.

TREATMENT OF PRURITUS ANI AND ANAL FISSURE:

THE USE OF ANAESTHETIC SOLUTIONS IN OIL.

BY

W. B. GABRIEL, M.S.,

SENIOR ASSISTANT SURGEON, ST. MARK'S HOSPITAL.

FROM a practical point of view cases of pruritus ani may be divided into two main groups:

1. The group in which some definite local cause is present. This is usually some lesion which causes an abnormal amount of perianal moisture; amongst the commoner of these may be mentioned prolapsing piles, external piles, fissure, fistula, hypertrophied papillae, rectal polypi and tumours, threadworms, proctitis, etc. Treatment of cases in this group, if limited to removing the exciting cause, together with treatment of the perianal region with simple lotions and powders, is usually effective.

2. The second group is that in which patients complain of persistent severe itching, but examination reveals none of the obvious causes enumerated above. The rectum is smooth, and examination, as a rule, is negative, except that some evidence is found to confirm the patient's complaint of irritation. The perianal skin is often somewhat thickened and thrown into rugae radiating from the anus, with a varying amount of excoriation of the skin or fissuring.

It is cases in this second group which are the great problem as regards treatment. It is probable that a chronic neuritis of the sensory nerves in the anal region is present in most cases of long duration, and hitherto the recognized treatment of cases which have failed to respond to local treatment of the dermatitis has been either (1) operation—Ball's operation or any of the various modifications designed towards sectioning the cutaneous nerves of the anus; (2) the subcutaneous injection of various solutions such as absolute alcohol, quinine-urea, hydrochloric acid, etc.; (3) x-radiation.

After working for the last eight years in the out-patient department of St. Mark's Hospital, I have been impressed with the unsatisfactory results generally obtained by these methods. Judging from the number of patients who attend who have previously had one or more courses of x-ray treatment, the conclusion is forced upon one that the relief obtained, if any, is often of short duration, and the necessity for repeated courses of x rays is dangerous. X-ray dermatitis and x-ray carcinoma of the anus are well-known catastrophes. As regards Ball's operation, it is true that, if properly done, patchy anaesthesia results at once in the flaps outlined by the operation, but it seldom lasts more than a few months; the extensive wound never heals by primary union, and although there may be relief for a time, the irritation often returns after a few months. There is then quite often the added trouble that an atrophic condition of the skin flaps is liable to develop; the perianal skin within the two curved incisions appears thin, shiny, and discoloured, often with patchy excoriation; this may be partly a trophic condition from section of the sensory nerves, and partly due to the impaired vascular supply; it is, I think, a serious objection to Ball's operation.

Pruritus being essentially a local condition, it is reasonable to expect a local treatment to relieve it, and subcutaneous injection of an anaesthetic solution appeals to me as being a sensible method likely to succeed. I was therefore greatly attracted by the report of Drs. Yeomans, Gorsch, and Mathesheimer¹ on the use of benacol in the treatment of pruritus ani. This solution was introduced to me last autumn by Dr. Frankfeldt of New York, who very kindly provided me with a supply for trial at St. Mark's. To quote Dr. Yeomans: "Benacol is a solution of equal parts of para-amino-benzoyl-benzoate and phenmethylol in 90 parts of rectified sweet almond oil. It is non-toxic, highly anaesthetic, moderately antiseptic, and, as far as investigated, has a mild depressant action on peripheral nerve endings."

The use of an oily as opposed to an aqueous solution would appear to be particularly valuable as a method of delaying absorption of the local anaesthetic, and therefore