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ARCHIVES

OF THE

Public Health Laboratory

Of the University of Manchester

EDITED BY

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INTRODUCTION.

During the last fourteen years many researches bearing upon the Etiology of Disease, Preventive Medicine and Public Health have been conducted in the Laboratory either by members of the staff or by research fellows and students. Much information bearing upon questions of general or local interest has gradually accumulated in the Laboratory books. The results of investigations carried out for public authorities have been communicated in the shape of private letters or reports summing up the main conclusions arrived at, but seldom containing a full account of the scientific work upon which these conclusions were based; a few of these reports have been made public. Some papers have been published in various periodicals and several researches have been embodied in dissertations. It has been found impossible up to this day to publish in a connected form an account of the scientific work done in the Laboratory. It seems, however, desirable that some record of this work be published from year to year, partly for the purpose of giving to the public authorities associated with the Laboratory information which cannot be supplied to them in abridged reports suitable for administrative purposes, partly for the purpose of allowing successive Laboratory workers to take full advantage of the labours of their predecessors, and partly also with the object of exchanging ideas with other investigators engaged elsewhere in work similar to our own.

As a teaching department of the University the Public Health Laboratory is concerned in the exposition of knowledge by means of lectures and demonstrations. Some of the lectures given at the Laboratory have been of special interest. With the object of bringing the student of Public Health in direct contact with scientific men whose personal experience and reputation allowed them to speak authoritively upon certain branches of Public Health, we have invited well-known hygienists to deliver, from time to

time, lectures upon subjects which they have made their own. The first volume of the Laboratory Archives is devoted to the publication of lectures delivered during the year 1904, and of a few reports which formed the basis of Demonstrations given by members of the Laboratory staff in connection with this course of lectures. On the occasion of the opening of the Laboratory, on January 27th, 1905, Professor Perroncito, of Turin, contributed an address on Intestinal Parasites, which is also included in this volume. These lectures, it is hoped, will form a suitable introduction to the Archives, for they deal with some of the most important branches of the work conducted in the Laboratory.

Original communications bearing upon diseases which are prevalent in the districts surrounding Manchester, or dealing with food and water supplies, air, disposal of refuse, sterilization and disinfection, etc., will be published in subsequent volumes. These communications will be based upon current work and investigations which have been carried out in the laboratory during the last 14 years for several County and Municipal Authorities.

The Editor will endeavour to publish the Archives in such a form as to facilitate reference to various questions which have proved of special importance or interest in the North-western Districts of England. The editorial work in connection with the first volume has consisted mainly in the selection of subjects, the arrangement of the text, the translation of Professor Perroncito's lecture, and the preparation of the index. No alteration has been made in the text without the sanction of the authors, who have been given the opportunity of revising finally their respective communications.

SHERIDAN DELÉPINE.

LECTURES AND OTHER COMMUNICATIONS CONTAINED IN VOLUME I.

Industrial Diseases due to certain Poisonous Fumes or Gases Phosphorus, Sulphuretted Hydrogen, Carbon Monoxide

BY

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Industrial Disease Due to Certain Poisonous Fumes or Gases.

Introduction.

Noxious gases or fumes are generated in the course of various industrial or other operations, and persons engaged in those operations are liable to certain forms of acute or chronic poisoning, to prevent which the State has sometimes to interfere. It is impossible in one hour to deal with the whole subject of industrial diseases due to gases or fumes. In this lecture three among the most important forms of intoxication are dealt with, namely poisoning by phosphorus, by sulphuretted hydrogen and by carbon monoxide.

I.—POISONING BY PHOSPHORUS.

Lorinser's (1845) and Samuel Wilks' Observation.

The discovery of the lucifer match marks an important epoch in civilization. It is difficult to say exactly where lucifer matches were first made, whether in Stockton in this country or on the Continent. They were made in Vienna in 1834, and we know that in Austria their manufacture had not proceeded very far when workers in the Industry began to suffer in health. In 1845, *i.e.*, eleven years after the commencement of the trade in that country, Lorinser, of Vienna, published a paper on phosphorus necrosis in match workers. Between 1839 and 1845, nine cases of necrosis had come under his observation. About this period Dr. (now Sir) Samuel Wilks had, in our own country, reported upon disease of the jaw-bone in a lucifer match maker.

Phosphorus is allotropic and exists in two forms (1) white or yellow, (2) red phosphorus. It is the white phosphorus used for heading ordinary strike-anywhere matches that is dangerous. From red phosphorus are made safety matches or those which strike only on the box. White or yellow phosphorus has been known for more than two centuries. To Brandt, of Hamburg, belongs the honour of its discovery in 1669. Phosphorus is made from bone ash by acting upon it with sulphuric acid, filtering and evaporating the product, heating this with charcoal and subsequently distilling it. Commercial phosphorus occurs in the form of white sticks 8 or 10 inches long, which become yellow on exposure to the air. During the absorption of oxygen from the atmosphere phosphorus glows or exhibits what is known as phosphorescence.

Toxic Properties of Oxides of Phosphorus.

In the act of becoming oxidized, phosphorus and oxides of phosphorus are formed. The greenish-white light evolved during phosphorescence can be checked by such oils as eucalyptus and turpentine. Phosphorus is extremely inflammable, it ignites at a temperature of 34°C. and forms, with a plentiful supply of air phosphoric oxide P_4O_{10} and, with a limited supply of air phosphorus oxide P_4O_6. On analysing the fume given off by phosphorus 8/10ths are found to consist of oxides of phosphorus. Until recently the whole of the strike-anywhere matches in this and other countries were headed by a

paste containing white phosphorus, and as this substance is volatile at ordinary temperatures it is to this fact, and the readiness with which the phosphorus enters into combination with oxygen, that the ill-health of workers in match factories has been attributed. The lower oxides of phosphorus are apparently more noxious than the higher, but upon what the specific action of the former depends it is difficult to say. Phosphorous oxide undergoes oxidation and in contact with moisture becomes phosphorous acid which is both a strong reducing agent and an active solvent. The peculiar odour of phosphorus is caused by a mixture of ozone and phosphorous oxide.

Phosphorus fumes are known to be harmful to animal tissues, including bone. Prof. Thorpe found, on exposing decayed teeth to the fumes of phosphorus for 12 hours that they lost 0·37 per cent. of their weight, also when carious teeth were crushed and exposed to a dilute solution of phosphoric acid (1 per cent.) that they lost 8·9 per cent. of their original weight.

In the air of a dipping room of a match factory Thorpe* found 0·02 milligramme of phosphorus per 100 litres of air, while the same quantity of air of the boxing room contained 0·12 milligramme of phosphorus. After working on an average four hours each, 22 employées in a match factory were caused to wash their hands in a particular basin of water. On analysing the water Thorpe found 37·3 milligrammes of phosphorus. The same observer showed that the composition of smoke produced in the burning of phosphorus matches depends to some extent on the conditions of combustion. In the case of a single match, for example, freely exposed to the air the smoke consists, or rather the phosphoric element consists, of phosphoric oxide P_4O_{10}, but in the "smothered" combustion of a lot of matches, such as frequently occurs in the boxing of matches, a considerable proportion of phosphorous oxide P_4O_6 is formed.

In the Match Factories of Great Britain and Ireland, during 1898, 4,270 persons were employed. Of these 1,166 were males and 3,104 were females. About 1,700 were working in phosphorus processes. The workers under 18 years of age were 466 males and 1,077 females. The match works were distributed as follows:—

England and Wales	17
Scotland	2
Ireland	5
Total	24

The number of match works in January, 1901, were:—

No. in which yellow phosphorus used	15
,, ,, ,, ,, discontinued	3
,, not yet working	1
,, closed temporarily	5
Total	24

* Reports to the Secretary of State for the Home Department on the "Use of Phosphorus in the Manufacture of Lucifer Matches," by Professors T. E. Thorpe, T. Oliver and Dr. Geo. Cunningham. Messrs. Eyre and Spottiswoode, 1899.

On 15th December, 1903, Dr. Whitelegge informs me that the number of match factories is 15. In 1901 the numbers of male and females employed in 12 factories were 868 and 2,604 respectively. Three firms (not large) had not sent in their returns. The declension in the number of match factories and of persons employed is explained by the absorption of some of the smaller factories, the greater use of machinery, and improvements in the methods of manufacture.

Processes Dangerous to Health.

The manufacture of phosphorus at Oldbury, near Birmingham, is attended with little risk to health as it is carried on mechanically and in covered-in vessels. Necrosis, however, is not unknown there. Ordinary white phosphorus is now made electrolytically in Pittsburg, probably, too, in England, and in France, on a small scale. The dangerous processes in a lucifer match manufactory, in which white phosphorus is used, are mixing, dipping, and boxing. The drying of matches is generally carried on in closed spaces from which the fumes are carried away outside the works altogether, so that this process in the manufacture is no longer dangerous to health. The paste for heading ordinary strike-anywhere matches is composed of white phosphorus, chlorate of potash, glue, ground glass, and colouring matter. In some factories the white phosphorus has been replaced by a, comparatively speaking, harmless substitute, viz., the sesquisulphide. In Britain frequently the paste does not contain more than 5 per cent. of white phosphorus, on the Continent it may contain treble that quantity or more. Formerly the phosphorus paste was mixed in an open mortar, now it is usually made in closed vessels that are well ventilated. The dipping of matches is a very simple operation. The "compo," as it is called, is spread upon an iron plate or table. Into this paste the ends of wooden splints projecting from a frame are dipped. Only male dippers are employed in this country. Owing to improved ventilation, and the use of fans, dippers can follow their employment for several years without experiencing any ill-effects. Years ago, before the introduction of adequate ventilation, a large number of dippers used to suffer from the effects of phosphorus poisoning owing to inhalation of the phosphorous oxide, formed during the slow oxidation of the paste on the dipping table. Similar oxidation occurs in the drying chambers to which the dipped matches are taken, but these chambers are well ventilated, and in Britain as a rule no person enters them. In the boxing-room women do the bulk of the work. Lifting up a handful of matches, they rapidly fill the boxes, and so dexterous do they become at this work that the number of matches in each box if counted would be found to be the same. Notwithstanding the aptitude of these women and the care they exercise, the matches they are handling often take fire. Although the flames are at once extinguished by wet cloths which are ready to hand, there is a considerable amount of smoke given off. We have already alluded to the chemical composition of this "smothered" smoke. The smoke irritates the throat, and makes the eyes smart. After a few hours' work in a boxing-room, the hands of the women are not only deeply stained by the dye from the match heads, they smell strongly of phosphorus, and in the dark are luminous.

Acute Phosphorus Poisoning is practically unknown in lucifer match makers; it is not an industrial disease. Phosphorus is an extremely fatal poison, whether it is swallowed or administered hypodermically. One-tenth of a grain, according to Zobel, of Jena, has caused death, while a fairly large piece of white phosphorus is stated to have passed down the length of the alimentary canal of a dog without causing symptoms. This, however, must be a most unusual event. Phosphorus produces its disastrous effects equally upon animals and men. When taken by the mouth the symptoms are those of irritant poisoning. The rapidity with which the symptoms develop depends largely upon the amount, and kind, of food present in the stomach. There is usually extreme pain at the epigaster, accompanied by vomiting, often coffee ground in character, and garlicky in odour. Three or four days afterwards jaundice develops in about 80 per cent. of the cases, and gradually deepens. There is considerable depression both mental and physical. The patient feels extremely wretched owing to pain in the abdomen and vomiting; he may pass into a state of somnolence which gradually deepens into coma, in which he dies. Women, if pregnant, miscarry and frequently die after the event. The liver in the early stages of acute phosphorus poisoning is usually found enlarged; later on it is diminished in size, and is olive green in colour and on section is greasy. The kidneys are large, pale, and fatty. There are numerous ecchymoses all over the body, in the pleura, pericardium, heart and muscles. On microscopical examination the cells in the liver and kidneys are observed to have undergone extensive fatty degeneration, while the cardiac muscle is seen to have lost its transverse striation.

This is acute phosphorus poisoning as it occurs in the accidental and suicidal forms of the malady. Persons breathing the vapour of phosphorus suffer in time from chronic phosphorus poisoning, but how the poison acts is not exactly known. In the ordinary metabolism of the body proteid is split up into a nitrogenous portion which is thrown off by the kidneys and a non-nitrogenous which is resolved into carbonic acid and water, and these are eliminated by the lungs and kidneys. "When a starving dog, living upon its own tissues, is poisoned with phosphorus, the proteid decomposition as indicated by the nitrogen in the urine is largely increased, while the amounts of carbonic acid given off and oxygen absorbed are largely decreased; on post-mortem examination the organs are found to contain excessive quantities of fat. We have here presumptive evidence that a part of the proteid molecule usually completely oxidized has not been burned but has been converted into fat."* In a word phosphorus interferes with the normal processes of oxidation that occur in living protoplasm. But phosphorus has, in addition, a special action upon the marrow of bone. Stockman and Charteris injected hypodermically into rabbits 1m.gm. of phosphorus dissolved in oil. They repeated the operation on a few occasions. The bone marrow was found to have undergone certain changes commencing primarily in a hyperæmia. The amount of change depended largely upon the length of time the phosphorus had been administered. There were usually distinct atrophy of the fat cells of the marrow and a great increase of the leucoblast marrow cells. These were followed by gelatinous degeneration

* "Text Book of Physiology," Howell, second edition. Vol. i., p. 513.

and a diminution of the giant cells and thickening of the connective tissue. The early changes, i.e., increase of leucoblasts, " indicate a reaction on the part of the marrow for the purpose of dealing with the poison. As the poisoning becomes more severe and obtains the upper hand, the marrow degenerates rapidly and its cellular structure disappears, just as happens in many other poisonings and cachectic conditions." Stockman and Charteris did not find any increase in the thickness of the bone.†

Industrial Phosphorus Poisoning. Phosphorus poisoning in match-makers differs considerably from acute phosphorus poisoning. It occurs under two forms—(1) in which the symptoms are more or less constitutional, and (2) local. To the first form French physicians have given the name of **phosphorisme.** Magitot depicted the malady as he observed it among the match makers at Pantin-Aubervilliers, near Paris, and Arnaud as he saw it in Marseilles. As a consequence of lengthened exposure to phosphorus fumes these writers maintain that there is induced, especially in female workers, a constitutional state characterised by anæmia, a yellow tint of the skin, loss of appetite, albuminuria, dyspepsia and headache followed by progressive emaciation. Although Arnaud‡ found albuminuria present in fully 70 per cent. of his cases of phosphorisme he did not find that the patients became ultimately the subjects of Bright's disease. Match makers have often an unpleasant garlicky odour about them which is mostly given off in the breath and not from the clothes they wear, for it is still present after the workers have had a bath and changed their raiment. One of the paths of elimination of phosphorus from the body is the lungs, and other paths equally important are the kidneys and skin. The odour of the urine in phosphorisme may at times be very unpleasant. The presence of albumin in the urine is explained by the fact of the kidneys being channels of elimination of phosphorus. Albuminuria can be produced experimentally in animals 48 hours after the introduction of phosphorus into the system. It has to be borne in mind that the particular channel by which the poison is introduced into the body is not without some influence in determining what organs shall suffer most. When phosphorus enters by the stomach the liver is the organ first and most profoundly affected; when administered hypodermically the kidneys are the first to suffer, but since in match makers the poisonous fumes are absorbed by the lungs the phosphorus passes into the blood and is eliminated by the breath and kidneys. Falck has described a cerebro-spinal form of phosphorisme, but it cannot be said that there is any decided clinical type. The commonest nervous symptoms are disorders of sensation, e.g., cutaneous or muscular hyperæsthesia, headache, pain in the spine, arms, and legs, accompanied by muscular weakness. Instead of hyperæsthesia there may be anæsthesia or loss of sensation in the lower extremities.

Opinion is divided as to whether pregnant females who are the subjects of phosphorisme exhibit a greater tendency to miscarry than healthy women employed in other occupations. Arnaud did not find that the match makers of Marseilles aborted in an unusually large proportion, a circumstance which

† "The Journal of Pathology and Bacteriology," December, 1903, p. 205.

‡ "Etudes sur le Phosphore et le Phosphorisme Professionel."

is rather interesting seeing that phosphorus matches are in our own country often resorted to as an ecbolic. The children of healthy female match makers in Marseilles were found to be just as strong as those of women following other trades. I have not found during my visits to the match works in our own country or in France and Belgium this form of constitutional poisoning or phosphorisme to any extent. When ordinary precautions are adopted, and the women are not of the very poorest classes, female workers in match factories are not more anæmic than those engaged in other trades.

Lucifer match making therefore does not appear to influence very adversely the general health where ventilation is good and personal cleanliness is observed. Men and women may work from 10 to 30 years without suffering either in their general health or from any local trouble, and yet, on the other hand, it must be admitted that in some of the workers there are induced physical changes in their body which create a predisposition to disease by diminishing the general and local resistance to infective organisms. Probably it requires a lengthened exposure to phosphorus fumes for this predisposition to be developed, but other agents may be co-operating, e.g., chloro-anæmia, bad food and alcohol, to bring about the altered state of body just referred to.

Phosphorus Necrosis or Phossy Jaw. The one malady which is special to lucifer match makers, and which has gained for the trade a bad name is necrosis of the jawbone, called in this country "phossy jaw" and in France "mal chimique." The disease usually begins with pains in the face, attended by a localized swelling of the gum, and followed by an abscess which may burst of itself, or be opened by a surgeon. The escape of pus is not succeeded by resolution, for the suppuration continues and there remains a fistulous opening. A suspected tooth is removed but even this does not lead to a cure for other teeth probably fall out, the suppuration continues, and months afterwards a sequestrum of bone is removed. There is nothing about this form of necrosis that is special to phosphorus poisoning unless it be the extreme slowness and the indefinite limit to which the disease may extend. Wounds in the mouth of match makers who have been exposed to phosphorus fumes heal very slowly. The wound caused by the extraction of a tooth in a presumably healthy match maker, instead of closing over in a few days, remains open for weeks or months, even if he absents himself from the factory. On the other hand, should the individual in this condition continue to follow his occupation, he is running a tremendous risk of becoming the subject of phosphorus necrosis. It is this defect in the processes of repair that suggests the existence of some local influence left upon the tissues by phosphorus fumes. In many match makers the saliva is acid, a circumstance which would not only tend to decalcify the teeth and favour the development of caries, but might at the same time exaggerate the virulence of microbes in the mouth. The defective processes of repair in the mouth may be due to the absence of diapedesis of the white corpuscles of the blood,and a diminution of phagocytosis. Arnaud is of opinion that the slowness of the cicatrization of wounds in match makers is not confined to the mouth but is also exhibited by wounds on the extremities, e.g., those caused by burns. Although there is nothing in phosphorus necrosis of the jaw different from that which occurs in syphilis and

in tubercle, there may yet be something in the anatomical condition of the jaw bones of man which render these bones specially liable to this affection, compared with animals upon whom experiments have been made to produce maxillary necrosis. It is an extremely difficult thing to induce necrosis of the jaw bone in animals even after submitting them for weeks or months to the fume of white phosphorus, to the fume of dry matches, or to the smoke from burning matches. Not even after the extraction of a tooth or two in a rabbit or a dog, laceration of their gums and subsequent exposure of the thus bared alveolar cavity to phosphorus fumes, do we find death of the bone readily brought about in animals. There is something that is peculiar to the human subject as regards his liability to phosphorus necrosis compared with the lower animals, but what that something is it is difficult to say. I have never succeeded in causing it, neither has Stubenrauch of Munich, nor Stockman of Glasgow, and yet a very small whiff of phosphorus fume in man may be followed by very painful if not serious symptoms. A patient of mine, a short while ago, lit his pipe and began to smoke. He had scarcely taken a draw or two when he complained of a most unpleasant taste and odour in his mouth. He continued smoking. Next day he had, what was most unusual to him, viz., violent toothache. On the day succeeding this his gums were suppurating, the teeth on the side he had held the pipe were tender and his jaw-bone was painful. The dentist he consulted could not explain the peculiar pathological state of the mouth. He was obliged, however, to remove five teeth, and although these were slightly carious they had never until the particular smoke of two days previously caused their owner the slightest trouble. An examination of the pipe was made, when it was found that a wax vesta, with its head toward the mouthpiece, had slipped into the rather wide canal of the pipe, and as there had been no trouble before, nor since the particular occasion referred to, and the peculiar state of the mouth was of such a character as to be outside of the ordinary routine of the dentist's experience, we cannot eliminate phosphorus fume from having played a special part in causing the pain in the teeth and the suppuration of the gums.

What then is the Cause of Phosphorus Necrosis? Wegner found that if he bared the tibiæ of dogs and exposed the animals to phosphorus fumes the periosteum and the bone became affected. This would suggest that there can be no necrosis without a channel of entrance for the phosphorus fumes, but as the malady has frequently developed in match makers after they have left off working in a factory, this circumstance would point to some additional coincidence, such as, for example, a general predisposition. There must be in operation (1) a causative agent, (2) a channel of entrance, and (3) a constitutional and local predisposition. There is a general consensus of opinion that more than one causative agent is at work. Although Stubenrauch placed white phosphorus into holes drilled through the teeth in the lower jaw of dogs and cemented the opening, he found that only the most limited necrosis occurred, and that it never extended as it does when necrosis is similarly induced by arsenic. It is more than likely therefore that the harmful agent is not so much phosphorus *per se* as its lower oxides. Add to the operation of phosphorus fumes, in the form of lower oxides, that of carious teeth providing

a channel by which these oxides may reach the alveolar cavity of the jaw, and we have, according to Roussel, who as far back as 1846 insisted upon dental caries as a primary necessity, an explanation of the necrosis and its occurrence in the jawbone.

Over and above these, additional local influences are probably at work. There are always myriads of micro-organisms in the mouth, some of which may be rapidly transformed by alteration of their surroundings into microbes of a most virulent character. Infection may therefore play a part. Phosphorus fumes by diminishing local resistance may prepare the way for the operation of infective micro-organisms. That the fumes themselves can inflict considerable local damage is clearly demonstrated by the case of the tobacco smoker already alluded to. If we accept the microbic theory, then phosphorus necrosis would be the result of an infective osteitis.

Professor Stockman, of Glasgow, found a few tubercle bacilli in the pus that escaped from a fistulous opening of the necrosed jawbone of a match maker. Stockman looks upon phosphorous necrosis as really a tubercular process. In the specially stained specimens of pus, similarly obtained from the necrosed jawbones of match makers, I have never succeeded in finding tubercle bacilli. Without therefore denying the possibility of the malady being tubercular I am disposed, owing to the large number of pus and other organisms in the mouth to regard phosphorus necrosis as the result of a multiple infection, in which tubercle when present is probably secondary.

It is more than likely that local causes are supplemented by a constitutional predisposition. In other words, the general health of the individual, as in ordinary infectious diseases, has something to do with local developments. Many match makers are exposed to phosphorus fumes for years, and yet it is only very few who suffer from necrosis of the jaw. In addition to penetrating a carious tooth, or acting upon an exposed alveolus and of thereby inducing local irritation, phosphorus fume is also capable of causing a mild form of constitutional poisoning, which if it does not always amount to phosphorisme is still capable of directing a local infection, by diminishing tissue resistance. Considerable time may be required before phosphorus can produce this general predisposition. Possibly it is this alteration of the general constitution that explains the difficulty of inducing in animals anything like phosphorus necrosis as met with in man. In order to bring about conditions as nearly as possible similar to those which men and women are exposed to, Stubenrauch, in his efforts to produce necrosis experimentally in animals, placed dogs in match works for six months and allowed them to breathe the air of the drying rooms daily from 6 a.m. till 6 p.m. In one dog, a wound was made by a surgeon on the forehead which exposed the periosteum, a small portion of the gum of the lower jaw was removed from another, two teeth were extracted from a third dog, while in the case of a fourth dog the artery entering the foramen of the inferior maxilla was tied. Not one of the animals suffered except the dog whose artery was tied. This animal developed a suppurating osteitis. Stubenrauch therefore concludes that phosphorus fume *per se* cannot be the cause of necrosis, that the malady is probably the result of infective organisms acting upon bone whose blood supply has been cut off, *e.g.*, by thrombosis, hence the greater frequency of phosphorus necrosis in anæmic women, and in those who have had hæmorrhage.

In Stubenrauch's experiments the animals were not exactly under the same conditions as men and women in a match factory, where, in addition to inhaling phosphorus fume, many of them are handling all day long dry matches, or standing over a dipping table.

That some constitutional change is induced as the result of the long-continued inhalation of phosphorus fumes is confirmed by the occurrence of spontaneous fracture of the long bones of match makers. When visiting the match works at Grammont, in Belgium, I had an interview with Dr. Brocoorens, medical officer to the works, who informed me that in 25 years he had treated 30 cases of fracture of the leg occurring spontaneously in men who were dippers, and who had previously suffered from necrosis of the jaw. In England this accident is comparatively rare to what it has been in Belgium. Dr. Garman, of Bow, who is medical officer to Messrs. Bryant and May, told me that he knew of two cases, and Dr. Dearden has reported two cases of double fracture of the thigh in match makers. All the accidents occurred under the most trivial circumstances. It would seem therefore as if phosphorus fume was capable not only of causing necrosis but of creating a constitutional state or cachexia, the outcome of deranged metabolism, or of causing structural changes in internal organs whereby a degree of fragility of the long bones is induced which makes them break on very slight exertion. Dearden * found that the relative proportion of phosphoric acid to lime is greater in the bones of match makers than in those of healthy persons, and is of the opinion that a chemical change in the bone precedes the necrosis. Gautrelet, a French chemist, gives it as his opinion based upon analysis that phosphorus necrosis is due (1) to a general condition of poisoning consequent upon hyperacidity of the blood, and (2) to a local action consisting in degeneration of the bony tissues with multiplication of the marrow cells.

Prevalence of Industrial Phosphorus Poisoning. The total number of cases of phosphorus poisoning in Britain coming under the provision of the Factory Act, and of which there are definite records during the 20 years ending December, 1899, is 102, and of these 19 terminated fatally. Since then the following cases have been notified to the Chief Inspector of Factories:—

	Cases.	Deaths.
1900	4	0
1901	4	0
1902	3	1
1903	0	0
From 1880–1903	113	20

Compensation to Lucifer Match Makers. In England lucifer match makers who have become ill through following their employment may receive voluntary help from their employers. There is no compulsory compensation they can claim. The money cost to Britain through industrial phosphorus poisoning has practically speaking been nil, compared

* "British Medical Journal," 1899. Vol. ii.

with the demands made upon the Treasury in France. The manufacture of matches is in France a Government monopoly. The principal match works are in Pantin-Aubervilliers, just outside of Paris, and in Marseilles, but the industry is also carried on at Begles, Saintimes, Trelayé and Aix-en-Provence. In 1896 there were working at Pantin 712 persons—78 men and 634 women—and at the time of my visit two years afterwards there were 400 women and 200 men. When I visited the Prado factory, Marseilles, in 1898, there were working 540 people, 460 women and 80 men. A few years previously the State had not only taken over all the match works in France, but had undertaken to indemnify the workers suffering from industrial phosphorus poisoning. Either as a consequence of this benevolent act, or as a coincidence, the number of reported cases of industrial phosphorus poisoning rose so high that in 1896 the French Government appointed a small Commission to enquire into the state of health of 226 workpeople who were said to be ill through having worked at Pantin. At the close of the year 1894 there were 32 cases of phosphorus poisoning. At the end of the following year the number had risen to 125, and to 226 at the close of 1896, or nearly one-third of the effective force of the factory. The French Government at this time was paying into a fund at the bank a sum of money to each match maker equal to 40 per cent. of his wages, quite apart from other gratuities that were allowed. To those in this country who are interested in compulsory compensation as applied to dangerous trades the following table shows how the disbursement of public monies was affected by the adoption of the measure in France.

Year.	Total Amounts Paid to Workers in Match Factories.
1890	572 francs 39 c.
1891	1,457 „ 12 „
1892	3,740 „ 59 „
1893	15,641 „ 64 „
1894	29,944 „ 69 „
1895	115,305 „ 26 „
1896	384,283 „ 83 „

It was scarcely to be expected that this increasing expenditure of public money could go on without attracting attention in Parliament. Not only did the extravagant payments lead to the appointment of a small Commission of Enquiry but it forced the hands of the Government to ascertain whether ordinary strike-anywhere matches could not be made from some substance other than the dangerous white phosphorus. In our own country, about the same period, there was considerable expression of public feeling against the manufacture of lucifer matches, owing to the sudden disclosure of a number of cases of phosphorus necrosis that had not been notified. It was this circumstance that brought Professor Thorpe and myself into official connection with the Home Office and supplied us with the opportunities of visiting match works both at home and abroad. In my Report to the Home Secretary there occur the words "There is no doubt that so long as ordinary white phosphorus is used in match works, even with all known precautions, absolute freedom from risk cannot be guaranteed to the workers. Total prohibition of the use of white phosphorus

is therefore the simplest and readiest way to obviate danger." At that time England was brought face to face with the question either of prohibiting the use of white phosphorus altogether as Denmark had done, or of producing a strike-anywhere match without white phosphorus. I had watched during my later visits to France the scientific and practical experiments that were being made in that country and the results that were being obtained. It was demonstrated that lucifers could be made, possessing all the qualities claimed for the ordinary strike-anywhere match, from the harmless sesquisulphide of phosphorus. It only required time to test the durability of the matches thus made. Nearly six years have elapsed since then, and what is the result? Not only has phosphorus necrosis disappeared from France, and the stringent regulations as regards match factories been withdrawn, but in our own country, in consequence of the substitution of a comparatively speaking harmless form of phosphorus for the deadly white by our largest manufacturers, industrial phosphorus poisoning in Britain has materially diminished, and match making has ceased to be the dangerous occupation it was. In addition to improvement in the health of match makers there has been, speaking for my own neighbourhood, a marked diminution in the number of cases of suicide from phosphorus poisoning. Not a year used to pass without several patients being admitted into the Newcastle Infirmary suffering from suicidal phosphorus poisoning, due to having drunk water in which lucifer matches had been soaked, and of these patients one or two would probably die. For the last two years, although cases of match poisoning have been admitted, the patients no longer present the serious symptoms as formerly, not because the cases are better treated outside before their admission into the Infirmary, but because many of the matches sold in the shops are not made from white phosphorus.

The Present Hygienic Aspect of the British Lucifer Match Industry. The lucifer match manufacturers of Britain are to be congratulated upon the effects of the improvements introduced into their factories as seen in the improved health of their workpeople. By having substituted comparatively speaking harmless compounds for dangerous white phosphorus, necrosis of the jaw and phosphorus poisoning in match makers are becoming each year rarer and rarer events. Messrs. Bryant and May, who are the largest manufacturers, have discontinued the use of white phosphorus in their works at Bow, and since doing this, the health of their employées has been extremely satisfactory. By the introduction of machinery whereby the mixing, dipping, drying, and boxing are done mechanically in large and well-ventilated rooms, the Diamond Match Company at Liverpool have demonstrated how the hygiene of match making can be promoted. Thus once again it is demonstrated how, under the stimulus of human necessity, science not only points the way but has shown herself capable of solving some of the numerous chemical problems that are constantly arising in the course of British industries. However satisfactory these industries may appear to be on the surface, they are like man himself ever undergoing a process of evolution and thus tending towards perfection.

II.—POISONING BY SULPHURETTED HYDROGEN.

Death from breathing sulphuretted hydrogen is fortunately a rare occurence, and yet in my own neighbourhood 18 months ago, and within the space of five weeks, this gas caused the death of four healthy men and imperilled the lives of three others. Sulphuretted hydrogen or hydrogen sulphide is a colourless transparent gas, burning with a blue flame; it has the peculiar odour which is given off by rotten eggs. When present to the extent of 1 in 100,000 of air it is recognizable by this unpleasant odour. It is readily soluble in water. At ordinary temperatures, water is capable of absorbing three volumes of the gas. Sulphuretted hydrogen is occasionally present in considerable quantities in the sewers of towns and in cesspools. It is evolved from the tank waste thrown out of chemical factories, and is given off from the slag of iron works. In chemical laboratories where the pure gas is made, inhalation of sulphuretted hydrogen by students, inattentive to ventilation, has been followed by serious symptoms.

Mode of Occurrence and Lesions.

It was my fortune to be brought into contact with the deadly effects of this gas a little over a year ago. I do not think that members of the medical profession are fully alive to the extreme danger that attends inhalation of sulphuretted hydrogen. In the "Lancet," of January 24th of last year, I published an account of the death of three workmen due to breathing hydrogen sulphide while engaged in making excavations for a graving dock at Hebburn-on-Tyne. One evening in July, 1902, a workman descended an open iron caisson or cylinder at 6-30. There were only a few inches of water in the cylinder at the time, which had oozed out of the surrounding soil largely made up of chemical waste and iron slag. This man had only been in the cylinder a few minutes when screams having been heard a fellow workman went to his rescue, but he could hardly have reached the scene of the accident, when screams again were heard. A third workman then descended the caisson; he too was heard to shout and immediately all was silence. In the space of a very few minutes three men were lying dead at the bottom of a cylinder which contained, as I have said, only a few inches of water. Notwithstanding numerous efforts made by the manager and gangs of workmen, some of whom were lowered down by ropes into the cylinder, it was found impossible that evening, on account of the over-powering effects of the gas, to remove the bodies. As naked lights burned well in the cylinder it was clear that the gas was not carbonic acid. Next morning the corpses were recovered, and on the following day, assisted by Mr. Malcolm, of Hebburn, I made a post-mortem examination on two of the bodies.

(1) A man 23 years of age; strongly built, muscles well developed; face pale and cyanosed; body not decomposing. On opening the cavities no special odour perceptible. Heart: right side flaccid and empty; left side hard, empty and contracted. Lungs œdematous and pale. Liver dark; abdominal viscera healthy. Blood fluid and dark.

(2) Youth aged 19, well developed; rigor mortis still present; hands clenched as if death had occurred during a convulsion; face cyanosed. No odour of

sulphuretted hydrogen on opening the cavities of the body. Heart: right side flaccid and empty; left side firmly contracted, hard and empty. Lungs pale and œdematous. Liver dark; abdominal viscera healthy. The blood was dark and liquid.

On making a spectroscopic examination, the blood, in both instances, gave the spectrum of ordinary oxyhæmoglobin, and as it was readily reduced by ammonium sulphide it was therefore quite free from carbon monoxide. As the air in the caisson smelt strongly of sulphuretted hydrogen I submitted some of the water taken from the bottom of the cylinder to Professor Bedson of the College of Science, who reported that in each 100 volumes of water there were 12·2 of sulphuretted hydrogen.

Five weeks after the death of these three men another accident occurred in the caisson whereby one man lost his life and three other workmen nearly theirs. On this occasion, owing to the progress of the excavations, the cylinder had sunk deeper into the earth, and it now contained 46 feet of water. A diver who had been at work had signalled that he was coming up, and was about to emerge on to a wooden platform in the caisson when a workman who had been standing there leaned down to open the diver's helmet, but being overcome by the fumes fell on to the diver in a state of unconsciousness. Help was shouted for, and in a very few minutes two other workmen were in the cylinder trying to render assistance. All four men, however, were overcome by the gas and had to be rescued. When they were removed it was found that the man who had tried to unscrew the diver's helmet was dead. By means of inhalations of oxygen, hypodermic administrations of ether and liquor strychniæ, warmth externally, etc., Dr. A. M. Walker, of Hebburn, succeeded in restoring the diver and two of the rescued workmen.

Shortly after the second accident, Professor Vivian B. Lewes, of London, was asked to report upon the chemical conditions present in the cylinder which at the time contained water to within 16 feet of the surface. Four hundred gallons of this water when agitated with air were found to be capable of giving off one cubic foot of H_2S, therefore it only required 800 gallons of the water to be agitated in order to yield sufficient gas to render the whole of the air in the cylinder fatally poisonous. As the depth of the water in the cylinder was 48 feet there would be approximately 37,000 gallons present. Very little agitation would be required to evolve a dangerous quantity of sulphuretted hydrogen. The ascent of the diver would be more than enough to do this. Lewes recommended a clearing away of the soil, over the area to be occupied by the dock down to the bed of clay, in the hope that by thus exposing the whole of the sulphuretted hydrogen yielding area to air, the gas would be oxidised and rendered harmless. This was done by the contractors, and it is gratifying to know that no further fatalities occurred.

Mode of Action

Here then within the space of five weeks were four men whose lives were suddenly terminated by breathing gas the nature of which was at once suggested by the peculiar odour that prevailed at the place. With the aid of my colleague, Dr. R. A. Bolam, I made a few experiments to test the toxicity of sulphuretted hydrogen. Until then I had had no experience of H_2S being

such a powerful poison. When we placed a dog in an atmosphere containing 0·15 per cent. of sulphuretted hydrogen, the animal very shortly afterwards, and without any signs of distress or warning, became rigid and fell apparently dead, its breathing having ceased. On removing the dog from the chamber, as its heart could be heard beating at very long intervals, artificial respiration was adopted, and by degrees life was restored. Some little time after this, on re-exposing the dog to an atmosphere containing 0·15 per cent. of sulphuretted hydrogen, apparent death occurred in 1min. 40sec. Death was in this instance preceded by a strong general muscular spasm and by cessation of respiration, but again life was restored. Subsequent exposure of the animal to an atmosphere containing 0·2 per cent. of the gas killed it suddenly and painlessly. Death appeared to be due to a strong impression made by the gas either upon the respiratory centre in the medulla, or upon the termination of the vagal nerves in the lungs, for the heart continued to beat, feebly and at long intervals, after the animal was apparently dead. At the autopsy made shortly after death, the heart was found to be flaccid, dilated and filled with liquid blood on both sides. The lungs were pale and presented nothing abnormal. The blood on examination gave the spectrum of oxyhæmoglobin and was easily reduced.

In order to ascertain whether blood if exposed to H_2S for a sufficient length of time would exhibit any particular spectrum we exposed to the gas some defibrinated ox blood freely diluted, and we found that after one minute's exposure the liquid became chocolate coloured and then green. On examining the altered serum, although it gave the spectrum of methæmoglobin, viz., one band in the red and two in the green, the hæmoglobin became slowly reduced after the addition of ammonium sulphide. Renewed experiments demonstrated to Bolam and myself that when sulphuretted hydrogen acts only for a very short time upon blood methæmoglobin is not immediately formed. It is probable therefore that in fatal cases of poisoning by sulphuretted hydrogen, owing to death coming so quickly, man is not exposed long enough to the gas for methæmoglobin to be developed, a circumstance which explains the absence of the spectrum of methæmoglobin in the blood of persons who have died from inhalation of the gas.

Toxity of H_2S.

Since H_2S is a frequent constituent of the air of sewers it is important to bear in mind the toxicity of the gas. The gaseous emanations from sewers when submitted by Layet to chemical analysis were found to contain sulphuretted hydrogen, ammonium, sulphide, carbon dioxide, nitrous oxide and phosphoretted hydrogen. According to Lehman* an atmosphere which contains 0·7 to 0·8 of H_2S per 1,000 litres of air is dangerous to human life, while air containing 1 to 1·5 per 1,000 destroys life rapidly. Vivian Lewes states that man is killed in $1\frac{1}{2}$ minutes after breathing air containing ·2 per cent. of H_2S. The sudden death of men when working in sewers is in most instances due to sulphuretted hydrogen. When only minute quantities of this gas are present

* "Archiv für Hygiene," 1892. Band xiv., p. 135.

in the sewer the workmen complain of vertigo, headache, and malaise w disappear shortly after the men have been taken out into the fresh air, occasionally it happens that a workman in the sewers suddenly falls a struck down in apoplexy and in a few seconds life is found to be ext Decomposition of fæcal matter is one of the principal sources of hydr sulphide in the sewers, but the air of these canals may be vitiated by c poisonous gases, e.g., carbon monoxide, which, coming from a leak in a that is carrying gas for illuminating purposes, has escaped into the soil a aspirated into the sewers.

Types of Poisoning and Treatment.

Two types of poisoning by sulphuretted hydrogen are met with. In form of poisoning death is immediate; the individual falls down dead struck by lightning; the pupil is dilated, the limbs are firmly contra breathing is arrested, but the heart still beats occasionally; the blood is c but its hæmoglobin is unaltered. Death under these circumstances is du the action of the gas upon the respiratory nerve centres. In the other for poisoning, death comes more slowly. To the nervous phenomena desc there are added those caused by asphyxia. The blood is not only dark bu hæmoglobin may be altered, while the urine may contain albumen or suga

No matter then what kind of work, at a particular time, men ma engaged in, detection of the odour of sulphuretted hydrogen should be rega as a danger signal. As the gas is heavy it lies low in confined spaces. Sa consists in the freest ventilation possible, but before allowing men to wo suspicious places it would be well to expose therein a piece of white filter p soaked in a solution of a lead or silver salt, blackening of which shoul regarded as prohibitory. Cylinders of oxygen should be at hand, and in event of men being overpowered by the gas, artificial respiration carried away from the immediate locality, should be resorted to, liquor stryc injected, and warmth applied externally.

III.—POISONING BY CARBON MONOXIDE: COAL GAS, WATER GAS.

Presence of Carbon Monoxide in Various Products.

Carbon monoxide is a very poisonous gas. It is formed when charco burned in an insufficient quantity of oxygen, and it owes its toxic properti the fact that the gas enters into direct combination with the hæmoglobin o blood, forming with it an extremely stable compound; once this has taken p the blood is useless for respiratory purposes. Carbon monoxide is colou odourless and tasteless. When it has caused poisoning it has generally bee association with other gases, one of the commonest of which is coal used for illuminating purposes, in which it may be present to the exte 5 to 10 per cent., while water gas, also an illuminant, may contain from 30 per cent. of carbon monoxide. It is the carbon monoxide which confers these gases their toxic properties. The products of combustion of these g do not contain carbon monoxide. The gas is present in the fumes give

from coke ovens and from charcoal stoves. Carbon monoxide unites with nickel to form nickel-carbonyl, an extremely toxic liquid as the recent deaths of workmen at Clydach show. Injurious effects from breathing carbon monoxide are sometimes observed in limestone burners and cement workers, coal miners after the explosion of fire-damp, coal gas makers, distillers of coal tar, lamp-black makers, labourers employed in iron smelting works, laundresses who use irons heated by gas, brickmakers, also in occupants of houses into which there has been an escape of coal gas.

Mode of Action. The hæmoglobin of the blood absorbs carbon monoxide in the same proportion as it does oxygen. When oxyhæmoglobin is treated by carbon monoxide this gas is substituted volume for volume of the oxygen that is displaced, the only difference being that a more stable compound is the result. The carboxyhæmoglobin thus formed, although capable of resisting the action of sulphuretted hydrogen, can yet be decomposed and its place taken by nitrous oxide. An atmosphere which contains 0·14 per cent. of carbon monoxide will transform half of the colouring matter of the blood into carboxyhæmoglobin. Carbon monoxide is a much more powerful poison than carbon dioxide.

The blood in carbon monoxide poisoning exhibits a beautiful cherry red colour, so, too, do the internal organs and the muscles of the body, and yet, although the blood has a redder tint than in health the tissues are unable to abstract oxygen from it, on account of the strong attachment that exists between carbon monoxide and hæmoglobin. In carbon monoxide poisoning therefore a person dies simply from want of oxygen. On examining the blood spectroscopically two bands are seen between the D and E lines, not unlike those observed in oxyhæmoglobin, but when the blood is subjected to the influence of such reducing agents as Stokes' fluid or ammonium sulphide, it is found that no reduction takes place, the two bands remain the same, whereas in oxyhæmoglobin that has undergone reduction, the two bands have been replaced by a broad one.

Toxic Quantities. Gréhant found that inhalation of an atmosphere containing 1 of CO for 275 of air was fatal to a dog, and that 1 in 70 killed a rabbit. Less than a gramme of carbon monoxide may kill a man. Breathing an atmosphere containing 0·05 per cent. of CO may cause unpleasant if not even serious symptoms. A few minutes after inhalation of the gas its presence may be detected in the blood.

Mode of Occurrence of Carbon Monoxide Poisoning. Illuminating gas is a frequent source of poisoning owing to its escape into a sleeping-room. If this occurs during the night and the sufferer is asleep he passes quietly from ordinary slumber into a state of profound coma, owing to the narcotising influence of the gas upon the nerve centres. Insidious as is the operation of ordinary coal gas, it is nothing to be compared with the subtle action of water gas. In this illuminant which is made by heating coke to a red heat, forcing air up through the coke, shutting off the air and allowing steam to pass down through the mass, subsequent removal of, and purification of, the gas, there are 50 per cent. of hydrogen and often as much as 40 per cent. of carbon monoxide.

Much sought after on account of its heating and brilliant illuminating properties, it is extremely dangerous (1) owing to the gas having little or no odour, and (2) owing to the extremely large percentage of carbon monoxide it contains.

In certain chemical works the manufacture of nickel carbonyl has been followed by rather serious results owing probably to inhalation of carbon monoxide, but as the manufacture is carried on in closed vessels, poisoning of the workmen has generally been due to some accidental and unforeseen breakdown of the machinery.

Men have been found dead near coke ovens and brick kilns, and the cause of death has generally been ascribed, and properly too, to inhalation of carbon monoxide. Within the last three weeks I have had the opportunity of examining the blood of a man aged 54, who was found dead close to a kiln in one of the large cement works on Tyneside. Dr. Inglis of Hebburn who made a post-mortem examination of the body was struck by the placidity of the features, the pallor of the general surface of the body, cherry-red colour of the blood, the rather pink colour of the brain at the junction of the white and grey substance, vivid carmine colour of certain portions of the lungs, the presence of a small quantity of reddish fluid in the pericardium, empty left heart. and the presence of a small quantity of cherry-red blood in the right ventricle. Dr. Inglis sent me a sample of the blood which, on being submitted to spectroscopic examination, was found to exhibit distinctly the two bands between the D and E lines. It was not reduced by ammonium sulphide and heating, a circumstance which clenched the diagnosis of carbon monoxide poisoning that had been advanced.

The Symptoms are due to asphyxia consequent upon want of oxygen to the nerve centres, and they depend upon the percentage of carbon monoxide in the air breathed, the rapidity of breathing, the presence of other gases, and the age of the individual. The quantity of CO present in the air is of more importance than the length of exposure to it. In acute intoxication the individual feels dizzy and complains of headache, noises in the ears, throbbing in the temples, a feeling of sleepiness and a sense of fatigue. There may be a feeling of sickness which culminates in vomiting, a sense of oppression at the chest with quickened or irregular breathing, palpitation, and an inability to stand or walk straight. Convulsions may or may not come on, or there may be only a few muscular tremors. There is a peculiar fixed look about the eyes, the pupils of which are dilated and their reaction slow. Consciousness by degrees is lost or it may be retained for some time, and yet owing to the great loss of motor power the individual, although aware of the danger, is often unable to escape from it. When a man has recovered from the effects of carbon monoxide, his life is still imperilled for some days to come. Not only does he run the risk of dying as late as eight days after the accident, he has still to face the risk of secondary maladies developing, such for example as glycosuria. Hasse found sugar present to the extent of 1 per cent. in the urine of men poisoned by carbon monoxide, and in animals experimented upon the quantity of sugar in the urine often rose to 4·2 per cent. I have known acute symptoms develop and paralysis follow in workmen who, when digging in the street, had been exposed

to the escape of coal gas from the soil impregnated by gas from a leaking pipe. Chronic carbon monoxide poisoning does not reveal itself by a very definite series of symptoms. There may be headache, sickness, diarrhœa, impaired digestion, dry throat, physical and mental depression, also anæmia.

A statement of the symptoms experienced by Dr. Inglis, who went to examine the place where the body of the man was found, whose blood I examined, may not be out of place here. Dr. Inglis could not have been in the place more than 30 seconds when he began to feel giddy, had a sense of throbbing and fulness about the head, and a feeling of great muscular weakness so that he had to catch hold of a workman's arm to save himself from falling. All the way home he had to walk very slowly on account of weakness in his legs and a feeling of oppression at the heart. His pulse, which on an average is 72, was reduced to 60 per minute. It was not until a good night's rest and sleep that he felt himself right again.

Post-mortem Appearances. Features placid, face and skin may exhibit a bright ruddy colour even when the body is decomposing, or be as in Inglis' patient, pale. The blood is of a bright cherry-red colour, both arterial and venous; a similar colour is exhibited by the muscles on section. There may be hyperæmia of the brain and membranes, and on section the white substance of the brain may be more pink than usual. Any serum present in the pleura or pericardium is tinged red. The myocardium is usually pale or red; the left side of the heart is empty, while the right heart may contain a fair quantity of cherry-red blood. The lungs may be emphysematous or there may be hyperæmia or œdema. Red patches may be observed on the surface of the abdominal viscera, and occasionally there are submucous hæmorrhages in the stomach and intestines.

Detection of Carbon Monoxide in the Air. Vogel's method was to agitate a small quantity of diluted healthy blood with some of the suspected air, and to examine the blood spectroscopically. Hempel, on the other hand, used to place a mouse in the suspected atmosphere and shortly afterwards examine its blood. Haldane, of Oxford, has shown that in man the symptoms of poisoning become alarming when half of the hæmoglobin has become saturated with carbon monoxide, or when the individual has been breathing an atmosphere containing 0·2 per cent. of carbon monoxide. About half of the carbon monoxide contained in the respired air is absorbed by the blood as it traverses the lungs. The disappearance of carbon monoxide from the blood when the individual is brought out into the open air, and when respiration is naturally or artificially carried on, is always slower than its absorption during the period of poisoning.

Prevention of Poisoning. To Haldane we are indebted for a very important and practical suggestion bearing upon this question. The time required for symptoms of poisoning to show themselves in warm-blooded animals, breathing a gaseous mixture in which there is a certain percentage of carbon monoxide, is inversely proportional to the value of the respiratory exchanges per unit of weight. This period is

20 times shorter in a mouse than in a man. A mouse will die in three minutes in an atmosphere in which man could live for one hour. The importance of this test therefore is apparent when, after explosion in coal mines, men, in order to rescue if possible their comrades, have to penetrate into the recesses of a coalpit. Carried in a small cage by the rescuing band a mouse is a practical indicator of the danger from this gas. If the animal continues to live the men may proceed, but even if it dies there is yet nearly an hour during which some good may be still effected. At any rate there is always time for the rescuing band to retire with safety to their own lives.

To those desirous of studying this group of diseases the following works may be recommended for consultation :—

"Traité des Maladies Professionnelles," par Dr. Theo. Sommerfeld. Tome 1. Translated by Dr. G. De Geynst. Brussels. Libraire Alfred Costaigne. 1901.
"Journal of Hygiene," Cambridge.
"Poisons : their Effects and Detection," by A. Wynter Blyth. London : Griffin & Co.
"Dangerous Trades," edited by Thomas Oliver, M.D. London : Murray. 1902.
"Récherches Experimentales sur la Respiration," par le Dr. L. G. de Saint Martin. Paris : Octave Doin. 1893.
"Les Asphyxies par les Gas, les Vapeurs, et les Anesthésiqnes," par Brouardel. Paris. 1896.
"Encyclopédie d'Hygiène et de Médicine Publique." Tome VI. Directeur Dr. Jules Rochard. 1894.
"System of Medicine." Vol. II. Article on Phosphorus. Edited by Clifford Allbutt. London : Macmillan & Co.
"Report to the Secretary of State for the Home Department on the use of Phosphorus in the Manufacture of Lucifer Matches." Messrs. Eyre and Spottiswoode. London, 1899.
"Reports to the Home Secretary on Water Gas and Colliery Explosions," by John Haldane, M.D.
"Les Gas du Sang." Gréhant.
"Annales d'Hygiène Publique et de Medicine Légale."
"Maladies Professionelles," Commission d'Hygiène Industrielle. Paris, 1903.
"Etudes sur le Phosphore et le Phosphorisme Professionel," par le Dr. François Arnaud. Paris. Balliere et Fils.
"Dictionnaire de Physiologie." Charles Richet. Paris : Felix Alcan.

Spread of Typhoid Fever, Dysentery, and Allied Diseases among large communities, with special reference to Military Life in Tropical and Sub-tropical Countries.

BY

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Spread of Typhoid Fever, Dysentery, and Allied Diseases among large communities.

Introduction.

It was with some diffidence that I accepted the position I occupy to-day, as I am fully aware of the responsibility attached to it, and not the least has been my difficulty in the selection of a subject on which to address you, which I could hope to present in a manner at all worthy of the occasion. I trust, therefore, the one I have chosen will not be thought foreign to this course of lectures. It is one of great interest to the Military Medical Officer, as well as to those of our own profession, whose lot is cast to live and combat with disease, not only at home but in tropical and sub-tropical climates.

General Considerations.

It is a well-recognised fact that certain diseases prevail in an endemic form within a measured range of distribution over the earth's surface, and from these endemic centres epidemics spread, either in isolated groups or devastating campaigns which have claimed its numberless victims.

Enteric Fever, as regards its frequency and diffusion, marks widely its difference from all other forms of disease; it knows no geographical limits, and its very universality and infectiveness makes it one of peculiar interest to Medical Officers of the Army, who, serving in various parts of the world, have ever to contend against this disease, often assuming as it does an epidemic form.

One of the most interesting points in connection with the literature upon enteric fever is the suggestion that many of the cases which are clinically diagnosed to be enteric, and presumably caused by infection, due to the bacillus of Eberth and Gaffky, are really instances of **infection by the colon bacillus, or possibly by both it and the bacillus typhosus.** Cases, reported by Burch in the *Medical Journal of New York*, May 31st, 1902, bear out this view. His patients all suffered from continued fever preceded by malaise, and invariably accompanied by some gastro-intestinal disturbance; the tongue was dry and foul, the abdomen was usually distended, there was gurgling and pain in the iliac fossa; headache and mild delirium were not unusual. Examination of the blood showed a diminution of leucocytes, while the urine swarmed with the bacillus coli communis. With the Grüber-Widal reaction the serum failed to specifically affect the bacillus typhosus, though it did agglutinate the urinary bacilli. None of the cases apparently terminated fatally.

Many of the Medical Officers of the Army are familiar with cases of this kind. Clinically and pathologically they are indistinguishable from classical enterica, but bacteriologically they are not, as the sera are specific only to the colon bacilli, while from the spleen and blood only these micro-organisms are recoverable. It is difficult to see why these cases should not be regarded as instances of pure infection by the colon bacillus, for it is exceedingly improbable that all of them should fail to react to the enteric bacillus, if in reality they were cases of enteric fever. Similar in nature and equally interesting are other cases, in which the infecting agent would seem to have been organisms intermediate between the enteric and colon bacilli, and which are variously termed para-typhoid and para-colon according to whether they approach culturally to one or other type.

It has for sometime been observed that the bacillus coli has a share in the causation of enteric fever, and Sanarelli's experiments on the influence of the coli toxins in raising the virulence of the bacillus typhosus has emphasized this fact. Man may have enteric bacillus in an attenuated form in his intestines and may remain in perfect health, but if other toxins invade the body, they may add new virulence and activity, and establish the specific disease.

In the Army in the field in South Africa many cases of enteric fever followed on an acute attack of dysentery, so that apparently the dysentery determined the attack of enteric fever. There appears, therefore, to be a causal relationship between both these diseases. Further, as to the nature of enteric fever we are indebted to Wasdin (*American Medicine*, February 8th, 1902) for the original and very interesting suggestion that the majority of cases of the disease are probably pneumonic in origin, the primary spot of infection being somewhere in the respiratory tract, whence the germ invades the general circulation. In support of this view he claims to have readily located, upon first examination, an area of lung corresponding to such infection. Wasdin says many of these cases might be overlooked as being merely simply bronchitis, but the condition is unilateral and the sputum contains bacteria indistinguishable from bacillus typhosus and accompanied by pneumococci.

In the "*Annales*" *of the Pasteur Institute*, Vol. ii., p. 55, 1897, P. Remlinger and G. Schneider discuss the question: **Does the bacillus typhosus exist in nature outside the sick man,** and the products which emanate from him? A series of experiments extending over many months was carried out on various materials, such as the public water supplies and wells of several towns having epidemics of typhoid, the soil and dust from different localities, the discharges from the digestive tract of persons not affected with typhoid, etc.; and a bacillus giving all the principal laboratory reactions of that of typhoid was obtained from all these sources. In 13 samples of soil and dust the bacillus typhosus was found seven times: (*a*) in the refuse from barracks where there were some cases of typhoid; (*b*) in dust from the laboratory floor; (*c*) in the space between the joists of a room in other barracks; (*d*) in four specimens of soil both superficial and a metre in depth from the courts and gardens of the Val-de-Grâce. These, in three instances, were pathogenic for animals. In the examination of the fæces of ten persons treated at the hospital for affections which had nothing in common with typhoid, five reacted like the bacillus typhosus. Thus

(*a*) in a case of leukæmia specimens examined at intervals of fifteen days, gave each time a positive result; (*b*) in one case of acute tuberculosis without intestinal lesions; (*c*) in a case of premonitory dysentery; (*d*) in two cases of chronic malaria. None of these patients had ever had typhoid fever. Of the bacilli from these five cases four were pathogenic for guinea pigs.

In many additional cultures from water, soil and the intestines, bacilli with every characteristic of the bacillus typhosus were found, except that they were not pathogenic for animals and were not agglutinated by the serum from a typhoid patient. In other words they seemed the same species but attenuated as to virulence. "It is allowable," they state, "to suppose that facts of a similar kind reproduce themselves in connection with the bacillus typhosus. The species of the bacillus of Eberth comprehends, perhaps, varieties more or less numerous, which do not probably react similarly under the influence of the serum of an animal immunised against a determined variety. The belief in the invariability of type in pathogenic microbes is to-day much weakened by many facts. The question of race, descendants from a common stock, but altered by unknown vicissitudes, acquires an importance which must not be underrated. Why should not this theory apply to the bacillus typhosus? We incline to think that bacilli, not pathogenic, and indifferent to the serum test which are encountered in water, soil, etc., are only varieties of the bacillus typhosus; at least, the parentage is evident even if the identity is not absolute. The diversity of fundamental type will, perhaps, serve to explain the variable forms of typhoid infection which are becoming recognised. If this interpretation of facts is exact the following conclusions will result. The bacillus typhosus is distributed in nature outside the human body; it is found in water, in soil, in the intestines of persons not attacked with typhoid, and without doubt forms part of the microbic flora of the media which surrounds us. This idea is not subversive of recognised facts as to the general etiology of typhoid, but rather enables us to conceive and comprehend facts otherwise inexplicable. Daily observations, especially noted in rural places, have set in relief the part played by contagion in the formation and extension of certain epidemic centres; their value remains. Modern researches have demonstrated the prime importance of impure waters in its development and spread; the character of the proof defies all question. But all the cases must originate from contagion or water polluted with the dejections of typhoid patients. Many times it breaks out in patients or groups worn out with fatigue, overwork, or privations, or after eating various foods, without its being possible to trace the origin of the contagion or the use of badly polluted water. The facts conform more easily with the widespread presence of the bacillus typhosus which accounts for its dispersion in surrounding media, and its presence eventually in our natural cavities. A water reputed pure may carry it. Thus introduced into the organism it will live there unoffensive till some depressing circumstance, a fortuitous assistance, perhaps the result of some associated microbe, will open up to it a career of action."

Influence of the Soil.

The next point to which I would invite your attention is the **influence of soil** upon what may be termed an ubiquitous disease, for the geographical distribution of enteric fever indicates that the incidence of the affection is as

wide as the globe, having the same characteristics and taking a leading place in the statistics of mortality equally in Northern Europe and America as in Australia, the Cape, India, Central Asia and the Mediterranean. For some years it was widely believed that the tropics enjoyed a more or less complete immunity from it; but from the facts carefully collected during recent times, and the overwhelming evidence as to the occurrence of enteric fever throughout the whole of India, in Burmah, the Straits' Settlements, Mauritius, the West Coast of Africa, Egypt, the West Indies, Brazil, the Transvaal, Orange-River Colony and the Cape, and the frequent cases of enteric sickness among crews of ships in harbours in the tropics, shows that the diffusion of the disease is limited by neither degrees of heat nor general atmospheric states. Observations made at the most diverse points of the globe, and the general distribution area of the disease, show that mere questions of elevation, or even configuration of ground, have little influence on the occurrence of the affection. On the other hand, the same observations go to show that the disease is met with oftener on more recent formations than on the older, which so far as concerns the physical conditions of the soil is identical with the questions of permiability to air and water. Magne, quoted by Hirsch, made enquiries into this matter, using as his material the epidemiological reports sent in to the Paris Academy of Medicine from 1841 to 1863, omitting 1858. Of the 757 epidemics reported 564 occurred on the more recent formations, 64 on the primary transition rocks, while the remainder were on soils belonging partly to the older and partly to the more recent formations.

An inquiry by R. H. Firth worked out on similar lines, chiefly from the Local Government Board Reports for the twenty years 1871—1890, as to the outbreaks of enteric fever in the British Isles, indicates, so far as the occurrence of the disease in different soils, the following facts:—

On Sandstones (Penrith, Oswestry)	5	outbreaks.
Millstone Grit (Garstang, Ripley, Pateley Bridge)	12	,,
Red Sandstone (Abergavenny, Middleton, Spode)...	19	,,
Variegated Marls (Drayton, Nottingham, Retford, Gainsboro')...	43	,,
Magnesium Limestone (Sedgefield, Wingate, Denton, Loughboro')	28	,,
Oxford Clay (Highworth, Wootton Bassett, Nowbridge)	32	,,
Oolite (Northampton, Southwick, Gloucester)	51	,,
Weald Clays (Neehurst, Caxton, Linfield, Hastings)	37	,,
Chalk & Chalk Marl (Hadham, Marlboro', Andover, Winchester)	103	,,
London Clay (Ealing, Hounslow, London generally)	152	,,
Middle Cœma (Ringwood, Portsmouth, Winckfield)	17	,,
Gravels (Tunstall, Lowestoffe, Hadleigh, Northwold)	136	,,
Gravel on Clay (Dublin, Ipswich, Spalding, Walton)	129	,,
Drift (Westbeach, Wolverton, Buxton, Peterboro')	122	,,

Even admitting the incomplete character of these data, yet their general tenour is deserving of consideration, which is such, that in regard to both upper and lower soils, the proper determining factor is the penetration of the ground by air and water as well as its hygroscopic character. On the oldest rocks, we must remember, there is always a more or less considerable layer of mineral and vegetable detritus, which gives to the soil the same physical characters as the more recent formations possess; and thereby the value of each kind of soil,

so far as concerns the presence of enteric fever, is reduced to the mean standard of porosity. Porosity is, in fact, practically identical with the capacity of any soil for water. Although enteric fever is as often prevalent on a wet ground as on a dry one, still we are unable to say that either absolute dryness or absolute dampness of soil appears to be decisive one way or the other. Notwithstanding this there have not been wanting authorities who have maintained that the disease is connected with **changes in the soil moisture and the rise and fall of the ground water.** The foremost exponent of this view has been Buhl, who, following Pettenkofer's suggestion and inquiries at Munich, formulated the following law: "Between the fluctuations of the subsoil water and the amount and severity of typhoid, there is an unmistakeable connection in this wise, that the total cases of sickness and death from typhoid falls with the rise of the subsoil water and rises with the fall of it; that the level reached by the disease is not in proportion, however, to the then level of the subsoil water, but only to the variation of it on each occasion; or in other words, that it is not the high or low level of the subsoil water that is decisive, but only the fluctuation."

This may be very true of Munich, and it seems, too, to be confirmed by other observers in regard to other parts of Germany, notably by Socin at Basle, Pribram at Pragne, Jacoby at Brislaw, Seidal at Leipzig, Virchow at Berlin, and Schiffendeker at Königsberg; still other workers, particularly in England and India, have not found evidence to support the principle as defined and laid down in Buhl's law. As Hirsch has remarked, this law cannot be expected to hold good equally for all times and places. Nor is it to be wondered at, considering that we are dealing with a factor of disease whose potency may be modified by many peculiarities of locality and soil. Moreover, fluctuations in the subsoil water, as well as mere saturated states of the ground in general, are not in any case the sole causes of the outbreak of a typhoid epidemic. We see examples of this where the disease has occurred in places where the depths of the water bearing stratum has been such as to quite preclude any question of the influence of soil water fluctuations on the surface soil; and, too, in cases where the disease has been traced to infection by other media, such as sewer emanations or personal contagion. As Buchanan pointed out, the experience of most English towns is that when by drainage operations the ground water has been lowered, typhoid, instead of increasing as it should do by Buhl's theory and law, has actually diminished; the reason being that these changes were coincident with the introduction of pure drinking water and other sanitary improvements. Both in this country and in India, that connection between the subsoil water fluctuations and the outbreak of enteric fever still remain to be determined, and that at any rate other conditions of diffusion appear to be far more common. On the other hand, remarkable evidences as to the power and influence of a water-logged soil or sewage, containing subsoil water, with daily fluctuations of soil water, in promoting the prevalence of enteric, are available from Dublin.

The report on **"The Distribution of Enteric Fever in the City of Dublin,"** by the late Dr. Grimshaw and Sir C. A. Cameron, shows that enteric fever prevails with great intensity over certain portions of Dublin, and is more or less absent in other parts. Examination of the geological structure of Dublin showed that

a considerable portion of the area was situated on a gravel bed, which is porous, and readily absorbs any fluids cast on its surface, these being retained in its porous structure owing to the fact that it lies upon an impervious clay. The rest of the Dublin district stands on the clay without the intervention of the gravel. The gravel bed is situated in the middle of Dublin, and the river Liffey, which receives nearly the whole of the city sewage, runs through its centre. This gravel bed is saturated with the leakage from sewers and drains, and with the equally dirty soakage from the tidal Liffey. The intensity of enteric fever prevalence falls on persons living on the porous sewage-water logged stratum, whilst those living on the impervious area are but slightly affected; while the deaths from enteric fever during the five years ending May, 1887, were at the rate of 1 in 365 of the population living on the gravel; the rate was 1 in 531 of those living on the impervious clay.

A contributory factor to this greater prevalence of the fever on the gravel is, that it is at a lower elevation than the clay, and in many places rises scarcely above high water in the river and its estuary. The gravels in Dublin are, in fact, water and sewage logged for a large portion of the 24 hours. The mouths of all the main sewers, that discharge their contents into the river, are furnished with valvular tidal gates which close automatically. When the tide rises to a certain height, the sewers which enter the river below the bridges are, on an average, closed for more than 12 hours daily. During this time, all the sewage entering the sewers from houses and other places, remains therein, except in the case of some of the easterly sewers, which are connected with the pumping stations. It is, however, not only the flow of sewage which is thus arrested for many hours daily, the subsoil water in the gravel of the low-lying localities is prevented from flowing seawards and the soil always is more or less water-logged. As the gravel everywhere interposes between the clay and the river, the whole of the drainage of the clay, except that portion which enters the sewers, passes into the gravel.

Still more interesting is the fact that the subsoil water level in the gravel area fluctuates as much as 7 feet each 24 hours; the figures being 15 and 22. Data like these, though not conforming with nor confirming Buhl's theory and law, are of great value as demonstrating the potency of marked fluctuations of the subsoil water whether at frequent or longer intervals in certain porous and polluted soils, such as the gravels in Dublin. The element of pollution of the soil is, however, essential, and the rationale of these fluttering fluctuations of subsoil water level producing evil, is explained by the power which a rise in the level of the subsoil water through a porous soil like gravel must have in pushing or forcing air (contaminated only too likely by specific germs) out of the soil into house basements, etc. Should the rise in soil water in circumstances such as exist in Dublin coincide with a sudden fall in the barometer, which of itself can aspirate the air out of soil, emission or suction of polluted and germ-laden soil air would be increased considerably. In truth, certain facts noted by Dr. Begbie, of Dublin, prove that the onset of actual cases of enteric fever have been coincident with such barometric and soil water fluctuations.

The more recent investigations by Firth and Horrocks, on the viability of the enteric bacillus in soil are given in the *British Medical Journal* of September

27th, 1902. The conclusions they arrived at are as follows: (1) that there is no evidence to show that the enteric bacillus in soil could increase or grow in different directions; (2) that the enteric bacillus is capable of being washed through at least 18 inches of closely packed soil by means of water; (3) that the bacillus survives in moist soil for varying periods, sometimes as long as 74 days; (4) that the survival of the organism in soil is independent of either pollution or the reverse; (5) that its survival is mainly dependent upon the amount of moisture present; (6) that the bacillus is recoverable from dry sands after twenty-four days, but if the sand is kept moist the bacillus is not recoverable after twelve days, probably from being washed down into the deeper layers; (7) that the bacillus rapidly dies out in peat; (8) that from ordinary soil kept damp with rain water the bacillus could be recovered on the sixty-seventh day, with dilute raw sewage on the fifty-fourth day, with dilute sterile sewage on the seventy-fourth day, and that in similar soil the bacillus disappeared at once from the surface layers after heavy rainfall; (9) that infective material could be readily carried from dried soil and sand by air currents; (10) that the bacillus is recoverable from air-dried fabrics, such as khaki drill or serge, after periods varying from twenty-four to eighty-four days; (11) that infected dry soil, if blown about as dust, is capable of infecting distant objects after twenty-four days from time of desiccation; (12) that ordinary house flies can convey infective matter from secreted or other polluted material to objects on which they may walk, rest, or feed, and that such infective matter appears to be attached to their heads, legs, bodies and wings, but there is no evidence to show that the enteric bacillus passes through their digestive tract. It is obvious that these facts, and the conclusions to be drawn from them, have an important bearing on practical sanitary questions in civil as well as in military life.

TYPHOID FEVER AND DYSENTERY IN SOUTH AFRICA DURING THE BOER WAR.

I propose now briefly to review some of the **facts connected with the outbreaks of enteric fever and dysentery which occurred in the Army in South Africa,** and to point out the preventive measures which it is advisable should be undertaken to mitigate the severity of these epidemics.

The conditions which produce dysentery in armies are akin to those which cause enteric fever. In the absence of a fuller knowledge of the nature of dysentery, it is sufficient to regard both this disease and enteric fever as being essentially filth diseases, and both due to bacillary organisms intimately associated with and given off from the excretory products of men and animals. Further, that as far as their general etiology is concerned, both diseases may be said to have a common origin.

Influence of Age.

At the outset it may be stated that, especially in the early part of the war, the Divisions and Brigades were very largely composed of young men. Very few appeared to be over 25 years of age, and a large number were considerably under it. No doubt many young men, in their anxiety to join the Army in the

Field, understated their age, but this could hardly account for the large numbers of young men in the ranks. Undoubtedly early adult life especially predisposes the individual to typhoid fever. Men from 15 to 30 years of age are in greatest danger from this disease. Murchison reported 52 per cent. of his cases among individuals from 15 to 25 years of age. Fielder found 58 per cent. of his cases to range from 20 to 30 years of age. The predisposition to this disease at an early age may partly be accounted for by the acquired immunity conferred on older men by a previous attack, the nature of which they were probably ignorant of.

The following table shows the admission-rate per 1,000 for enteric fever in the Army in India from 1897—1901:—

Years.	Under 20.	20—25.	25—30.	30—35.
1897	33·0	44·4	19·6	7·6
1898	24·7	53·3	22·2	9·0
1899	18·8	31·3	11·5	5·8
1900	9·0	23·1	12·0	6·0
1901	12·70	18·6	10·7	5·4

In 1900–01 only few men were sent from England; owing to the war in South Africa the time-expired men were retained, and thus the average age of the soldier serving in India was increased. But, if we may draw any conclusions from the sickness and mortality statistics of the Army in India, we find that there is even a more important factor than age as a predisposing cause.

Enteric fever most frequently attacks new arrivals. In India the newly-arrived soldier suffers most from enteric fever is evident from the following table, which shows the ratio per 1,000 of admissions from 1898—1901.

Year.	Under 1 year.	2nd year.	3rd year.	4th year.	5th year.
1898	91·3	38·0	26·6	23·1	20·6
1899	51·6	30·3	11·6	10·7	8·2
1900	30·4	28·3	20·5	11·8	12·1
1901	37·8	12·5	18·1	11·9	10·2

These figures show that there is a certain immunity afforded by residence, and this appears to be much more perfect in tropical and sub-tropical regions than in higher latitudes. The protection acquired through **acclimatisation and seasoning** cannot be denied, though what influence of its own a tropical climate has in this respect is uncertain. The increased prevalence of enteric fever in India has always been accompanied by, and is possibly dependant on, an increase in the number of *young* and *recently* arrived soldiers in that country.

It is not possible to give a comparative table for South Africa, but personal observation and enquiry goes to show that a certain protection is also acquired there by acclimatisation. A dominant factor in the prevalence of this disease was the immense numbers of *young* and *recently* arrived soldiers for the Army there.

Influence of Flies.

In no previous war has the composition of the Army been the same as in the great Boer War. Owing to the nature of the country, the character and mode of life of the Boers during peace, and the training by their Commandos, it was absolutely necessary to employ large numbers of mounted troops, and these were out of all proportion to the numbers that compose an Army Corps as it then or now exists. The absence of food supplies for men and animals throughout the vast areas traversed by these mounted columns in the Transvaal, Orange River Colony and the Cape necessitated hugh convoys of Kaffirs and ox-waggons to accompany the columns on the march. In a country so exposed and with an enemy so agile, it was necessary to form camps on the line of march in close formation. These circumscribed camps involved the presence of large numbers of animals in close proximity to the men, and with these came myriads of flies which have not only a most troublesome cause of discomfort, but were also the means of distributing and spreading disease among the troops. Flies swarmed over infected fæcal matter in the trench latrine pits, and fed upon the food issued to the men in tents. The use of quicklime did not prevent their invasion. Flies can readily carry enteric infected matter from specific excreta and deposit it on the food of men. Such infected matter appears to be attached not only to their heads but also to their legs, wings and bodies. I have seen in Bloemfontein and Pretoria where the pail system for the removal of excreta is the one in use, swarms of flies pass between the houses and the latrines which are in close proximity.

Disposal of Excreta. In camps, especially in the tropics and subtropics, enteric fever and dysentery are always more prone to assume an epidemic form than in civil life in England. The reason for this is that camp life does not lend itself to the disposal of excreta and refuse as carried out usually in towns and villages. Enteric fever and allied diseases bear a direct relationship to the means adopted for the disposal of excrementitious matters. Once a regiment or company becomes infected the latrine is the common focus from which the disease is spread, and this infection may occur before the disease is recognised. Much has been said and written on the dissemination of these diseases by water among the troops in South Africa. Without desiring in the least to minimise the absolute necessity that must always exist for an ample supply of pure water, I am convinced that it is to the trench latrine pits, to the general fouling of the soil, to direct personal infection, and to infected clothing and blankets, is to be attributed the spread of these diseases in an Army in the Field. Trench latrines are always a source of danger, however well arranged for, and in South Africa they were certainly no exception to this rule. To them and to personal contact, either with individuals in the early stages of the disease, or with infected clothing, I attribute the large numbers of admissions to hospital. So subtle is enteric fever in its onset that it may not be recognised until long after the enteric bacilli have infected the latrine, and thus an individual becomes a source of danger to others. To disinfect, burn, or otherwise destroy the bacilli in the excreta from patients in hospitals, affords but very partial means of protection in camps. To eliminate with any degree of certainty this source of danger *all* excreta should be thoroughly disinfected.

The system of trench latrines is the one universally adopted in all Armies. With an Army in the Field, when the site has to be for only a few days, no other system appears to be practicable. In standing camps, it will be necessary to employ men (natives) under a proper organisation, and arrange for the removal of all excreta in sanitary carts for cremation or other disposal outside the camp. There is no more important question than this in the hygiene of camps, and if our object is to prevent as far as possible these diseases we must be prepared to accept the principle that only a proper conservancy, with the complete removal and destruction of filth can effect fewer deaths and less sickness in an Army in the Field.

In the Military Hospitals in South Africa two methods were adopted for the disposal of all excreta: one by boiling—the solid and liquid excreta, after disinfection in the ward, were boiled in ordinary pot boilers;—this method is unsightly and rather offensive; the ladeling out of the boiled fæces is a dirty and somewhat disgusting process and is open to objection. If this plan is adopted where a system of sewerage exists, and where the boiled fæces could be passed directly into the sewers, this objection would not hold good. It would, however, be necessary to adopt some means for cooling the liquid in the container, otherwise this liquid passed into the drains, hot, may crack them or their expansion would displace the joints. A far more cleanly method is to mix the fæces with ashes or other dry material and burn them together in a small cremator. This plan was adopted most successfully in many of the large hospitals.

The nature of the soil in the Transvaal and Orange River Colony added to the difficulties of camp sanitation. The soil itself, which in most parts is composed chiefly of alluvial clay, is deficient in the salts of lime and potash—the alkaline basis;—there is little limestone in the Transvaal or Orange River Colony. This kind of soil does not lend itself to the disintegration and breaking up of organic matter. Dead animals and organic filth remain for months without undergoing the natural process of decay, and lie as putrifying masses, apparently slowly melting away. In the same way bodies buried in cemeteries emit foul emanations from the ground, so much so that lime had to be freely used at the time of burial to prevent a nuisance. Those plants which are indicative of nitrogen ferments in the soil, such as the vetches and the *Leguminosæ* generally are conspicuous by their absence. There appears to be an absence of nitrifying organisms in the soil, and this is further shown by the small amount of nitrogen acids found in water.

All these conditions indicate that the soil itself, uncultivated and not submitted to the action of growing plants, is not in a condition to receive and render harmless organic filth deposited on circumscribed and small areas, densely populated and without any sewerage system available. This soil, too, has little or no absortive power.

Density of Population. The density of population in camps is an important factor in furthering the spread of any infective disease. On active service an ordinary bell tent is assumed to afford sufficient protection for 15 men. It is made of "duck," provided with six-inch eaves to carry off water clear of the walls, and has these small ventilators covered with bibs. The tent weighs

41½lbs., has an internal capacity of 623 cubic feet, and its diameter is 13ft.; the height of the walls is 2ft. 2ins., and the pole is 9ft. 9ins. long. Ventilation is most imperfect, as the holes are so small that the movement of air is almost imperceptible. There is little ventilation through the canvas, and none at all when it is wet with dew. Some of the newer pattern tents have a few openings for ventilation made in the canvas near the pole, and this is an improvement on the older form.

There are 3,097,600 square yards in a square mile, and assuming, as was most frequently the case in South Africa, that there were 15 men in each bell tent, the following table gives the surface area per tent for different densities of population per square mile.

	Strength	Square Yards	Acres	Men per Acre	Men per Sq. Mile
Infantry battalion, full size...	1,011	21,600	4·46	226	144,640
„ „ minimum..	1,011	7,800	1·61	628	401,920
Cavalry regiment, full size ...	630	34,000	7·02	89	56,960
„ „ minimum..	630	15,000	3·09	204	130,560
Battery, R.A.	154	11,200	2·31	67	42,880
Field Company, R. E.	182	7,500	1·54	118	75,520
Bearer Company	66	8,400	1·73	38	24,320
Field Hospital	145	11,200	2·31	62	39,680

This table shows that a cavalry regiment encamped upon its maximum area is nearly as densely populated as Liverpool, which has a population density of 97 persons to an acre; and when occupying its minimum space would have as dense a population as Whitechapel, which is the most crowded part of London. It is therefore not difficult to understand the danger of infection from close proximity of infected to healthy men in tents, particularly in the early stages of the disease and before they realise that they are sick. The enteric bacilli are able to exist in cloth and woollen clothing for upwards of two months, and in cloth fouled by liquid fæces the micro-organism is recoverable in sixteen days.

The evidence that enteric fever is communicable from person to person has been slowly but surely accumulating, and there are few now who would deny this source of dissemination. Many instances of this occurred in South Africa, and the tendency too often was, to neglect the personal factor of direct infection and to seek a possible cause outside the camp itself. This factor of personal infection was very clearly demonstrated at Green Point, Capetown. This camp had an excellent supply of water from the Capetown Water Works, and no fault could be found with the conservancy arrangements. Over and over again outbreaks of enteric fever occurred, and these in every instance followed on the arrival of an infected party of convalescents sent down from up country, and who were detained on arrival at Green Point until they could be disposed of. There can be no doubt that the infection was conveyed by these convalescents in their clothing or by personal contact. Confirmatory of this is the later evidence investigated and reported by Dr. Collingridge of an epidemic of enteric fever on the " Corwall " Training Ship. In the course of his injury he was able to trace the source of infection to condemned army blankets returned from South

Africa. These blankets were found to be in a filthy condition and fouled with blood and excrement. They were submitted to Dr. Klein, who reported that they were stained with fæcal matter and urine, and contained innumerable typhoid bacilli.

It may be of interest in this connection to quote a paragraph from the report of the Commissioners appointed to enquire into the origin and spread of enteric fever in the military encampments in the United States during the Spanish War of 1898:—

"Out of 1,608 cases most thoroughly investigated more than half were due to direct and indirect infection in and from tents. Of all the conclusions this last is the most momentous and most instructive. The evidence indicates that when once the infection has been established in a camp, it will continue to spread in spite of shifting camp, provision of pure water supply, and other sanitary improvements, unless the infected men be separated and the contaminated clothing, bedding, and tentage thoroughly disinfected. Practically the evidence and the conclusions imply that direct infection from man to man in the tents, and indirect infection through the contaminated clothing, bedding, tentage and soil of the tent were the most important factors in the spread of the disease throughout the United States camps, and that this mode of infection is probably common and persistent in camp life."

Water Supply in Relation to these Diseases. The supply of pure, potable water, is, especially in South Africa, a very difficult problem. In Pretoria and one or two other towns good water is available, but in the large majority of towns the supply is taken from rivers, which are open to pollution on every side. Troops in camps outside towns and on the line of march had to depend on surface supplies from shallow wells, from spruits or streams, or from ponds. It was impossible to connect cases of enteric fever with the use of such water supply. As a rule the men on the march were healthy and did not suffer. Very many men and convalescents from enteric fever and dysentery, whom I closely interrogated, denied drinking any water except in the form of tea or coffee. One medical officer, suffering from a severe attack of enteric fever was so impressed with the idea that water was the most probable cause, that he saw all the water he used boiled, and had never taken any to drink except in the form of tea; he could not understand, with all the precautions he had taken, how he had contracted the disease.

It was remarkable how few men in the blockhouses were attacked with enteric fever; the reason for this was, I believe, that few men (10—12) occupied them. They were removed from the danger of contracting the disease either by personal contact or from clothing, etc., and they were, comparatively speaking, free from flies, which found little refuse around these structures on which to congregate. As a rule all isolated places and posts escaped. This fact clearly shows that density of population is a very important factor in the dissemination of these diseases.

Very stringest orders were issued to all units that water used for drinking purposes should be boiled before distribution, and for this purpose Sawyer's boilers were provided and extra fuel allowance issued. Berkefeldt filters were also provided.

From a very careful consideration of the whole question, and by tracing as far as possible, the admissions to hospital from the various camps and stations, I am forced to the conclusion that water played a comparatively insignificant part in the diffusion and spread of enteric fever in the Army in South Africa, and that other causes operated to a far greater extent. As a further proof of this, figures, as far as I have been able to analyse them, show that officers suffered in much the same proportion as the rank and file. The returns have not yet been classified, so that no positive statement can yet be made.

Of course there were exceptions to these facts. In such towns as Bloemfontein, Kronstad, Standerton, and many others, when the supply was taken from rivers and was largely polluted, enteric fever has been endemic for a long time.

In a country as large as that in which the army operated in South Africa, it is necessary to summarise our experiences on the whole of the facts under review, and not to emphasise individual cases, dogmatise on them, and make them applicable to the entire army scattered over half a continent.

