to our therapeutic armamentarium—an agent which, although not by any means a general panacea, is the more valuable in that many of its successes are those very chronic and difficult cases in which the prognosis has hitherto been so hopeless.

A bibliography of the literature of ultra-violet therapy in ophthalmology is appended.

It is a pleasure to thank the members of the staff at Moorfields for their cordial assistance and advice, and for according me the privilege of making use of and reporting their cases.

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DEATH FROM FULMINATING PNEUMONIA AFTER BRIEF NITROUS OXIDE ANAESTHESIA.

BY

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An apparently healthy lad was anaesthetized with nitrous oxide and the stumps of a molar tooth extracted. He never recovered consciousness and died in thirty-seven hours. The main features of this remarkable case are as follows.

A.B., aged 17, the brother of a dental student of Liverpool University, was seen by a dentist in June, 1923; he unsuccessfully attempted to remove two stumps of a lower molar tooth. A local anaesthetic was used. Six months later the jaw was x-rayed at the Liverpool Dental Hospital and an appointment made for extraction. On Saturday, January 26th, 1924, the patient came to the hospital as arranged. At 11 a.m. he was anaesthetized with pure nitrous oxide, and the stumps extracted without difficulty. The administration of the anaesthetic and the extraction took a minute or slightly more.

The patient began to recover from the anaesthetic, but soon went a "nasty colour, then black," and collapsed. As air failed to enter the lungs properly he was placed on the floor and artificial respiration resorted to; 15 minims of ether were injected subcutaneously. A few minutes later Dr. Earlam, a house-surgeon at the Liverpool Royal Infirmary, was summoned. He found that the patient was cyanosed. There was a gag in his mouth; but owing to an unusual deformity of the lower jaw the chin was pressing on the larynx. On removing the gag and pulling the chin forward, breathing became much freer.

At 1.30 the patient appeared to be progressing satisfactorily, but was still drowsy, so he was put to bed in the Royal Infirmary under the care of Dr. Douglas Bigland; the temperature was then

102°, and the pulse 118 (see chart). Soon after admission he had a generalized convulsion, lasting one or two minutes. At 3 p.m. the convulsions recurred for about fifteen minutes with incontinence of urine and faeces; they were so violent and persistent that a strait-jacket was necessary.

The patient was treated with wet packs, and the temperature fell a little, but at midnight it was again 102°; the respirations were 45 (see chart). Lumbar puncture was then performed; the fluid, though under slightly increased pressure, appeared normal, but unfortunately the tube containing it was broken immediately afterwards. Throughout January 27th there were no more convulsions, but the coma persisted and the temperature rose still higher; the respirations were about 50. The breath smelt strongly of acetone, and the urine was found to contain a large quantity of it, but no sugar, no albumin, and no urobilin. More wet packs were employed, also intravenous salines, insulin, and adrenaline. The skin became a peculiar "yellowish tinge," and the patient died at 12.15 a.m., January 28th, thirty-seven hours after the anaesthesia.

Necropsy.

The post-mortem examination was made fourteen hours later by Dr. Howel Evans, lecturer in pathology, Thompson Yates Laboratory, Liverpool University.

The brain was congested and contained a few punctiform haemorrhages; the membranes were normal. The thymus was big "but not definitely enlarged." The tonsils were not enlarged. The trachea and bronchi were intensely congested; there was no blood and no other foreign body present in them nor in the larynx. The lungs (weight, right 1 lb. 4 oz., left 1 lb. 8 oz.) showed moderately extensive early lobar pneumonia of both bases, especially the left—portions were red and sank in water; the margins were moderately emphysematous. Petechial haemorrhages were present on both pleura, but no fibrin. The heart, stomach, intestines, pancreas, and suprarenal glands were normal. The liver (weight 4 lb.) was congested and soft. The spleen (weight 7 oz.) was dark and softer than normal. The kidneys (weight 6 oz.)

were congested. Some of the mesenteric glands showed caseating tubercle with commencing calcification. The mandible was malformed and underdeveloped, particularly posteriorly; it had no angle, for the ascending and horizontal rami lay in a practically straight line.

Bacteriological Investigation.