Great difficulties were experienced in carrying out the regulations with regard to the boiling of water by the columns on the line of march. In many districts traversed there was no wood or other fuel, not even bushwood, and the difficulties of transport and the necessity for speedy movements was such as frequently to place men on short rations. Those men did not suffer. They were healthy while on the march, but sickness appeared when they came into standing camps in towns for the purpose of refitting.

Our experience in South Africa was that infected water played a very subordinate part in the dissemination of enteric fever in the army there. There certainly were local supplies that became specifically infected, but infected water was not the dominant factor in the causation of this disease. And this is practically the experience of the United States' officers in the Spanish War. The Commission reported that: "Infected water was not an important factor in the spread of typhoid fever in the national encampments in 1898."

It must, I think, be admitted that notwithstanding the greatest care for the safeguarding of the generally accepted sources of these diseases, it is practically impossible to completely prevent their occurrence in any army, hastily mobilised, operating in a country in which water is scarce, and whose ranks are largely filled with young men at an age notoriously liable to be affected by diseases of this nature. Much, however, may be done to lessen both the incidence and mortality from these diseases by suitable and adequate sanitary measures.

Feeding in Relation to the Health of the Young.

BY

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Feeding in Relation to the Health of the Young.

Introduction.

In the present lecture I propose to consider the feeding of children in the poorest class of the community, treating the subject chiefly from the public health point of view. Experience shows that where the earnings of a family exceed 35s. a week, unless there be some special source of drain, such as illness or excessive drinking, children generally receive sufficient food and a sufficient variety.

There is a widespread belief that children are degenerating, owing to the increasing aggregation in towns, and to loss of that simplicity in living which was practised in former days. There are unfortunately no measurements of height and weight at different ages on a scale to give us any exact information on the subject, and we are compelled, therefore, to fall back on the statistics of mortality.

Works consulted (see last page).

I. PHYSICAL CONDITION AS INDICATED BY DEATH-RATES, FROM 1838 TO 1901.

Statistics of Mortality from 1838 to 1901. In the Annual Reports of the Registrar-General we find tables showing the death-rates for decennial and also for quinquennial periods since the year 1840, at groups of ages, and we are thus able at a glance to see the history as regards mortality of each period of life.

TABLE I.

England and Wales. Annual Death-rates at Twelve Age-periods and General Death-rates corrected for Age-Constitution in Groups of Years 1838–1901. Males and Females.

MALES. Deaths to 1,000 living.

Period	All ages.	0—	5—	10—	15—	20—	25—	35—	45—	55—	65—	75—	85 & up
1838–1901	21.1	68.4	7.1	4.0	5.6	7.6	8.9	12.7	18.9	33.3	68.3	147.4	308.6
1841–50	22.2	71.2	9.2	5.1	7.1	9.5	9.9	12.9	18.2	31.8	67.5	148.3	312.3
1851–60	21.9	72.7	8.5	4.9	6.7	8.8	9.6	12.5	18.0	31.0	65.5	146.7	308.2
1861–70	22.3	73.5	8.2	4.5	6.2	8.5	9.9	13.5	19.2	33.1	67.1	147.2	315.0
1871–80	21.5	68.5	6.7	3.7	5.3	7.4	9.4	13.8	20.1	34.9	69.7	150.8	327.4
1881–90	19.7	61.6	5.4	3.0	4.3	5.7	7.8	12.4	19.4	34.7	70.4	146.6	305.8
1891–1900	19.2	62.7	4.3	2.4	3.8	5.1	6.8	11.5	19.0	35.0	70.4	146.1	286.8
1841–45	21.3	68.7	8.8	4.8	6.8	9.0	9.4	12.2	17.2	30.3	65.5	143.7	305.1
1846–50	23.1	73.8	9.5	5.4	7.3	10.0	10.5	13.6	19.2	33.2	69.5	153.0	319.5
1851–55	22.4	73.9	8.8	5.1	7.0	9.2	10.0	12.9	18.6	31.5	66.6	150.8	311.0

MALES—*Continued.*

Period	All ages.	0—	5—	10—	15—	20—	25—	35—	45—	55—	65—	75—	85 & up
1856–60	21.4	71.5	8.3	4.6	6.4	8.4	9.2	12.2	17.4	30.4	64.3	142.6	305.4
1861–65	22.3	74.0	8.5	4.7	6.4	8.7	9.7	13.2	18.9	32.8	66.3	145.8	316.4
1866–70	22.2	72.9	7.9	4.3	6.0	8.3	10.1	13.8	19.6	33.5	67.8	148.7	313.6
1871–75	22.0	69.9	7.1	4.0	5.7	8.1	10.0	14.3	20.3	34.8	70.0	149.5	323.3
1876–80	21.0	67.0	6.3	3.4	4.9	6.7	8.7	13.4	19.8	34.9	69.4	152.2	331.6
1881–85	19.7	61.3	5.8	3.2	4.5	6.0	8.2	12.8	19.3	34.2	68.7	145.4	297.8
1886–90	19.6	61.9	4.9	2.8	4.1	5.5	7.4	12.0	19.4	35.2	72.1	147.9	313.8
1891–95	19.6	62.9	4.5	2.6	4.0	5.2	7.1	12.0	19.6	35.9	72.5	149.3	291.0
1896–1900	18.8	62.4	4.1	2.3	3.6	4.9	6.5	11.1	18.3	34.1	68.3	142.9	282.6
					FEMALES. Deaths to 1,000 living.								
1838–1901	19.3	58.5	6.9	4.1	6.0	7.1	8.8	11.5	15.4	28.1	60.0	133.3	281.9
1841–50	21.0	61.1	8.9	5.4	7.9	9.1	10.6	12.9	16.1	28.4	60.9	135.9	293.3
1851–60	20.6	63.0	8.4	5.1	7.4	8.6	10.0	12.2	15.3	27.1	58.9	134.5	288.9
1861–70	20.4	63.7	7.8	4.5	6.7	8.0	9.7	12.1	15.6	27.9	59.1	134.9	285.1
1871–80	19.3	58.4	6.3	3.7	5.5	6.8	8.6	11.6	15.6	28.7	61.0	135.4	296.4
1881–90	17.6	52.0	5.3	3.1	4.4	5.5	7.4	10.6	15.1	28.5	60.4	130.6	270.8
1891–1900	17.0	52.8	4.4	2.6	3.7	4.5	6.1	9.6	14.8	28.5	60.7	130.6	261.4
1841–45	20.1	58.6	8.6	5.2	7.7	8.6	9.9	12.2	15.1	27.2	59.1	138.8	288.6
1846–50	21.9	63.7	9.2	5.7	8.1	9.6	11.2	13.7	17.0	29.7	62.8	140.1	297.9
1851–55	21.0	63.8	8.5	5.3	7.8	8.9	10.3	12.6	15.8	27.8	59.6	137.1	292.0
1856–60	20.1	62.3	8.3	4.8	7.1	8.2	9.6	11.9	14.8	26.5	58.1	131.9	285.8
1861–65	20.6	64.1	8.3	4.8	6.9	8.2	9.8	12.1	15.5	27.9	59.1	133.7	287.7
1866–70	20.3	63.3	7.4	4.3	6.4	7.8	9.6	12.1	15.8	27.9	59.1	136.1	282.4
1871–75	19.8	60.0	6.6	4.0	5.9	7.4	9.2	12.0	15.9	28.7	61.2	135.3	293.8
1876–80	18.7	56.8	5.9	3.5	5.0	6.2	8.0	11.2	15.4	28.6	60.8	135.5	299.0
1881–85	17.8	51.9	5.7	3.3	4.7	5.9	7.9	11.0	15.2	28.1	59.0	128.9	265.4
1886–90	17.5	52.0	4.9	2.9	4.1	5.2	6.9	10.3	15.0	28.8	61.7	132.3	276.2
1891–95	17.5	52.8	4.6	2.8	4.0	4.8	6.6	10.2	15.2	29.5	63.1	134.4	264.2
1896–1900	16.5	52.7	4.2	2.4	3.3	4.1	5.6	9.1	14.3	27.4	58.4	126.8	258.5

We thus perceive that at all the earlier ages an immense advance has occurred, both in males and females, but greater in the case of the latter. This is most marked at the ages 5 to 9, 10–14 and 15–19, being less noticeable at earlier ages, and gradually diminishing till it ceases at the age group 35–44 in the case of males.

No doubt many factors have been at work, such as Sanitary Administration, factory legislation, better housing, etc., but if we follow the course of the decline we are obliged to conclude that some other general causes must have been operative, which have acted over so long a period, and have penetrated into counties where Sanitary Administration has been comparatively little in evidence, and the effects of factory legislation have been but little felt. The improvement is particularly observable in the five years 1876–80, and though this is coincident with a period of much Sanitary activity, it follows too close on this to be ascribed to it. The decline must be ascribed to increasing prosperity, to improving wages, and to cheapening food.

It is perhaps not without significance, and appears to suggest increased physical well-being, that the great drop in the mortality at ages 0–5 takes places in the next quinquennium. The same continued improvement is manifest in Manchester, and the statistics also show that the children in the Central Districts of Manchester had in the last decennium a lower death-rate than the children throughout England 60 years ago. This is also visibly evident in photographs taken by Mr. Ellwood, of the Public Health Office, of slum children fifteen years ago and in October, 1903. I am informed by Mr. Wyatt that photographs of school children in the poorest districts taken 20 years ago and recently give the same results.

TABLE II.

Death-rates in Age Groups in Manchester. The rates for 1851 to 1890 are based on the deaths occurring in the Manchester, Chorlton and Prestwich Unions; in 1891–1900 they are for the City of Manchester.

Years	All ages.	Under 5.	5—	10—	15—	20—	25—	35—	45—	55—	65—	75—	85—
1851–1860	28.59	105.95	11.61	5.18	7.72	8.75	11.28	16.62	26.46	44.82	86.60	172.30	344.75
1861–1870	29.44	100.87	11.48	5.44	7.61	9.21	12.03	18.31	27.80	49.82	94.64	181.48	294.05
1871–1880	27.06	86.90	8.97	4.45	6.22	8.42	11.70	18.22	28.29	49.98	95.64	180.51	
1881–1890	24.15	77.52	7.50	3.67	5.12	6.47	10.19	17.33	27.30	49.39	93.33	176.39	
1891–1900	23.27	82.11	5.62	2.93	4.39	5.33	8.36	16.40	26.87	49.27	94.96	181.06	306.36

TABLE III.

Death-rates in the Manchester Township in Age Groups.

Years.	All ages.	Under 5 years.	5—	15—	25—	45—	65—
*1851–1860	31.48	117.24	9.22	8.98	15.24	36.08	116.16
*1861–1870	32.80	111.88	9.63	9.44	16.96	39.99	122.48
*1871–1873	31.46	103.82	7.81	9.01	17.07	39.93	113.73
1874–1880	32.16	103.52	7.84	8.72	18.59	44.93	126.34
1881–1890	31.73	96.69	6.32	7.81	19.66	49.43	123.18
*1891–1900	30.10	104.33	5.25	5.66	16.67	46.48	142.47

* Up to the end of 1873 the Prestwich Union formed a part of the Manchester Union. These figures are, therefore, for the two Unions combined.

Influence of Prosperity and Abundance of Food. The relative lowering of the death-rate of women and children to those of men is in inself a proof of growing prosperity. It might be supposed that the view that increasing prosperity has determined largely the lowered death-rate amongst the young implies that the most marked advance should have been noted in the years 1870–74, the year of overflowing trade. But on consulting Mulhall's *Dictionary of Statistics* we find that in those years prices, and particularly the prices of articles of food, sustained an increase, so that while improved wages affected only a part of the people, another part suffered from the increased prices.

How great is the gulf between families who have only enough to get sufficient food, and those in which food is deficient can be seen any day by

visiting poor households, examining the children, and enquiring into the circumstances of health of members of the family. There is not a shadow of doubt that many children annually perish from what is virtually starvation, and equally no doubt that the Society for the Prevention of Cruelty to Children, the Police, Health Visitors, District Nurses, the Boys' Refuge, Strangeways, and many other agencies, in addition to the Administrators of the Poor Law, partly by the strong hand, and partly by kindly aid, do much more now than formerly to avert this result. It suffices to see the transformation effected by Industrial Schools in the wretched children brought under their care, to realise what a difference to the death-rate, and to well being generally, the difference in diet produces, aided, it is true, by other means. And that it is diet chiefly anyone may convince himself by an examination of children in poor homes, some sufficiently and others insufficiently fed.

It is, therefore, easy to understand how any difference in the wages earned by the poorest class, or in the price of food, or in the capacity of wages to obtain food, must be reflected in the death-rate. We may assume that the death-rate is a measure of physical condition generally, though, doubtless, the improvement in the former is greater than in the latter.

Correspondence between Poverty and Death-rate. The correspondence between poverty and death-rate is well seen if we compare the mean death-rates at different groups of ages for the years 1891–1900 in the Manchester Township, South Manchester and North Manchester.*

TABLE IV.

Average Annual Death-rates for the years 1891–1900 in the City of Manchester and its three Main Divisions, in Age Groups and from certain Diseases.

ALL CAUSES.

Division.	All ages.	0-5	5-15	15-25	25-45	45-65	65 and up.
Manchester Township	30.05	104.72	5.26	5.68	16.69	45.88	127.89
Northern	18.71	66.24	3.80	4.37	8.74	27.32	101.22
Southern	21.92	78.46	4.03	4.67	10.65	32.23	112.45
City	23.27	82.19	4.30	4.86	11.77	34.94	113.87

MEASLES.

Division.	All ages.	0-5	5-15	15-25	25-45	45-65	65 and up.
Township	1.11	8.75	0.22	0.01	0.00	—	—
Northern	0.65	4.80	0.16	0.00	0.00	—	—
Southern	0.63	5.09	0.15	—	0.00	—	—
City	0.77	6.01	0.17	0.00	0.00	—	—

WHOOPING COUGH.

Township	0.74	5.84	0.15	—	—	—	—
Northern	0.45	3.40	0.06	—	—	—	0.03
Southern	0.58	4.74	0.10	—	—	—	—
City	0.59	4.66	0.11	—	—	—	0.01

* See a paper on this subject by Alderman McDougall, *Brit. Med. Journ.*

PHTHISIS.

Township	...	...	3.21	...	0.47	...	0.53	...	1.84	...	5.68	...	6.22	...	3.85
Northern	...	...	1.29	...	0.23	...	0.26	...	1.40	...	2.21	...	1.99	...	0.76
Southern	...	...	1.87	...	0.47	...	0.35	...	1.54	...	3.12	...	3.17	...	1.41
City	...	...	...	2.08	...	0.40	...	0.38	...	1.58	...	3.57	...	3.75	...	1.91

NERVOUS DISEASES.

Township	...	...	1.91	...	8.84	...	0.45	...	0.23	...	0.81	...	2.15	...	5.15
Northern	...	...	1.32	...	5.47	...	0.37	...	0.22	...	0.52	...	1.77	...	4.99
Southern	...	...	1.42	...	6.49	...	0.33	...	0.21	...	0.58	...	1.42	...	5.85
City	...	...	...	1.52	...	6.85	...	0.37	...	0.22	...	0.63	...	1.71	...	5.44

HEART AND BLOOD-VESSEL DISEASES.

Division.			All ages.		0-5		5-15		15-25		25-45		45-65		65 and up.
Township	...	...	3.06	...	0.28	...	0.40	...	0.57	...	2.13	...	9.82	...	28.35
Northern	...	...	2.08	...	0.32	...	0.24	...	0.45	...	1.16	...	6.85	...	27.19
Southern	...	...	2.61	...	0.32	...	0.29	...	0.44	...	1.46	...	8.06	...	30.61
City	...	...	...	2.59	...	0.31	...	0.30	...	0.48	...	1.56	...	8.27	...	29.15

PNEUMONIA.

Township	...	...	3.52	...	12.70	...	0.75	...	0.79	...	2.22	...	5.21	...	9.28
Northern	...	...	2.00	...	8.05	...	0.44	...	0.46	...	1.10	...	2.81	...	4.59
Southern	...	...	2.23	...	8.32	...	0.37	...	0.55	...	1.29	...	3.33	...	7.48
City	...	...	...	2.52	...	9.43	...	0.49	...	0.59	...	1.49	...	3.74	...	7.25

ALL RESPIRATORY DISEASES EXCEPT PNEUMONIA.

Township	...	...	3.81	...	10.52	...	0.31	...	0.17	...	1.04	...	8.35	...	33.48
Northern	...	...	2.18	...	7.04	...	0.17	...	0.07	...	0.51	...	4.33	...	21.19
Southern	...	...	2.60	...	8.35	...	0.20	...	0.13	...	0.58	...	4.90	...	24.58
City	...	...	...	2.82	...	8.57	...	0.22	...	0.13	...	0.68	...	5.75	...	26.17

Average Annual Death-rates for 1891–1900 amongst persons dying at home, in Workhouses and in Hospitals for the City and its three Main Divisions.

				Home death-rate.		Death-rate in Union Hospitals.		Death-rate in other Institutions.		Total death-rate.
Township	...	...	...	21.45	.....	6.09	.....	2.51	.....	30.05
Northern	...	...	...	16.64	.....	0.81	.....	1.26	.....	18.71
Southern	...	...	...	18.26	.....	2.36	.....	1.30	.....	21.92
City	...	...	...	...	...	18.69	.....	2.96	.....	1.62	.....	23.27

The Institution death-rate in the Manchester Township is 8·6 per 1,000, in the Southern Division 3·7 per 1,000, and in the Northern 2·1 per 1,000. Corresponding differences show themselves in the death-rates at each age group. Special attention may be directed to the correspondence in the death-rates from Measles and Whooping Cough at early ages, with the figures showing death-rates at all ages occurring in the Unions. There is every reason to believe that these diseases are as prevalent in one district as in another, and that the differences in mortality are due to the differences in nutrition. The same difference

is observed in Scarlet Fever, and Dr. Gordon has informed me that he has observed that the number of deaths per 1,000 cases is higher in the poorer districts owing to the more defective nutrition of the children attacked.

The statistics for the Central Districts of Manchester show, what indeed may easily be seen by direct observation, that the problem of improving the nutrition of the children in the poor districts is still with us. But even in this area, which relatively to the total population is becoming poorer and poorer, there has been immense improvement. In 1861–70 the death-rate at ages five to fourteen was 9·63. In 1891–1900 it was 5·25. At the former period at ages fifteen to twenty-four, it was 9·44, at the latter 5·66. There is, therefore, every encouragement to endeavour.

II. THE FEEDING OF ADULTS AND CHILDREN.

Dietary of a Man doing moderate Labour. In considering the diet possible for children in the poorest families it will be necessary to take the dietary of a man doing moderate labour and to subdivide it among the children. This is the more permissible as I find through the enquiries of the health visitors that, in general, the labourer shares with his family. In many instances, it is true, he gets what is believed to be a more nourishing meal. In any case we shall not be overstating what the children get or what they can have, and there is no other way possible. This is the method used by Mr. Rowntree in his book on poverty by which I was possibly unconsciously directed to it, but I do not see how else one could proceed.

Foods are substances capable, when eaten, of producing or repairing tissue, and generating heat and work. They are resolved by chemical analysis into proteids, fats, carbo-hydrates, salts and water. Of these the proteids alone are available for the building up and repair of living cells, while the salts are also necessary to life. These include calcium, iron, phosphorus, sodium and potassium, the first three being in organic combinations. They are present in sufficient amount in all ordinary diets. Proteids, fats, and carbo-hydrates are all available as sources of energy, though, on account of the excess of nitrogenous waste produced as well as on the score of expense, proteids are not possible as the sole source of energy.

For the maintenance of a high standard of health both fats and carbo-hydrates are necessary, though life can be sustained with either alone. There is, however, no fixed proportion, and convenience and individual tastes largely determine the proportions in which they are taken. The amounts of these constituents of food required vary under different conditions, including sex, age, weight, weather and conditions of work.

Inasmuch as they are burned up in the system to produce energy, the amounts must be increased with increased work and production of heat, while with increased work tissue waste also increases, so that the demand for an increase of proteid coincides with the demand for more fat and carbo-hydrate.

The daily amount required for any given condition, such as a man of average weight (eleven stone) doing a moderate day's work, may be determined by measuring the total carbonic acid and urea which he excretes in a day on a

freely chosen diet, and then determining the quantities of proteid necessary to yield the amount of nitrogen present as urea and the quantities of fat and carbo-hydrates needed to make up the carbon excreted. In this way Playfair arrived at the following diet:—

Proteid.		Fat.		Carbohydrate.
119	.....	51	.....	531

While Atwater, as the result of many observation, gives:—

Proteid.		Fat.		Carbohydrate.
125	.....	125	.....	450

A Calorie being the amount of heat required to raise the temperature of 1 kilogramme (litre) of water 1°C from 0° to 1°C, the first diet yields 3,140 Calories and the latter 3,520.

One gramme of proteid gives	4·1	Calories.
One gramme of fat gives	9·3	„
One gramme of carbohydrates gives...	4·1	„

Fat being reckoned as 2·4, Playfair's diet gives for proteid a ratio of 1 to 6·3 of other constituents, Atwater's of 1 to 6. In the discussion of diets I propose to use Atwater's dietary. It approximates much more nearly to that freely chosen by persons of the requisite means, and we may believe, especially in dealing with Manchester families, that we are thus not under-estimating the amount of food required. It will be seen presently that fat can now be obtained as cheaply as carbohydrate.

The amount of food required by the various members of the family is given by Atwater in terms of the above as follows:—

A child	under 2	requires	0·3	the food of a man doing moderate work.	
„	of 3 to 5	„	0·4	„	„
„	of 6 to 9	„	0·5	„	„
„	of 10 to 13	„	0·6	„	„
A girl	of 14 to 16	„	0·7	„	„
A boy	of 14 to 16	„	0·8	„	„

A woman may be taken to consume 0·9 except when with child, in which case her diet should be increased. This is also true of lactation.

Current Prices of Ordinary Foods. We now require a statement of the ordinary foods, with their current prices, so that we may construct dietaries on the scale given above, and square the price with the means of the family.

The following table, collected from Hutchinson's book, gives the necessary materials for a number of typical diets, and the lowest prices are attached, at which wholesome articles can be obtained at present in Manchester:—

TABLE V.

Composition of Foods.

100 grammes of	Prices.	Water.	Proteid.	Fat.	Carbo-hydrate.	Salts.
Beef, medium fat	6d. per lb.	76.5	20.0	1.5	—	1.3
Mutton, lean	5d. per lb.	75.0	18.0	5.7	—	1.3
,, medium fat	5d. per lb.	65.2	14.5	19.5	—	0.08
,, very fat	5d. per lb.	46.0	10.2	43.2	—	0.06
Pork, medium fat	6d. to 8d. per lb.	60.9	12.3	26.2	—	0.05
,, very fat	—	44.4	9.7	45.5	—	0.04
Bacon	6d. per lb.	22.3	8.1	65.2	—	4.4
Liver, ox	4d. per lb.	71.2	20.7	4.5	1.5	1.6
Heart, ox	3½d. per lb.	62.6	16.0	20.4	—	1.0
Tripe	3d. per lb.	74.6	16.4	8.5	—	0.5
Herring, cooked	2 for 1d. = ½lb.	52.99	23.67	8.72	—	—
Cow's milk, average	3½d. quart	87.4	3.5	3.5	5.0	0.6
Butter	1s. per lb.	15.0	2.0	82.0	—	—
Margarine	8d., 6d., 4d. per lb.	9.3	1.3	82.7	—	6.7
Cheese, American	6d. per lb.	26.9	32.9	31.0	—	4.5
,, Cheshire	6d.—9½d.	33.2	29.4	30.7	—	4.3
Wheat meal	1d. per lb.	12.1	12.9	1.9	70.3	1.2
Fine flour	1⅛d. to 1½d. lb.	13.0	9.5	0.8	75.3	0.7
Oatmeal	2d. per lb.	7.2	14.2	7.3	65.9	1.9
Barley	2d. per lb.	12.3	10.1	1.9	69.5	2.4
Barley meal	2d. per lb.	11.9	10.0	2.2	71.5	2.6
Pearl barley	3d. per lb.	12.7	7.4	1.2	76.7	1.2
Fine corn meal	—	12.5	6.8	1.3	78.0	0.6
Rice, husk removed	3d. per lb.	12.0	7.2	2.0	76.8	1.0
Bread, white	2d. = 2 lb.	40.0	6.5	—	52.5	1.0
Whole meal	,, ,,	45.0	6.3	—	47.5	1.2
Hovis	3½d. = 2 lb. (Flour 2d.)	45.0	9.9	—	42.3	1.2
Macaroni	4d. = 1 lb.	12.0	10.89	0.65	75.70	0.50
Semolina	3d. = 1 lb.	10.6	11.96	0.60	75.79	0.65
Dried peas	1½d. split peas; 2d. & 2½d. marrowfat.	13.0	21.0	1.8	55.4	2.6
Lentils	2d. per lb.	11.7	23.2	2.0	58.4	2.7
Haricot beans	2d. per lb.	11.7	23.0	2.3	55.8	3.2
Potatoes	1s. 2d. per score, best	76.7	1.2	0.1	19.1	0.9
Carrots	2 lb. for 1d.	85.7	0.5	0.3	10.1	0.9
Turnips	2 lb. for 1d. (given as 5 lb. for 2d.)	90.3	0.9	0.06	5.0	0.8
Cabbage	1d. to 2d. for 1	89.6	1.8	0.4	5.8	1.3
Cauliflower	3d.	90.7	2.2	0.4	4.7	1.2
Apples	2d. per lb. (very dear)	82.5	0.4	0.5	12.5	Acids. 1.0
Currants	5d. per lb.	27.9	1.2	3.0	64.0	2.2
Raisins	6d. per lb.	14.0	2.5	4.7	74.7	4.1
Best sugar	1 lb. for 2d.	2.9	—	—	92.0	2.56
Treacle	,, ,,	23.4	—	—	69.7	—
Honey	1 lb. 6d.	19.98	—	—	73.95	—
Lard	4d. to 6d.	—	—	—	—	—
Suet	6d. to 8d.	—	—	—	—	—
Dripping	4d. to 6d.	—	—	—	—	—
Eggs	One = 2 ozs. for 1d.	65.5	13.1	9.3	—	—

In applying this dietary to childhood we have first to consider the question of **milk.** As a source of proteid milk is expensive, and children over 3 years of age get very little of it in the poorest class. Dr. Ashby says it should be used all through childhood. It is especially rich in lime and phosphates, and when it can be got fresh is a valuable addition to the diet. **Eggs** are also a valuable food, being rich in iron, lime and phosphorus, but are still more expensive as a source of proteid.

An effort should be made to supply milk (which should, as usually supplied, always be boiled, cooled and kept covered over in a cool place) up to the age of six, but probably amongst the very poor up to that age it will not he possible. It should be given without fail up to the end of the third year.

Fat is on the whole a more expensive article of diet than carbohydrate. According to Ashby, it is an indispensable one to children, as a deficiency of fat in the diet is liable to produce scurvyrickets. Children, however, often have a great objection to crude fat, though it is always possible to get them to take it in combination, as in pastry, pasty, pudding, on bread or toast, or in combination with potatoes, pulses, and cabbage, and it may usefully be given as toffee or chocolate, where the means will allow.

In the case of **meat** not too fat, it may be given as mince, in which form it may be economically cooked as a pasty.

Children do not always find **peas** or **beans** easy to digest, and care must therefore be expended in cooking these. Even the poorest Lancashire people prefer marrowfat peas to split peas, and very possibly, when soaked for 12 hours, and then cooked, they are easier to digest, as they are certainly more appetising.

To children, quite as much as to adults, variety of foods is essential, perhaps more so. By examination of the fæces it is ascertained that a certain proportion of the proteid, fat and carbohydrate is not absorbed, and that this proportion is different for different foods. The proteid in pulses is imperfectly absorbed, about 20 per cent. escaping; with brown bread the proportion may be 20 per cent. or more, while with potatoes it amounts to 30 per cent. But this proportion is reduced when these articles form part of a mixed diet. Fats and carbohydrates are much more completely absorbed.

Care of Teeth and Intestine. Children require training in masticating their foods, and parents need to pay special attention to this matter. Further, the importance of attending to their teeth should be recognised, and arrangements should be made at Hospitals where children are treated, for the purpose. It is of the greatest consequence that parents should be taught to see that the bowels are regularly moved, as neglect in this particular leads to serious depression in health. This is of especial importance where the diet is but little varied, and is abundant.

Regularity of Meals, Variations in the Diet. The meals should be taken at regular times, though probably no great harm is done by a piece of bread and butter being given between meals, provided the child takes his regular meals well. Not only should the food be varied as much as the means will allow, but by variety in preparation of the same article of food, the distaste which sameness produces may be avoided. Thus herring may be steamed, fried, stewed in vinegar or baked in a pasty or grilled. Flour may be used as

bread, cake, pudding, pasty, pastry, gravy. Oatmeal may be used as cake, porridge, pudding, or fried with fat, onions, pepper, and salt. Peas or beans may be steamed, made into soup, or taken as a stew.

In the preparation of a cheap dietary a knowledge of cooking, therefore, plays a primary part, and it should be a necessary part of a girl's education, as well as of a boy's, if he so wishes. These elementary, but important, points, girls should be taught as part of their training in domestic economy and hygiene.

Diet appropriate to different ages. It is necessary that the proteid consumed should be varied at different periods of growth in accordance with the requirements of proteid for growth. It is probable that this would, in many instances, be the most economical mode of arranging the diet, but practically it is not possible, and the simpler way is to make an increased allowance for proteid, fat and carbohydrate, which is no doubt what Atwater has done in his scale. It may, however, be interesting to see what would be the maximum change produced in the diet at different periods of growth. In Dr. Clement Duke's book on school diet is given a table of average height and weight of boys and girls of English-speaking races, calculated from the total of British and American Statistics.

TABLE VI.

Showing the average and mean height and weight, and the annual rate of increase, of 7,709 boys and men, between the ages of 10 and 30 years, of the most favoured classes of the English population—public school boys, naval and military cadets, medical and university students. (Dukes on School Diet).

	Height without shoes.			Weight, including clothes of 9 lbs.		
Age last birthday	Average inches	Growth inches	Mean inches	Growth inches	Average lbs.	Growth lbs.	Mean lbs.	Growth lbs.
10	53.40	—	53.00	—	67.4	—	67.0	—
11	54.91	1.51	54.50	1.50	72.9	5.50	73.0	6.0
12	56.97	2.06	56.50	2.00	80.3	7.39	80.0	7.0
13	58.79	1.82	58.50	2.00	88.6	8.27	88.0	8.0
14	61.11	2.32	61.00	2.50	99.2	10.61	98.0	10.0
15	63.47	2.36	63.50	2.50	110.4	11.21	110.0	12.0
16	66.40	2.93	66.50	3.00	128.3	17.92	126.0	16.0
17	67.84	1.46	68.00	1.50	141.0	12.69	140.0	14.0
18	68.29	0.43	68.50	0.50	146.0	4.97	146.0	6.0
19	68.72	0.43	68.75	0.25	148.3	2.20	148.0	2.0
20	69.13	0.41	69.00	0.25	152.0	3.87	150.0	2.0
21	69.16	0.03	—	—	152.3	0.27	152.0	2.0
22	68.93	—	—	—	154.7	2.44	—	—
23	68.53	—	—	—	151.7	—	—	—
24	68.95	—	—	—	149.2	—	—	—
25–30	69.06	—	69.00	—	155.2	0.42	154.0	2.0

The greatest increase in weight in the case of boys occurs at the age of 15 when the gain in weight is 15·40 lbs. and in girls at the age of 12 when the gain is 10·47 lbs. The boy of 15 taking 100 grammes of proteid a day, and the

girl of 12 taking 75 grammes, the total amount used by the former in his fifteenth year of age is 81 lbs., by the girl in her twelfth year 60·8 lbs. Supposing that these amounts were required for repair and energy independently of growth, the boy requires nearly one-fifth and the girl nearly one-sixth additional proteid for growth, on the assumption that the increase in weight is entirely in proteids, which is not quite correct. The nutritive ratio for the boy becomes 1 to 5 and for the girl 1 to 5·14.

The matter is, however, not of much practical moment. Carbohydrates act as proteid sparers, and enable the system to do with less proteid for immediate purposes. The quantity required for repair and building is thus reduced, and nitrogenous equilibrium may be established on a smaller amount of proteid. The use of a certain amount of proteid for immediate work appears to increase vitality and resistance to disease, so that it is not desirable to go very far in this direction. If, however, the boy or girl consumes the additional food in the shape of bread, the rest of the diet being sufficiently varied, all practical purposes will be served.

Diet from the economical point of view. I propose now to discuss the subject briefly from the point of view of economy. Proteid being the most essential article of diet, and also the most expensive, it is desirable to know how it may be most cheaply obtained. This is shown in the following table, modified from Hutchinson's figures:—

TABLE VII.

(Modified from Hutchinson on Dietetics.)

Taking the analyses and prices given in Table V., we find that a pound of proteid costs:—

	s.	d.	
Beef	2	6	
Frozen Mutton	2	$3^{4}/_{5}$	
Liver, Ox	1	$7^{3}/_{10}$	
Heart, Ox	1	$9^{9}/_{10}$	
Herring	0	$8^{45}/_{100}$	to 5d.
American Cheese	1	$6^{1}/_{10}$	
Cheshire ,,	1	$8^{4}/_{10}$	
1d. Wholemeal	0	$8^{1}/_{5}$	allowing for 20% loss $10\frac{1}{4}$d.
Fine Flour	1	$0^{3}/_{10}$	allowing for 10% loss 1s. $1^{6}/_{10}$d.
2d. Oatmeal	1	$2^{1}/_{10}$	
White Bread	1	$3^{4}/_{10}$	
Hovis	About same.		
	s.	d.	Not absorbed.
Dried Peas, split	0	$7^{1}/_{10}$	—20%, $8^{9}/_{10}$d.
,, Marrowfat	0	$9\frac{1}{2}$	—20%, $11^{9}/_{10}$d.
Lentils	0	$8^{6}/_{10}$	—10%, $9^{5}/_{10}$d.
Haricot Beans	0	$8^{7}/_{10}$	—30%, 1s. 4d.
Milk	3	4	
Eggs	5	1	

As regards the total calories derivable from the amount of different foods which can be bought for the same sum, these are as follows:—

TABLE VIII.

(Modified from Hutchinson on Dietetics.)

The number of Calories obtainable for a shilling without deductions:—

Refined Cotton Seed Oil	16,740
Fine Flour at 1/2 the dozen lbs.	15,636
Hovis Bread at 1½d. per lb.	10,764
Oatmeal	10,894
Beet Sugar	10,186
Peas	8,921
Lard, Dripping, Margarine at 4d. per lb.	8,652
Herrings, 2 for 1d.	4,811
Potatoes	3,796
Milk	3,000
Butter	2,884
Cheese	2,638
Apples	1,529

We thus see that flour is the cheapest source of energy in general use, and that bread, oatmeal and sugar occupy the same rank, while the cheaper fats and oils are not much behind, ranking with the pulses.

Having thus obtained a clear idea of the foods which may be selected to give the cheapest foods, we note that the following simple combinations are useful:

Bread and Milk.
Bread and Cheese.
Bread and Herrings.
Bread and Margarine or Oil.
Oatcake and Margarine.
Oatcake and Cheese.
Oatmeal as porridge and Sugar.
Peas and Margarine or Oil.

From the table showing the analysis of foods into water, proteids, fats, carbohydrates and salts we can construct dietaries, and I show diets, for a man of average weight (11 stone) doing moderate work, for every day in the week, giving a fair amount of variety. They are arranged to contain a quart of milk so as to allow each of the children to get a share. The result of this is to raise the price of the dietary from 8d. to 10d. as may be seen on substituting for a quart of milk at 3½d., giving P 34·5grm., F 35·4grm., C 50·25grm., the following:

		P	F	C
		grm.	grm.	grm.
Cheese	20ȝ for ¾d.	18.51	17.44	—
Beans	20ȝ for ¼d.	14.85	1.3	32.96
Sugar	10ȝ for ⅛d.	—	—	25
Dripping	10ȝ for ¼d.	—	24	—
	Price 1⅜d.	33.36	42.74	61.08

In this way we save 2⅛d.

As these diets with milk do not depart much in price from 10d., in applying them to families we shall treat them as 8d. diets, the above substitution being supposed, assuming that Atwater in his dietary scale has made allowance for the necessity of giving milk to very young children.

I have then endeavoured to reduce the price of the diets as much as possible by varying the combination of cheaper foods, and seven such dietaries are shown with their prices, the utmost reduction being to 4¼d., as shown on cheap diet 6. A fair diet may, however, be given for 3¾d., as shown in No. 7.

It is conceivable that this might be depressed a little further by using flour instead of bread, and refined cotton seed oil as a fat. Such vegetable oil is the cheapest of all sources of energy. It is of the greatest importance to depress the price of the dietary as far as possible, since every fraction of a penny that we can save can be expended on vegetables, such as cabbage, turnips, carrots, or on some cheap fruit, such as apples, oranges or bananas. Cabbage may be obtained at 1d. each, turnips at 2d. for 5 lbs., oranges at four for 1d., and bananas, I am told, at four for 1d. A small portion of these make a valuable addition to the dietary.

The following are the dietaries constructed to yield, most of them, P 125, F 125, C 450.

TABLE IX.

Monday's Diet.

Materials.	Price.	Proteid.	Fat.	Carbohydrate.
Bread, 1 lb.	1d.	29.75	4	232.25
Potatoes, 1 lb.	¾d.	5.4	—	85.95
Milk, 1 quart	3½d.	35.4	35.4	50.25
Rice, 25 grammes at 3d. per lb.	⅙d.	1.8	0.25	19.2
2 herrings = ½ lb. cooked	1d.	50.12	19.6	—
Bacon, 2 ozs.	¾d.	4.56	36.68	—
Margarine, 1½ ozs.	$^{7}/_{16}$d.	—	36	—
Sugar, 2 ozs.	¼d.	—	—	56.25
Tea, ¼ oz.	¼d.	—	—	—
Price	$8^{5}/_{48}$d.	127.03	131.93	443.9

Meals.

Breakfast: Milk 1 pint, 1 herring, ¼ lb. bread, ½ oz. margarine.
Dinner: Bacon 2 oz. fried, potatoes ½ lb., rice pudding ($^{1}/_{16}$ lb.), milk 1 pint.
Tea: Tea ⅛ oz., sugar 1 oz., margarine ½ oz., bread ½ lb., 1 herring.
Supper: Potatoes fried in bacon fat ½ lb., margarine ½ oz., bread ¼ lb.

Tuesday's Diet (for an adult man).

Materials.	Price.	Proteid.	Fat.	Carbohydrate.
Bread, 1 lb.	1d.	29.75	4.0	232.25
Milk, 1 quart	3½d.	35.4	35.4	50.25
Lean Mutton at 6d., ⅜ lb.	2¼d.	30.375	9.42	—
Peas, at 2d., $^{5}/_{16}$ lb.	⅝d.	29.5	2.5	77.9
Margarine, 3½ ozs.	¾d. + ⅛d.	—	84.0	—
Vegetables	¼d.	—	—	—
Sugar 2.6 oz.	2¾d.	—	—	—
Tea, ¼d.	¼d.	—	—	—
Price	9⅛d.	125.025	135.3	440.4

Breakfast : Bread ¼ lb., milk 1½ pints, tea ⅛ oz.

Dinner : Lean mutton ⅜ lb. fried and then stewed, with the peas previously cooked, cabbage separate and margarine 1½ oz., bread ¼ lb.

Tea : Tea ⅛ oz., bread ½ lb., margarine 1 oz., sugar 2.6 oz.

Supper : Bread ¼lb., margarine 1 oz., milk ½ pint.

Wednesday.

Materials.	Price.	Proteid.	Fat.	Carbohydrate.
Pork ¼ lb.	2d.	13.84	29.48	—
Milk, 1 quart	3½d.	35.4	35.4	50.25
Cheese, 2 ozs.	¾d.	18.51	17.44	—
Windsor Beans, ⅛ lb.	¼d.	14.85	1.3	32.96
Bread, 1 lb.	1d.	29.75	4.0	232.25
Margarine, 1½ ozs.	⅞d.	—	36.0	—
Cabbage	¼d.	—	—	—
Potatoes, 1 lb.	¾d.	5.4	—	85.95
Sugar, 2 ozs.	¼d.	—	—	56.25
Tea, ¼ oz.	¼d.	—	—	—
Price	9⅞d.	117.75	123.62	457.66

Breakfast : Bread ¼ lb., 1 pint milk, ½ oz. margarine, tea ⅛ oz.

Dinner : Pork ¼ lb., beans ⅛ lb., bread ¼ lb., caggabe and margarine.

Tea : Tea ⅛ oz., bread ¼ lb., margarine ½ oz., sugar 2 ozs.

Supper : Fried potatoes 1 lb., milk 1 pint.

Thursday.

Materials.	Price.	Proteid.	Fat.	Carbohydrate.
Mutton, ½ lb.	3d.	32.63	43.87	—
Milk, 1 quart	3½d.	35.4	35.4	50.25
Suet Pudding, 2 ozs. (¼ oz. suet, 1 lb. flour.	0.35d.	5.80	10.98	—
Porridge (2 ozs. oatmeal)	0.⅜d.	7.99	4.11	37.07
Rice, 2 oz., husk removed	⅜d.	4.05	1.13	43.2
Bread, 1 lb.	1d.	29.75	4.0	236.25
Sugar, 2 ozs.	¼d.	—	—	56.25
Tea, ¼ oz.	¼d.	—	—	—
Cheese, 1½ ozs.	$^{9}/_{16}$d.	13.424	12.55	—
Turnip, ¼ lb.	¼d.	1.01	—	5.62
Margarine, 1½ ozs.	⅝d.	—	36.0	—
Cost of diet	10.6d.	130.05	148.04	428.64

Breakfast : Porridge (2 ozs. oatmeal), milk 1 pint, bread ¼ lb., margarine ½ oz.

Dinner : Mutton ½ lb., turnip ¼ lb., suet pudding 2 ozs., sugar 1 oz.

Tea : Tea ¼ oz., bread ¼ lb., margarine ½ oz., sugar 1 oz.

Supper : Bread ¼ lb., cheese 1½ ozs., margarine ½ oz., rice pudding (2 ozs. rice), milk 1 pint.

Friday.

Materials.	Price.	Proteid.	Fat.	Carbohydrate
Beef, ½ lb.	3d.	45.0	3.37	—
Macaroni 1 oz.	¼d.	3.06	—	21.29
Milk, 1 quart	3½d.	35.4	35.4	50.25
Cheese, 1 oz.	⅜d.	9.25	8.72	—
Bread, 1 lb.	1d.	29.75	4.0	232.25
Sugar, 2 ozs.	¼d.	—	—	56.25
Potatoes, 1 lb.	¾d.	5.4	—	85.95
Margarine, 3 ozs.	¾d.	—	72.0	—
Orange	½d.	—	—	—
Tea, ¼ oz.	¼d.	—	—	—
Price	10⅜d.	127.86	123.5	445.99

Breakfast: Milk 1 pint, bread ¼ lb., margarine 1 oz.
Dinner: Beef ¼ lb., potatoes 1 lb., macaroni 1 oz., milk ¼ pint with sugar, orange.
Tea: Tea ¼ oz., sugar 1 oz., bread ¼ lb., margarine 1 oz.
Supper: Bread ¼ lb., cheese 1 oz., margarine 1 oz.

Saturday.

Articles.	Price.	Proteid.	Fat.	Carbohydrate.
Bread, 1 lb.	1d.	29.75	4.0	232.25
Milk, 1 quart	3½d.	35.4	35.4	50.25
Bacon, 2⅔ ozs.	1d.	4.30	36.67	—
Liver ¼ lb.	1d.	25.98	10.12	5.62
Potatoes, 1 lb.	¾d.	5.4	—	85.95
Cabbage	¼d.	—	—	—
Cheese, 3 ozs.	1⁵/₁₆d.	26.84	25.1	—
Sugar, 2.6 ozs.	³/₁₆d.	—	—	80.0
Margarine, ½ oz.	½d.	12.0	—	—
Tea, ¼ oz.	¼d.	—	—	—
Price	9¾d.	139.67	111.29	454.07

Breakfast: Bread ¼ lb., milk 1 pint.
Dinner: Liver and bacon (2⅔ ozs. and ¼ lb.), potatoes 1 lb., cabbage.
Tea: Tea ¼ oz., bread ½ lb., margarine ½ oz., sugar 2 ozs.
Supper: Bread ¼ lb., cheese 3 ozs., milk 1 pint.

SUNDAY.

Materials.	Price.	Proteid.	Fat.	Carbohydrate
Herring 1	½d.	25.06	9.8	—
Bread, 1 lb.	1d.	29.75	4.0	232.25
Milk, 1 quart	3½d.	35.4	35.4	50.25
Pastry: dripping 1 oz.,, flour 2 oz., mutton ¼ lb.	$2^{1}/_{16}$d.	23.56	48.31	39.54
Margarine, 1 oz.	¼d.	—	24.0	—
Egg 1	1d.	—	—	—
Potatoes, ½ lb.	½d.	2.7	—	42.97
Sugar, 2.6 ozs.	$^{5}/_{16}$d.	—	—	80.0
Orange	½d.	—	—	—
Cheese, 1 oz.	⅜d.	9.25	8.72	—
Tea, ¼ oz.	¼d.	—	—	—
Price	10¼d.	125.72	130.23	445.01

Breakfast: Herring 1, bread ¼ lb., milk 1 pint.
Dinner: Pastry, potatoes ½ lb., orange.
Tea: Tea ¼ oz., egg 1, bread ½ lb., margarine 1 oz.
Supper: Bread ¼ lb., cheese 1 oz.

To these I have added the minimum expenditure on which an adult can subsist, which can be extended to children over 3.

CHEAP DIET NO. I.

Articles.	Price.	Proteid.	Fat.	Carbohydrate
Bread, 1 lb.	1d.	29.75	—	232,25
Peas, ¼ lb.	⅜d.	23.6	—	69.2
Turnip, ½ lb.	$^{1}/_{3}$d.	2.02	—	4.02
Margarine, 4 ozs.	1d.	—	101.0	—
Potatoes, 1 lb.	¾d.	5.4	—	85.9
Cheese, 2 ozs.	¾d.	18.51	17.44	—
2 Herrings	1d.	50.12	19.6	—
Tea	⅛d.	—	—	—
Sugar, 2 ozs.	¼d.	—	—	56.25
		129.40	138.04	447.62

The peas in the above are split peas at 1½d. per lb., and are to be made into Soup with the Turnip.

The cost of the above is $5^{3}/_{20}$d. If we add ½d. for share of bone we may call it 6d. But in that case the diet will be increased in point of fat.

CHEAP DIET No. II.

Articles.	Price.	Proteid.	Fat.	Carbohydrate.
4 Herrings	2d. ...	104.26 ...	39.24 ...	—
Bread, 1 lb.	1d. ...	29.75 ...	— ...	232,25
Margarine, 4 ozs.	1d. ...	— ...	101.0 ...	—
Sugar, 4 ozs.	½d. ...	— ...	— ...	96.0
Potatoes, 1 lb.	¾d. ...	5.4 ...	— ...	85.9
Tea	⅛d. ...	— ...	— ...	—
Flour, 3 ozs.	$^{3}/_{16}$d. ...	8.4 ...	— ...	59.31
Price	$5^{9}/_{16}$d. ...	147.81 ...	140.24 ...	473.46

Two of the herrings are here to be made into pasties. It is not a diet which could be often repeated, though an abundant one.

In the above diet it is important to retain all the salts. Hence there should be a steamer for the potatoes.

CHEAP DIET No. III.

Articles.	Price.	Proteid.	Fat.	Carbohydrate.
½ lb. Haricot Beans	1d. ...	51.75 ...	5.175 ...	316.35
¼ lb. Bacon	1½d. ...	8.11 ...	73.35 ...	—
1 lb. Bread	1d. ...	29.75 ...	4.0 ...	232.25
Herring, 1	½d. ...	25.06 ...	9.8 ...	—
Margarine, 1 oz.	¼d. ...	— ...	24.0 ...	—
American Cheese, 6d., 1 oz.	¾d. ...	9.25 ...	8.72 ...	—
Tea	⅛d. ...	— ...	— ...	—
Sugar, 4 ozs.	½d. ...	— ...	— ...	112.5
Total price...	5⅝d. ...	123.92 ...	125.045 ...	470.28

Breakfast: Tea $^{1}/_{16}$d., herring 1, bread 6 ozs.
Dinner: Bacon and beans as above, bread 2 ozs.
Tea: Tea $^{1}/_{16}$d., bread 4 ozs., margarine ½ oz.
Supper: Bread 4 ozs., cheese 1 oz., margarine ½ oz.

Still cheaper dietaries could be constructed.

CHEAP DIET No. IV.

Articles.	Price.	Proteid.	Fat.	Carbohydrate.
1 lb. flour, baked into cakes = 20 % proteid.	1⅙d. ...	46.44 ...	8.55 ...	316.53
1 lb. beans boiled = 20 % proteid ...	2d. ...	82.8 ...	10.35 ...	251.1
Margarine, 2 ozs.	½d. ...	— ...	48.0 ...	—
Dripping, 2 ozs.	½d. ...	— ...	48.0 ...	—
Sugar, 1 oz.	⅛d. ...	— ...	— ...	28.12
Tea, ¼ oz.	¼d. ...	— ...	— ...	—
Price	$4^{13}/_{24}$d. ...	129.24 ...	114.90 ...	595.57

DIETARY No. V.

Articles.	Price.	Proteid.	Fat.	Carbohydrate.
2 Herrings	1d. ...	50.12 ...	19.6 ...	—
Beans, ½ lb.	1d. ...	40.0 ...	5.175 ...	125.0
Bread, 1 lb.	1d. ...	29.7 ...	4.0 ...	232.5
Sugar, 2 ozs.	¼d. ...	— ...	— ...	56.25
Tea, ¼ oz.	¼d. ...	— ...	— ...	—
Dripping, 1 oz.	¼d. ...	— ...	24.0 ...	—
Margarine, 2 ozs.	½d. ...	— ...	48.0 ...	—
Treacle, 2 ozs.	¼d. ...	— ...	— ...	39.275
Price	4½d. ...	119.82 ...	100.78 ...	453.2

CHEAP DIET No. VI.

Articles.	Price.	Proteid.	Fat.	Carbohydrate.
Peas, 1 lb.	1½d. ...	94.5 ...	8.1 ...	249.3
Bread, ½ lb.	½d. ...	14.875 ...	2.0 ...	116.125
Herring, 1	½d. ...	25.06 ...	9.8 ...	—
Fat, 2½ ozs.	½d. + ⅛d. ...	— ...	60.0 ...	—
Bone	¼d. say. ...	5.0 ...	10.0 ...	—
Turnip	⅛d. ...	— ...	— ...	—
Sugar 2 ozs., Treacle 2 ozs.	½d. ...	— ...	— ...	95.0
Tea, ¼ oz.	¼d. ...	— ...	— ...	—
Price	4¼d. ...	139.44 ...	89.9 ...	460.4

Calories, 3385.4

CHEAP DIET No. VII.

Articles.	Price.	Proteid.	Fat.	Carbohydrate
Bread, 1½ lbs.	1½d. ...	45.0 ...	6.0 ..	348.0
Sugar, 4 ozs.	¼d. ...	— ...	— ...	103.0
Herring, 2	1d. ...	50.12 ...	19.6 ...	—
Margarine, 1 oz.	¼d. ...	— ...	24.0 ...	—
Oatmeal, 2 ozs.	¼d. ...	7.99 ...	4.11 ...	37.07
Peas, 2 ozs.	¼d. ...	11.2 ...	5.0 ...	31.18
Tea, ¼ oz.	¼d. ...	— ...	— ...	—
Price	3¾d. ...	114.3 ...	58.7 ...	519.2

Calories = 3141.0

Breakfast: Porridge and sugar 3 ozs., bread 6 ozs., and 1 oz. margarine, tea ⅛ oz.
Dinner: Herrings and bread 6 ozs., peas and ¼ oz. margarine.
Supper: Bread 6 ozs. and margarine ½ oz., tea ⅛ oz., sugar 1 oz.

Examples of dietaries of poor families. These dietaries are applied to the circumstances of families thus:—By means of enquiries instituted through the Health Visitors and Sanitary Inspectors it has been ascertained in a number of poor families how much income there is, and what is the amount available for and expended on food. Where the latter is in excess of the former it may be assumed that the income is at present below

the normal level and that the family is falling into arrears. The articles of diet consumed by the family are ascertained and by comparison of the foods used with those in which the diet on Atwater's Scale can be given, we may ascertain whether the children are receiving enough food.

The particulars for a few families are as follows:—

FAMILY 1.

	m.	f.	f.	m.	f.	f.	
Members, with ages	49 ...	50 ...	18 ...	12 ...	11 ...	9	
Earnings	17/-		6/-	5/-		Total	28/-

No. of rooms, 4.

Expenditure: Fire and light, 3/0½; rent, 4/-; clothes, 1/8.

Available for food, under 19/2½. Expended on food, 20/- per week.

Foods used: Beef, 8d.; bacon, 8d.; liver, 6d.; cod, 3d.; milk, 3½d.; margarine, 6d.; lard, 8d.; potatoes, 1s. a score; jam, 4½d.; sugar, 2d.; American cheese, 8d.; cabbage, 1d.; carrots and turnips, 2 lbs. for 1½d.; tinned milk, 2½d. per tin; tea, 1/2 per lb.

At 7½d. per head of the household, the expense on Atwater's Scale equals 19/8¼. At least 7d. is available, and this should give a good and varied diet, though not in the above articles.

The family do not appear well nourished. Very little alcohol used.

FAMILY 2.

	m.	f.	f.	f.	m.	f.	f.	m.	f.
Age	39 ...	39 ...	13 ...	11 ...	8 ...	6 ...	4 ...	2 ...	3m.

Earnings, 19/-.

No. of rooms,4.

Expenditure: Rent, 4/9; clothing, 10d.; Fuel, etc., 2/2; total, 7/9.

Available for food, 11/3; expenditure, about 12/-.

Food used:—Breakfast: Dry toast and tea. Dinner: Potato hash, sometimes pea soup. Tea: Tea, bread and margarine. Supper: Children have bread, parents tea and bread.

On a milk diet at 10d., with Atwater's Scale, this family would cost 37/0½ at 7d., with the three youngest children on milk diet it would cost 24/6. With the six older members at 4½d. per day and the three young children specially dieted on milk 14s. 6d.

The family can be adequately fed for 3¾d., for which, as we have seen, a possible diet can be given. But with the materials actually used the family is hopelessly underfed.

FAMILY 3.

	m.	f.	f.	f.	m.	f.	f.	f.	f.
Age	41 ...	40 ...	20 ...	18 ...	13 ...	9 ...	6 ...	4½ ...	1

Rooms, 5. Earnings, 29/6.

Deducting 10/2 for rent, fuel, clothes, club and school money, there remains 19/4 available. Actual sum expended 20/-.

On a 5d. diet the family could be supported for 17/2½. A 6d. diet would cost about 20/8.

The expenditure here in detail is:—Breakfast: Tea, bread and bacon (6d.). Dinner: Stew, broth, pea soup, potatoes, cabbage. Tea: Tea, bread and

margarine or dripping. This is not so conspicuously deficient, and the family, as a matter of fact, appears well nourished.

The prices are of interest: Beef, 2½d.; bacon, 6d.; liver, 4d.; bones, 1d. worth; herrings, 3 and 4 for 1d.; cod, 2d.; bloaters, 2 for 1½d.; savoury duck, 1d. each; margarine, 6d.; suet given in; flour 1¼d.; oatmeal, 2d.; rice, 2d.; potatoes, 1s. per score; bread, 2 lbs. for 2d.; sugar, 2 lbs. for 3½d.; peas, 3d. and 1½d.; cabbage, 1d.; turnips and carrots, 2 lbs. for 1d.; tea 1s. 4d. The utmost economy is shown here, and one cannot improve very much on it.

In some cases the mother has found out for herself how to make the money go as far as possible, but it is not possible to pursue this matter into more detail. I therefore throw the material into a table:—

TABLE X.

No.	No. in family	No. of children not working	Money available for for food	Rate at which Atwater's Diet Scale can be given	Expenditure economical
4	7	4	15/9	5½d.	No.
5	8	4	19/9	6d. = 18/10.8d.	No.
6	5	3	16/7½	7½d.	No, healthy.
7	8	5	17/9	5¼d.	Yes, healthy.
8	8	3	31/-	About 8d.	Yes, healthy.
9	9+ lodger	5	25/5	7d.	No, healthy.
10	7	2	24/-	8d.	Fairly healthy.
11	5	3	22/6	8d. = 18/1.6	Yes, healthy.
12	5	3	13/9	7d.	No, healthy.
13	8	5	24/- to 14/-	8d. to 4¾d. now.	No, not healthy.
14	9	7	8/1	3d. yields 9s. 4¹/₅d. on Atwater's scale.	Yes, not healthy.
15	6	4	7/6½	3¾d. yields 7/7⅞d.	Not very
16	6	2	9/9	3d. yields 8/9	No.
17	9	6	11/2	4d. gives 11/5	Yes, proteids deficient.
18	7	4	12/-	4½d. sufficient.	Yes.
19	6	4	7/3	3½d. yields 7/1¾	No.
20	6	4	16/6	7d. yields 15/11d.	No.
21	6	4	12/-	6d. yields 12/11d. (5½d.)	Fairly.
22	5	3	17/9	8d. yields 14/11½d.	Yes.
23	6	4	11/3	Diet 5½d.	No.
24	5	3	5/6	4d. would require 7/-	Yes.
25	3	0	1/6 + (?)	4d. would require 7/-	
26	4	2	12/-	8d. = 12/1.6d.	No.
27	7	5	14/-	5d. = 14/1	No.
28	8	5	16/-	5¾d. = 15/11	No.
29	7	4	16/-, spent 15/-	5½d. = 16/1	No.
30	5	3	16/-, spent 15/-	8d. = 14/5½	Fairly.
31	7	5	12/6	5d. = 13/1.5	No.
32	6	4	Available 24/-, spent 18/-	8d. = 16/9½	No.
33	7	5	Available 27/6, spent 20/-	7d. = 19/7.2d.	No, more food required.
34	7	2	Available 22/-, spent 20/-	7d. = 20/9.8d.	No.

From a study of the particulars given in these and other enquiries it appears that when the pinch begins the stress does in reality often fall on the wife and children or on the children who are not yet able to earn money.

Out of the 34 given in table 10, which are taken as they come, it will be seen that only in four instances is it quite impossible to provide an adequate diet, in which case it may be said that the families are destitute. The standard taken is $3\frac{3}{4}$d. per man. In the majority of cases, however, owing largely to want of knowledge of the means by which a sufficient diet may be secured, and judging from the articles used, the diet value is far below Atwater's standard. A strenuous effort should therefore be made to impart the necessary knowledge, both to parents and to school children.

Expensive articles of food. The great choice of tasty but expensive articles in towns appears to me to be a serious evil, and goes far to explain the inferior physique of the townsman. Efficiency in diet is sacrificed to quality. It is very essential in particular that the poorer classes should be made acquainted with the value of the cheaper sources of proteid, such as herring, the pulses, oatmeal, flour, how to cook them, and to what extent they can afford to use meats.

Physical exercise. Some years ago I had an enquiry made by the health visitors into the relative healthiness of native children and of the children of immigrants from the country, and no differences could be perceived. I infer that there is no natural inferiority in the townsman, and I think it is probable that, if properly fed and cared for the town child would have nearly as good a physique as the country child.

If children are to reap the benefit of physical exercises they must receive sufficient food, and, provided they are taking exercise, there is no reason why they should not take that food in the most economical forms. Exercise with a sufficient diet is in general highly beneficial, without it injurious.

I am satisfied that at present the poorer classes require clear instruction in dietetics and cooking, and in a large number of cases assistance as well. But judicious guidance alone would be very useful.

Alcohol. In the above table I have given the money available, after known sources of expenditure had been deducted from the wages. It is rarely that anything is left for alcohol, or it is at a most a very small amount. But there are doubtless many destitute families rendered so through drink. These the health visitors would not be able to investigate for evident reasons. I am satisfied, however, that the money stated to be expended on food in the returns used is so expended within narrow limits.

Housing in relation to feeding. It will have appeared that diet has been treated as the largest factor in the gain and loss of health of children, and it is evident that this must be so. It is on the quantity and quality of the food that the greatest change can be imposed. The question of housing is no doubt an important one, and it is interlocked with that of food. People want more house-room and better surroundings, and if their means allow they

will generally have both. If their means are inadequate, according to their tastes, some will put up with poorer housing, others with more meagre fare. The latter is probably the more dangerous policy to pursue.

If, however, they are compelled to avoid crowding, they must, when pinched, get less food, and *vice versa*. No doubt a good deal, both in food and housing, might be extracted from the alcohol now consumed, but a very large number of cases will remain, in which the course to be pursued on the total income has to be carefully weighed, in regulating the housing and diet of families.

Preserved foods. It is found that not a few, even of the poorest families, use jams which are, of course, made from glucose. In temperance drinks, as well as beer, glucose is used. Tinned foods are also largely used. They save trouble, and are not always uneconomical. Condensed milks again are largely used. It is clear that these substances require to be sampled and analysed. If, also, we are to put no hindrance in the way of using cheap oils, lard, dripping and margarine, their composition and safety should be ascertained. The presence and amount of preservatives present in foods should be determined by analysis. The bacteriological safety of foods also requires investigation. A specially watchful attention should be directed to preserved foods, and cheap articles of diet. But for our immediate purpose, these are not the most important matters.

III. THE FEEDING OF INFANTS.

Death Rates of Children under 1 year. We now come to consider the feeding of infants under one year of age. There has been no reduction in the mortality of infants as measured by the number of deaths in the first year of life per 1,000 births, corresponding to that which has taken place in children at higher ages. The facts for the whole country are as follows:— I give the number of deaths under one year per 1,000 births in successive decennia, and also the deaths less diarrhœal diseases. The effect of decennial seasonal variations are in the latter figures to some extent eliminated. The general effect is one of improvement up to the last decennium, when retrogression has occurred. This is more clearly perceived if diarrhœa be excluded.

TABLE XI.

Deaths per 1,000 births in decennial periods, England and Wales.

	Births	Deaths under one year	Rate per 1000 births	Rate less diarrhœa and cholera
1851–60 ...	6,471,650 ...	996,630 ...	154 ...	141
1861–70 ...	7,500,096 ...	1,155,182 ...	154 ...	137
1871–80 ...	8,588,782 ...	1,277,326 ...	149 ...	132
1881–90 ...	8,890,238 ...	1,259,860 ...	142 ...	128
1891–00 ...	9,155,153 ...	1,403,719 ...	153 ...	135

In Manchester City, since 1891, the total effect is that the mortality rate has increased, though, if diarrhœa be excluded, a marked improvement is shown.

TABLE XII.

Death-rates under 1 year per 1,000 Births, Manchester.

Year	City. All causes.	All causes less Diarrhœa.
1891	192	176
1892	178	162
1893	201	165
1894	159	145
1895	202	166
1896	176	151
1897	195	156
1898	196	150
1899	205	157
1900	189	153
1901	198	150
1902	151	138
Average, six years, 1891–1896 ...	184.6	160.8
,, ,, 1897–1902 ...	189.0	150.7

The figures are not available further back than 1871. Since that time the decennial data agree with those for England and Wales, as shown by the following figures:—

Comparison of deaths per 1,000 of infants (born) from all causes and from diarrhœa, during the decennia 1871–80 and 1881–90 (Manchester, Chorlton and Prestwich Unions).

	Deaths under 1 year per 1,000 births. All Causes.	Diarrhœa & Cholera.	Less Diarrhœa.
1871–80	176	26·0	150
1881–90	169	19·1	150
1891–1900	189·47	31·45	158

The first question we must ask relative to these figures is:—Are they correct? It is possible that they depend to some extent on a changing proportion of infants dying within a few days of birth, who have been registered as still-born. It may be possible to get some information on this point by taking the death-rate of still-born children. The information which the Registrars of the Cemeteries have kindly furnished shows that within the past ten years no material alteration has taken place in the numbers registered. It is improbable, however, that this can have largely affected the figures, and we have to ask how it is that diarrhœa has exacted an increasing infantile mortality, although infant mortality as a whole has declined, in Manchester at all events.

Diminution in maternal lactation. I have been informed by Medical Practitioners that there is a diminution in maternal lactation, a much smaller proportion of mothers now suckling their infants than was formerly

the case, and we know that diarrhœa is powerfully affected by this factor. I have endeavoured to ascertain the cause of this alleged increase in the number of persons who do not suckle their offspring. By some it is ascribed to a lowered vitality, by others to a disinclination to give up their accustomed pleasures.

If the fact be as stated, it will account for the increased diarrhœal mortality. It is to be observed, however, that the increase in the last decennium is a general one over the whole country, and may, therefore, be due to seasonal changes.

Defective feeding. On the other hand, the diminution in the infantile death-rate in Manchester during the 12 years, 1891 to 1902, other than diarrhœal, may reasonably be ascribed to public instruction in infant feeding since 1895. If, as is generally accepted, fatal summer diarrhœa is due directly to impure food, what are the factors which have been at work?

The death-rates per 1,000 births in the City of Manchester and its three Main Divisions in the decennium 1891–1900 are given in the following table:—

TABLE XIII.

Annual Death-rates under 1 year per 1,000 Births for the years 1891–1900 from all causes, and from four selected causes in the City of Manchester and in each of its three Main Divisions.

Disease.	City.	Township.	Northern.	Southern.
Diarrhocal Diseases	31.45	36.83	26.74	30.73
Convulsions	13.07	15.96	9.71	13.19
Lung Diseases	34.91	40.12	31.73	33.39
Found dead in bed	6.00	8.59	3.79	5.62
All causes	189.47	218.93	160.95	187.18

It appears that the total infantile death-rates in the three main divisions of Manchester do not differ from each other nearly to the extent that the diarrhœa death-rates do, and that more than half the differences are to be found under the four headings—diarrhœa, convulsions, lung diseases and found dead in bed. Of these causes we may say that the last is in special relation to intemperance, and here the differences are most profound. Scarcely less so are the differences under convulsions, a cause of death in special relation to errors and neglect in feeding. Lung disease, doubtless, stands in special relation to defective nutrition, and the differences under this head are of the same order as under diarrhœa.

It is impossible to doubt that defective nutrition, from whatever causes arising, is a potent determining factor in fatal diarrhœa, and when I have animadverted on the condition of the milk supply to dealers, the answer has been that there are plenty of infants who get no milk, and they die faster than those do who get the milk so much blamed. This is, of course, true, though all it proves is a deeper poverty and misery. It does not even prove that milk is the best food for infants.

It is worth noting that in North Manchester, which is par excellence the

industrial division of the City, the infantile death-rates are far the lowest from each cause named, and indeed our enquiries show that mothers in these districts with rare exceptions do stay at home and tend their children.

Factors believed to influence Diarrhœa Death-rates. The factors which have been believed to exercise an influence on the diarrhœa death-rates are the annual mean temperature, the mean rainfall, geological conditions of the soil, insanitary conditions such as overcrowding and dirt, defective methods of storing and removing excreta, defects in the milk supply, defective methods of infant feeding.

On the relation of **temperature** and **rainfall** to diarrhœa, it is not necessary here to enlarge. There can be no doubt, I think, that defective nutrition is the largest avoidable factor concerned. There is equally little doubt that a pure **milk supply** would often get contaminated by keeping, and by dirty feeding utensils.

As regards conditions of the **soil,** the soil is probably of importance in regard chiefly of the amount of filth which it can hold near the surface in a moist condition, and its importance becomes comparatively small when suitable methods for the removal of refuse are adopted.

Our observations seem to show that it is possible to overestimate this factor which can only act when the air is grossly contaminated, and when milk has been exposed to the contaminated air for a considerable period. It seems probable, however, that the infection may be carried by **flies.**

Foods such as **milk, fruit, meat pies,** meat shapes, may easily get contaminated in shops, where infection may develop for 12 hours or longer before the food is disposed of.*

The infective matter of diarrhœa can thus be introduced into houses much more effectively than through the air carrying particles of excreta, and the milk and teat may be alike infected from contaminated fruit, the one by means of the mother's hands and the other through both fruit and milk being kept in the same cupboard.

Not only may diarrhœa be communicated in other ways than by food, as by sucking dirty clothes and by other food than milk, but even when milk in the safest forms is given the disease occurs. It occurs comparatively, but not absolutely, rarely when the infant is taking the breast. This is no doubt due to contamination of the nipple. It occurs also not rarely when, so far as can be ascertained, only boiled milk has been used. This may be due to a dirty tube or teat or bottle. But it occurs also not rarely when a spoon is used. It is also not rare in connection with the use of condensed milk.

Important then as the purity and freshness of the milk are, we have by no means done with the causation of summer diarrhœa in infants when we have secured for them a pure milk. On the other hand, milk is probably the most important factor in determining a high death-rate from diarrhœa, not on account so much of the immediate chances of escaping infection as on account of the improvement in nutrition which a good milk supply would produce, and

* See Delépine on "Food Poisoning and Diarrhœa," *Journal of Hygiene*, Vol. iii., No. 1.

the effect of this improvement on the death-rate. Professor Delépine considers from his examination of milk that the freshness of milk is all important in the causation of diarrhœa, and, on the whole, I believe his contention to be sustained by the facts.

We may sum up the most important factors in the causation of fatal diarrhœa as:

1. Insufficient food, overcrowding.
2. Dirt.
3. Milk not sufficiently fresh.
4. Want of knowledge of the conditions requisite in preparing food for infants.

It must be admitted that advance will necessarily be very slow in this direction.*

Malnutrition. It is, of course, not a mere question of diarrhœa. In an excellent paper contributed to the *Medical Chronicle*, Dr. Railton traces to malnutrition the excessive mortality in infants from wasting, convulsions and bronchitis, a subject on which he is well qualified to pronounce an opinion. On convulsions as a sign of bad feeding he lays special stress, and, indeed, I have used this sign as a test of the value of our feeding instructions year by year. In these regards we can show improvement. But a severe task lies before us in trying to reduce the death-rate still further.

Before entering on the subject of the steps which I believe to be necssary to still further reduce infant and child mortality. I must briefly mention to you the efforts which have been made to improve our milk supply.

Improvement of Milk Supplies. They are either in their infancy or are abortive, but in view of the importance of the subject they require attention.

1. *The Pure Milk Supply of Copenhagen.* This Company, whose continued success is due to the energy of Mr. Busck, at one time supplied about a quarter of the milk consumed in Copenhagen. The milk was sold at current prices. The chief features are these:—The cows are kept clean and free from disease, under veterinary inspection. The milk is kept comparatively free from micro-organisms by the use of ice from milking to delivery. Every inducement is given to farmers and employes of the company not to conceal infectious disease.

2. *The Walker-Gordon Laboratories.* Of these, according to Rotch, there were in 1902 eighteen in the States and Canada, and one in London. At these laboratories the whole milk, which must contain only a small number of bacteria, is divided by centrifuging into cream and separated milk, and the caseinogen is removed from the separated milk by the use of rennet. The compounder thus has at his disposal cream mixtures, whole milk, whey, milk sugar, sterilised water, lime water, and can make up from these, within certain limits, any prescription for modified infants' milk which is desired.

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* For a full discussion of the causes of summer diarrhœa see Newsholme, *Public Health*, 1899.

The most elaborate precautions are taken in regard to avoidance of impurities. The cows are stall-fed. Ice is freely used.

3. *The Municipal Sterilisation of Milk.* This was introduced into this country by Dr. Drew Harris, of St. Helens, and is in use also in Liverpool, Battersea, and Ashton-under-Lyne. Whole milk obtained from contractors is taken to municipal stations, is mixed with certain proportions of cream and sugar according to the age of the infants to be fed, is run into bottles, sterilised, sealed and sent to distributing stations. Each bottle contains one feed, and the requisite number of bottles for a day's supply is put up in baskets. Two teats are supplied with the basket. The parent must pay in advance, and the milk is only supplied under stringent conditions, a record being kept of the progress of the infant.

4. *General Administrative Action, common to Manchester and a few other Towns.* This includes ample and successful enforcement of the Sale of Food and Drugs Acts, measures for the prevention of tubercular infection in milk, enforcement in Manchester of regulations under the Dairies, Cowsheds and Milkshops Orders, stringent so far as cowsheds and cows are concerned, and extensive as well as practical instruction in the modification and sterilisation of milk at home. Something is also effected in the way of improving farms outside.

5. *Modification and sterilisation of milk at home* are carried out at home under medical supervision according to methods such as those given in Dr. Ashby's book on "Health in the Nursery."

6. Much good may be effected merely by addition of small amounts of bicarbonate of soda and starch.

Only in the first two methods are milks used which are quite fresh. The milk must therefore be sterilised, and it is not sufficient to Pasteurise it.

Now Professor Delépine has shown the immense importance of using only fresh milk in the reduction of its infectiveness, and has demonstrated that this may be obtained in two ways—either by obtaining the milk from some neighbouring farm, or by keeping it cooled down from milking to delivery by the use of ice or by some other means of refrigeration. The latter procedure remains therefore with our present milk supply, even if it were much improved, an irreducible need.

From the point of view of digestion it is no less important, as a milk which is not fresh produces a hard leathery curd.

Summary of Recommendations regarding the milk supply. *These recommendations may be summed up thus:—It is not sufficient to provide for poor persons a modified milk sterilised. Such milk should be clean and fresh and should be absolutely under control. It should be supplied only under certain conditions, such as those in use in Liverpool, or still more stringent. The condition of the milk supply will not be reasonably satisfactory until the necessity for keeping it cool from milking to delivery is generally recognised. As many persons as can do so should get their milk from

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*In connection with this subject I desire to draw attention to the admirable report on the milk supply of Finsbury, by Dr. Newman, reproduced in the February number of *Public Health*.

the neighbourhood, and should satisfy themselves as to the cleanliness of the cows, cowsheds, milk cans, and water used. Milk kept for a great number of hours in summer, at ordinary temperature, gives a hard, leathery curd, very difficult of digestion, and causes gastric and intestinal irritation. The curd may be softened by the addition of alkali and a little starchy flour to the food, and possibly this may often enable the infant to digest the milk without modification. These additions are much more likely to be effective with clean fresh milk recently drawn from the cow. Home modification on the lines suggested by Dr. Ashby is also much more likely to be effective if the milk is clean and fresh. It is a question whether an effort should not be made to get a number of milk farms established near towns, the the desire for pasture being put aside and the cows stall-fed on a selected and fairly uniform diet. The enforcement of a sufficient degree of cleanliness in milk will only be possible when the milk must pass a certain standard of bacteriological purity.

Instruction of Girls. To feed an infant successfully, however, the person in charge must be trained not only to prepare the food skilfully, but also to keep the infant's person and clothes thoroughly clean, and otherwise to care for it. Experience has abundantly shown that the most careless class do not take advantage of modified milk. Where children are seriously neglected, it is usually the result of drink. In dealing with such persons the Society for the Prevention of Cruelty to Children will be of more avail than lady inspectors.

More instruction is needed, and more, I hope, will be forthcoming. But the Education Authority can do infinitely more in this matter than the Sanitary Committee. Whatever subject girls are or are not instructed in, they ought to be taught the elements of domestic economy and hygiene before they leave school, and such instruction should be practical. They should be taught in regard to infants the vital necessity of cleanliness in and about the house, what they ought to wear and how to dress them, the necessity for keeping their persons and clothing thoroughly clean, especially in the case of posseting babies. The most skilful feeding may be defeated by the absence of such care.

As regards feeding, I see no reason why they should not be taught how to modify milk by some such processes as are described in Ashby's book; each step taken in the storage of milk, in its sterilisation, and in the cleaning of materials being thoroughly explained and practised. It is hopeless to think of overtaking the feeding of infants in any other way than through the schools. They should receive elementary instructions in the value of the different constituents of food, and should be made to use their arithmetic to construct dietaries. It is merely a sum in addition, with the interest of a not very difficult puzzle added. Given the ratio of diets for different ages they could be set to find out how much must be brought in for a family of given ages in a day or a week, and how much it would cost. To most of them the arithmetic which they have learned must be of very little value indeed, if it will not stretch so far as this. In this way money would be made to go as far as possible in providing a sufficient diet. This would do much to avert the craving for liquor which is much more intense in persons insufficiently fed and working hard, than in persons getting enough food. There is no doubt whatever that

not merely something could be done by this means, as Mr. Rowntree says, but a vast economy of health and a great increase of productiveness could be brought about.

As regards the working out of diets, boys should be set the same tasks. They should also be made to calculate out dietaries in use. Further, it should be possible in a certain number of cases to get the home dietaries completely tabulated, and so to ascertain whether the diet was sufficient. These most useful proceedings require absolutely nothing except such arithmetic as school children in the upper standards are supposed to possess, with some practice. It would be no less interesting, and much easier, to calculate out the constituents in the diet of institutions, than to construct fresh dietaries, and would give the opportunity for judicious criticism.

Another immense economy could be effected in cooking. There surely can be no doubt that every girl should be taught practically how to cook. In the cooking of peas and beans to make them palatable, whether as soups or as separate dishes, considerable ingenuity may be usefully expended, while herring may be cheaply served in a number of appetising ways.

Special attention should be directed to the cooking of the cheaper foods which are rich in proteid in the poorest schools, that is to the pulses, herring, flour, oatmeal, cheese. It is astonishing what variety may be derived from these. Meat, especially beef and mutton, should, however, in all cases be included, as well as milk and eggs, and the cheaper vegetables and fruits. It is the woman's privilege and pleasure to cook well, and if education means drawing out the natural faculty in the most useful directions, then, if this is not education, I don't know what is.

Then a girl should be taught sewing, mending and washing. By these means she will be made to utilise her arithmetic which, in nine cases out of ten, she makes no use of and presently forgets, her life will be filled with varied demands and utilities, she will not find time hang heavy on her hands, and when she comes to be married and have children she will know, to a certain extent, what to do. Probably she will be able to make both ends meet, and perhaps lay something by. Finally, as far as woman is concerned, this is the higher and holier and nobler life, of which one hears in educational questions.

Physical Exercise and Supervision of the Health of Children. Teaching should be modified in the same direction. The new system of physical exercises imposes necessarily on teachers the gravest duties. So long as only mental studies were concerned, to which an ill-fed or ailing child could oppose an impenetrable stupidity, possibly no great harm was done to an ill-fed child himself by the failure of teachers to detect his condition.

Now, I quite agree that physical exercises are an admirable thing for well-fed children. They are liable, on the other hand, to be most deleterious to ill-fed or ailing children. Moreover, any conditions of ill-health which interfere with receptivity interfere with education, while they cause much misery to the individual child. It has, in my opinion, on both grounds, become most essential that teachers should be carefully trained to detect signs of illness or weakness, and to assign them to the true cause as a first approximation, that is to disease or privation. This knowledge should be obtained by instruction

given to teachers by medical experts while examining classes of children, and the action to be taken in individual cases should be explained.

It is, in my opinion, not possible in any other way to overtake the work. The teacher sees the child at study and sees him taking exercise, and has many opportunities of observation which fall to him, or her, alone. But to give precision and confidence in forming a judgment on physical condition requires careful training.

If a child is suffering from insufficient nourishment, it should be fed. Nothing will excuse a child being starved, whether on the ground of humanity or on the ground of efficiency, and as a preventative of vice, crime and disease. Careful enquiry should be made by the School Attendance Officers as to the nourishment which the child receives, and here I wish to enter a note of warning.

Many persons lament the good old days of oatmeal porridge, and it is necessary, therefore, to be careful to ascertain in such foods as porridge and pea soup, how much oatmeal and how much peas falls to the share of the child. But very little oatmeal can be got into a reasonable quantity of palatable porridge, and in the same way we must be careful that pea soup is made sufficiently thick to be nourishing. Oatcake is a different matter, and the cooking of oatcake should be carefully taught. The means by which the ability of the parent to supply the necessary amount of food of suitable quality present features of great difficulty.

IV. SCHEME FOR IMPROVING THE PHYSICAL CONDITION OF SCHOOL CHILDREN.

The scheme which I have in view is something like the following, which is indeed only an extension of that of the Manchester Education Authority, in administering their Free Food Fund:—

The trained teacher is of opinion, let us suppose, after observing a child at his studies and during exercise, that he is not receiving sufficient nourishment. He would thereupon communicate with the Director of Elementary Education, who would instruct the School Attendance Officer to ascertain the income of the family, and the expenditure per week other than on food which is absolutely necessary, also the age, sex, and conditions of work of each member of the family. These particulars are returned to the Director, who refers them back to the head teacher to determine whether the parents can provide a sufficiency of food, and on what scale of diet. For this purpose the teacher might be furnished with a set of model diets, and all necessary particulars guiding him in the determination of a suitable diet for the family and the amount which can be expended. He or some member of his staff must be able to work out the diet required, and special training for this purpose is required. The parent is then to be informed how a sufficient diet can be procured for his family on the money available, and required to see that the child s properly fed. If, after some weeks, it is found that the child is still insufficiently fed the parent is to be compelled to furnish the food necessary or to defray the cost of feeding him. For this purpose powers are to be

conferred to Educational Authorities by Parliament, and they are to be required to carry them into effect. If the powers were carried into effect in one district and not in another the results would not be satisfactory.

Clearly in cases of doubt or difficulty the Medical Officer of the School Board would have to determine the doubtful issues and advise as to proceedings, or where the Education Authority does not possess a Medical Officer, the Medical Officer of Health. Is the School Attendance Officer quite the suitable person for such delicate enquiries, and what are the enquiries which might be supposed to be necessary?

In addition to the enquiries above named, the food alleged to be given to the child would form matter for investigation. Deficiency might arise from ignorance, and therefore I consider that the first step which must be taken is to place within the reach of every family in the community so much knowledge of dietetics as will enable the parents to use their means to the best advantage, in such a shape as that, if they cannot themselves utilise the information given, they may readily get assistance in doing so. The utilisation of the family income to secure, where possible, a sufficiency of nutriment to the growing child is of such far-reaching importance that where a child appears to be suffering from deficiency of food this must override the reluctance of the Educational Authority to interfere further with the affairs of families.

The question then narrows itself to this: Are the School Attendance Officers persons who are suitable for such enquiries? But that is merely a detail of administration which can receive due attention. Especially at the present time advice as to the wise expenditure of money in food is of immense importance to the population, and I am of opinion that the subject should be well discussed by all those gentlemen who have taken a special interest in these questions. In a very large number of instances, at present at all events, it would become clear that the means of the parents, when fully utilised and stretched to the utmost limit of their buying capacity, are inadequate to find the children in food.

We are then brought face to face with the question, "Is the Educational Authority to rely on charity for the funds required, or should these be supplied out of the heavily burdened rates?" The Scottish Commission have answered this question in the sense that charity must be relied upon. On the whole, I am inclined to think otherwise, though I quite realise the difficulties. If the burdens of parents are removed, it may be said, you are making them so much better off, and you are thus giving an impulse to the increased production of children. Is it so? Does the birth-rate increase or decrease with increased well-being?

If we look at the birth-rates in our richer and poorer districts we find that the birth-rate amongst well-to-do people is smaller than amongst the poor. In accord with this is the reduction in the birth-rate as the circumstances of the nation have improved. I will not stay to discuss the reasons for this apparent anomaly, though I think it is quite explicable. Suffice it to say that the birth-rate reaches a maximum amongst moderately well-to-do artisans, and declines therefrom in both directions. We must conclude, then, that this argument is not without some foundation.

On the other hand, you are not lifting the burden to an extent materially

to affect the birth-rate. There still remain the children under school age, and in those families in which there are no such children, in most cases, the older children will have begun to earn wages, and assistance will not be required. The gain in wealth to the community, by passing school children on to the working period in a condition of physical efficiency, would be so enormous as to quite justify, in my opinion, the additional immediate burden on the rates. How is the necessary assistance to be given, by one, two, or more school meals? It is useless entering on a course of feeding of children if it is not to effect the purpose.

Suppose, for example, one meal of porridge with sugar is supplied to a child age seven, what relation does this bear to the 62·5 grammes of proteid, and 1,750 Calories of energy which he requires for full efficiency? Let us suppose that at such a meal he gets 2 oz. of Scotch oatmeal and 2 oz. of sugar.

Take the analysis:—Oatmeal, P. 14·5, fat 10; carbohydrate, 65 per cent; beet sugar carbohydrate, 92 per cent.

First meal. The total proteid in this meal=8·16 grammes; fat=5·62; Calories=287·57. The proteid, then, without deductions=one-eighth of that required for a day's diet, and the total energy=one-sixth of that required.

Second meal. Suppose, at another meal, he gets one pint of pea soup without bone or meat ($\frac{1}{2}$ lb. of peas per quart) and $\frac{1}{2}$ lb. of bread.

Peas: P. 21, C. 55·4, fat 1·8 per cent.

Bread: $\frac{1}{2}$lb.=P. 15, C. 116·2, fat 2, in grammes (not per cent.).

The total proteid=38·6 grammes, the total fat=4 grammes, and the total Calories=987·5.

From this meal he gets 59 per cent. of the total proteid required and over 56 per cent. of the total calories required.

Third meal. Suppose, before leaving school, he gets $\frac{1}{2}$ lb. of bread and 1 oz. margarine, he gets 15 grammes of proteid and 804·6 Calories.

By these means he will have got a practically complete diet, though one poor in fats.

It is evident that, if it is desired to give a sufficient amount of food, an arrangement must be made to supply breakfast, dinner, and a sort of tea, but that one good meal in the middle of the day will go far. It is further evident, on grounds of economy and for educational purposes, that the Educational Authority should cook the meals.

In providing the means of cooking, regard should, of course, be had to the habits of the people, and the cooking appliances should approximate as nearly as possible to those which they are accustomed to and can afford, except that cheap and necessary additions may be made, such as a pan-steamer.

V. SUMMARY AND ENLARGEMENT.

1. It is of supreme importance for the welfare of the nation that the children should be adequately fed, so as to develop, as far as heredity and environment will permit, sound minds in sound bodies.

2. It is the duty of parents to provide adequately for their offspring, and

this duty they should not be relieved of further than is imperatively demanded in the interests of the State.

3. There is a great surplus of unskilled labour, and the wages of unskilled labour are thus depressed to such a point that, in a high percentage of instances, say 5 to 10 per cent., it is impossible for the parents continuously to provide for their children. It has been suggested that a minimum wage of 25s. per week for an unskilled labourer should be provided. The proposal is impracticable, and it is not certain that if it were practicable the result would not be to lead to increased waste.

4. If, however, children during school life were adequately fed so that they could profitably receive instruction, and if that instruction were such as to educate them, as far as practicable, so as to make them fit for skilled work, wages would be more equalised, and a higher standard of living would result.

5. There are at present various sources of enormous waste going on, of which two stand out very clearly:—(*a*) Drink. If the amount of alcohol consumed in this country could be diverted to the feeding of children it would maintain 20,000,000 in food, and if reckoned only as beer and spirits it would maintain 11,000,000, at a charge of 6d. per day, which would be an ample allowance. It may be said broadly, therefore, that the amount of alcohol consumed is the chief cause of deficiency of food for the young. The remedy is bound to be slow, and a long process of education is required. (*b*) But a great waste also occurs from want of knowledge on the part of mothers of how economically to select, buy and prepare food. This knowledge should be imparted in the schools. In association with this is the waste due to want of knowledge of the elementary principles of health in relation to breathing, clothing, exercise, cleanliness, and so forth.

6. The Education Authority possesses machinery which, when improved and extended, will enable them to detect illness and innutrition amongst children, and to apply remedies.

7. Teachers should be trained to look after the physical conditions as well as after the mental instruction of their scholars, and that training should be practical.

8. Legislation is required enabling the Educational Authority to enforce the provision of sufficient food and the maintenance of cleanliness in school children by parents.

9. The Educational Authority should also be empowered to obtain from the rates sufficient provision for the free feeding of children under certain definite conditions. In all schools kitchens should be provided for instruction in the elements of cooking.

10. In all schools children should be weighed and their height measured once a term, and a record should be kept of the age, height and weight. By this means it would be possible to gauge the condition of the children, both collectively and individually. These observations might usefully be made more frequently in cases where a deficiency of nutriment or disease was suspected.

11. A prolonged and practical course of domestic economy and hygiene should be compulsory for girls. It should include cooking, construction and criticism of diets, management and feeding of infants, methods of cleanliness, sewing, mending and washing. I am fully aware of the excellent work which has been done by the Education Authority in their Evening Continuation Classes, and which is already bearing fruit. But, in my opinion, such instruction should be a necessary part of education.

12. The poorer population generally should be instructed as to the diet requisite and the means by which it may be most cheaply obtained.

Works consulted:

"Report of the Royal Commission on Physical Training" (Scotland).
Hutchinson on "Dietetics."
"Poverty: "A Study of Town Life." Rowntree.
"Health in the Nursery." Ashby.
"School Diet." Dukes.

The Role of "Missed" Cases in the Spread of Infectious Diseases.

BY

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The Role of "Missed" Cases in the Spread of Infectious Diseases.

Introduction.

The subject of my address is one with which all Medical Officers of Health are familiar. We can all adduce numerous instances in which preventive measures against the spread of infection have been rendered partially futile owing to the failure on someone's part to recognize disease. I should not, therefore, have ventured to address you on this subject, did I not increasingly perceive that the importance of this cause of partial or complete failure in preventive measures is being constantly overlooked or under-estimated; and that neglect to look for slight cases of infectious disease and for microbe-bearers who may appear to be in good health, is responsible for a very large share of our failures to preventing the spread of disease.

Preliminary Remarks. I shall deal more particularly with two such common diseases as Scarlet Fever and Diphtheria, in which this lesson is pre-eminently important, though I may, if time permits, give similar instances from experience in Small-pox and Enteric Fever. I do not propose to discuss the differential diagnosis of each of the diseases dealt with, nor to attempt to give special hints as to diagnosis. We have doubtless all learnt in this regard that the only safe rule is to use every available aid to diagnosis, whether clinical, bacteriological or other, and to attach as great importance to the history of the mode of onset and of possible exposure to infection as to the actual symptoms when the patient is examined.

The possibilities of missed or unsuspected cases may be better understood by considering them in relation to the various stages of an infectious disease after the reception of infection.

Latent Period as Distinguished from Incubation Period.

We are accustomed to speak of 1st an incubation period, and 2nd a period of actual symptoms, the latter alone being regarded as infectious. This, although usually true, is not always so, and our conceptions on this subject, particularly in regard to scarlet fever and diphtheria, need modification. Some years ago Mr. F. Vacher read a paper* in which considerations were advanced in favour of the view that an infectious disease may be communicable in the incubation period. The apparent discrepancy between this opinion and the ordinary opinion which, in the majority of instances, accords better with experience, can, I think, be reconciled if we divide the period between the reception of infection and the development of symptoms into two:—

(1) A *latent period* or period of "latent microbism"; and
(2) A period of *true incubation*, during which the vital changes resulting in the first symptoms of the particular disease are progressing.

* "The Duration of the Latent Period . . . . of the Exanthemata," by F. Vacher, F.R.C.S. (North-Western Association, Medical Officers of Health, June, 1884).

Instances which, I believe, illustrate the above distinction are given in Appendix A; and here the general remark may be made that illustrative cases are throughout given in Appendices, in order that the general argument of my paper may not be lost in detail.

It may be objected that these were instances (*a*) of protracted true incubation, or (*b*) of infection from the same source as supplied the first case in each household, or (c) that there had been some failure in disinfection. There was every reason for deciding against the last supposition; the second supposition was eliminated so far as possible in each instance; and, in regard to (*a*), it appears to be more reasonable to suppose that in each instance the patient was a mechanical "carrier" of infection on or in his mucous membranes for some days or even weeks, rather than that the true incubation period varies so enormously as these cases would appear to suggest. The possibilities of fresh external infection or of failure of disinfection can seldom be eliminated with absolute certainty; but in my opinion we have to deal in most of these instances either with instances of protracted true incubation of infection, or a period in which the prospective patient is a mere mechanical "*carrier*" *of infection* on or in his mucous membranes for some days or even weeks before true incubation begins, this being, in my opinion, the more probable supposition. If this be so, we may suppose that stray diphtheria bacilli or scarlatinal germs are present in the mucous membrane of the naso-pharynx, these "taking root," so to speak, under the influence of a slight catarrh or other influence lowering vital resistance.

In some instances the latently microbic person never becomes ill, so far as can be ascertained, and yet he may cause illness in others. This is shown for diphtheria in the illustrations given in Appendix B.

There is no reason to suppose that the same is not true for scarlet fever, though the possibility of infection by mucus containing stray scarlatinal germs cannot at present be certified. It is probable that in certain instances the period of latency may overlap into the period of incubation, which would explain the cases in which infection has occasionally appeared to be produced during the incubative period of disease.

Infection in Period of Invasion. The amount of spread of infection under the above conditions is relatively small when compared with that occurring during the *period of invasion* of infectious diseases. The number of cases which at this stage are temporarily "missed" is very large, and most infectious diseases are specially infectious at this stage. The symptoms at this stage are commonly not characteristic; the disrepute to the family practitioner from a mistake, and the desirability of caution are to him so obvious, that we cannot be surprised, however much we may regret it, when diagnosis and notification are postponed until the case has been watched. Were it a universal law of medical ethics that precautionary measures of isolation should be urged on the family while the symptoms are maturing, no great objection could be taken to this course; but unhappily this is not so in most instances.

Although it is widely believed, both by doctors and the public, that infectious diseases are only infectious to a slight extent at this stage, there can

be little doubt that both scarlet fever and diphtheria are, from the onset, intensely infectious, more so than at later stages of the disease. Of 38 cases of scarlet fever investigated by me, in which secondary cases subsequently occurred in the same family, 2 were at the end of one day, 6 at the end of two days, 3 at the end of four days, 6 after five to seven days, 4 in the second week, 8 in the third week, and 9 later. Thus, in this experience 45 per cent. of all the secondary cases occurred within a week of the first case; and it is clear, that if this be regarded as a fair sample of the experience of others, nearly half the secondary cases of scarlet fever have been produced before effective isolation, whether at home or in hospital has been seriously attempted.

After the period of invasion we arrive at the stage at which the ordinary symptoms, such as rash in scarlet fever and membranous exudate in diphtheria usually appear. In not a few instances, however, the classical symptoms do not appear or are overlooked. Ordinary overlooked cases I do not propose specially to discuss. They are instances of careless diagnosis. I am chiefly concerned with cases in which characteristic symptoms do not appear, as in scarlet fever or smallpox without rash and diphtheria without membranous exudate. The consideration of such cases is of the essence of my subject.

Professor Theobald Smith has suggested a formula which enables us to consider this question with an approach to exactitude. Thus if D = disease, M = the micro-organism, which causes it, and R = the vital resistance of the person attacked

$$\text{Then } D = \frac{M}{R}$$

Dr. W. T. Sedgwick has extended the formula as follows:—

If N = number of micro-organisms, and V = their virulence

$$\text{Then } D = \frac{M \cdot N \cdot V}{R}$$

Missed Cases in Tuberculosis. Sir Dyce Duckworth, in an address to the Liverpool Medical Institution, in 1901, on "The Personal Factor in Tuberculosis," has reminded us that in considering the two pathogenic factors of host and infecting parasite, the younger generation of pathologists are apt "to reckon without their host." This is a useful illustration and enables me to take tuberculosis as an example of the importance of "missed" cases in spreading disease. A large proportion of the population are doubtless infected by tuberculosis. Of those infected, many are able at once to destroy the invading tubercle bacilli. R is very potent, or being less potent, N is limited, or V is attenuated. Many others harbour the bacilli, become the victims of a small patch of disease, which, when the patient subsequently dies of another disease, is discovered in the mortuary. Here R has operated though tardily, helped or not helped by attenuated V or fewness of N. We need not discuss the fully-developed disease; but the second series of cases are of the utmost importance from the view-point of preventive medicine. Before the

disease becomes quiescent, the potentiality for spreading infection of the patients suffering from slight tuberculosis has been very considerable, and no scheme for preventing the spread of tuberculosis can be considered to approach completeness which does not lay down as a medical axiom that every patient with a troublesome cough should, even in the absence of physical signs of phthisis, have his sputum examined and rigid precautions taken in regard to it.

Scarlet Fever Without Rash. Returning to the acute infections, I do not propose to attempt an account of cases of scarlet fever in which infection has been spread by atypical cases; but in the *Journal of Hygiene*,* I have described a scarlatinal outbreak, the majority of patients in this outbreak not showing a rash, but only a severe sore throat. We have then to bear in mind that while we are isolating patients with fully developed attacks of scarlet fever in hospital, other members of the same family occasionally are keeping alive the infection in what may appear to be an innocent catarrhal condition of the mucous membrane of the naso-pharynx.

Small-pox Without Rash. The case given in Appendix C illustrates the possibility of spread of infection by abortive attacks of small-pox. It further shows that the very success attending vaccination may, unless the utmost vigilance be exercised, lead to an occasional though rare failure of preventive measures. We are familiar with this risk in *Varioloid cum eruptione*. It must also be noted for *Varioloid sine eruptione*. The three cases given in Appendix C, whose temperature charts are shown, would probably have been overlooked had not the entire family of which they were members been taken into a quarantine ward for observation, and might then have caused further cases of small-pox.

Missed Cases of Diphtheria. Diphtheria is, however, the disease in which we have the most exact data as to "missed" cases, and the part they play in spreading infection. It is quite clear that in this disease, as in tuberculosis, the soil is of equal importance with the seed, and that only a relatively small proportion of those receiving infection actually develope disease to a recognizable extent, some not at all. Whether variations in N or in V or in R, or various combinations of these three factors are the cause of the failure of the contagion to take root or to grow sufficiently to produce recognizable results, it appears to me that these unrecognized and often unrecognizable cases are a frequent means of spreading infection.

Diphtheria Without Exudate. Diphtheria without exudate or with overlooked exudate is an extremely common means of spreading diphtheria, and I regard the instances given in Appendix D as illustrations of the necessity of the axiom which I would venture to formulate that *every sore throat should be regarded as possible diphtheria until it is proved not to be so*. This appears

* Vol. II., No. 2, p. 150.

fairly obvious, but is so frequently disregarded, that it is necessary to state instances in some detail. In Appendix D (*a*) are summarised first a number of cases missed by parents, no blame in most instances attaching to them, as the symptoms were very slight. In some of these doubtless membranous exudate was overlooked, in others this may have been absent. In Appendix D (*b*) are given instances of cases missed by the doctor, and in Appendix D (*c*) I give an instance of a case overlooked in my own department, although carefully investigated at the time, except that unfortunately no swab was taken from G.Y., a doctor being at the time in attendance, and the symptoms, enlarged post-cervical glands, etc., being regarded as sufficiently explained by the eczema capitis.

In nearly all the instances given in Appendix D the missed cases had symptoms of some degree, possibly slight and transient. It is probable that such cases are a much more frequent means of spreading infection than mere "carrier" cases. In these slight unrecognized cases the possibility of diagnosis depends on a careful investigation of the history of each case, the association of the case with more typical cases in the same household or school, the repeated use of bacteriological aids to diagnosis, and the presumption on the side of diphtheria which I have ventured to suggest should always be entertained.

"Carrier" Cases. There remain, however, certain cases in which so far as can be ascertained no symptoms whatever have occurred. In some of the cases under this head given in Appendix E, cases with slight symptoms are mixed up with cases which apparently had no symptoms ("carrier" cases).

The instances given in Appendix D and E sufficiently emphasize the fact that the M.O.H. who awaits notification certificates and secures isolation of notified cases and subsequent disinfection has not exhausted the possibilities of preventing the spread of infection. Even when more minute investigation is made, and "missed" cases are looked for, our network of preventive measures is still too coarse to prevent the overlooking of a certain proportion of cases; and it is only by repeated visits to infected houses, by complete and active co-operation between the family practitioner and the Medical Officer of Health, and between these and the head of the household, that we can hope to bring our preventive measures nearer to perfection.

Limits of Utility of Bacteriology. I do not propose to give instances on the same lines relating to scarlet fever and enteric fever, but such instances will occur to many of you. In diphtheria we are fortunately aided by bacteriological methods of diagnosis. It is necessary to note, however, that this means of diagnosis may occasionally, when misused, become an aid to the spread of infection. The following underscored statement is attached to every certificate sent out from my laboratory giving the result of an examination of a "swab."

"*In all instances in which the Diphtheria Bacillus is not found, its absence does not imply that the case is not Diphtheria. This must be decided on clinical*

grounds, or a further specimen sent. Three consecutive failures to find the Diphtheria Bacillus are required before any importance can be attached to negative results."

And yet occasionally one meets with instances in which the doctor is stated to have informed the patient that "the test shows there is no diphtheria," and the patient, on the strength of this statement, is released from isolation.

If the bacteriological test is employed to relieve the doctor from the onus of diagnosis in fairly characteristic clinical cases of diphtheria, to that extent it is harmful, because it leads to twenty-four hours' delay in treatment by antitoxic serum, and may, if a negative result be obtained, mean a dangerous withholding of specific treatment. The true scope of bacteriological diagnosis is for atypical cases which would otherwise be missed; and we cannot afford to displace clinical diagnosis from its first and most important position.

When a case of an infectious disease is recognised, attempt is made at isolation, either at home or in hospital, until the period of infection has passed. The old idea that this period can be strictly delimited is in the light of modern bacteriology necessarily doomed. There can be no arbitrary line between the infectious and post-infectious period. The magical period of six weeks for scarlet fever has no exact correspondence with facts: it may be three or even thirty weeks in exceptional cases. Each case must be decided on its merits, and we are hampered in scarlet fever by the lack of an exact bacteriological diagnosis. Even in diphtheria, in which this aid is available and most valuable, we have to remember the limitations of the bacteriological test. We cannot too often endeavour to impress on medical practitioners the importance of the following rule: *If diphtheria bacilli are found in a case with any catarrhal abnormal condition of the throat or nose the evidence of diphtheria must be regarded as indubitable; but if diphtheria bacilli are not found, the onus of diagnosis still rests on the practitioner; and the negative result, unless several times repeated, must not enter into the judgment.*

I have already alluded to the erroneous deductions frequently drawn from negative swabs. Of 342 consecutive cases admitted to our isolation hospital, 26 were bacteriological cases in which no evidence of disease, apart from the laboratory test, was present except in some instances slight catarrhal symptoms; 51 cases were apparently not diphtheria, and 10 cases were clinically diphtheria although they were repeatedly examined without success for diphtheria bacilli (each case at least five times).

Recurrence of Infection in Diphtheria. The occasional recurrence of infection in cases of diphtheria is a still more important illustration of the limitations of negative bacteriological examinations. The most remarkable instance of this with which I am acquainted is the following. There are clearly possibilities of other overlooked sources of infection, but I am myself inclined to take the view that the chain of events shown in the following chart, and given more fully in Appendix F, corresponds to the facts. If so it illustrates very forcibly the limitations of negative bacteriological results.

B. G., æt. 4, of 27, D. Street.
Onset Diphtheria Aug. 19.
Hospital Aug. 25, slight throat exudate on admission.
Four negative swabs.
Sent home Sept. 7.
Resumed school attendance (Babies' Class) Sept. 24.

W. M., æt. 3, of 6, T. Street.
Onset Diphtheria Sept. 26.
Last attendance (Babies' Class) Sept. 25.
Diphtheria bacilli present.

H. W., æt. 5, G. Street.
Onset Diphtheria Oct. 3.
Last attendance (4th Class) Oct. 1.
Diphtheria bacilli present.

B. G., *negative swab* Oct. 7.
Returned to school Jan. 25 until Feb. 5.
Negative swab Feb. 12.

O.S., æt. $2\ ^{11}/_{12}$, of 18, R. Street.
"Cold" Feb. 9.
Seen by a doctor Feb. 10.
Away from school (Babies' Class) Feb. 9—22, except 2 half-days, Feb. 16 and 17.
Examined Feb. 23, large cervical glands and *Diphtheria bacilli present.*

F. D., æt. 3, of 18, S. Street.
Onset Diphtheria Feb. 2.
Last at school (Babies' Class) Feb. 3.
Diphtheria bacilli present.

B. N., æt. 4, of 34, M. Street.
Onset Diphtheria Feb. 6.
Last attendance school (Babies' Class) Feb. 5.
Diphtheria bacilli present.

F. S., æt. 6, of 18, R. Street.
Onset Diphtheria Feb. 18.
Hospital Feb. 22.
Diphtheria bacilli present.

B. S., æt. 10, of 18, R. Street.
Onset Diphtheria Feb. 21.
Hospital Feb. 22.
Diphtheria bacilli present.

G. S., æt. 1.
Onset Diphtheria about Feb. 20.
Diphtheria bacilli present.

G. B., æt. $5\frac{1}{2}$.
"Bronchitis" Dec. 10.
"Cold on chest" Jan. 26.
Nasal discharge Feb 7.
Hospital Feb. 10.
Numerous *consistent negative swabs* after admission.
Nasal voice a fortnight after admission.

This case illustrates also a very important variety of missed cases—those which have been released from isolation. It may appear to be stretching the scope of a paper under the present title to introduce the subject of "return cases," but the significance of these must be faced, if every possibility of missed infection is to be investigated. If the above sequence of events is correctly indicated, a diphtheria patient may firstly, three weeks after release from isolation, and secondly, 146 days after release from isolation, cause small outbreaks of the same disease. Of course there may have been other overlooked sources of infection, though these were carefully and unsuccessfully sought for. The association of B.G. with the events following her return to school is confirmed by the fact that on each occasion the outbreak ceased when she was again excluded. A second instance of the same kind is given in Appendix F, and in this instance the recurrence of infection was proved by the detection of diphtheria bacilli.

"Return" Infection not a Hospital Phenomenon in Diphtheria. In diphtheria such return or persistence of infection is not a hospital phenomenon, whatever it may be in scarlet fever. I have in my notebooks numerous instances of re-infection of others after release of diphtheria patients from home isolation; and the fact is so well known that it need not be laboured. If not a hospital phenomenon in diphtheria, why is it to be regarded as such in scarlet fever? During 1903, four "return cases" of scarlet fever have occurred in my local experience, two in cases in the best circumstances treated at home, and two in hospital experience. There is, in my judgment, no justification in fact for the opinion so frequently expressed that increased and protracted infectivity is a consequence of aggregation of cases in hospital. Such a theory is analogous to that which assumes that when more than say twenty cases of acute small-pox are aggregated in a small-pox hospital aerial convection of infection is rendered increasingly possible. It is a theory to support a theory, and so in the main is the theory of hospital origin of "return cases." My own views are expressed in the following extract from my annual report for 1902, and I contend that these views are more consistent with the known facts as to the natural history of these two diseases than what we may describe as the hospitalism theory.

"It is probable that, although in a certain proportion of these "return cases" the patient may serve merely as a carrier in his secretions of infection from the ward, in the majority of instances when "return" cases occur he is at the time of discharge, and for some time afterwards, an active incubator of scarlet fever organisms in his throat and nose. On this point I would lay stress on the analogy between diphtheria and scarlet fever. In diphtheria it is a well-known fact, irrespective of whether the patients are treated in hospital or at home, that a certain number remain infectious for protracted periods, and in these cases there is usually rhinorrhœa. In such chronic cases the infection may persist for three or more months. What occurs in diphtheria almost certainly occurs in scarlet fever in a small proportion of the total cases; and in scarlet fever, as in diphtheria, it is not, in my opinion, as a rule a question of hospital influence, but of the natural history of the disease.

"These chronic cases of scarlet fever and diphtheria represent sports of the disease. The great difficulty in scarlet fever is that, unlike diphtheria, one has at present no trustworthy bacteriological test when the patient is discharged; hence the greater difficulty in controlling "return" cases in scarlet fever than in diphtheria."

It may be added that such prolonged infectivity in a certain proportion of cases of scarlet fever and diphtheria is consistent with what we know of the occasional prolonged continuance of infection in enteric fever. Several outbreaks of enteric fever in this country have been traced to enteric patients who were convalescent before leaving South Africa for home. We know again that typhoid bacilli may persist for many months in the gall bladder, etc *

Relapses and "Missed" Cases. I can only briefly allude to relapses as an occasional "missed" cause of spread of infection. Such definite relapses not infrequently occur near the end of the period of isolation in these diseases, and they are known in home as well as in hospital treatment. I described in the *British Medical Journal* for Oct. 10th, 1896, a case of relapse of small-pox occurring after a patient had left a metropolitan small-pox hospital, where he had suffered from an undoubted attack of small-pox. The relapse was mistaken for chicken-pox and gave rise to a small outbreak of small-pox. I have only been able to find three or four similar cases in medical literature.

Chronic Infection of Ear Discharges. Before concluding my imperfect review of "missed" cases of infection, a number of instances in which the only bacteriological evidence of diphtheria was in ear discharges from which the patients were suffering may be mentioned. In some instances such an ear discharge had been the only obvious evidence of disease. See Appendix G.

The above cases illustrate the important fact that we have to deal not only with cases never recognised which ought to have been recognized, with other cases which could only have been recognized by calling in the aid not only of bacteriology but of complete information as to the history and environment of the patient, but also with cases which may have been under our own care, whether isolated at home or in hospital, which continue to be infections or resume infection after being released from isolation.

We come to consider in the last place what remedies are practicable for the evils of which I have given numerous examples. By all means let us reform our isolation hospital administration and make it as perfect as possible. This hospital isolation is, in my opinion, a most potent means of preventing the spread of infection; the occasional failures to prevent the recurrence of infection after cessation of isolation, and the still more frequent failures which have nothing to do with hospital isolation, form a deeper and more extensive evil. In part they are inherent in the extremely variable manifestations of each infectious disease. In part they might be obviated by improved measures, which I can, in conclusion, only briefly indicate.

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*This subject is treated more fully in a paper read by the writer before the Royal Medico-Chirurgical Society, June 14th, 1904, and published in the Transactions of the Society.

Difficulties of Diagnosis. Our difficulties come under three heads. These are first the difficulties of *diagnosis*. How are we to aid and accelerate diagnosis? How are we to persuade parents of the necessity of having a doctor for what may appear to be a trifling ailment? I have elsewhere advocated a system of free medical diagnosis,* and no complete remedy appears practicable apart from a much more complete use of medical men in relation to public health.

Difficulties of Notification. There are secondly the difficulties of *notification*. Notification by the householder is a dead letter, and must remain so, until free medical diagnosis is given and the last excuse for neglect is thus removed. Notification by the doctor is commonly belated, and will remain so, until or unless the notification of doubtful cases is rendered obligatory. There is precedent for the adoption of this line of action. In fact a step in this direction has been already taken by the legislature. Under the Infectious Disease Notification Act the practitioner is required to notify a case "*as soon as he becomes aware.*" Under the Factory and Workshops Act, 1901, he must notify patients "*whom he believes*" to be suffering from lead, arsenic, or mercury poisoning or anthrax. Farmers are required to notify each *suspected case* of glanders or foot and mouth disease. In Sydney and the whole of New South Wales the practitioner must notify every case of small-pox and every disease which may reasonably be suspected of being small-pox. In the New Zealand Public Health Act of 1900 the same obligation is extended to other infectious diseases, the practitioner being under an obligation to notify the case of "any person found to be sick of any infectious disease, or of any sickness the symptoms of which raise a reasonable suspicion that it may be an infectious disease."

But if such notification of doubtful cases is to be enforced, the responsibility of the final diagnosis must be partially placed on the shoulders of the Medical Officer of Health; and this implies a large addition to the medical portion of the public health service. That such an addition is required has long been my opinion; and I should welcome the day when candidates were only permitted to take the D.P.H. after having worked day by day for a year under a Medical Officer of Health. To give every possible help to the medical practitioner in the diagnosis of infectious diseases is the natural corollary of the fact that we have minimised his opportunities of obtaining experience in regard to them.

Difficulties of Supervision. There are thirdly, difficulties of *supervision and examination*. A complete system of preventive measures implies a complete list of contacts, and the power, where necessary, of examining these.

Suggested Remedies. The remedies are:—

1. The universal adoption of bacteriological aids to diagnosis, negative results being discounted.

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* *British Medical Journal*, Portsmouth Meeting.

2. The experimental adoption of a system of free medical diagnosis in cases of sore throat.

3. The tentative notification of all cases suspected to be infectious, a fee of 1s. being given for such notification, a further fee being given if the diagnosis is confirmed.

4. The appointment of Assistant Medical Officers of Health in large districts to visit doubtful cases.

5. Statutory power to be given to examine contacts.

6. A strict supervision to be kept on all past patients in the post-isolation period, whether this has been at home or in hospital.

This list is obviously incomplete, but a second lecture would be necessary for anything more than a passing indication of remedies.

My sketch of this difficult subject would be incomplete and lacking in perspective, were I to give the impression that preventive measures hitherto employed have been a failure. Where relative incidence of disease can be tested this is far from being the case. Scarlet fever is, in recent epidemics, not only less fatal but also less prevalent than formerly.* The same is almost certainly true of diphtheria. Enteric fever and tuberculosis notwithstanding failures in diagnosis are both rapidly declining. Small-pox, in which the further aid of vaccination is available, can be rigidly controlled when desired. The danger of dealing solely with one aspect of the case, as in the present paper, is that the importance of the one missing measure is magnified, and the ninety-nine instances in which preventive measures have not gone astray are apt to be overlooked. How to deal with the exceptional "missed" cases, is, however, a most pressing problem in preventive medicine, and we cannot hope for more than relative success until and unless special efforts are made in this direction.

APPENDIX A.

CASES OF PROBABLE LATENT MICROBISM.

1. Scarlet Fever.

First instance.—G.R., æt. 5, failed with scarlet fever on December 6th, removed to hospital 8th. F.R., æt. 7, failed with scarlet fever December 25th, removed December 26th. K.R., æt. 4, failed January 7th. There were no other children in this house. Father and mother had no sore throat or other symptoms. Thus secondary cases occurred at intervals of 18 and 14 days respectively. No outside infection after the first case. Disinfection very thorough.

Second instance.—B.C., æt. 5, failed with scarlet fever on September 24th, admitted to hospital September 29th. H.C., æt. 4, failed with scarlet fever October 13th. Is stated to have "had a cold" all the time since the removal of B.C. When seen by me on September 30th had a "stuffy nose." A brother,

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* "The Epidemiology of Scarlet Fever, etc." By A. Newsholme. *Trans. Epidem. Society*, Vol. xx., p. 59.

æt 8, remained well; father and mother same. Interval of 15 days between the two cases. Disinfection thorough.

Third instance.—O.D., æt. 9, failed with scarlet fever September 21st, admitted to hospital September 23th. B.D., æt. 6, failed with scarlet fever October 6th, thirteen days interval. In this and the above cases, no evidence of further outside infection. B.D. stated to be "poorly" when O.D. was removed.

2. Diphtheria.

Fourth instance.—W.K., æt. 6, failed with diphtheria March 13th, admitted to hospital March 16th. Was still there when a brother, R.K., æt. 3, failed with diphtheria on April 14th. No history of intervening cases in the household consisting of mother and father, a girl æt. 11, and the above patient. No outside fresh infection discovered.

APPENDIX B.

INSTANCES OF TRUE "CARRIER" CASES.

First instance.—During the course of an outbreak of diphtheria in a particular school, M.W., a girl aged 11, was attending Standard III. She sat next M.M., who attended school for several days while suffering from unrecognised diphtheria up to and including the 20th October. On 23rd October a young man lodger at M.W.'s home failed with diphtheria. He only arrived in Brighton on the 16th October, and had lodged since his arrival in this house with five other young men. He only had one meal in the house, viz., breakfast brought up to his bedroom. This was brought up by M.W. and by her older sister. None of the other lodgers had diphtheria, and of the large family to which M.W. belonged (father and mother and eight children varying in age from 2 months to 12 years) all remained apparently well.

For further illustrations of "carrier" cases, see under Appendix E.

APPENDIX C.

Family Outbreak of Small Pox, showing Cases without Eruption.

(b) Patient introducing the infection into the house.	(c) Members of same household infected by the First Patient.	(d) Members of other households infected by Patients in Columns b and c.	(e) Non-infected members of same household.	Vaccinated. (f) (1) Before being exposed to infection.	Vaccinated. (f) (2) Within 3 days after being exposed to infection.	Vaccinated. (f) (3) More than 3 days after being exposed to infection.	Re-vaccinated. (g) (1) Before being exposed to infection.	Re-vaccinated. (g) (2) Within 3 days after being exposed to infection.	Re-vaccinated. (g) (3) More than 3 days after being exposed to infection.	(h) Date of appearance of Small Pox Rash.	(i) Nature of Attack.	(j) Result.	
Man aged 36	...	...	...	In infancy	...	...	Never	Never	Never	May 7	Discrete (numerous pustules)	,,	Isolated from evening of May 9.
...	Wife	...	...	,,	...	...	...	...	May 9	May 19	Slight (20 to 30 pustules)	,,	Isolated from onset.
...	Girl aged 15	...	...	,,	...	...	...	...	May 9	May 19	Very slight (a dozen pimples)	,,	,, ,,
...	Baby aged 3 weeks	...	...	...	...	May 9	...	...	...	May 19	Severe, discrete	,,	,, ,,
			Boy aged 17	In infancy	...	...	...	...	May 10	None ever appeared, but on May 17, each of these boys had headache and high temperature, and in two days were quite well again.			
			Boy aged 9	,,	...	...	...	...	May 9				
			Boy aged 4	,,	...	...	...	...	May 17				
			Boy aged 14	,,	...	...	Feb., 1902	...	...	Remaining well.			
			Girl aged 5	,,	...	...	...	...	May 9				

APPENDIX D.

MISSED CASES.

Diphtheria.—(*a*) *Cases missed by parents.*

First instance.—The cook at 9 T. Road had a "very bad throat" three weeks before Christmas, 1901. Was very ill for a fortnight, but no doctor was called in. Nothing further happened until Easter, when, in the same house, M.D., æt. 14, had a bad cold and sore throat with enlarged glands. No doctor. Diphtheria bacilli were found in her throat on June 12th, by which time she or other members of the same family had infected a number of children at a Kindergarten School. The following scheme gives the order of events. The first six are members of this particular household; all the other cases attended the above school.

		Onset.	Recognised as	
Cook	9 T. Road	Christmas	"Sore Throat"	
M. D., æt. 14 ...	do.	Easter ...	do.	Evidence of diph. bacilli persisting 73 days later. Returned to Kindergarten School May 16
Nursemaid ...	do.	May 22...	do.	
E. D., æt. 10...	do.	May 25...	do.	Away from School May 26 to June 2. Returned June 3
N. D, æt. 5 ...	do.	May 26...	"Cold"	
L. D., æt. 7 ...	do.	June 6 ...	"Diphtheria"	
N. Co., æt. 14...	39 F. Road	June 5 ...	do.	
M. H., æt. 9 ...	13 R. Road	June 6 ...	do.	
E. W., æt. 8 ...	71 T. Street	June 7 ...	do.	
T. T. æt. 8 ...	S. Road ...	June 10...	do.	

There were in the kindergarten class of the above school 21 children, of whom five, including one member of the infecting family, had diphtheria more or less lightly. Only two cases, including a member of the infecting family, occurred in the higher classes. In one member of the infecting family the presence of diphtheria bacilli, after a sore throat 73 days previously, was demonstrated.

Second instance.—N.W., æt. 3, failed with diphtheria October 20th. and was notified October 25th. J.W., æt. 2, failed October 25th. and was notified October 27th. Inquiry into these cases led to the discovery that the brother G.W., æt. 5, had come home from school with enlarged glands on October 7th, but no doctor was called in. Diphtheria bacilli were found in his throat on October 25th.

Third instance.—F.T., æt. 4, failed with diphtheria November 16th, hospital November 28th. I.T., æt. 7, on November 8th had headache followed by nasal discharge. He continued to attend school until the 18th, and infected three other children in the same class, who failed two on November 14th and one on the 19th. Nasal swab from I.T. on November 19th showed diphtheria bacilli.

Third instance.—D.B., æt. 3, failed with diphtheria August 21st, admitted to hospital August 24th. W.B., æt. 16, failed on August 26th, admitted to hospital August 26th. B.B., a boy æt. 14, complained of "headache and cold" about August 1st. Diphtheria bacilli found in swab from his throat August 25th. An infant aged 5 months, a boy aged 9, and a girl aged 7 remained well.

Fourth instance.—D.R., æt. 8, was away from school January 6th with sore throat and headache. He was only absent from school on one day. On the 11th his sister, C.R., æt. 7, failed with diphtheria and was notified on the 14th. This led to a swab being taken from D.R.'s throat, in which diphtheria bacilli were found.

Fifth instance.—R.G., æt. $8^{11}/_{12}$years, failed with diphtheria June 16th, admitted to hospital 19th. His sister, F.G., æt. 4, attended school until June 12th, and next day "began with a cold." A swab from her throat on June 20th and a second July 16th showed diphtheria bacilli. In this, as in other cases, there is the fallacy that she might have acquired diphtheria bacilli from her brother; but she was ailing before he was.

Sixth instance.—G.J., æt. 5, failed with diphtheria March 30th, had not been to school since the 18th March, admitted to hospital 1st April. He was the only child in this family, but in the same house lived another family having two children. Of these two, B.C., æt. 7, attended a class of an infant school which at that time was infected with diphtheria; last attendance on April 3rd, when the school was closed. Examined by me on 7th April, she had some eczema naris, also conjunctivitis, but there was no history of sore throat. Negative swabs were obtained from both nose and throat. Her brother C.C., æt. 5, examined same day, had enlarged cervical glands. No history of sore throat. Diphtheria bacilli in throat. He had not attended school since March 18th. The probable sequence of events in this family was that B.C. brought infection in an unrecognised form from school and gave it to G.J. and C.C.

(*b*) *Cases missed by practitioner.*

First instance.—F.B.W., æt. $2\frac{1}{2}$, failed with diphtheria June 10th, notified by Dr. X and removed to hospital June 15th. No school attendance. The brother, B.W., æt. 5, had a sore throat for which he was taken to Dr. X on 6th June, having been ailing since 2nd June. A swab was taken from his throat on 17th June, no growth; swab on 23rd June, diphtheria bacilli present. Was this due to engrafting of diphtheria bacilli on a previously innocent sore throat? Almost certainly not. No source of infection to F.B.W. could be discovered except B.W.

Second instance.—F.R., æt. $1^{10}/_{12}$year, had a "cold" on February 14th, followed by nasal discharge. She was seen by Dr. Y on 16th February. On the 12th March the child seemed worse, had difficulty in swallowing and regurgitation of food. Dr. Y. resumed attendance on the 17th, but did not notify the case until March 25th. Three days later he notified the case of G.R., a brother, æt. 2, who failed on the 27th.

Third instance.—F.T., æt. 5, attended an infected infants' school, last attendance February 9th. On this day he was hoarse; his mother thought he was choking and called in Dr. Z. Next day two doctors saw this child together. The mother states that he then had enlarged cervical glands and that there were white patches on the throat at the onset. The nature of his attack was not recognized by the medical attendant until two other children were infected. He was then admitted to hospital (February 20th) suffering from laryngeal and nasal diphtheria, for which tracheotomy was required. The attack proved fatal on 27th February. On the 12th February, N.T., æt. 3, failed with diphtheria and was admitted to hospital on 17th February, and on the 18th a baby, aged 10 months failed with diphtheria and was admitted on the 20th.

(*c*) *Case missed by M.O.H.*

On December 6th a letter was received from another M.O.H. stating that on December 4th G.Y., æt. 20, of J., had been notified with diphtheria, and that on November 28th and 30th he slept at No. 174 X. Street, where his sweetheart's parents lived. His case was of a very severe type. Enquiries were at once made at this house by my assistant. It was found that there were living in this house Mr. and Mrs. G., their five children, and two unmarried sisters of Mrs. G. All these had been well, except O.G., æt. $2\frac{1}{2}$, who was being attended by the Dispensary Doctor for eczema capitis and enlarged post-cervical glands. No swab was then taken, but one on the 16th December gave a negative result. This boy will be further considered after the history of other members of the family has been given. On or about December 11th F.R., æt. 20, the sister of G.Y.'s sweetheart, failed with diphtheria, and was removed to hospital on December 14th. Then G.R., æt. 22 (G.Y.'s sweetheart) was swabbed and diphtheria bacilli found in her nasal secretion. She had two minute dots (? pent-up follicles) on her throat. Removed to hospital on December 16th. It transpired that while G.Y. was staying in this house the two sisters had slept together, and I leaned to the view that one sister had become a "carrier" of infection from the young man to the other sister, who developed diphtheria eleven days later. Subsequent events, however, put a different complexion on the matter. On January 5th, I received a letter from the same doctor who previously attended O.G. and who notified F.R., that O.G. was now suffering from what "looks as if it might be post-diphtheritic paralysis; general weakness "difficulty in swallowing liquids, and no knee jerks." It is probable, therefore, that O.G. was the first case of diphtheria, and that although he was seen by two doctors, one of whom had the possibility of diphtheria before him, diphtheria was overlooked.

APPENDIX E.

FURTHER INSTANCES OF "CARRIER" CASES.

First instance.—F.W., æt. 6, attended until March 13th a class of a school in which cases of diphtheria were occurring at the time. She was then kept at home because she had ringworm, but is stated to have had no cold or sore throat. On the 19th March her sister, E.W., æt. 4½ years, failed with diphtheria. E.W. had not been attending school, and had been kept strictly indoors owing to a recent attack of chicken-pox. There was one other child in the house, a brother, æt. 7, who attended the same school as F.W., but in a class in which no cases of diphtheria had recently occurred. It may be argued that both brother and sister were infected from the clinical case E.W. This child, however, had been kept isolated for over three weeks, and I do not think it can be doubted that F.W. carried the infection from the school to E.W., although she was never ill.

Second instance.—O.N., æt. 3, failed with diphtheria 4th April, removed to hospital 9th April. Had not attended school, but two sisters had been attending the infected infant school mentioned above. N.N., æt. 6, was said to have had a "cold," onset about 8th March. Had continued to attend school until April 3rd. Negative swabs from her throat were obtained on 11th and 14th April. W.N., æt. 5, had a "cold," onset about March 15th, last attended school March 18th. On April 11th diphtheria bacilli were found in her throat. A sister, B.N., æt. 10, was never ill, but on April 14th diphtheria bacilli were found in her throat. It is fairly clear that N.N. infected the three other children, one with typical, one with atypical diphtheria, and a third with apparently only a bacteriological infection. N.N. was continuing to attend school, along with two other unrecognised cases, and these three between them caused a serious school outbreak of diphtheria.

Third instance.—B.N., æt. 16, failed with diphtheria November 14th, admitted to hospital November 17th. There were in the same house five members of the same family, and a second family consisting of Mr. and Mrs. W. and baby. It was now found that E.N., aged 12, had had nasal discharge and sore throat beginning about November 1st. This was put down to a cold, as she always "had trouble with her nose." She was away from school from November 6th, returning November 15th to 17th. In the same class was a girl who was last at school on November 5th, and soon afterwards died of diphtheria. E.N. gave a positive swab (nasal) on November 19th and she went to the isolation hospital. Swabs were then taken from a brother aged 5 (negative) an older sister, æt. 27, negative swab same date, and Mrs. W., who gave a positive swab on November 19th. With the exception of Mrs. W., who had been "rather poorly" none of these had any symptoms of illness. The interest of this group of cases is increased by the fact that E.N. had a definite attack of diphtheria in August of the year previous.

See also under Appendix B.

APPENDIX F.

RECURRENT OR PROTRACTED INFECTION IN DIPHTHERIA.

First instance.—B.G., aged 4, failed with diphtheria on August 19th, admitted to hospital on the 25th. On admission, moderate exudate on tonsil. No diphtheria bacilli found. A second negative swab on the 26th. On September 5th Hofmann bacilli found; on September 6th no diphtheria bacilli found. The child returned home on September 7th, directions being given that she was not to resume school attendance for six weeks from the following Monday.

B.G. resumed attendance in the Babies' Class of School on September 24th, contrary to directions, the teacher conniving at this. This class and the Fourth Class of the same school have a common cloak-room. On September 26th W.M., aged 3, failed with diphtheria in the Babies' Class, and on October 3rd H.W., aged 5, failed with the same disease in the Fourth Class. Diphtheria bacilli were found in each of these. No actual contact between these children and B.G. could be proved. It was now discovered that B.G. had improperly resumed attendance at school. A swab was taken from her throat with a negative result. She was quite well, and had no nasal discharge.

B.G. did not again attend school until January 25th of the following year, attending until February 5th. The following cases occurred in the same class at this school:—F.D., aged 3, onset February 2nd; O.S., aged 3, onset February 9th, and B.U., aged 4, onset February 6th. Diphtheria bacilli were found in each of these. No other likely sources of infection for the above children were discovered except B.G., and no further cases occurred when B.G. was again excluded from school, except that as shown on the chart, p. 9, three secondary cases occurred in O.S.'s family.

B.G.'s family consists of father and mother, boy aged 5, and a child aged 2½ years. These have all remained well, except that the child aged 2½ had "a cold" on February 3rd, when a negative swab was obtained from him, and a negative swab once more from B.G.

In the same house lives another family consisting of father and mother, a child aged 2, who had bronchitis near Christmas, and a boy, G.B., aged 5½, who had "bronchitis" beginning about December 10th, had again a "cold on his chest" onset January 26th, nasal discharge beginning February 7th, seen by a doctor on February 10th, and notified as diphtheria the same day. No diphtheria bacilli were found in his nasal or throat secretions, although these were examined repeatedly after his admission to hospital. He was very pallid and a fortnight after admission developed a nasal voice.

Second instance.—F.M., aged 10, failed with diphtheria on November 19th, admitted to hospital on November 20th. Infected at school. Had pharyngeal diphtheria; no nasal discharge. Three consecutive negative swabs were

obtained from her throat on December 14th, 15th, and 16th, and she was discharged on the 17th.

In the same family were father and mother, and two sisters, aged 8 and 7, none of whom had diphtheria. The two sisters returned to school on December 20th. The ex-patient returned to school on February 1st, six weeks after leaving hospital, in accordance with our usual procedure.

F.S., who is in the same class with F.M. at school, failed with diphtheria on February 4th. No other cases occurred in this class, and no other source of infection for F.S.'s attack could be discovered. Suspicion being directed to F.M,. a swab was taken from her throat on February 8th, *with a positive result.* She was carefully examined, and found to have no nasal discharge, and had been quite well since returning home. Her two sisters have slept in the same bed with her since the week before Christmas. On February 10th, swabs were taken from the three sisters again, with a negative result in each case. On February 23rd F.M. again gave a positive swab, and on March 7th a negative swab.

APPENDIX G.

PROTRACTED INFECTION OF DIPHTHERIA IN EAR DISCHARGES.

First instance, simulating a "return" case.—N.B., æt. 5, failed with diphtheria on February 5th, admitted to hospital February 10th, and discharged March 18th. She slept at home this night in a separate bed and next morning was taken to another town, returning home on March 28th. On the 25th March K.N., æt. 2, a step-sister of the above, failed with diphtheria, and it was suspected that she might be a "return" case. The above dates, however, do not fit in with this supposition, and the following appears to be the probable source of K.N.'s infection. The baby brother of N.B., æt. 9 months, began with discharging ear on or about February 24th, a fortnight after N.B.'s admission to hospital. When K.N. failed with diphtheria this baby's ear was examined, and a pure culture of diphtheria bacilli obtained by cultivation of the ear discharge on blood serum.

Second instance.—K.S., æt. 4, was infected with diphtheria at —— School, and failed on November 14th. He was admitted to hospital on November 15th, and discharged on December 21st, after three consecutive negative swabs from the throat. Immediately after K.S.'s admission to hospital the brother, D.S., æt. 4, and sister, W.S., æt. 1, were given prophylactic doses of antitoxic serum. At this time a swab from D.S.'s throat gave a negative result. K.S., while in hospital, had left otorrhœa, which began November 28th, and on December 11th had ceased. He had profuse nasal discharge on admission, but this had ceased when he went home. The otorrhœa returned after he came home. He occupied a separate bedroom, but played with D.S. and W.S. during the day. W.S.

remained well, but on January 3rd, 14 days after K.S. came home, D.S. failed with diphtheria and was removed to hospital next day. K.S. was now re-examined and found to be anæmic, and still had left otorrhœa. A swab from this ear on January 5th showed diphtheria bacilli.

Third instance. D.R., æt. 1 year, failed with diphtheria on November 7th, admitted to hospital on November 18th with pharyngeal and nasal diphtheria. On November 28th right otorrhœa began; on December 9th there was double otorrhœa, and some evidence of pharyngeal paralysis. On 31st December a negative swab was obtained from the throat, but the discharge from both ears showed diphtheria bacilli. On January 5th throat and ear swabs all negative. On January 7th throat swab negative. January 9th throat swab negative, ear swab growth too scant for diagnosis. January 11th, swabs from both ears negative; only very slight ear discharge and no nasal discharge. The patient was sent home on January 12th. The serum-growth taken on the 31st December was tested by Dr. Eyre, and found to be virulent.

The Application of Chemical Analysis to the Study of Biological Processes of Sewage Purification.

BY

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The Application of Chemical Analysis to the Study of Biological Processes of Sewage Purification.

Introduction.

Owing to the general adoption of water carriage and the increasing scarcity of land in the vicinity of large towns, the question of sewage purification has of recent years become of such far reaching importance, and so many new developments have taken place in connection with it, that a long course of lectures would be required to deal with the subject at all adequately.

At Professor Delépine's suggestion I have selected one branch of the subject upon which more particularly to speak, viz., *the chemistry of the Bacterial treatment of sewage.* As the matter of the lecture developed in my mind, it has seemed better to modify slightly the title as originally advertised, and my subject will be best defined as "The Application of Chemical Analysis to the study of the Biological Processes of Sewage Purification."

My intention is not to enter into the details of specific methods of analysis, but rather to show how the results obtained by the various methods in use for the examination of sewage and effluents may serve as a guide to those who have to advise or report upon questions of sewage purification. At the same time I hope to be able to indicate directions where further knowledge is desirable.

Methods of Sewage Purification. It will be useful at the outset to summarise briefly the various methods of sewage purification available, and for that purpose I have prepared the following table:—

Preliminary Treatment.	Purification Process.	Typical Examples.
Screening	Dilution by Discharge into river	Hamburg
Settlement	„	Cassel
Chemical Treatment	„	London
Septic Tank	„ sea	Morecambe
Screening	Broad Irrigation over Land	Paris
Settlement	„	West Derby (near Liverpool
Chemical Treatment	„	Wolverhampton
Septic Tank	„	Birmingham

Preliminary Treatment.	Purification Process.	Typical Examples.
Screening	Intermittent Filtration through Land	Dewsbury
Settlement	„	Berlin
Chemical Treatment	„	Bolton
Septic Tank	„	Prestwich
Screening	Contact Beds	Hampton-on-Thames
Settlement	„	Oldham
Chemical Treatment	„	Heywood
Septic Tank	„	Manchester
Screening	Continuous Filters	Leeds
Settlement	„	Malvern
Chemical Precipitation	„	Salford
Septic Tank	„	Accrington

Many other equally typical and instructive examples might, of course, be given besides the particular ones chosen. I have been guided chiefly by my own knowledge, and I have endeavoured to choose one instance in each case where the works are noteworthy either from their size or completeness, rather than to multiply instances. The final purification in all these cases is biological. In the case of discharge into large bodies of water the purification is effected by the action of various organisms in presence of oxygen, dissolved in the water. In the other cases a similar final result is obtained through the action of organisms which develop on the extended surfaces offered by land or the material of artificial filters.

Objects of Sewage Analysis. The objects of sewage analysis may be broadly defined as follows:—

1. To determine the character of the sewage to be treated.
2. To determine the efficiency of purification works.
3. To determine the effect of discharge of sewage or effluents into various bodies of water either river, lake or sea.

In connection with all of these questions special problems are constantly arising demanding careful scientific investigation.

DETERMINATION OF THE CHARACTER OF THE SEWAGE.

The first application of sewage analysis we have to consider then is the determination of the character of a sewage with a view to deciding what is the best and most economical method to adopt for its purification. With this problem is bound up the third above mentioned, viz., the investigation of the stream into which the effluent will flow, but it will be convenient to take the first part of the question by itself and two cases present themselves:—

(*a*) The case of a town already sewered.

(*b*) The case of a town not yet sewered.

Town already Sewered: Taking of Samples. The first of these two cases is the simpler, as here representative samples of the sewage may be taken and directly analysed.

To obtain a true idea of the character of the sewage it is necessary to take samples at least every hour during the 24, and if an average sample is required the separate samples should be mixed in proportion to the flow, the latter being determined by hourly gaugings, or by a recording instrument.

Differences between Night and Day Sewage. The following tables of results obtained in Manchester will indicate the difference in character and quantity between the sewage flowing during the night and during the working hours of the day.[1]

Time.	Rate of flow in gallons per 24 Hours.	4 Hour's Oxygen Test.	Chlorine.
a.m.		Parts per 100,000.	
1	25,000,000	7·7	15·4
2	23,000,000	7·4	16·6
3	21,500,000	7·5	16·4
4	20,500,000	7·3	15·7
5	20,000,000	7·3	16·0
6	20,000,000	6·7	16·0
7	20,000,000	6·9	13·7
8	22,500,000	6·0	11·6
9	26,000,000	6·0	11·3
10	32,500,000	14·1	12·4
11	37,750,000	12·3	15·9
noon 12	40,000,000	10·6	16·0
p.m.			
1	42,500,000	13·9	17·4
2	43,000,000	14·0	17·9
3	41,000,000	14·6	19·9
4	39,500,000	15·3	18·9
5	41,000,000	15·3	18·0
6	42,000,000	13·0	16·6
7	41,500,000	13·9	18·0
8	38,000,000	13·7	18·3
9	35,000,000	12·7	17·6
10	32,500,000	14·1	18·9
11	29,000,000	11·4	18·9
mdn. 12	26,500,000	8·1	17·1

Influence of Rainfall. The difference between the composition of average samples made up in proportion to the flow, and average samples

[1] Annual Report of Manchester Rivers Committee, March 28th, 1900, p. 8, Diagram A. City Surveyor's Annual Report on Treatment of Sewage at Davyhulme for year ending December 31st, 1897, pp. 18–19. Supplementary Report of Superintendent, January 16th, 1900, p. 10.

made up in equal proportions will vary according to circumstances, notably rainfall. Thus, while in dry weather the average composition of a day's hourly samples will indicate greater impurity when these are taken in proportion to the flow than when they are taken in equal volumes, the reverse may easily be the case when the sewage is diluted by rain; though here no general rule can be given, as different results will be obtained according to whether a storm occurs after a period of dry weather, when large quantities of accumulated filth may be swept from the sewers, or whether the increased flow continues after the first flush has passed.

Influence of Trade Effluents. Further exceptions may occur through the irregular discharge of trade effluents on different days of the week or at different hours of the day.[2] One day of the week, usually Monday, is observed as washing day by the majority of people, and the composition of the sewage is affected thereby. It is necessary, therefore, that samples should be taken as above described, if possible, on every day of the week and under all conditions of weather.

Suspended Matter. In sampling sewage into ordinary bottles much of the suspended matter is apt to be missed. If a thorough examination is to be made it is well to take larger quantities and examine by sieving. Experiments in this direction have been made in Berlin by Dr. Monti[3] using sieves varying in mesh from 7mm. to 0·5mm. and about 400 litres of sewage. He obtained the following results:—7mm. sieve retained fæces, plant fragments, paper, etc.; 2·4mm. sieve retained plant fragments, leaves, seeds, twigs, etc.; 1mm. sieve retained a sludgy smooth deposit consisting partly of cotton, flax and wool fibres, muscle fibres and particles of humus; 0·5mm. retained a muddy, dark grey slime with more humus than the 1mm. residue. Both the last two residues increased in volume on drying. On exposure for some time they became gradually black and smelt of rancid fat.

In general it may be said that the substances retained on the sieves are those which contain most nitrogenous and fatty organic matter.

Fat and other Organic Products. The fat content of Berlin sewage has been carefully investigated by Schreiber,[4] and he finds the mean percentage of fats in the dry residue of sewage to be 13·8. When it is remembered that the whole of the soap consumption of a town finds its way into the sewers, besides much of the waste fat from ordinary households

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[2] Supplementary Report of Superintendent, *loc. cit.*, pp. 20—25. Annual Reports of Rivers Department, March 28th, 1900, Diagram B; March 27th, 1901, p. 33. Experts' Report on Treatment of Manchester Sewage, October 30th, 1899, Diagrams 4, 5, 7, 8, 20.

[3] "Ueber die Schwimm-u-Schwebestoffe des Berliner Sielwassers." Dr. Monti, *Archiv für Hygiene*, Band xlv.

[4] "Ueber den Fettreichtum der Abwässer und das Verhalten des Fettes im Boden der Rieselfelder Berlins." Dr. Karl Schreiber, *Archiv für Hygiene*, Band xlv.

and from restaurants, and that to this must be added in many cases the refuse from abattoirs and from soap, candle and leather works, it will be seen that the recovery of grease from sewage sludge is a problem worth consideration. It has been successfully attempted at Cassel, where only domestic sewage has to be dealt with. At Bradford, where exceptional quantities of grease are present in the wool-scouring waste liquors, a large percentage of the grease is recoverable economically by simple methods, and steps are being taken to extract the whole.

Decomposition of Samples. Before considering the more ordinary methods of sewage analysis it should be mentioned that as biological fermentation takes place very rapidly, especially in domestic sewage, the analysis should be made at the latest within 24 hours of taking the sample. Where this cannot be done it is necessary to stop or retard fermentation changes by addition of antiseptics.

Special Objects of Analysis. In order to arrive at a knowledge of the character of a sewage it is necessary to determine:—

1. The amount and nature of the organic and mineral solids in solution and suspension.
2. The amount and nature of the oxidisable matters.
3. The amount and nature of the nitrogenous matters.
4. The chlorine content.

1. Solids in Solution and Suspension. The ordinary methods for the determination of these, viz., filtration, evaporation and ignition of the suspended and dissolved matter are liable to various errors and must be used with judgment. Ammonium salts, especially carbonate of ammonium tend to be evolved on evaporation, though this may be to some extent avoided by addition of known quantities of oxalic acid before evaporation.

Ignition of the suspended and evaporated solids is accompanied by numerous changes, e.g., water of crystallisation and hydration is driven off, iron compounds are oxidised, sulphides are oxidised to sulphates and oxides. Due allowance being made for these facts, however, useful comparative results may often be obtained.

Complete analyses are much more tedious but more useful. Thus Rubner,[5] by careful precipitation with basic ferric acetate, and analysis of the clear liquid and precipitate, has been able to show that 40 per cent. of the combustible matter in the precipitate consisted of cellulose, 20 per cent. of fat, and the remainder mostly albumen. The phosphates will also be in this precipitate.

2. The Amount and Nature of the Oxidisable Matters. Rubner was able to determine the carbon and nitrogen in his precipitate, the carbon by moist combustion by chromic acid, and the nitrogen by the Kjeldahl

[5] "Das Städtische Sielwasser und seine Beziehung zur Flussverunreinigung." M. Rubner, *Archiv für Hygiene*, Band xlv.

method, as well as to determine separately cellulose, fat, etc., as above mentioned. He obtained also interesting comparative values by combustion of the precipitates in the calorimetric bomb. The residue on evaporation of the precipitated liquid can be similarly dealt with. As a rule, simpler and more rapid methods are made use of, the amount of oxygen absorbed from a known volume of acid permanganate being taken as a measure of the oxidisable matter present.

Broadly speaking the test is applied in three ways, the sample is left in contact with excess of permanganate at the ordinary temperature or slightly above it, for four hours and for three minutes respectively, or it is boiled ten minutes with excess of permanganate (Kubel method) the excess of permanganate being suitably determined in each case. As a rule the Kubel method gives considerably higher results than the four hours test, but the difference depends to some extent on the nature of the oxidisable matter.

There are, of course, many trade effluents which are oxidisable by permanganate. The following are equally capable with ordinary sewage of putrefactive decomposition, viz.:—Brewery waste, yeast, etc.; size manufacturers' refuse; abattoir refuse; paper waste; distillery waste; tannery and fellmongering refuse; wool-scouring refuse.

Other substances may be present which are not putrefactive but which absorb oxygen. Such are the following:—Various coal-tar products, *e.g.*, phenol, naphthylamine, naphthols, naphthalene sulphonic acids, pyridine bases; dyes, *e.g.*, indigo, logwood, etc.; inorganic salts, *e.g.*, thiocyanates, sulphites, iron pickle, nitrites.

A very good indication of the presence of such bodies as these is afforded by the ratio of the four hours to the three minutes' test, a much greater proportion of the total absorption taking place, as a rule, in three minutes, when oxidisable trade effluent is present than when only domestic sewage has to be dealt with. Examples:—

	Manchester Sewage, containing Oxidisable Trade Effluents.		Urmston & Flixton Domestic Sewage.
Oxygen Absorbed from Permanganate.	Parts per 100,000.		Parts per 100,000.
4 Hours' Test	12·7	.....	5·2
3 Minutes' Test	6·0	.....	2·0
Ratio 4 hours to 3 minutes	2·0:1	.....	2·5:1

The difference between these ratios is more marked when the samples are filtered before analysis, as the matter in solution is affected by the permanganate more rapidly than matters in suspension.

The majority of the trade effluents above mentioned, especially the non-putrefactive effluents absorb oxygen from permanganate instantly, and consequently affect the 3 minutes' test to a greater proportionate extent than the 4 hours'. For the same reason there is less difference between the Kubel and the 4 hours' test with sewage containing trade effluents than with domestic sewage. Examples:—

Manchester Sewage.			Hamburg Experiments.[6]		
4 Hours' Test.		Kubel Test.	4 Hours' Test.		Kubel Test.
9·4	.....	11·5	8·8	.....	13·0

A further differentiation between putrefactive and non-putrefactive oxidisable matter is arrived at by means of the dissolved oxygen test, or biological oxygen absorption test which we shall have to consider later when speaking of filtrates.

3. Total Nitrogenous Matter. The actual composition of the nitrogenous organic matter in sewage as it enters the works is not too well known. Certain bodies are known to be originally present as products of excretion, and some indication of their quantity and the degree of decomposition is arrived at by the three customary methods of determining ammonia, viz., as free ammonia, albuminoid ammonia and organic or Kjeldahl ammonia. The final products of oxidation of nitrogenous organic matter, viz., nitrites and nitrates are seldom found in sewage except when diluted by rain.

The free ammonia is derived in large measure from the ammoniacal fermentation of urea, a change which takes place with great rapidity, and also in some measure perhaps from incipient decomposition of albuminoids.

The albuminoid ammonia affords some index to the quantity of complex nitrogenous matter present, and to the degree of decomposition which has taken place, to which further reference will be made when considering the changes taking place in the septic tank. It is, however, a somewhat indefinite number, and depends to a great extent on the details of the analytical method used. If the boiling with alkaline permanganate is continued indefinitely there is a corresponding evolution of ammonia, and with increasing concentration of the alkaline solution the process merges into a modification of the Kjeldahl.

By the Kjeldahl method if proper precautions are taken the total nitrogen of the nitrogenous organic matter may be obtained. There is a large number of different modifications of this process. We may hope to have some information with regard to it in the report on analytical methods shortly to be published by the Royal Commission on Sewage Disposal. The method is, it must be confessed, somewhat difficult and tedious, but either some such method or Frankland's combustion method must be used if absolute figures are required.

Many attempts have been made to work out a definite ratio between the Kjeldahl and albuminoid figures, especially in America, where it has been found that in the case of the majority of Massachusetts sewages[7] the following formula may be used with fair results:—

Organic nitrogen = 12 times the square of the Nitrogen as Albuminoid Ammonia divided by the Nitrogen as free Ammonia.

In England, according to Scudder,[8] whose evidence I can confirm, it has

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[6] "Leitfaden für die Chemische Untersuchung von Abwasser," Farnsteiner, Buttenberg and Korn. R. Oldenbourg, Munich and Berlin.

[7] Fuller. "The Composition of Sewage, in relation to Problems of Disposal." *Technology Quarterly*, Vol. xvi., No. 2, June, 1903.

[8] Royal Commission Reports, Vol. ii., Q5950, p. 329.

not been possible to find a fixed ratio. It will depend no doubt on the dilution of the sewage and the degree of decomposition of the albuminoids as above mentioned. In Manchester and in other manufacturing towns the character of the trade waste will be of influence. Ammonia recovery liquor from the gas works contains residual ammonia and sulphocyanate of lime, which will respectively affect the free and albuminoid ammonia figures. Amido compounds such as naphthylamine will be of influence, also brewery and tannery waste.

Acidity, Alkalinity, Chlorine. Other factors in the composition of sewage besides those already mentioned, which have to be taken into account are the *acidity* and *alkalinity*, especially when trade effluents are present, and also the *chlorine number*. This is useful in affording an index to the dilution of the sewage, though allowance must be made in the case of some sewage for the presence of bleaching liquor, iron pickling liquor (when hydrochloric acid is used) and also for sulphocyanates in ammonia recovery liquor, which are estimated along with the chlorine in the ordinary titration with silver nitrate. A large increase is sometimes observed in the chlorine figures during a thaw of snow, owing to the salt used for melting.

By all these methods we may obtain a fairly complete idea of the character of the sewage to be treated. Here the methods of analysis used, if they are to be really informing, should be as complete as possible. As a matter of fact thorough data for a number of towns of varying character are still lacking. Until these are obtained there will always be some amount of uncertainty in dealing with the second problem in this division of our subject, viz.:—

The Estimation of the Composition of the Sewage in a Town not yet Sewered. In this case the effect of certain factors on the composition of the sewage has to be carefully weighed.

Human Excreta. At the basis of the calculation there are, of course, certain physiological constants. Thus, according to Michael Foster[9] the urine passed per 24 hours by an ordinary individual contains 1,500 grams water and 72 grams solids, of which about 33 grams consist of urea. The weight of fæces amounts to about 150 grams per diem,[10] and according to Rubner[11] the amount of paper used amounts to 6·8 grams per diem.

Domestic Water supply. The character of the sewage to be treated will depend upon the water supply per head. This varies greatly under different conditions. In America, where there is a lavish use and sometimes waste of water, it may be as much as 100 gallons per head; in England it is commonly from 20 to 40 gallons; while in India and the Colonies it may be much less.

Experiments have been made by A. E. Silk with latrine sewage in connection with the Presidency Jail at Calcutta, where only half a gallon per head of

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[9] "Physiology," Book ii., p. 653.

[10] Gillespie. "Natural History of Digestion," p. 203.

[11] *Loc. cit.*

water was used. No figures are published for the raw sewage, but the following results of the examination of the contents of the tank carts in Johannesburg will give some idea of the character of the liquid, although probably the latrine sewage was even stronger. The analysis was made by Dr. Pakes, Government Analyst, and has been sent me by the courtesy of Dr. Porter, the Medical Officer of Health.

	Parts per 100,000.		
	No. 1.	No. 2.	No. 3.
Total Solids	289·6	405·8	273·65
Loss on ignition	104·40	174·15	168·30
Solids in suspension	135·4	264·2	281·4
Loss on ignition	16·05	86·2	77·05
Solids in solution	154·2	141·6	150·55
Loss on ignition	88·35	87·95	91·25
Oxygen absorbed (4 hrs. 27 C.)	20·5	—	29·5
Chlorine	29·5	31·5	29·5
Saline Ammonia	15·25	9·12	18·21
Albuminoid Ammonia	5·60	10·07	12·10
Nitric Nitrogen	Trace	Trace	Trace
Nitrites	Nil	Nil	Nil

We may compare with this Rideal's figures for Exeter sewage, a moderately strong domestic sewage.[12]

Total Solids in Solution	46·8
Oxygen Consumed	6·56
Free Ammonia	3·6
Albuminoid Ammonia	1·4

It may be noted here that a certain minimum dilution is necessary for purification as Warington[13] has shown that a 12 per cent. solution of urine was the highest strength nitrifiable.

Soap, Grease and Trade Effluents. In the case of the total sewage of a large town the amount of soap and grease passing away from the houses must be considered. As a result of the investigations of Berlin sewage above referred to, Schreiber concludes that domestic grease production amounts to 8·4 grams per head per day. The amount and character of the various trade effluents must be carefully ascertained.

Rain Water. The problem is greatly complicated if the combined system of sewerage is in contemplation. Regard must then be had to the incidence and amount of the rainfall, whether seasonal as in the tropics, or fairly evenly distributed as in Manchester. The amount of sub-soil water likely to enter the sewers under different conditions of weather and season must

[12] Royal Commission Report, 1902, Vol. ii., Q4135.

[13] *Journ. Chem. Soc.*, 1884, p. 661.

also be allowed for. The figures arrived at in the manner thus indicated may be checked by reference to similar towns already sewered.

Thus Fuller (*loc. cit.*) gives comparative data of grams per capita of the various elements as determined by ordinary analysis, and finds a fair agreement between the data for towns of a corresponding character. Such data can, of course, be only approximately comparative, as local conditions vary more or less from town to town, especially where the sewerage is on the combined system. As further exact data accumulate for different conditions, it should be possible to assess with considerable accuracy the composition of the prospective sewage of a town still short of a sewerage system.

THE APPLICATION OF CHEMICAL ANALYSIS TO THE DETERMINATION OF THE EFFICIENCY OF PURIFICATION PROCESSES.

We have now to deal with *the application of chemical analysis to the determination of the efficiency of purification processes.* Here we are chiefly concerned with obtaining comparative numbers for control purposes, and rapidity is one of the chief requisites in the methods used. In order better to appreciate the meaning of the analytical results obtained it is necessary here to consider briefly the character of the changes taking place in the septic tank, and in the bacterial filters, whether these latter are intermittent (contact beds) or continuous (percolating filters).

It must be at once confessed that all the chemical and other changes which take place during these purification processes are by no means fully understood. I will, however, in brief outline give what in the light of our present knowledge seems to me to be the fairly well ascertained course of things.

Changes in the Septic Tank. Taking first the septic tank, here the sewage passing slowly through deposits the greater part of its matters in suspension. These falling to the bottom enter in time into anærobic decomposition. Albuminoids are broken down by the action of various organisms and enzymes yielding numerous amido compounds of more or less complexity, *e.g.*, glycocoll, leucine, asparagin; also compounds such as phenyl-acetic acid, indol and skatol, and finally ammonium salts, amines, hydrogen sulphide, mercaptans, marsh gas, free nitrogen, hydrogen and carbon dioxide.[14] These changes take place to some extent, and the conversion of urea into ammonium carbonate almost entirely, in the sewers, and it will depend on the length of travel of the sewage between the town and the sewage works how far the changes have progressed. The conversion of cellulose into methane, hydrogen and CO_2 by the action of two specific organisms,[15] one of which produces methane and the other hydrogen, takes place more slowly and is chiefly confined to the septic tank. The slow

[14] Rideal, Royal Commission Report, Vol. ii., p. 250. Emmerling Berichte, 1897, xxx., p. 1863. *Mittheilungen aus der Königlichen Prüfungsanstalt*, Berlin, Heft 1, 1902, p. 81. Fowler, Manchester Rivers Department Annual Report, March 27th, 1901, pp. 29—42. Buxton, *American Medicine*, Vol. vi., No. 4, pp. 137—142, July 25th, 1903, No. 15, pp. 381—583, October 10th, 1903.

[15] Omeliansky. *Centralbl. Bakt.*, 1902 (ii.), p. 193 and *seq.*

splitting up of fats by suitable enzymes yielding butyric, acetic and c acids,[16] will also take place chiefly in the tank.

There is reason to think that the chief bacterial activity is in the depo sludge. After this has begun to ferment portions are carried up by the g which surround the particles, and finally a crust is formed consisting la of matted fibrous material full of organic life. There is a continual interch between the sludge and scum, and thus the liquid constantly passing thr becomes also affected and changed in character. The matters in solution ap to break down to some extent and the liquid is readily purified in bact filters.

Changes in Bacterial Beds or Filters. Here the cha are even more complex. As far as our present knowledge goes we hav distinguish at least three classes of change, *physical*, *chemical* and *biolog* Among the latter may be distinguished changes due to *bacterial* or plant and changes due to animal life.

In a new unripened filter the *physical* effect is first apparent. The ma in emulsion and in many cases in actual solution are retained by the me by a process of *physical absorption*, akin to the condensation of CO_2 on r glass wool observed by Bunsen.[17] Purely *chemical* oxidation of certain compo may take place owing to condensed oxygen, *e.g.*, ferrous compounds such as are oxidised, also H_2S to H_2SO_4 and certain leuco bases to the correspon dyes.[18]

A very interesting experiment has been devised by Lübbert to show powerful oxidising action which takes place on the surface of cinders. 2 per cent. solution of dimethyl-aniline in ordinary alcohol be brougl contact with clinkers of about 2mm. diameter, the dimethyl-aniline is oxi to methyl violet. Lübbert has obtained the effect in a few hours. In atte ing to repeat the experiment I could not obtain the colour till after more a week's contact, it was then, however, quite strong, and no change took in a portion of the same solution left out of contact with clinkers. In ordinary way it is necessary to heat dimethyl-aniline with potassium chl to obtain the colour.

Hypochlorites are oxidised to chlorates, and thus a means is found purifying hospital sewage sterilised with chloride of lime.[18] Whereas pl will, when present in large quantities, inhibit the action of the bacterial fi these are practically unaffected by the presence of reasonable amount chloride of lime. These apparently purely physical and chemical cha require further study.

The *bacterial* changes are chiefly those concerned in the gradual conve

[16] Herfeldt. *Journ. Soc. Chem. Ind.*, May, 1895.

[17] Kattein and Lübbert. "Zur Bedeutung der Absorptions Vorgänge bei der biolog Abwasserreinigung." *Ibid*, 1903, No. 25. Fowler, Manchester Rivers Department Annual R March 25th, 1903, p. 22.

[18] Dunbar. "Zur Beurteilung der biologischen Abwasserreinigungs methoden." *Gesund Ingenieur*, 1903, Nos. 33 and 34. Dunbar and Korn, "Zur Desinfektion von Abwässer gleichzeitiger Reinigung derselben." *Ibid*, 1904, No. 2.

of ammonia into nitric acid, the process known as *nitrification*, and the subsequent oxidation of organic matter by means of the nitrate formed. But purely carbon oxidation changes must go on, *e.g.*, the breaking down of dextrine and sugar, etc.,[19] and no doubt the processes started in the septic tank are carried on and complete themselves in the primary bed or in the upper layers of a continuous filter. The ammonia thus set free is partially oxidised. It has never been possible actually to account for the total quantity of nitrogen disappearing. Some of it has been proved to be liberated in the free state,[20] no doubt during the oxidation of organic matter by nitrates, in accordance with the earlier observations of Gayon and Dupetit, some will be stored in the filter as a constituent of the residual humus, and a certain quantity undoubtedly is taken up by the higher forms of life in the filter which themselves probably feed on the bacteria, and here we are confronted with changes due to animal life as distinct from bacterial life. Some very interesting observations in this direction have been made by Letts[21] and Lorrain Smith[22] in Belfast. They, in common with other workers, have noticed the numerous forms of life, other than bacteria, present in contact beds. Letts noticed swarms of minute, black, wingless insects, probably "Podura Aquatica"; also numbers of worms. Besides these higher organisms there were countless animalculæ present.

Lorrain Smith instituted experiments on the rate of disappearance of unoxidised nitrogen in dilute broth inoculated with sewage, and found it far less than when the broth was brought in contact with material from the bacteria beds.

A curious biological succession has been observed at Accrington. On the continuous filters there, at certain seasons of the year, there are crowds of small flies. These give place later to spiders, which finally become the prey of birds. Flocks of wagtails have been observed on the bacteria beds at the Manchester Corporation Sewage Works.

The Application of Analysis to the Control of the Septic Tank cannot be said yet to be fully worked out, the process being complicated by the fact that changes are going on simultaneously both in the deposited solids and in the liquid passing through. Researches have been directed to determining the amount of solids digested by ascertaining the quantity of suspended matters passing in and out of the tank during a specified time and the amount of sludge present in the tank at the end of that time. Unless the flow through the tank is constant or easily measurable there is considerable difficulty in sampling, as, in order to obtain correct results, samples must be taken in proportion to the flow.

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[19] M. E. Rolants. "De l'Epuration Biologique des Matierès Hydro-carbonées dans les eaux résiduaires industrielles." *Revue d'Hygiene*, Dec., 1902.

[20] Letts. Report on Belfast Sewage Scheme. Third Report of Royal Commission, Vol. ii., p. 132. Gayon and Dupetit, Station Agronomique de Bordeaux, 1886.

[21] *Loc. Cit.*

[22] Report to Corporation of Belfast re Bacteriological Investigation of the Experimental Contact Beds. Third Report of Royal Commission, Vol. ii., p. 147.

Careful measurements made in London,[23] Manchester,[24] and at Worcester (Mass.)[25] appear to show that a considerable percentage destruction of organic matter takes place even with sewage containing manufacturing refuse. With purely domestic sewage ordinary experience would indicate a much greater percentage destruction, but exact figures are still to seek. Some difference of opinion indeed exists on the data already available, the point of discussion appearing to resolve itself into a question of the conditions and limitations of septic action. There is no doubt, for instance, that if certain septic tank effluents are kept in a closed bottle they develope H_2S, become exceedingly offensive, and deposit considerable quantities of matter originally in solution. An effluent in such a condition may be termed "over-septicised," and there is no doubt that it is possible to hold sewage too long in the septic tank when nuisance is apt to arise and the effluent is very difficult subsequently to purify on ærobic beds. There is some evidence that toxic products, *e.g.*, amines, are formed,[26] the H_2S and other gases present also quickly use up the oxygen supply in the filters.[27]

But at present, I am bound to say, analytical methods have not been developed enough in the direction of determining when a septic effluent is in the best condition for subsequent purification. Possibly the composition of the gases evolved from the tank may give some indication, but these arise chiefly from the solids and have no necessary reference to the condition of the liquid. Thus, if the flow through the tank is very irregular the liquid may be over-septicised at one time and not enough at another, while the process remains the same in the deposited sludge throughout.

Further research is needed in this direction, especially as regards the ratio of the nitrogen figures. Possibly some help may also be forthcoming from the bacteriologist. At present the routine tests, the oxygen absorption test, albuminoid ammonia, etc., merely indicate the percentage purification on the raw sewage, and the character of effluent to be put upon the beds.

Of considerable importance is the determination of *suspended matter*, as showing the need or otherwise of removing some of the deposited sludge and as indicating the quantity of solids passing on to the beds.

Analysis in the Control of Biological Filter Beds. Analysis is, however, of the greatest value in *controlling the work of biological filter beds* whether *intermittent or continuous*. Here we are concerned with the actual disappearance of oxidisable matters, and ascertaining, to some extent

[23] Clowe's Fourth Report to Main Drainage Committee of the London County Council, April 17th, 1902, p. 21, 22—39.

[24] Annual Report of Rivers Committee March, 1901, pp. 29—42.

[25] Kinnicutt and Eddy. "The Action of the Septic Tank on Acid Iron Sewage." Third Annual Report of the Connecticut Sewage Commission, 1902.

[26] Rideal, *loc. cit.*, Q4146, cf. Scott-Moncrieff, ditto, Q3242.

[27] Annual Reports Massachusetts State Board of Health, No. 33, 1902, pp. 286–7, 291-2; No. 31, 1901, pp. 387—392.

at any rate, the quantity of oxidised material formed. Thus the percentage purification, either as measured by the oxygen absorbed or by the albuminoid ammonia, gives an excellent idea of the work done. It has, in fact, been argued by Dunbar and Thumm,[28] that if a certain per cent. purification, *e.g.*, 60—65 per cent., of any given tank effluent, as measured by the Kubel method, after filtration, through paper, has been effected by the purification process, the final effluent is non-putrefactive whatever the absolute numbers may be. This may hold in certain cases, but hardly in all. In Manchester, *e.g.*, the indications of the four hours and albuminoid ammonia test are complicated by the presence of trade effluents, which affect the indications of the two tests, the former especially, and render such a method of determining effective purification uncertain.

The amount of *nitrate* present and the *behaviour on incubation* in closed bottles either with or without addition of aerated water are safer guides in such cases. There can be no doubt that the amount of nitrate appearing in an effluent is the residuum left over after a certain proportion has been employed in oxidising carbonaceous impurities. This is especially so in the case of the contact bed. If the material of a contact bed is washed out after rest with ordinary town's water, much more nitrate will be obtained in solution than if, say, septic tank effluent were employed. This denitrification change is no doubt necessary for the effective oxidation of many substances, *e.g.*, cellulose.[29] In the case of the contact bed the two changes nitrification and de-nitrification take place alternately, and possibly also simultaneously in the same bed, and consequently after double contact an effluent is obtained where both carbonaceous and nitrogenous organic matters have been well oxidised.

In the case of continuous filters, at any rate those of the more open kind, the ammonia is rapidly oxidised to nitric acid, but the conditions do not seem so favourable to carbon oxidation, therefore, these filtrates often have a rather high oxygen absorption and albuminoid ammonia, and incompletely oxidised organic suspended matter passes through and has to be strained out or settled. During the straining process some amount of de-nitrification takes place and the final effluent, though containing less nitrate, is of greater general purity. It is here that the sequence of changes in biological filters appears to be different from that taking place during direct oxidation in mixtures of sewage or effluent with oxygenated water, which we shall have to consider in the next section. The precise change going on depends greatly on the size of material in the filter, as on this depends the extent of surface of contact and the amount of liquid held up in the interstices of the material and consequently the interactions which take place.

In judging of the working of a filter, then, we shall be guided by the percentage purification, the disappearance of free ammonia, and the appearance of nitrate. If the free ammonia does not decrease or if the nitrites increase at the expenses of the nitrates, it is generally a sign that the filter is overworked.

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[28] Abwasserreinigungsfrage, Munich, 1902. R. Oldenbourg, pp. 18, 19; cf. "Leitfaden für Chemische Untersuchung von Abwasser," *loc. cit.* pp. 54, 55.

[29] G. van Iterson. "Proc. K. Akad. Wetensch.," Amsterdam, 1903, v., 685—703 *Journ. Chem. Soc.*, Abs. 1903, Part ii., p. 503.

If a good supply of nitrate is present the effluent will rarely, if ever, putrefy on incubation, the oxygen of the nitrate being sufficient to oxidise the residual impurity, in any case the degree of putrefaction indicated by the increase in the three minutes oxygen absorption test before and after incubation will be less.[30]

The Matters in Suspension in an Effluent from Bacterial Filters are of great importance. It may frequently happen, especially in the case of continuous filters that considerable quantities of suspended matters pass away in the effluent. It may also be that the effluent, when incubated along with these suspended matters, is not putrefactive. It must not, however, always be concluded that the suspended matter is harmless, as if allowed to settle, the clear filtrate poured off and the suspended matter separately incubated with distilled water, then it will often be found to be putrefactive. The character of suspended matter which separates from filtrates under various conditions requires careful study. Perfectly clear land filtrates may, on standing, give ferruginous deposits, partly due to oxidation of iron present originally as organic ferrous salts, and partly due to the presence of bacteria which collect and separate iron from solution, *e.g.*, crenothrix.