Direct examination at the Thompson Yates Laboratory of the juice of the consolidated portion of the lungs showed many organisms like pneumococci. The colonies of bacteria on a Petri plate culture, using "chocolate" agar* as a medium, were approximately 70 per cent. pneumococci, 25 per cent. *Staphylococcus aureus*, and 5 per cent. streptococci. The presence of pneumococci was also demonstrated by inoculation of lung juice into a mouse; it died in three days and the organism was isolated from the peritoneal fluid. The pneumococcus cultures were bile-soluble, and when tested with type serums proved to belong to Group IV. The streptococcus was non-haemolytic. No bacilli of the influenza group were found, though specially looked for.

Histological Investigation.

Consolidated portion of a lung showed great congestion; the alveoli were more or less completely filled with exudate, which consisted chiefly of red cells; fibrin was scanty; the bronchioles contained many leucocytes. There was no pleurisy. Numerous pneumococci were demonstrated in the alveoli by Gram's stain, but no streptococci or staphylococci.

The polyhedral cells of the liver showed much fatty degeneration, the fat being deposited in small droplets round a central nucleus. The kidney showed marked cloudy swelling and some congestion.

Examination of the Brain.

Though death was clearly due to fulminating pneumonia, yet, "to make assurance doubly sure," the brain was examined microscopically, to eliminate the possibility of encephalitis or other lesions. This was because the writer once made a *post-mortem* examination on an infant aged 6 months who died unexpectedly thirteen hours after a talipes tenotomy. A big thymus of 27.4 grams suggested status lymphaticus; but a microscopical examination of the brain—normal macroscopically—revealed encephalitis lethargica. Ether was the anaesthetic. (Glynn and Dun, 1923, Case 2.) Nevertheless, a section of A.B.'s cerebral cortex—also examined by Dr. G. A. Watson, pathologist to the Rainhill Mental Hospital—showed neither meningitis nor encephalitis. There was, however, a diminution of Nissl granules in the nerve cells—indicating exhaustion from the fits and toxæmia.

1. The Unusual Virulence of the Pneumonia.

Coroners and even medical men often fail to realize that sudden and unexpected death is not infrequently due to acute bacterial infection; so the cause may be overlooked *post mortem*. Death from pneumococcal pneumonia sometimes occurs in six or seven hours, therefore before the development of lung consolidation, particularly in children and young adults (McGowan and McNeil, 1913; Glynn, 1913; Glynn and Dun, 1923; also Wilson and Glynn, 1926).

A. B. was infected with Group IV pneumococcus. But the mortality of Group IV pneumococcal pneumonia is lower than that of the two other common types; for in 17.6 per cent. of 1,993 English and American adults it was contracted with 22.9 per cent. Type I and 35.2 per cent. Type II (Glynn and Digby, 1923, M.R.C. Special Report No. 79, Table XXIV).

The streptococci found in the sputum most probably did not increase the virulence of the pneumonia because they were non-haemolytic; also because they were present in relatively small numbers—namely, 5 per cent. in culture; besides pneumococci only were found in the lung section.

Glynn and Digby have produced evidence that the streptococci they cultivated from the sputum of 77 per cent. of 92 adults with pneumococcal lobar pneumonia, usually exert "little or no pathogenic effect" and were "confined to the trachea and bronchi" (Table XXXVI, also Table XXXV, pp. 132 and 155). They also cultivated the sputum of 26 adults with pneumococcal lobar pneumonia who eventually died, and found streptococci in 73.1 per cent. and *Staphylococcus aureus* in 7.6 per cent. The relative distribution of the pneumococci, streptococci, and *Staphylococcus aureus* in the positive plate cultures was 70.4, 19.5, and 25 per cent., which is similar to that in A. B.'s sputum—namely, pneumococci 70 per cent., streptococci 5 per cent., and *Staphylococcus aureus* 25 per cent.

The unusual virulence of A. B.'s pneumonia was probably due to the patient's low "resistance," and also to the partial asphyxia from the anaesthetic and from the subsequent respiratory obstruction by the deformed jaw, and lastly to the acidosis. It is interesting that even partial

asphyxia with non-irritating gases may produce severe pulmonary congestion (David, 1912; Yandell Henderson, 1926).

2. The Date of the Onset of the Pneumonia.

The lad's health had been good; but on Thursday evening, January 24th, he complained of headache, and his mother found the temperature "a little over 99°." Next day, however, he appeared to be in normal health, so the temperature was not taken; he played bridge till 2 a.m. On the following morning a friend who met him going to the Dental Hospital for the extraction noticed that he was "very flushed." Probably, therefore, the lad's temperature was up at the time of the anaesthetic, especially as it was 102° three hours later.