The same may take place in the effluent from contact beds, especially where the sewage contains iron pickling refuse in solution. In the case of open, continuous filters actual suspended matter from the septic tank may find its way in an unoxidised condition through the filters. In all these cases the organic and volatile matter may also be in large degree due to growths of organisms of one kind and another, sometimes, it may be, of a fairly high order, *e.g.*, infusoria, etc.

In general it may be said that the more perfect the action of the bed the more harmless or more thoroughly oxidised will the suspended matter be, until finally it may be rightly described as organic residuum or debris. It must, however, if present in more than traces (3 grains per gallon have been suggested as a limit), be removed from the effluent by straining or settlement.

The experience of Leeds would show that an effluent containing large quantities of suspended matter can be strained through 6in. sand at the rate of two million gallons to the acre, or about six hours settlement is necessary. Much less than this is generally given, but as a rule it is not adequate.

THE EFFECT OF AN EFFLUENT UPON A BODY OF WATER, RIVER, LAKE OR SEA.

In deciding upon the fitness of an effluent to enter a body of water a great number of questions have to be borne in mind, involving really as much study as the purification process itself.

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[30] See p. 116.

Discharge into Sea. Here the following matters have to be considered:—

1. The set of tides and currents.
2. The character of the foreshore.

The discharge of sewage into the sea is by no means the simple problem it is often supposed to be. If not discharged into a strong seaward current it is apt to return with the tide and cause nuisance on the foreshore. I will refrain from mentioning specific instances where this has occurred, but the difficulty has been sought to be remedied in some cases, *e.g.*, at Morecambe by the provision of septic tanks, the effluent from which passes into the sea. In this way the solids are disintegrated and quickly disseminated, but even here the point of discharge must be carefully chosen, as it is hardly sufficiently understood that nitrification and oxidation of organic matter go on very slowly in sea water.[31]

With the bacteriological side of the question in relation to oysters I am not competent to deal. Unfortunately the remedy is here hardly to be found in the provision of biological purification plant, unless carried to the point of a water works, as it must, I fear, be conceded that *b.coli* and presumably other organisms of a more dangerous character pass unchanged through ordinary biological filters.

In regard to the character of the foreshore the special case of Belfast presents most interesting features. Here a very serious nuisance is caused in the summer months not directly by the decomposition of sewage but by the decomposition of the sea-weed "ulva latissima." This weed contains a very high percentage of nitrogen, and has been found by Dr. Letts[32] to owe its nourishment to sewage, as it absorbs both ammonia and nitrate. An ordinary well-nitrated effluent would therefore be as good food for this weed as sewage itself. The problem in Belfast therefore is not to oxidise nitrogen but to *disperse* it by the kind of reactions already described. It is probable that the mode of construction and working of the beds may be modified to suit this end. It is further suggested that the effluent may be allowed to flow through a pond containing the "ulva" before passing it away into the Lough. The final traces of nitrogen are thus absorbed by the controlled weed, and none is left to stimulate growth on the foreshore.

Discharge into Lake, River or Stream must be conditioned by the volume of the body of water with reference to the volume and character of sewage or effluent required to be discharged, the conditions as to currents, banks, etc., and finally whether the water is to be used for drinking purposes. In the study of the conditions of this problem the determination of *dissolved oxygen*

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[31] Letts *loc. cit. cf.* Letts, Blake, Caldwell and Hawthorne. "The Nature and Speed of the Chemical Changes which occur in Mixture of Sewage and Sea Water." Scientific Proceedings of the Royal Dublin Society, Vol. iv. (N.S.), Part iii., No. 24.

[32] Interim Report of Royal Commission, Vol. ii., Q8574 and *seq.*, also Proceedings of the Royal Society of Edinburgh, 1900–1.

is of the utmost value. When sewage is mixed with thoroughly oxygenated tap water a series of changes take place, probably identical with many occuring in biological filters, but apparently less complex and occupying more time. They have been very carefully studied by Adeney,[33] whose method of experiment consisted in making various mixtures of sewage and aerated tap water, and determining the composition of the dissolved gases after various periods. He made similar experiments with organic substances of known composition and drew the following among other conclusions:—

"(*a*) That when the atmospheric oxygen is present as dissolved oxygen, in sufficient quantity in proportion to that of polluting matter, only two of the three possible fermentative processes can take place, and these two not simultaneously but progressively, viz., (1) bacteriolysis, (2) nitrification.

"(*b*) That by bacteriolysis the unfermented organic matters are completely broken down, the products being carbon dioxide, water, ammonia and relatively small quantities of organic matters in a much altered form.

"(*c*) That nitrification sets in only after the completion of bacteriolysis.

"(*d*) That it may be concluded both from experimental evidence and from the chemical nature of the process of nitrification that if the polluting matters present in the water have already undergone the process of bacteriolysis, no secondary process such as putrefaction can set in, even if the dissolved oxygen in the water containing them be consumed before they become completely nitrified, and that hence, fermented organic matters and ammonia need not be regarded as presenting the same danger to waters that unfermented organic matters undoubtedly do."[34]

It follows that if an effluent is to be discharged into a stream there must be sufficient oxygen present in the effluent either dissolved or as nitrate, or there must be sufficient in the stream to completely oxidise the impurity.

The power of a stream to successfully deal with pollution is very hard to gauge as so much depends on whether thorough mixing takes place, whether eddies or back waters are frequent or whether there are rapids when thorough oxidation can take place. Much will also depend on the flora of the river, *e.g.*, whether many green plants are present which can assist oxidation.

Mutual Effects upon each other of Sewage and River Flora. Our knowledge of the mutual effect upon each other of sewage and river flora is, however, limited. From the careful researches of Kolkwitz[35] we know that thorough oxidation and consequent breaking down of putrefactive matters in sewage is inimical to the growth of sewage fungus such as *leptomitus lacteus*. On the other hand it may well be that many green river

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[33] "The Course and Nature of Fermentative Changes in Natural and Polluted Waters and in Artificial Solutions, as indicated by the Composition of the Dissolved Gases." Scientific Transactions of Royal Dublin Society, 1895—7.

[34] "Recent Advances in the Bacteria-Chemical Study of Sewage and other Polluted Waters." Dublin, John Falconer, 53, Upper Sackville Street, 1896.

[35] "Mitteilungen aus der Königlichen Prüfungsanstalt." Berlin, Heft 2, p. 34.

plants flourish, like the *ulva latissima* of Belfast Lough, on nitrates and consequently it is conceivable that even a well purified effluent may in certain cases unduly encourage the growth of weeds. No doubt where possible the manurial value of the nitrates should be used judiciously upon the land. In the case of the Manchester Ship Canal and other streams one could mention there is not likely to be an excess of nitrate for some time to come.

In the very interesting report to the Royal Commission on their investigation of the River Severn in the Shrewsbury District, Professor Boyce and Doctors Grünbaum, MacConkey, and Hill[36] carefully discuss the various influences at work in the self purification of rivers. The researches of Spitta[37] are also of considerable value in this direction.

The investigation of the sediment at the bottom of a stream is of the very greatest importance. An instructive case is cited by Marsson[38] of an effluent from a works producing nitro-naphthalene. The effluent, when diluted by cooling and condenser water, was apparently harmless to fish under the conditions at the point of discharge. On investigation of the mud near the outfall, however, it was found to have a strong smell of naphthalene products, and all signs of the organic life, necessary for the gradual breaking down of putrefactive matter, had disappeared. This effect was discernable 400 metres below the outfall.

Arbitrary Limits of Impurities fixed for Administrative Purposes. The whole conditions are so difficult that it is the custom of the various River Authorities to lay down limits of impurities which should not be exceeded by the effluent irrespective of the stream into which it flows. There is a gradual consensus of opinion however that these limits should not be merely empirical, *e.g.*, so much oxygen absorption, so much albuminoid ammonia, but should indicate the actual condition of the effluent as regards its capacity of putrefaction, or to use Dr. Adeney's words, they should indicate the amount of "unfermented organic matter" present. An effluent may contain high albuminoid ammonia for instance and yet be almost completely fermented, as is indeed often the case in effluents from strong domestic sewage. Thus the effluent from the installation in connection with the new Christ's Hospital at Horsham has an albuminoid ammonia of 0·33 and yet will hold its dissolved oxygen and support fish life.[39] A more striking case is that of the Calcutta experiments already quoted,[40] where filtrates yielding, according to the published analyses, 1 or 2 parts per 100,000 of albuminoid ammonia were found to be non-putrefactive.

The putrescibility of a filtrate may be determined by what is known as the incubator test, and by the dissolved oxygen test, already referred to, and which on account of their special importance may be here briefly described. The

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[36] Second Report of Royal Commission.

[37] *Archiv. für Hygiene*, Band xxxviii. and xlvi.

[38] "Mitteilungen aus der Königlichen Prüfungsanstalt." Berlin, Heft 2, p. 28—31.

[39] *Journal of the Royal Institute of British Architects*, third series, Vol. xi., No. 8, p. 193.

[40] *Loc. cit.*

"*incubator test*" introduced into general practice by F. Scudder is simple and useful where no antiseptic agents are present in the sample. It consists briefly in determining the three minutes oxygen absorption test of the sample, completely filling a small bottle with the sample, incubating at 22—26°C. for five or six days, and re-determining the three minutes oxygen absorption at the end of that time. If putrefaction has taken place the three minutes absorption will have increased owing to the easier oxidisability of the products of putrefaction, *e.g.*, H_2S. In general if the sample is a good one the three minutes' test is less after incubation, in consequence of the oxidation of organic matter which has taken place during incubation through the agency of dissolved oxygen or nitrates.

The *dissolved oxygen test* consists in mixing the sample with enough oxygenated tap water to complete the oxidation of impurities, incubating for one or two days and determining the loss of dissolved oxygen.

A rapid qualitative indication of the true purity of an effluent is afforded by the addition of a trace of *methylene blue* sufficient to faintly colour the sample. On keeping the coloured sample for a few hours in a closed bottle at incubation temperature the blue colour quickly disappears if the effluent is putrefactive.[41] The test has quantitative possibilities about it which have yet to be worked out.

Although the incubator test and the dissolved oxygen test are of the greatest value, it is necessary to mention that they are subject to certain limitations which have yet to be investigated, due to the effect of certain trade-effluents either on the oxygen present or on the bacteria. Used with judgment they are in my opinion the best criteria of the purity of an effluent.

Concluding Remarks. From a careful examination of effluents by tests of this description, and by giving at the same time thorough consideration to the conditions existing at and near the point of discharge, the necessary degree of purity for every case should be capable of being exactly assessed, and thus the limit of necessary expenditure defined. For in my view the sewage problem is essentially a cost problem.

It is comparatively easy in the light of our present knowledge to purify sewage to any degree of purity provided cost does not enter into the question. The true solution is *to purify sewage to well within the limits of safety for any given set of conditions at the lowest possible cost.*

I am conscious that this lecture has been of a very sketchy and fragmentary nature. Many questions which I have had to dismiss with the briefest reference are in themselves sufficient for an extended discussion. But I trust I have been able to show the wide field which is open for exact scientific investigation. The necessary urgency of the case has compelled much of the experimental work done in recent years with regard to the purification of sewage to be largely empirical in its character.

The sewage problem, like any other scientific or practical question can only be solved by the patient and thorough elucidation of facts.

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[41] Spitta. *Archiv für Hygiene*, Band xlvi.

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Vaccination:

Its Pathology and Practice.

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Vaccination: Its Pathology and Practice.

Introduction.

As a preliminary to the consideration of questions relating to the practice and pathology of Vaccination it is perhaps desirable to define as accurately as possible what, at the present time, we understand by the term vaccination in the Jennerian sense. This is the more necessary seeing that of recent years, in consequence of the writings of Pasteur and his pupils, the word has come to be commonly employed in a wide sense to signify the inoculation of infective material of any kind. But in its narrower significance, as introduced by Jenner, vaccination may be defined as *the term originally devised for a method of protective inoculation against small-pox, consisting in the intentional transference to the human subject of a disease of milch-cattle, called cow-pox.* In similar fashion, to the serous fluid containing the specific virus of vaccinia which he obtained from vesicles on the teats or udders of cows suffering from this disease, Jenner gave the name of "Vaccine-lymph."

Consideration of the pathological questions raised by the wording of my definition, I will, however, defer to a later section of my paper.

VACCINATION.

Historical Remarks: Legislation and Administration.

As is, of course, well known, the introduction of Vaccination dates from the publication, in 1798, of Dr. Edward Jenner's historic pamphlet, the original draft of which had shortly before been rejected by the Royal Society. It is doubtless true that Dr. Jenner had been to some small extent anticipated by Jesty and others in the employment of the virus of cow-pox as a prophylactic against small-pox, but he it was who first realised the possibility of carrying on the disease through a series of human beings, by transferring the contents of the vaccine vesicles from arm to arm, and who, by his strenuous advocacy of the methods introduced by himself, first brought the matter prominently before the public. Early in the following year (1799) an extensive series of public vaccinations were initiated in London, mainly as the result of which Dr. Jenner was enabled to state, in 1801, that upwards of 6,000 persons had been inoculated with the virus of cow-pox, and that the far greater part of them had since been inoculated with that of small-pox, and exposed to its infection in every rational way that could be devised, but without effect. For this rapid extension of the practice of Vaccination Dr. Jenner was not a little indebted to the activity and enthusiasm of Dr. Pearson of St. George's Hospital, and Dr. Woodville of the Small-pox Hospital, who, when Jenner's stock of original lymph had come to an end, and opportunity for replenishing it did not immediately offer, were enabled, as the result of their discoveries of cow-pox outbreaks in various parts of London, to start independent series of vaccinations. In 1802 Dr. Jenner's

discovery was brought to the notice of the Legislature, with the result that a Committee of the House of Commons, after examining a number of witnesses eminent in the profession, issued a report entirely corroborative of Dr. Jenner's statement. In 1806, again, in consequence of an address to the King, voted by the House of Commons, the Royal College of Physicians of London were instructed to inquire into the matter. Concerning the outcome of this inquiry, which extended over some nine months, it is stated in the Appendix to the Report of the Select Committee on Vaccination of 1871, that "the College of Physicians felt it their duty strongly to recommend the practice of vaccination They have been led to this conclusion by no preconceived opinion, but by the most unbiassed judgment, formed from an irresistible weight of evidence that has been laid before them." Sir John Simon has placed it on record that in consequence of this report, which was presented to the House of Commons in July, 1807, "the public mind was apparently quite satisfied on the subject, and from this period begins to date the almost universal vaccination of children of the educated classes in this country."

In 1809 the so-called National Vaccine Establishment was founded, of which Dr. Jenner was at first appointed Director, although he shortly after resigned the post. From this period onwards, in spite of a certain amount of opposition, the practice of vaccination became gradually more popular. But notwithstanding the fact that the value of the operation received ample recognition from Parliament, by whom its discoverer was substantially rewarded, it was not until the year 1840, in which the first Vaccination Act was passed, that the subject was dealt with by legislative enactment. By this Act, which was to some extent amended in the following year, every inhabitant of England and Wales was afforded the opportunity of obtaining vaccination at the public cost, although the question as to whether any person availed himself of its advantages or not was left entirely to his own option. Another important provision of this Act was that the operation of small-pox inoculation, which prior to the introduction of vaccination had obtained a considerable hold on the populace, was now prohibited, the transference of small-pox virus from one person to another being made a penal offence. Not until thirteen years later, in 1853, did vaccination become compulsory. The Act passed in this year made provision for the establishment of stations in each of the districts into which guardians and overseers were required to divide their unions and parishes in order to afford increased facilities for vaccination of the poorer classes. At these stations medical officers were to attend to perform the operation, and again for inspection of the result; and parents or others having charge of children, failing to cause them to be vaccinated, or, subsequently to vaccination, omitting to have them taken for inspection, rendered themselves liable to a penalty. Certain further enactments, to which specific reference is unnecessary, came into force in 1858 and 1861. Six years later, in consequence of certain difficulties having arisen, more particularly in regard to the enforcement of penalties, a Bill to consolidate and amend the law relating to vaccination was brought before the House of Commons. This, after reference to Select Committee of both Houses, was passed, and received the Royal assent on August 12th, 1867. Hardly, however, had it become law before strenuous opposition to its provisions was raised, Parliament being eventually petitioned to repeal it. Under these circumstances

a Select Committee, to which reference has already been made, was appointed in the session of 1871 to inquire into the operation of the Act. As the result of careful consideration of the evidence brought before them, the Committee reported that in view of the great, though probably not absolute protection afforded by vaccination against attacks of small-pox, together with the almost absolute protection against death from that disease, it was the duty of the State to endeavour to secure the careful vaccination of the whole community. They were further of opinion, however, that multiple penalties should not be imposed in the case of the same child. These suggestions were embodied in a Bill which passed the House of Commons, and after amendment in the Upper House, involving some disagreement between the two legislative assemblies, became law in 1871. By this Act the Local Government Board were substituted for the Poor Law Board and the Lords of Her Majesty's Privy Council in the administration of the Vaccination Acts, and the powers of the Board were extended. Under the provisions of this Act and a further Act of 1874, the Local Government Board, in October of the latter year, issued an Order regulating the appointment, tenure of office, duties, and remuneration of vaccination officers, and also the institution and conduct of proceedings by them. In February, 1887, an Order dealing with the duties of public vaccinators was issued by the Board, and in February of the following year (1888) yet another Order altered the age limits at which re-vaccinations could be performed at the public expense.

Meanwhile, however, opposition to vaccination had been steadily increasing, and the administration of the Vaccination Acts became more and more difficult. Thus, although in the year 1872, the first year for which complete returns were made after the passing of the Act of 1871, the number of children in England and Wales whose vaccination was not finally accounted for amounted to 5·1 per cent. of the total number of births, and the same percentage was unaccounted for in 1883; subsequent to this date the percentage of cases not fully accounted for mounted gradually higher and higher.

It was under these circumstances that Mr. Ritchie, the then President of the Local Government Board, came to the conclusion that further investigation of the whole subject from every possible point of view was desirable, and accordingly in May, 1889, a Royal Commission was appointed, consisting of fifteen members, under the presidency of the late Lord Herschell. The terms of reference were as follows:—To inquire and report as to (1) the effect of vaccination in reducing the prevalence of and mortality from small-pox; (2) what means, other than vaccination, can be used for diminishing the prevalence of small-pox, and how far such means could be relied on in place of vaccination; (3) objections made to vaccination on the ground of injurious effects alleged to result therefrom, and the nature and extent of any injurious effects which do, in fact, so result; (4) whether any, and if so what means should be adopted for preventing or lessening the ill effects, if any, resulting from vaccination; and whether, and if so by what means, vaccination with animal vaccine should be further facilitated as a part of public vaccination; (5) whether any alterations should be made in the arrangements and proceedings for securing the performance of vaccination, and in particular in the provisions

of the Vaccination Acts with respect to prosecutions for non-compliance with the law.

After sittings extending over a period of six years the Royal Commission published their final Report in August, 1896. Once again the value of vaccination as a protection from small-pox was re-affirmed, the actual amount of such protection being declared to depend on the efficiency and thoroughness of the operation and its repetition after an interval of from seven to ten years. Injury following on the operation was declared to have been insignificant in the past, and under better precautions, which should be adopted in the future, likely to disappear completely. Certain suggestions also were put forward as to the best means of popularising vaccination, and as to alterations in regard to the infliction of penalties on defaulters, and the affording of a loophole to the "conscientious objector."

Purification and preservation of Vaccine-lymph.

Before this Royal Commission I had the honour of giving evidence, mainly in reference to the outcome of research work which I had been carrying on for several years previously. This work had dealt with methods of testing the potency of various kinds of lymph, whether of human or bovine origin, and their relative protective powers against the virus of small-pox, and more particularly with the question of the purification and preservation of vaccine lymph derived from the calf.

Years before, the occurrence of certain fatal cases of erysipelas, following on vaccination, in the practice of the then public vaccinator at Norwich, concerning the origin of which an official inquiry was held at the time, had, boy though I then was, made a great impression on me. When commencing work on the subject of vaccination, the remembrance of this unfortunate event caused me to turn my attention to the possibility of avoiding the accidental transference of erysipelas and other diseases from one child to another, in the operation of vaccination, by the substitution of some preparation of animal lymph for the arm-to-arm method of vaccination with human lymph, which, prior to the passing of the Vaccination Act of 1898, was the only method officially recognised outside the metropolis.

On making inquiry into the matter, I found that there prevailed very commonly an idea that the use of crude calf lymph was undesirable for the reason that it "took" more strongly than human lymph; that, in other words, more reaction and more generally "bad" arms were believed to be likely to result in cases vaccinated with lymph of bovine origin. In the course of a lengthy series of bacteriological inoculations of different culture media from various samples of vaccine lymph, carried out originally in the hope of isolating the specific contagium of vaccinia, I found that my culture tubes were apt to show abundant growth of micro-organisms which proved to be in no sense peculiar to vaccine lymph, consisting in part, indeed, of forms apparently identical with certain microbes commonly associated with suppurative processes. Moreover, a further point was noted that in plate cultures inoculated from specimens of fresh calf lymph the number of individual colonies which resulted was almost invariably much greater than in similar plate cultures, the

nutrient medium of which had been inoculated with an equal quantity of fresh human lymph. And it appeared to me not improbable that this fact might have relation to the observed tendency of calf lymph to "take" strongly, to which I have already referred, and might be due, in turn, to the greater difficulty in keeping clean the skin of the calf, as compared with that of a child. The further fact was also noted that when vaccine lymph of either bovine or human origin was stored for any length of time in capillary glass tubes, the opacity which usually resulted after a longer or shorter period (an occurrence usually associated with deterioration of the efficiency of the lymph for purposes of vaccination) was due, in large part at any rate, to an enormous multiplication of extraneous micro-organisms, which found in the serum of which the liquid portion of the lymph consisted, a favourable medium for their continued growth and multiplication.

Glycerinated Lymph.

With the object of obtaining, if possible, inhibition of these extraneous micro-organisms without injuriously affecting the specific contagium of vaccinia, and thus providing myself with material of more hopeful nature for the purposes of my research, I carried out, some years ago, a series of experiments, which were first demonstrated to the International Congress of Hygiene in 1891, and subsequently to the Royal Commission. The results have since been set out fully in the Milroy Lectures for 1898. To these experiments, therefore, it is unnecessary for me on the present occasion to refer in detail. Suffice it to say that eventually the desired result was obtained by submitting the epithelial pulp of the vaccine vesicle, after careful trituration, to the continued action of a sterilised 50 per cent. watery solution of chemically pure glycerine for about four weeks, such treatment having the effect of at first inhibiting, and ultimately destroying altogether, the numerous extraneous micro-organisms originally present in the lymph material. Later it became apparent that such an emulsion, if preserved and stored under favourable circumstances, of which a low temperature and protection from light are of special importance, was usually capable of retaining its efficiency as vaccine unimpaired for considerable periods. Still further investigations proved that not only was it possible to kill off all the bacteria ordinarily to be found in crude lymph material, but also the streptococcus of erysipelas and the bacillus of tubercle, even when, for experimental purposes, these micro-organisms had been added in relatively huge quantities to the lymph pulp prior to glycerination. Thus was gradually elaborated the method of ensuring the bacteriological purification of vaccine lymph to which the Royal Commissioners make reference in their final report, and which, as the result of further investigations carried out at their suggestion on behalf of the Government, has now been officially adopted in this country for the purposes of public vaccination.

National Vaccine Establishment and Public Vaccination.

In order to make adequate provision for the manufacture of glycerinated lymph on a sufficiently large scale it became necessary to revise and largely augment the arrangements previously in force under the auspices of the

Government. Thus the **Animal Vaccine Establishment** in Lamb's Conduit Street, which had been founded in 1881 under the direction of the late Sir George Buchanan and Dr. Cory, mainly for the performance of vaccinations direct from calf to arm, was entirely renovated in order to comply with modern aseptic requirements, and provision was made for the accommodation of a largely increased number of calves. Also additional premises were leased of the British (now the Lister) Institute of Preventive Medicine, and what are now known as the **Government Lymph Laboratories** were founded.

My late official chief, Sir Richard Thorne, and I, had previously, at the instance of the Government, made a tour of inspection of the principal vaccine establishments on the Continent, more especially those under Government control in Germany, where we found that the methods devised by myself had already been adopted. As the outcome of the practical experience thus acquired, supplemented by the results of a somewhat similar, although less extended series of visits made by Dr. F. R. Blaxall, subsequent to his appointment as Bacteriologist to the Department, the laboratories were equipped so far as possible with everything in the way of apparatus and accessories that appeared likely to prove useful in commencing operations on a large scale. Exactly how large that scale was to prove, however, we hardly realised at the time, and very considerable augmentation of the modest staff with which the work was commenced, and large expenditure for the installation of additional instruments, have been found necessary in order to ensure the enormous demand for lymph being met as promptly and efficiently as is now invariably the case.

Each public vaccinator receives, in response to application made to the National Vaccine Establishment, a consignment of lymph, together with a schedule in which to record the results of its use, and these schedules, after having been examined at the National Vaccine Establishment, are sent to the laboratories. The schedules indicate the series number of the lymph, the date of its despatch from the National Vaccine Establishment, the name of the public vaccinator to whom it was supplied, the number of tubes sent, the dates when the several tubes were used, the number of persons vaccinated, the number of scarifications made, and the number of vesicles obtained. All these details are recorded at the laboratories, and from the last two items information as to the success which has resulted, both as regards individuals vaccinated and insertions of lymph made, is obtained and set forth, both in full and in the form of a percentage. In addition to these records a register is kept stating the particulars of the calves employed, the details of the lymph obtained from each calf, including the results of the bacteriological examinations, the results of the use of the lymph at the Animal Vaccine Establishment, and also the number of tubes of each series despatched to the National Vaccine Establishment.

Statistical Returns.

During the first year of operations nearly 500,000 tubes of glycerinated lymph were sent out from the Government Laboratories. Notwithstanding the difficulties that had naturally to be overcome in the inauguration of work of a character entirely new to practically all those engaged upon it, the success attending the use of the lymph at the hands of public vaccinators throughout the country was distinctly gratifying, the returns made by them to the National

Vaccine Establishment showing that a case success of 93 per cent. and an insertion success of 83 per cent. had been attained. With a particular lymph series concerning which it became necessary to make special inquiry, and which had been distributed to 160 public vaccinators, the case success and insertion success were found to be 98 per cent. and 93 per cent. respectively. And at the present time it is by no means an unusual experience at the laboratories for returns showing complete case and insertion success to be received.

Effects of the Vaccination Act, 1898.

Under the provisions of the Vaccination Act, 1898, which came into force in January, 1899, for a period of five years, a period which has recently been extended for another year, and of the Vaccination Order (1898) of the Local Government Board, numerous changes in connection with vaccination administration and with the performance of the operation were introduced, in addition to the supersession of arm-to-arm vaccination by the use of glycerinated calf lymph. Thus, whereas by the Vaccination Acts of 1867 and 1871 the parent or person having the custody of any child was required to procure its vaccination within three months of birth, this period, by the Act of 1898, has been extended to six months. Again, no parent is now liable to a penalty under the compulsory clauses of the Vaccination Act who affords proof that he has, within four months of the birth of a child, satisfied a stipendiary magistrate or two justices in petty sessions that he conscientiously believes that vaccination would be prejudicial to the health of the child. Moreover in no case can proceedings now be taken more than twice against a defaulting parent, namely, once under Section 29 of the Act of 1867, and once under Section 31 of the same Act, provided that the child has reached the age of four years. When first propounded, the so-called "Conscience Clause" was received with considerable opposition, ; but it is now, I think, generally admitted that the cause has jusiified its existence, since the operation has practically done away with "martyrdom," and so has weakened to no slight extent one of the principal weapons in the armoury of the anti-vaccinators.

Operative Procedure.

The operative procedure in public vaccinations was formerly based on the necessity of carrying on a weekly series of transferences of vaccine lymph from arm to arm, this method having been originally introduced as the best means then attainable of insuring the activity and comparative purity of the lymph. In large urban districts, therefore, vaccinations were performed week by week throughout the year, while in small towns and rural districts quarterly or half-yearly periods, each comprising several weeks, usually sufficed. In the latter cases material for starting the series was generally obtained by vaccinating one or more infants a week previously, and if fresh lymph for this purpose could not be obtained from a private case or through the good offices of a neighbouring practitioner, a few doses of human or calf lymph, stored in tubes or on points respectively, were provided on application being made to the National Vaccine Establishment.

Again, for the purposes of arm-to-arm vaccination the provision of stations to which children were brought, first for the performance of the operation, and again after a week's interval for inspection of the results, was an essential. The occasional hardships to the mothers and a somewhat remote possibility of danger to the children involved in being taken long journeys to a vaccination station in bad weather, or arising from the collecting together in one room of a number of children and adults, one or more of whom might happen to be suffering at the time from some infectious disorder, are a few of the reasons which appeared to render a change in this regulation desirable; as a matter of fact, it would appear that nothing but good has arisen from the substitution of domiciliary for stational vaccination, coupled as it is with the use of glycerinated calf lymph, "or such other lymph as may be issued by the Local Government Board." It may here be mentioned, however, since the fact appears to be comparatively unknown, that it is not essential for a public vaccinator to employ the lymph issued by the Government unless the parent or person in charge of the child exercises his right of insisting on its use. But the public vaccinator "must not employ lymph supplied by any person who does not keep an exact record of its source," and in any case he "must keep such record of the lymph he uses for vaccinating," as will enable him always to identify the origin of the lymph used in each operation. Further, the operator is enjoined never, when he has unsealed a tube of lymph, to attempt to keep any part of its contents for the purposes of vaccination on a future occasion. Moreover, he is required to use an artificial blower for the purpose of expelling the lymph, instead of applying his mouth to the tube.

Two other sections of the "Instructions to Vaccinators under Contract" in the third schedule of the Vaccination Order of 1898 are of special importance. One of these refers to the aseptic precautions with which every stage of a vaccination should be carried out. "These should include (1) the cleansing of the surface of the skin before vaccination; (2) the use of sterilised instruments; and (3) the protection of the vaccinated surface against extraneous infection, both on the performance of the operation and on inspection of the results." The other section to which I would specially refer requires that, whenever possible, four separate good-sized vesicles, or groups of vesicles, not less than half-an-inch from one another, must be produced, and that in any case the total area of vesiculation resulting from the vaccination should not be less than half a square inch. As regards the standard thus set up and insisted on in the case of those vaccinators whose work comes under periodical inspection at the hands of the Medical Inspectors of the Local Government Board, it may at once be admitted that there is no special magic in the particular number "four." But an area of half a square inch having been recognised in the Report of the Royal Commission as probably the least extent of vesiculation, absorption from which is capable of affording adequate protection to the individual against subsequent invasion by small-pox, the regulation requires that this area should be distributed over four insertions, mainly for the reason that experience has shown that less inflammatory reaction and permanent destruction of skin tissue is likely to ensue than if the attempt be made to secure the same area by means of a less number of insertions of lymph.

Efficient Vaccination.

This leads us to the consideration of what constitutes "efficient" vaccination. The answer may be summarised as follows:—The clinical activity and bacteriological purity of the lymph employed for vaccination; the skilful performance of the operation itself; the making an adequate number of insertions of lymph over a sufficient area; the observance of precautions needful for ensuring strict asepsis, both at the time of vaccination and subsequently, until the vaccination wounds are soundly healed; all these are matters to be regarded as essential to "efficient vaccination." But, as has been well said by a writer in the *Edinburgh Review*, "even after efficient vaccination a slow progress away from safety and towards danger is inevitable, and re-vaccination at least once after childhood is necessary if protection is to be maintained."

As regards the carrying out of the operation itself, it is somewhat unfortunate that there exists no official definition of what constitutes a "successful vaccination," and in consequence it is open to any practitioner to give a certificate of successful vaccination in cases where but one minute vesicle may have been produced. It is to be feared that such certificates are too frequently given, and it cannot be too strongly urged that vaccination of this sort involves incomplete protection. The standard laid down by the Local Government Board, and to which reference has already been made, has for the most part proved easily attainable in practice, and it is much to be desired that in private as in public work the attainment of this standard should be aimed at in every instance. The suggestion has been made that, in the form employed in certifying to the success of a vaccination, the medical man performing the operation should be required to state the number of insertions of lymph made and the number of vesicles or groups of vesicles resulting therefrom. But it does not appear that the value of such a regulation would be very great in the absence of a further regulation requiring that all vaccinators, public and private alike, shall conform to a definite standard. The further suggestion has been made that every medical man should become a public vaccinator, to the extent that he should have the right of claiming a fee from public funds for every vaccination performed by him, provided that he was willing that his work should be subject to inspection on behalf of the Government. But the originators of this idea can hardly have realised the magnitude of the inspectorial staff that would be required if such an arrangement were to be put in force.

Preparation and Treatment of the Vaccinated Arm.

The treatment of the arm, at the time of vaccination and subsequently during the progress of the case, is another subject which has aroused considerable controversy, and concerning which much divergence of opinion would appear to exist. Thus, in some quarters, the initial cleansing of the arm is said to be objected to by the parents as a reflection on the care, or want of care, on their part, as regards the condition of their children; but in general it is found that a little tactfulness in explaining the difference between ordinary and surgical cleanliness has sufficed to overcome the difficulty. In addition to this aspect of the case the friction employed in the process is of value in causing

a slight capillary dilatation which undoubtedly contributes to the success of the operation. Water, soap and water, spirits of wine, or antiseptic solutions, of greater or less potency, containing boric or carbolic acid, lysol or perchloride of mercury, for instance, are employed by different operators for the purpose, of which, in all probability, a warm solution of boric acid is the most generally useful—a stronger antiseptic, such as corrosive sublimate, unless removed by the subsequent use of sterilised water or alcohol, being liable to exert a somewhat deleterious effect upon the lymph.

The method to be employed at the operation and during the maturation of the vesicles for the protection of the vaccinated area from extraneous infection has not been defined by the regulations, for the reason that it appeared probable that each man would best attain the desired end by the same methods that he would ordinarily employ in the treatment of any other case of minor surgical injury. As was to be expected, therefore, the means adopted for the protection of the vaccination wounds have been very various, and different trade firms have undoubtedly reaped an extensive harvest by the introduction and energetic advertisement of special dressings of one and another kind.

At the Government Station in Lamb's Conduit Street a dressing composed of a couple of layers of boric lint, kept in place by means of pieces of rubber strapping which do not entirely encircle the arm, is applied at the time of vaccination, and this is replaced by another exactly similar dressing when, a week later, the case returns for inspection of the result. But, whatever be the nature of the dressing, the free use beneath it of a dusting powder of boric acid has a most beneficial effect in preventing any undue amount of inflammatory reaction.

Operation.

Concerning the nature of the instrument best adapted for the purpose of vaccination I need only say that each operator will probably attain the greatest measure of success with that instrument to the use of which he has been accustomed. But, speaking generally, the less complicated it be, the better. Again, it is desirable that it should be formed entirely of steel, so that it may be readily sterilised by boiling or by heating to redness in the flame of a spirit lamp, the first method being preferable as not tending to injure the temper of the metal.

The manner of operating which affords the most generally successful results consists in blowing out the lymph from the capillary tube in which it is stored on the surface of the skin at different points, the number and situation of which must correspond with those of the vesicles it is desired to obtain. The skin, put slightly on the stretch, is then gently scarified, *through each droplet of lymph*, with the needle or other instrument, first in one direction and then in another more or less at right angles to the first, the drawing of blood being avoided as far as possible. In this way the corium or superficial layer of the skin is thoroughly opened up and in some measure removed, and thus the emulsion is brought into intimate relation with the cells of the true skin beneath. Operating in this fashion, and employing lymph of normal potency, it is quite easy to obtain an area of vesiculation satisfying official requirements. Many operators,

I find, reverse the procedure somewhat, first making their scarification and then rubbing on the lymph. But whether it be that some of the minute scratches are thus closed up as the lymph is applied, or whatever else the reason, certain it is that the results obtained are usually by no means comparable with those following on the method of scarification *through* the beads of lymph previously dropped upon the skin.

The operation having been completed, it is well to avoid too great haste in applying the protective dressing, especially if this be of the nature of an absorbent pad, or if it be impregnated with some powerful germicide, as in the case of sal-alembroth wool. Some little time necessarily elapses before complete absorption of the vaccine emulsion and the exuded lymph has taken place, and, to ensure the best results, a little exercise of patience is essential. With the object of hastening this period of drying, I, some time ago, devised a method of removing the glycerine from the lymph emulsion, after such time as bacteriological tests showed that it had fulfilled its purpose of destroying extraneous micro-organisms, and replacing it with an equal amount of an inert fluid of similar specific gravity. But the more rapid drying of the vaccinated area thus obtained appeared hardly to compensate for the extra trouble involved in the special preparation of the lymph.

Vaccination Scars.

When every care has been taken to protect the arm during the progress of the vaccination, and to prevent the premature detachment of the crusts, the amount of permanent scarring of the skin which remains may be astonishingly slight. This is, I think, one of the results of our present methods of vaccination to which, as yet, attention has hardly been sufficiently directed, although in the future it is likely to prove a matter of considerable importance. There can be little doubt but that the huge and deep scars which not infrequently resulted from the vaccination of former years, were due, to some extent, to excessive destruction of skin tissue by micro-organisms other than that specific to vaccinia. If this be so, then it becomes apparent that persistence of such large and deep scars, practically throughout life, does not necessarily afford evidence that any equivalent degree of immunity against the infection of small-pox is enjoyed by their possessor.

Recent Statistical Returns.

The question naturally arises as to whether appreciable advantage to the community can be proved to have resulted from the adoption of our more modern methods of vaccination. It is for several reasons somewhat difficult to give, as yet, any very definite answer, although such statistics as are at present available are decidedly encouraging. There can be no doubt, as will be seen from the figures set out in the forthcoming Report of the Medical Officer to the Local Government Board, that the operation of the Vaccination Act of 1898 has been accompanied by a very considerable increase in the number of vaccinations performed, although it should be borne in mind that this increase was, in the earlier years, in some degree at any rate, to be accounted for by the vaccination of cases previously in default, a source from which it is unlikely that the figures can be continuously augmented. None the

less, however, it is matter for satisfaction that the vaccination of these outstanding cases should have been secured. In 1902, an epidemic of small-pox was accountable for a specially large amount of vaccination and re-vaccination.

From a return presented to the House of Commons on August 6th, 1900, we learn that in the year 1899, the first during which the Act of 1898 was in force, the number of certificates of successful primary vaccinations received showed an increase of 169,035, or no less than 33·8 per cent., over the figures for the previous year. Later returns exhibit continued and marked increase in the total number of certificates of successful primary vaccination, at all ages, as shown in the following table:—

1898	500,314
1899	669,349
1900	676,807
1901	710,785
1902	826,771

But there is another source from which we may learn something as to the value of modern methods of vaccination. I refer to the Annual Reports of the Registrar-General, under the heading "Deaths attributed to cow-pox and other effects of vaccination." For a series of years deaths thus registered averaged one every week for the whole of England and Wales, but whereas in 1889 and 1892 the actual number of deaths included under this heading was 58, and in 1893 it was 59, in 1900 the number was 25, while in 1901, the last year for which data are available, the figures had sunk still further to 17.

II.—ETIOLOGY OF VACCINIA AND VARIOLA.

Pathology of Vaccinia.

I may now revert to the pathological questions raised by my definition of "vaccination"; and, as a preliminary and without going into great detail, it may be well in the first place, to set out some account of the chief macroscopic appearances which are to be observed in the skin during the local development of the vaccine vesicles. As the experimental observations, by Dr. Gustav Mann and myself,* on which the histological portion of this account is based, were made, for obvious reasons, on material obtained from the calf, it should be borne in mind that in this animal the whole process of vaccination runs a distinctly shorter course than in the human being, a fact that is probably in some measure dependent on the normally higher body-temperature of the calf as compared with that of man. Thus, in the human subject the vaccine vesicle ordinarily attains maturity on the eighth day [(24 hours × 7) = 168 hours], while in the calf the vesicle will have attained a similar stage of development on the sixth day [(24 hours × 5) = 120 hours.)]

Although doubtless well known to all here present, I may, perhaps, in the first place, briefly note the varying naked-eye appearances of the vaccine vesicle. Within an hour after vaccination, more particularly in the calf, the skin immediately bordering on the inoculation wound not infrequently becomes

* Annual Report of the Medical Officer of the Local Government Board, 1898-99—Appendix C.

somewhat raised, owing to a transient local urticaria. This, however, rapidly passes off, and, in the human subject, practically no further change becomes obvious before the third day after the insertion of the vaccine lymph, by which time a small inflamed spot or "papule" may usually be observed at the point where the vaccination was performed. Next day this spot appears more florid, and on passing the point of the finger over it a certain degree of hardness and swelling is perceptible. By the fifth day the papule develops into a small, pale vesicle. This vesicle has a milky white colour, it is eventually somewhat depressed in the centre, and its edges are distinctly elevated above the level of the surrounding skin. As yet the vesicle has no inflammatory zone around it.

For the next two days the vesicle increases in size; assuming, if the vaccination was performed by the method of puncture, a circular form; if done by an incision, an oval shape. But in both cases the margin is regular and well defined. About the eighth day an inflammatory zone of a bright red colour, termed the "areola," begins to appear around the base of the vesicle; this increases in extent for two or perhaps three days more, by which time it may extend for about a couple of inches from the vesicle. The latter still retains its concave appearance, but a crust of a brownish colour will have commenced to form in the centre. By about the eleventh day the vesicle has attained its greatest magnitude, and the surrounding inflammation begins to abate. The fluid contained in the vesicle, or "pustule" as it is now called, which before was thin and transparent, becomes more viscid and somewhat turbid. After this period the whole becomes quickly converted into a smooth, shining, dry crust, of a dark brownish colour. This crust, unless forcibly removed, will adhere for a week or more and then fall off, leaving the skin beneath, apparently sound, but livid for a time, and afterwards more or less permanently scarred.

Histology of the Vaccine Vesicle.

During the evolution of the local changes which result from the insertion of vaccine lymph beneath the surface of the skin, it is possible, as previously indicated, to recognise three more or less definite stages of papule, vesicle, and pustule.

The same statement holds good with reference to the eruption of small-pox, whether this be local, *i.e.*, due to intentional inoculation of the virus on the skin; or general, as the result of casual infection, with the exception that the edge of the vesicle resulting from inoculation is apt to be less regular in outline than is that of the vaccine vesicle.

In each instance the appearance of the first or *papular* stage is brought about by inflammatory reaction, causing an increase of intercellular fluid, together with concomitant increase in volume and number of epithelial cells, of the *rete Malpighii*, more particularly. The papule gradually becomes enlarged by a circumferential extension of the same process, and owing to further changes in the cells first affected, vacuoles arise in the central portion of the papule, by the extension of which this ultimately becomes a vesicle.

The *vesicle* is a multilocular structure, the dissepiments, by means of which its interior is divided up, being formed from the thinned and extended remains of the original epithelial cells. Owing to the fact that the process of vacuolation

increases, for a time, more extensively at the advancing edge of the vesicle, the central portion remains somewhat less elevated, thus giving rise to the appearance termed "umbilication."

At a somewhat early stage of the process, an outflow of leucocytes takes place towards the point of injury. In time, each blood-vessel becomes the centre of an aggregation of leucocytes, which, by the rapid increase in their numbers, eventually transform the originally clear inflammatory exudation into a purulent fluid. The vesicle is said now to have become converted into a *pustule*.

By the thinning and ultimate rupture of its tubercle the pustule finally becomes unilocular. The turbid fluid contained in it now gradually dries up, and, together with the necrosed remains of epidermal cells, takes part in the formation of the *crust*, which, under the microscope, appears as a homogeneous mass which takes an intense colouration when treated with the ordinary laboratory stains.

Meanwhile a regeneration goes on underneath the crust, the new epidermis being formed by an ingrowth from the surrounding *stratum lucidum*. The extent to which the *cutis vera* has been involved determines the depth of the resulting scar.

Bacteriology.

The bacteriology of the vaccine vesicle, is a question concerning which on the present occasion nothing more than a short resumé can be attempted owing to the immense amount of work on the subject which has been published even within recent years.

It may be stated, however, that the evolution and development of the vesicle is usually accompanied by the appearance of vast numbers of bacteria of one and another kind, the ancestors of which were probably, to some extent at any rate, introduced beneath the surface of the skin at the time of performance of the vaccination, since the purer the lymph and the greater the antiseptic precautions observed in connection with the operation, the less as a rule will be the number of bacteria in the resulting vesicle.

During the year 1897, I carried out, in conjunction with Dr. Blaxall, an exhaustive series of investigations on the bacterial flora of calf vaccine, with the outcome that, as the results of former work had led us to anticipate, and as has been repeatedly shown by other observers, calf lymph (or rather, vesicle pulp) may contain a large number of micro-organisms which are in no way concerned with its specific activity and to which, for this reason, I originally applied the term "extraneous." Those species which are most commonly met with are, morphologically and culturally, identical with the staphylococci found in pus, viz., the staphylococcus pyogenes aureas, albus, and cereus flavus. Occasionally the streptococcus of pus may be present as also the staphylococcus citreus. It should be stated, however, that the fact that in vaccine lymph micro-organisms are found, which, under certain circumstances, are known to be provocative of suppuration, does not in any way imply that the lymph in which they occur is purulent, or that, if such staphylococcus-infected lymph were employed for vaccination of the human being, suppuration would necessarily ensue.

Other microbes of a purely saprophytic nature that are met with, include the common hay bacillus, bacillus mesentericus, varieties of proteus, yeasts, moulds, and sarcinæ. These, however, can for the most part be excluded by careful manipulation, so that in vesicle pulp, which has been collected with all due precautions, nothing is usually found beyond some one or more of the four first-named staphylococci, of which a white staphylococcus is perhaps most common, and possibly one or more yeasts.

To the method of glycerination of the lymph, by the employment of which it is freed, prior to use in vaccination, from these "extraneous" micro-organisms, I have referred in an earlier section of this paper, but I may here refer to an improvement on my original method, devised by Dr. Green,* and consisting in the use of chloroform vapour, by means of which the process of purification is much hastened; thus rendering it possible to cope with sudden demands for large quantities of vaccine lymph such as are likely to occur at times of small-pox prevalence. But in ordinary *routine* work of lymph-production the extra labour involved in the use of the chloroform method would appear to afford hardly commensurate advantage.

Discovery of a Bacillus possibly the cause of Vaccinia and Variola.

In 1894 I showed, concurrently with Dr. Klein, though independently, that in specially stained specimens of vaccine lymph taken at a period antecedent to full maturity of the vesicles, the presence of bacilli of extremely small size and in practically pure cultures can be demonstrated. These bacilli cannot be found, or only with difficulty, in mature lymph, for the reason, probably, that they have, by then, given place to spores. For some time it appeared impossible to obtain further evidence as to the rôle of these bacilli, as they altogether refused to grow on any of the ordinary culture media, and under either aërobic or anaërobic conditions, though this very fact obviously tends to show that they are not of a merely saprophytic nature.

Cultures of these small bacilli, as also of apparently quite similar ones from small-pox material, were eventually obtained, in some few out of a large number of experiments, by inoculation into hens' eggs, which were subsequently incubated at a temperature of 37°C. for periods ranging from a fortnight to a month. Such cultures, however, almost invariably failed.

Still, as recorded at the time, I was able in some instances to successfully inoculate calves with the contents of these eggs, and ultimately lymph, taken from vesicles at the third remove from the egg-culture, was successfully employed for the vaccination of a large number of children. For these reasons I came to the conclusion that the bacilli found by Klein and myself were, in all probability, specific to vaccinia and variola respectively, although for various reasons into which it is needless to enter here, my work in this direction can hardly be regarded as conclusive.

* Proceedings of the Royal Society, Vol. lxxii., pp. 1—4, and Vol. lxxiii., pp. 342—346.

III.—RELATION OF VARIOLA AND VACCINIA.

Previous researches.

It is evident from Jenner's writings that he believed vaccinia to be nothing more nor less than "small-pox of the cow," but his theory would appear not to have received general support even in his own day. And from this time onwards the value of the practice of vaccination has now and again been impugned by certain persons, on the plea that inoculation of one disease—"cow-pox"—could not be expected to exert any really protective influence against the ravages of small-pox, a disease supposed by them to be of totally different origin. And if the thesis of essential difference between these maladies were capable of demonstration, no doubt the objection would be of considerable weight, and *vice versâ*. Accordingly, during the long period which has now elapsed since the introduction of vaccination, many observers have set themselves the task of attempting, by experimental methods, to solve the problems of the true relationship of variola to vaccina. These attempts have, almost without exception, been directed to the possibility of giving rise to cow-pox by the introduction, in one or another manner, of the virus of small-pox into the system of the bovine animal. In the great majority of such attempts, which have been vastly more numerous than is generally supposed, the results have been entirely negative, although from time to time apparently successful results have been recorded. It is impossible, on the present occasion, to describe in detail the various series of inoculation experiments to which I refer, and indeed it is unnecessary since they are to be found admirably epitomized in the Final Report of the last Royal Commission on Vaccination, to which, or (for a more detailed account) to my Milroy Lectures, delivered before the Royal College of Physicians in 1898, and since published in book form, I would refer anyone desiring precise information as to the actual methods employed and the results obtained.

Some years ago, Sir Michael Foster suggested to me that in view of possible deterioration of lymph-stocks, after employment through a long series of generations, it would be well, if possible, to devise some practicable means of investigating the efficiency of the protection afforded by some of those, then in use, against the infection of variola.

Author's researches.

The inoculation of small-pox on the human subject having been made a penal offence by the Vaccination Act of 1840, I bethought myself of the monkey tribe as possibly, on account of their similarity in many respects to man, being available for experimental purposes, although at the time I was assured, on high authority, that they were not susceptible to either vaccinia or variola. On putting the matter to the test, however, I was agreeably surprised to find that this was not the case, inoculations of both vaccine and variolous lymph having each of them given, in my hands, successful results in every instance in which I have tried them on the monkey.

Experiments on Monkeys.

The method of operation which I adopted in the first instance was precisely similar to that usually adopted in the case of a vaccination on the human being, the seats of election in the monkey being either the upper arm or the inner surface of the thigh; the skin in either case having first been shaved to free it from hair. In the case of more recent experiments, however, the operation has been carried out by means of parallel incisions on the skin of the back, between the shoulder blades, where the monkey is least able to interfere, by scratching, with the normal evolution of the eruption experimentally produced.

The chief difference noted between the effects resulting from the local inoculation of these two diseases is that in the case of variola, vesiculation is usually less marked than in vaccination; that about the ninth to the eleventh day a general eruption may appear, which, in some few instances, has covered almost the whole surface of the body; and that the final "crust" at the site of inoculation is not so elevated in the variolated as in the vaccinated animal.

Having thus proved to my own satisfaction that monkeys are susceptible not only to vaccination but also to small-pox, I next determined to make trial as to the protection against small-pox afforded in the monkey by previous vaccination, and the protection against vaccination afforded it by variolation, and I went on to compare the effect produced by the use of human and calf vaccine respectively.

Without entering into details of the numerous experiments which were performed at intervals of one to five months after the primary operation, whether vaccinal or variolous, I may state that in no instance did anything in the nature of a successful result follow the first or subsequent re-inoculations. From these experiments it would appear therefore that the protective power of lymph obtained from these three different sources, when inoculated on the monkey, so far as can be judged from the necessarily limited duration of the experiments, is practically identical in all respects.

These experiments having demonstrated to my satisfaction that the monkey was available as a control in place of the human being, I realised that it would be possible to put to the test a point that had occurred to me some time previously, and which involved a modification of previous attempts at conversion of human small-pox into cow-pox.

Experiments on Calves and Monkeys.

I have already referred to the considerable difficulty experienced by myself and numerous other investigators in endeavouring to transmit human small-pox directly to bovines, whether cows or calves, and to the fact that this acknowledged difficulty has from time to time been cited with the object of discrediting the theory expounded by Jenner, that cow-pox, whether carried through the horse as intermediary or not, was originally derived from small-pox in the human being.

But a great deal, at any rate, of the small-pox which was prevalent at the time that Jenner lived and wrote, was of that comparatively mild variety which, under the name of inoculated small-pox, was intentionally produced in healthy

subjects, with the object of thereby conferring protection against subsequent attack by the disease in virulent form. So mild, indeed, at times, were the results of inoculations in the hands of such operators as Adams and the brothers Sutton that, as we learn from contemporary records, in many instances but little obvious effect was observed, with the exception of the local vesicle arising at the site of insertion of the small-pox virus, and the patients suffered but little inconvenience. Thus, more particularly in certain of Adams' cases, as may be gathered from his own account of the circumstances, the visible effect produced so closely resembled the results then beginning to be known as following on the Jennerian process of vaccination, that numbers of his patients were with difficulty persuaded that he had not, contrary to their desire, intentionally vaccinated rather than variolated them. The gradual evolution of a strain of lymph of such tenuity, according to Adams himself, was obtained by attention to the mode of life and general treatment of persons undergoing the process, together with careful selection of the "source" (preferably the "primary vesicle") from which the virus was obtained.

The majority of persons thus inoculated are not likely to have been incapacitated as the result of the operation, to a much greater extent than are those who undergo efficient vaccination at the present day, and, doubtless, therefore, they would be, for the most part, capable of following their ordinary avocations during the process of the induced disorder. On the other hand, this would have hardly been possible in the case of persons contracting small-pox in the ordinary way, among whom the disease was apt to exhibit such virulence as to account for the death of perhaps 50 per cent. of those attacked.

Not only were the effects following on inoculation comparatively mild, but the disease, in this form, was intentionally brought into many country districts which otherwise might not have become invaded by small-pox. In the light of these facts, it had for some time past been borne in to my mind more and more convincingly that it was probably from the *inoculated* form of small-pox, rather than from the ordinary variety of the malady, that much, at any rate, of the cow-pox, in the pre-vaccination era was derived. It is not difficult to understand how the cracks so often found on the udders of cows might become infected by a milker with fingers contaminated by contact with the inoculation sore upon his arm.

I determined, therefore, if possible, to put the matter to the test, and learning that in Nubia, in Burmah, and in certain parts of India, the inoculation of small-pox is still practised, I made numerous endeavours to obtain the necessary material, but unfortunately without success.

In default, therefore, of inoculated small-pox in the human subject, I made trial of the monkey, which, as I had previously shown is readily susceptible to the disease, the various phases of which in this animal closely resemble those observed in man, but in a milder form; the occurrence of a generalised eruption being exceptional.

The different series of experiments, a complete record of which has been published elsewhere,* were carried out at intervals, determined mainly by the possibility of procuring the necessary small-pox material. For the various

* Proceedings of the Royal Society, Vol. lxxi., pp. 121—133 and 190.

supplies received I am indebted to the Medical Officers of Health of Middlesborough and Glasgow, and to the Medical Superintendents of the Small-pox Hospital at Glasgow, of the West Ham Small-pox Hospital at Dagenham, near London, and of the Hospital Ships of the Metropolitan Asylums Board.

The most satisfactory material was found to be vesicle pulp obtained in the *post-mortem* room from cases of discrete small-pox that had died during a comparatively early stage of the eruption. This pulp was triturated with dilute glycerine after the fashion employed in making glycerinated vaccine lymph, and the resulting emulsion was found to remain active often for considerable periods. Inoculation of this specially prepared small-pox lymph was usually carried out, as previously mentioned, by means of linear incisions on a previously shaved and cleansed area of the back of a monkey, between the shoulder blades.

After two, three or more passages through the monkey the now strictly localised disease was transferred to the skin of the calf, further transferences from calf to calf to the number of two, three or even more, being usually required before the most typical vesiculation was obtained.

The results of my experiments may be briefly summarised as follows:—

In each of four separate series of experiments the human small-pox lymph or pulp was first inoculated directly on calves, and, in every instance, so far as could be observed, with altogether negative results. But with monkeys, success was as invariably obtained, and when, after one or more passages through this animal, the contents of the local inoculation vesicles were employed for insertion on the calf, an effect was now produced which, after two or three removes in that animal, was indistinguishable from typical vaccinia.

Vaccinia derived from Variola after passage through Monkeys.

Moreover, from the contents of vesicles raised in this manner on the calf, a number of children have, in turn, been vaccinated, some of whom were afterwards kept under observation for as long a period as a couple of months.

Every such vaccination "took" normally, and in no case was any bad result subsequently observed by myself or reported by the parents of the children; no "generalization" of the eruption occurring in any instance. But none of the strains of vaccine lymph derived originally from human small-pox in the manner described have been brought into general use.

My first series of experiments had not long been concluded when I learnt of similar work which had been carried out in Batavia by Dr. Eilerts de Haan.* This proved of special interest to me, for the reason that, quite independently, we have been able to corroborate one another's work, except as regards the transference of the strain of variola-vaccine to the human subject—a final test which Dr. de Hann did not, as he says, feel justified in attempting, in view of the unfortunate experience of Chauveau in connection with his abortive attempts at variolation of the cow.

In conclusion I desire to call attention to the somewhat remarkable fact that a mild and strictly localised form of small-pox, such as is induced in the

* *Centralblatt für Bakteriologie*," March 28th, 1899, p. 380, *et seq.*

monkey by the inoculation of material from cases of the generalised disease in man, should, when transferred to the calf, "take" readily with the production of a vesicular eruption of non-infectious character in that animal, whereas it is well known that successful transference of small-pox, direct from man to calf can only be accomplished with the utmost difficulty.

The experimental results obtained in the course of this research all tend, then, to confirm the view that the vaccinia of Jenner's time was derived, in all probability, from a comparatively mild form of human small-pox.

In addition, I think it will be admitted that the work has afforded conclusive evidence of the essential identity of the virus of small-pox and cow-pox or vaccinia.

On the Construction of Life-Tables, and on their Application to a Comparison of the Mortality from Phthisis in England and Wales, during the decennia 1881-90 and 1891-1900.

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On the Construction of Life-Tables, and on Their Application to a Comparison of the Mortality from Phthisis in England and Wales, during the decennia 1881-90 and 1891-1900.

Introduction.

The subject-matter of this Lecture, which is honoured by being accorded a place in the series of "Advanced Lectures on Public Health," inaugurated under an ægis so distinguished as that of the Victoria University of Manchester, can scarcely, in the nature of things, compete in general interest or practical usefulness with most of the other Lectures of the series.

Yet, however distasteful descriptions and explanations of statistical methods may be, it must at least be conceded that without statistics the science of Public Health would scarcely have been founded, and that without them it would be scarcely possible to measure its progress.

Without further preface or apology the present lecturer may be permitted to proceed with the task set before him, conscious, however, that the subject is one which lends itself to addressing *readers* rather than *hearers*.

From the title of the Lecture it will appear that it may be divided into two parts:—

1. An attempted description and explanation of some technical points relating to the construction of Life-Tables.

2. A demonstration of the application of Life-Tables, among other methods, to comparing the mortality from some special cause, such as phthisis, in two successive decennia.

It may be that the results set forth in Part II. will interest many who do not care to grapple with the explanations given in Part I. as to *how* these results have been obtained.

PART I.—ON THE CONSTRUCTION OF LIFE-TABLES.

This course of Lectures being described as "advanced," some degree of knowledge must be assumed to be possessed by those addressed, as to what a Life-Table is, and how its different columns are built up from the foundation numbers of population and deaths. Moreover, the available limits of space and time would hardly suffice for a successful attempt to lead a student, in one lecture, from the starting-point of complete ignorance to the position of full knowledge of the subject. It may be possible, however, as the different parts of the subject are being successively dealt with, for the Lecturer to give such references to what has been previously written by others or by himself, as shall suffice to amply supplement the necessary deficiencies of this lecture.

Construction of an Extended Life-Table. The problem of constructing an extended Life-Table practically resolves itself into calculating from the data the series of values tabulated under the heading of "the p_x column."

Now p_x simply means the chance, or probability, of surviving from age x to age $x+1$, and is expressed by the ratio,

$$\frac{\text{number living at end of } x^{\text{th}} \text{ year of age}}{\text{number living at beginning of } x^{\text{th}} \text{ year of age}}.$$

If the conditions assumed in the familiar illustration of a ledger account, starting with a certain number of infants born on the first day of January in a given year, and supposed to be made up yearly, could be realized, then, when the account, after a century or so, was closed, we should have the complete l_x and d_x columns of a Life-Table, and the p_x values could be calculated from these.

$$\text{Thus, } p_x = \frac{l_x - d_x}{l_x} = \frac{l_{x+1}}{l_x}.$$

On the other hand if we had given the p_x values, the l_x and d_x columns could be calculated thus:—

$$l_x \times p_x = l_{x+1}\,; \text{ and } l_x - l_{x+1} = d_x.$$

As it is practically impossible to construct a Life-Table by this method of *continuous* observation, a method has to be used which assumes that rates of mortality at different ages, which exist *simultaneously* during a limited period of observation, will come *successively* to affect a supposed generation of individuals from birth to the end of life.

In order to see how an absolutely perfect and accurate series of p_x values might be obtained from facts observed during a limited period of time, if the requisite conditions could be realized, let it be supposed that a census of the whole country had been taken at the middle of the year 1891, and that the age of every individual of the population had been accurately stated, also that the results had been correctly tabulated for each year of age.

We should then have what may be called a P_x column, P_x denoting the mean population for the year at age x to age $x+1$, who may be of any age at and over age x and under age $x+1$.

Let it be further supposed that the numbers and ages of those who had died during the same calendar year had been recorded with equal accuracy for each year of age, thus giving a d_x column.

(In order to avoid confusion of ideas it must be noted that the symbols P_x and d_x, as applied to a census population and registered deaths, must be distinguished from the same symbols as used in Life-Table terminology. In the latter case certain definite relations at successive ages are implied which do not exist in the former.

When used with reference to an actual census population and death records the letters P and d simply mean population and deaths, some indication being always added of the ages to which they relate, but in a Life-Table, P_x and d_x refer to population and deaths in a single year of age, and another letter Q is introduced to denote population in a group of ages or the total population at any age and upwards.)

When dealing with sufficiently large numbers, such as those in the case supposed would be, it would be found (1) that if the average age of all the individuals included in each of the P_x groups were reckoned up, this would not for any year of age, except the first, differ very much from $x+\frac{1}{2}$; (2) that the average age of each of the d_x groups, except the first, would not be far from $x+\frac{1}{2}$; and (3) that apart from accidental disturbing influences, such as epidemics, the numbers of deaths would be nearly equally divided between the two halves of the year.

Such being the case, it is easily seen that we may take l_x, that is the number living at the beginning of the year 1891 at exact age x, as represented by $P_x+\frac{1}{2}d_x$, and l_{x+1}, that is the number living at the end of the year 1891 at exact age $x+1$, as represented by $P_x-\frac{1}{2}d_x$, and therefore.

$$p_x=\frac{P_x-\frac{1}{2}d_x}{P_x+\frac{1}{2}d_x}=\frac{2P_x-d_x}{2P_x+d_x}.$$

If we may further suppose that the same process of census taking and accurate tabulation of P_x and d_x columns had been repeated in each of the succeeding years of the decennium 1891—1900, then the mean results would serve to eliminate any disturbing influences in single years. Such an ideal mode of arriving at a series of p_x values is, of course, scarcely within the possibility of practical realization, but although unattainable, there are certain characteristics which we may almost certainly assume that a series of p_x values thus obtained would possess.

1. When plotted out to scale on paper ruled into squares the series would become visible as points in a continuous curve ("the p_x curve") without any breaks or angularities, or in other words, the successive p_x values would change by gradual transitions.

2. Suppose the numerical expressions of the p_x values (or preferably their logarithms) to be set down in a column, and that they are "differenced" by successively changing the sign of the upper of each successive pair of values, and taking the algebraical sum of the pair, the results being set down in a parallel column, we should have "the first order of differences." If a similar

process be applied to this last-mentioned column, we should have "the second order of differences," and so on.

Let it be supposed that the process has been carried on until the last possible difference has been obtained. It is obvious that if we had given only the first term of the series, and the leading differences in a line with it, the original series of p_x values could be readily reconstructed, that is, it is as easy to obtain the terms from the differences as the differences from the terms.

To deal thus, however, with a continuous series of terms numbering a hundred or more would be a practicable impossibility. Suppose, therefore, that the whole series were to be split up into sections of ten or twenty each, and that the values for the first five years of age were eliminated. It would be found in each of the sections that the orders of differences after the first five or six became very small, so small that they might be neglected, that is to say that the original series of p_x values might be reconstructed, without any very material variation, by only taking into account five orders of differences.

When we turn from the *ideal* to the *actual* we find that the data practically available for calculating the series of p_x values are population and death numbers (1) not given for each separate year of age but in groups of ages, and (2) vitiated by mis-statements of age in the census returns and in the death registers. The latter point has simply to be mentioned and left.

The problem then is to so deal with the foundation numbers of population and deaths given in age-groups as to obtain a series of p_x values for each year of age, conforming as nearly as may be to the conditions already laid down for the ideal curve.

It is, of course, obvious that it is easy to obtain a series of values each one expressing the mean chance of surviving a year in an age-group x to $x+n$, from the total population and death numbers for the age-group, by the formula

$$p_{x \text{ to } x+n} = \frac{2P_{x \text{ to } x+n} - d_{x \text{ to } x+n}}{2P_{x \text{ to } x+n} + d_{x \text{ to } x+n}}.$$

(In order to avoid this somewhat cumbrous notation it is proposed from this point to use the symbol x/n instead of x to $x+n$. Thus the above formula may be written

$$p_{x/n} = \frac{2P_{x/n} - d_{x/n}}{2P_{x/n} + d_{x/n}}.$$

It must be understood that whatever x as a suffix means, x/n means that same thing for a group of persons beginning at age x and going on for n years beyond.

Thus p_x meaning the probability of people aged x living one year, $p_{x/n}$ would mean the probability of an n-year group, *beginning with age* x, living for one year, *not* the probability of persons aged x living for n years.

Similarly E_x, denoting the mean after-lifetime of persons aged x, $E_{x/n}$ would denote the average after lifetime of a number of persons included in a group of ages beginning with age x and going on for n years beyond).

If such a series of values be represented diagrammatically, instead of a continuous curve, we shall have a series of straight lines which, when their extremities are joined, will show step-like ascents or descents.

It will be obvious that any yearly series of p_x values deduced from the data in groups of ages should not only conform to the conditions already laid down for an ideal p_x curve, viz., that (1) when plotted out they must indicate a curve without breaks or angularities, and (2) when differenced they must show regular graduations of differences, such differences after the first few orders becoming very small, but also (3) the continuous curve must have some rational relation to the diagram of mean $p_{x/n}$ values.

Graphic Method. One mode of dividing up the numbers of population and deaths given in age-groups into numbers belonging to each separate year of age is the so-called "graphic" method employed by Milne in the construction of the Carlisle Life-Table, and (as well as by others) by Dr. A. Newsholme in the construction of his two Brighton Life-Tables.

For a full description of this method see Newsholme's "Vital Statistics," third edition, pp. 265—269, and a paper by Drs. Newsholme and Stevenson in "The Journal of Hygiene," Vol. iii., No. 3, July, 1903.

However, if the p_x curves taken from the Life-Tables which have been constructed in this way be tested by the methods previously indicated, there are none of them, with the exceptions of Dr. Newsholme's second Life-Table for Brighton, and Dr. Barclay's Life-Table for New Zealand, 1891—95, which give at all satisfactory results, and these latter have been made satisfactory by departures, to some extent, from the original method.

Method of "Finite Differences." The "graphic" method is *one* way of measuring, but there is another and *much more accurate* way of measuring, by employing interpolation by the method of "finite differences."

If anyone will take the trouble to set down a series of say six numbers and difference them by the method already indicated, and then proceed, from the first term of the series and the five differences in a line with it, to reconstruct the series of numbers originally given, the first step will have been taken towards comprehending what is meant by interpolation by the method of finite differences.

If he will then test the following three formulæ by the concrete numbers and differences before him a sufficient knowledge of the theory of interpolation will have been obtained. Let the six given numbers be denoted by the symbols u_0, u_1 . . . u_5, and the line of differences opposite to u_0 by the symbols δu_0, $\delta^2 u_0$ . . . $\delta^5 u_0$, then

$$(1) \qquad u_x = u_0 + x\delta u_0 + \frac{x(x-1)}{2}\delta^2 u_0 + \frac{x(x-1(x-2)}{2.3}\delta^3 u_0 + \ldots .$$

This formula follows the law of the Binomial Theorem. If it be expanded and reduced to its simplest form, it can be expressed by

$$(2) \qquad u_x = A(i.\ e.,\ u_0) + Bx + Cx^2 + Dx^3 + \ldots .$$

which means that each power of x has a *constant coefficient*, whatever the value of x.

The next formula is what is known as "Lagrange's formula," by which having given any series of numbers (linear quantities) denoted by the symbols u_0, u_a, u_b, u_c . . . .

$$(3) \qquad u_x = \frac{(x-a)(x-b)(x-c)\ldots}{(o-a)(o-b)(o-c\ldots}u_0 + \frac{(x-o)(x-b)(x-c)\ldots}{(a-o)(a-b)(a-c)\ldots}u_a + \ldots$$

Some elementary teaching as to interpolation is attempted in papers contributed to "Journal of Royal Statistical Society," Vol. lxii., part 3, pp. 454—464, and "Journal of Hygiene," Vol. ii., No. 1. Those who may desire to go thoroughly into the matter may consult Bowley's "Elements of Statistics," pp. 242—249, and the chapters entitled "Formulas of Finite Differences," and "Interpolation," in the "Institute of Actuaries' Text-Book," by George King, F.I.A., etc.

To return to the point at which it was supposed that perfectly accurate P_x and d_x numbers had been obtained for each year of age by exact census enumeration and registration of deaths. Let it be supposed that the compilers of these numbers had by some freak first of all for each year of age put the numbers in the form of two columns, giving respectively twice population minus deaths, and twice population plus deaths, and then, assuming the last age to have been 100, had added the values in each of these columns for age 99 to those for age 100, and then the values for age 98 to the sum of those for ages 100 and 99, and so on, and then had finally published the figures in two columns giving the numbers of twice population minus deaths $(2P-d)$, and twice population plus deaths $(2P+d)$, *at each age x and upwards,* under headings respectively of "$\psi(x)$" and "$\phi(x)$."

It is obvious that from these two columns by taking their first differences we should obtain respectively the numerators and denominators of the p_x fractions. Thus

$$p_x = \frac{\psi(x) - \psi(x+1)}{\phi(x) - \phi(x+1)} = \frac{2P_x - d_x}{2P_x + d_x}.$$

Suppose, further, that instead of publishing the two columns giving respectively $2P-d$ and $2P+d$ at *each* age x and upwards, the compilers had only published them for a *few selected ages,* say for ages 4, 5, 10, 15, 25 . . . 85 and upwards, it would then be possible, by formulæ of interpolation, such as are to be hereafter given, to very nearly reconstruct the original complete series of values for each year of age from age 5 onwards.

But it may further be supposed that the compilers had not troubled to publish the population and death numbers at all, but only the p_x values for the same selected ages, viz., 4, 5, 10, 15, 25 . . . 85. It would then be possible by interpolations in this limited series of p_x values to very nearly reconstruct the original complete series of such values.

The processes of interpolation may therefore be applied, (1) so as to obtain from the foundation numbers of population and deaths, given in age-groups, values of $2P-d$ and $2+d$ for each year of age, and from these the values of p_x for each year of age, or (2) so as to obtain values of $2P-d$ and $2P+d$ only for certain selected years of age, separated by intervals, and from these the corresponding p_x values, and then by applying the formulæ of interpolation to these latter, the complete series of p_x values may be obtained. There are theoretical reasons for (1), but for practical advantage in working (2) is to be preferred.

Interpolation may, of course, be effected in the population and death numbers at age x and upwards *taken separately,* but for practical convenience it is better to take population and deaths *in combination,* arranged as $2P-d$ and $2P+d$ at age x and upwards. It is also best to apply the formulæ not to the

numbers but to the logarithms of the numbers, as it is only thus possible to obtain rational results in continuing the series after age 85.

Let it be understood then that the symbols $\psi(x)$ and $\phi(x)$ are used to denote respectively $2P-d$ and $2P+d$ at age x and upwards, and that $u_x = \log\ \psi(x)$ and $U_x = \log\ \phi(x)$, also that the given values of x are 4, 5, 10, 15, 25 . . . 85.

Suppose that in the u_x series we wish to interpolate u_6. There are given eleven values of u_x, viz., u_4 . . . u_{85}, which may be considered as ordinates of a curve, the equation to which is $a+bx+cx^2+ \ldots +kx^{10}$. If it were proposed to fix the ordinate u_6 with relation to the whole number of given ordinates this would entail very laborious calculations involving ten orders of differences. In the scheme to be afterwards explained it is only proposed to use six orders of differences, which means that the ordinate u_6 is to be determined only with relation to the ordinates u_4, u_5, u_{10}, u_{15}, u_{25}, u_{35}, u_{45}. When u_6 is determined its numerical value is $\psi(6)$, and $\psi(5)-\psi(6) = 2P_5 - d_5$. Similarly $\phi(5)-\phi(6) = 2P_5 + d_5$.

$$\text{Therefore } p_5 = \frac{\psi(5)-\psi(6)}{\phi(5)-\phi(6)}.$$

$$\text{and } \log p_5 = \log\Big(\psi(5)-\psi(6)\Big) - \log\Big(\phi(5)-\phi(6)\Big).$$

Now $\psi(5)-\psi(6)$, and $\phi(5)-\phi(6)$, giving the numbers respectively of $2P-d$ and $2P+d$ at and over age 5 and under age 6, may be considered as approximately the numbers at age $5\frac{1}{2}$.

Suppose, however, that we wish to determine the values of $2P-d$ and $2P+d$ *at the exact age 5*. This can be done by a simple application of the differential calculus, for the knowledge of which the writer is indebted to Mr. A. C. Waters.

It is not possible here to make an attempt to explain the matter fully, but something may be attempted on the same lines as in a paper already contributed by the writer to the "Journal of the Royal Statistical Society," Vol. lxiii., Part 4, December, 1900.

The following brief explanation can only be completely intelligible to those who have some knowledge of the elementary principles of the differential calculus, but it may at least be partly comprehended by those who have not such knowledge. We have already used the symbols $\psi(x)$ and $\phi(x)$ to denote respectively $2P-d$ and $2P+d$ at age x and upwards. Therefore the numbers of $2P-d$ and $2P+d$ in any age-group x to $x+h$ will be denoted respectively by $\psi(x)-\psi(x+h)$, and $\phi(x)-\phi(x+h)$, and the mean chance of living a year possessed by those included in the age-group x to $x+h$ will be represented by

$$\frac{\psi(x)-\psi(x+h)}{\phi(x)-\phi(x+h)}.$$

By changing all the signs and dividing the numerator and denominator by h, this expression becomes the precisely equivalent one

$$\frac{\dfrac{\psi(x+h)-\psi(x)}{h}}{\dfrac{\phi(x+h)-\phi(x)}{h}}.$$

This is true for any value of h, no matter how large or how small; we may suppose, therefore, h to become smaller without limit until finally when h

vanishes, *i.e.*, when $h=o$, or at the *exact moment* of age x, the expression becomes

$$\frac{\psi'(x)}{\phi'(x)}.$$

$$\text{now } \frac{\psi'(x)}{\phi'(x)}=\frac{2P-d \text{ at exact age } x}{2P+d \text{ at exact age } x}.$$

Therefore $p'x$, *i.e.*, the chance of living a year which exists at exact age x,

$$=\frac{\psi'(x)}{\phi'(x)}.$$

In order to be able to calculate the values of $\phi'(x)$ and $\psi'(x)$ the *form* of the functions ψ and ϕ must be fixed.

We have taken the common log of $\psi(x)$ as u_x, and u_x is capable of being interpolated for any age (or value of x) in a series of $n+1$ terms with n orders of differences.

$$\text{Therefore } \psi(x)=10^{a+bx+cx^2+\dots}$$

Now when this is the form of the function ψ it can be shown that

$$\psi'(x)=10^{a+bx+cx^2+\dots}\times \log_e 10\times(b+2cx+3dx^2+\ \dots$$

which may be written

$$\psi'(x)=\psi(x)\times \log_e 10\times(b+2cx+3dx^2+\ \dots)$$

similarly

$$\phi'(x)=\phi(x)\times \log_e 10\times(B+2Cx+3Dx^2+\ \dots)$$

$$\text{Therefore } p'_x=\frac{\psi'(x)}{\phi'(x)}=\frac{\psi(x)\times \log_e 10\times(b+2cx+3dx^2+\ \dots)}{\phi(x)\times \log_e 10\times(B+2Cx+3Dx^2+\ \dots)}$$

Now although the suffix x is usually used to indicate the exact *age*, it may be also used to indicate *relative distance* in a series of ordinates. Instead of reckoning from birth as o we may take any age as o and reckon by distances in years from it. Therefore, in any given series of u_x and U_x values we may make any member of the series to be u_0 and measure the distances of the others from it. For example in the series u_4, u_5, u_{10}, u_{15}, u_{25}, u_{35}, u_{45}, by taking u_5 as u_0, the series becomes u_{-1}, u_0, u_5, u_{10}, u_{20}, u_{30}, u_{40}, and the expression for p'_5 is simplified to

$$p'_0=\frac{\psi(o)\times b}{\phi(o)\times B}.$$

and $\log p'_0=(u_0+\log b)-(U_0+\log B)$.

The problem, therefore, of calculating a series of $\log p'_x$ values is to be solved by (1) making each value of x to be o in the series of u_x and U_x values taken as the foundation series for the calculation, and (2) calculating by appropriate formulæ the values of b and B for each value of x when taken as o.

Since p'_x is the chance of living a year which exists at *exact age* x, it may be taken as very nearly representing the mean chance of living a year possessed by the individuals included in any small age-group of which x is the centre, as the year of age $x-\frac{1}{2}$ to $x+\frac{1}{2}$.

It is thus possible to obtain a number of fixed points in the p_x curve determined with absolute accuracy (in so far as the data are accurate), and from these the complete series for each year of age may be measured either (1) by a "graphic" process of drawing a curve through the given fixed points (*i.e.*, log p'_x values) and measuring the ordinates $5\frac{1}{2}$, $6\frac{1}{2}$, $7\frac{1}{2}$ . . ., or preferably (2) by interpolation.

For a description of (1) see "Journal of Hygiene," Vol. ii., Nos. 2 and 3. The special applications of (2) which have been adopted in working out the Life-Tables in Part II. of this Lecture, are to be soon described.

It has been thought desirable to dwell at some length on the theoretical principles underlying the construction of the p_x curve, as they are of essential importance, and it now remains to proceed to deal more briefly with other points.

Compilation of the Data required for the Construction of a Life-Table. Reference may be made to "Journal of Royal Statistical Society," Vol. lxii., part 3, pp. 446–447, or to "Journal of Hygiene," Vol. ii., No. 1, pp. 2, 3.

Calculation of Mean Population Numbers from Census Figures. On this point see paper by Mr. A. C. Waters in "Journal of Royal Statistical Society," Vol. lxiv., Part 2, June, 1901, also papers in same Journal, Vol. lxiv., Part 3, September, 1901, and Vol. lxv., Part 2, pp. 356—358. Also "Journal of Hygiene," Vol. ii., No. 1, pp. 3—8.

The method adopted for the succeeding Life-Tables is that of Mr. A. C. Waters, which is based on the two assumptions (1) that the population of the whole of England and Wales during the interval from the census of 1891 to that of 1901 changed in Geometrical Progression, and (2) that the proportions of the parts (*i.e.*, age-groups or separate districts) to the whole was changing uniformly in Arithmetical Progression.

On these two assumptions it can be shown that two constant multipliers "m" and "n," can be attained, applicable respectively to the census numbers of 1891 and 1901, and that the sum of the multiples, whether applied to the whole or to the parts gives the true mean annual population, which, when multiplied by 10 gives the "years of life" for the decennium.

$$\text{when } r = \frac{\text{Total Population at census of 1901}}{\text{Total Population at census of 1891}}$$

$$m = \frac{(r-1) + \dfrac{r-1}{\log_e r} - \dfrac{39r+1}{40}}{r^{\frac{1}{2}} \cdot \log_e r}.$$

$$n = \frac{\dfrac{39r+1}{40} - \dfrac{r-1}{\log_e r}}{r \cdot r^{\frac{1}{2}} \cdot \log_e r}.$$

By applying these formulæ to the total census numbers for England and Wales for 1891 and 1901

$$r = \frac{32{,}527{,}843}{29{,}002{,}525} = 1{\cdot}121552106239 \ldots$$

$\log_e r = 0{\cdot}1147135351 \ldots$
$r^{\frac{1}{10}} = 1{\cdot}0028719455 \ldots$
$m = 0{\cdot}544606333$
$n = 0{\cdot}456486649$
Total years of life for 1891—1900 = 306,434,848.

Calculation of the p_x Values for the First Five Years of Age. Descriptions of the methods applicable to this part of "Life-Table construction" are to be found (1) in Dr. Newsholme's "Vital Statistics," 3rd edition, pp. 271—273; (2) in "Journal of Royal Statistical Society," Vol. lxii., part 3, pp. 451—454; (3) in "Journal of Hygiene," Vol. ii., No. 1, pp. 10—14. The description in (3) is the most complete.

PRACTICAL WORKING FORMULÆ, BASED ON THEORETICAL CONSIDERATIONS PREVIOUSLY EXPLAINED, WHICH HAVE BEEN USED IN CALCULATING THE p_x VALUES OF THE LIFE-TABLES GIVEN IN PART II.

This may be called Method I. It is assumed that the numbers representing $2P-d$ and $2P+d$ at age x and upwards have been compiled for the ages 4, 5, 10, 15, 25 . . . 85, and that the corresponding logarithms have been taken, these latter being denoted by the symbols u_x and U_x.

It is first of all necessary to continue the u_x and U_x series to values for ages 95 and 105.

This is done by differencing the series u_{45}, u_{55}, u_{65}, u_{75} and u_{85}, and carrying down the differences for two stages.

It is to be understood, so as to avoid needless repetition, that which is stated concerning the u_x series also applies to the U_x series.

The log p'_x values for ages 5, 10, 15, 20 and 25 have been calculated from the series u_4, u_5, u_{10}, u_{15}, u_{25}, u_{35}, u_{45}.

The least laborious method is to interpolate u_{20} and u_{30} in this series, which may be effected by the following formulæ:—

Let the following multiples of the respective u_x values be taken, and let each multiple of u_x be denoted by the symbol v_x

$$\begin{aligned}
u_4 &\times 625{,}000 && = v_4\\
u_5 &\times 97{,}867 \text{ or } 11(10{,}000-1{,}103) && = v_5\\
u_{10} &\times 223{,}696 \text{ or } (44\times 5{,}084) && = v_{10}\\
u_{15} &\times 177{,}940 \text{ or } 20(10{,}000-1{,}103) && = v_{15}\\
u_{25} &\times 139{,}810 \text{ or } (11\times 12{,}710) && = v_{25}\\
u_{35} &\times 12{,}628 \text{ or } 44(300-13) && = v_{35}\\
u_{45} &\times 341 \text{ or } (11\times 31) && = v_{45}
\end{aligned}$$

Then $$u_{20} = \frac{[10v_5 + 6(v_{15}+v_{45}) + 2v_{25}] - [v_4 + 2(2v_{10}+v_{35})]}{782{,}936}$$

and $$u_{30} = \frac{4v_4 + 13[(v_{10}+v_{25}+3v_{35}) - (3v_5+v_{15}+5v_{45})]}{1{,}565{,}872}.$$

The arithmetical labour may be diminished by subtracting u_{45} from all the terms of the series, thus reducing itself to zero. The u_{20} and u_{30} of the formulæ must then be *added* to u_{45}.

Next let the seven equidistant terms u_5, u_{10}, u_{15}, u_{20}, u_{25}, u_{30}, u_{35} be set down and differenced. If the differences be carried down for two stages u_{45} will be obtained and the correctness of the calculations for u_{20} and u_{30} will be proved.

Let the lines of differences opposite to u_{10}, u_{15}, u_{20} and u_{25} be completed in the table, as shown in the following illustrative example in which, for the sake of space, the three extra places of decimals have been omitted.

		Δ	Δ^2	Δ^3	Δ^4	Δ^5	Δ^6
u_5	8·4128672 :	− 616603 :	− 68473 :	+ 8428 :	− 7794 :	+ 7827 :	− 11652 :
u_{10}	8·3512069 :	− 685075 :	− 60045 :	+ 634 :	+ 33 :	− 3825 :	− 11652 :
u_{15}	8·2826993 :	− 745121 :	− 59411 :	+ 667 :	− 3792 :	− 15477 :	− 11652 :
u_{20}	8·2081872 :	− 804532 :	− 58744 :	− 3125 :	− 19269 :	− 27129 :	− 11652 :
u_{25}	8·1277340 :	− 863276 :	− 61869 :	− 22394 :	− 46398 :	− 38781 :	− 11652 :
u_{30}	8·0414064 :	− 925145 :					
u_{35}	7·9488919 :						

(In order to avoid the repetition of cyphers : is used to indicate the end of seven places of decimals.)

Then the values of b, when u_5, u_{10}, u_{15}, u_{20} and u_{25} are respectively made to be u_0 in the series, can be obtained by applying to each of the lines of differences in succession the formula

$$5b = \Delta u_0 - \tfrac{1}{2}\Delta^2 u_0 + \tfrac{1}{3}\Delta^3 u_0 - \tfrac{1}{4}\Delta^4 u_0 + \tfrac{1}{5}\Delta^5 u_0 - \tfrac{1}{6}\Delta^6 u_0.$$

The result will be a − quantity, but it may be taken as +, and the division by 5 need not be made, since

$$\frac{-5b}{-5B} = \frac{b}{B}$$

As it is of essential importance to make perfectly clear the mode of calculating $\log p'_x$ the following example may be given.

In the above table we have $u_5 = 8{\cdot}4128672$: The application of the given formula to the line of differences opposite to u_5 gives the result $5b = -574099{:}8$ From the corresponding table dealing with U_x values we have $U_5 = 8{\cdot}4186377{:}$, and $5B = -571210{:}1$. Therefore from the formula $\log p'_o = (u_o + \log b) - (U_o + \log B)$ we have

$$\begin{aligned}\log p'_5 &= (8{\cdot}4128672 + \log 574099{:}8) - (8{\cdot}4186377 + \log 571210{:}1)\\ &= (8{\cdot}4128672 + \bar{2}{\cdot}7589874) - (8{\cdot}4186377 + \bar{2}{\cdot}7567958)\\ &= \bar{1}{\cdot}9964211.\end{aligned}$$

The calculations for the $\log p'_x$ values for ages 35, 45, . . .85 are much easier, as each is to be derived from a series of five equidistant terms of which the central term in each case is taken as u_0.

Thus $\log p'_x$ is to be calculated from the series u_{15}, u_{25}, u_{35}, u_{45}, u_{55}, in which u_{35} is taken as u_0. The general formula for b in such a series is:—

$$b = \frac{8(u_{10} - u_{-10}) - (u_{20} - u_{-20})}{120}.$$

or, changing all the signs, and arranging for convenience in working:—

$$-120b = 10(u_{-10} - u_{10}) - [2(u_{-10} - u_{10}) + (u_{-20} - u_{20})].$$

At this stage we shall have obtained a series of log p'_x values for ages 5, 10, 15, 20, 25, 35 . . . 85.

It is required to continue the series as far as age 105 and to interpolate intermediate terms corresponding to ages 30, 40 . . . 100. The following special formulæ may be used for ages 30 and 40. (In order to avoid confusion it must be noted that in the following formulæ u_x is used to denote log p'_x.)

$$u_{30} = \frac{[21(u_5 + 25u_{15} + 50u_{25} + 10u_{35}) + u_{55}] - [160(u_{10} + 6u_{20}) + 15u_{45}]}{672}.$$

$$u_{40} = \frac{(320u_{20} + 1050u_{35} + 630u_{45} + 7u_{65}) - (63u_{15} + 525u_{25} + 75u_{55})}{1344}.$$

which may be thus written :—

$$u_{40} = \frac{5[(64u_{20} - 15u_{55}) + 105(2u_{35} - u_{25})] + 7[9(10u_{45} - u_{15}) + u_{65}]}{1344}.$$

The values of log p'_{50} and log p'_{60} may be obtained by the following general formula for interpolating a central term u_0 in a series of six equidistant terms u_{-5}, u_{-3}, u_{-1}, u_1, u_3, u_5.

$$u_0 = \frac{300(u_{-1} + u_1) - 50(u_{-3} + u_3) + 6(u_{-5} + u_5)}{512}.$$

For example, the unit of interval being 5,

$$u_{50} = \frac{300(u_{45} + u_{55}) - 50(u_{35} + u_{65}) + 6(u_{25} + u_{75})}{512}$$

The following special formulæ may be used for log p'_{70} and log p'_{80}.

$$u_{70} = \frac{3(u_{45} + 30u_{65} + 20u_{75}) - 5(4u_{55} + u_{85})}{128}.$$

$$u_{80} = \frac{5(3u_{65} + 9u_{75} + u_{85}) - (u_{55} + 40u_{70})}{24}.$$

Next by differencing the five equidistant terms u_{65}, u_{70}, u_{75}, u_{80}, u_{85}, it is easy to carry on the series to u_{90}, u_{95}, u_{100} and u_{105}.

Having proceeded so far we shall have a complete series of log p'_x values at 5-yearly intervals from age 5 to age 105.

The next step is to interpolate in this series a further series of intermediate terms corresponding to ages $7\frac{1}{2}$, $12\frac{1}{2}$ . . . $92\frac{1}{2}$.

The same general formula as that already given is applicable from ages $17\frac{1}{2}$ to $92\frac{1}{2}$.

Thus, the unit of interval being $2\frac{1}{2}$,

$$u_{17\frac{1}{2}} = \frac{300(u_{15} + u_{20}) - 50(u_{10} + u_{25}) + 6(u_5 + u_{30})}{512}$$

For $u_{12\frac{1}{2}}$ and $u_{7\frac{1}{2}}$ the following formulæ may be used :—

$$u_{12\frac{1}{2}} = \left[\frac{20(u_{10} + u_{20}) + 90u_{15} - (u_5 + u_{25})}{64}\right] - u_{17\frac{1}{2}}.$$

$$u_{7\frac{1}{2}} = \left[\frac{(u_5 + u_{20}) + 15(u_{10} + u_{15}) - 20u_{12\frac{1}{2}}}{6}\right] - u_{17\frac{1}{2}}.$$

Having now obtained a series of log p'_x values for ages $7\frac{1}{2}$, $12\frac{1}{2}$, $17\frac{1}{2}$ . . . $92\frac{1}{2}$, it may be noted that since the chance of living a year at *exact age* $7\frac{1}{2}$ may be

taken as the chance of living from age 7 to age 8, we may change the notation from log p'_x to log p_x, and we may use the series of log p_x values for ages 7, 12, 17 . . . 92 to interpolate the required complete series for each year from age 5 onwards.

The scheme which has been adopted in dealing with the given eighteen log p_x values consists, (*a*) in taking them in groups of six each (five orders of differences); (*b*) in making the successive series overlap each other; and (*c*) in welding the series together by pairs of multipliers calculated from the "curve of cosines," according to the method devised by Mr. A. C. Waters.

The following is a tabular representation of the scheme, in which it must be understood that for the sake of conciseness the figure 7 is intended to denote log p_7 and so on. The portions of each sub-series actually used are indicated by brackets, and the places at which the sub-series are welded are indicated by under-and-over lines.

(1) 7, 12, 17, 22, 27], 32.

(2) 17, [22, 27, 32, 37], 42.

(3) 27, [32, 37, 42, 47], 52.

(4) 37, [42, 47, 52, 57], 62.

(5) 47, [52, 57, 62, 67], 72.

(6) 57, [62, 67, 72, 77], 82.

(7) 67, [72, 77, 82, 87, 92. . .

The first step is to set down the six terms of each sub-series in a column and to difference them.

From the differences corresponding to 5-yearly intervals thus obtained, which may be denoted by the symbol Δ, the sub-divided differences corresponding to yearly intervals, denoted by the symbol δ, may be obtained by the following simple formulæ.

$$\delta^5 u_0 = \cdot00032\Delta^5 u_0$$
$$\delta^4 u_0 = \cdot0016\Delta^4 u_0 - 8\delta^5 u_0$$
$$\delta^3 u_0 = \cdot008\Delta^3 u_0 - 6\delta^4 u_0 - 18\delta^5 u_0$$
$$\delta^2 u_0 = \cdot04\Delta^2 u_0 - 4\delta^3 u_0 - 8\delta^4 u_0 - 10\delta^5 u_0$$
$$\delta u_0 = \cdot2\Delta u_0 - 2\delta^2 u_0 - 2\delta^3 u_0 - \delta^4 u_0 - \cdot2\delta^5 u_0.$$

In sub-series (1) the formulæ are to be applied to the *first* line of differences opposite to u_7, but in the other sub-series (2) to (7) they are to be applied to the *second* line of differences, completed by inserting the constant $\Delta^5 u_0$.

Before proceeding to carry down the differences it is desirable to check them by the following formulæ:—

For sub-series (1),

$$u_{25} = u_0 + 25\delta u_0 + 300\delta^2 u_0 + 2{,}300\delta^3 u_0 + 12{,}650\delta^4 u_0 + 53{,}130\delta^5 u_0.$$

For sub-series (2) to (7),

$$u_{20} = u_0 + 20\delta u_0 + 190\delta^2 u_0 + 1{,}140\delta^3 u_0 + 4{,}845\delta^4 u_0 + 15{,}504\delta^5 u_0.$$

The scheme for "welding" the respective overlapping parts of two sub-series is illustrated by the following example:—

	in sub-series (1)	in sub-series (2)
$\log p_{23} =$	$\log p_{23} \times \cdot 904$	$+ \log p_{23} \times \cdot 096$
$\log p_{24} =$	$\log p_{24} \times \cdot 654$	$+ \log p_{23} \times \cdot 346$
$\log p_{25} =$	$\log p_{25} \times \cdot 346$	$+ \log p_{24} \times \cdot 654$
$\log p_{24} =$	$\log p_{26} \times \cdot 096$	$+ \log p_{26} \times \cdot 904$

The calculations are facilitated by deducting the largest possible common value from each pair of log p_x values before multiplying, and then adding the two products to the common value.

Seeing that sub-series (1) starts with log p_7, the values of log p_5 and log p_6 may either be obtained by carrying back the differences, or preferably by the following formulæ:—

$$u_5 = \left(\frac{2u_4 + 5u_{11}}{7}\right) + 5u_7 + 9u_9 - (10u_8 + 4u_{10}).$$

$$u_6 = \left[\frac{(u_4 + 6u_{11}) + 70(u_7 - \frac{1}{2}u_{10})}{21}\right] + 4u_9 - 5u_8.$$

Exactly how far sub-series (7) must be carried on can only be determined when the l_x column of the Life-Table is calculated.

Having now obtained the complete series of log p_x values the remaining stages in the construction of a Life-Table may be illustrated by the following simple scheme which has already been used in a paper in "Journal of Hygiene," Vol. ii., No. 2.

An Easy First Lesson in Constructing a Life-Table from the Series of p_x Values when obtained.

Age	p_x	l_x	P_x	Q_x	$\frac{Q_x}{l_x} = E_x$
x	$\frac{8}{10} = 0{\cdot}8$	10	9	25	$\frac{25}{10} = 2{\cdot}5$
$x+1$	$\frac{6}{8} = 0{\cdot}75$	8	7	16	$\frac{16}{8} = 2{\cdot}0$
$x+2$	$\frac{4}{6} = 0{\cdot}\dot{6}$	6	5	9	$\frac{9}{6} = 1{\cdot}5$
$x+3$	$\frac{2}{4} = 0{\cdot}5$	4	3	4	$\frac{4}{4} = 1{\cdot}0$
$x+4$	$\frac{0}{2} = 0$	2	1	1	$\frac{1}{2} = 0{\cdot}5$
$x+5$		0			

The processes of calculation are as follows:—

(1) $l_x \times p_x = l_{x+1}$; $l_{x+1} \times p_{x+1} = l_{x+2}$; &c.

(2) $P_x = \frac{1}{2}(l_x + l_{x+1})$; $P_{x+1} = \frac{1}{2}(l_{x+1} + l_{x+2})$; &c.

(3) $Q_{x+4} = P_{x+4}$; $Q_{x+3} = Q_{x+4} + P_{x+3}$; &c.

(4) $\frac{Q_x}{l_x} = E_x$; $\frac{Q_{x+1}}{l_{x+1}} = E_{x+1}$; &c.

It is to be understood, of course, that the multiplications and divisions are in actual practice to be effected by means of logarithms.

It is of importance to comprehend:—

(1) that the p_x values have relation to the *present* tense; *i.e.*, each value depends upon the rate of mortality at the middle of the age x to $x+1$.

(2) that the l^x values have relation to the *past* tense, in that they are only affected by the *preceding* p_x values, from p_0 to p_{x-1};

(3) that the E_x values have relation to the *future* tense, in that they are only affected by p_x and the *following* values to $p_{x+\omega-1}$ (where $x+\omega$ is the age at which there are no more survivors).

It is obvious that the value $l_x=10$ can only depend upon the arbitrary number l_0 with which the Life-Table will have been commenced, and upon the values of p_0 to p_{x-1}. Assuming these to have been so different that l_x has become 20 instead of 10, this could not affect the values of E_x to E_{x+4}, for the values of p_x to p_{x+4} remain the same, and therefore all the results of the calculations in the above Table will be doubled, and $E_x=\frac{50}{20}=2{\cdot}5$ as before.

Therefore Q_x is is always in direct proportion to l_x.

There is a special point to note concerning P_0 which is not truly represented by $\frac{1}{2}(l_0+l_1)$. It is best obtained by the following formula, derived by a simple application of the integral calculus:—

$$P_0=l_1+\frac{(l_0-l_{\frac{1}{2}})+5(l_{\frac{1}{2}}-l_1)}{6}.$$

With this formula in view it is desirable to have calculated p_0 in two stages:—(1) the chance of living from birth to age 6 months, and (2) the chance of living from age 6 months to age 1.

The values of $E_{x/n}$ are derived from the Q_x and P_x values by the formula

$$E_{x/n}=\left[\frac{Q_x+Q_{x+1}+\ldots+Q_{x+n-1}}{P_x+P_{x+1}+\ldots+P_{x+n-1}}\right]-\frac{1}{2}.$$

On some other Methods of Applying the General Principles of Interpolation to Obtaining a Series of Log p_x Values. The evolution of the scheme which has already been described was largely guided by its originally intended application to arriving at the complete yearly series of log p_x values by measuring ordinates in a curve graphically drawn through a limited number of fixed points determined by exact calculation.

In its subsequent development the general principle has been to do as little interpolation as possible in the foundation series of u_x and U_x values, to get away from these as speedily as may be, and to reserve all subsequent processes of interpolation to the log p'_x values.

This scheme has been described so fully, because *in its earlier stages it is the most easily applicable to the method to be described in the next section of this Lecture of working out an abbreviated Life-Table in 5-yearly stages.*

Moreover, with the prospect in view or working out three concurrent Life-Tables by an extended method, it was of importance to adopt a scheme entailing the minimum amount of labour. Still this is only one of many possible

methods, and it is now proposed to briefly indicate the main lines of three other schemes, which have as their first stage the interpolation of intermediate terms at 5-yearly intervals in the original series of u_x and U_x values. These may be called Methods II., III. and IV.

The scheme of interpolation will be apparent when set down as below in a tabular form, the figure 4 being for the sake of conciseness used to denote u_4 and so on.

The interpolated terms are indicated by thicker type.

4	5	10	15	**20**	25		35		45									
	5	10	15	**20**	25	**30**	35		45		55							
			15		25		35	**40**	45		55		65					
					25		35		45	**50**	55		65		75			
							35		45		55	**60**	65	**70**	75	**80**	85	

The formulæ of interpolation are given below. (The formula for u_{20} is another form of that already given. Seeing that u_{30} is not required in the same series this new form is to be here preferred.)

$$u_{20} = \left[\frac{12{,}710(77u_5 + 84u_{15} + 22u_{25}) + 2{,}046u_{45}] - [625{,}000u_4 + 3{,}608(248u_{10} + 7u_{35})}{782{,}936}\right].$$

$$u_{30} = \frac{[21(u_5 + 25u_{15} + 50u_{25} + 10u_{35}) + u_{55}] - [160(u_{10} + 6u_{20}) + 15u_{45}]}{672}.$$

(The u_{20} in the above formula is that already calculated by the preceding formula). u_{40}, u_{50}, and u_{60} are to be obtained by the general formula already given for interpolating a central term in a series of six equidistant terms.

$$u_{70} = \frac{21(u_{45} + 10u_{65} + 5u_{75}) - [3u_{35} + 7(10u_{55} + u_{85})]}{256}.$$

$$u_{80} = \frac{7[u_{35} + 9(2u_{55} + 5u_{75} + u_{85})] - [5(9u_{45} + 42u_{65})]}{256}.$$

If now the six equidistant terms u_{55}, u_{60}, u_{65}, u_{70}, u_{75}, u_{80} be differenced the series can be carried on at 5-yearly intervals to u_{85} (proving the correctness of the interpolation of u_{70} and u_{80}) and then to u_{90} . . . u_{120}.

Next by differencing the seven equidistant terms u_5, u_{10}, u_{15}. u_{20}, u_{25}, u_{30}, u_{35}, the series can be carried back to u_0, u_{-5}, and u_{-10}.

In order to secure accuracy four extra places of decimals must be used in these differencings.

Method II. Being now provided with a continuous series of equidistant terms of u_x and U_x extending at 5-yearly intervals from u_{-10} to u_{120}, it is possible to calculate the values of log p'_x from age 5 to age 105 by the application of the following formula for obtaining the values of b and B in a series of seven equidistant terms of which the central one is taken as u_0.

$$b = \frac{45(u_1 - u_{-1}) - 9(u_2 - u_{-2}) + (u_3 - u_{-3})}{60n}.$$

which, when the interval $n = 5$ may be written thus for convenience in working:

$$-300b = \left[\frac{100 - 10}{2}(u_{-5} - u_5) + (u_{-10} - u_{10}) + (u_{-15} - u_{15})\right] - 10(u_{-10} - u_{10}).$$

Having obtained the series of log p'_x values from age 5 to age 105 the interpolation of the intermediate values for ages $7\frac{1}{2}$ to $92\frac{1}{2}$ may be proceeded with as in the previous scheme already described.

Method III. Another method is after having obtained the consecutive series of u_x and U_x values at 5-yearly intervals (which in this case need only extend from u_{-5} to u_{105}), to proceed to interpolate a further series of terms in each series, $u_{7\frac{1}{2}}$, $u_{12\frac{1}{2}}$, . . . $u_{92\frac{1}{2}}$.

This can be readily done by the application of the general formula already given.

$$\text{Thus } u_{7\frac{1}{2}} = \frac{300(u_5 + u_{10}) - 50(u_0 + u_{15}) + 6(u_{-5} + u_{20})}{512}.$$

It is then possible to obtain *directly* from the u_x and U_x series, the series of log p'_x values for ages $7\frac{1}{2}$, $12\frac{1}{2}$ . . . $92\frac{1}{2}$ required for the scheme of yearly interpolation, by the application of the following formula for obtaining the values of b and B in a series of seven terms, of which six are equidistant, and the seventh, taken as u_0 is the central term.

$$b = \frac{2250(u_1 - u_{-1}) - 125(u_3 - u_{-3}) + 9(u_5 - u_{-5})}{3840n}$$

which, when the interval $n = 5$ may be thus written for convenience in working:

$$-76800b = 9\Big[1000(u_{-5} - u_5) + 4(u_{-25} - u_{25})\Big] - 500(u_{-15} - u_{15}).$$

Methods II. and III. have the theoretical advantage, as compared with Method I. of keeping longer in touch with the foundation numbers of population and deaths. If this advantage be desired Method II, while also being applicable to the construction of an extended Life-Table, may be substituted for Method I. in constructing an abbreviated Life-Table by an application of the integral calculus to be hereafter described.

If it be proposed to construct an extended Life-Table from census data, Method III. may perhaps be considered as an approach to an ideally perfect application of the differential calculus. It has been found by actual trial that the p_x values obtained by the three methods, while not absolutely identical, agree very closely. They may all be considered as rational methods. That Method I. can give satisfactory results will be evident by the three concurrent Life-Tables to be afterwards given, which have been worked out by it. Methods II. and III. have been mentioned partly with the view of showing how much less labour is involved by Method I, which is therefore the method to be preferred from this point of view.

Various other methods of obtaining log p'_x values have been tried involving fewer as well as more orders of differences, but it would occupy too much space to attempt to allude to them in detail.

Method IV. for obtaining the complete series of p_x values directly from Population and Deaths.

If it be desired to effect a complete series of interpolations at yearly intervals in the foundation u_x and U_x values (which are respectively the logs of $2P - d$

and 2 $P+d$ at age x and upwards), and then, by taking the first differences of the numerical values of the resulting series of logs, to obtain $\frac{2P-d}{2P+d}$ for each year of age, the following plan may be adopted.

After having interpolated u_{20}, u_{30}, ... u_{80} in the original series by formulæ already given in the description of Methods II. and III., the scheme of interpolation indicated below may be applied. (It is to be understood that the numeral 5 stands for u_5, &c.).

5, 10, 15, 20, 25], 30,
15, [20, 25, 30, 35], 40,
25, [30, 35, 40, 45], 50,
35, [40, 45, 50, 55], 60,
45, [50, 55, 60, 65], 70,
55, [60, 65, 70, 75, 80. . . . .].

The formulæ for effecting this series of interpolations, and for welding together the overlapping parts, have already been given in the description of Method I., as applied to obtaining a yearly series of log p_x values.

The series of p_x values obtainable by the above method would have a close relation to the foundation numbers of population and deaths, and would very nearly indicate the true p_x curve in so far as the data were true.

ON ABBREVIATED METHODS OF LIFE-TABLE CONSTRUCTION.

It has been shown by the present writer that by very simple modifications of the late Dr. Wm. Farr's original method of constructing "short" Life-Tables it is possible to arrive at E_x values at quinquennial or decenneal intervals approximating very closely to those which would be obtained by the much more laborious "extended" methods.

For description of these modifications see (1) "Public Health" for July, 1898, (2) "Journal of the Royal Statistical Society," Vol. lxii., Part 3, pp. 470—480, (3) "Journal of Hygiene," Vol. ii., No. 1, pp. 34—42, (4) Newsholme's "Vital Statistics," third edition, chapter xxiii. It was afterwards shown how, by equally simple methods, $E_{x/n}$ values could be obtained. See (5) "Journal of Royal Statistical Society," Vol. lxv., Part 4, pp. 680—684.

To give one more example of the value of these methods the following tables may be presented, showing the differences from the E_x and $E_{x/n}$ values obtained for England and Wales, 1891—1900, by the extended method herein described of the corresponding values obtained by the shortened methods described at the above-given references, of which (2) and (5) may be especially referred to.

Ages.	E_x Males.	E_x Females.		Ages.	$E_{x/n}$ Males.	$E_{x/n}$ Females.
0	+0·01	−0·01	.....	0—5	+0·02	+0·01
5	+0·03	±0·00	.....	5—10	+0·03	+0·01
10	+0·01	−0·01	.....	10—15	+0·01	−0·01
15	+0·01	−0·01	.....	15—20	+0·05	+0·01
20	+0·07	+0·02	.....	20—25	+0·04	±0·00
25	+0·02	±0·00	.....	25—35	−0·02	−0·03
35	−0·06	−0·07	.....	35—45	−0·04	−0·07
45	−0·05	−0·06	.....	45—55	−0·03	−0·06
55	−0·08	−0·12	.....	55—65	−0·03	−0·07
65	−0·05	−0·08	.....	65—75	+0·01	±0·00
75	−0·02	−0·04	.....	75—85	+0·03	±0·00
85	+0·08	+0·02	.....	85 and upwards	+0·05	+0·02

For merely local Life-Tables the results thus indicated may be regarded as sufficiently close approximations, especially when it is considered that *much larger differences are to be obtained between the results of different extended methods.* However, the empirical and somewhat crude methods by which these results have been obtained do not give true l_x values, although when two Life-Tables calculated by their means are compared with each other, the differences of the l_x values are rightly indicated in direction if not in precise degree.

As an improvement upon these methods it is now proposed to explain how, by simple applications of the integral calculus it is possible to obtain a series of l_x and E_x values at 5-yearly intervals almost exactly coinciding with those which are to be obtained by interpolating p_x values for each year of age. Indeed, if there be found differences between the two sets of results, those obtainable at 5-yearly stages by the methods to be described must be regarded as the more accurate, because in the process of working out an extended Life-Table in yearly stages, the 5-yearly sections of the true p_x and l_x curves are measured by interpolating only four intermediate ordinates, whereas, by the use of the integral calculus, the five-yearly sections of the curves are measured with absolute and perfect accuracy, taking into account an infinite number of intermediate ordinates.

Let us go back to the point at which in the construction of the extended Life-Table by Method I. we had obtained the series of log p'_x values at quinquennial intervals from age 5 to age 105, and had then interpolated a further series of central terms corresponding to ages $7\frac{1}{2}$, $12\frac{1}{2}$ . . . $92\frac{1}{2}$, each of which had been made to depend upon three values on either side of it.

Using the symbol u_x to denote log p'_x and considering the given six equidistant log p'_x values as ordinates of a curve, the equation to which is $u_x = A + Bx + Cx^2 + \ldots$ we have already found the exact value of the central term, or ordinate, according to this equation, by the interpolation formula:—

$$u_0 = \frac{300(u_{-1} + u_1) - 50(u_{-3} + u_3) + 6(u_{-5} + u_5)}{512}.$$

Having therefore given the seven ordinates u_{-5}, u_{-3}, u_{-1}, u_0, u_1, u_3, u_5, which form a series with five orders of differences, the problem is to measure the sum of all the ordinates between u_{-1} and u_1.

Now it can be shown that the general expression for such a sum,

$$\int u_x dx, \text{ when } u_x = A + Bx + Cx^2 + Dx^3 + Ex^4 + Fx^5, \text{ is}$$

$$\left(Ax + \frac{Bx^2}{2} + \frac{Cx^3}{3} + \frac{Dx^4}{4} + \frac{Ex^5}{5} + \frac{Fx^6}{6}\right).$$

and therefore the sum between the limits $-n$ and n, expressed by

$$\int_{-n}^{n} u_x dx \text{ is equal to}$$

$$\left(nA + \frac{n^2B}{2} + \frac{n^3C}{3} + \frac{n^4D}{4} + \frac{n^5E}{5} + \frac{n^6F}{6}\right)$$

$$-\left(-nA + \frac{n^2B}{2} - \frac{n^3C}{3} + \frac{n^4D}{4} - \frac{n^5E}{5} + \frac{n^6F}{6}\right)$$

$$= 2nA + \frac{2n^3C}{3} + \frac{2n^5E}{5}.$$

After working out the values of C and E in terms of the given seven ordinates,* and taking $A = u_0$, this last expression is found to be reduced to

$$\frac{n}{15}\left[\frac{111{,}872u_0 + 30{,}690(u_{-1} + u_1) - 235(u_{-3} + u_3) + 9(u_{-5} + u_5)}{5{,}760}\right].$$

which, when $n = 2\frac{1}{2}$, can be written thus for convenience in working.

$$\frac{10{,}230[11u_0 + 3(u_{-1} + u_1)] + 9(u_{-5} + u_5) - 47[14u_0 + 5(u_{-3} + u_3)]}{34{,}560}$$

This will give the sum of all the ordinates between u_{-1} and u_1 in terms of

$$u_{-5},\ u_{-3},\ u_{-1},\ u_0,\ u_1,\ u_3,\ u_5,$$

However, since u_0 has already been determined with relation to the six original ordinates, a sufficiently near approximation to the value required can be obtained by only bringing u_{-1}, u_0, and u_1 into the integration formula, which is thus reduced to the much simpler expression,

$$\int_{-n}^{n} u_x dx = \frac{n}{3}(4u_0 + u_{-1} + u_1).$$

and which when $n = 2\frac{1}{2}$ becomes

$$\frac{5(4u_0 + u_{-1} + u_1)}{6}.$$

In order then to obtain the sum of all the possible log p'_x values between log p'_5 and log p'_{10}, the rule simply is, *take the sum of the two outside terms plus 4 times the central term, multiply this sum by 5, and divide the product by 6;* the quotient is the logarithm which will enable us to pass by one step of addition from log l_5 to log l_{10}. By a similar process the remaining steps may be taken until log l_{105} is reached.

* This has been done by means of some general algebraical formulæ for expressing B, C, D, &c., in terms of u_0, u^a, u_b, &c., which are too long to quote here.

Before applying the formula it is best to put each logarithm into the negative form, thus

$$\log p'_{5} = \bar{1}\cdot 9964211 = -\cdot 0035789$$
$$\log p'_{7\frac{1}{2}} = \bar{1}\cdot 9983127 = -\cdot 0016873$$
$$\log p'_{10} = \bar{1}\cdot 9989816 = -\cdot 0010184$$

By applying the formula the result is $-\cdot 0094554$, which is easily changed back into $\bar{1}\cdot 9905446$.

As an example of the different results obtained by different methods take the interval between $\log p_{60}$ and $\log p_{65}$. The results are:—

(1) by the complete formula of integration ... ... $\bar{1}\cdot 9097524$

(2) by the short formula ... ... $\bar{1}\cdot 9097519$

(3) by yearly interpolations, *i.e.*, the sum of the $\log p_x$ values for ages 60, 61, 62, 63 and 64 ... ... $\bar{1}\cdot 9097727$

It may be noted that the values of $\log p'_{97\frac{1}{2}}$ and of $\log p_{102\frac{1}{2}}$ may be obtained by the application of the following special formulæ:—

$$u_{97\frac{1}{2}} = \frac{5(u_{90} + 9u_{95} + 3u_{100}) - (u_{105} + 24u_{92\frac{1}{2}})}{40}.$$

$$u_{102\frac{1}{2}} = u_{92\frac{1}{2}} - \left[\frac{5(u_{95} - u_{100}) + (u_{90} - u_{105})}{4}\right].$$

By taking out the numerical values of the series of $\log l_x$ values now to be readily obtained we shall have a complete series of true l_x numbers at 5-yearly intervals from age 5 to age 105.

These l_x numbers also may be regarded as ordinates of a curve, and exactly the same integration formulæ may be applied to them (to the *numbers*, not the logarithms), so as to obtain the true years of life lived by l_x persons in the age-period from age x to age $x+5$, *i.e.*, the value of $Q_x - Q_{x+5}$ (or $Q_{x/5}$). Before this can be done, however, intermediate values must be calculated, $l_{7\frac{1}{2}}$, $l_{12\frac{1}{2}}$, . . . $l_{92\frac{1}{2}}$. Those for ages $17\frac{1}{2}$ . . . $92\frac{1}{2}$ inclusive may be readily calculated as the central terms in an equidistant series of six by the general formula already given. For ages $7\frac{1}{2}$ and $12\frac{1}{2}$ the following special formulæ may be used.

Having already calculated $l_{17\frac{1}{2}}$,

$$l_{12\frac{1}{2}} = \left[\frac{20(l_{10} + l_{20}) + 90l_{15} - (l_5 + l_{25})}{64}\right] - l_{17\frac{1}{2}}.$$

$$l_{7\frac{1}{2}} = \left[\frac{(l_5 + l_{20}) + 15(l_{10} + l_{15}) - 20l_{12\frac{1}{2}}}{6}\right] - l_{17\frac{1}{2}}.$$

As it is scarcely necessary to obtain a later value of E_x than E_{95}, Q_{95} may be obtained from l_{90}, l_{100} and l_{105} by applying the formula of integration

$$\int_{-n}^{n} u_x dx = \frac{n}{3}\left(4u_0 + u_{-1} + u_1\right)$$

with the value of $n=5$, and which then becomes

$$\frac{5(4u_0+u_{-1}+u_1)}{3}.$$

$$\text{Therefore} \quad Q_{95}=\frac{5(4l_{100}+l_{95}+l_{105})}{3}.$$

$$\text{Then} \quad Q_{90}-Q_{95}=\frac{5(4l_{92\frac{1}{2}}+l_{90}+l_{95})}{6}.$$

and so on until Q_5-Q_{10} has been worked out. Q_0-Q_5 will have been obtained already as the sum of P_0, P_1, P_2, P_3 and P_4.

The Q_x column can now be constructed at 5-yearly stages by successive additions, and then the series of E_x values can be calculated by the formula

$$E_x=\frac{Q_x}{l_x}.$$

Thus with very much less labour than that involved by the complete extended method a series of l_x and E_x values at 5-yearly intervals can be obtained with perfect accuracy in so far as the data are accurate.

In order to complete the work, however, it is desirable to obtain also values of $E_{x/n}$ that is values expressing the mean future lifetime of all the individuals included within the limits of the usual age-groups.

This can readily be done by a further application of the same method already employed to obtain l_x and E_x values.

$$\text{since } E_x=\frac{Q_x}{l_x} \text{ it follows that } E_{-n/n}=\frac{\int_{-n}^{n} Q_x dx}{\int_{-n}^{n} l_x dx}.$$

As has already been noted Q_x is always in direct proportion to l_x. From a geometrical point of view an integration of l_x is represented by an *area*, and Q_x is a *sum of areas*, therefore an integration of Q_x is represented by a *solid* figure. In actual practice, as the successive values of l_x are not precisely the same function of x, an integration of Q_x can only be an approximate value, but it will be so near to the true value that the error may be neglected.

In order by this method to obtain values of $E_{x/n}$ the Q_x numbers must be dealt with exactly as the l_x numbers are dealt with.

It is first of all necessary to interpolate intermediate Q_x values for ages $7\frac{1}{2}$, $12\frac{1}{2}$, $17\frac{1}{2}$ and $22\frac{1}{2}$ by similar formulæ to those already used for the corresponding l_x numbers. Then, beginning with the age-period 5/10 the quotient obtained by dividing the integrated value of $Q_{5/10}$ by the integrated value of $l_{5/10}$ will give the value of $E_{5/10}$. It may be noted that by striking out the common factor $\frac{5}{6}$ in the numerator and denominator of the expression for $E_{x/5}$, the work may be simplified. Thus

$$E_{5/10}=\frac{\frac{5(4Q_{7\frac{1}{2}}+Q_5+Q_{10})}{6}}{\frac{5(4l_{7\frac{1}{2}}+l_5+l_{10})}{6}}=\frac{4Q_{7\frac{1}{2}}+Q_5+Q_{10}}{4l_{7\frac{1}{2}}+l_5+l_{10}}$$

Similarly for the age-periods 10/15, 15/20 and 20/25. The value of $E_{0/5}$ must be obtained as in the method already described for the extended Life-Table. From the Q_5 of the abbreviated Life-Table by successive additions of P_4, P_3, P_2, P_1 and P_0 the values of Q_4 . . . Q_0 are obtained.

$$\text{Then } E_{0/5} = \frac{Q_0+Q_1+Q_2+Q_3+Q_4}{P_0+P_1+P_2+P_3+P_4} - \tfrac{1}{2}.$$

When we come to the age-period 25/35, instead of taking the sum of the integrations of 25/30 and 30/35, to obtain which would necessitate interpolating $Q_{27\frac{1}{2}}$ and $Q_{32\frac{1}{2}}$, the integration for the 10-yearly period may be effected in one stage.

Thus after eliminating the common factor $\frac{5}{3}$ in the numerator and denominator,

$$E_{25/35} = \frac{4Q_{30}+Q_{25}+Q_{35}}{4l_{30}+l_{25}+l_{35}}.$$

Similarly for the age-periods 35/45 . . . 75/85.

E_{85} and upwards may be obtained by the formula

$$\frac{(4Q_{90}+Q_{85}+Q_{95})+(4l_{100}+l_{95}+l_{105})}{(4l_{90}+l_{85}+l_{95})+(4l_{100}+l_{95}+l_{105})}.$$

Of various other formulæ of integration which have been worked out and tried only the following one need be mentioned, which has been obtained by substituting the expression for u_0, given at the foot of page 161, in place of u_0 in the complete integration formula, given at the middle of page 162, and then reducing, thus *combining* in one formula the two processes of interpolation and integration. The result arrived at is:—

$$\int_{-n}^{n} u_x dx = \frac{n}{15}\left[\frac{802(u_{-1}+u_1)-93(u_{-3}+u_3)+11(u_{-5}+u_5)}{48}\right]$$

when $n = 2\frac{1}{2}$ the denominator becomes 288.

This formula may be used in the integrations of log p'_x and l_x values as an alternative to first interpolating a central term in a series of six, and then using the short integration formula.

The method of working out an abbreviated Life-Table which has now been described, it is hoped with sufficient clearness to enable it to be followed, has been tested by actual trial, and its advantage will be made apparent by an inspection of the following table of comparison.

In columns (*a*) are given the figures obtained by the extended Method I., and in columns (*b*) the corresponding values obtained by the method of integration just described. The table, in order to save space, only applies to males, but the figures for females have also been worked out with equally close results.

ENGLAND AND WALES, 1891—1900.—MALES.

Age x	l_x (a)	l_x (b)	E_x (a)	E_x (b)	Age x/n	$E_{x/n}$ (a)	$E_{x/n}$ (b)
0	100,000	100,000	44·11	44·10	0—5	52·66	52·65
5	75,093	75,093	53·41	53·42	5—10	51·62	51·61
10	73,482	73,475	49·54	49·55	10—15	47·33	47·33
15	72,592	72,584	45·12	45·12	15—20	43·02	43·01
20	71,235	71,227	40·93	40·93	20—25	38·93	38·92
25	69,446	69,438	36·91	36·92	25—35	33·07	33·07
30	67,323	67,316	33·00	33·01	35—45	25·71	25·71
35	64,716	64,710	29·22	29·24	45—55	19·01	19·01
40	61,505	61,497	25·62	25·62	55—65	13·14	13·13
45	57,655	57,648	22·16	22·17	65—75	8·37	8·36
50	53,015	53,008	18·87	18·87	75—85	5·02	5·00
55	47,424	47,417	15·79	15·79	85—	2·94	2·93
60	40,821	40,813	12·93	12·93	—	—	—
65	33,163	33,156	10·33	10·32	—	—	—
70	24,589	24,581	8·05	8·04	—	—	—
75	15,813	15,806	6·14	6·13	—	—	—
80	8,207	8,202	4·61	4·60	—	—	—
85	3,121	3,118	3·44	3·42	—	—	—
90	767	766	2·56	2·54	—	—	—
95	104	104	1·92	2·08	—	—	—
100	6	6	—	—	—	—	—

It is thus evident that practically all that is required in constructing local life tables may be obtained by this abbreviated method. The only real discrepancy is at E_{95}, which is not a matter of great practical moment.

ADDENDUM.

During the months which have elapsed since this lecture was delivered some further experimental work has been done, directed to finding the extent of differences in the l_x and E_x values which may be deduced from the same data by varying methods.

(1) In the first place an extended Life-Table for England and Wales, 1891—1900 (Males), has been worked out by "Method IV." (see page 159). This method, an extremely laborious one, is essentially similar to one which was suggested by the writer some years ago in a paper read before the Royal Statistical Society in May, 1899, and published in the Journal of that Society, Vol. LXII., Parts III. and IV., and which was afterwards employed in the construction of the Life-Table for London. The writer is now of opinion that the altered details in modes of working of "Method IV.," as compared with those of the earlier method described at the above given reference, are to be preferred.

It is necessary to have some standard of accuracy, and it is proposed to take the results of "Method IV." as the standard to which the results of the other methods which have been employed may be referred. In the succeeding table

the l_x and E_x values of "Method IV." are given at 5-yearly intervals in columns (a), and the differences from these of the results of three other methods are indicated in columns (b), (c), and (d).

(2) The differences in columns (b) are those of the extended method which has been previously described as "Method I."

(3) The differences in columns (c) are those of the values obtained by the abbreviated method which consists in first obtaining a series of log p'_x values at 5-yearly intervals by "Method II." (see pages 157—159), and then integrating in succession the log p'_x and the resulting l_x series.

However, instead of using the process of integration to obtain the logs for passing from log l_5 to log l_{10}, and from l_{10} to log l_{15}, it has been found preferable to adopt the simple process of taking 5 times the log of the mean p_x value obtained from the "years of life" (or "lives at risk"), and the total deaths for each of the age-groups 5-10 and 10-15 respectively, by the formula

$$[\log (2P - d) - \log (2P + d)] \times 5.*$$

The process of integration is to be first applied to the log p'_x series for ages 5, 10, 15, 20, 25 and 30, so as to get the integrated value between ages 15 and 20.

The work of calculating the log p'_x series might be lessened by obtaining the required values after log p'_{85} by differencing the values obtained for ages 60, 65, 70, 75, 80 and 85, and carrying on the series by means of the differences.

The work of intregation has been actually done by the methods of calculation which have been described (see pages 161—163), but the last formula given on page 165, would enable the work to be more readily accomplished, as it dispenses with the necessity of interpolating central terms in each series.

In applying the same formula to the l_x series of numbers in order to avoid the labour of obtaining the hypothetical terms l_0 and l_{-5} by the method of differencing, the following simple formulæ may be used:—

$$l_0 = 6(l_5 + l_{25}) + 20l_{15} - [15(l_{10} + l_{20}) + l_{30}],$$
$$l_{-5} = 6(l_0 + l_{20}) + 20l_{10} - [15(l_5 + l_{15}) + l_{25}].$$

The series does not require to be carried on beyond l_{105} (see formula for Q_{95} on page 164).

(4) The differences given in columns (d) are those of l_x and E_x values obtained by a STILL SIMPLER ABBREVIATED METHOD which only involves using three or four orders of differences, the formulæ required for which will now be given.

In the first place it is to be noted that u_{20} and U_{20} in the foundation series of logs of $2P - d$ and $2P + d$ at age x and upwards are to be derived from the actual data and not by calculation.

Although it was found necessary in the comparative series of Life-Tables, in Part II., to obtain u_{20} by calculation (see second paragraph on page 175), no apparently irrational results were obtained by taking u_{20} from the actual data *as regards Life-Table I. taken alone*, and justification for

* If extreme accuracy be desired it is best to obtain the separate log p_x values for ages 5, 6, 7, 8 and 9, by "Method IV.," and then to sum them.

taking u_{20} from the actual data is found in the final "General Report of the Census for 1901" (see page 63 of that Report).

Formulæ for completing the u_x and U_x series at 5-yearly intervals:—

$$u_0 = 5(u_5 - u_{20}) + u_{25} - 10(u_{10} - u_{15})$$

$$u_{30} = \frac{5(u_{15} + 9u_{25} + 3u_{35}) - (24u_{20} + u_{45})}{40}$$

$$u_{40} = \frac{9(u_{35} + u_{45}) - (u_{25} + u_{55})}{16}$$

$$u_{50} = \frac{9(u_{45} + u_{55}) - (u_{35} + u_{65})}{16}$$

$$u_{60} = \frac{9(u_{55} + u_{65}) - (u_{45} + u_{75})}{16}$$

$$u_{70} = \frac{3(u_{45} + 30u_{65} + 20u_{75}) - 5(4u_{55} + u_{85})}{128}$$

$$u_{80} = \frac{5(3u_{65} + 9u_{75} + u_{85}) - (u_{55} + 40u_{70})}{24}$$

$$u_{90} = u_{65} + 10(u_{75} - u_{80}) - 5(u_{70} - u_{85})$$

$$u_{95} = u_{70} + 10(u_{80} - u_{85}) - 5(u_{75} - u_{90})$$

The correctness of the results of the last four formulæ is to be checked by finding an identical value of u_{95} by $u_{95} = u_{45} + 10(u_{65} - u_{75}) - 5(u_{55} - u_{85})$, or by differencing the series from u_{65} to u_{95} inclusive and finding that there is a constant fourth difference.

Calculation of log p'_x values.

From the u_x and U_x series thus completed it is now easily possible to calculate a series of log p'_x values for ages 10 to 85 inclusive at 5-yearly intervals by a formula already explained at the foot of page 153, each value being calculated from a series of five consecutive equidistant values of u_x and U_x of which the x of log p'_x is taken as 0 in the series u_{-10}, u_{-5}, u_0, u_5, u_{10}. Thus

$$\log p'_0 = \left\{u_0 + \log[8(u_{-5} - u_5) - (u_{-10} - u_{10})]\right\} - \left\{U_0 + \log[8(U_{-5} - U_5) - (U_{-10} - U_{10})]\right\}$$

The series can be completed for ages after 85 by differencing the values for ages 65, 70, 75, 80 and 85, and carrying on the series to log p'_{110}.

Integration of log p'_x series.

The summed log p_x values between ages 5 and 10, and 10 and 15, are to be obtained by the simpler method described under (3) on page 167.

The integration between ages 15 and 20 may then be effected from the values for ages 10, 15, 20 and 25, which correspond to the *relative* ages, $-7\frac{1}{2}$, $-2\frac{1}{2}$, $2\frac{1}{2}$ and $7\frac{1}{2}$.

The formula of integration is, u_x denoting log p'_x

$$\int_{-n}^{n} u_x dx = \frac{n}{3}\left[\frac{13(u_{-1} + u_1) - (u_{-3} + u_3)}{4}\right]$$

which when $n = 2\frac{1}{2}$ becomes

$$\frac{130(u_{-2\frac{1}{2}} + u_{2\frac{1}{2}}) - 10(u_{-7\frac{1}{2}} + u_{7\frac{1}{2}})}{48}$$

The simple processes of calculation therefore to be applied to each series of four log p'_x values are (1) to add together the two middle and two outside logs, (2) to take the "co-logs" of the two sums (*i.e.*, their differences from 0), (3) to apply the formula to the co-logs, the result being the co-log of the required integrated value.

EXAMPLES.

x	log $p'x$		
10	$\bar{1}$·9989149	$\bar{1}$·9987360	$\bar{1}$·9989149
15	$\bar{1}$·9987360	$+\bar{1}$·9980524	$+\bar{1}$·9975809
20	$\bar{1}$·9980524	$\bar{1}$·9967884	$\bar{1}$·9964958
25	$\bar{1}$·9975809	0·0032116	0·0035042
		$\times 130$	
		963480	
		32116	
		4175080	
		-350420	
		0·3824660 ÷ 48 = 0·0079680	
		∴ required log = $\bar{1}$·9920320	

x	log $p'x$		
95	$\bar{1}$·7970020	$\bar{1}$·7213533	$\bar{1}$·7970020
100	$\bar{1}$·7213533	$+\bar{1}$·6251749	$+\bar{1}$·5050623
105	$\bar{1}$·6251749	$\bar{1}$·3465282	$\bar{1}$·3020643
110	$\bar{1}$·5050623	0·6534718	0·6979357
		$\times 130$	
		196041540	
		6534718	
		84·9513340	
		$-6·9793570$	
		77·9719770 ÷ 48 = 1·6244162	
		∴ required log = $\bar{2}$·3755838	

Integration of l_x numbers.

The log l_x values being now readily obtained at 5-yearly intervals by successive additions of the integrated log p_x values, the corresponding numerical values are to be taken out.

Having completed the required series by interpolating a hypothetical l_0 value (which of course must not be confounded with the true l_0 number

of the Life-Table), by the formula $l_0 = 4(l_5 + l_{15}) - (6l_{10} + l_{20})$, the same formula of integration may be applied to the series l_0, l_5, l_{10}, l_{15}, so as to obtain the years of life lived by l_5 persons in the interval from age 5 to age 10, and so on to the interval between age 100 and age 105.

The remaining stages in the construction of Life-Tables have been already described.

Calculation of $E_{x/n}$ values.

Having completed the Q_x series by interpolating a hypothetical Q_0 value by a formula corresponding to that above given for l_0,

$$E_{5/10} = \frac{13(Q_5 + Q_{10}) - (Q_0 + Q_{15})}{13(l_5 + l_{10}) - (l_0 + l_{15})}$$

(The l_0 value is of course the calculated one).

By similar formulæ the values of $E_{10/15}$, $E_{15/20}$, and $E_{20/25}$ may be obtained.

The remaining $E_{x/n}$ values are to be obtained as already described on page 165.

Table showing l_x and E_x values for England and Wales, 1891—1900 (Males), calculated by extended "Method IV.," and the differences from these of the corresponding figures obtained by other methods as previously indicated.

	l_x				E_x			
	(*a*)	(*b*)	(*c*)	(*d*)	(*a*)	(*b*)	(*c*)	(*d*)
0	100,000	0	0	0	44·17	−0·06	0	+0·03
5	75,093	0	0	0	53·50	−0·09	0	+0·04
10	73,494	− 12	− 2	− 2	49·62	−0·08	0	+0·04
15	72,599	− 7	+ 2	+ 2	45·20	−0·08	−0·01	+0·04
20	71,245	− 10	0	+36	41·01	−0·08	−0·01	+0.02
25	69,454	− 8	− 6	+50	37·00	−0·09	−0·01	+0·01
30	67,357	− 34	+11	+76	33·07	−0·07	−0·01	0
35	64,876	−160	−24	− 2	29·24	−0·02	0	+0 03
40	61,637	−132	−16	+14	25·64	−0·02	0	+0·02
45	57,758	−103	−13	+22	22·19	−0·03	−0·01	+0·01
50	53,181	−166	−19	+14	18·88	−0·01	−0·01	+0·02
55	47,622	−198	−20	+23	15·78	+0·01	0	+0·02
60	40,970	−149	−14	+52	12·93	0	−0·01	+0.01
65	33,264	−101	−15	+71	10·33	0	−0·01	+0·01
70	24,668	− 79	−14	+57	8·05	0	0	0
75	15,874	− 61	−14	+32	6·14	0	0	+0·01
80	8,244	− 37	−12	+ 7	4·62	−0·01	−0·02	0
85	3,136	− 15	− 8	− 2	3·44	0	−0·02	0

The above is presented as the most complete example of a number of comparative Tables which have been prepared.

The conclusions to which the writer has finally been led by inference from them are:—

(1) That for the purposes of Public Health comparative statistics it is scarcely necessary to use any extended method in constructing a Life-Table.

(2) That if extreme accuracy be desired the shortened method by which the results indicated under columns (*b*) have been obtained may be used.

(3) That for ordinary practical purposes the simple and easy method by which the results given under columns (*d*) have been obtained is amply sufficient.

(4) That for the sake of minute accuracy in comparison it is desirable that different Life-Tables shall have been constructed by the *same* method.

(5) That when this has been the case the differences between two Life-Tables compared are nearly the same by all the methods which have been tried, and therefore the differences of Life-Tables Nos. II. and III. from Life-Table No. I., as given in Part II. of the Lecture, may be taken to be practically identical with those which would have been obtained if another method had been used in their construction.

PART II.

COMPARISON OF THE MORTALITY FROM PHTHISIS IN ENGLAND AND WALES DURING THE DECENNIA, 1881—1890 AND 1891—1900.

The most simple and obvious method of making such a comparison is to contrast the death-rates per million from this cause for each of the usual age-groups, as shown in the following table.

From the necessity of economising space the foundation numbers and the foundation work are not here set down in detail.

It must be understood, however, that the "years of life" in each decennium (*i.e.*, ten times the mean annual population), have been calculated from the data of the censuses of 1881, 1891 and 1901, by the method of Mr. A. C. Waters, already alluded to, and that the numbers of deaths for the decennium 1891—1900 have been compiled from the Annual Reports of the Registrar-General.

ENGLAND AND WALES. DEATH-RATES FROM PHTHISIS PER MILLION LIVING.

	Males			Females		
Ages	1881—90	1891—1900	Changes per cent	1881—90	1891—1900	Changes per cent
0—5	553	441	−20·19	518	385	−25·64
5—10	254	174	−31·64	328	239	−27·30
10—15	344	234	−32·12	703	502	−28·61
15—20	1294	995	−23·14	1810	1290	−28·71
20—25	2342	1887	−19·46	2327	1591	−31·63
25—35	3039	2369	−22·05	2803	1923	−31·39
35—45	3579	3095	−13·50	2741	2121	−22·62
45—55	3507	3144	−10·34	2063	1642	−20·42
55—65	2921	2618	−10·39	1516	1239	−18·30
65—75	1824	1584	−13·17	980	807	−17·68
75—85	744	587	−21·09	428	371	−13·17
85 & upwards	273	282	+ 3·23	193	208	+ 7·34
All ages	1853	1580	−14·75	1615	1214	−24·86

From the preceding table it is evident (1) that the comparison of the figures for the two decennia shows a very marked decrease in the mortality from Phthisis at all age-periods except for ages 85 and upwards, such decrease affecting females much more than males, (2) that notwithstanding the decrease, Phthisis is still exacting a very large death-toll.

It may be within the knowledge of some of those addressed that the present writer contributed a paper to the "Transactions of the British Congress on Tuberculosis" in 1901, in which it was sought to measure the effects of Phthisis as a cause of mortality during the ten years 1881—90 by finding answers to the questions, supposing that Phthisis had been abolished as a cause of mortality: (1) how much longer would people live? and (2) how many more would be alive at certain ages?

It was shown that question (1) could be answered with a very near approach to accuracy by the modified short method of Life-Table construction employed, but it was only claimed as regards question (2) that by this method the answers would be right in *direction*, but not in precise *degree*.

When the work embodied in this Lecture was first contemplated the writer proposed to undertake a similar task based on the data for 1891—1900, but instead of using the short method, to employ an "extended" method for the sake of obtaining with accuracy answers to question (2), *i.e.*, correct l_x values

On subsequent reflection, however, it appeared that Life-Table methods might be applied to the question of Phthisis mortality in yet another way.

The total extinction of Phthisis may be regarded at present as an almost Utopian dream, or scarcely within measurable distance of realization, and while it may be of interest to employ exact statistical methods to measure the total effects of Phthisis on longevity, it may be of still more interest and advantage to apply these methods in measuring the effects of such an actually observed concrete instance of *diminished* mortality as is set forth in the preceding table.

The complete carrying out of this idea has necessitated the construction of three concurrent Life-Tables, both for males and females, by methods already described in detail.

(1) A Life-Table for England and Wales based on the total mortality from all causes during 1891—1900.

(2) A Life-Table based on the hypothesis that during this period Phthisis as a cause of mortality had been totally abolished.

(3) A Life-Table based on the hypothesis that during 1891—1900 Phthisis had been prevailing as a cause of mortality with the same proportionate intensity as in 1881—90.

These three Life-Tables will, in what follows, be denoted respectively as I., II., and III.

Before proceeding to give the results obtained it is necessary to give some explanation as to the modes employed for obtaining the numbers of deaths as the foundations for Life-Tables II. and III.

First, as regards II., to take an example, we find that out of a total number of years of life for males living in the age-group 25 to 35 of 22,724,929, there occurred 153,545 deaths, of which 53,832 were due to Phthisis.

Now it will be obvious that the supposed extinction of Phthisis could not possibly mean a nett gain of 53,832 lives, for if there had been no Phthisis, these 53,832 individuals would each have been exposed, on the average for half the period of time involved, to the same risks which accounted for 153,545—

53,832 = 99,713 deaths, and therefore a deduction must be made from the 53,832 of those of their number who would have died from other causes if they had not died from phthisis.

It will also be obvious that the years of life out of which the deaths occurred will have been increased by $\frac{1}{2}$ 53,832 = 26,916.

The deduction to be made from 53,832 will be, therefore

$$\frac{99{,}713 \times 53{,}832}{22{,}724{,}929 + 26{,}916} = 118.$$

and the total number of deaths in the age-group on the hypothesis in question will be 153,545 − (53,832 − 118) = 99,831.

To represent the calculation in a general formula let

P = the years of life for any age-group,
D = total number of deaths from all causes,
d = number of deaths which have occurred from some cause supposed to be eliminated,
D' = total number of deaths after elimination of this cause.

$$\text{then} \quad D' = (D - d) + \frac{(D-d) \times d}{2P + d},$$

and the rate of mortality per unit in the age-group will be represented by

$$\frac{D'}{P + \frac{1}{2}(D - D')}.$$

In the next place to illustrate the work required for Life-Table III., the corresponding instance may be taken.

In 1881—90, out of 19,456,609 years of life for males in the age-group 25—35 there occurred 59,126 deaths from Phthisis. From these figures, in conjunction with those already quoted for 1891—1900, it may readily be calculated that the number of deaths in 1891—1900, at the rate of 1881—90 would have been 69,058 that is an increase of 15,226 on the number actually recorded. But we cannot take the number 15,226 as the correct number of *extra* deaths which would have occurred on the hypothesis.

If we look at the separate causes of mortality as "machine-guns," each mowing down so many individuals in a certain time, and suppose that one of these machine-guns, Phthisis, had been made to fire more rapidly during 1891—1900, it is evident that the other machine-guns would not have had the same chance of killing the same number of individuals as those actually killed by them.

In order, therefore, that we may obtain the correct number of extra deaths, on the hypothesis, a deduction must be made from the 15,226 of the number of those among them who would have died from other causes if they had not died from an increased intensity of Phthisis as a cause of mortality.

The mean population, or years of life, out of which the extra deaths are to occur, having been reduced by half the number of actual deaths from phthisis, the deduction from the number 15,226 will be found thus: —

$$\frac{99{,}713 \times 15{,}226}{22{,}724{,}929 - 26{,}916} = 33,$$

and the corrected number of extra deaths on the hypothesis is 15,226 − 33 = 15,193.

In order to represent this calculation, too, by a general formula, let

P = years of life in the age-group for the decennium,
D = total number of deaths from all causes,
d = number of deaths which have actually occurred from Phthisis,
d' = some additional number of deaths supposed to have occurred from Phthisis,
D' = total number of deaths in the age-group by hypothesis,

$$\text{then } D' = D + d' - \frac{(D-d)\times d'}{2P-d}.$$

and the rate of mortality per unit for the age-group will be represented by

$$\frac{D'}{P - \frac{1}{2}(D'-D'}$$

(In actual practice it is found that in the above formulæ the denominators $2P-d$ and $2P+d$ may be replaced by $2P$ without obtaining more than a fractional variation in the results. In the work for the succeeding tables $2P$ has been used.)

The work done in calculating the numbers of deaths as foundations for Life-Tables II. and III. may be first of all set forth in the form of comparative death-rates reckoned per million living, as shown in the following table.

In columns (*a*) are given the death-rates from *all causes.*

In columns (*b*) are given the differences from (*a*) on the hypothesis that Phthisis had been eliminated as a cause of mortality during the decennium, thus *measuring the proportionate numbers of lives which would have been saved if there had been no Phthisis.*

In columns (*c*) are given the differences from (*a*) on the hypothesis that Phthisis had prevailed as a cause of mortality during the decennium with the same proportionate intensity as in 1881—90, thus *measuring the proportionate numbers of lives actually saved* during 1891—1900 by the reduction in Phthisis mortality as compared with 1881—90.

England and Wales, 1891—1900. Mean Annual Death-rates per million.

	Males.			Females.		
Ages.	(*b*)	(*a*)	(*c*)	(*b*)	(*a*)	(*c*)
0—5	− 428	62710	+108	− 375	52797	+129
5—10	− 173	4309	+ 80	− 238	4370	+ 89
10—15	− 233	2448	+110	− 501	2566	+201
15—20	− 993	3789	+299	−1288	3665	+519
20—25	−1884	5060	+455	−1589	4461	+735
25—35	−2364	6757	+669	−1919	6082	+878
35—45	−3082	11498	+481	−2113	9593	+618
45—55	−3119	18946	+360	−1631	14741	+418
55—65	−2576	34949	+299	−1222	28438	+274
65—75	−1529	70386	+232	− 783	60723	+168
75—85	− 544	146118	+145	− 347	130598	+ 53
85 & upwards	− 241	286872	− 6	− 180	261418	− 10
All ages	−1569	19316	+323	−1206	17142	+440

In the calculation of the figures for the above table the foundation numbers for the age-group 0—5 have been dealt with *collectively*, but in the calculations for the Life-Tables to follow the foundation numbers have been treated for the *separate* age-groups 0—3 mos., 3—6 mos., 6—12 mos., 1—2 yrs., 2—3 yrs., 3—4 yrs., and 4—5 yrs.

It may also be noted that in the above table, the foundation numbers for the age-groups 15—20, and 20—25, have been taken *separately*, whereas in the calculations for the Life-Tables they have been taken *in combination* as 15—25, since it was found, after the expenditure of a good deal of labour, that it was only thus possible, in the case of the *female* Life-Tables, to arrive at rational results, this pointing almost certainly to the existence of mis-statements of age in the census Reports and in the Death Registers.

Reserving until the end the presentation of the actual results of the three Life-Tables I., II. and III., for each successive year of age, as regards p_x, l_x, and E_x values, it is proposed at this point to set forth some of the most interesting and useful deductions from these results, especially such as may be made to apply to *groups of ages*.

In the first place there will be given a table showing

(*a*) the mean numbers living within the limits of the usual age-groups *out of a million born*, according to Life-Table I., obtained from the P_x column of the Life-Table by taking the sum of $P_x + P_{x+1} + \ldots + P_{x+n-1}$ and dividing this by n. (From a geometrical point of view this is, of course, simply taking the mean height of the ordinates of a section of the l_x curve between the limits x and $x+n$, by a method not quite so accurate as that depending upon an application of the integral calculus already described, but giving results not very greatly differing from those to be obtained by this last-named method.)

(*b*) The corresponding numbers in *excess* over Life-Table I. according to Life-Table II.

(*c*) The corresponding numbers of *deficiency* from Life-Table I., according to Life-Table III.

ENGLAND AND WALES, 1891—1900.

Mean numbers of those living within the limits of the age-groups indicated *out of a million born*.

Ages.	Males. (*b*)	Males. (*a*)	Males. (*c*)	Females. (*b*)	Females. (*a*)	Females. (*c*)
0—5	+ 1,057	798,466	− 334	+ 930	828,357	− 359
5—10	+ 1,984	741,167	− 555	+ 1,887	772,396	− 681
10—15	+ 2,512	730,570	− 881	+ 3,117	760,867	− 1,155
15—20	+ 4,489	719,563	−1,488	+ 6,110	749,486	− 2,392
20—25	+ 9,504	703,672	−2,780	+11,435	734,263	− 4,621
25—35	+21,212	672,405	−5,750	+21,373	704,880	− 8,894
35—45	+37,453	613,971	−8,801	+33,740	651,265	−12,966
45—55	+49,961	528,527	−9,731	+41,354	577,856	−14,343
55—65	+51,345	406,411	−8,765	+40,729	468,771	−13,177
65—75	+36,633	245,506	−5,915	+29,931	306,683	− 9,264
75—85	+13,835	86,422	−2,235	+12,593	121,843	− 3,802
85 and upwards	+ 879	5,370	− 142	+ 907	8,578	− 269

The next table shows the results of the preceding table in proportions *per million in each age-group.* Taking as a standard a million living in each age-group according to Life-Table I., columns (*b*) show how many *more* than a million are living in each age-group according to Life-Table II., and columns (*c*) how many *fewer* than a million are living according to Life-Table III.

	Males.		Females.	
Ages.	(*b*)	(*c*)	(*b*)	(*c*)
0—5	+ 1,324	− 418	+ 1,123	− 433
5—10	+ 2,677	− 749	+ 2,443	− 882
10—15	+ 3,438	− 1,206	+ 4,097	− 1,518
15—20	+ 6,239	− 2,068	+ 8,152	− 3,192
20—25	+ 13,506	− 3,951	+ 15,573	− 6,293
25—35	+ 31,546	− 8,551	+ 30,321	− 12,618
35—45	+ 61,001	− 14,335	+ 51,807	− 19,909
45—55	+ 94,529	− 18,412	+ 71,565	− 24,821
55—65	+ 126,338	− 21,567	+ 86,885	− 28,110
65—75	+ 149,214	− 24,093	+ 97,596	− 30,207
75—85	+ 160,087	− 25,861	+ 103,354	− 31,304
85 and upwards	+ 163,687	− 26,443	+ 105,736	− 31,359

The next table shows the average expectation of life, or mean after-lifetime of all the individuals included within the limits of the usual age-groups ($E_{x/n}$), the values given for "all ages" representing the average Life-capital of the Life-Table population:—

Columns (*a*), according to Life-Table I.

Columns (*b*), the *increase* over Life-Table I., according to Life-Table II.

Columns (*c*) the *decrease* from Life-Table I., according to Life-Table III.

ENGLAND AND WALES, 1891—1900. $E_{x/n}$.

	Males.			Females.		
Ages.	(*b*)	(*a*)	(*c*)	(*b*)	(*a*)	(*c*)
0—5	+ 2·70	52·66	− 0·53	+ 2·27	55·05	− 0·80
5—10	+ 2·83	51·62	− 0·56	+ 2·36	53·92	− 0·82
10—15	+ 2·84	47·33	− 0·54	+ 2·31	49·70	− 0·80
15—20	+ 2·74	43·02	− 0·52	+ 2·14	45·42	− 0·74
20—25	+ 2·49	38·93	− 0·45	+ 1·84	41·31	− 0·62
25—35	+ 1·92	33·07	− 0·29	+ 1·33	35·38	− 0·39
35—45	+ 1·19	25·71	− 0·14	+ 0·73	27·86	− 0·18
45—55	+ 0·60	19·01	− 0·07	+ 0·33	20·73	− 0·08
55—65	+ 0·22	13·14	− 0·03	+ 0·12	14·31	− 0·03
65—75	+ 0·05	8·37	− 0·01	+ 0·03	9·08	− 0·01
75—85	+ 0·01	5·02	± 0·00	± 0·00	5·45	± 0·00
85 and upwards	± 0·00	2·94	± 0·00	± 0·00	3·20	± 0·00
All ages	+ 0·98	32·32	− 0·18	+ 0·71	33·52	− 0·23

In the next place the $E_{x/n}$ values given in the preceding table will be applied to the *census* population, as enumerated in 1901, to deduce the average Life-capital for males, females, and persons.

It will be seen that these values are considerably in excess of those of the *Life-Table* population, because in a census of a population among whom the births have been exceeding the deaths, there will be a larger proportion of those living in the earlier age-groups of life than in a stationary Life-Table population.

Average Life-capital of the population of England and Wales as enumerated at the census of 1901

(*a*) according to Life-Table I.,
(*b*) *excess* according to Life-Table II.,
(*c*) *deficiency* according to Life-Table III.

	(*b*)		(*a*)		(*c*)
Males	+1·96	.....	36·13	.....	−0·35
Females	+1·46	.....	37·59	.....	−0·48
Persons	+1·71	.....	36·88	.....	−0·42

The last of this series of comparisons will be devoted to showing how the total expectation of life *at birth* is, on the average, distributed over the main age-periods of life

(*a*) according to Life-Tables I.,
(*b*) differences of *excess* according to Life-Table II.,
(*c*) differences of *deficiency* according to Life-Table III.

	Males.				Females.		
Age-periods.	(*b*)	(*a*)	(*c*)		(*b*)	(*a*)	(*c*)
0—5	+0·01	3·99	−0·01	.....	+0·01	4·14	±0·00
5—15	+0·02	7·36	−0·01	.....	+0·02	7·67	−0·01
15—65	+1·67	29·33	−0·35	.....	+1·46	31·45	−0·53
65 and upwards	+0·52	3·43	−0·08	.....	+0·44	4·47	−0·14
Totals	+2·22	44·11	−0·45	.....	+1·93	47·73	−0·68

The leading points brought out by this series of comparisons are:—

1. If there had been no mortality from Phthisis in England and Wales during 1891—1900

Males would have had

(1) expectation of life at birth increased by 2·22 years,

(2) average existence during working period of life lengthened by 1·67 years,

(3) average ife-capital increased by 1·96 years.

Females would have had

(1) expectation of life at birth increased by 1·93 years,

(2) average existence during working period of life lengthened by 1·46 years.

(3) average life-capital lengthened by 1·46 years.

2. As the result of the decrease in the mortality from Phthisis during 1891—1900 as compared with 1881—90,
Males have had

(1) expectation of life at birth increased by 0·45 year,

(2) working period of life prolonged by 0·35 year,

(3) average increase in life-capital of 0·35 year.

Females have had

(1) expectation of life at birth increased by 0·68 year

(2) working period of life prolonged by 0·53 year,

(3) average increase in life-capital of 0·48 year.

An inspection of the diagrams and detailed Life-Tables will serve to demonstrate many other interesting points which time and space will not permit to be fully referred to.

The diagrams showing p_x, l_x, and E_x curves have only been prepared for males. Those drawn on a large scale shown at the Lecture extend from age 0 to age 85. On the smaller scale upon which they are published they have had to be curtailed.

The Lecturer desires to make acknowledgment of the help received in preparing the diagrams from Mr. W. R. Maddison of Haydock, and also of the great aid given by Mr. Chas. Dickinson, Sanitary Inspector of Haydock, in performing a large portion of the routine calculation in the working out of the series of Life Tables. Without such efficient help it would not have been possible to complete the task.

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THREE LIFE-TABLES FOR ENGLAND AND WALES, 1891—1900.

No. I. based on the total mortality from all causes.

No. II. based on the hypothesis that there had been no deaths from Phthisis during 1891—1900.

No. III. based on the hypothesis that Phthisis had prevailed during 1891—1900 with the same proportionate intensity as in 1881—90.

In order to economise space it is only proposed to give the p_x, l_x and E_x values for each separate age from age 0 to age 85, and to indicate

in columns (*a*) the results of Life-Tables I.,

in columns (*b*) the differences from Life-Table I. of the corresponding values of Life-Tables II.,

in columns (*c*) the differences from Life-Table I. of the corresponding values of Life-Table III.

Chance of Living One Year at Each Age x

p_x.

Age x.	Males. (b)	Males. (a)	Males. (c)		Females. (b)	Females. (a)	Females. (c)
0	+·00061	·82861	−·00028	.....	+·00050	·85963	−·00025
1	+·00061	·94696	−·00012	.....	+·00053	·95061	−·00018
2	+·00034	·97922	−·00005	.....	+·00029	·97990	−·00007
3	+·00023	·98685	−·00003	.....	+·00024	·98669	−·00003
4	+·00021	·99033	−·00002	.....	+·00021	·99047	−·00006
5	+·00025	·99295	−·00006	.....	+·00021	·99305	−·00010
6	+·00021	·99482	−·00008	.....	+·00021	·99484	−·00010
7	+·00016	·99612	−·00009	.....	+·00022	·99605	−·00008
8	+·00011	·99698	−·00009	.....	+·00023	·99685	−·00008
9	+·00009	·99749	−·00008	.....	+·00027	·99732	−·00009
10	+·00010	·99774	−·00009	.....	+·00032	·99756	−·00012
11	+·00014	·99778	−·00010	.....	+·00039	·99761	−·00015
12	+·00021	·99767	−·00010	.....	+·00049	·99753	−·00019
13	+·00031	·99746	−·00013	.....	+·00060	·99737	−·00024
14	+·00044	·99718	−·00015	.....	+·00072	·99715	−·00030
15	+·00060	·99686	−·00017	.....	+·00085	·99691	−·00036
16	+·00076	·99654	−·00021	.....	+·00100	·99666	−·00042
17	+·00095	·99622	−·00026	.....	+·00115	·99641	−·00048
18	+·00115	·99591	−·00030	.....	+·00129	·99618	−·00054
19	+·00134	·99563	−·00035	.....	+·00142	·99597	−·00060
20	+·00154	·99537	−·00040	.....	+·00156	·99577	−·00065
21	+·00173	·99513	−·00045	.....	+·00167	·99559	−·00070
22	+·00191	·99491	−·00050	.....	+·00176	·99543	−·00076
23	+·00207	·99470	−·00054	.....	+·00183	·99526	−·00084
24	+·00221	·99451	−·00059	.....	+·00189	·99509	−·00084
25	+·00232	·99431	−·00062	.....	+·00192	·99490	−·00087
26	+·00241	·99409	−·00065	.....	+·00195	·99469	−·00090
27	+·00250	·99383	−·00067	.....	+·00197	·99446	−·00091
28	+·00257	·99355	−·00068	.....	+·00199	·99420	−·00091
29	+·00262	·99325	−·00069	.....	+·00201	·99391	−·00089
30	+·00267	·99291	−·00068	.....	+·00203	·99361	−·00088
31	+·00270	·99255	−·00067	.....	+·00205	·99329	−·00085
32	+·00273	·99216	−·00065	.....	+·00206	·99296	−·00082
33	+·00277	·99174	−·00063	.....	+·00208	·99262	−·00079
34	+·00280	·99129	−·00060	.....	+·00209	·99227	−·00075
35	+·00282	·99083	−·00058	.....	+·00210	·99192	−·00072
36	+·00285	·99036	−·00055	.....	+·00211	·99158	−·00069
37	+·00289	·98988	−·00052	.....	+·00211	·99126	−·00065
38	+·00293	·98939	−·00050	.....	+·00209	·99095	−·00062
39	+·00296	·98889	−·00048	.....	+·00207	·99064	−·00060

p_x.

Age x.	Males (b)	Males (a)	Males (c)		Females (b)	Females (a)	Females (c)
40	+·00301	·98836	−·00047	.....	+·00203	·99033	−·00058
41	+·00305	·98780	−·00045	.....	+·00200	·98999	−·00056
42	+·00309	·98720	−·00043	.....	+·00195	·98963	−·00054
43	+·00312	·98656	−·00042	.....	+·00192	·98922	−·00052
44	+·00315	·98586	−·00040	.....	+·00188	·98877	−·00050
45	+·00317	·98511	−·00039	.....	+·00184	·98826	−·00048
46	+·00317	·98430	−·00038	.....	+·00181	·98768	−·00046
47	+·00317	·98342	−·00037	.....	−·00176	·98704	+·00045
48	+·00316	·98248	−·00036	.....	+·00171	·98633	−·00044
49	+·00314	·98147	−·00035	.....	+·00166	·98554	−·00042
50	+·00310	·98040	−·00035	.....	+·00161	·98469	−·00040
51	+·00307	·97925	−·00034	.....	+·00156	·98377	−·00038
52	+·00302	·97803	−·00033	.....	+·00151	·98277	−·00036
53	+·00296	·97674	−·00033	.....	+·00146	·98170	−·00035
54	+·00290	·97536	−·00032	.....	+·00141	·98055	−·00033
55	+·00283	·97389	−·00031	.....	+·00136	·97932	−·00033
56	+·00277	·97231	−·00031	.....	+·00133	·97797	−·00030
57	+·00270	·97060	−·00030	.....	+·00128	·97651	−·00029
58	+·00262	·96875	−·00029	.....	+·00124	·97491	−·00028
59	+·00252	·96675	−·00029	.....	+·00119	·97316	−·00028
60	+·00243	·96456	−·00028	.....	+·00116	·97122	−·00026
61	+·00233	·96217	−·00028	.....	+·00111	·96909	−·00025
62	+·00223	·95956	−·00027	.....	+·00107	·96674	−·00024
63	+·00213	·95669	−·00026	.....	+·00103	·96414	−·00023
64	+·00201	·95356	−·00026	.....	+·00098	·96128	−·00022
65	+·00190	·95013	−·00025	.....	+·00094	·95813	−·00020
66	+·00178	·94638	−·00024	.....	+·00089	·95468	−·00019
67	+·00166	·94230	−·00023	.....	+·00084	·95091	−·00018
68	+·00154	·93786	−·00023	.....	+·00078	·94679	−·00017
69	+·00142	·93304	−·00022	.....	+·00074	·94232	−·00016
70	+·00130	·92780	−·00021	.....	+·00069	·93746	−·00015
71	+·00118	·92213	−·00020	.....	+·00064	·93219	−·00013
72	+·00106	·91601	−·00020	.....	+·00059	·92651	−·00012
73	+·00094	·90941	−·00019	.....	+·00054	·92039	−·00010
74	+·00083	·90230	−·00017	.....	+·00049	·91381	−·00009
75	+·00073	·89467	−·00016	.....	+·00044	·90676	−·00008
76	+·00063	·88650	−·00015	.....	+·00039	·89921	−·00007
77	+·00053	·87776	−·00014	.....	+·00034	·89115	−·00006
78	+·00045	·86843	−·00013	.....	+·00031	·88256	−·00004
79	+·00037	·85851	−·00012	.....	+·00027	·87344	−·00004

p_x.

Age x.	Males (b)	Males (a)	Males (c)		Females (b)	Females (a)	Females (c)
80	+·00031	·84796	−·00010	.....	+·00024	·86377	−·00003
81	+·00026	·83679	−·00009	.....	+·00021	·85354	−·00002
82	+·00021	·82498	−·00007	.....	+·00018	·84274	−·00001
83	+·00018	·81253	−·00007	.....	+·00015	·83137	±·00000
84	+·00016	·79942	−·00005	.....	+·00014	·81941	+·00001
85	+·00015	·78566	−·00004	.....	+·00013	·80688	+·00001
90	+·00036	·70717	+·00005	.....	+·00018	·73559	+·00001
95	+·00092	·61409	+·00010	.....	+·00041	·65099	−·00001
100	+·00172	·51049	+·00011	.....	+·00081	·55602	−·00006

Number of Survivors at Each Age x out of a Million Born

l_x.

0	—	1,000,000	—	.....	—	1,000,000	—
1	+ 608	828,613	− 279	.....	+ 505	859,627	− 244
2	+ 1,083	784,666	− 366	.....	+ 941	817,167	− 387
3	+ 1,322	768,364	− 400	.....	+ 1,166	800,740	− 439
4	+ 1,483	758,262	− 417	.....	+ 1,341	790,082	− 461
5	+ 1,635	750,926	− 423	.....	+ 1,490	782,554	− 502
6	+ 1,812	745,633	− 465	.....	+ 1,646	777,118	− 580
7	+ 1,963	741,771	− 519	.....	+ 1,799	773,109	− 655
8	+ 2,071	738,896	− 582	.....	+ 1,957	770,058	− 718
9	+ 2,149	736,664	− 644	.....	+ 2,128	767,630	− 778
10	+ 2,212	734,818	− 705	.....	+ 2,326	765,577	− 847
11	+ 2,280	733,157	− 767	.....	+ 2,565	763,709	− 934
12	+ 2,376	731,530	− 835	.....	+ 2,860	761,886	− 1,048
13	+ 2,524	729,826	− 908	.....	+ 3,223	760,010	− 1,194
14	+ 2,743	727,972	− 1,004	.....	+ 3,669	758,012	− 1,377
15	+ 3,055	725,919	− 1,096	.....	+ 4,208	755,854	− 1,600
16	+ 3,476	723,645	− 1,221	.....	+ 4,844	753,519	− 1,866
17	+ 4,019	721,142	− 1,370	.....	+ 5,585	751,001	− 2,175
18	+ 4,693	718,415	− 1,547	.....	+ 6,430	748,307	− 2,525
19	+ 5,503	715,479	− 1,757	.....	+ 7,376	745,451	− 2,919

l_x.

Age x	Males (b)	Males (a)	Males (c)		Females (b)	Females (a)	Females (c)
20	+ 6,451	712,351	−1,998	.....	+ 8,417	742,447	− 3,351
21	+ 7,531	709,052	−2,274	.....	+ 9,545	739,312	− 3,822
22	+ 8,737	705,599	−2,581	.....	+10,750	736,056	− 4,326
23	+10,057	702,010	−2,920	.....	+12,014	732,690	− 4,861
24	+11,477	698,295	−3,287	.....	+13,325	729,216	− 5,421
25	+12,077	694,463	−3,679	.....	+14,663	725,634	− 6,004
26	+14,542	690,512	−4,089	.....	+16,009	721,936	− 6,603
27	+16,154	686,431	−4,513	.....	+17,359	718,105	− 7,210
28	+17,806	682,201	−4,943	.....	+18,710	714,125	− 7,815
29	+19,484	677,806	−5,373	.....	+20,059	709,982	− 8,410
30	+21,178	673,231	−5,797	.....	+21,405	705,661	− 8,987
31	+22,879	668,460	−6,209	.....	+22,741	701,154	− 9,541
32	+24,578	663,479	−6,604	.....	+24,068	696,452	−10,067
33	+26,268	658,277	−6,979	.....	+25,381	691,551	−10,560
34	+27,943	652,842	−7,331	.....	+26,681	686,448	−11,019
35	+29,600	647,160	−7,656	.....	+27,965	681,141	−11,443
36	+31,236	641,226	−7,953	.....	+29,231	675,636	−11,832
37	+32,850	635,045	−8,224	.....	+30,473	669,947	−12,187
38	+34,444	628,621	−8,470	.....	+31,678	664,095	−12,512
39	+36,018	621,955	−8,692	.....	+32,842	658,088	−12,807
40	+37,568	615,046	−8,893	.....	+33,959	651,932	−13,076
41	+39,093	607,887	−9,071	.....	+35,024	645,626	−13,319
42	+40,588	600,471	−9,230	.....	+36,030	639,164	−13,538
43	+42,048	592,786	−9,369	.....	+36,977	632,533	−13,734
44	+43,463	584,819	−9,489	.....	+37,862	625,716	−13,908
45	+44,827	576,553	−9,590	.....	+38,683	618,690	−14,059
46	+46,128	567,971	−9,672	.....	+39,440	611,428	−14,187
47	+47,354	559,054	−9,734	.....	+40,126	603,899	−14,292
48	+48,492	549,788	−9,779	.....	+40,739	596,072	−14,370
49	+49,532	540,157	−9,804	.....	+41,271	587,922	−14,425
50	+50,462	530,151	−9,811	.....	+41,719	579,422	−14,453
51	+51,273	519,760	−9,799	.....	+42,081	570,551	−14,455
52	+51,956	508,978	−9,768	.....	+42,353	561,289	−14,430
53	+52,504	497,799	−9,718	.....	+42,535	551,618	−14,381
54	+52,909	486,221	−9,651	.....	+42,625	541,523	−14,304
55	+53,179	474,241	−9,564	.....	+42,623	530,990	−14,201
56	+53,276	461,859	−9,459	.....	+42,526	520,007	−14,073
57	+53,227	449,070	−9,335	.....	+42,335	508,554	−13,919
58	+53,014	435,870	−9,193	.....	+42,045	496,611	−13,739
59	+52,635	422,252	−9,031	.....	+41,659	484,152	−13,532

l_x.