Now the incubation period of pneumococcal lobar pneumonia, though short, appears to be two to three days or more (Osler and McCrae, 1907). It would also take at least one to two days for the pneumonic consolidation, found *post mortem*, to develop (Aschoff, 1921). Most probably, therefore, the disease had actually begun before the anaesthesia, and the lungs were in the first or congested stage of the inflammation. The usual initial pleurisy and rigor were absent, otherwise the patient might have realized that he was ill and his chest affected. It is interesting that the initial rigor is less frequent in Group IV pneumococcal lobar pneumonia than in the other two common types—namely, 42.9 per cent., contrasted with 77.8 per cent. Type I and 52.6 per cent. Type II (Special Report No. 79, Table XLIV).

3. The Epileptiform Convulsions.

They probably signified the entrance of many pneumococci into the general circulation. It is well known that acute pneumonia in young children is sometimes ushered in by convulsions; but the lad A. B. was 17. Nevertheless, convulsions occurred in a boy of 15 who died of fulminating pneumonia seven hours after the first symptom (Glynn, 1913, Case 2).

4. The Ketosis.

This was severe and first noticed about 11 a.m. on January 27th. It presumably signified acidosis—confirmed by the fatty degeneration of the liver. The ketosis was not the result of the anaesthetic. It was partly due to starvation; the patient had a light breakfast of tea and toast the day before and no food afterwards, except glucose injections at 1 p.m. and 6 p.m. on January 27th. It was also partly due to the fever and to the toxæmia. Terminal acidosis frequently occurs in dying persons.

5. The Source of the Infecting Organisms was most probably Autogenous.

This is because Group IV pneumococcus is the "type" commonly present in the saliva. Pneumococci were demonstrated in 43.5 per cent. of 485 specimens of saliva; 67.4 per cent. of the positive specimens contained Group IV pneumococcus—some of those containing other types were from pneumonia contacts (Special Report No. 79, Table XX). Non-haemolytic streptococci are invariably present and *Staphylococcus aureus* sometimes present in normal saliva.

SUMMARY.

An apparently healthy lad of 17 was briefly anaesthetized with pure nitrous oxide and two molar stumps extracted without difficulty. But he never recovered consciousness. Three hours later his temperature was 102°; then epileptiform convulsions occurred, and finally acidosis and hyperpyrexia. He died of pneumococcal lobar pneumonia thirty-seven hours after the anaesthetic.

The bases of both lungs showed early red hepatization; the pneumococcus was of Group IV and therefore probably an autogenous infection. Most probably the pneumonia had begun at the time of the anaesthesia, especially as the patient was indisposed two days before.

The virulence of the pneumonia was probably due to the patient's low "resistance" and also to the partial asphyxia from the anaesthetic and the subsequent temporary respiratory obstruction by the deformed jaw, and lastly to the

* Chocolate agar is a mixture of agar and boiled blood; it is especially suitable for growing bacilli of the influenza group (W. Levinthal, *Zeits. f. Hyg. u. Infektionskrankh.*, 1919).

But for the *post-mortem* examination, death might have been attributed to an extraordinary susceptibility to nitrous oxide poisoning.

I wish to thank Dr. Douglas Bigland for kindly giving me the notes of the case.

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A NEW SYSTEM OF TREATMENT OF DIABETES MELLITUS.

BY

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THE following is an outline account of a method of treatment which, although not claimed as a cure for diabetes, has been employed in over 160 cases of varying degrees of severity with considerable success since 1923.¹

If insulin be administered in large doses before the two principal meals of the day, over unbroken periods, it has been observed that a certain summation of effect can be obtained. That the actual basal trouble is diminished in intensity is shown by the progressive lowering of the "ketonic threshold value"²—the point at which ketonuria appears when a patient is placed on a diet containing a known quantity of protein and fat and no carbohydrate. This value is expressed as the patient's blood sugar content at this moment, and is a direct method of expressing the intensity of the basal factor at any given period.

This action of insulin can be pushed to such an extent that the patient may be transformed, metabolically speaking, into a normal person whilst under its influence; this condition being capable, in favourable cases, of persisting for variable periods after the cessation of injections. This experience led to the idea that treatment might be attempted in the form of "series" of injections, somewhat in the same way that syphilitic courses are arranged. The results which have been obtained appear to justify the adoption of this method of treatment, at any rate in a majority of cases.