Age x.	Males (b)	Males (a)	Males (c)		Females (b)	Females (a)	Females (c)
60	+52,083	408,212	−8,849	.....	+41,171	471,155	−13,297
61	+51,357	393,745	−8,647	.....	+40,578	457,596	−13,034
62	+50,452	378,850	−8,426	.....	+39,880	443,451	−12,742
63	+49,370	363,528	−8,185	.....	+39,072	428,700	−12,421
64	+48,109	347,785	−7,923	.....	+38,153	413,327	−12,070
65	+46,672	331,633	−7,642	.....	+37,120	397,323	−11,689
66	+45,061	315,095	−7,342	.....	+35,973	380,688	−11,278
67	+43,287	298,201	−7,023	.....	+34,714	363,436	−10,839
68	+41,357	280,996	−6,686	.....	+33,345	345,594	−10,370
69	+39,283	263,537	−6,334	.....	+31,870	327,207	− 9,874
70	+37,082	245,890	−5,966	.....	+30,297	308,333	− 9,355
71	+34,773	228,137	−5,586	.....	+28,635	289,050	− 8,813
72	+32,374	210,374	−5,197	.....	+26,902	269,451	− 8,252
73	+29,912	192,705	−4,800	.....	+25,092	249,651	− 7,677
74	+27,413	175,247	−4,399	.....	+23,242	229,776	− 7,091
75	+24,904	158,126	−3,999	.....	+21,362	209,972	− 6,501
76	+22,414	141,471	−3,603	.....	+19,471	190,394	− 5,912
77	+19,973	125,414	−3,216	.....	+17,590	171,204	− 5,329
78	+17,609	110,083	−2,840	.....	+15,738	152,568	− 4,759
79	+15,350	95,600	−2,480	.....	+13,945	134,651	− 4,207
80	+13,220	82,073	−2,140	.....	+12,219	117,611	− 3,680
81	+11,239	69,595	−1,823	.....	+10,585	101,589	− 3,182
82	+ 9,425	58,237	−1,532	.....	+ 9,058	86,710	− 2,718
83	+ 7,790	48,045	−1,269	.....	+ 7,650	73,074	− 2,291
84	+ 6,339	39,038	− 994	.....	+ 6,372	60,752	− 1,906
85	+ 5,076	31,207	− 808	.....	+ 5,231	49,781	− 1,561
90	+ 1,260	7,667	− 203	.....	+ 1,517	14,303	− 448
95	+ 176	1,039	− 27	.....	+ 264	2,444	− 77
100	+ 12	64	− 2	.....	+ 24	211	− 6

TABLE SHOWING AT CERTAIN AGES THE PROPORTIONAL INCREASE AND DECREASE OF THE l_x VALUES OF LIFE-TABLES II. AND III. RESPECTIVELY AS COMPARED WITH LIFE-TABLE I., EXPRESSED AS CHANGES PER CENT.

	Males		Females	
Ages	(*b*)	(*c*)	(*b*)	(*c*)
5	+ 0·21	−0·06	+ 0·19	−0·07
10	+ 0·30	−0·10	+ 0·30	−0·12
15	+ 0·42	−0·16	+ 0·55	−0·22
20	+ 0·90	−0·28	+ 1·13	−0·46
25	+ 1·86	−0·53	+ 2·02	−0·83
30	+ 3·14	−0·87	+ 3·03	−1·28
35	+ 4·57	−1·19	+ 4·10	−1·68
40	+ 6·10	−1·45	+ 5·20	−2·01
45	+ 7·77	−1·67	+ 6·25	−2·28
50	+ 9·51	−1·86	+ 7·20	−2·50
55	+11·21	−2·02	+ 8·02	−2·68
60	+12·75	−2·17	+ 8·74	−2·83
65	+14·07	−2·31	+ 9·34	−2·95
70	+15·08	−2·43	+ 9·82	−3·03
75	+15·74	−2·53	+10·17	−3·09
80	+16·10	−2·61	+10·39	−3·12
85	+16·26	−2·65	+10·50	−3·13

Mean Expectation of Life or Mean After-Lifetime at Age x

E_x.

Ages x.	Males (b)	Males (a)	Males (c)		Females (b)	Females (a)	Females (c)
0	+2·22	44·11	−0·45	.....	+1·93	47·73	−0·68
1	+2·63	52·15	−0·52	.....	+2·22	54·45	−0·77
2	+2·75	54·04	−0·54	.....	+2·30	56·26	−0·80
3	+2·79	54·18	−0·55	.....	+2·33	56·40	−0·81
4	+2·82	53·89	−0·55	.....	+2·34	56·16	−0·83
5	+2·83	53·41	−0·55	.....	+2·36	55·69	−0·83
6	+2·83	52·79	−0·56	.....	+2·36	55·08	−0·83
7	+2·84	52·06	−0·56	.....	+2·36	54·36	−0·83
8	+2·84	51·26	−0·56	.....	+2·35	53·58	−0·83
9	+2·84	50·42	−0·56	.....	+2·35	52·74	−0·82
10	+2·85	49·54	−0·55	.....	+2·35	51·88	−0·82
11	+2·85	48·65	−0·54	.....	+2·33	51·01	−0·82
12	+2·84	47·76	−0·54	.....	+2·32	50·13	−0·81
13	+2·84	46·87	−0·54	.....	+2·30	49·25	−0·80
14	+2·83	45·99	−0·54	.....	+2·27	48·38	−0·79
15	+2·82	45·12	−0·54	.....	+2·24	47·52	−0·78
16	+2·80	44·26	−0·53	.....	+2·22	46·66	−0·76
17	+2·77	43·41	−0·52	.....	+2·17	45·82	−0·75
18	+2·74	42·57	−0·51	.....	+2·12	44·98	−0·73
19	+2·69	41·75	−0·50	.....	+2·07	44·15	−0·71
20	+2·65	40·93	−0·49	.....	+2·01	43·33	−0·69
21	+2·60	40·11	−0·47	.....	+1·95	42·51	−0·66
22	+2·53	39·31	−0·46	.....	+1·88	41·70	−0·64
23	+2·47	38·51	−0·44	.....	+1·81	40·89	−0·61
24	+2·40	37·71	−0·42	.....	+1·75	40·08	−0·58
25	+2·33	36·91	−0·40	.....	+1·68	39·27	−0.54
26	+2·25	36·12	−0·38	.....	+1·61	38·47	−0·52
27	+2·17	35·33	−0·37	.....	+1·54	37·67	−0·48
28	+2·09	34·55	−0·34	.....	+1·47	35·88	−0·45
29	+2·01	33·77	−0·32	.....	+1·41	36·09	−0·42
30	+1·93	33·00	−0·30	.....	+1·34	35·31	−0·39
31	+1·85	32·23	−0·28	.....	+1·27	34·54	−0·37
32	+1·77	31·47	−0·26	.....	+1·21	33·76	−0·34
33	+1·70	30·71	−0·24	.....	+1·14	33·00	−0·31
34	+1·62	29·96	−0·22	.....	+1·08	32·24	−0·29

E_x.

Age x	Males (b)	Males (a)	Males (c)		Females (b)	Females (a)	Females (c)
35	+1·55	29·22	−0·20	.....	+1·02	31·49	−0·26
36	+1·47	28·49	−0·19	.....	+0·96	30·74	−0·25
37	+1·40	27·76	−0·18	.....	+0·90	30·00	−0·23
38	+1·33	27·04	−0·17	.....	+0·84	29·26	−0·21
39	+1·26	26·33	−0·16	.....	+0·79	28·52	−0·20
40	+1·21	25·62	−0·15	.....	+0·73	27·79	−0·19
41	+1·13	24·91	−0·13	.....	+0·69	27·05	−0·16
42	+1·06	24·21	−0·12	.....	+0·64	26·32	−0·15
43	+1·00	23·52	−0·12	.....	+0·59	25·59	−0·14
44	+0·94	22·83	−0·11	.....	+0·54	24·87	−0·13
45	+0·87	22·16	−0·11	.....	+0·51	24·14	−0·12
46	+0·81	21·48	−0·09	.....	+0·47	23·42	−0·11
47	+0·76	20·81	−0·08	.....	+0·43	22·71	−0·10
48	+0·70	20·16	−0·08	.....	+0·39	22·00	−0·09
49	+0·64	19·51	−0·07	.....	+0·36	21·30	−0·08
50	+0·59	18·87	−0·07	.....	+0·33	20·60	−0·07
51	+0·54	18·24	−0·07	.....	+0·30	19·92	−0·07
52	+0·50	17·61	−0·06	.....	+0·27	19·24	−0·06
53	+0·45	17·00	−0·06	.....	+0·25	18·57	−0·06
54	+0·41	16·39	−0·05	.....	+0·23	17·90	−0·05
55	+0·37	15·79	−0·04	.....	+0·20	17·25	−0·05
56	+0·34	15·20	−0·04	.....	+0·19	16·60	−0·04
57	+0·30	14·62	−0·04	.....	+0·16	15·97	−0·04
58	+0·27	14·05	−0·04	.....	+0·15	15·34	−0·03
59	+0·24	13·48	−0·03	.....	+0·13	14·72	−0·03
60	+0·21	12·93	−0·03	.....	+0·12	14·11	−0·03
61	+0·18	12·39	−0·03	.....	+0·10	13·52	−0·03
62	+0·16	11·86	−0·03	.....	+0·09	12·93	−0·02
63	+0·14	11·33	−0·02	.....	+0·08	12·36	−0·02
64	+0·13	10·82	−0·02	.....	+0·07	11·80	−0·02
65	+0·10	10·33	−0·02	.....	+0·06	11·26	−0·02
66	+0·09	9·84	−0·01	.....	+0·05	10·73	−0·02
67	+0·08	9·37	−0·01	.....	+0·05	10·21	−0·01
68	+0·06	8·92	−0·01	.....	+0·04	9·71	−0·01
69	+0·06	8·47	−0·01	.....	+0·04	9·23	−0·01
70	+0·04	8·05	−0·01	.....	+0·02	8·77	−0·01
71	+0·03	7·64	−0·01	.....	+0·02	8·32	−0·01
72	+0·03	7·24	−0·01	.....	+0·02	7·89	−0·01
73	+0·02	6·86	−0·01	.....	+0·02	7·47	−0·01
74	+0·02	6·49	±0·00	.....	+0·01	7·08	−0·01

E_x.

Age x	Males (b)	Males (a)	Males (c)		Females (b)	Females (a)	Females (c)
75	+0·02	6·14	—	.....	+0·01	6·70	−0·01
76	+0·02	5·80	—	.....	+0·01	6·33	±0·00
77	+0·01	5·48	—	.....	+0·01	5·99	—
78	+0·01	5·18	—	.....	+0·01	5·66	—
79	+0·01	4·89	—	.....	+0·01	5·35	—
80	+0·01	4·61	—	.....	±0·00	5·05	—
81	+0·01	4·35	—	.....	—	4·77	—
82	+0·01	4·10	—	.....	—	4·50	—
83	±0·00	3·87	—	.....	—	4·25	—
84	—	3·65	—	.....	—	4·01	—
85	—	3·44	—	.....	—	3·79	—
90	—	2·56	—	.....	—	2·84	—
95	—	1·92	—	.....	—	2·15	—
100	—	1·43	—	.....	—	1·63	—

TABLE SHOWING AT CERTAIN AGES THE PROPORTIONAL INCREASE AND DECREASE OF THE E_x VALUES OF LIFE-TABLES II. AND III. RESPECTIVELY, AS COMPARED WITH LIFE-TABLE I., EXPRESSED AS CHANGES PER CENT.

Ages	Males (b)	Males (c)		Females (b)	Females (c)
0	+5·03	−1·02	.....	+4·04	−1·42
5	+5·29	−1·02	.....	+4·23	−1·49
10	+5·75	−1·11	.....	+4·52	−1·57
15	+6·25	−1·19	.....	+4·71	−1·64
20	+6·47	−1·19	.....	+4·63	−1·59
25	+6·31	−1·08	.....	+4·27	−1·37
30	+5·84	−0·90	.....	+3·79	−1·10
35	+5·30	−0·68	.....	+3·23	−0·82
40	+4·72	−0·58	.....	+2·62	−0·68
45	+3·92	−0·49	.....	+2·11	−0·49
50	+2·96	−0·39	.....	+1·60	−0·34
55	+2·34	−0·25	.....	+1·16	−0·28
60	+1·62	−0·23	.....	+0·85	−0·20
65	+0·96	−0·19	.....	+0·53	−0·17
70	+0·49	−0·11	.....	+0·22	−0·11

According to Life-Table II. the greatest percentage increase is, for males, 6·48 at age 21, and for females, 4·75 at age 16. According to Life-Table III. the greatest percentage decrease is, for males, 1·19 at the ages from 15 to 20, and for females, 1·64 at age 15.

England and Wales, 1891—1900.

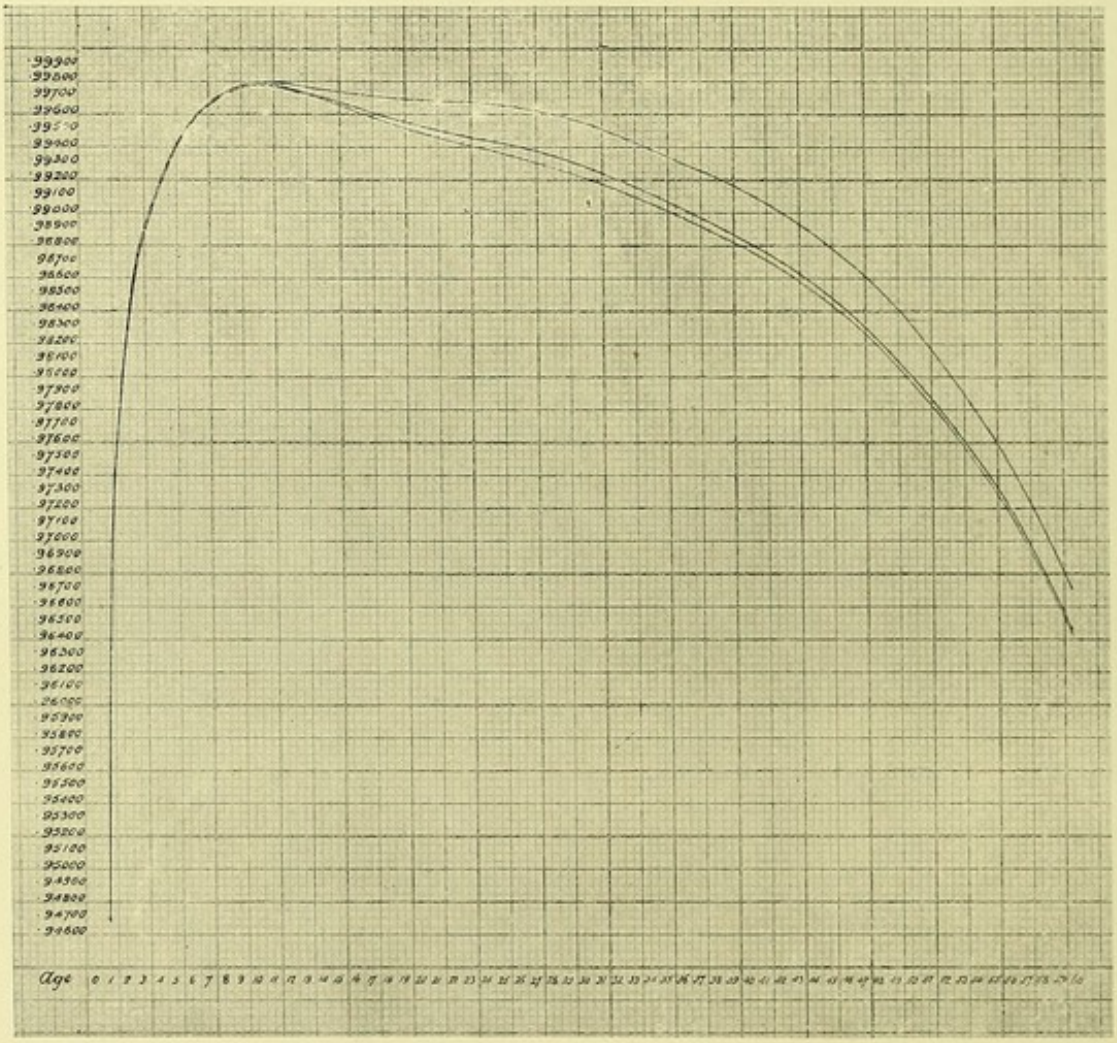
Males.

p_x Curves.

(*a*) based on the total mortality from all causes (*Middle curve*).

(*b*) based on the supposition that Phthisis had been eliminated as a cause of mortality (*Upper curve*).

(*c*) based on the supposition that the mortality from Phthisis had been the same as in 1881-90 (*Lower curve*).

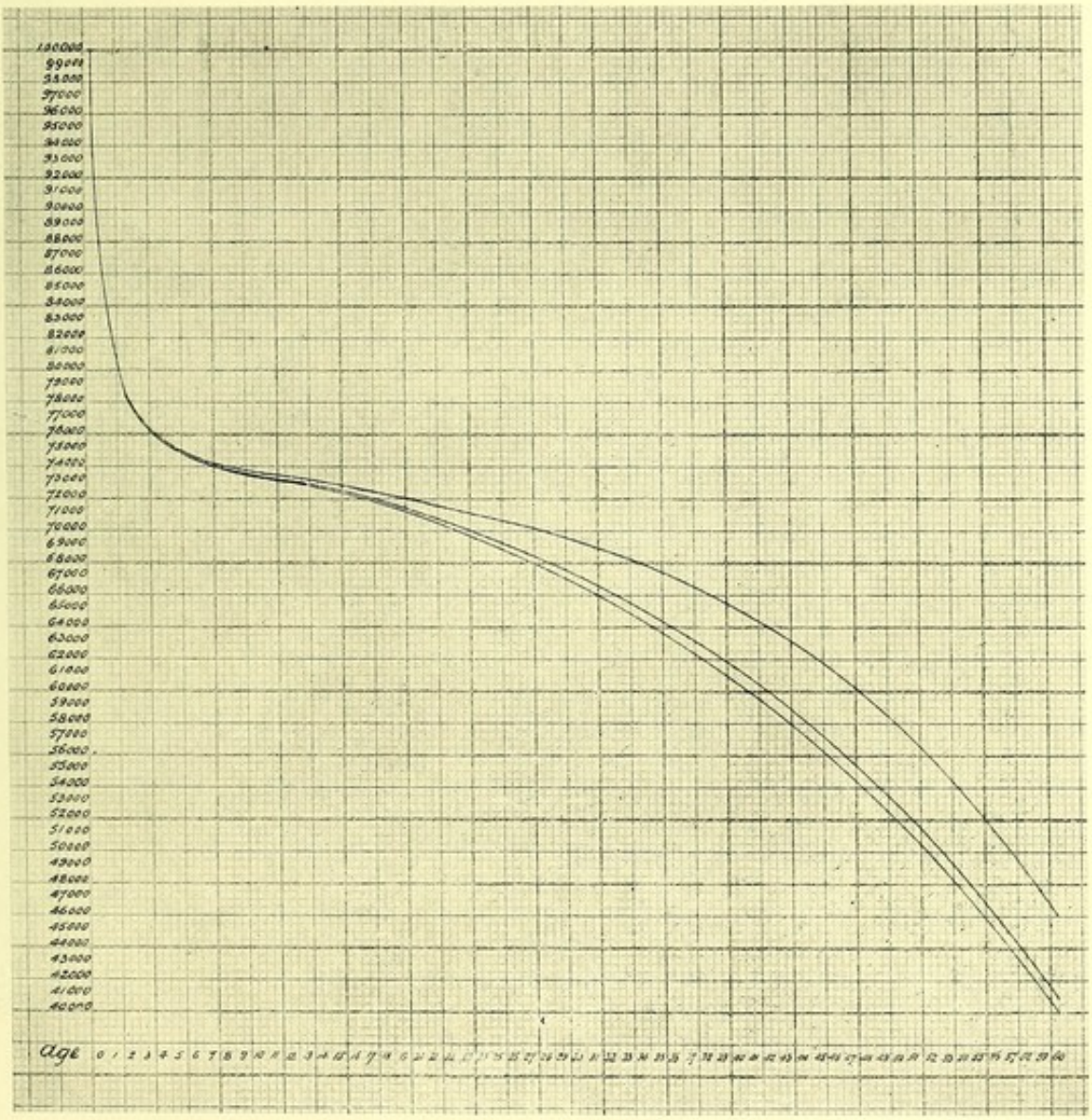


ENGLAND AND WALES, 1891—1900.

MALES.

l_x Curves.

(*a*) based on the total mortality from all causes (*Middle curve*).
(*b*) based on the supposition that Phthisis had been eliminated as a cause of mortality (*Upper curve*).
(*c*) based on the supposition that the mortality from Phthisis had been the same as in 1881-90 (*Lower curve*).

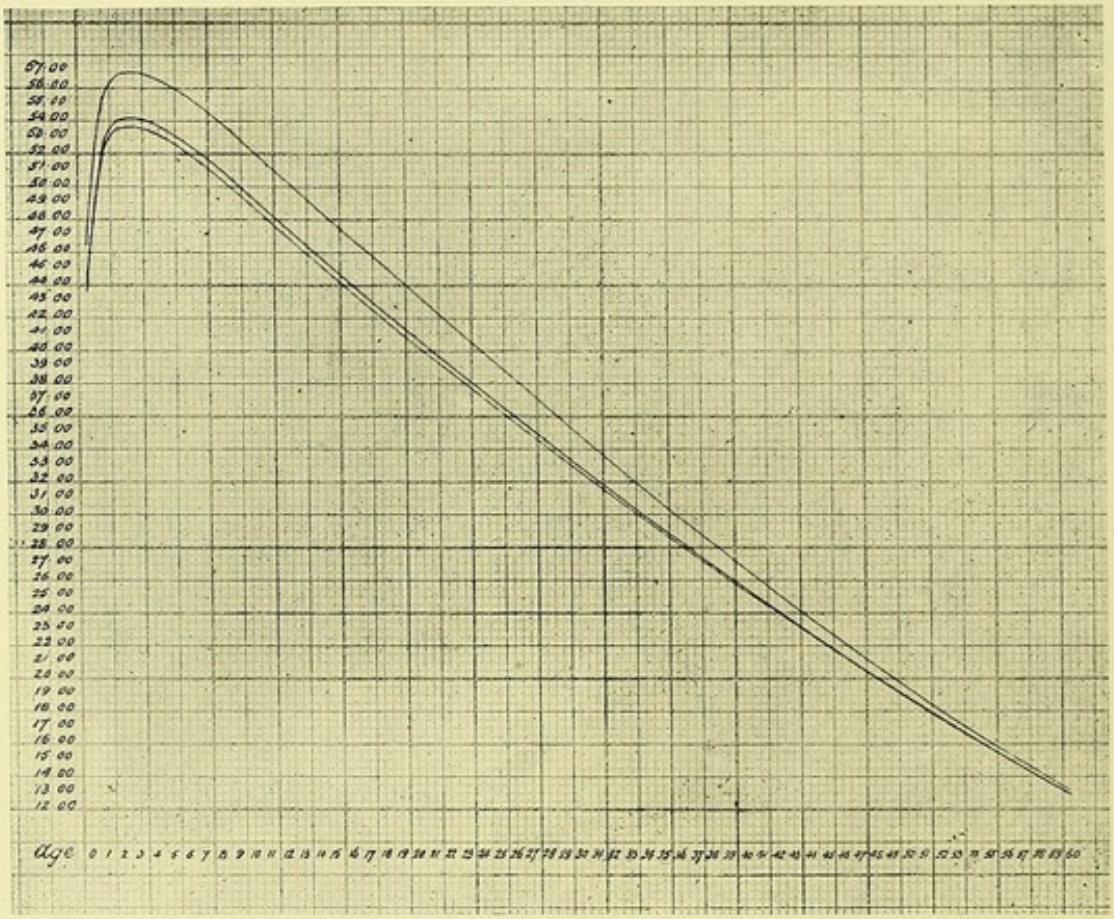


England and Wales, 1891—1900.

Males.

E_x Curves.

(*a*) based on the total mortality from all causes (*Middle curve*).
(*b*) based on the supposition that Phthisis had been eliminated as a cause of mortality (*Upper curve*).
(*c*) based on the supposition that the mortality from Phthisis had been the same as in 1881-90 (*Lower curve*).



Water Filtration in Connection with Public Supplies.

BY

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Water Filtration in Connection with Public Supplies.

Historical Remarks. The process of filtration for the purification of water appears to be of very modern origin. The extensive remains of ancient aqueducts, reservoirs, tanks, etc., do not indicate that any system of filtration such as is now practised was adopted at the time of their construction, nor do we find in the ancient description of these waterworks any reference to filtration. Forbes has suggested that the " castella " found along the course of the Roman aqueducts were places in which the water was filtered, but they are far too small to have served for any such purpose. They may, however, have been used as straining chambers to remove visible particulate matter suspended in the water. Probably at this early period this was all that was considered necessary or possible. The problem of converting the sewage polluted water of a river into a good and safe drinking water may not have presented itself to the engineers of Babylon, Nineveh, Rome, or other cities. In any case the first systematic attempt to remove something more than the visible suspended matter appears to have been made by one of the London Water Companies in 1829.

English System. The engineer of the Chelsea Company having found that the Thames water did not become clear by mere subsidence, experimented with sand filters and found that satisfactory clarification could be obtained by passing the Thames water through layers of sand at not too great a rate. It is probable, however, that sand filtration had been tried, and was actually in use, in a few places in Lancashire, as Simpson, the engineer above referred to, says that he was led to make his experiments after examining filters used by manufactories and some waterworks in the north of England. The Chelsea Company adopted the system and other Companies speedily followed their example.

At this time there was no idea that the process was other than a mere fine straining, the sand particles keeping back the suspended matter. Methods of determining the organic matter in water were not known, and bacteria had not then been heard of. With the advance in knowledge of the chemistry of water and the elaboration of methods intended to estimate the organic matter in water, it was found that by filtration something more was accomplished than the mere removal of visible particles. The free ammonia decreased or entirely disappeared, and the organic matter dissolved in the water suffered marked diminution. The efficiency of the filtration was no longer measured merely by the physical effects produced, but by the chemical, and chemical standards were set up, which at the time served a useful purpose, but which now ought to be obsolete. How this improvement in the quality of the water was brought about was difficult to explain, and whilst engineers and chemists were discussing the possible explanations the bacteriologist appeared upon the scene and speedily found the true solution of the problem. The chemical

action was shown to be quite insignificant and almost entirely secondary to the action of the lowest forms of life. The problem proved to be primarily biological and not chemical, and the standard of purification now recognised is bacteriological, though stress is still laid upon the results of chemical examinations in so far as they indicate the extent to which the water has been purified by the biological changes which take place in it as it traverses the filters. When it is recognised that the chief water-borne diseases, Typhoid fever, Cholera, and Diarrhœa, are due to the action of bacteria, and that the small amount of organic matter found in solution in potable waters is quite harmless, it is obvious that bacterial purification is far more important than physical or chemical purification.

In a large number of cases, however, water not seriously liable to pollution by excremental matter requires filtration in order to render it fit for domestic purposes; in these the efficiency may be chiefly judged by the result of the physical and chemical examination, the bacteriological results being of secondary importance.

The system of slow filtration through sand has proved so generally applicable throughout Great Britain, and the use of sand filters has become so common in this country that it is now usually spoken of as the " English " system. Over large areas on the continent and in the United States, where the river waters are almost invariably turbid from the presence of clay and alluvial matters, it has been found that the English system entails too great labour and expense to produce satisfactory results, the sand beds speedily clogging and requiring too frequent cleaning.

American System. To obviate these difficulties the Americans have largely adopted a system entailing the use of coagulants and mechanical filters, and this is so widely used in the States that it is spoken of as the "American" system. The coagulant, sulphate of alumina generally, when added to the water, causes a precipitate of aluminic hydrate which carries down with it nearly all the suspended matter. Subsequent rapid passage through a mechanical filter then suffices to produce a clear and palatable water.

Continental System. Since the historical outbreak of Cholera at Hamburg, due to the use of polluted river water by the inhabitants, very serious attention has been directed by the French and Germans to the subject of water filtration; the English sand process has been so improved and the bacterial efficiency so increased by the introduction of scientific methods of working that the advanced system is now generally referred to as the " Continental system." No one system can be said to be applicable in all cases, though one may be more generally applicable than another. That system which, when applied to any given water, will render it hygienically satisfactory, and which will do this with the greatest certainty, and at the smallest cost, is the best system for that particular water. Much depends upon the definition of a hygienically satisfactory water. I consider that no water derived from a public supply is hygienically satisfactory unless it is at all times delivered to the consumers in a bright and palatable condition, free from any obvious colour and containing no saline or organic matter of an objectionable character.

Sources of Drinking Water. Before describing more fully the systems of filtration it will be well to consider briefly the various sources from which water is derived and the conditions which may render filtration necessary or desirable.

Deep Wells usually yield water of such a character that it can be supplied direct to the consumer. Very occasionally it may be turbid from the presence of sand, clay, or chalk, rendering filtration necessary; more frequently the water is ferruginous, containing in solution ferrous carbonate, which becomes deposited as an oxycarbonate when the water is exposed to air, rendering it turbid and unsightly. These waters are very common from certain formations, and no engineer would select such a source if any other were available. When no other water is available the iron must be removed. This can be done by adopting any of the well-known devices for aerating the water and afterwards allowing it to clear by subsidence or removing the deposited oxycarbonate by means of a filter.

Deep wells may yield waters of excessive hardness, and if a softening process is adopted subsequent filtration may be necessary. I know of no deep well water which requires filtration on account of its being polluted with sewage matter. When such a source yields a polluted water it is usually abandoned, this undoubtedly being the wisest course.

Subsoil Water and Spring Water. In the great majority of cases spring water is the natural overflow of the subsoil water, and when springs are utilized for public supplies it is not usual to attempt to derive from them more than the natural yield, in which case the water is generally of such a character that filtration is not necessary. But where an attempt is made to draw the water from the subsoil faster than the natural flow, turbidity may ensue, and filtration be required. Wells, sunk in superficial sands and gravels, especially if connected with adits, and from which water is pumped at such a rate as to greatly depress the water level during pumping, have a tendency to yield waters which either are slightly turbid or may contain the spores of low forms of vegetable life capable of developing in service and other reservoirs, and possibly in the water mains themselves. I am constantly investigating such cases. In one instance I found that sand filtration had been resorted to, the water being passed through the filter bed in the summer only. When the filter was again used in the following summer the water had a green tint, and when allowed to stand in jugs and caraffes the sides became covered with a green slime. I found that during the spring the sand of the filter bed had become permeated with a protococcus which, when the beds were again used, was washed through into the supply. Recently I saw another sand filter used for subsoil water, and the surface was covered with a dense mass of fresh water algæ, which necessitated very frequent scraping of the beds. In another case the water contained fine clay in suspension, which the small sand filters used failed to remove. Larger filters and the use of polarite got over this difficulty.

Surface Waters. Waters derived from mountain sides and from moorlands must be collected in reservoirs of considerable capacity, since sufficient must be collected during the rainy period to tide over the longest drought. If the moorland surface is uncultivated and the reservoir properly protected, such waters are of the highest degree of hygienic purity, and admirably adopted for the purpose of public supplies. But even in such cases low forms of vegetable life may appear in the reservoirs, and be discovered in the water supplied to the consumer. Occasionally these may be so numerous as to affect the colour, odour, or even the taste of the water. In summer they are always to be found if sought for. It is only necessary to tie a fine linen strainer over the mouth of the water tap and remove this in a few days to discover them. Usually the surface is covered with organisms, animal and vegetable, which, when seen by a total abstainer, are almost sufficient to make him decide to abstain from drinking water also. It is a moot point whether such waters should be filtered. It is usually with the engineer a question of expense. So far as we know these unpolluted waters containing diatoms, desmids, protococci, etc., etc., are quite without effect upon health, and the organisms so rarely become sufficiently numerous as to impair the physical properties of the water that it may be doubtful whether the expense of filtration is justifiable. As I prefer a water which is always bright and clear, and which contains none of these low forms of life, I should like to see such water filtered before being supplied to the consumers. Accidents will happen, and if the body of a man or animal happens to get into the reservoir I think the filter would at least keep back 98 per cent. of the organisms of putrefaction, and this, to me, would be a source of satisfaction.

When any portion of the collecting area is under cultivation or is largely used for grazing cattle, the necessity for filtration is increased, even if no sewage of human origin gains access to the water. The manurial matter introduces bacteria and other organisms, and also phosphates and organic matter which tend to increase the luxuriance of the growth of all low forms of life. Waters of this character are very prone to become infected with organisms to such an extent as to impair their palatibility, and I certainly think that water from such sources should be submitted to some process of filtration.

If upon the collecting area there are houses, the sewage from which, directly or indirectly, gains access to the supply, filtration is imperative, and the nature of that filtration we shall immediately proceed to consider.

River Waters. The waters found in rivers consist of surface water and subsoil water. There may possibly be a few cases in which such waters may be impounded and used without filtration, but such cases are very rare. Before any river in this country has attained such dimensions that water can be abstracted from it for the purpose of a public supply, it must have traversed inhabited regions and have been subjected to pollution. From time to time also it must be in a condition of flood and yield a turbid water, utterly unfit for domestic purposes. Large impounding reservoirs may obviate the necessity for taking water from the river whilst in flood, and may permit of all visible sedimentary matter being deposited, but this does not, in my opinion, obviate the necessity for filtration.

Objects of Filtration. This brief review of the sources from which public water supplies are derived has shown us that filtration may be necessary for one or more of the following purposes:—

1. The removal of very fine suspended mineral matter.
2. The removal of low forms of animal and vegetable life other than bacteria.
3. The removal of bacteria.
4. The removal of organic matter in solution.

The latter object is generally required when water is derived from small lakes or reservoirs in which vegetable growth is abundant. The death and decay of this vegetable matter introduce an amount of organic matter into solution which may be harmless, but which is undesirable. Such cases are far more common in America, where swamp water has often to be utilised, than in England, but they are not unknown in this country, though probably they are very few in number.

It will save time, perhaps, if the various methods of filtration already referred to are described in some detail and the effects they are capable of producing discussed. It will then be a simple matter to decide in any given instance the method of filtration which should be adopted to produce the desired result, viz., a water at all times bright and palatable and free from all deleterious or possibly deleterious bacteria.

English Sand Filters. The English sand filter consists of a layer of fine sand resting upon a layer of coarser sand, and this in its turn upon shingle or gravel contained in a tank upon the bottom of which pipes are laid to carry away the water. Larger gravel is laid over these under-drains so that the water may have a uniformly free exit from the beds above. Were one portion better drained than another, there would be a tendency for that portion of the filter to be overworked.

Massachusetts Experiments. The fine sand is the most important element in the construction of the filter, as it is chiefly upon this that the efficiency depends. Mr. Hazen, of the Massachusetts State Board of Health, who has conducted long series of experiments upon sand filtration, says: "We find that both the quality of the effluent obtained by filtration and the cost of filtration depend upon the size of the sand grains. With a fine sand the sediment layer forms more quickly and the removal of bacteria is more complete, but, on the other hand, the filter clogs quicker and the dirty sand is more difficult to wash, so that the expense is increased." As we shall see later, it is the layer which Hazen calls the sediment layer and which the Germans call the "schlammdecke" which is the true bacterial filter, but there is no doubt that the sand itself, if not too coarse and not in too thin layers, has great purifying powers, and "in addition acts as a safeguard by positively preventing excessive rates of filtration on account of its frictional resistance" (Hazen). The Lawrence experiments showed that with very fine sands of an

effective size varying from ·09 to ·14mm., and 4 to 5 feet deep, it was almost impossible to drive bacteria through, the water passing being practically sterile, but of course the rate of filtration was too slow for practical work. With fine sands, although the upper layer more quickly becomes clogged, the matters removed from the water including the bacteria remain very near the surface and are easily removed by scraping, whereas with a coarse sand the suspended matter penetrates further and thicker layers have to be removed for cleansing purposes. Hazen says, " It is obvious that the minimum expense for cleansing will be secured with a sand which just does not allow this deep penetration, and the coarsest samples in actual use, having effective sizes of about ·40mm., represent the practical limit to the coarseness of the sand, and that any increase above this size would be followed by increased expense for cleansing as well as by decreased efficiency."

Effective Size of Sand. By the effective size of a sand is meant " The size of a grain such that 10 per cent. by weight of the particles are smaller and 90 per cent. by weight larger than itself," whilst the "uniformity co-efficient " signifies the ratio of the size of the grain which has 60 per cent. finer than itself to the size which has 10 per cent. larger than itself." The sands used by the London companies have an average effective size of ·37mm. and a uniformity co-efficient of 2·6. Sand of this character is apparently obtained to the following specification :—" The whole of the sand to be of such fineness that it will pass through a wire sieve having 400 meshes to the square inch, and 80 per cent. of it through a sieve having 900 meshes to the square inch, and none of it through a sieve having 3,600 meshes to the square inch." The best sand in this country apparently comes from Leighton Buzzard, but sea sand from certain sources has a high reputation.

Thickness of Sand Layer. The sand layer may be of any thickness from 12 to 48 inches. Usually when the bed is first made it has a thickness of 30 to 40 inches, and this is gradually reduced by scraping, about three-quarters of an inch being removed each time, until it is reduced to 12 inches, when the bed is again made up. If after scraping the sand is immediately washed and returned to the bed the thickness is usually kept at about 24 inches.

Head of Water. With a freshly constructed bed the rate of filtration increases with the head of water, or the difference in level between the water on the sand and in the outlet chamber, with the temperature of the water, the effective size of the sand, and decreases with the thickness of the layer of sand.

Temperature. The warmer the water the more rapid the rate of filtration; other things being equal, where 100 gallons would pass at 50°F. only 70 would pass at 32°F., whilst 145 would pass at 77°F.

Filtering Film. After the filter has been in use a few days the rate of filtration is affected by another and much more important

factor, the *schlammdecke*, of German writers, which forms over the surface of the sand. As this increases in thickness the rate of filtration decreases until a point is reached when the filter must be scraped. A filter may have to be cleaned every few weeks or it may remain effective for months, the difference depending upon the character of the water and the amount of sedimentary matter which it contains.

Packing of Sand and Filling of Filters. The packing of sand upon the filter must be carefully done or lines of lesser resistance may be left leading to defective filtration. In most beds air tubes are inserted to allow of the air being driven through them as the water descends when the bed is being filled from the top, but these tubes allow dust, small animals, etc., to gain access to the unfiltered water, and if not carried up sufficiently high may permit unfiltered water to pass through. As it is found that it is much better to fill the beds in the first instance, and after scraping, from below upwards, there is no necessity for such tubes, and the beds are better without them.

Working of the Filter. After filling, the water must be applied in such a way that the sand surface suffers no disturbance. The water which passes through after filling or cleansing will still contain a large proportion of the bacteria, but after two or three days' filtration at a slow rate a deposit forms upon the surface of the sand which retains the bacteria. This *schlammdecke* has such power of retaining these minute organisms that it has frequently been asserted that the bacteria found in the filtrate from a "ripe" filter are simply those which have grown in the gravel layer and under-drains. That the filtering film does not stop all bacteria has been many times demonstrated by adding to the unfiltered water organisms not found in the filtrate, such as the bacillus coli communis and the bacillus prodigiosus. These, of course in much diminished numbers, are soon found in the filtered water and must have passed through the filtering film.

Rate of Filtration. In a "ripe" filter the number of bacteria which pass through depends chiefly upon the rate of filtration, hence to maintain a uniform standard of efficiency the rate of filtration must admit of being fairly constant. This can only be done by regulating the head of water in the filter. In most works this is entrusted to the man in charge, but it is far better to affix some automatic regulating apparatus.

Automatic Regulation of Rate of Filtration. Each filter bed must have its own regulating apparatus. Without this apparatus a considerable depth of water has to be kept upon the filter, but with a regulating appliance the depth need not exceed 3 feet. The Massachusetts experiments showed that there was a "marked decrease in efficiency with increasing rates, the number of bacteria passing increasing in general as the square of the rate" (Hazen). This does not appear to be always the case and Kirkwood asserts that every water has its own special rate of filtration which must be determined by experiment. With clear waters containing comparatively few bacteria a high

rate of filtration may obtain, whereas with water containing larger numbers of micro-organisms a much slower rate must be adopted to secure efficient filtration. At Zürich, where the lake water contains only from 150 to 250 bacteria per c.c. the number in the effluent was found to be practically constant whatever the rate of filtration, consequently when the lake water is filtered at the rate of 7·5 million gallons per acre daily the bacterial contents only average 25 per c.c. This is an exceptionally high rate, 2·5 millions is about the average at the Berlin works and 1·6 millions at Altona. The average rate for the London Water Companies is about the same as that at Altona, and it is found that at this rate a water bacterially very impure may be filtered and delivered into the reservoir with an average of less than 100 bacteria per c.c.

Bacteriological Standard of Purity. This brings me to the question of the Bacteriological standard of efficiency. By a general consensus of opinion a properly filtered water should contain less than 100 organisms per c.c. capable of growing on nutrient jelly at 20°C. We may accept this standard provisionally, but more important than the fixing of this definite number is the decision as to the method of ascertaining the number. In the German regulations, Article 4 says: "To insure uniformity in the method of bacteriological analysis, the following process is recommended. The culture medium should be peptone jelly with extract of meat. The plates to be kept at about 20°C. and the colonies counted with a magnifying glass at the end of 48 hours." In England the colonies are usually counted at the end of the fourth day and the difference between the readings at the end of the second and fourth days is often considerable. There are advantages in the German method since it is often a serious inconvenience to wait until the end of the fourth day before deciding whether the results are satisfactory or not.

Control of Filtration. To obtain a water of this bacterial purity at all times many points require attention. At most of the important water-works on the Continent biologists are employed constantly in making the necessary observations. In this country we are satisfied with the examination of occasional samples, and I doubt whether there are any works in which the character of the water from each filter bed is daily determined.

Such examinations should be made so that any bed not working properly can be cut out until the cause is ascertained and remedied. Unfortunately two days must at least expire before a defect is discovered, and meanwhile imperfectly filtered water is being distributed, but this at present appears to be unavoidable. To enable such examinations to be made each filter must be so constructed that a sample of the filtrate can be taken at any moment. This is done by providing each with a special well through which the filtered water must pass on its way to the filtered water reservoir.

The outlet from this well should also be constructed so that the filtered water may be discharged either into the filtered water reservoir or over the surface of a second filter. In such case when a filter is found to be ineffective or is known not to be passing a water of a sufficiently high standard of purity (as after scraping, or after renewal of the sand) the water need not be wasted but can be passed through a second and "ripe" filter and thus utilized.

Formation and Composition of the Filtering Film. The filter will not be efficient or "ripe," as it is termed, until the filtering film ("schlammdecke") has formed. This is produced by the accumulation upon the surface and in the interstices of the upper layer of sand of bacteria and algæ derived from the water. Each particle of sand acquires a glutinous covering to which apparently bacteria, etc., adhere, and, over the whole surface a fine net-work of algæ and other filaments is formed, which forms the effective filtering layer. The entangled bacteria are not necessarily destroyed, on the contrary there is reason to belive that many forms grow luxuriantly, but the green algæ undoubtedly destroy microbes continuously, and if the filters are open, as is the case in this country, light also will exercise its germicidal powers.

"At Hamburg it has been shown that there is a certain regularity in the nature of the plants constituting the filtering film" (Hazen). In winter the film consists chiefly of a few varieties of diatoms. In the early spring green algæ appear and continue through the summer. Blue algæ are numerous in the hottest months, continue through the autumn, but disappear in winter. The diatoms alone are constantly present in large numbers, and possibly they are the most important as filtering agents. Dr. Kemna, who is the Biologist in charge of the Antwerp waterworks, confirms these results. He found upon examining the Antwerp film quantitatively that it contained:—

Melosira varians	50 per cent.
Fragilaria capucina	40 „ „
Spirogyra	10 „ „

that is 90 per cent. of the organisms were diatoms.

I have not had occasion to make any such observations. My examinations of the "schlammdecke" have been conducted for the purpose of ascertaining the nature of the organisms removed from waters from different sources.

* Fig. 1 shows the nature of the organisms found in the surface waters from Dartmoor.

Fig. 2 shows the organisms found in the surface water from a Welsh moorland. The pleurococcus, here so abundant, was shortly afterwards found by me to be penetrating deeply into a sand filter used for purifying water derived from a gravel subsoil.

Fig. 3 shows the organisms contained in the film covering the surface of large sand filters used for the purification of the water supply to an important town. Some of this water was derived from the surface of cultivated ground. The felted mass consisted chiefly of an alga (Rivularia) and a fungus (Beggiatoa).

Fig. 4. Altogether different is the nature of the film derived from a filter bed treating water from an upland surface free from contaminating matter. Here the diatoms and desmids are the dominant forms with an abundance of Spirogyra.

* These plates are reproduced by kind permission of the publishers, Messrs. J. & A. Churchill, from the Author's work on "The Examination of Water and Water Supplies."

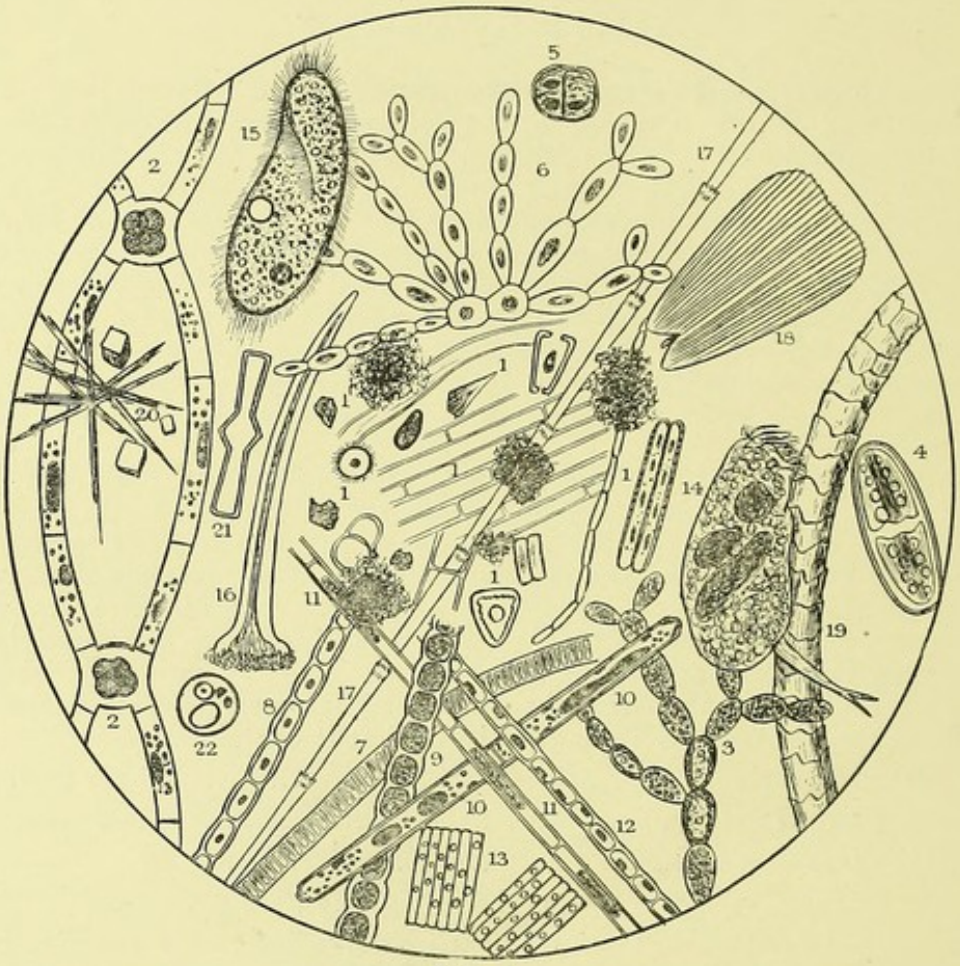

FIG. I.

1. Vegetable débris—remains of cells, fragments of diatoms.
2. Staurospermum viride. F.W.A.[1] N.O. Zygophyceæ.
3. Lemanea torulosa. F.W.A. N.O. Lemaneaceæ.
4. Desmid. F.W.A. Species of Penium.
5. Pleurococcus. F.W.A. N.O. Palmellaceæ.
6. Dead form of 3. Cells plasmolysed.
7. Ulothrix tenuis. F.W.A. N.O. Ulotricheæ.
8–12. Various forms of algal filaments. 9 is evidently a species of Ulothrix. 10 was motile.
13. Diatom. F.W.A. Sub-family Fragilarieæ.
14. A rotifer or wheel animalcule. Animal of sub-kingdom Annuloida.
15. Paramœcium (Nassula?). S.K. Protozoa. Class Infusoria.
16. An animal spine.
17. Hair of insect.
18. Wing scale of insect.
19. Fibre of wool.
20. Crystal: probably calcium sulphate.
21. Skeleton of a diatom.
22. Not identified. Free swimming, but no visible cilia.

All magnified 500 diameters.

[1] F.W.A. = Fresh Water Algæ.

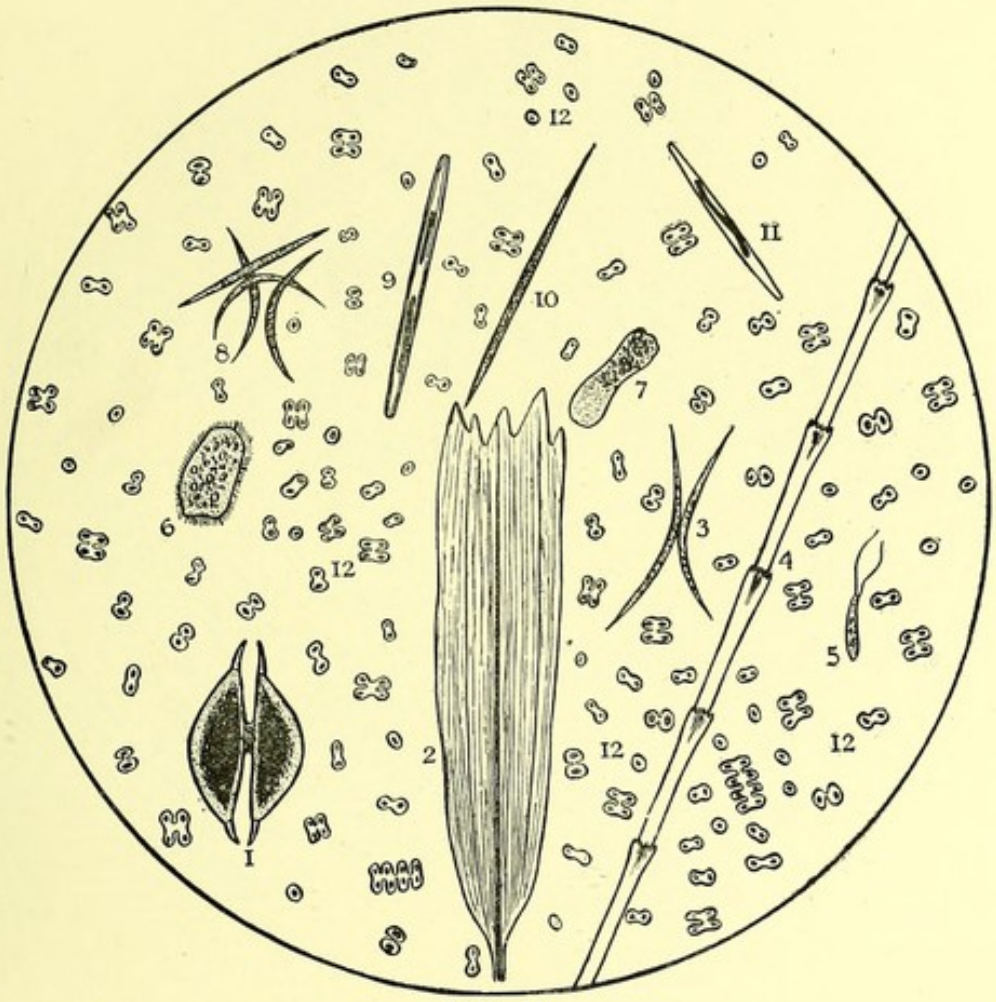

FIG. II.

1. A desmid (Arthrodesmus).
2. Scale from wing of moth or butterfly.
3. A desmid (Ankistrodesmus).
4. Hair of insect.
5. Minute protozoon.
6. A ciliate infusorian.
7. An amœba.
8. As fig. 3.
9, 11. Diatoms.
10. A small desmid.
12. Pleurococcus. The members of this genus of the order Palmellaceæ are very frequently met with in waters.

The description of the genus given by Cooke is :—"Cells gregarious, globose, or angular; single or associated in small families. Cell-contents green, or oily red. Multiplication by division in alternate directions. Propagation by gonidia."

This water formed a green deposit in any jug or vessel in which it was left, and in glass vessels exposed to light the pleurococcus grew luxuriantly.

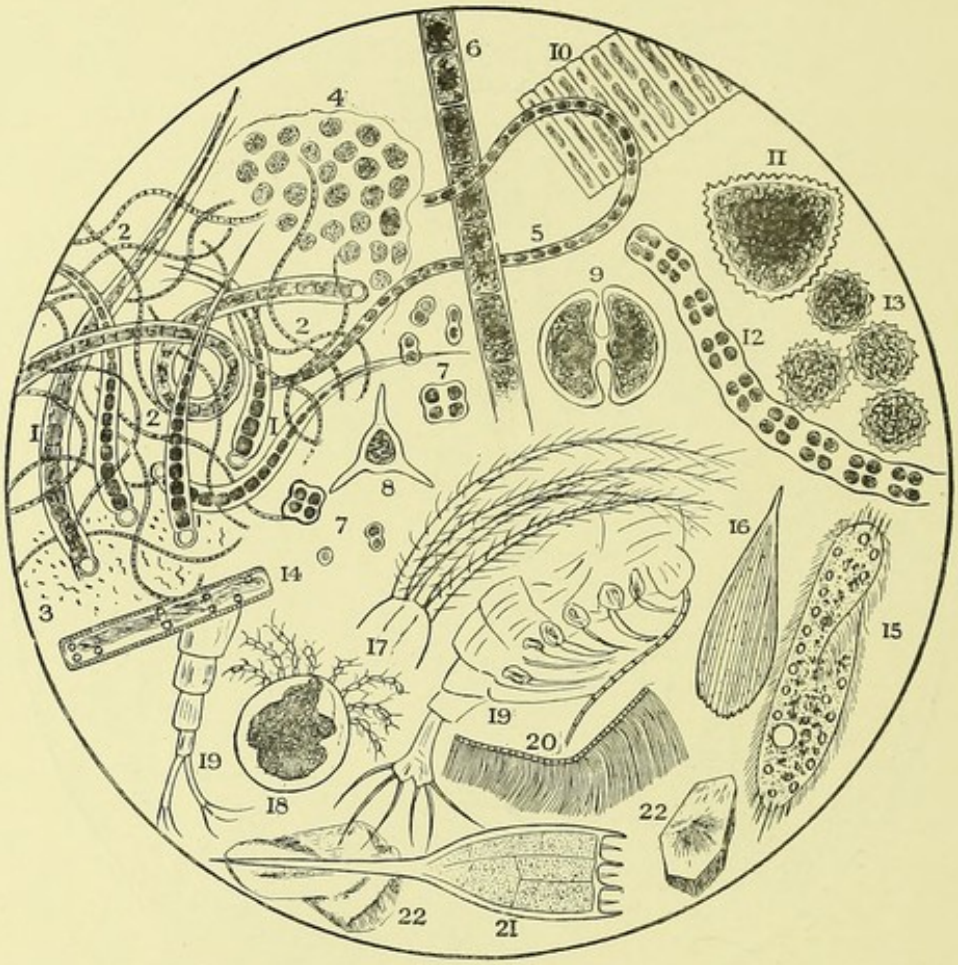

FIG. III.

SCRAPING FROM THE SURFACE OF A LARGE SAND FILTER USED FOR THE PURIFICATION OF THE SUPPLY TO A LARGE TOWN.

The water was in part derived from springs and in part from surface of more or less cultivated land. The dried surface of the sand was covered with a thin semi-transparent film of a brownish green colour.

1. Rivularia and allied forms, which with
2. Beggiatoa formed the larger portion of the felted mass covering the sands.
3. Bacilli and spirilla.
4. Palmella embedded in jelly.
5. A conferva.
6. Probably a species of Zygnema.
7. Pleurococcus.
8. A desmid (Staurastrum).
9. A desmid (Cosmarium).
10. A diatom (Fragilarieæ).
11. A desmid.
12. An algal filament.
13. Probably pollen.
14. Diatom (Pinnularia).
15. Stylonychia.

16–21. Dead and disorganised animal remains. (16 Scale from wing of moth. 18. A mite 19. Cyclops).

21. Sheath of rotifer.
22. Sand particles.

1–15 magnified about 50 diameters.
16–21 magnified about 500 diameters.

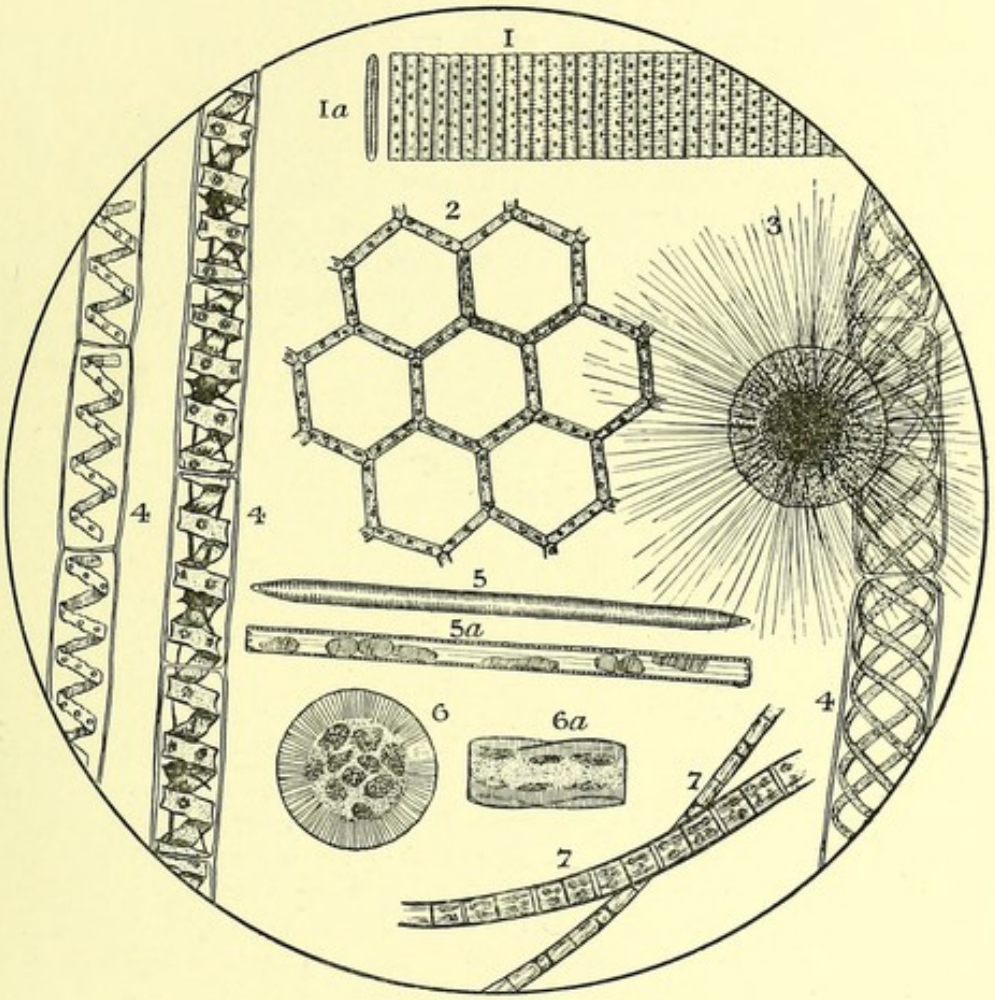

FIG. IV.

SCRAPINGS FROM THE SURFACE OF A FILTER BED USED IN CONNECTION WITH A LARGE STORAGE RESERVOIR COLLECTING UPLAND SURFACE WATER.

This plate contains only a few of the organisms identified, but they were the most abundant Associated with these was much vegetable débris, not shown in the drawing.

1. Bands of diatoms (Fragilarieæ). Girdle view.

1*a*. Separate diatom. Valve view.

2. Hydrodictyon. This organism is, by many, considered to be a desmid, in which the cells after division remain connected and form a network. It is a freshwater alga. Cooke regards it as a sub-family of the Palmellaceæ.

3. Actinosphærium. A rhizopod.

4. Filaments of a Spirogyra.

5. Diatom (Synedra). Valve view.

5*a*. Diatom (Synedra). Girdle view.

6. Cyclotella, a discoid diatom. Valve view.

6*a*. Cyclotella, a discoid diatom. Girdle view.

7. Melosira. Discoidal diatoms forming filaments. Closely allied to No. 6.

Nos. 2 and 3 magnified 100 diameters; No. 4, 200 diameters; Nos. 1, 5, & 7, 250 diameters No. 6, 500 diameters.

Conditions Disturbing the Filtering Film. The filtering film may become affected and its continuity broken in various ways, hence the necessity for constant watchfulness. In summer the felted mass of algoid filaments may entangle such a quantity of *liberated oxygen* that ultimately the mass becomes detached and rises to the surface. The exposed area then allows imperfectly purified water to pass.

Kemna mentions two *insects* as sometimes affecting sand filters. "One is a kind of gnat, *chironomus*, having an aquatic larva of red colour (commonly called blood worms). These worms burrow in the sand, cementing the grains into the form of dwelling tubes. . . . When the larvæ are transformed into winged insects they come to the surface, and the tubes remaining empty and open, the filtering surface is riddled with innumerable holes. The second is the hemipterous *Corixa*. This is an exceedingly active insect which rapidly breaks up the slime film, bringing it up in small particles to the surface." *Sticklebacks and eels* have been known to gain access to the water over filters, and to affect the filtering layer. That such should be the case indicates the desirability of the raw water passing through a fine strainer before being passed on to the beds. *Frost*, sudden changes in the rate of filtration, accidents, etc., may interfere with the efficient action of a filter.

Automatic Regulation and Recording of Rate of Filtration. Continuing our study of the condition necessary for efficient filtration, I would urge the necessity for some apparatus for regulating automatically the rate of filtration, or rather I should say for regulating the head of water so as to maintain a constant rate of filtration. Few works in this country have any arrangement of this kind, and such as I have seen do not appear to me to be efficient. There are many forms in use in America and on the Continent. **Langford's Automatic Filter Regulator** (Fig. 5) floats on the water in the well and carries a three-legged syphon (B). The middle leg acts as the discharge pipe into the stand pipe (C), the outer legs dipping into the well water. When the arms of the syphon are charged water passes up the outer limbs and down the centre limb to the filtered water reservoir. As the tank (A) rises and falls with the water in the well, the discharge is maintained constant. In the discharging limb there is a valve which can be regulated so as to permit of any required quantity being discharged per day. An index plate affixed shows the rate of discharge and therefore of filtration.

Figs. 6 and 7 show the arrangement in use in *Bremen*. The vertical arm of the outlet pipe from the filtered water well has within it a second tube with a telescopic action. This inner tube is attached to a float which keeps its mouth at a definite distance below the water surface. As the flow of water down this tube varies only with the head of water above it, if this is kept constant, the flow must be constant. When the clogging of the filter reduces the rate of filtration the level of the water in the filter well falls until a pressure head is obtained capable of passing the requisite quantity of water. In this telescopic pattern, there is a risk of the inner tube sticking and working irregularly. The figure shows how by a simple arrangement the rate of filtration and the pressure head can be recorded.

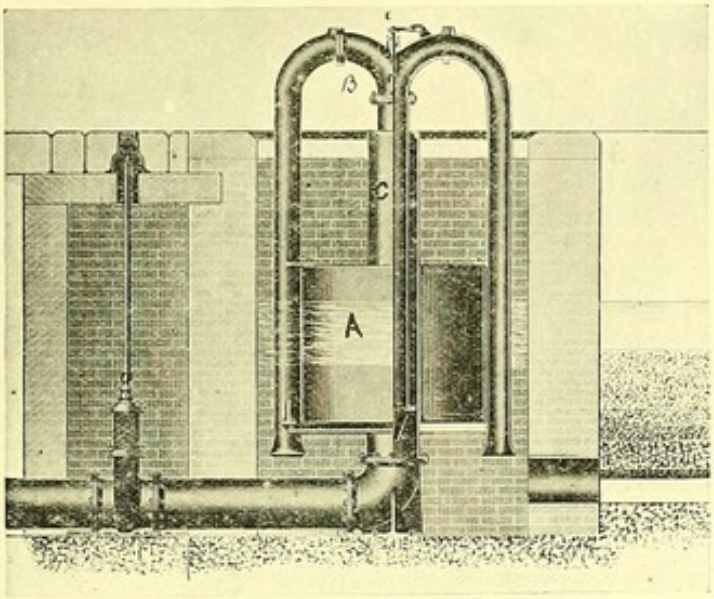

Fig. 5.

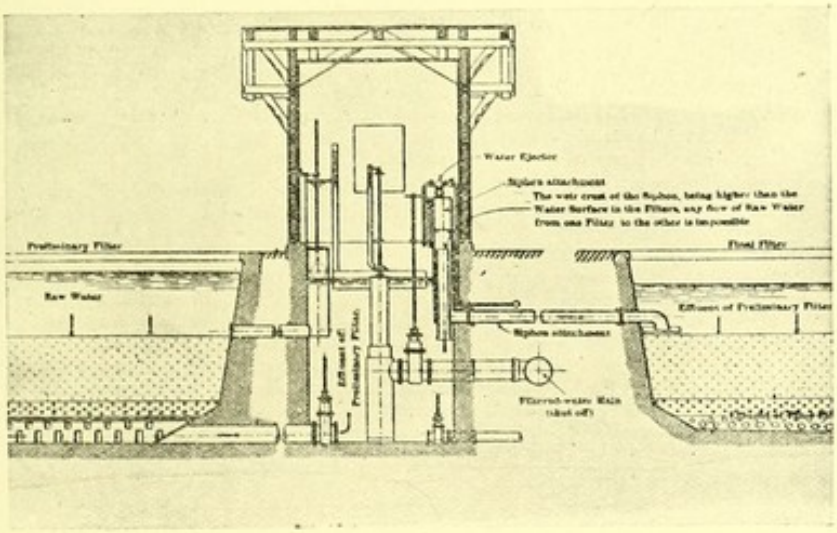

Fig. 6.

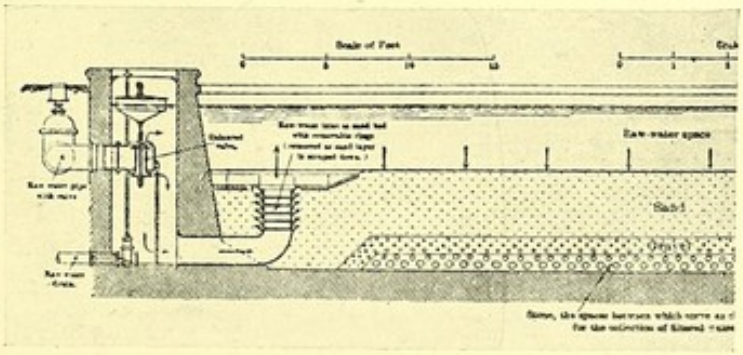

Fig. 7.

If there is a filtered water reservoir of sufficient capacity to balance the varying demand during the 24 hours, the filter can, by an appliance of the above nature, be caused to work continuously at a definite rate. When the filter can no longer pass water at this rate the supply to the bed is cut off and the water drawn away until nearly the whole of the sand is drained. The surface layer is then removed. To set the filter in action again filtered water is allowed to flow into the well, and the filter bed filled again from below upwards. When the sand is covered to a sufficient depth the unfiltered water is again run upon it. There should be a further arrangement whereby the water which now passes through the filter can be passed on to a "ripe" filter until the primary filtrate has attained the requisite bacterial standard.

The filtered water well should not be of so great a depth as to render it possible for such a head to be obtained as would rupture the filtering film.

When the sand bed is scraped and the sand not washed and returned, naturally the filter decreases in depth. When the effective sand layer is reduced to 15 inches the bed should be re-made. If, on the other hand, the removed sand is immediately washed and returned, it is advisable to remove a second layer of sand and substitute the washed sand for this layer, the unwashed sand of the second layer then being spread upon the surface. By this means the filter more rapidly "ripens" or acquires the necessary "schlammdecke."

In all cases the greatest care must be taken to maintain the upper portion of the bed in an efficient condition. If the filter becomes frozen over the surface will almost certainly be damaged, hence in countries where the winters are severe the filters are usually covered. Even in our climate there is little doubt that covered filters would be an advantage, but probably this would not be commensurate with the increased cost.

Finally, the number of filters and the total filtering area should be such as to supply the maximum demand for water of satisfactory bacterial purity, allowing for filters out of use for cleansing and for any accidental breakdown in a filter.

Data Relating to Filtering Area and Rate of Filtration in Several Waterworks. The following statistics concerning filter beds are taken from a lengthy table given in Hazen's "The Filtration of Public Water Supplies":—

City	Year	Area of filters in acres	Average daily rate of filtration in million gallons per acre	Av'ge amount of water filtered, in million galls., between scrapings
Altona	1898 ...	3·1 ...	1·72 ...	53
Berlin	1898 ...	27·0 ...	1·34 ...	—
Bremen	1898 ...	3·2 ...	1·28 ...	41
Hamburg	1898 ...	43·0 ...	·76 ...	48
Laurence (U.S.A.) ...	1897 ...	2·5 ...	1·22 ...	27
Liverpool	1896 ...	10·9 ...	2·14 ...	54
Lübeck	1898 ...	1·4 ...	3·4 ...	45
Rotterdam	1893 ...	6·3 ...	2·1 ...	—
Stockholm	1897 ...	3·6 ...	2·1 ...	36
Zürich	1898 ...	1·66 ...	4·5 ...	58

The following figures are calculated from those given in the Report on Metropolitan Water Supply contained in the L.G.B. report for 1902-3 :—

Name of Company.	Capacity of subsidence reservoirs. days supply	Area of filters in acres	Thickness of sand ft. in. to ft. in.	Rate of daily filtration per acre in million galls.
Chelsea	16	8	3 3 to 4 3	1·47
East London	30	31	1 6 to 2 6	1·3
Grand Junction	3·2	24½	2 3 to 3 0	·75
Lambeth	4·4	12⅓	2 6 to 3 0	2·33
New River	4·2	16⅝	1 7 to 2 3	2·38
Southwark & Vauxhall	13·4	37½	2 3 to 3 0	·9
West Middlesex	18·0	23⅓	2 6 to 2 9	1·0

It will be observed that the rate of filtration in the first list varies from 760,000 gallons per acre daily at Hamburg to 4,500,000 gallons at Zürich. This latter rate has, I believe, since been increased. It must not be forgotten, however, that in Hamburg a polluted river water containing thousands of bacteria per c.c. is being treated, whereas at Zürich lake water containing very few hundreds is being filtered. It is well to remember that with any given supply the most suitable rate of filtration can only be determined by experiment. In London the rate of infiltration varies from 750,000 gallons per acre by the Grand Junction Company to 2,380,000 gallons per acre by the New River Company, but here again the bulk of the New River Company's unfiltered water is much less impure bacterially than that of the Grand Junction Company.

The first Table also shows that with rapid filtration cleansing is much more frequent than when the filtration is slower. The Zürich filters pass but little more water between successive cleansing periods than the filters at Altona and Hamburg. Probably, however, the lake water, though containing fewer bacteria than the Elbe water, contains more spores of algæ, and these by their growth on the filter bed tend to impede the filtering process.

The sand filtration process as generally conducted in England is done by rule of thumb. There are few automatic regulators or rate or pressure recorders, there are few places in which there are arrangements for collecting samples of water from each filter, or for passing the water from one filter over the sand of a second. Samples are taken for bacteriological examination only at comparatively long intervals, there is no resident biologist and no attempt to work the process on strict scientific principles.

In what I have called the Continental system it is not unusual to find a skilled biologist in charge, nothing is done by rule of thumb, every filter is carefully watched and the filtrate therefrom daily examined, and the results together with the rate of filtration, filter head, etc., are daily recorded.

Control of Filtration in Bremen. At Bremen (as shown on Figs. 6 and 7), the several filters are connected by syphons, one leg of which is in the regulating well for the effluent water of one filter, while the other leg is in the raw water compartment of the adjoining filter. The syphons are inserted in such

a manner that the filtrate of one filter may be led upon one or other of the neighbouring filters, so as to have a choice in case one of them is out of service or is not yet ripe.

After a filter has been scraped and when the river water is turbid, as also during periods of frost, double filtration is resorted to. The following are a few of the results recorded in Tables given by Herr Goetze, Chief Engineer in charge of the Waterworks in Bremen, in "Water," January, 1904.

Bacteria per cc.

	Raw water.	Primary filters. No. 5.	Primary filters. No. 4.	Final filters.
During frost	650 ...	920 ...	58 ...	23
„ „	950 ...	1000 ...	144 ...	8
„ „	775 ...	280 ...	90 ...	25
Average for a month...	6150 ...	232 ...	117 ...	35

In the Table from which this is taken the bacteria in the raw water varied from 650 to 18,200 per c.c., in filtrate from No. 5 from 28 to 1440, in filtrate from No. 4 from 16 to 330, whereas in the final filtrate the variation was only from 8 to 67.

Effect of scraping filter. Scraped March 22nd.

	Raw water.	Primary filter (scraped).	Secondary filter.
March 23	18,200	1,720	50
„ 24	12,200	1,450	71
„ 25	14,600	1,120	90
„ 26	13,400	790	64
„ 27	9,800	480	63
„ 28	4,600	210	70
„ 29	5,300	130	44
„ 30	3,400	92	57
„ 31	4,500	82	51

This table shows well the effect of the scraping and the time required for the scraped filter to become effective. It also shows that the effect of the second filter is little influenced by the number of bacteria in the primary effluent.

Similar tables are given showing the effect of double filtration at a time of high and turbid water. For example, on one occasion the raw water contained nearly 40,000 bacteria per c.c., the primary effluent contained 525, and the secondary effluent only 15.

Puech's System of Filtration. The advantages of a preliminary filtration before the final sand filtration are apparently sufficiently great to warrant its adoption in certain cases, if not in all. It removes the coarser sedimentary matter and some of the bacteria, and enables the final sand filters to pass a much larger volume of water between the periods of cleansing.

The system designed by Mr. Armand Puech, and known as Puech's system, was first tried at Mazarnet, in France, afterwards at Nantes and other towns,

then in Paris, and recently by the East London Water Company, at their works at Hanworth. At Hanworth there are no storage subsidence reservoirs, and it was found very difficult to filter the requisite quantity of water per day when the river water was turbid. The filter beds, six in number, are about five acres in extent, and two of the beds were often out of use at one time for cleansing purposes. By the introduction of Puech's preliminary filters this difficulty has been successfully overcome, and a satisfactory water can apparently be produced at all times.

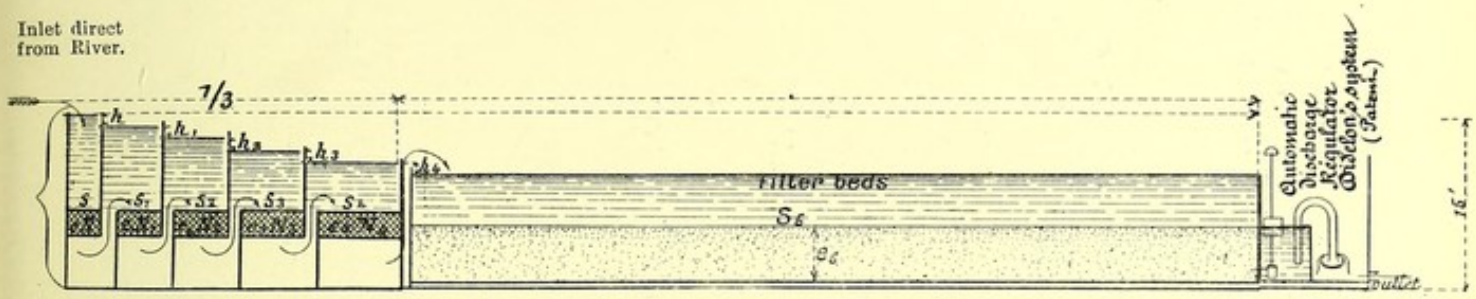

Fig. 8.

In the Puech filter (Fig. 8) there is a false bottom of perforated steel, upon which rests a layer of washed gravel. In the first filter this gravel is coarse, in the second finer, and in the third the gravel is about the size of peas. A fourth and fifth filter can be provided, if necessary, covered with still finer material. At Hanworth three filters have been found sufficient for the preliminary filtration. The three Puech filters have one-tenth the area of the sand filters. The raw water from the river passes into the first filter and through the course gravel downwards into the cavity below the steel plate. Thence it passes to the surface of the second filter, with little loss of head, traverses this, again passes forward to the third filter, from which it is discharged upon the sand filter. When the river water is turbid the effect of the Puech filters is most marked, and bacteriologically there is a great improvement. The greater portion of the organic matter in suspension is retained by the Puech filters, and the sand filters can do their work very much better, besides leaving a much greater area always in use. The gravel filters can be cleaned easily and quickly by mean of a hose pipe, one filter being cleaned while the other two are working. The following tables give details of various installations: —

Water supply.	Source of water.	Proportionate area of Puech filters to sand filters.	Daily average output, million gallons.	Quantity filtered between scrapings in million gallons.
Paris ...	R. Seine ...	$^1/_{10}$ ...	4 ...	100 to 125
„ Suburban ...	„ ...	$^1/_3$ ...	$7\frac{1}{2}$ ...	300 to 400
East London ...	R. Thames ...	$^1/_{10}$ ...	$10\frac{1}{2}$ ...	100 to 125
Nantes ...	R. Loire ...	$^1/_{10}$ ...	4 ...	125 to 165
Annonay ...	Brook water ...	$^1/_5$ ...	$1\frac{1}{4}$ ...	165 to 190
Bombaste ...	R. Meurthe ...	$^1/_2$ ...	·165 ...	415 to 500

Mr. Bryan, the engineer to the East London Company, has kindly given me the following table showing the bacteria in the river water before and after it has passed through the Puech and sand filters from the date of installation to February last. The results appear to be eminently satisfactory:—

		No. of bacteria in 1 c.c. of Unfiltered water.	Filtered water.			No. of bacteria in 1 c.c. of Unfiltered water.	Filtered water.
1902				1903			
Sept.	6	1667	73	June	6	1824	45
„	13	2086	77	„	13	6960	119
„	20	1300	40	„	20	26686	245
„	27	5246	29	„	27	5334	46
Oct.	4	1500	27	July	4	1706	12
„	11	25060	5	„	18	1413	16
„	18	1553	14	„	25	2360	13
Nov.	1	5446	5	Aug.	1	2466	57
„	8	1266	15	„	8	1376	90
„	15	1303	62	„	15	26207	60
„	22	2300	11	„	22	13227	4
„	29	4413	62	„	29	4293	7
Dec.	6	7566	32	Sept.	5	1906	15
„	13	6546	17	„	12	10136	5
„	20	4500	106	„	19	2423	40
„	27	7330	14	„	26	1680	24
1903							
Jan.	3	2866	4	Oct.	3	23646	115
„	10	12280	41	„	10	10546	28
„	17	5573	46	„	17	29580	85
„	24	31180	77	„	24	9953	29
„	31	8200	17	„	31	12953	76
Feb.	7	7620	10	Nov.	7	8026	12
„	14	11656	6	„	14	11400	22
„	21	20220	7	„	21	28093	9
„	28	23100	52	„	28	12573	10
March	7	17026	68	Dec.	5	72404	67
„	14	7006	56	„	12	41936	54
„	21	3753	13	„	19	17645	22
„	28	2093	11	„	26	4590	9
				1904			
April	4	12773	16	Jan.	2	2966	32
„	11	5080	4	„	9	9613	106
„	18	1008	28	„	16	9253	29
„	25	1920	8	„	23	5420	22
May	2	3713	8	„	30	13260	16
„	9	7373	14	Feb.	6	57187	74
„	16	4553	22	„	13	6120	25
„	23	4245	11	„	20	15033	17
„	30	2246	20	„	24	2716	12

The averages for the 18 months give

In the river water	10,121 bacteria per c.c.
In the filtered water	36 ,,
Percentage bacteria removed	99·65 ,,

On five occasions only have the bacteria exceeded 100 per c.c., and on one occasion only has this figure been more than slightly exceeded.

Dividing the above period into two of equal length, we find:—

	Average No. of bacteria in River water.	Filtered water.	Percentage of bacteria removed.
Sept., 1902, to May, 1903, incl. ...	7,225 ...	30 ...	99·59
June, 1903, to Feb., 1904, incl. ...	13,550 ...	44 ...	99·68

The first period covers nine months, when the river water rarely contained over 10,000 bacteria per c.c., whilst the second covers a period during which the river was very often in flood, and the water contained a very excessive number of bacteria. Although a few more bacteria were contained on an average in the filtered water during the second period than in the first, the percentage purification was higher.

From a previous table we have seen that filters require scraping when, on an average, 40 to 50 million gallons of water have passed through per acre; whereas the above table shows that with the addition of Puech's filters, 100 to 500 millions of gallons may be passed. The advantages therefore of a preliminary rough filtration are undoubted.

Mechanical Filters. Time will not permit of my dwelling upon the subject of sand filtration at greater length, as the "mechanical filter" still remains to be considered. I may say at once that I have never examined one of these mechanical filters, treating a polluted water, which gave satisfactory results, but it does not follow that there are not many cases, even in this country, where they could be used with advantage. The number of these filters is legion, but they resemble each other in these respects that the filtering material is contained in a tank or cylinder, usually of iron, that the water is passed far more rapidly through the material than through an ordinary sand filter, and that at short intervals the direction of the current of the water is reversed so as to wash out of the filtering material the matter which it has removed. Where very turbid waters have to be treated it is usual to add a little aluminic sulphate as a coagulant, allow the alumina to deposit in a subsidence tank and pass the supernatant water through the filter. In this country the use of coagulants is probably never necessary, we need not therefore give it further consideration.

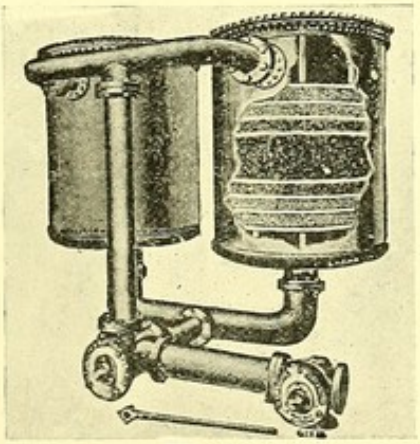

Fig. 9.
Type of Mechanical Filter.

Jewell Filter. One of the best types of mechanical filter is that made by the Jewell Company, of New York, who have recently fixed some at the Wolverhampton and York Waterworks, but merely for effecting a preliminary clarification before submitting the water to sand filtration. The Jewell filter (Fig. 10) consists of a tank containing sand of great uniformity resting upon a suitable screen carrying a series of radiating pipes with nozzles for collecting the water. The thickness of the sand is four feet. The rate of filtration may be varied from 1,600 to 3,200 gallons per square foot per diem, equal to 70 to 140 million gallons per acre. To maintain this rate a head of from 6 to 14 feet of water is required, but it does not follow that there must be this head above the sand surface. A relatively small head upon the sand with a larger negative head below is found to give the best results. The upper three inches of the sand is found to strain out most of the suspended matter, and when this upper surface becomes too clogged for filtration to be economically continued, the sand bed is washed by reversing the flow of water. A current, preferably of filtered water, is forced upwards with considerable velocity through the sand, washing into the drain provided for the purpose the accumulated filth. From time to time the sand can be further cleansed by putting into the water a little caustic soda and blowing in steam.

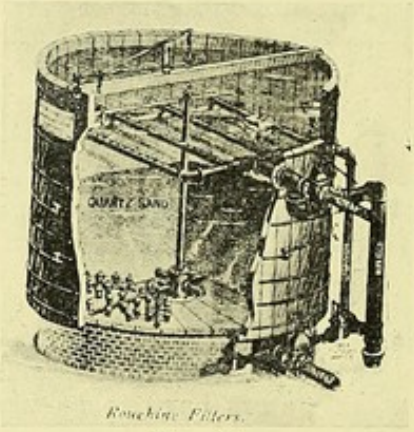

Fig. 10.

Even with these mechanical filters it is found necessary, for producing uniform results, to maintain a uniform rate of filtration, by increasing the pressure gradually as the porosity of the sand decreases. The Jewell filter has an automatic controller and gauges showing the regularity of the increase of the filtering head. Each filter can be controlled independently. It is claimed that this system of filtration possesses the following advantages over the sand filters: —

1. They occupy a very small area of ground.

2. They are not affected by weather.

3. Objectionable growths cannot take place in the filter, and therefore there is less risk of waters acquiring odour or taste.

4. They are easily and rapidly cleaned without risk of contamination by workmen.

5. There is absolute control over each filter.
6. The sand can be easily sterilized if required.
7. Water can be sent directly from the filter to the consumer with the least possible delay and exposure.
8. They can easily be adapted for removing lead or for filtering water after softening.
9. Small subsidence reservoirs only required, if at all.
10. Small cost of construction and maintenance.

The Jewell filters installed at York to filter the Ouse water were guaranteed to give, when using the coagulant, a filtrate free from colour and visible suspended matter (not more than 10 parts per million by weight), and if the unfiltered water contained 1,000 or more bacteria per c.c. to remove at least 90 per cent. of these. If the unfiltered water contained less than 1,000 bacteria per c.c., the filtered water was not to contain more than 100. Further, the filtered water should not contain any trace of the coagulant, nor be increased in hardness. Official tests were made before accepting the filters, with the following results:—

	Bacteria per c.c. Unfiltered water.	Filtered water.	Rate of filtration per sq. ft. gallons.	Grains of alumic sulphate per gall.
Aug. 27	440 ...	20 ...	2,070 ...	1
Sept. 1	372 ...	7 ...	3,080 ...	·75
Sept. 3	190 ...	23 ...	2,780 ...	·75

Professor Bitter, reporting upon the suitability of these filters for the Nile water to supply Cairo, states that he obtained results better than those obtainable from the best working English sand filter beds. Using the B. prodigiosus, he found that after the filter had been in use $2\frac{1}{2}$ hours, although the unfiltered water contained 100,000 of the bacilli per c.c., the filtrate only contained 4. It is doubtful, however, whether his other results, if published, would have led us to the same conclusion, since he remarks: "The first experiments made by me with Jewell filter very soon showed that no definite conclusions as to the value of the system could be arrived at on the mere basis of the number of bacteria contained in the filtered water."

Pittsburg Experiments. Many extended series of experiments have been made in America with these mechanical filters. Of these the Pittsburg experiments are probably the most interesting. The water to be filtered is derived from the Rivers Alleghaney and Monogahela. It is always turbid, the turbidity usually varying from ·1 to ·5, but occasionally, after heavy rains, becoming 1·0 or more. The results obtained were as under:—

Kind of Filter.	Effective size of sand.	Uniformity co-efficient.	Coagulant used.	Rate of Filtration in mill. gall. per acre, per diem.	Bacteria per c.c. River water.	Filtered water.
English Sand Filter, with preliminary sedimentation	·30 m.m. ...	2·0 ...	None ...	2 to 5 ...	16,340 ...	129
Ditto without preliminary sedimentation	·63 m.m. ...	2·0 ...	None ...	2 to 5 ...	16,340 ...	177
Warren Mechanical Filter...	·63 m.m. ...	1·1 ...	1·36 ...	115 ...	11,427 ...	262
Jewell Filter	·46 m.m. ...	1·4 ...	1·0 ...	104 ...	11,427 ...	459

After washing a filter the water was found to be of an inferior quality for about 20 minutes. The English sand filters clogged very rapidly, especially the one using unsettled water, and the bacterial efficiency was not so marked as when settled water was used, notwithstanding that the settling removed few bacteria.

Mr. Hazen, who reported on the results of these experiments concludes:— "With an amount of sulphate of alumina which makes the cost of the two processes substantially equal, the mechanical filters yield effluents containing from two or three times as many bacteria as the sand filters, and are consequently 2 to 3 times as likely to transmit disease germs, while, on the other hand, the effluents are clearer and more nearly colourless. If the raw water were very much more muddy than it is and contained less sewage, the advantage would be on the side of the mechanical filters. If, on the other hand, the water was less turbid and contained more sewage bacteria the advantage would be decidedly with the sand filters." I am afraid this opinion would not help the Pittsburg people much in deciding which system to adopt.

York Observations. At York, where the water is first "passed through mechanical filters and then through sand filters, a water of exceptional bacterial purity is obtained, as is shown by the following results published by Mr. Humphreys, the Engineer.

		Number of bacteria per c.c. in Unfiltered water.		Filtered water.
1902	July	198	.....	5
	August	410	.....	0
	September	211	.....	2
	October	289	.....	1
	November	742	.....	1
	December	913	.....	2
1903	January	698	.....	2
	February	245	.....	4
	March	560	.....	3
	April	2,270	(heavy floods)	15
	May	295	.....	1
	June	203	.....	4

Candy Filter. There is one other system of mechanical filtration which is worthy of mention, since it originated in England, and does not entirely depend upon sand for its efficiency and is in use in several important towns. Possibly also the fact that the two towns in which it has been longest in use have the lowest death-rates from typhoid fever may also justify me in selecting it for special mention. I refer to the Candy filter, in which polarite (a magnetic oxide of iron) and sand form the filtering medium. The closed tank is divided into chambers containing polarite and sand. The unfiltered water enters under slight pressure, and in doing so is sprayed so as to become charged with compressed air. Each compartment of the cylinder is a separate filter. The water charged with dissolved oxygen from the air traverses these

filters, and not only are bacteria removed, but a considerable portion of the dissolved organic matter is oxidised. By reversing the direction of the current of water the suspended matter which has been removed by the filter is carried away. Polarite is also used in the construction of slow filters, layers of polarite being laid under the sand. At Hastings and Leighton Buzzard polarite filters treat ferruginous waters very successfully. At Reading the very impure water of the River Kennett is converted into a water of great organic purity, and containing very few bacteria. The South-West Suburban Water Company have also constructed polarite filters, and have improved the quality of their water.

Water of River Kennett, before and after treatment through Polarite.
In parts per 100,000.

	Before.		After.
Free Ammonia	·0001	.....	·0000
Organic Ammonia	·0214	.....	·0043
Oxygen absorbed...	·391	.....	·124

Thames water after polarite filtration:—

	Waterworks, Egham.
Free Ammonia	·0000
Organic Ammonia	·0020
Bacteria per c.c.	40

Selection of Method of Filtration. Having thus discussed, though briefly, the various methods of filtration, we are in a position to consider the circumstances under which each is specially applicable. First, however, it must be understood that the mechanical system is cheaper than the English sand system, both in first cost and in upkeep. Other things being equal, therefore, this alone would decide in favour of the mechanical system.

In the first group of cases in which filtration is necessary merely for the removal of mineral matter in suspension some form of mechanical filtration would obviously suffice and be cheaper than ordinary sand filtration. The mechanical filter would possess all the advantages enumerated when we were considering such filters. In the second group of cases where the object is the removal of low forms of vegetable life other than bacteria I am inclined to think that the mechanical filter has other advantages besides the cost. There is less probability of any organism predominating to such an extent as to cause the water to acquire an odour or taste, since the filtration takes place in a dark enclosed chamber. With excessive growth of minute vegetable organisms the ordinary filter bed would very speedily clog, giving much extra trouble, whereas in the mechanical filter the extra trouble involved in cleaning more frequently is negligible. This applies to many water supplies from uncultivated moorland surfaces, but not to such supplies if any considerable proportion of the collecting area consists of cultivated ground.

These latter would be included in our third group, in which the object of the filtration is chiefly the removal of bacteria of objectionable origin. Cases are conceivable in which the amount of pollution is relatively so small that

mechanical filtration might suffice to ensure safety, but where the pollution is so gross as is the case in most of our rivers mechanical filtration alone could not be trusted. At present slow sand filtration should be insisted upon, and all the precautions now taken at so many places on the Continent adopted. Further experiments with dual filtration, as in Puech's system at the Hanworth works, may show that such uniformly satisfactory results can be obtained, that all the precautions now considered necessary may not be imperative.

The final group of cases, in which the object of filtration is the removal of organic matter in solution, necessitate special treatment. At present the only way of removing such matter in which I have any confidence is filtration through a polarite medium. Probably in most such cases there will be also objectionable matters present derived from sewage or manure, rendering efficient bacterial treatment also necessary. Slow filtration through sand and polarite may suffice in such cases, but it would be safer to submit the water first to the action of polarite in a mechanical filter and then to pass it through a sand filter. The former would remove much of the organic matter and most of the bacteria, and the final sand filtration would ensure a filtrate of a high degree of bacterial purity.

No hard and fast lines can be laid down. Every supply necessitates a special study, and in many cases experiments would have to be conducted before an opinion could be given as to the best system or combination of systems to be adopted. Mechanical filters have been greatly improved during recent years, but where dangerous bacteria may gain access to a water we are not in a position yet to say that such appliances can be trusted to remove them, whilst in these, the most important cases, we know that sand filtration can be so conducted as to be an absolute safeguard.

The literature of the subject is very voluminous, but much of it is of little importance. Results obtained are so often buried in masses of tables and in successions of reports that it is exceedingly difficult to draw any useful conclusions from them. The time at my disposal has not permitted me to deal very fully with the subject, but I believe that I have discussed all the points of major importance, and probably I may atone for my shortcomings by adding to my lecture, a list of some of the works and reports which may with advantage be consulted by those who wish to give the subject further study.

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Atmospheric Carbonic Acid, its Estimation and Variation.

BY

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Atmospheric Carbonic Acid, its Estimation and Variation.

Historical Remarks: Joseph Black. It is just 150 years since Professor Joseph Black first demonstrated the presence of Carbonic Acid in the air. Black was recognised as one of the most expert investigators of his day, and his discovery of carbonic acid or "fixed air" as he called it was one of his several most important achievements. His investigations on the Carbonic Acid in air and on the composition of carbonate of lime were done when he was a young man at Glasgow University. On his promotion to be Professor of Chemistry at Edinburgh University Black appears to have given up original research work, and to have devoted all his energies to teaching an art in which he became as accomplished and as successful as he was in investigation work.

Black and those who followed him appear to have enormously over-estimated the amount of Carbonic Acid in the air. From 50 to 1,000 volumes of Carbonic Acid per 10,000 volumes of air were supposed to be present by the various investigators who worked at the subject during the 18th century.

John Dalton. It was not till the inventive genius of John Dalton of Manchester was brought to bear on the subject that approximately accurate results as to the amount present were obtained. Dalton was engaged in investigating the subject in 1802, and he soon demonstrated that instead of the atmosphere containing one per cent. of Carbonic Acid it contained less than one-tenth of a per cent. by weight, *i.e.*, 6·8 volumes per 10,000 volumes.

Dalton collected the air in a bottle of a capacity of 471 cub. in.—far too small as we now know. Lime water or Baryta water was added in known amount and of known strength, and after absorption of CO_2 the solution was titrated against dilute sulphuric acid. He collected the air sample by first filling the bottle with water and then emptying out the water in the situation where he wanted to collect his air sample. This was a bad plan, and the error it gave rise to was demonstrated by Dalton's distinguished pupil, Hadfield, in 1828.

Hadfield. Hadfield improved the method of collection by using a dry bottle and filling it with air by means of a bellows.

Dalton's process with the modifications suggested by Hadfield is as good and even better than many of the processes described in our text-books to-day. I will ask you shortly to consider whether we in this country are not entitled to speak of Dalton's method rather than Pettenkofer's.

De Saussure. Between 1810 and 1830 De Saussure, of Geneva, published the results of his work with a gravimetric method. He precipitated the Carbonic Acid in about 34 litres of air and weighed the resulting barium carbonate. By this method he showed that the mean amount of CO_2 in air was about 4·03 volumes per 10,000 volumes of air.

Pettenkofer. The next important and far-reaching researches in atmospheric carbonic acid were made by Pettenkofer by a method almost identical with that of John Dalton and his pupils—a dry bottle, the air forced in by means of a bellows, Lime or Baryta water used as an absorbent and "any non-volatile dilute acid for titration." Angus Smith says that the use of the oxalic acid for titration is the essential of the Pettenkofer process, but obviously that is wrong for Pettenkofer himself says "any non-volatile acid" will do.

In view of all the circumstances I now ask you whether we, in this country, should not associate Dalton's name with the bottle method of determining CO_2?

Angus Smith. You have in Manchester another very interesting link in the investigation of Atmosphere Carbonic Acid, for it was here that Angus Smith did much of his work on the subject of the variation of Carbonic Acid in the air. Those who may not have read his "Air and Rain" will find that it contains much valuable information in regard to Carbonic Acid.

Having now indicated very briefly the scope of the older work on Atmospheric Carbonic Acid, I will next describe some of the more recent methods and afterwards the results.

Methods in Use at the Present Time. The methods may for the sake of convenience be divided into (*a*) those devised to secure the greatest possible accuracy irrespective of the amount of trouble involved, and (*b*) those designed for practicable purposes.

Those of us who are engaged in practical public health work, and who may be required to make Carbonic Acid determinations in examining as to the efficiency of methods of ventilation should have a knowledge of the degree of accuracy of the process we employ. Most of our text-books are vague and insufficient, and it will probably be found that the best results will be obtained by studying carefully, first, those methods which have the merit of scientific accuracy and then seeing how our available working methods differ in accuracy. In this way many errors will be avoided and confidence in our results obtained.

Haldane and Pembrey's Method. Of the accurate methods probably one of the best is that described by Dr. J. S. Haldane, F.R.S., and Dr. M. S. Pembrey in the "Philosophical Magazine," Vol. xxix., p. 306. In this method one or more absorption tubes are used filled with carefully prepared soda lime. In order to prevent the absorption of moisture from the air by the soda lime the air is passed through absorption tubes filled with pumice soaked in sulphuric acid. These observers aspirated 90 litres of air at the rate of one litre per minute. The soda lime tubes were weighed on a chemical balance before and after the absorption, and the difference taken as the weight of CO_2 in the air. As an additional precaution control absorption tubes should be

submitted to the same temperature and other conditions without being used for CO_2 absorption.

In comparing the accuracy of this method with the bottle method Haldane and Pembrey show that for air which is pure or relatively pure, the excess of CO_2 as indicated by the latter varies from 7 to 18 per cent.

There are obvious disadvantages in the use of the method for practical purposes. The amount of air 30 to 90 litres is inconveniently large, and the time required for the absorption of the Carbonic Acid too long for every-day work.

I recommend the method as one eminently suitable for checking the accuracy of any of the ordinary working tests. Since it is possible that any of us may now be required to take action and possibly prosecute on our results of CO_2 estimation, it is most important to be able to check our work against a reliable method.

Demonstration of the method was here given.

Dalton's Method and Modifications of It. The Dalton (or Pettenkofer) method has now been so improved as to be a reliable and quite satisfactory means of estimating the amount of CO_2 in air if care and attention are paid to the numerous details. A good deal of experience is required in order to avoid serious error.

Collection of Samples. (*a*) The *bottle used for the collection* of the sample of air should be large—at least capable of holding five litres and have a wide mouth. Its capacity should be accurately measured by filling it with distilled water at 60°F. to the top. An indiarubber cap is more convenient than any form of cork or stopper. The bottle should be washed clean and dried inside thoroughly. It is advisable that acid be not used for washing these collecting bottles as it is difficult to get rid of traces of it.

(*b*) The air may be filled into the bottle by using a *bellows* as a force pump, or by using it as an *aspirator*. The most convenient and best form is that suggested by Angus Smith. Such a bellows folds into small space, and when used as an aspirator enables the operator to place the bottle where the sample has to be collected while he himself may stand seven or eight feet away. (It is most important at all times to bear in mind the danger of error arising from one's own expired air.) The above form of bellows aspirator is used extensively in obtaining samples of gases in chemical works. One stroke of the bellows aspirates 1 cubic-foot of air. If the bellows is filled 10 times when working with a 5-litre bottle the air in the bottle will have been changed at least 20 times—sufficient to obtain a reliable sample.

Reagents and Procedure. After filling with air the bottle is capped and taken to the laboratory where 100c.c. *Baryta water* is added by means of a pipette. The absorption of the CO_2 is allowed to go on for an hour, the Baryta solution being shaken up at intervals. The Baryta solution is removed from the absorption bottle with the same pipette and filled into a bottle of about 120c.c. capacity from which all CO_2 has been previously

removed. This bottle has a well-vaselined stopper and the contained solution is allowed to deposit its carbonate by remaining quiescent for 24 hours.

Every precaution must be taken to prevent so sensitive a solution coming in contact with air containing CO_2 and specially with the expired air of the operator. When dealing with samples of pure air if the text-book descriptions of the process are followed the error will never be less than 25 per cent. and often will be as much as 50 per cent.

The solutions required as are follows:—

1. *Baryta solution* about 1 per cent. This should be stored in a large bottle having a soda lime inlet for air, and a special long outlet to admit of the pipettes being filled direct without suction being required.

2. The acid which I think is the best for all-round work is twice recrystalized *oxalic acid* of the strength 1·41 grammes per litre.* This should be dissolved in boiled-out distilled water so as to free it as far as practicable from CO_2. Much of the commercial acid is impure. It is easier to obtain an approximately correct oxalic acid than a hydrochloric acid when working in a laboratory where correct solutions are not kept in stock. Weak solutions of oxalic acid have the great disadvantage of not keeping well. They should therefore always be used fresh. Each c.c. of this acid = 1 volume of CO_2 per 10,000 when using 25c.c. of Baryta.

The titration. 25c.c. of the Baryta solution should be placed in a flask from which all CO_2 has been washed out and titrated with the oxalic acid, phenolphthalein being used as an indicator. Having obtained the accurate titre of the Baryta exactly similar procedure should be adopted with 25c.c. of the supernatent solution used as an absorbent. The 100c.c. allows of at least three such determinations. Quickness and care in preventing expired air gaining access to the Baryta is all important.

At one time the error in my experiment with the Dalton method amounted to 0·9 volume of CO_2 per 10,000. With the precautions I have above described it is less than 0·25 volume of CO_2 per 10,000 volumes of air when dealing with pure air. I have not estimated what the error is when impure air is being dealt with but anticipate it will not be greater.

Demonstration of method was given here.

Method Adopted by the Author. Another very convenient method which I have used a good deal within recent years and found even less liable to error is as follows. The air is collected in an empty india-rubber balloon inside a wide-mouthed bottle by being pumped in by means of a hand-bellows. The india-rubber bag, filled with the air, is taken and attached to an absorption tube so arranged that the air is passed through the Baryta solution in small bubbles at a slow rate.

The *absorption tube* was first devised by Pettenkofer and recently greatly improved by Dr. Gibson.

This absorption tube is filled to a mark with the Baryta solution in the same way as one would fill a pipette from the Baryta stock-bottle. Five litres

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*The acid should be tested against a standard acid and proved to be $C_2H_2O_4 2H_2O$.

of the air are drawn from the india-rubber bag through the absorption tube and into a graduated aspirator bottle. The absorption is very complete. Its efficiency may be controlled by means of a second similar tube connected with the first. After the whole of the air was passed through the contained Baryta solution is treated in exactly the same manner as that from the bottle. The advantage of this method is the ease with which an accurate sample can be obtained. A more accurate sample is obtained than with the bottle method. Its chief disadvantage lies in the fact that the passage of the air through the absorption tube takes at least an hour.

The above absorption tube is admirably adapted for the estimation of CO_2 in ground air. The absorption tube is directly attached to the air tube in the ground and the air is measured by means of a bottle aspirator.

Letts' and Blake's Method. Quite recently Letts and Blake have described an important modification of the Dalton process which will be found useful in ascertaining one's own working error by the ordinary process.

All bottles used to contain or receive Baryta solution were coated with paraffin wax to prevent the Baryta acting on the glass. The collecting bottles were filled by a method of exhaustion, 50c.c. of Baryta were run in from a special measuring pipette so that the Baryta did not come in contact with the air at all. After absorption is complete the whole of the Baryta is sucked into a special vacuous flask and then titrated. The whole process is carried out with the least possible chance of error. It is however tedious and the apparatus expensive. With known volumes of CO_2 added to air these observers found that the mean error per 10,000 volumes of air was only 0·04 volumes.

Haldane's Method. No account of modern methods for the estimation of CO_2 would be complete without a reference to the excellent work done by Dr. J. S. Haldane, F.R.S. in this connection.

The apparatus which he has devised is most ingenious and excellent in its arrangements. It enables an estimation of the amount of CO_2 in the air to be made in about five minutes, and in addition it has the advantage of enabling the examination to be made on the spot and without any calculation.

Dr. Haldane's description of the apparatus is so complete in the *Journal of Hygiene*, or in the report on the "Ventilation of Factories and Workshops," that it will be unnecessary to give here a detailed description of it.

Briefly it consists of an accurately graduated measuring pipette which allows of exactly 25c.c. of air to be admitted for examination. Having collected the amount of air the CO_2 is absorbed in a caustic potash solution. After complete abstraction of CO_2 the volume of the air is again read off at the same temperature and pressure. The difference between the first and second readings giving the volumes of CO_2 per 10,000 volumes of air.

This appears to be very simple and easy of application, but in practice great care is required to ensure that the air under examination is kept at exactly the same temperature and pressure and that the whole of the apparatus is in good working order.

My own personal experience with the apparatus has not been very great though I have used it at intervals during the past three years. I have had

some obviously inaccurate results when I least expected them, and it is the fear of a repetition of these results which makes me stick to one of the older methods which, though much more tedious, is, I think, more under control.

Demonstration of Haldane's apparatus was given here.

There are on the market many *other varieties of apparatus* for estimating the amount of atmospheric CO_2. Most of these have some defect rendering them inaccurate or else they are mere modifications of the Dalton process.

Corrections for Temperature and Pressure. A point which will arise in these determinations is as to whether it is necessary to correct for temperature and pressure. For all work where scientific accuracy is required such corrections must be made. On the other hand in estimations made with a view to ascertaining the amount of CO_2 for the purpose of judging of the sufficiency or otherwise of the means of ventilation correction to normal temperature and pressure is unnecessary as the temperature and pressure at which the samples are analysed are relatively uniform and therefore the results are fairly comparable.

Personal Error. A much more important point is to know accurately the amount of one's own experimental error. This will vary for each observer. If a uniform amount of care is observed the amount of the error will be consistently uniform. My own error with the Dalton method is 0·24 volume of CO_2 per 10,000 volumes of air, *i.e.*, if I find 3·24 volumes of CO_2 in air I know I am dealing with air containing three volumes only; if 20·74 the corrected figure would be 20·5.

It is most important to test one's results against such a gravimetric method as that of Haldane and Pembrey or that of Professor Letts and Mr. Blake.

The effect of experimental error is indicated by the statements in most of our text-books that pure air contains about four volumes per 10,000, when, as a fact, such air contains rather less than three volumes—an error of about 33 per cent. when pure air is being dealt with, or of 7 per cent. when air containing 14 volumes of CO_2 per 10,000.

Amount of Carbonic Acid in Air. Pure air was shown by Thorpe in 1866 to contain 2·95 volumes of CO_2. Numerous other observers who have avoided the most obvious errors in the Dalton method have obtained similar results, the two most recent in this country being:—

1. Haldane, who gives the following results:—

		No. of Analyses.	Average CO_2 per 10,000 vols.	
April 1st to September 30th	day.....	29	2·88	2·98
	night ...	22	3·08	
December to January	day.....	6	2·99	3·00
	night . .	5	3·01	

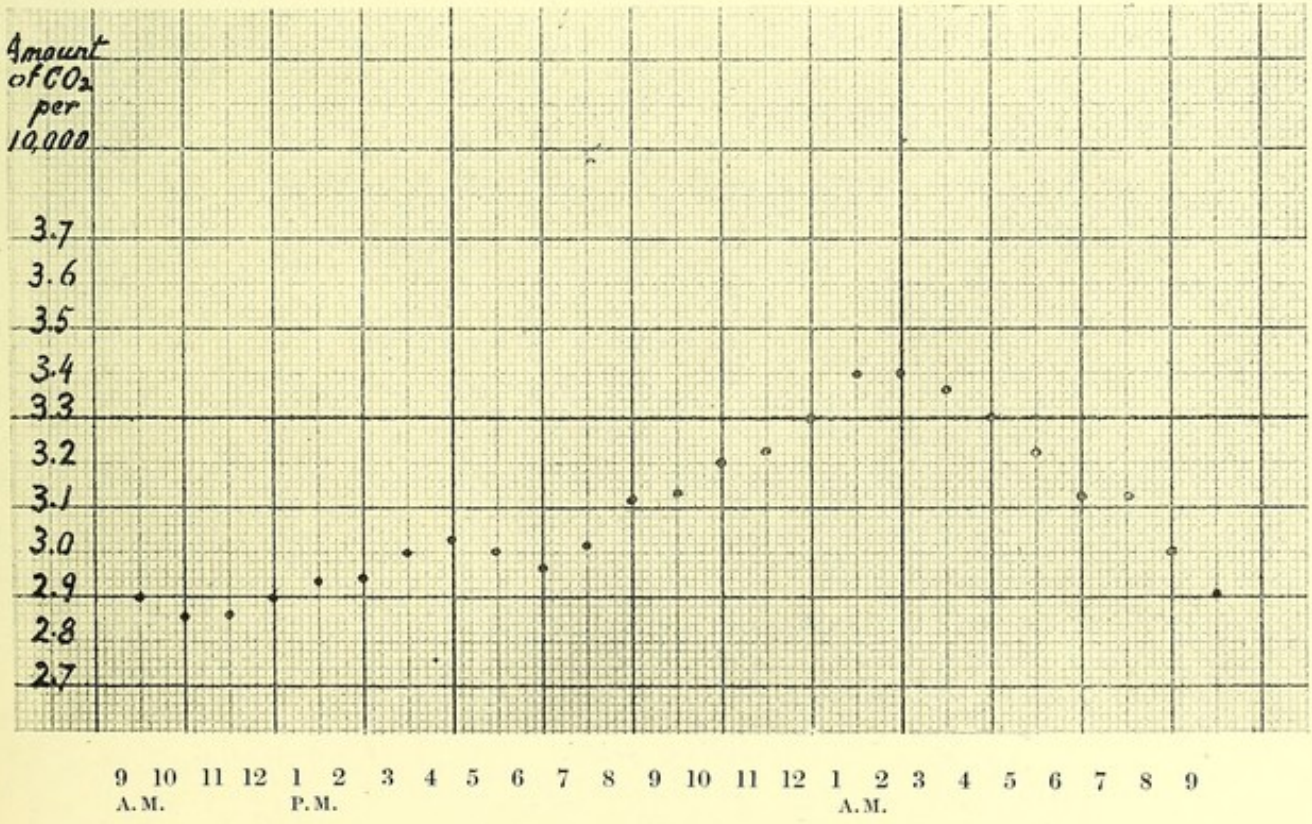
2. Prof. Letts and Mr. Blake ..... 46 2·91

Variations Due to Seasons and Weather. The variation in the amount of carbonic acid in the pure air of the country is extremely small from day to day. In the observations of Haldane noted above it varied from 2·58 to 3·55 volumes per 10,000 at different times of the year, and in different weather conditions. It is difficult to make use of the results of other workers for they are not strictly comparable owing to variations in methods.

My own observations are not sufficiently numerous but they indicate that during clear weather in summer-time the variation is slight if the observations be taken at the same time each day.

All observers are agreed that there is in summer-time more CO_2 in the air at night than during the day, due to the fact that plants only take up CO_2 during daylight and also to the escape of ground air from the warm soil into the cold atmosphere. There appears to be little doubt, however, that the amount of this variation is small and for practical purposes unimportant. My own figures show an absolute variation of 0·5 volume, *i.e.*, from 2·9 to 3·4.

But while the variations in atmospheric CO_2 in pure air are small and practically unimportant, there are many conditions in nature which produce departures from the normal.



Hourly variation in amount of CO_2 in atmosphere in Summer time.

Influence of Towns. All observers are agreed that the air of towns contains appreciably more CO_2 than does that of the country. The following are a few averages:—

Observer.	*District.*	*Town.*	*Country.*
Angus Smith	Manchester	4·3	3·69
Muntz	Paris	3·19	2·84
Spring and Roland ...	Liege	3·35	2·94
Williams	Sheffield	3·85	3·29

Influence of Large Water Surfaces. The amount of CO_2 is least over large water surfaces such as the ocean, and the diurnal variation is, as expected, absent.

Influence of Vegetation. The amount of CO_2 taken up by green leaves is very considerable. Under favourable conditions as much as $\frac{1}{2}$ litre of CO_2 can be decomposed by a square metre of leaf surface. This ought to play a very important part in influencing the amount of CO_2 in air, but air currents both upwards and lateral reduce the effect so that in practice the variation is unimportant.

Influence of Fog. Angus Smith was probably the first to demonstrate the importance of fog in increasing the amount of CO_2 in the atmosphere. His figures are for Manchester: During fogs, 6·79 vols. CO_2; ordinary weather, 4·03 vols. CO_2

Dr. W. J. Russell, F.R.S., made observations at St. Bartholomew's Hospital in 1882—84, and found that as much as 14·1 vols. of CO_2 may be found in a London fog, and that the mean results of 29 examinations showed 7·2 vols. of CO_2 per 10,000 vols.

The importance of recognising this fact will be obvious when estimations of the amount of CO_2 in the air are made for ventilation purposes.

The excess of CO_2 during fogs has been shown to be due to the accumulation of the products of combustion and decomposition due to the absence of air currents.

Influence of Rain. Rain appears to diminish the amount of CO_2 in the air by washing out a small proportion.

Von Fodor, at Buda Pest, gives the following results:—

	Before rain.	*During rain.*
1877	4·6	4·15
1878	3·86	3·53
1879	3·68	3·60

Influence of Season. There is a slight increase in CO_2 in winter. Peterman, working at Gembloux, in Belgium, gives the following:—

Spring	2·95
Summer	2·91
Autumn	2·92
Winter	2·95

Storage of CO_2 in the Soil. The great carbonic acid storehouse is the soil. Here bacterial life is constantly producing enormous quantities of the gas which concentrates in the lower layers, and in the upper layers is diluted by transfusion. From this storehouse CO_2 is given off during the night and during windy weather. Von Fodor, who has done so much in regard to the CO_2 in the soil has published a diagram of the season changes in the amount of CO_2 in the soil at varying depths.

My own observations indicate that the amount yielded by the soil varies very much in different soils and even in any particular soil the amount varies enormously from year to year with temperature, rainfall, and wind.

Influence of Respiration and Combustion. The question of vitiation of air by CO_2 derived from combustion and respiration is from the practical point of view the most important source of CO_2. Here again some most excellent work has been done in Manchester by Mr. Francis Jones, who has published in his "Airs of rooms" a most important contribution to our knowledge of the influence of combustion and respiration on the air in rooms.

Any consideration of the effects produced by combustion in the air of rooms would lead us too far away from the subject proper of our lecture. The amount of CO_2 in air from combustion and respiration is indicated by the difference actually found between the air in the centre and that in the suburbs of a large City. Specially is this noticeable when the movement of the air is a slow one and when convection currents are reduced to a minimum by the presence of fog.

Defective Sanitary Appliances.

BY

FRANCIS VACHER,

County Medical Officer of Health, Cheshire.

Defective Sanitary Appliances.

Introduction.

"Sanitary science and plumbing are synonymous," remarks an American writer, with amusing freshness. There are probably few on this side the Atlantic who would venture to endorse this statement, yet one may admire its spirit, and wish that plumbing were a little more sanitary and a little more scientific.

All defects in house construction are in one way or another dangerous to health. Plumbing and drainage work, however, seems to require very exceptional knowledge, skill and judgment, and when these happen to be wanting the work is done under conditions which make good work impossible, and the outcome is that health and comfort are affected in no small degree. The defective work may be due to ignorance, unhandiness, negligence, carelessness, hurry or deliberate wrong-doing. The blame attaching thereto is not wholly, or indeed primarily, the artisan's. The work may be ill-planned by the architect, or not planned at all. The contract to do the work may be at an impossibly low price, necessitating the employment of inferior material, or the time limit allowed for the work may be too short. Again, the artisan, though duly apprenticed, may never have been properly taught. Of course occasionally the actual operative is solely to blame—he may be idle and demoralised, or slovenly, and his work is but half done, or simply left undone.

The subject I am to bring before you is defective sanitary appliances. Whether these be due to lack of art, or lack of industry or lack of conscience it is not my present purpose to discuss. Whatever the cause, immediate or remote, the defective sanitary appliances are in our homes and our neighbours' homes now or lately. Wherever we live they are abundant, if not ubiquitous. I lay claim to no special information on this subject, but since 1885 it has much interested me, and I have been, so to speak, on the look out for defective appliances. The instances of faulty arrangements or apparatus which I have discovered, or had communicated to me, form, I think, an instructive collection, and some of these I now propose to bring before you. The first question I had to consider was how to represent the defective appliances effectively. I tried photography, but that did not seem well suited to illustrate this particular subject; then I tried lantern slides and again was not satisfied with the results. Finally, I decided that the most direct and simple way of illustrating such a topic was by means of diagrams showing each object to be displayed as it actually appeared or in section. The next question was one of order, *i.e.*, how to group my exhibits. For the systematic study of defective apparatus and arrangements the more convenient way of grouping examples is probably under

such headings as "rain conducting," "yard and area drains," "waste pipes," "soil pipes," "domestic cisterns," etc. For a lecture some other way of grouping seems better calculated to arrest attention and fasten on the memory, and the one I have chosen may answer the purpose as well as another. Any arrangement will prove arbitrary and in a measure unscientific, as an example often shows many defects and with equal fitness might be classed in two or three groups.

Antiquated and Obsolete Appliances. In a search of the kind I have undertaken some of the first appliances which appealed to me as worthy of remark were those which come under this heading. I give an illustration of antiquated traps occasionally found in connection with sink waste pipes. Fig. 1 shows what is called a *Buxton trap* in section. It consists of a pipe bent as indicated, and provided with a tightly-covered chamber, in which is a conical plug attached to a float. It was no doubt intended that when the water was discharged from the sink the float would rise and settle into position again as the water escaped. The plug was made exactly to fit the outlet and packed to be water-tight. This appliance was said to have an advantage over other traps as it would allow solids as well as liquid to pass it. However, there came a day when certain solids did not pass, but prevented the plug from fitting tight, and the return of foul air ceased to be checked. Fig. 2, a *duck-bill* trap, though a very different sort of trap, is quite as inefficient. The top of it is a strong metal ring, fitting close to the entrance to the sink water-pipe. To the lower part of this ring two curved plates are hung, which in the first instance rested one against the other, and when wet closed the mouth of the sink pipe; but very shortly small pieces of solid matter must have prevented the exact apposition of the curved plates, and the trap at once ceased to fulfil its functions.

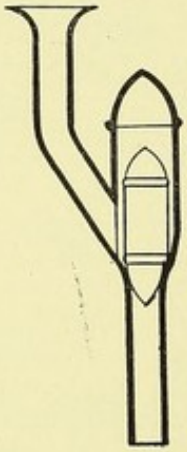


Fig. 1.

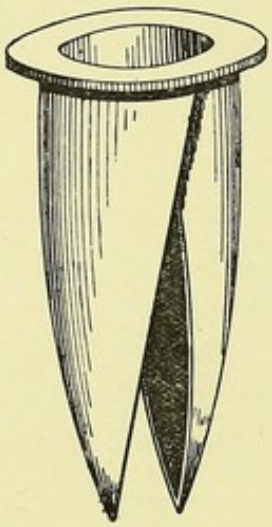


Fig. 2.

Both these traps are so manifestly bad that they are now rarely seen. The next example of a defective trap which I give is still frequently unearthed. It is apparently intended to serve as a ventilated trap, but as long as it contains any trapping-water neither the house side nor sewer side of the drain would be ventilated. The perpendicular portion, if continued to the surface, admits rubbish and does not facilitate the cleansing of the drain. When the perpendicular portion is completely sealed, as it is occasionally, no ventilation can take place even when the drain is dry. Should this sealing be incomplete, results may follow similar to those indicated in Fig. 3. The drain (6 inches in

diameter), a portion of which is here represented, was constructed of glazen earthenware pipes, cement-jointed, and having sufficient fall. However, as it passed under a plantation of young trees, a trap had been inserted as shown. The mouth of the perpendicular pipe was 6ft. from the ground surface, and being merely covered with a piece of slate (S), the roots (R) of the young trees in search for water found their way in, and a dense mass of fine root ends, felted together, filled the pipe, entirely obstructing the drain (D) which, on the house side of the trap was soon filled.

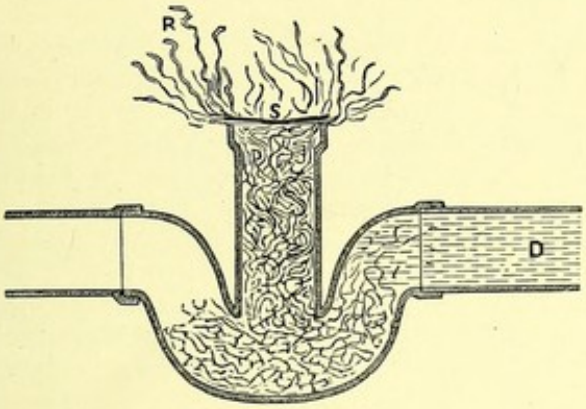


FIG. 3.

Many more examples of antiquated apparatus could be given, but I pass on now to

Castings Defective in Design. I give three illustrations under this heading, and I admit that before I found the three traps I am now to refer to I was quite unaware that various forms of cast-iron traps were quite ordinarily bought and sold which could by no possibility act as traps or trap anything except the purchaser. One of these traps is shown in section in Fig. 4. It was fixed in the rear of a house bearing a rental of about £65. It was properly connected to the drain, and certain waste-pipes, delivering on the top, kept it well supplied with water. A brief examination showed the trap to be a delusion, as the water stood an inch too low to form a seal. Effluvia in the drains were thus brought close up to the dwelling-house and discharged directly under the scullery window. The workman had set the trap quite square and made good connections, but no intelligent workman does his duty honestly who fixes a trap without first testing it. Oddly enough this trap went by the name of "Smith's Gully," and I found that Smith was a man who had received some training as a civil engineer, and who acted for many years as District Surveyor to a Local Board.

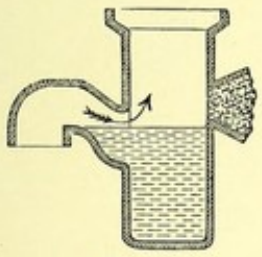


FIG. 4.

Fig. 5 depicts in section a so-called *box-trap*. When discovered it was supposed to be trapping the yard-drain of a house in an important thoroughfare in the centre of the town. A is the lid, perforated with 16 small holes, and carrying the useless box B on the under surface; C is the reservoir of water ($2\frac{1}{4}$ins. deep) into which the box should dip, but which it does not touch; and D is the outflow from the trap leading to the drain.

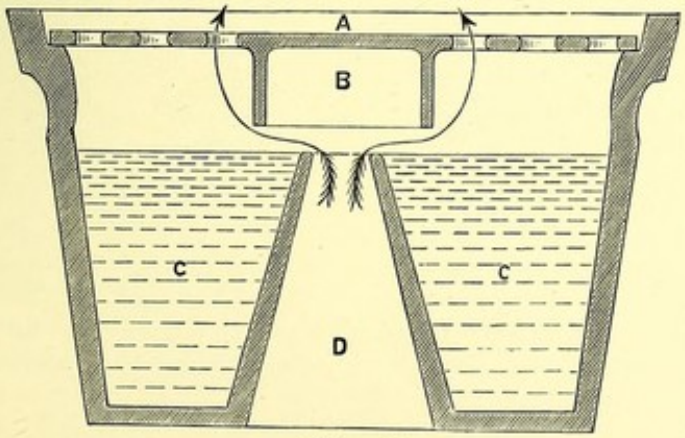


FIG. 5.

Fig. 6 shows in section a typically bad trap. Box-

traps are seldom fixed now, but *bell-traps* are still supplied by the hundred, and trusted to guard the sink waste-pipe. The trap depicted I examined and measured with great care, and then drew the section given. A is the perforated grating, B the bell, C the water reservoir — which the rim of the bell could never reach, — and D the portion of the casting which was continuous with the sink waste-pipe going straight to the drain. It will be noted that the distance between the rim of the bell and the surface of the water, when the reservoir was full, would be one-quarter of an inch.

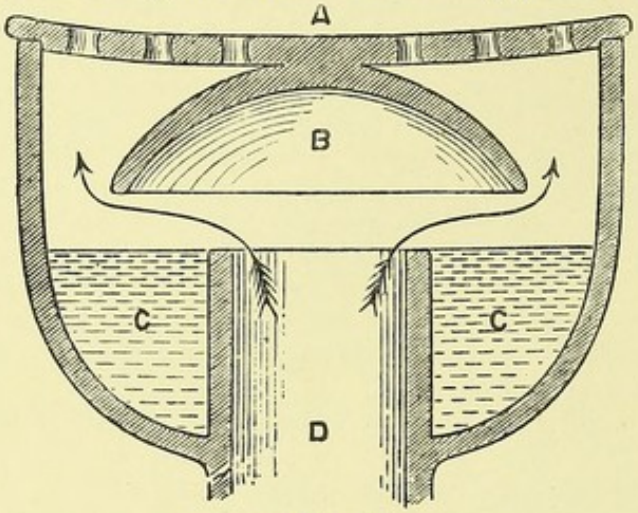


FIG. 6.

Flawed or Broken Appliances. When appliances are fixed which are either flawed or broken, it must be due to gross carelessness or deliberate wrong-doing. Flaws in lengths of rain-pipe and rain-guttering are apparently not uncommon. Fig. 7 represents one I saw. In the wall of a new public building flush with the street was a chase containing a 4-inch iron rain-spout. In this was a double crack, 13ins. long, which in course of time became converted into a hole as shown at A. The hole being 3½ft. from the pavement, P, caused an effluvium nuisance to foot-passengers, as the rain spout was directly connected to a soil drain. Such a pipe should never have been accepted and used.

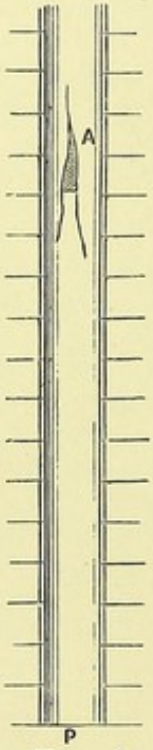


FIG. 7.

Fig. 8 is a sectional view of a glazed earthenware trap I discovered at the mouth of a yard-drain. A notice had been served on the owner to provide a proper yard drain, and I called to see if the work had been done. The tenant said the workman had finished the job a day or two ago, and took me into the yard where was the new glazed earthenware trap. As it did not hold water, even when freshly poured in, I had it removed and found a large hole in the back. I questioned the tenant as to whether anyone belonging to the house had broken the trap and he said it had not been touched since the workman left. The only conclusion open to me was therefore that a tradesman had been guilty of the fraud of selling and fixing a broken trap.

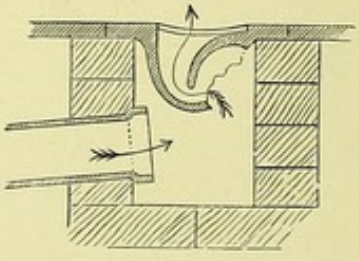


FIG. 8.

Appliances Put in Wrongly. Fig. 9 shows the uselessness of trapping a yard-drain with a loose trap of any pattern. The trap indicated in section, is a so-called *dish-trap* of cast-iron. It easily gets obstructed, and for this reason it is commonly inserted without any setting, so that it may be taken up and cleansed. Foul air and gases in the drain are, of course, not kept back, but find their way out external to the trap on all sides as shown in the illustration. The trap pictured was found on premises owned by well-to-do people who would hardly have objected to the cost of providing and fixing an efficient gully-trap if the defects of the loose trap had been pointed out to them.

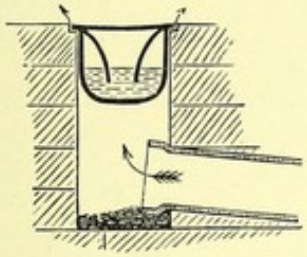


Fig. 9.

Fig. 10 is a sectional view of a trap which holds too little trapping water to be secure even had it been properly set. However, the workman who fixed it had done his work so negligently that owing to uneven setting the water-seal was completely broken. Such a blunder could hardly be made except in a yard paved with small boulders or very badly levelled.

Fig. 10.

Planning Defective to Save Material or Time. Defects appear to be often due to this cause. I am reminded of two in particular, both a little striking. Fig. 11 shows a back kitchen wall in section. On one side is the bottom of an area (A) and on the opposite side is a sinkstone (C) draining direct to a trap (D) fixed in the floor. This area, instead of being drained in a proper way, was drained to the sink-stone by a short lead pipe (B). What led to this part of the house being examined was an effluvium nuisance in the back kitchen which was traced to the trap. The trap was obviously sealed by a decomposing animal liquid, and the readiest explanation of this seemed to be that the area had been used after dark for a purpose for which it was certainly not intended.

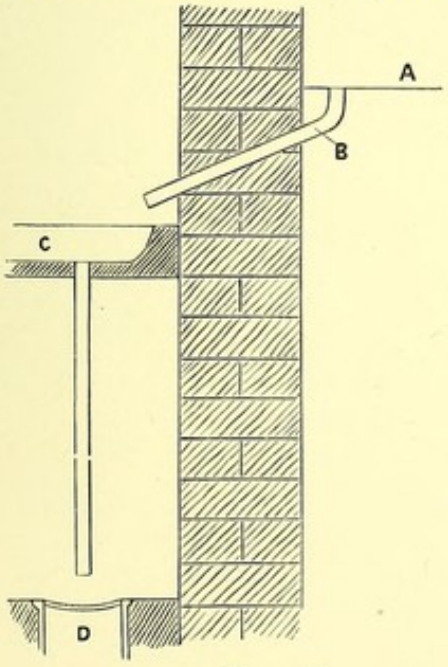


Fig. 11.

Fig. 12 illustrates a mistake, the exact opposite of the above. A sink in the basement of a large house had to be provided with a waste-pipe. As there was an " air-drain " all round the house, fixing this waste-pipe was not as simple a job as it might have been otherwise. The plumber apparently saw no

difficulty but did his work in the shortest and cheapest way. He pierced the wall and carried a short waste-pipe (B) into the "air-drain" A. The house where this occurred was a handsome suburban villa, exceedingly well built and provided with many modern conveniences. The air-drain about 14ins. wide, communicated with the air above by iron gratings (C) situated opposite the

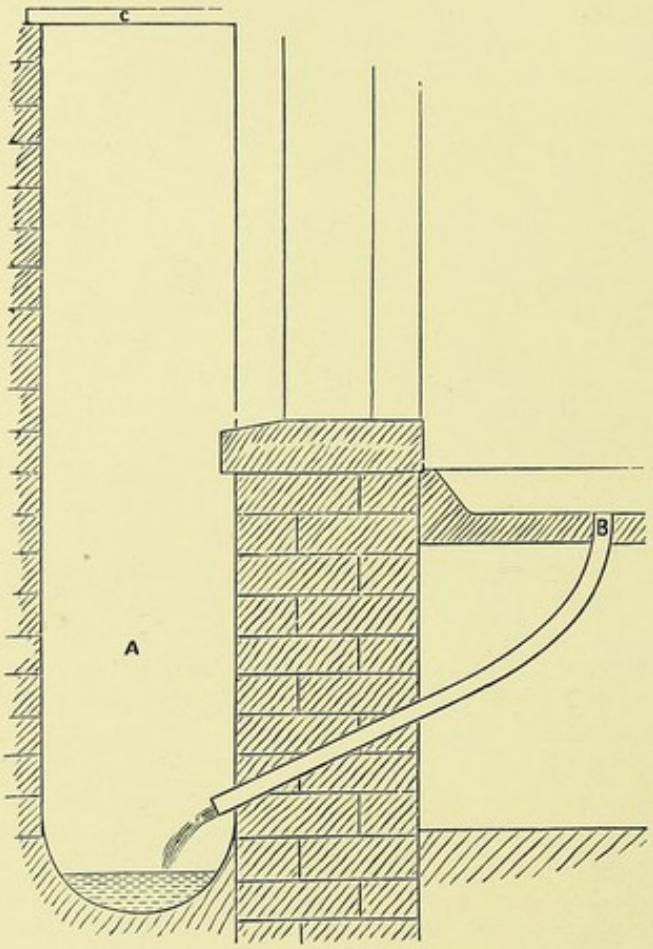


FIG. 12.

basement windows. The bottom of this area was cemented over and finished off as a channel—the area draining by a very slight incline to a trap placed in an inaccessible position. The trap got choked, and foul, soapy water, collected in the channel to the depth of some inches, and effluvia came into the premises through the basement windows. The house was indeed an island, surrounded on all sides by very foul stop-water.

A third example of a defect of this kind is shown in Fig. 13. I found the

arrangement in a house rented at £65 a year. The pantry was under the bathroom. One day soapy water was ejected from the sink waste-pipe (D) with considerable force, and as the bath (A) had just been used it was obvious that the soapy water came from the bath. On investigation it was found that the bath waste-pipe (B) had been connected with the sink waste-pipe above the trap (E), and this had become obstructed at the point marked (F). The pantry sink (C), being much smaller than the bath, was soon full and overflowing. The motive of this arrangement was probably to save a few feet of lead pipe, and make one trap serve both waste-pipes.

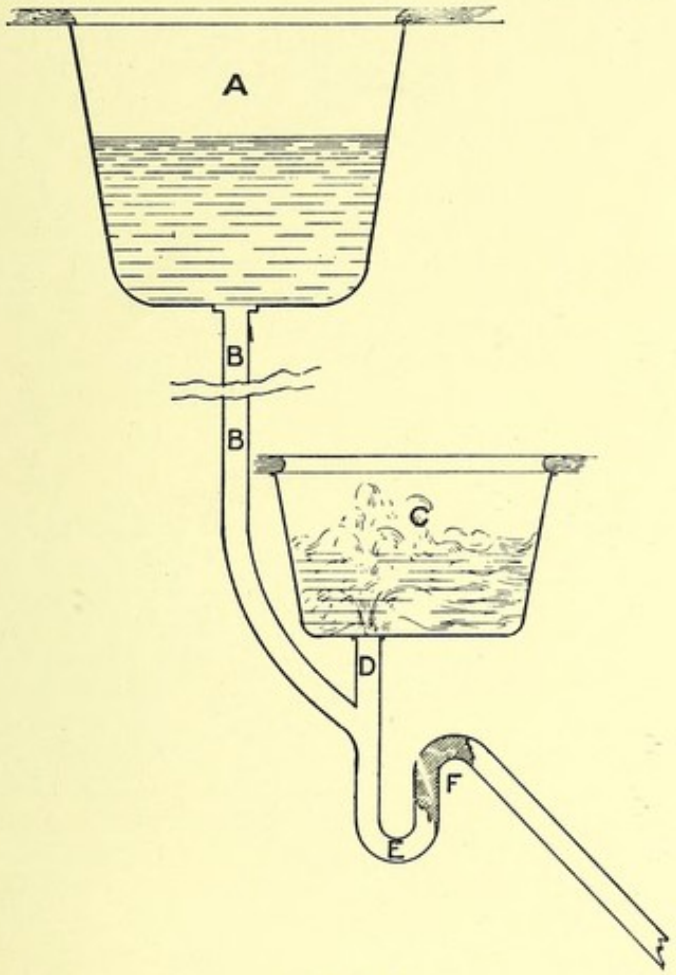


Fig. 13.

The fourth example I show under this heading must surely be unique. It is a curiously ill-planned service to a closet: the arrangement is shown in Fig. 14. Complaint was received from the tenant of an eight-roomed house in a terrace, of an effluvium nuisance chiefly noticed in the basement. The nuisance on an examination of the premises was traced to a portion of the ceiling in the basement, where the water-pipe, after being branched to the sink, disappeared. A rough hole had been knocked in a 4in. earthenware soil-pipe (D), and through this the lead-pipe had been passed. It was carried up inside the soil-pipe through two floors, and re-appeared (C) piercing the top of the lead trap (B), which was jointed to the basin (A).

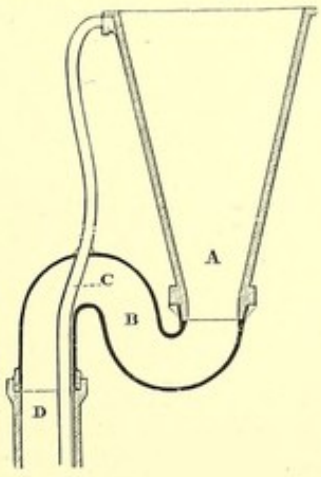


Fig. 14.

Planning Defective Owing to Misdirection. Delivering a bath or lavatory waste-pipe, as indicated in Fig. 15, is approved of in model bye-laws, yet waste-pipes thus treated may cause a great nuisance. The bath and hand-basin waste-pipes are brought outside to discharge on the area flags (B), which

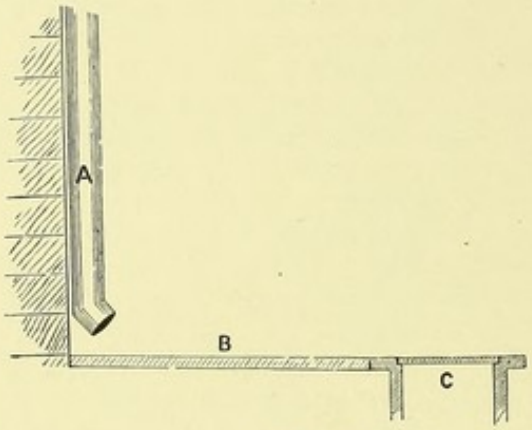


Fig. 15.

are channelled to conduct the water to a trap (C). Only one waste-pipe (A) is represented, but I have seen three together treated in the same manner. In some instances I have seen the trap a yard or more from the end of the pipe, and sometimes no channel or conductor to the trap is provided. In any case the flagging between the pipe and the trap is always foul, and effluvia find their way into the house at the kitchen or pantry window.

The method of dealing with the sink-waste shown in Fig. 16 is advocated by a distinguished engineer and enjoined by many Sanitary Authorities, yet the method is wholly bad. The arrangement will be seen at a glance. The sink-stone (A) has a short waste-pipe (B) which is directly connected with the basin of a closet (C) adjoining. In the case referred to the mouth of the waste-pipe had once been guarded by a bell-trap, but the top was lost when the premises were visited. The effluvia from the closet basin were directly conveyed to the sink-stone, and the only water the closet had to flush it was the more or less greasy slop-water poured down the sink.

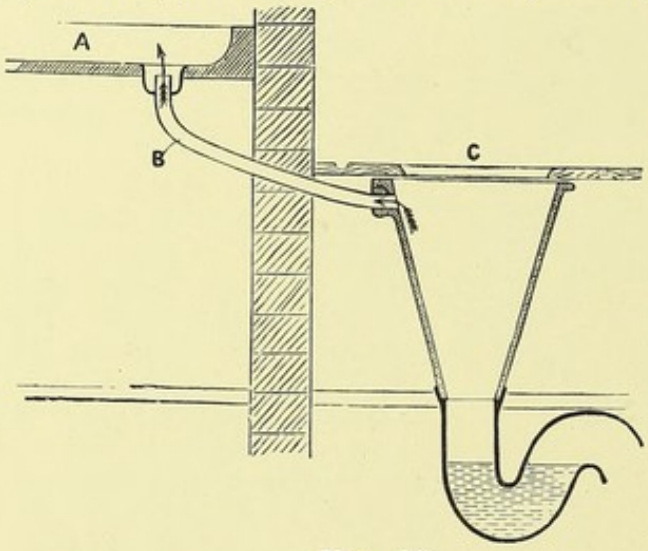


Fig. 16.

Planning Defective Owing to Gross Carelessness. It would be difficult to find a worse instance of gross carelessness than the one shown in Fig. 17. A large house was under inspection when unoccupied. Six months before a new bath had been fixed, and it was found that the waste-pipe from this (B) had been connected, to a rain-water pipe (A) close by. It chanced that the rain-water pipe discharged into a cistern under the house, but some distance off. As the bath had been in use for three or four months before the house was vacated, the cistern was much polluted. Connecting the waste-pipe with the rain-pipe was doubtless done in the belief that the rain-water ran to waste, but no serious effort could have been made to see if this were so.

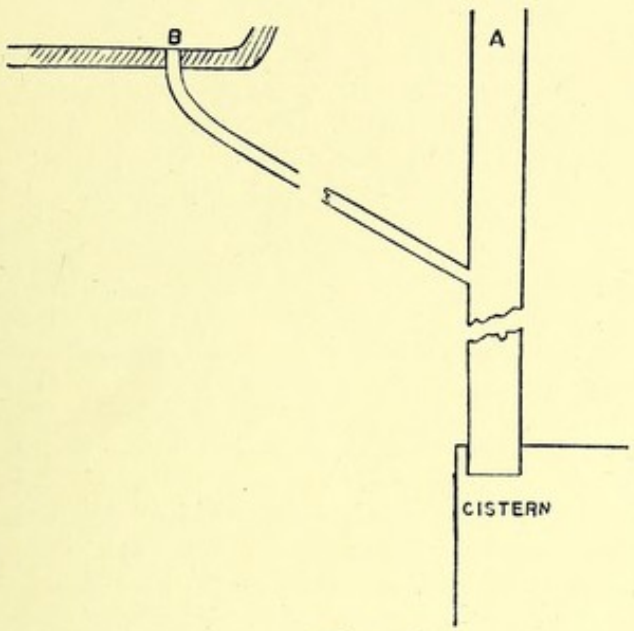


FIG. 17.

The only other example I shall give under this heading is perhaps the most interesting record I have. A case of typhoid fever had occurred in a very old house in a poor neighbourhood, and after removal of the patient to hospital the usual report as to the state of the premises was submitted to me. From the report I gathered that there was a water-closet built out from the back wall, a separate service cistern for the same, and a pipe from the main for drinking and domestic purposes. Not satisfied with this report, I examined the premises myself. I found the closet was entered from a small landing on the stairs, between the ground floor and the first floor. On looking to see what was under this I saw what is depicted in Fig. 18, viz., a sink-stone, with tap from the main, and immediately over the sinkstone the lead trap fixed under the closet-basin, and a transverse portion of soil-pipe connected therewith, which was carried through the brick wall into the house. In the soil-pipe close to the trap was a corroded hole (S). Through this hole foul air must often have been discharged, and foul liquids from the closet must often have overflowed. Indeed, here were all the conditions present which one would expect to find resulting in

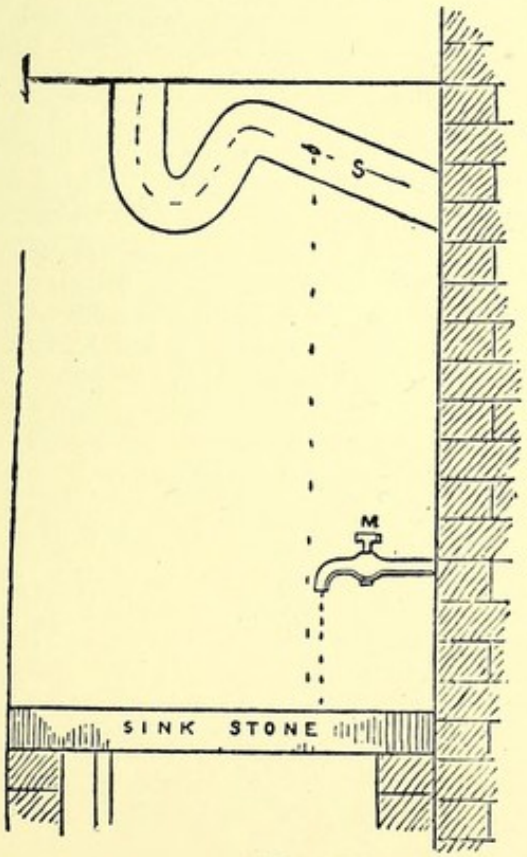


FIG. 18.

an outbreak of typhoid fever. What in all probability happened is that drinking water drawn from the tap was made foul by excremental liquid dropping into it, and the water thus polluted was actually drank. The illustration shows how easily a jug of water drawn from the main tap might be polluted by foul liquid from the hole in the soil-pipe.

Ventilating with Polluted Air. I give two illustrations of this. Fig. 19 shows what I saw at a suburban villa, rented at £70 a year. Complaint was made that water was coming in through the external wall of a room in the basement used as a washhouse. On investigation it was found that the water came through an iron grating that had been put in to ventilate the basement.. On the outside of this grating was a small area, which had been made to receive the gully-trap on which the sink waste-pipe delivered. As this area was covered by an iron grating, no attempt to cleanse it or the trap it contained had been made for a long time. The result was that the trap got filled to the brim with fat and other offensive matter, and then foul water discharged by the sink waste-pipe filled the area, till it reached the level of the ventilator and overflowed. Of course a ventilating-grating should never have been fixed in such a position.

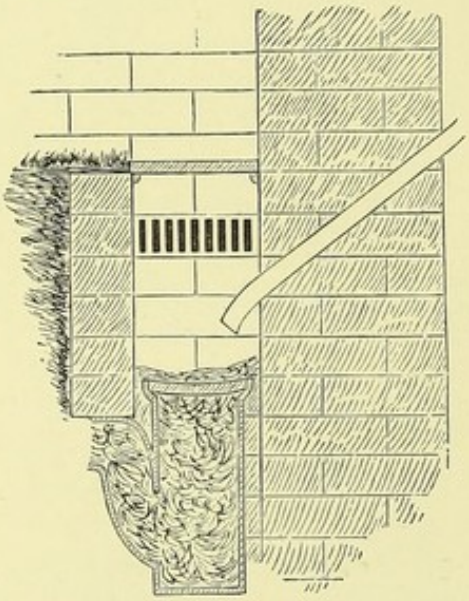


Fig. 19.

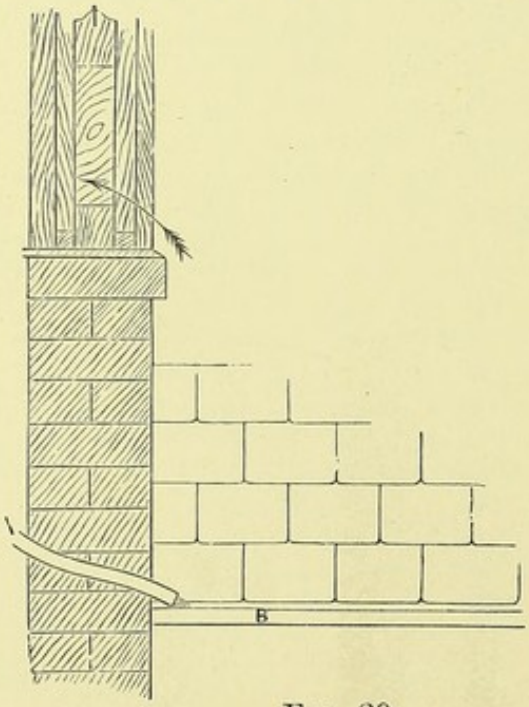


Fig. 20.

You will probably be surprised to hear that Fig. 20 illustrates a comparatively common fault. The sketch was taken from a large building used as a hospital. The waste-pipe (A) from two hand basins is seen discharging on a roof gutter (B). The foul water ran along the open gutter for five feet before it reached the downspout. Two feet above the gutter, which was ordinarily in a very foul state, was a window, which was often left open to air the room. The waste-pipe should have been brought down to a trap on the ground level.

Admission of Sewer-gas to a House. This is done frequently and in various ways. Two examples may suffice. Fig. 21 represents in section the top of a dwelling-house wall with slates and rain-gutter. It is thus seen how a rain spout carried direct into a drain or sewer delivers gases therefrom into the house. To simplify the sketch the framework of the roof is not indicated. B is where the framework should be, and A is the 3ins. rain-spout.

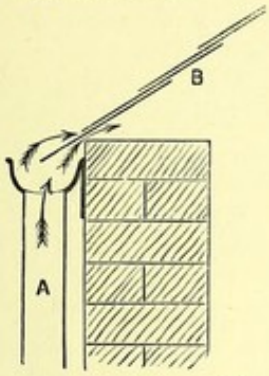


FIG. 21.

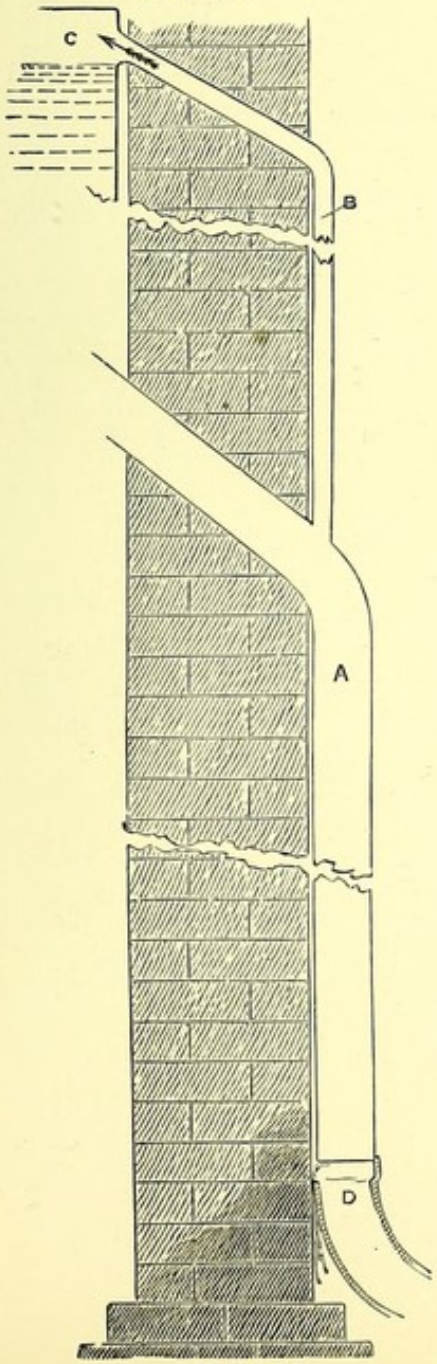


FIG. 22.

Fig. 22 shows an unventilated soil-pipe (A) taken outside the house directly it leaves the closet. As there was no proper connection between this and the house drain (D), the contents were allowed to escape to some extent and polluted the wall from opposite the leak to the footings. However, this did not prevent the sewer gases going straight up into the cistern room, for as will be seen the cistern overflow pipe (B) went direct to the soil-pipe, the effect being that the sewer was ventilated into the cistern room. There is nothing very unusual about this arrangement, bad as it is, except that where a cistern overflow is carried into a soil-pipe, the connection is usually made inside the house, the plumber probably being conscious that he is doing something irregular. To bring a cistern overflow through an external wall, carry it down some distance and connect it with an unventilated soil-pipe, points to an unconsciousness of wrong-doing in the workman, indicating that he had never been properly instructed.

The Defect of Trap-Circumvention. One would think that every plumber knew that the intention of a trap was to form a water-seal to keep back foul air. However, it is not unusual, when a trap is in position, for a workman to come round on the other side of the trap making it quite useless. Fig. 23 illustrates the most common way in which this is done. The bath (A) has a trap (B) on the waste-pipe, but this trap has been made of no avail owing to the overflow being connected to the waste-pipe between the trap and the house drain. The

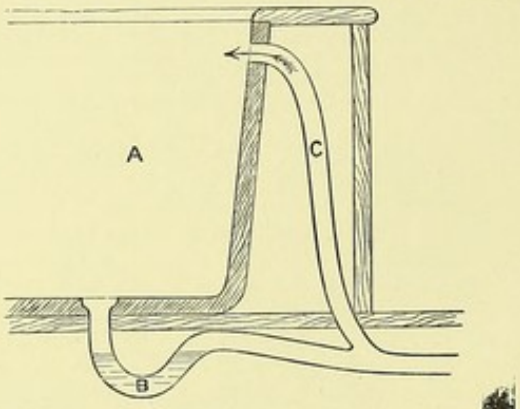


Fig. 23.

outcome was that the said drain was ventilated into the bath-room. Fig. 24 shows another way of making the same mistake. The old form of closet apparatus, with a container (C) below the basin (B), was ordinarily fitted with a "safe," or tray, to catch any foul water, etc., overflowing from the basin. The plumber apparently thought this tray wanted draining, and to drain it he carried a short pipe (D) therefrom into the soil-pipe (E), consequently circumventing his trap.

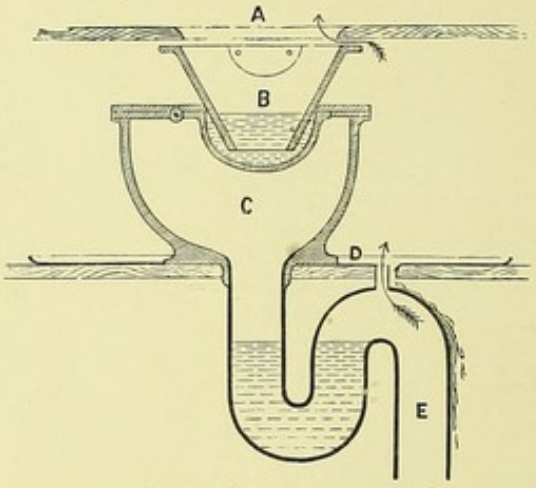


Fig. 24.

Yet another way in which the same result was achieved is indicated in Fig. 25. A closet basin (B) and trap beneath (C) were in one piece of glazed earthenware, and there was an opening (D) behind the trap for connecting a ventilator. In this instance no ventilator had been connected. A great nuisance was experienced, and on uncovering the apparatus it was found that the man who fixed it had filled up the hole (where the ventilator should have been connected) with *paper*, and covered this over at the top with putty. In course of time the putty loosened, and then the foul air from the soil-pipe had ready access into the house. Of course this foul air was rendered more foul by passing through a plug of paper saturated with the filth of years.

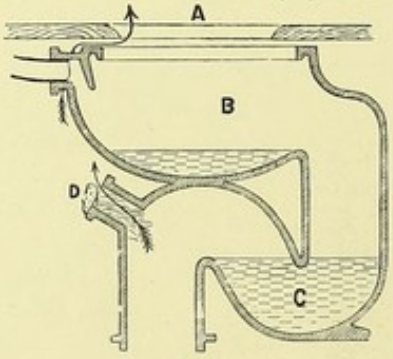


Fig. 25.

Unsealing a Trap by Syphonage. The possibility of a trap being unsealed by syphonage should always be kept in view. It happens under various circumstances, but its occurrence may, with care, be prevented. I have notes and sketches referring to many cases where it occurred, but I have only space for one example. The arrangements are shown in section in Fig. 26. The apparatus depicted was of the kind ordinarily supplied many years ago, but instead of having a D or S trap under the container, there was a sort of P trap. It is quite possible this form of trap may have been put in, instead of an S, as being less liable to syphonage. However, when the closet was flushed, so much of the trapping-water was swept forward out of the trap that the seal was broken. The basin is marked B, the container C, the trap D, the soil-pipe F, and E is the ventilator, carried right up through the roof. No doubt the plumber thought that this ventilator would prevent the trap seal being broken.

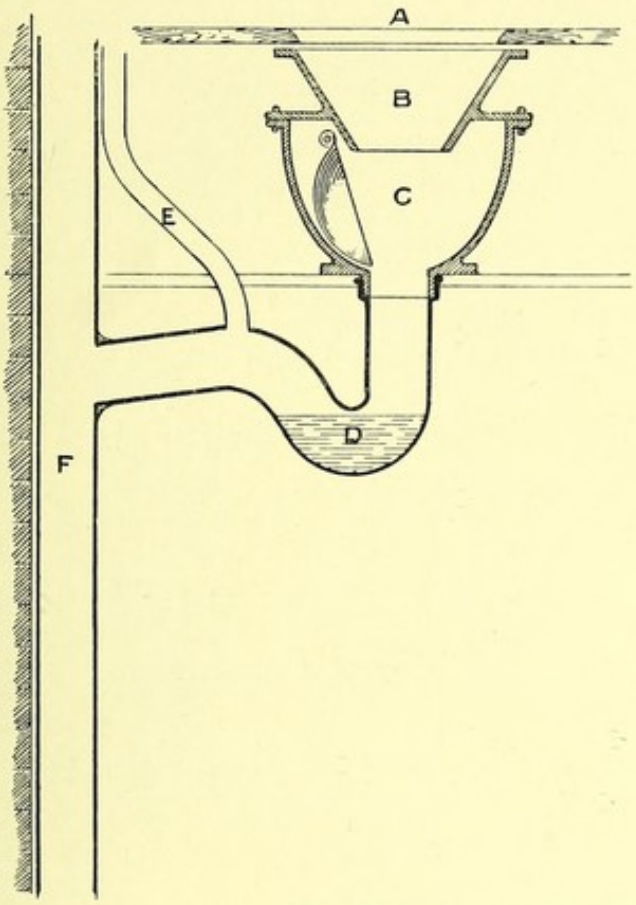


FIG. 26.

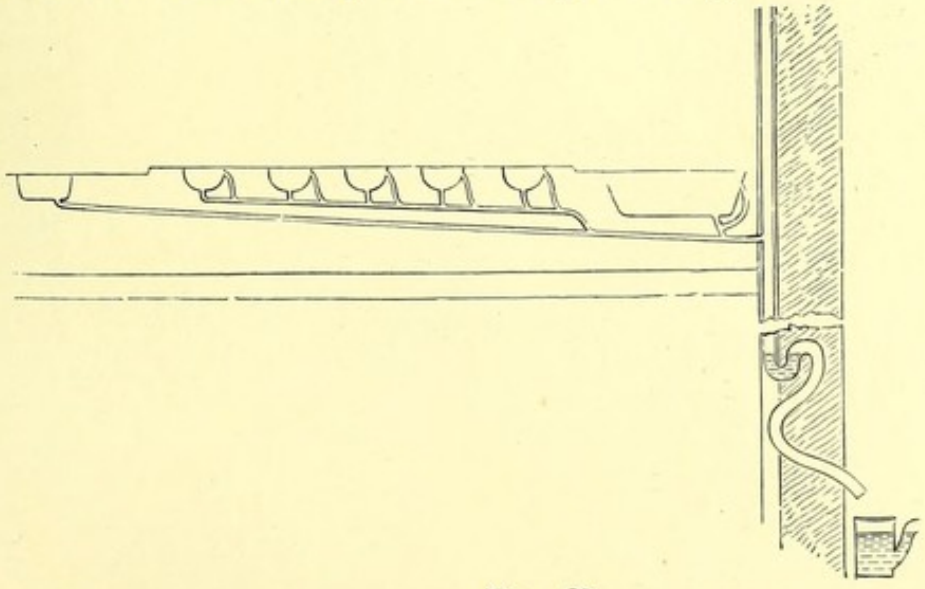


FIG. 27

Foul Air from Waste Pipes Driven into the House. This is often done, and nearly always in the same manner, by gathering many waste-pipes into one and trapping the outlet. Fig. 27 shows in section a typical instance of this defect. The waste-pipes and overflow-pipes from a bath, row of hand basins and housemaid's sink discharged into a 2½in. pipe in a chase in the wall. There were three stories, each provided with a similar range, all draining into the same 2½in. pipe, which was ventilated by being continued through the roof. As, however, this pipe was provided at the foot with a trap (embedded in the wall) there was no passage of air through it. The consequence was that when the ranges in the upper and middle stories were used, foul air was forced through the untrapped waste and overflow-pipes in the lower storey, and when the range in the upper storey only was used, foul air from the untrapped waste and overflow-pipes was perceived in the middle and lower storeys.

Not Making Provision for the Effects of Frost and Snow. This is accountable for much damage and inconvenience. What defect could be more obvious than that illustrated in Fig. 28? A service had been provided for the garden, and the tap not being placed at the lowest part of the branch, a portion remained which could not be emptied. The water had been run off from the pipes and cistern, as a frost was expected, but of course the *cul de sac* remained full and froze, and the lead was burst as indicated. As soon as the water was turned on again the rent was made manifest.

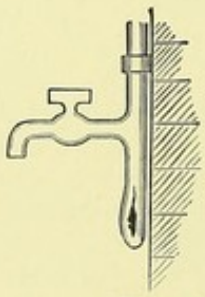


FIG. 28.

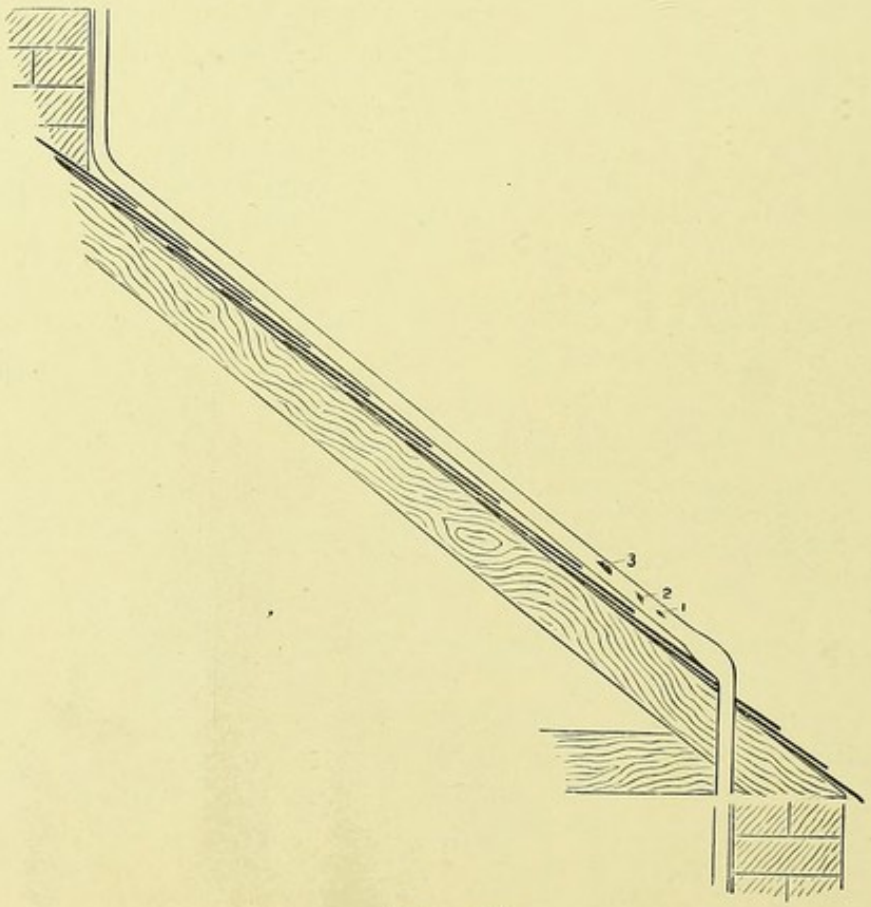


FIG. 29.

Fig. 29 shows the expansion pipe from a hot-water cylinder, which was ruptured by frost three times between Christmas and the middle of January. Every plumber knows that an expansion pipe should not be exposed to the action of frost, yet the pipe shown was carried along the roof, unprotected, for 5ft., and then up a chimney for 4ft. Just beyond the point where it pierced the roof there was a joint, and the first rupture occurred close to this at 1. After it was mended a second rupture occurred at 2, and after this was mended the pipe burst for the third time at 3. The chimney was from a kitchen and was practically always warm, and if the pipe, instead of being carried along the slates, had been made to pierce the roof where the chimney came through, it would have been sufficiently protected from the action of frost.

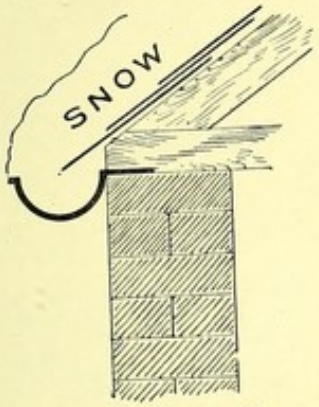


Fig. 30.

Fig. 30 indicates a defective way of fixing an ordinary half-circle eaves gutter, which I have seen at several houses. The gutter was fastened just too high, and in time of snow prevented the snow sliding off the roof. In some instances, had the gutter been placed two inches lower, it would have offered no obstruction to the roof clearing itself after a snowfall. When the extent of roof-surface is considerable, gutters may be wrenched out of position by the weight of snow held up.

Removing an Appliance after Inspection. This is not as exceptional as it should be. I only give one example. The defects produced in this instance were simply the result of an impudent attempt to evade the law. I noticed four houses where the soil-pipe ventilators terminated close to back windows. In three cases the ends were on a level with the tops of the windows, and in one case a foot above the top. As the requirement with respect to soil-pipe ventilators is that they shall be carried up not less than 2ft. above the roof eaves, and the houses were quite new, special inquiries were instituted. Under the local Act, passed in 1881, it is "not lawful to let or occupy as a dwelling-house any building, not so occupied before the commencement of the Act, until such building has been certified by the surveyor, or other officer appointed for the purpose, to be as to drainage, supply of water for domestic purposes, and in every other respect fit for human habitation, and constructed and completed in conformity with the provisions of the Act and the local bye-laws." It transpired that these four houses had been examined, and as the soil-pipes there terminated as required (2ft. above the roof eaves) the houses were duly certified as fit for habitation. However, shortly afterwards other houses approached completion, and iron castings necessary to carry the ventilators 2ft. above the roof eaves not being

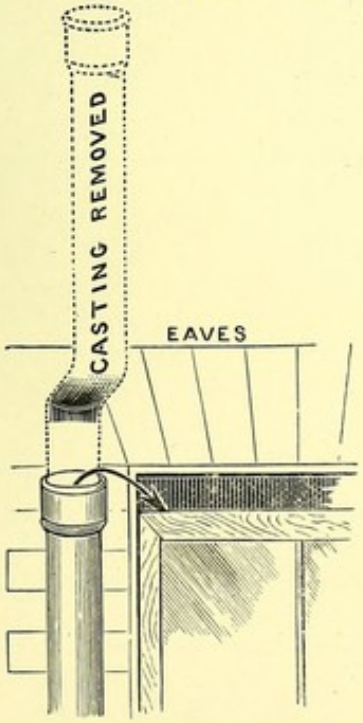


Fig. 31.

ready to hand, those belonging to the four houses already certified were removed, and made to do duty in obtaining a certificate for other houses. When the houses thus robbed were inspected, three were visited, and in each of these three the window next the soil-pipe end was open for two or three inches at the top. Fig. 31 explains itself.

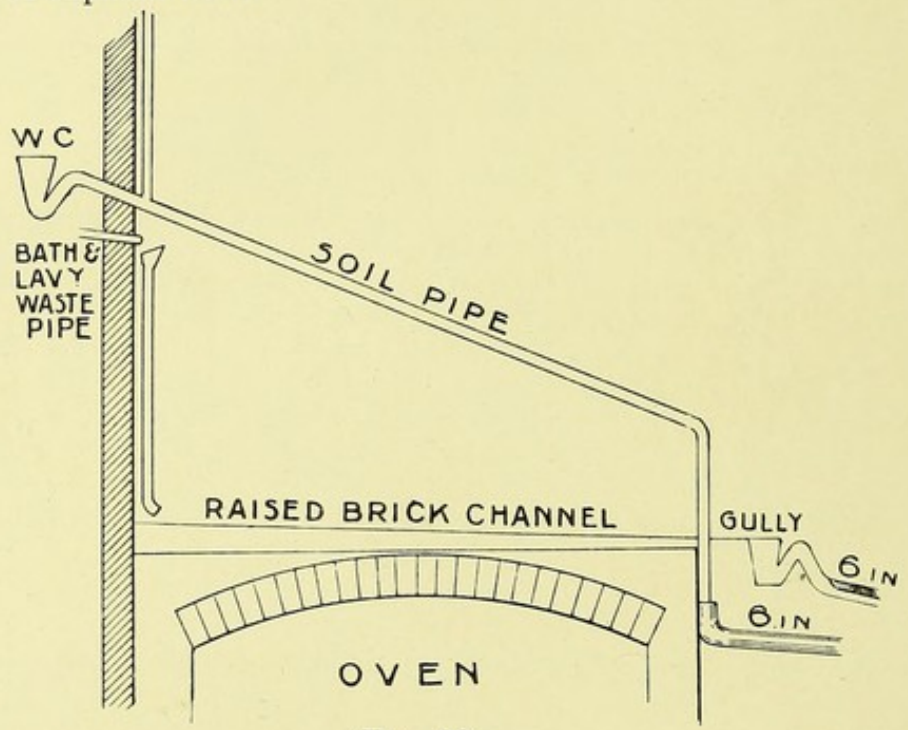


Fig. 32.