Advantage is, further, taken of the fact that the patient is temporarily transformed into a normal individual to increase considerably the ingestion of carbohydrate which can, under the influence of the large doses of insulin administered (80 units per twenty-four hours), be easily tolerated and utilized to improve the general condition and resistance of the patient. It is found that in spite of the fact that, in certain instances, the regime is increased to almost normal proportions, the urine becomes sugar-free, and the blood sugar falls to the region of the normal in a short time.

It is considered that every case of diabetes mellitus presents two essential elements: (a) a basal, or metabolic, element; (b) a renal element.³ The former consists in the defective power of the tissues to consume glucose in normal ratio to their blood sugar content—for quantitatively this consumption is often found to be equal to the normal. The renal element is a phenomenon similar to that observed in cases of "renal glycosuria," but with the basal element superimposed.

These two elements may be combined in varying proportions, the preponderance, or otherwise, of the renal element tending to determine the clinical type of the disease (*D. gras*, or *D. maigre*). This being so, it is found advisable, when fixing the initial regime of a diabetic patient, to estimate approximately the intensity of this renal factor, since it is found experimentally that in any given case the time taken for the glycosuria to become normal is in inverse ratio to the importance of the renal diabetic

element. This is estimated very simply by comparing the blood sugar of the patient taken whilst fasting with his twenty-four hourly output of urinary sugar. If the difference is considerable, the renal element is said to be pronounced, and the carbohydrate content of the diet is modified somewhat in consequence, in order to allow of the glycosuria becoming normal in a minimum of time. It is seldom, however, found necessary to reduce it below a daily ration of about 100 grams, whilst if the renal element is found to be of moderate importance only, a ration of between 150 and 250 grams may be allowed from the start.

The insulin "series" is then commenced, the injections being given twice a day by the intramuscular route, generally into the buttock, and before the principal meals of the day in which most of the carbohydrate is massed. Hospital out-patients bring with them the 150 grams of milk which is specially introduced into their regime for the purpose, and which they drink immediately after the injection, by this means avoiding the risk of hypoglycaemic accidents which might otherwise intervene before they could reach their homes. Accidents of this nature, it may be mentioned, have been observed in only 7 cases amongst the 50,000 or so injections given between 1923 and 1925.

When the glycosuria has been *nil* during a minimum period of fifteen days, and if the blood sugar is within reasonable limits, the injections may be suspended, pending a return of the symptoms, which must be carefully watched for; a further "series" being commenced under the same conditions as the former on the slightest indication. In this way the free intervals between the series will be found gradually to increase, whilst the duration of the series can be progressively diminished to a minimum of about ten days, until a patient with a basal trouble of moderate severity, who has come under treatment early, may be enabled to go three months or so at a time without insulin. Longer than this it is not considered advisable to permit the patient to go, although it is occasionally quite possible—two patients, for instance, having actually been able to dispense with insulin for over a year without return of their symptoms. In the intervals between the "cures" the diet is modified merely to the extent necessary to maintain the glycosuria and blood sugar at the same level as they were whilst the patient was receiving insulin.

Indications.

It is considered that every case of diabetes mellitus presents an indication for treatment by insulin, however slight it be. Contrary to the classical conception of insulin as "a useful adjunct to diabetic treatment," insulin is here considered as the factor *par excellence* of the treatment, dietetic methods being only of secondary importance, and never insisted on to the degree which is at present usual; certainly never to the detriment of the patient's general condition.

In states of pre-coma and coma insulin is naturally indicated, although it is not considered to be by any means the best indication from the point of view of ultimate results, since so much damage is frequently sustained by essential viscera, such as the kidney or the heart, during the presence of a state of acidosis, that delayed collapse is frequent even after apparent cure.

In cases of mild diabetes of the type known clinically as "*gras*" it is considered that insulin treatment should be instituted even when the glycosuria can be controlled, as it generally can, by dietetic methods alone. This is particularly true in the presence of any of the ordinary diabetic complications, such as pruritus or carbuncle. It is in cases of severe diabetes (*maigre*), with acetonuria, that this treatment finds its best indication, if the patients come under observation early,⁴ since in these cases a very real benefit will generally result from repeated "cures," at increasing intervals if this be found to be possible.

Results.

The immediate results of this system of treatment are estimated as the result of observations of (1) the ketonuria, (2) the glycosuria, (3) the general condition of the patient, and (4) his blood sugar content.

1. The effect on the condition of ketonuria, if present, is extremely marked. The ketone bodies of the urine result, it is generally believed, from the incomplete oxidiza-