Polluting a Baker's Oven. Much attention has of late been given to underground ovens, and it is hoped that in a short time they will be altogether things of the past. The last diagram I produce indicates how liable to pollution these ovens are. The baker's oven to which Fig. 32 refers was situated immediately under the back yard of the baker's house and shop—a back yard which was just big enough to hold it. The bath and lavatory waste water was discharged on an open brick channel, and so conveyed to the gulley. Any little obstruction in the channel made it overflow. The lower part of the soil-pipe and the upper end of the 6ins. drain to which it was connected adjoined the oven wall. Both 6ins. drains discharged into a 6ins. main drain in the back passage.

In conclusion, it only remains for me to say that barely the fringe of this important subject has been touched in my lecture. However, it has not been my object to deal with defective plumbing systematically, but rather to awaken an interest in it and show what very curious arrangements there are hidden and unsuspected in many modern dwelling-houses.

It may be well to add that more efficient training in plumbing and kindred work is now having the attention of the Worshipful Company of Plumbers, London, and many Technical Instruction Committees throughout the country.

Report upon an alleged Effluvium Nuisance attributed to the use of Yeast in a Tannery, and upon an Outbreak of Diphtheria.

BY

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Report upon an alleged Effluvium Nuisance attributed to the use of Yeast in a Tannery, and upon an Outbreak of Diphtheria.

Preliminary Remarks.

Certain manufacturing processes are associated with the production of gases which have an unpleasant smell and are, on that account, sometimes supposed to be detrimental to health. The occurrence of an outbreak of disease in the neighbourhood of a factory where strong smells are generated may, at times, strengthen a mistaken belief in the existence of danger. This was actually the case with regard to an alleged nuisance supposed to arise from a tannery, and which was investigated for the Rural District Council of Runcorn in January, February and March, 1903. An account of that investigation has already been given to the Council in March, 1903, in a Report where full details about procedure and documents consulted may be found. In this communication a shorter account of the same investigation is given. The specimens exhibited in the laboratory are samples of the various kinds of tanning material, tan liquors, tannery effluents, which are mentioned in the Report. The collection of yeasts isolated from these various substances has been prepared by Dr. Carver, and a description of these yeasts will be found in the special report contributed by him. (See *The Characters of Yeasts Occurring in Tanning Material*, etc., by J. R. Carver, p. 279.)

I. INTRODUCTION.

During the month of November, 1902, I was requested by Dr. Adams on behalf of the Runcorn Rural District Council to investigate whether the smells arising from Mr. L.'s Tannery, Cliff Lane, Grappenhall, constituted an effluvium nuisance dangerous to health. This request was made owing to a complaint made by Mr. N., a gentleman residing in the neighbourhood of the tannery.

Summary of the Complaint. 1st. The smell from Mr. L.'s tannery is a very great nuisance to the neighbourhood, it constitutes an *effluvium nuisance dangerous to health*, which has rendered Mr. N.'s house virtually unfit for habitation.

2nd. This smell is not an ordinary one; it strikes people in various ways, causing painful choking sensations, nausea, vomiting, diarrhœa, faintness, etc.

3rd. The smell has been the cause of much fatal disease in the Thelwall district.

4th. The nuisance appears to be due to changes in the method of tanning. Prior to these changes no inconvenience was caused by the tannery.

5th. This effluvium nuisance is due "*to a septic ferment, in this case yeast Cells.*"

The smell is described variously as:—

Horrible stench, putrid smell, smell of sulphuretted hydrogen, smell burning throat and chest, keen acrid stench, putrid stench mixed with burnt stench, etc.

The effects produced on various persons are said by them to be:—

Burning of throat and chest, sore throat, diphtheria, diarrhœa, faintness, choking, loss of voice, dyspnœa, depression, flow of tears, etc., etc.

The smell has been attributed successively to decayed foreign hides left on a heap of manure, to old soaks, to secret tanning processes and the use of yeast, to the tannery brook (ditch along Waste Lane), to one tip of spent tan (in the fields) the deepest parts of which are supposed to generate the stench.

Specific statements have been made regarding some of the dates upon which the smell had been noticed to be very bad. I give them with the names of the persons responsible for these statements:—

1899 December 17. (Scarle).
1900 January. (Leah).
1901 February 16. (Vickers).
1901 August 17. (12-30 to 5 a.m.). (Leah).
1902 August 1. (Leah).
1902 August 23. (Scarle).
1902 December 22. (Leah, oral statement to me on the day of my visit).
1902 December 30. (Mr. N. from reports).
1903 January 2. (9-30 to 12 a.m.) id. id.
1903 January 4. (Leah).
1903 January 6. (Leah).
1903 January 10. (Leah).
1903 January 14. (Mr. N. from reports).
1903 February 21 and 22. id. id.
1903 February 27 and 28. id. id.
1903 March 4, 7, 11. id. id.

It will be noticed that these dates correspond to the period during which I conducted my investigation. Also to the period during which the tannery was watched closely day and night on behalf of the County Medical Officer of Health.

II. INSPECTION OF THE TANNERY.

Description of the Locality in 1900. As I had no opportunity of seeing the state of things which existed when the alleged nuisance was first formally complained of, and as the existence at that time of a certain brook and cesspool at one side of a portion of Waste Lane is a matter of importance, I think it best to reproduce here parts of Dr. Vacher's report to the Runcorn Rural District Council, dated July 12th, 1900, and which has been communicated to me by the district Medical Officer of Health. *(See Map, p. 23).*

"Leaving the Knutsford Road, we came to a highway, which we were "informed was known as Waste Lane, and on private land, but close to the "lane was a polluted water course or ditch, about 85 yards in length. Waste "liquor from the tannery, and sewage from four houses were discharged "into this ditch at one end, and polluted it for the greater part of its length. "A large sewer from Cuerden Hall and four other houses, and a small "sewer from a house and 10 cottages, discharged sewage into this ditch at "the other end, and polluted it for a short distance only. The polluted "liquor from both ends of the ditch was received into a round tank about "12 feet in diameter, and holding about a foot of liquor. This tank was "arched over, and difficult to obtain access to, and there was no evidence "that it was periodically cleansed. This tank when inspected was full of "an exceedingly foul semi-liquid material. At the side nearest the road "was a fixed weir, and over this the tank overflowed into a channel, under "Waste Lane, and under the Bridgewater Canal, to a brook which eventually "reaches the river Mersey. That the brook is polluted in this way there is "no doubt.

"We next carefully inspected the tannery, being shown over it by "Mr. L. We noted the various processes to which the skins were "subjected, from the time they arrived as salted dry hides, till seven or "eight months later when they are finished leather. The tannery is a large "one, turning out as we are informed from 250 to 300 hides per week, but "the whole manufacture appeared to be conducted in a very cleanly manner, "and was as free from effluvium nuisance as any tannery I have hitherto "inspected.

"We were shown the small stream above as it reaches the tannery; very "little water was coming down, and what there was was not free from "pollution. This water not being sufficient for the purposes of their "business, the Messrs. L. have a deep well, and from this they pump "about 11,400 gallons daily. The effluent being discharged at the time of "our visit was (judging by its appearance and smell) a good one for a "tannery, though it is probable that it would not have passed Sir H. "Roscoe's standard on analysis."

"Is there an effluvium nuisance? As the result of my inspection of the "tannery, in company with your Committee, I am bound to say that there "was no effluvium nuisance created by the tannery at that time. It may be

" that the tannery and its environments were in exceptionally good order,
" and I gather from your Medical Officer's Report that on another occasion
" ' the process of drying fleshings appeared to cause a distinct smell nuisance
" from the tannery.' There is therefore evidence from your Medical Officer
" of Health, as well as from the owner of Cuerden Hall, that there has been
" an effluvium nuisance in the past, but the fact that there was no effluvium
" nuisance at the tannery on the afternoon of the 11th inst. indicates that
" the business can be conducted without effluvium nuisance.

" However, on walking along Waste Lane, bounded for 85 yards or
" thereabouts by a ditch or watercourse, containing the tannery effluent and
" the sewage from 20 houses or cottages, I did perceive an effluvium nuisance.
" Is there pollution of a brook? Obviously this question must be answered
" in the affirmative. This little brook appears to be polluted to some extent,
" before it reaches the tannery, and, there, is polluted by the tannery effluent,
" and the sewage from twenty houses or cottages."

State of the Tannery on September 6th, 1902. Dr. Vacher visited the tannery again on September 6th, 1902. I think it is desirable to quote the following passages from that Report:—

" The County Medical Officer of Health visited Messrs. L.'s tannery,
" Grappenhall, on September 6th, and examined the premises as he had done
" on many previous occasions. The tannery, so far as he could judge,
" seemed conducted in the usual way. The skins are soaked, and kept in
" lime and water till the hair can be readily removed, the hair is then
" scraped off, and the skins are trimmed and put in the tan pits, and when
" the tanning is completed the skins are dried and dressed. Comparatively
" little effluvium was noticed, and what there was seemed similar to the
" effluvium in other well-conducted tanneries. What is trimmed off the
" skins is classed as ' picked sizings,' ' rough sizings,' and ' fleshings.' The
" ' sizings,' after being in lime and water for many days, are dried first in
" the open-air and then in a warm chamber, and the fleshings after being in
" lime and water for two or three weeks, are carted away wet once or twice a
" week. There was a little effluvium from the ' fleshings,' but practically
" none from the ' sizings.' The lime, after being drawn from the liming
" pits, is deposited in a field near the tannery. The deposit was visited,
" and no effluvium was noticed therefrom.

" The Medical Officer was informed that the material used for tanning
" alters from time to time according to prices. The materials at present in
" use in Messrs. L.'s tannery are ' Valonia,' ' Myrobolams,' oak bark, and
" extract of bark. The Manager stated yeast had been used in the past, but
" none had been used for fully two months. The spent tan is deposited in
" a field about 400 yards off. This deposit was inspected, and was not
" noticed to give rise to an effluvium nuisance."

. . . . . .

" The Runcorn Rural District Council have just laid a pipe sewer ($2\frac{1}{2}$ feet
" in diameter) in the ditch which used to receive the effluent at the side of
" Waste Lane. The public road will be widened and extend over this

"sewer. The sewer will convey Cliff Lane Brook along Waste Lane, and "the contents of Mr. N.'s sewer, under the canal. A drain from the "tannery will discharge the tannery effluent into the sewer. On September "6th the connection had not been made, but the Rural District Surveyor "showed the County Medical Officer where the drain would come in.

"The Tannery Manager stated that Mr. N. and Messrs. L. had drawn "the attention of the Local Government Board to the alleged nuisance, but "that so far as he knew the Board had not investigated it.

"Mr. N. used to reside at Cuerden Hall, in Thelwall, but has lately "resided in London, having, he alleges, changed his residence on account of "the effluvium nuisance he complains of. A ditch which used to divide "Grappenhall from Thelwall was converted into a pipe drain or sewer by "Mr. N.

"Mr. L. states that no change has been made in his method of tanning "which would produce an effluvium nuisance, or poison the effluent, and "that the effluent has been much improved in recent years. As regards the "beginning of his business at Grappenhall, Mr. L. states that Mr. Leigh, "of Belmont, purchased the Cliff Lane Tannery in 1845 or 1846 when the "buildings were enlarged for Mr. L.'s father's firm. The tanyard was "bought of Mr. Peter Wright (and his son-in-law John Wilmot) who was "then 80 years old and had tanned there for many years. Mr. John N., "father of Mr. N., built the house known as Cuerden Hall probably about "1866, when the effluent from the tannery was discharged into the brook in "a very crude state. Mr. N. altered and enlarged Cuerden Hall about 1893, "and the first intimation of complaint from him was in 1900, when he was "invited to call and see the arrangements then existing for clarifying the "effluent. . . . . .

"In conclusion, the County Medical Officer of Health submits:—

"That neither on September 6th, nor on any previous occasion when he "visited Messrs. L.'s tannery, was he able to detect an effluvium other "than that which would be produced at a tannery conducted in a proper "and cleanly manner."

* * * * * *

State of the Tannery at the End of 1902, and Beginning of 1903. At the time of my first visit on December 22nd (*i.e.*, nearly four months after Dr. Vacher's last visit) I found that the drains from the tannery had been connected with the Waste Lane sewer, otherwise things were in the same state as on the occasion of Dr. Vacher's September visit. The **plan of the tannery,** in which I have indicated the various effluents, will show the relations of the parts referred to. (*See Plan, p.* 257). A general statement would add nothing to what has already been so clearly and concisely stated by Dr. Vacher. I will therefore add only a few remarks regarding special points not mentioned in the above reports.

I went down the manhole at the junction of the **tannery drain,** and of the **Waste Lane Sewer,** which at the time of my visit had been connected, and smelt closely the tannery effluent, which had the usual smell of tan mixed with the slightly ammoniacal smell noticeable over the settling tanks. I placed my face

at the opening of the drain from the tannery, noticed no exceptional smell, and suffered no bad effects from the experiment. I went down the sewer, lower down, at its junction with the new sewer coming down from Cuerden Hall, and did not notice any exceptional smell. On the occasion of my second visit I smelt again the tannery effluent as it entered the sewer and noticed no alteration in its odour.

The tannery is provided with **earth closets.** I was informed that none of the solid excreta of the men employed at the factory pass into the sewer, they are disposed of on the land about. I may mention here that the only houses with water closets are Cuerden Hall and Millington House, and in both these cases the solid excreta are not at the present time allowed to pass directly into the sewer.

As **soak pits** when improperly managed may be a source of nuisance I enquired specially on two different occasions as to the time the hides were allowed to soak. I was informed that the water was changed, and the pits cleaned, every other day, also that the skins were never allowed to soak for more than three days. The state of the water in the two pits I examined on two different occasions, and the examination in the laboratory of 4 samples *(see Table I., p. 262)* taken at various times confirmed entirely that statement. The smell of the soak pits, and that of the **salted hides** which were immersed in them, resembled closely that of salted fish in a good state.

The soaked hides are left in the **lime pits** for 15 to 21 days. The smell of the lime pits cannot be well described, such a smell is always produced when animal matter is treated with lime, and is neither distinctly objectionable nor detrimental to health. The smell of pits in which skins had remained longest was not so strong as that of the others. Lime therefore acted as it usually does, *i.e.*, as an antiputrid agent.

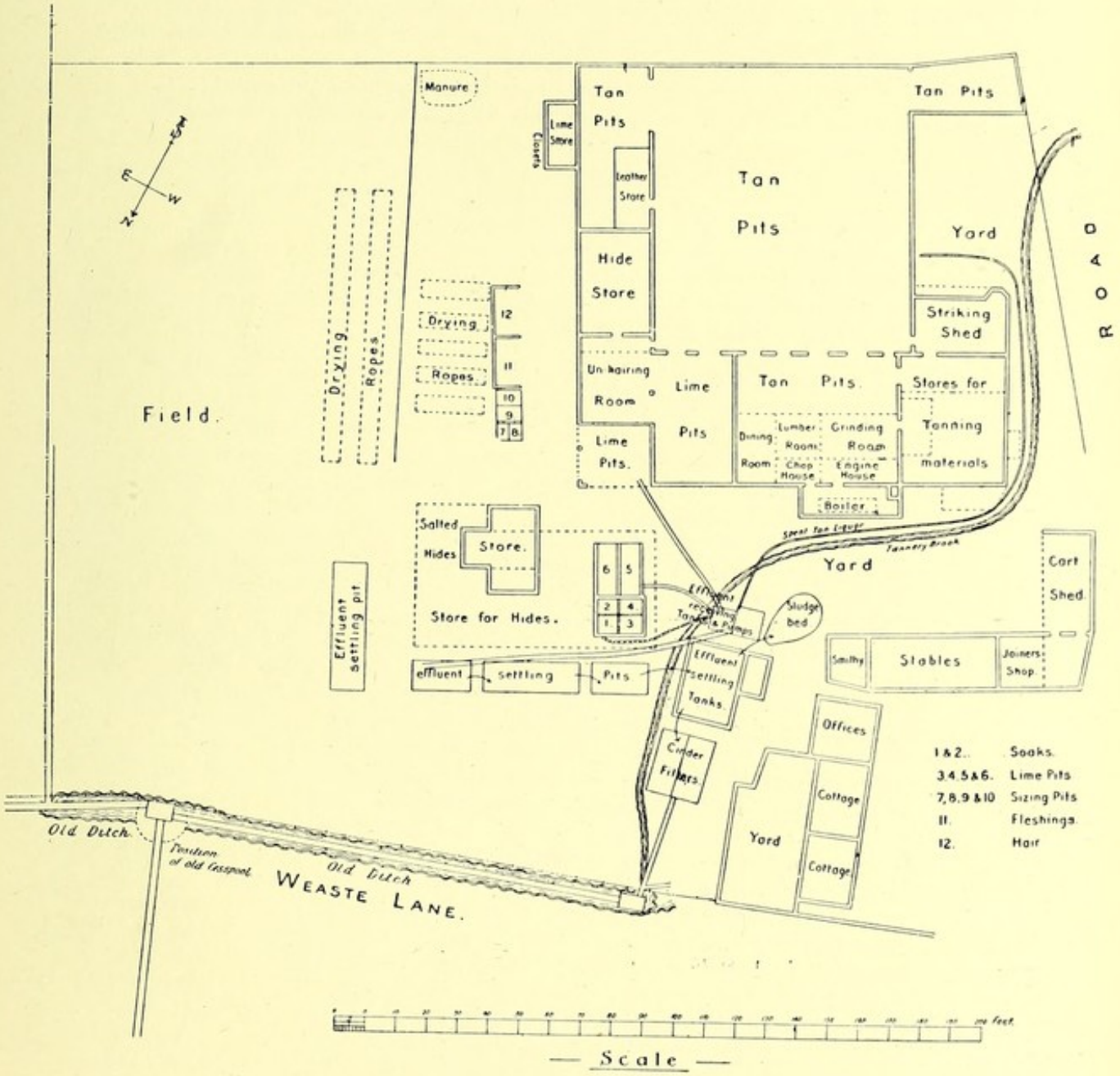
Before the skins are treated the **tails and other useless parts of the hides** are cut off, and left in barrels in the open until removed, the smell of these tails is the same as that of the hides. In a few cases the **horns, and part of the skull** are bought with the hides, and have also to be removed. The second time I was at the tannery I saw a few horns in a heap, which emitted a putrid smell. This was the only time I found in the yard anything having a putrid smell, and I was assured that the thing was quite exceptional. The amount of putrid matter was, however, so small that the smell was not noticeable at the distance of a few yards, and unless large accumulations of horns were allowed to take place no serious nuisance could arise from this cause.

I smelt closely on three occasions the **sizings** hanging on ropes in the yard, and could not detect any objectionable smell.

The **fleshings** were also remarkably free from any putrefactive smell. At the time of my second visit, there was a larger accumulation than usual, about 18 inches deep. I had the fleshings turned over, and smelt the deepest layer without detecting any putrid smell.

The **hair** was treated in the same way, both fleshings and hair had a slightly ammoniacal smell, difficult to recognise and due to the action of lime.

The places in which these operations were conducted had the same smell as the lime pits. These operations, which may be strongly objectionable when



PLAN OF THE TANNERY.

putrefactive fermentation is allowed to take place during the previous stages, were at the time of my three visits conducted without producing any nuisance. The methods adopted at the tannery did not seem to me to allow of the possibility of such a nuisance being produced.

The **tan pits** are entirely covered, and it is in this part of the tannery that I noticed the strongest smell. But the smell was that of tan materials, and is quite inseparable from the tanning process. There was nothing nauseous or actually very unpleasant in it. I inspected a number of tan pits separately, and more specially those in which the strong liquors were prepared from valonia and gambier, and also the pits where yeast was added to the tan liquor.

On my first visit I found that the use of **yeast** had been discontinued for six months. Mr. L. informed me that the discontinuance was not due to any fear on his part that a nuisance was produced by the yeast. He had some doubts as to the advantages of using it. Mr. Scudder thought that, this being the case, it would be well to discontinue it for a time. I closely questioned the men employed about the yeast pit as to whether they preferred the smell of the liquor when the yeast was used, or when it was not. They did not seem to have noticed much difference. About one part of strong "**carbolic acid**" is added to every 10 or 11 parts of yeast thrown into the tan liquor.

It seemed difficult to believe that brewer's yeast added to tan liquor could be a source of danger to health, but in order to satisfy myself that the men had really experienced no inconvenience from the use of yeast, and that some unexpected reaction, giving rise to some bad smelling gas, did not take place, I asked Mr. L. to resume the use of yeast exactly as he did before June, 1902. This experiment I was sure would be without danger to anybody.

I visited the tannery again one month after this, and found practically no difference in the smell of the tan liquor or of the tannery as a whole. On March 9th, *i.e.*, more than two months after the yeast had been resumed, I found absolutely no alteration in the smell of the tannery.

The **effluents** from the tan pits, the lime pits, and the soak pits are led to a well in the yard, and the mixing of these fluids is followed by a considerable amount of precipitation. The mixed or **crude effluent** is pumped up, and led by an open conduit to a number of settling pits and tanks which are in the open. The smell of the crude effluent as it runs from the well to the **settling tanks** is not strong, it is difficult to describe it except as a mixture of the smells of tan, lime pits, and soaks, which have been already described, the result being a slightly ammoniacal, mawkish smell. The smell is not very strong, but is unpleasant, there is, however, nothing putrid or actually nauseous about it; I exposed myself repeatedly to its action without suffering from it at any time, and I did not notice that any of the visitors who were with me suffered in any respect, although I watched them closely the whole time.

The crude effluent, after settling, is passed through two **cinder filters,** from which it escapes comparatively free from suspended matter. The smell of the treated effluent is more distinctly ammoniacal than that of the crude effluent; it is certainly not more objectionable than the smell of ordinary tannery effluent.

The **sludge** consists of lime and organic matter more or less coloured by tan. This material is allowed to drain for some time in order to make its removal

easier. It is then carted away to adjoining fields. There are one earth pit and one sludge bed in the yard where the drying process is carried out. I examined the lime at various depths in the sludge beds, and found it free from any strong or objectionable smell. I also examined a large accumulation of sludge in a field across Cliff Lane, and could not detect any effluvium arising from it.

The **spent tan** is heaped up for a time at the side of the tannery, and is then carted away to a field at a distance of 350 to 400 yards from the tannery on the other side of the road. This tan is used to fill up a disused pit, several such pits have been filled up in the same way in past years. The smell of the spent tan tip which I examined was that of tan; a deep hole was dug in it to ascertain whether putrid smells were generated in the deeper parts of this accumulation. At a depth of four to five feet, a stratum of dark fluid which had drained from the tan was reached. Some gas bubbles escaped from that fluid but the gas had no smell and was apparently chiefly composed of carbonic acid. The dark fluid was found to drain slowly into the ditch or small brook running in the direction of the tannery. The water and the banks of that brook had a dark-brown colour owing to this.

The smell of the water of that **brook** resembled that of tan liquor, mixed with that of the decomposing vegetable matter which had fallen from the banks, and of drainage from **manured fields.** It is probable that at times the smell of this brook is unpleasant owing to the accumulation of decomposing matter. If this be the case the banks and bed might with advantage be made more even. I did not, however, at the time of the two visits I paid to this brook, notice any smell that might fairly be called a nuisance. The turbid water from this brook had a dark-brown colour and an acid reaction.

None of the **tanning material** used at Mr. L.'s tannery differed from those in common use in various tanneries. In the following list those which I found employed at this tannery are marked by the sign.*

* Oak Bark. The bark of the British oak (*Quercus robur*, and other varieties).
* Oak Wood Extract.
* Valonia. } The cups of the acorns of an evergreen oak (*Quercus*
* Valonia Extract. } *aegilops*).
* Myrobolams. Myrobalan, the fruit of trees of the genus *Terminalia*.
Myrobalan Extract.
Mimosa Bark. The bark of various trees of the group of *Mimosae*.
* Mimosa Extract.
Hemlock Bark. The bark of the Hemlock Spruce (*Abies canadensis*).
* Hemlock Extract.
* Sumach. The powdered bark, etc., of the Sumach (*Rhus coriaria*).
Sumach Extract.
* Block Gambier. Obtained from the *Uncaria gambier*.

All these substances are strong astringents, with well known properties, and incapable of producing noxious effluvia. In addition to these tannin-containing substances *brewer's yeast* and crude *carbolic acid* are also used in the treatment of the skins. I need not make any remark about carbolic acid, the use of which as a disinfectant and antiseptic are generally well known.

With regard to **yeast**, however, I found it necessary to make some investigations. I am not acquainted with any fact or scientific research which could justify the opinion that "yeast when mixed with the waste liquors from soak pits, tan pits, etc., *produces an effluvium nuisance*, having all the characters of the smell complained of, when let off into any tank or drain," and that yeast acted under those circumstances as a " septic ferment." This theory, which I heard and read of for the first time in Dr. Penny's report upon this tannery, seemed all the more strange to me because yeasts must always be present in tan liquors, owing to the general distribution of yeasts in nature, and specially their almost constant presence on the surface of barks, and fruits of various kinds. In view, however, of the positive statement made by Dr. Penny, I did not think it right to assume that he was in error without investigating the matter fully.

1. I have already stated that I was quite unable to detect any effluvium or irritating vapour in connection with the **yeast tan-pit**, nor with the treated or untreated effluent into which some of the spent tan liquor found its way. I have incubated samples of all the fluids containing yeasts, at various temperatures up to high summer temperature, for over six weeks, and have not observed any generation of bad smelling gases. Carbonic acid appeared to be the only gas produced in any quantity.

2. I have also been unable to detect any alteration in the smell of these fluids after the use of yeast had been resumed at my own request.

3. To complete the evidence necessary to ascertain whether yeast did not introduce some unsuspected alterations in the tanning process, I tested by various methods a number of samples of tan liquors, of various effluents, of tanning material, etc. I also obtained from Mr. D.'s tannery where brewer's yeast had never been used, samples of tan liquors for comparison with the tan liquors obtained from Mr. L.'s tannery. The results of my examinations are summed up in the following table. *(See Table I., p. 262.)*

From this table it will be seen that yeasts of various types were present in all the tanning material and liquors examined. Many of the yeast cells were budding. As very little attention seems to have been paid to the presence of these various kinds of yeast in tan liquors, I asked Dr. Carver, senior assistant in my Laboratory, to isolate these yeasts and study their characters. The results of his observations are embodied in his report upon the subject.

None of these yeasts grew well in animal media or produced a putrid fermentation giving rise to bad smelling gases.

With regard to the nature of the gases produced I have not investigated it fully; but Mr. Scudder having made a number of analyses, I have summarised the results which he has kindly communicated to me in the following table. *(See Table II., p. 264.)*

From these results it appears that yeasts of various kinds are generally present in tan liquors and tanning material, and that these materials are all capable of gaseous fermentation producing inodorous, harmless gases, whether brewer's yeast is added to them or not.

From Messrs. L.'s books which I was allowed to inspect, it appears that yeast began to be used in 1895.

During the year 1899-1900, *i.e.*, at the time when the diphtheria epidemic

occurred, the amount of yeast used was less than half of what had been used in the two previous years.

I was informed that no important alterations had been made in the premises since 1895, and no new processes used, with the exception of what has already been mentioned in connection with yeast.

It is generally thought that the **health of men employed** in carrying out a manufacturing process is no absolute criterion of the danger associated with their occupation and that they gradually acquire a certain amount of tolerance. It appears to me, however, that if it were true that the introduction of yeast in 1895 had caused *a new element of danger*, the men employed at the tannery would not have been more protected than other individuals, and if, *for the sake of argument*, it was supposed that the germs of diphtheria could be generated by the addition of yeast to tan liquor or tannery effluents, the men, even if protected, would have carried about with them the germs of that disease, and infected the young members of their families. The families living on the premises, or near the premises would, on such a supposition, have been specially liable to infection.

In order to test whether there was any such evidence of deleterious action, I asked Messrs. L. to supply me with a complete list of their men, of the children of those that were married, and of their addresses. I also asked them to obtain a complete list of the children that had died in these families, their age at death and the supposed cause of death.

From the documents so supplied to me I have obtained the following information.

At the present time there are 54 workmen employed at the tannery. These men have worked in the tannery for various lengths of time.

1 year or less	... ...	12 men	2 of them married.
over 1 year to 2 years	... ...	5 „	4 „
„ 2 years to 3 „	... ...	5 „	1 „
„ 3 „ 5 „	... ...	4 „	2 „
„ 5 „ 10 „	... ...	12 „	6 „
„ 10 „ 15 „	... ...	7 „	7 „
„ 15 „ 20 „	... ...	1 „	1 „
„ 20 „ 30 „	... ...	5 „	5 „
„ 36 years ...	... ...	1 „	1 „
„ 45 „ ...	... ...	1 „	1 „
„ 57 „ ...	... ...	1 „	1 „
		54 men	31 married.

None of the twelve men I questioned could remember having suffered from sore throat, sickness, diarrhœa, or loss of appetite on first entering the tannery.

Twenty-six of the married men have children. During the last twelve years the number of persons connected with the tannery has been over 54 men, 31 women, and 118 children.

Deaths Occurring among the Children of Workmen. During these twelve years twelve children have died. Some of the births and some of the deaths occurred before the men had entered the tannery, but it is

TABLE I.—Examination of tan liquors and various effluents

Ref. No.	Material Examined.	Tannery.	Smell.	Reaction.
375	Brewer's yeast	L. ...	Pleasant, of fermenting wort	Acid ... .
376	Recent tan liquor and yeast ...	„ ...	Pure tan, no putrid smell	Strongly acid ..
377	Tan liquor (7 days old) and yeast	„ ...	Id., slight smell, resembling that of phenol	„ ..
406	Tan liquor and yeast	„ ...	„ „ „ ..	„ .
379	Tan liquor, no yeast added ...	„ ...	Pure tan smell...	„ ...
405	Recent tan liquor, no yeast added	„ ...	„ „	„ ..
409	Recent tan liquor, no yeast added	D. ...	„ „	„ .
410	Old tan liquor, no yeast added...	„ ...	„ „	„ ...
380	Liquor from soak pit, 1 day old	L. ...	Slight mawkish smell ...	Strongly alkaline
384	„ „ 2 days old	„ ...	„ „	„
385	„ „ 3 days old	„ ...	„ „	„
381	Liquor from lime pit...	„ ...	Slightly ammoniacal ...	„
379 380 381	Tan liquor, soaks and lime water mixed in equal parts...	„ ...	Tan smell modified by others	Slightly acid ...
382	Crude effluent	„ ...	Faint mawkish smell, slightly ammoniacal ...	Strongly alkaline
407	Crude effluent	„ ...	„ „ „ ...	„
383	Settled effluent	„ ...	Very slight, as above ...	„
408	Settled effluent	„ ...	„ „ „	„

Tanning

396	Valonia extract	—	—	—
397	Myrobalan extract	—	—	—
398	Mimosa extract	—	—	—
399	Sumach extract	—	—	—
400	Myrobalan, incubated in wort...	—	—	—
401	Sumach „ „ ...	—	—	—
403	Valonia „ „ ...	—	—	—
404	Gambier „ „ ...	—	—	—

obtained at Messrs. L.'s and Mr. D.'s Tanneries.

Sp. Gr.	Yeast found by direct observation, size and shape of cells.	Production or Absorption of Gas on Incubation.	Smell of Gas.
—	Typical yeast cells	Abundant production (after addition to wort)	None
1110–1112	Abundant yeast cells of 3 different shapes	Abundant production...	,,
1108–1110	,, ,, ,,	Moderate production	,,
1120	,, ,, ,,	—	—
1108–1110	,, ,, 2 ,,	Very abundant production	None
1130	Fairly abundant yeast cells of 3 shapes	—	—
1090	Yeast cells abundant of 3 shapes and sizes	Scanty production	None
1110–1112	Yeast cells of 1 shape, various sizes	Abundant production...	,,
1010	No yeast found	None	—
1034	Very few yeast cells...	,, ...	—
1034	,, ,, ...	Gas absorbed	—
1012	,, ,, ...	,, ...	—
1049	Various yeasts fairly abundant ...	Scanty production of gas, followed by absorption	—
1023	Yeast cells few...	Gas absorbed	—
1018	Yeast cells, few and small	,, --	—
1011	Yeast cells, few, small, 2 shapes ...	,, --	—
1007	(?) ...	,, --	—

Materials.

—	Yeast cells scanty, 2 shapes and sizes	—	—
—	Yeast cells abundant, 3 shapes ...	—	—
—	,, ,, ...	—	—
—	Yeast cells scanty, 2 shapes	—	—
—	,, ,, ...	Gas produced	None
—	,, ,, ...	,, ...	,,
—	,, ,, ...	,, ...	,,
—	,, ,, ...	,, ...	,,

TABLE II.—Production of Gas by Tan Liquors and Effluents.
Analyses made by Mr. Scudder.

Material Tested.	Quantities.		Duration of Experiment.	Temperature.		Percentage Composition of Air at End of Experiment.			H.	H_2S.	CH_4	Smell.
	Fluid C.C.	Air C.C.		F.	C.	N.	O.	CO_2				
1. Tan liquor without yeast or carbolic acid...	200	130	11 days	60°	15·5°	64·50	10·90	24·60	0	0	0	0
2. Tan liquor + 2 grammes yeast	200	140	11 „	„	„	66·0	11·50	22·50	0	0	0	0
3. Liquor from soakpit + 2 grammes yeast...	200	150	11 „	„	„	80·40	19·20	0·40	0	0	0	0
4. Limepit liquor + 2 grammes yeast... ...	200	170	11 „	„	„	81·60	18·40	0·0	0	0	0	0
5. Crude effluent + 2 grammes yeast	200	120	11 „	„	„	85·60	14·40	0·0				
6. Settled effluent + 2 grammes yeast... ...	200	160	11 „	„	„	80·80	17·20	2·00				
7. Tan liquor without yeast or carbolic acid...	250	270	7 days	75°	24°	76·06	16·90	7·04	0	0	0	0
8. Tan liquor + 2·5 grammes yeast	250	200	7 „	„	„	72·20	16·40	11·40	0	0	0	0

difficult to allow accurately for these (three of the deaths at least occurred before the father had begun working at the tannery). The following table gives the causes of death among the children and approximately the distance of their dwellings from the tannery:—

A.—Families living on the spot or across the road.

No. of families.	No. of children.	Deaths since 1890.	Age at death.	Reported cause of death.	Year of death.
4	16	1	3½ years	Hypertrophy of tonsils, adenoid dis. of naso-pharynx. Ulceration of larynx. Operation (for adenoids) 1 month before death.— (From Death Certificate).	Nov. 1899.

B.—Families living within ½ mile (mostly ¼ mile).

6	29	0	—		

C.—Families living between ½ and 1 mile (mostly ½ mile).

6	32	5 in 2 families (*a* and *b*)	*a* (All 4 cases in a family of 12)	A few days	Weakness.	1891.
				12 months	Measles.	1898.
				15 „	Gastro-enteritis	1900.
				13 „	Weakness	1901.
			b	8 „	Convulsions	1891.

D.—Families living over 1 mile (mostly 2 miles (Lymm)).

10	41	6 in 3 families (*a*, *b*, & *c*)	*a*	4 months	Convulsions	1891.
				8 „	After vaccination (cause?)	1899.
			b	18 months	Weakness	1890.
				7 „	„	1895.
			c	11 months	Convulsions	1894.
				7 „	„	1896.

From this summary it appears that all the deaths occurred in six families out of twenty-six, and that the deaths were fairly equally divided among the twelve years.

1 in 1890	... ...	1 in 1896	
3 „ 1891	... ...	1 „ 1898	
1 „ 1894	... ...	2 „ 1899	
1 „ 1895	... ...	1 „ 1900	
	... ...	1 „ 1901	
6		6	

A further analysis shows that seven deaths only occurred during twelve years among seventy-two children who were young enough to be easily affected by infectious germs, a mortality which is much below the average mortality among children. These deaths all occurred in three families only. There is therefore no evidence of any deleterious influence existing at the tannery since 1895, nor at any time since 1890.

Only three adults have died since 1895.

(*a*) A man, aged 82, who died in 1901, after working 50 years at the tannery.

(*b*) A wife, aged 80, who died from the effects of a severe burn.

(*c*) A wife, of consumption.

(I understand that there has been no report of death among the men who have left the tannery, *i.e.*, within a few months of their departure).

Bacteriological Examination of the Throat of Workmen. To complete the above evidence I examined bacteriologically the throat of seven of the workmen in case some evidence of possible infection had been over-looked. I selected these seven men as they were leaving the tannery, and after cultivation of the secretions obtained from their throats found no evidence whatever of diphtheria bacilli nor of any other unusual bacteria.

Watching the Tannery at Night. I have received several letters stating positively that effluvia arose from the tannery mostly in the middle of the night, and on my discussing with Dr. Vacher the advisability of obtaining evidence on this point, he informed me that he had already done so, and has allowed me to state that during the month of December, 1902, he had the tannery watched night and day by an absolutely reliable person upon twenty different occasions. The watcher reported that upon each occasion he had failed to detect any disagreeable smell beyond the ordinary tannery smell.

Summary of Report upon the Cliff Lane Tannery.

1. There is no evidence of any effluvium being generated at the tannery, nor by the effluent from the tannery.

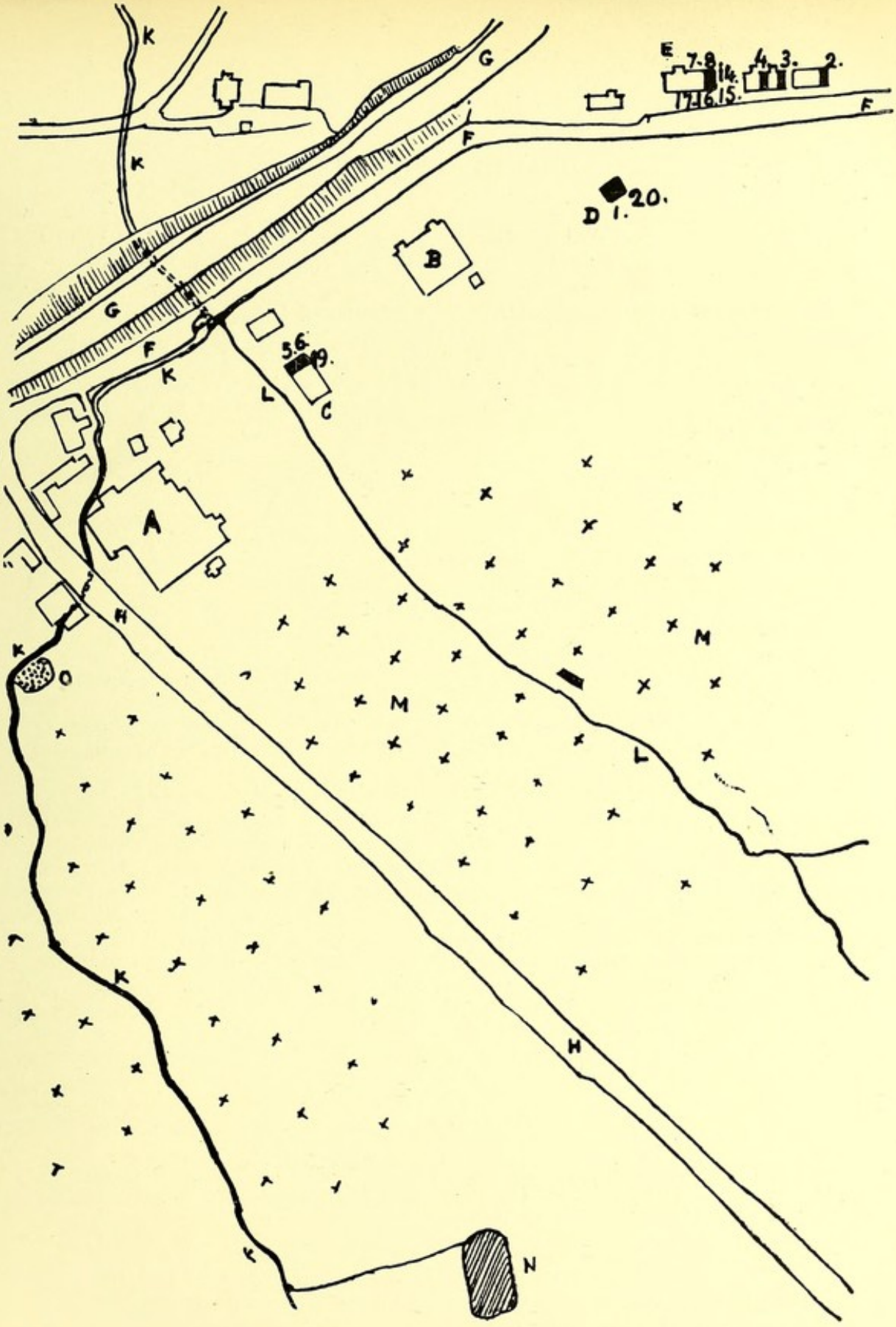
2. There is no evidence of any unusual process being employed at the tannery, with the exception of the use of yeast, which began in 1895. As yeasts are naturally present in all tan liquors and materials, the addition of yeast does not seem to be material, at any rate, it does not produce any nuisance.

3. There is no evidence of any excessive amount of illness among Messrs. L.'s employés, on the contrary, the amount of illness and death among these people during the last 12 years has been very small. I find no evidence that the introduction of yeast in 1895 has had any effect upon their health.

4. There is nothing in the tanning processes used at the tannery that could give rise to an outbreak of diphheria.

5. The tannery is well conducted and clean. Considerable trouble is taken to treat the effluent so as to diminish as far as possible the smell which is necessarily connected with tanning.

6. I am convinced that there can be no connection whatever between the tannery and the outbreak of diphtheria which has afflicted Thelwall.



Spot-Map shewing part of Thelwall adjoining the Tannery.

The houses are outlined only. The houses in which Diphtheria occurred are in black. The numbers adjacent to the Diphtheria houses indicate the order in which the cases occurred. They correspond to the numbers given to the notified cases in the text, p. 22.

A, Cliff Lane Tannery; B, Cuerden Hall; C, the Coach House; D, the Lodge; E, Opposite the Temperance Hall in Anderton Place; F, Waste Lane; G, Canal; H, Knutsford Road or (Cliff Lane); K, Tannery brook or Cliff Lane Brook; L, Boundary Brook; M, Manured fields, also indicated by XX; N, Spent tan; O, Lime and sludge;

III. NATURE OF THE OUTBREAK OF DISEASE AT THELWALL in 1899—1900—1901 and 1902.

Extent of the Outbreak. The extent and severity of this outbreak can be gathered from the following tabulated statements prepared after consultation of official documents, and visits to the various families affected:—

A. *Cases of Diphtheria or "Diphtheritic Throat" which had occurred within a radius of 1 mile around the Tannery, and had been certified to the Medical Officer of Health.**

	Date.	Name of deceased.	Address.	Certification.
1.	Dec. 29th, 1899	Emma Scarle (Taken ill, December 18th)	The Lodge, Cuerden Hall	Diphtheria notified as Diphtheritic sore throat.
2.	Dec. 29th, 1899	Quennell (Taken ill, December 25th)	Anderton Place	,,
3.	Dec. 30th, 1899	Cissy Hewitt,	Anderton Place	,,
4.	,,	Edith ,,	,,	,,
5.	Jan. 5th, 1900	Lucy Leah	The Stables, Cuerden Hall	Diphtheria
6.	,,	G. Leah	,, ,,	,,
7.	,,	(-) Vickers	Caretaker, Temperance Hall	,,
8.	,,	(-) ,,	,, ,,	,,
[9.	Feb. 11th, 1900	(-) Rigby	Near Ship Canal, Grappenhall	,,]
[10.	,,	,,	,, ,,	,,]
[11.	Feb. 12th, 1900	,,	,, ,,	,,]
[13.	Nov. 3rd, 1900	(-) Ellison	Near Schools, Thelwall	,,]
14.	Feb. 24th, 1901	R. Vickers (Taken ill, February 16th)	Caretaker, Temperance Hall, Thelwall	,,
15.	Mar. 2nd, 1901	B. Vickers (Taken ill, March 1st)	Caretaker, Temperance Hall, Thelwall	,,
16.	Mar. 6th, 1901	O. Vickers (Taken ill, March 9th)	Caretaker, Temperance Hall, Thelwall	,,
17.	Mar. 8th, 1901	Mrs. Vickers	Caretaker, Temperance Hall, Thelwall	,,
[18.	April, 1901	(-) Allan	At Ferry's Cottage	,,]
19.	August, 1902	May Leah (Taken ill, August 1st)	The Stables, Cuerden Hall	,,
20.	,,	Mary Scarle	The Lodge, Cuerden Hall	,,

*The numbers on left side refer to the spot-map, part of which is reproduced in this report. Cases within brackets were unconnected with the outbreak near the Tannery.

B. *Copies of the Entries in the Health Register of the persons who had died in the neighbourhood of the Tannery since the beginning of the outbreak.*

Date.	Name of deceased.	Age.	Address.	Certification.
Dec. 25th, 1899	F. Hewitt	$\frac{11}{12}$	Anderton Place, Thelwall	Bronchitis, Croup.
[Dec. 27th,	F. J. Tighe (Mrs. Tighe is daughter to Mr. Scarle.) (Taken ill, Dec. 17th, 1899)	4	Church Street, Altrincham (Child had been staying from Dec. 14th to Dec. 19 with Mrs. Scarle.)	Laryngitis.] Case added here for reference.
Jan. 6th	Lucy Leah (Taken ill, about Jan. 4th) ?	4	The Stables, Cuerden Hall	Diphtheria.
Jan. 10th, 1900	G. Leah (Taken ill, about Jan. 5th) ?	7	,, ,,	,,
Feb. 27th, 1901	R. J. Vickers (Taken ill, Feb. 16th)	5	Waste Lane	,,
Mar. 9th, 1901	Bertie Vickers (Taken ill, March 4th)	$1\frac{1}{2}$	,,	Diphtheria, Meningitis.
Aug. 15th, 1902	May Leah (Taken ill, August 1st)	2	The Stables, Cuerden Hall	Diphtheria.

General State of Health of the Locality. Thelwall is composed of a number of dwellings scattered over a large area. There are, however, two distinct groups of houses, a larger one on the banks of the Ship Canal, and a smaller one about the tannery.

If diphtheria be excluded, Thelwall and Grappenhall have been during the last eight years remarkably free from notifiable infectious diseases. The total number of cases, including diphtheria, occurring in Grappenhall (population 987) in the eight years since 1895 being 16, of which 3 were due to diphtheria and 13 to other diseases.

The total number of cases of notifiable infectious diseases occurring in Thelwall (population 481) was during the same time 22, of which 17 were attributable to diphtheria, and 5 to other diseases. If, therefore, diphtheria be excluded Thelwall appears to have been proportionately freer from infectious diseases than Grappenhall (I will show later that Grappenhall itself was much more healthy than many other localities in the district). All the cases of diphtheria occurred in Thelwall since the end of 1899. *It is, therefore, entirely to diphtheria that the excess of disease which has occurred during that period is due.*

Incidence of Infectious Diseases in Thelwall and other localities compared. A severe outbreak of diphtheria occurring among a small community may be attributed to several causes, the most important and probable of which is the *importation of diphtheria from outside*, either by a resident or by a visitor. In addition to this, a locality may become specially affected owing to *predisposing conditions* rendering the inhabitants more liable to the disease than average individuals. In order to find out if there had been serious chances of importation, and, if the inhabitants of Thelwall were specially

TABLE III.

PLACE	Population		1895		1896.		1897.		1898.		1899.		1900.		1901		1902		Eight years, 1895—1902.		Percentage	
	1891	1901	Total	Diph.	Total	Diph.	Total	Diph.	Total.	Diph.	Total.	Diph.	Total	Diph.	Total	Diph.	Total	Diph.	Total.	Diph.	Total 8 Years	Diph 8 Years
Aston	260	249	1	—	8	1	—	—	—	—	1	1	—	—	—	—	—	—	10	2	4·0	8
Aston Grange	35	40	—	—	—	—	1	—	—	—	—	—	—	—	—	—	—	—	1	—	2·5	—
Clifton	215	213	3	—	1	1	5	**5**	—	—	—	—	1	—	—	—	3	—	13	6	6·1	2·8
Halton	1,555	1,238	21	—	13	—	5	—	15	**14**	9	**9**	9	1	15	—	89	—	170	24	12·6	1·9
Norton	430	294	7	—	—	—	1	—	—	—	—	—	—	—	—	—	3	—	11	—	13·7	—
Stockham	47	38	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
Sutton	471	405	2	—	20	**12**	3	3	3	1	1	—	5	2	1	1	2	2	37	21	9·1	5·1
Weston	2,222	2,075	43	3	2	—	11	**7**	14	**12**	4	—	2	—	4	—	8	1	88	23	4·2	1·1
Alvanley	328	319	3	—	8	**8**	1	1	—	—	—	—	4	—	—	—	1	1	17	10	5·3	3·1
Frodsham	3,333	2,728	36	4	42	6	50	**11**	16	7	37	**17**	10	3	33	**30**	21	**15**	245	93	8·9	3·4
Frodsham Lordship	1,513	1,403	19	4	23	**8**	18	1	10	3	5	1	1	—	7	4	2	—	85	21	6·0	1·4
Helsby	1,154	1,372	3	—	7	2	10	2	13	**5**	1	—	2	1	3	1	13	**5**	52	16	3·3	1·0
Kingsley	1,111	1,066	7	—	2	—	4	3	2	1	3	3	—	—	6	—	5	1	29	8	2·7	7
Kingswood	233	248	—	—	1	—	4	—	—	—	—	—	1	—	—	—	2	2	8	2	3·2	8
Manley	296	303	2	—	2	—	—	—	—	—	1	—	—	—	1	—	1	—	7	—	2·2	—
Newton-by-Frodsham	105	130	5	**5**	—	—	—	—	—	—	—	—	1	1	1	1	2	2	9	9	6·9	6·9
Norley	689	656	5	—	2	—	2	—	5	—	3	1	4	—	8	—	25	—	54	1	8·2	·1
Acton Grange	453	144	3	—	8	—	1	—	1	1	1	—	—	—	1	—	3	—	18	1	12·5	6
Antrobus	425	373	—	—	1	1	—	—	—	—	1	—	—	—	—	—	—	—	2	1	5	2
*Appleton	2,759	744	19	—	20	2	10	3	7	3	56	2	2	—	1	—	2	—	117	10	15·7	1·3
Bartington	83	75	—	—	—	—	—	—	—	—	—	—	1	—	—	—	—	—	1	—	1·4	—
Budworth	510	476	—	—	3	—	2	2	2	—	2	—	1	—	—	—	1	—	11	2	2·3	4
Crowley	190	182	—	—	—	—	3	1	—	—	2	—	—	—	—	—	—	—	5	1	2·7	·5
Daresbury	190	153	1	—	5	—	1	—	—	—	—	—	1	—	—	—	1	—	9	—	5·8	—
Dutton	416	426	—	—	6	1	3	—	1	—	—	—	—	—	1	—	—	—	11	1	2·5	·2
Hatton	327	319	7	—	9	—	1	—	1	—	—	—	4	—	1	—	2	—	25	—	7·8	—
Keckwick	82	65	3	—	9	—	—	—	1	—	—	—	2	—	—	—	—	—	15	—	23·0	—
Moore	526	406	10	—	10	—	8	—	—	—	2	—	1	—	1	1	2	—	34	1	8·3	·2
Newton-by-Daresbury	185	169	—	—	4	—	3	—	—	—	—	—	—	—	—	—	1	—	8	—	4·7	—
Preston-o'-the-Hill	493	454	—	—	17	—	2	—	1	1	2	—	3	—	2	—	4	—	31	1	6·8	·2
Seven Oaks	177	162	1	—	1	—	1	—	—	—	—	—	—	—	—	—	—	—	3	—	1·8	—
Stretton	396	310	—	—	7	—	2	—	—	—	3	1	—	—	3	—	2	—	17	1	5·4	3
Walton Inferior	425	719	2	—	2	1	1	1	3	—	6	—	1	—	1	—	5	2	21	4	2·9	5
Walton Superior	225	215	—	—	—	—	—	—	—	—	—	—	3	—	—	—	1	—	4	—	1·8	—
Whitley Inferior	205	201	1	—	2	—	3	**3**	—	—	—	—	—	—	1	—	1	—	8	3	3·9	1·4
Whitley Superior	303	323	—	—	4	—	—	—	—	—	—	—	—	—	—	—	2	—	6	—	1·8	—
Grappenhall	984	987	1	—	2	—	—	—	—	—	1	—	5	3	6	—	1	—	16	3	1·6	·3
Thelwall	770	481	—	—	—	—	2	—	—	—	6	**4**	6	**5**	6	**6**	2	2	22	17	4·5	3·5
Latchford Without	490	—	1	—	—	—	—	—	—	—	1	—	1	1	—	—	2	—	5	1	—	—
*Stockton Heath	1,615	2,543	—	—	—	—	—	—	—	—	—	—	8	—	8	—	17	2	33	2	1·2	·08
Totals for each year	24,665	23,203	266	16	241	43	158	43	95	48	148	39	79	17	111	44	226	35	1264	285	5·4	1·2
Fatal Cases	—	—	7	2	24	11	12	4	4	2	12	3	9	6	15	8	14	7	—	—	—	—

* Before 1901, Stockton Heath was included in Appleton.

The Black Figures indicate special incidence of Diphtheria

liable to infection, I have prepared, on the basis of Dr. Adams' annual reports, a table indicating (1) the total number of cases of illness attributable to notifiable infectious diseases, (2) the number of cases notified as having been affected with diphtheria, (3) the population in 1891 and 1901 in each one of the localities included in the Rural District of Runcorn. *(See Table III.)*

The figures indicating that the incidence rate of diphtheria in any year has been excessive are in black figures.

From this table it will be seen that several places too distant from the tannery to be influenced by it, have, during the last eight years, suffered from Diphtheria as much as, or more than, Thelwall.

These places are Clifton (1897), Halton (1898–99), Sutton 1896), Alvanley (1896), Frodsham (1901), Newton-by-Frodsham (1895), Whitley Inferior (1897). It will be noticed that before and during the epidemic at Thelwall many places in the district had an unusual amount of diphtheria. The table also shows that the total amount of notifiable infectious illness at Thelwall and Grappenhall since 1895 was below the average of the district.

Possible Sources of the Outbreak of Diphtheria at Thelwall. There remains to account for the appearance of diphtheria in the houses at Anderton Place (Waste Lane) and at the stables, Cuerden Hall.

The points which seemed to have attracted attention chiefly were, according to the documents in my possession and the opinions which I have heard expressed :—

1. That these houses were exposed to the smells emanating from the tannery, and which are carried over Cuerden Hall and Anderton Place when the wind is in the south-western direction.

2. That the persons affected were all living in houses connected with a defective sewer.

Both statements are correct, there is no doubt that the smell of the tannery can be recognised at times over the greater part of the area affected. It is also certain that the system of drainage and sewerage was extremely deficient. It is unnecessary for me to deal here with the sewerage question which has been reported upon by the Medical Officer of Health, the Rural District Surveyor, and others. *(See Dr. Vacher's 1st report, p. 253).*

3. There is a third possible factor which I think deserves of very special attention, and that is the importation of diphtheria into one of the families living along Waste Lane, and its spread from individual to individual.

I have already given a full account of my investigation of the tannery, and the entire absence of any evidence pointing to the tannery as a possible source of diphtheria. I feel absolutely certain that there was nothing in the tannery capable of causing diphtheria, or of predisposing to that disease.

Opinions are divided regarding the share which **sewer gas** has in the production of diphtheria. The only evidence deserving of credence which has been obtained on this point tends only to show that sewer gas may predispose to attacks of sore throat. Ordinary sewage does not contain the germs of diphtheria, and could only contain virulent diphtheria bacilli if these had been introduced into it by discharges coming from persons already affected with diphtheria. In my opinion, therefore, the bad state of the sewer, even when

the ditch and cesspool along Waste Lane were still in existence, is not sufficient to explain the appearance of diphtheria in the locality.

Was Diphtheria introduced from outside? No case of diphtheria was notified during the years 1895, 1896, 1897, 1898, and up to December, 1899, as having occurred in Thelwall. That this was not the result of errors of diagnosis is shown by the fact that during the same four years only two cases of infectious disease had been notified, both occurring in 1897.

No evidence of any unusual deleterious influence being at work before 1899 can be found in statistical returns.

This almost complete absence of infectious diseases was suddenly followed by an explosive outbreak, affecting nine children in the course of two weeks. To these local children must be added a grand-daughter of Mr. Scarle who was staying from the 14th to the 19th of December at the Lodge, and who died of "Laryngitis" at Altrincham, on December 27th. These children belonged to the following families: Scarle, 2; Hewitt, 3; Quennell, 1; Vickers, 2; Leah, 2. All these people were in the habit of meeting each other at the Temperance Hall. There were special meetings for children on the Mondays, parents and children met on the Sundays. They also met sometimes during the week.

Of the children affected, the one *who seems to have been the first taken ill, and who was the second to die*, was a little girl, Florence Jessie Tighe, aged 4, granddaughter of Mr. Scarle. Mrs. Tighe had come for a visit to the Lodge, Cuerden Hall, on Thursday, December 14th, 1899. The child is said to have been then apparently in good health. It appears from what Miss Mary Scarle told me, that Florence Tighe was much with Emma Scarle.

On Sunday, December 17th, the child was distinctly out of sorts, on Monday the 18th, she had to be kept in bed, on that day Emma Scarle became ill, and her case was notified as one of diphtheritic sore throat. Mrs. Tighe returned to Altrincham on Tuesday, December 19th, and her child died on the 27th, her death being given in the Death Certificate as due to "laryngitis."

The child Florence was, according to Miss Scarle, well till the Sunday, she then became feverish (the mother states that the skin of the child was very hot and dry). Florence was dressed on Monday morning by Emma Scarle, who carried her downstairs and nursed her, but the child was too ill to sit up and had to be taken back to bed. On Tuesday morning the child looked better and was taken back to Altrincham.*

Judging by the course followed by the illness, which I have purposely given in detail, it is impossible to state positively whether the child was already ill or not before arriving at the Lodge. The period of incubation of diphtheria

* The illness continued to the last to have an insidious course. Monday and Tuesday, the little patient suffered from severe headache; Wednesday, she was better and allowed to get up; Thursday, not so well and kept in bed; Friday, better and got up; Saturday, scarlet fever suspected; Sunday, kept in bed; Monday, child very quiet, not complaining of any pain; Tuesday, same state, the mother "suspected" that the throat was bad; Wednesday, same state, but child obviously very weak and exhausted. Doctor diagnosed croup and ordered usual treatment. The child died soon after the doctor's visit. (December 27).

is very variable, it may last only one or two days or extend over a week. The child is said to have been well for two whole days after reaching the Lodge. On the supposition that the incubation period had been a short one, the disease might have been contracted at Altrincham, during the journey, or at Thelwall. It is, however, remarkable that no child was known to be suffering from diphtheria at Thelwall before Florence Tighe came, and that four days after her arrival Emma Scarle who had nursed her became affected with diphtheria. F. Hewitt was 11 months of age, big and fat, and still breast-fed. It had never been away from home. It was taken to a tea-party at the Temperance Hall, on Thursday, the 21st of December, and died on the 25th. The child had been ill from bronchitis before going to the party, but was not seriously ill. On the 24th it became suddenly much worse. Mrs. Hewitt was on friendly terms with the Scarle's and the Quennells. The Quennells appear to be on friendly relations with the Scarles, and a son of the Quennells was notified as suffering from diphtheria on December 29th. I am informed that he was taken ill on December 25th.

On the supposition that Florence Tighe brought diphtheria to Thelwall, the occurrence of the other cases is quite easy to explain.

On the other hand, it may be argued that Emma Scarle or the Hewitt baby were previously ill, and that Florence Tighe was infected by one of these. All the evidence I have obtained points in the opposite direction.

Whether, however, diphtheria was brought or not to Thelwall by Florence Tighe, all the facts indicate its introduction from outside.

After the first sharp outbreak which affected five families, the disease seems to have remained confined to three families, *e.g.*, the Scarles, the Leahs, and the Vickers. Mr. Vickers is caretaker of the Temperance Hall, where many meetings were taking place at the time of the beginning of the outbreak (week before Christmas) and his family was severely affected.

Regarding the **continuance of the disease from year to year,** the probable explanation is that the patients' rooms and their contents had become infected. This explanation is in perfect accordance with well-known facts. Active bacilli may persist for more than two months in the nose of patients who have suffered from diphtheria. It is also well known that diphtheria bacilli may be present in the throat of persons who have been exposed to infection, without giving rise to a typical attack of the disease and sometimes even without giving rise to any disease at all. In addition to all this it is also recorded that diphtheritic products, when they have been allowed to dry, may retain their virulence for months and even more than a year (Heubner, Proust, *Traité d'Hygiène*, Paris, 1902, i., 159), and render rooms, in which patients affected with diphtheria have lived, infections for many months.

I was specially struck by the fact that Leah's children all slept in two small rooms communicating through a small landing, and that the three children lost by him had all died in the same room.

It will be noticed that the same continuance of diphtheria for a certain number of years in one place is not special to Thelwall. It is sufficient to glance at the table of notified cases of diphtheria in the various localities comprised in the Runcorn Rural District to see that when once diphtheria has

been introduced into a small locality it has a tendency to persist for several years in that place.

I conclude from the above evidence that the Thelwall diphtheria outbreak was in all probability the result of infection brought to the locality by a diseased person, probably Florence Tighe.

In this abstract I have not thought it necessary to give a detailed list of all the documents consulted, nor any detailed account of the procedure adopted in conducting the investigation. This information will be found in the original report to the Council of the Rural District of Runcorn. It is, however, my pleasant duty to acknowledge the assistance I have invariably received from Dr. J. Adams during my enquiries.

IV. CONCLUSIONS.

1st. I can find no evidence of Messrs. L. & Sons' tannery having been at any time since 1900 a danger to the health of people living in neighbouring houses.

2nd. I can find no evidence of the use at the tannery of any process capable of generating a dangerous effluvium. The tannery is well conducted and every reasonable care is taken to purify the effluent.

3rd. I have been unable to detect any general nuisance at the time of any of my visits. I noticed, however, that a bad smell might arise at times from horns and bones, if ever allowed to accumulate to any large extent.

4th. From the evidence I have obtained, I think it most probable that a nuisance did actually occur at times, before the open ditch and the cesspool along Waste Lane had been converted into a well constructed sewer. The stench arising from this open sewer and partly covered cesspool was certainly in part due to sewage. The mixture of tan smell with that of putrid organic matter may have given rise to the honest belief that the smell arose from the tannery.

5th. The sanitary measures taken by the Runcorn District Council with regard to sewerage are, in my opinion, sufficient to remove the nuisance mentioned under 4 (it may, however, become necessary to improve the brick sewer under the road and canal if there is any evidence of deposit). The soil on the sides of the ditch and of the old sewer had, however, been so saturated with decomposing organic matter, that entire disappearance of the smell resulting from this may not be complete at once.

6th. I am convinced that the severe outbreak of diphtheria, which has afflicted Thelwall since 1899, was originally the result of infection imported from outside. I would recommend a very thorough disinfection of the five houses in which cases of diphtheria have occurred, also a thorough inspection of the throats of the inhabitants, followed by disinfection of the throat and nose of any suspicious case.

7th. It is obvious that the smell of a tannery is not pleasant, and may be very disagreeable to persons living in the neighbourhood. Whether such a smell, when not proved to be noxious, constitutes a nuisance against which

persons coming to live near a pre-existing tannery have a remedy, is question of common law, which is entirely outside the province of public health.

February, 1903.

Nearly a year having elapsed since the completion of the report, it is now possible to test the accuracy of the conclusions arrived at, in the light of the experience gained during the year 1903. Dr. Adams has kindly written to me the following letter in answer to my enquiries:—

January 23rd, 1904.

"DEAR PROFESSOR DELÉPINE,—

"I enclose on separate sheets, the cases for comparison with Table III. "of your Report. (*See p. 276.*)

"In accordance with recommendations in your Report, I made house-to- "house visitation of the premises mentioned therein, for the purpose of "throat examination, etc. I succeeded in obtaining swabs from the throats "of Emily Quennel, Emma Scarle and Ethel Vickers, on July 16th and 15th, "which were examined and reported on by you.* . . . . . J. Leah flatly "refused to allow his children's throats to be touched in any way. . . . . .

"All the affected houses were thoroughly disinfected with formalin "spray.

"I have just heard from the surveyor in reply to my enquiries; he "writes:—'I believe the sewers are all working well, and I have not heard "'of any complaints.'

"JOSEPH ADAMS, *Medical Officer of Health.*"

From the data furnished at various times by Dr. J. Adams I have constructed the following short tables:—

CASES OF NOTIFIABLE INFECTIOUS DISEASES REPORTED TO THE MEDICAL OFFICER OF HEALTH OF THE RURAL DISTRICT OF RUNCORN.

Place	Years	Total No. of Cases		No. of Cases of Diphtheria	
		Actual Number	Per-centage	Actual Number	Per-centage
Whole district, population 23,203 (1901 census)	8 years (1895–1902) yearly average	158	0·67	35·6	0·15
	1902	226	0·97	35	0·15
	1903	**88**	**0·38**	**28**	**0·12**
Thelwall, population 481 (1901 census)	8 years (1895–1902) yearly average	2·7	0·56	2·1	0·41
	1901	6	1·23	6	1·23
	1902	2	0·41	2	0·41
	1903	**2**	**0·41**	**0**	**0·00**

These figures show that during the year 1903 the incidence of cases of infectious diseases was below the yearly average for the previous 8 years, both in Thelwall itself and in the whole district. As during the year

* The results of these examinations were as follows:—A. E. Quennel, æt 11, no diphtheria bacilli found; Emma Scarle, æt 17, no diphtheria bacilli found; Ethel Vickers, æt 11, diphtheria bacilli, long and short, present, but not virulent.

1903 the number of cases notified in the whole district was below the average, it is well to note that notwithstanding this general improvement there are some localities in which an increased incidence of Diphtheria was observed.

	Total Cases Percentage		Cases of Diphtheria Percentage	
	8 years average	1903	8 years average	1903
Halton, population 1,238	1·57	0·64	0·23	0·56
Daresbury, population 153 ...	0·72	2·61	0·00	2·00
Walton Inferior, population 719	0·36	0·83	0·06	0·55

Every fact observed during the year 1903 seems, therefore, to indicate that the conclusions arrived at with regard to the cause of the Thelwall outbreak were correct, and that the locality was not under the influence of specially noxious effluvia but had simply suffered from the effects of a contagious disease imported into it.

February, 1904. S. D.

NOTIFIED CASES OF DIPHTHERIA, SCARLET FEVER, TYPHOID FEVER AND ERYSIPELAS, IN 1903.

Place.	Total.	Diphtheria.
Aston	1	—
Aston Grange	1	—
Clifton	—	—
Halton	8	7
Stockham	1	—
Sutton	1	1
Weston	10	1
Alvanley	—	—
Frodsham	9	6
Frodsham Lordship	3	2
Kingsley	1	—
Newton-by-Frodsham	1	1
Norley	2	—
Acton Grange	1	—
Antrobus	1	1
Appleton	1	—
Budworth	1	—
Daresbury	4	3
Hatton	2	—
Moore	3	—
Newton-by-Daresbury	3	—
Preston-o'th'-Hill	2	—
Walton Inferior	6	4
„ Superior	1	—
Whitley „	3	—
Grappenhall	3	—
Thelwall	2	—
Latchford Without	1	—
Stockton Heath	16	2

Names omitted = No case notified. J. ADAMS.

The Characters of the Yeasts Occurring in Tanning Materials and in Tannery Liquors and Effluents.

BY

J. R. CARVER, M.D., D.P.H.,

Assistant in the Public Health Laboratory, Manchester.

The Characters of the Yeasts Occurring in Tanning Materials and in Tannery Liquors and Effluents.

Introduction.

The following observations were made at the request of Professor Delépine. In the course of an investigation upon the possible relations between the use of brewer's yeast in a tannery and the production of noxious gases, he ascertained that various yeasts were present in all the materials used in two tanneries as well as in all the liquors and effluents; and that the fermentations to which they gave rise were not accompanied by the generation of noxious gases. (See Report upon an alleged effluvium nuisance, etc., *(p. 251)*.

The object of my investigation was:—

1. To ascertain whether the various forms of yeast cells observed by Professor Delépine belonged to separate kinds of races of yeasts having distinct cultural characters or properties.

2. To determine the characters of the yeasts which could be isolated from the various tanning materials, liquors and effluents.

3. To ascertain, if possible whether yeasts played any useful part in the tanning processes.

Occurrence of Yeasts in Tanning Materials, etc. First of all I give a list of the various samples submitted to me for examination, and the results of my first rough examination for the presence of yeasts and attempts to isolate and cultivate them.

No.	Nature of sample.	Yeasts seen by Microscope.	Yeast isolated.
375	Brewers' yeast	Abundant	Nearly pure growth
376	Recent tan liquor and brewers' yeast	Many	None (seen on culture)
377	Same (7 days old)	Many	None
379	Tan liquor, without added yeast	Many	One variety
381	Liquor from lime pits	Few	One variety
382	Crude effluent	Many varieties	None
383	Settled effluent	Few	None
385	Soaks (3 days old)	Many	None
396	Valonia extract	Few	One variety
397	Myrabolam extract	Several varieties	No growth
398	Mimosa extract	Few	One variety
399	Sumach extract	Few	No growth
401	Sumach powder	—	One variety
403	Valonia cups	—	One variety
404	Block Gambier	—	One variety
405	Recent tan liquor	Several varieties	One variety
406	Tan liquor and brewers' yeast	Several varieties	One variety
407	Crude effluent	Several varieties	None
408	Settled effluent	Several varieties	None
409	Recent tan liquor, D.	Few	One variety
410	Old tan liquor, D.	Few	One variety
444	Waste tan	Many varieties	4 varieties

These samples were the same as those collected by or for Professor Delépine, who handed them over to me. (For fuller details about materials see his own report).

Thus, out of 22 samples examined, yeasts were seen in all of them, but were isolated and cultivated from 13 only. In two of the thick, concentrated extracts, no growth at all could be obtained. It will be seen that with these two exceptions yeasts were grown from all the tannery materials (in 376 one was isolated but accidentally lost); but in all the waste materials with one exception, No. 381, where the yeast may have been of atmospherical origin, no yeast was isolated, owing to the great number of other organisms present.

Methods of Investigation. The first method used in attempting to isolate the yeasts was to make successive strokes on a large disc of sterilised potato in a Petri capsule, so as to obtain isolated colonies; these were incubated at various temperatures. This method was not very satisfactory, as the yeast colony on potato is not very characteristic, and when small is almost invisible. Later I made plates of wort-gelatin 10 per cent., and incubated at 22°, and I found that on this medium the yeast colonies are fairly characteristic and could be made out even when quite small.

A rapid microscopical examination of the various materials showed that all the yeasts were not of the same size and shape, and this was confirmed when the yeasts were isolated and pure cultures obtained.

Determination of the Kinds of Yeasts. We are met with the difficulty of there being no complete classification based on morphological and structural characters; most of the work on the subject has been done in connection with brewery yeasts under the lead of Hansen's classical work. Hansen divides the yeasts occurring in the brewery into three great classes, to which he gives the names adopted by Reess:—

1. Saccharomyces, Cerevisiae,
2. „ Ellipsoideus,
3. „ Pastorianus,

the first being the true brewing yeast while the others are the so-called "wild yeasts" which are injurious to the beer. Hansen declares that it is impossible to classify these brewing yeasts by their morphology and characters of growth in different media and at different temperatures. He proceeds by first producing cultures from single cells and distinguishes yeasts by their power of forming surface films on wort in a certain number of days and of forming endospores at different temperatures. Joergensen, on the basis of Hansen's researches gives 27 to 30 kinds of yeasts.

I had thought at first that it might be possible to differentiate the yeasts by the appearance of their colonies in plate cultures, but this has been found impossible except in two or three cases, as nearly all of them have the same appearance, colonies of various sizes being spherical with a smooth outline and colour varying from white to brown. Some of the surface colonies Nos. 403

and 406 had a tendency to form a fringed outline; and the red yeasts produce characteristic pigment. The oidium No. 444A formed a very characteristic colony of stellate appearance, looking to the naked eye like a small mould.

In well-grown cultures, on the surface of wort-gelatin and other media, there is an obvious broad division of the yeasts into three groups:—

1. Those forming a dry, hard growth, which is very difficult to remove with the needle, the cells seem to be cohesive, and this gives a hard, granular appearance to cover glass films.

2. Those forming a soft, moist growth, with gradual liquefaction of gelatin media.

3. Those producing a characteristic pigment.

There also appears to be striking differences in the power of fermenting glucose and in the growth at different temperatures.

The yeasts have grown well in all the usual laboratory media, and the most favourable temperature appears to be about 26°C. I have found wort-gelatin 10 per cent. to be the best medium, there is good growth in liquid wort, and in this certain differences may be noticed:—

1. The surface growth or formation of a film.

2. Alcoholic fermentation.

3. Sour or acetic fermentation.

Morphological Characters. I now come to a more detailed description of the different types. Some of the yeasts isolated from different materials are evidently identical, such as 409 and 410 from two tan liquors in Mr. D.'s tannery, and 381 and 344c, the pink yeasts. Some others which appeared different at first have approached a common type in later cultures and have been classed together. It must be noticed that in all the yeast cultures we see cells which are exceptions to the general type of cell found, to which the description applies. These types are as follows:—

Type	No.	Material.		Description.
A	375	Brewers' yeast	(fig. 1 in plate)	Large round and oval cells
B	444B	Waste tan		Large round and oval cells
C	401	Sumach powder	(fig. 4)	Small round
D	403	Valonia cups	(fig. 3)	Large oval and ellipsoid
	406	Tan liquor and brewer's yeast		
E	379	Tan liquor (no added yeast)		Medium size oval
	396	Valonia extract		
	405	Recent tan liquor (no added yeast)		
F	381	Liquor from lime pits	(fig. 2)	Medium oval (pink yeasts)
	444C	Waste tan		
G	398	Mimosa extract	(fig. 5)	Small oval
	304	Block Gambier		
H	409	Recent tan liquor	(fig. 7)	Medium or small size ellipsoid
	410	Old ,, ,,		
I	444A	Waste tan	(fig. 6)	Oidium

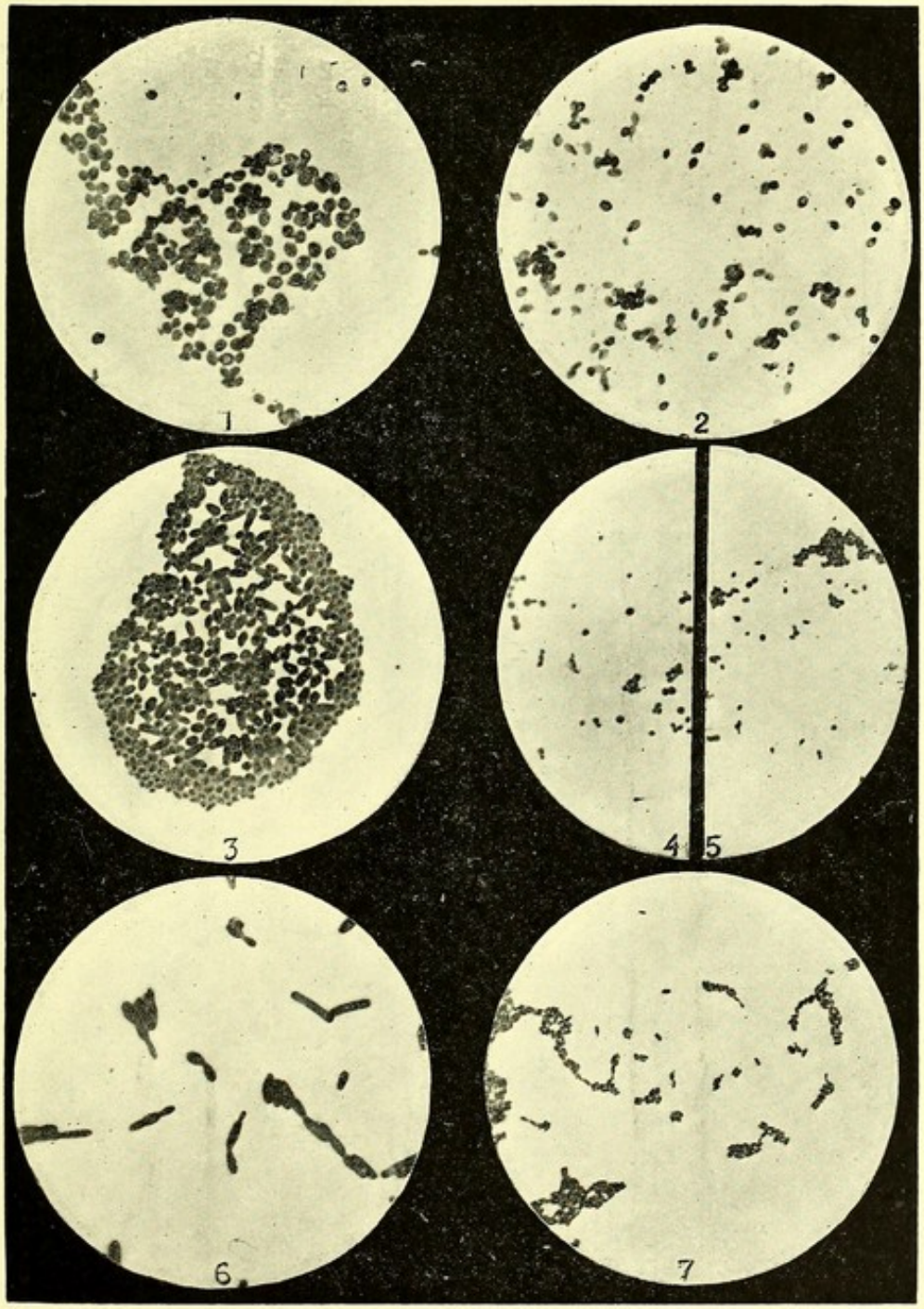
Relation of Yeasts to the Souring of Tan Liquors. It had long been surmised that bacteria played some part in the tanning process, it was found that there was a gradual souring of the bark liquors, so that it has

been the custom to add old liquor to the new batch. Gmelin and Wladika found that acetic and lactic acids were largely produced and were derived from the saccharine constituents of the bark. Haenlein isolated a bacillus, the "bacillus corticalis," which he says plays an important part in the souring process. It acts on saccharose, glucose, and lactose producing acids and a large quantity of gas, chiefly hydrogen, but tannin is not attacked by the organism.

Acid-forming bacteria have also been described occurring in "the soak pits, and are supposed to have an important action on the 'plumping' process, causing the hides to swell.

It was thought desirable to find if the yeasts which are present in the liquor have any important action in the souring process. In wort and in glucose broth there is a slight acid formation with most of the yeasts, but there is none with lactose gelatin nor in a litmus milk medium. As there is no acid fermentation of lactose and milk, it is improbable that the yeasts play an important part in the formation of lactic acid.

The following tables (I.—V.) show, more clearly than a written description, the results of my examinations of these different yeasts.



Some of the Yeasts from Tanning Materials, etc., × 280.
See table on page 284.

Table I.—Giving general character of Yeasts and their growth on various Media.

No.	Material.	Type	Size. L'ght Micron.	Size. Brdth. Micron.	Appearance on Wort Gelatin.	Growth on Wort Gelatin.	Growth in Wort: Sediment.	Growth in Wort: Film.	Growth in Wort: Odour.	Growth on Potato.	Growth on Serum.
375	Brewer's Yeast.....	A	8	4-8	large, round, and oval	heavy, white, dry, hard	abundant	fair	alcohol as in beer	dry, hard	good growth
379	Recent tan liquor with added Brewer's Yeast	E	6-2	2	medium size, oval	pale, moist, creamy	moderate	slow, small	sour beer	moist, cream	good
381	Liquor from lime pits	F	5-2	3-2	medium, oval	bright red colour, moist, liquefying	moderate	slow, moderate	no alcohol smell	thick, moist, bright red	small
396	Valonia extract	E	4-8	2	medium, oval	pale, moist, creamy	moderate	slow, small	marked amyl, odour	thick, moist, cream	very small
398	Mimosa extract	G	4	2	small, oval	heavy, moist, creamy	moderate	slow, small	beer with slight amyl, smell	thick, moist, cream	very small
401	Sumach powder	C	3-2		small, round	slow, white, moist	abundant	fair	sour beer	thick, moist, brown	good
403	Valonia cups.....	D	7-2	4	large, oval and ellipsoid	heavy, white, dry, hard, fringed edge	abundant	slow, small	beer alcohol strong	dry hard, heavy	good
404	Block Gambier.....	G	4	2-4	small, oval	slow, pale, transparent, moist	abundant	moderate	slight alcoholic sweet herbaceous odour	slow, dry, hard	very small
405	Recent tan liquor.....	E	5	2-8	medium, oval	heavy, creamy, moist	abundant	rapid, heavy	sour bee	heavy, moist, creamy	good
406	Recent tan liquor Brewer's Yeast added	D	7-6	3-2	large, oval and ellipsoid	heavy, thick, white, dry, hard, fringed edge	very large	slow, small	beer alcohol strong	white, dry, hard	good
409	Recent tan liquor..... (D. Tannery)	H	4-8	2-4	medium, ellipsoid	pale, moist, liquefying, transparent	moderate	slow, small	slight odour of beer alcohol	slight creamy	good
410	Old tan liquor ..... (D. Tannery)	H	5	2	medium, ellipsoid	pale, moist, transparent, liquefying	moderate	slow, small	slight odour of beer alcohol	slight creamy	good
444A	Waste tan.....	I	14	4	very long	pale, dry, branching colonies, growing into mediu m	small	none	no alcoholic odour	hard, grey	good
444B	Waste tan.....	B	6-8	5-2	large, round and oval	thick, moist, cream	abundant	slow, small	marked odour of beer	thick, moist, buff colour	good
444C	Waste tan.....	F	5-2	3-2	medium, oval	bright red, moist	moderate	slow, moderate	no alcoholic odour	brighred, thick, moist	very small

Table II.—Showing the fermenting power of the Yeasts grown in gelatin to which various sugars had been added.

No. of Type.		Production of Gas. Glucose gelatin.	Wort gelatin.	Lactose gelatin.	Alteration in the specific gravity of wort after incubations at 25° cent. Sp. gr. of wort after 4 days. Control = 1080	Another experiment after 4 weeks growth. Control = 1084
375	A	abundant	abundant	none	1022	1026
379	E	slight	slight	none	1070	1072
381	F	none	none	none	1078	1084
396	E	slight	very slight	none	1072	1080
398	G	moderate	abundant	none	1050	1040
401	C	slight	moderate	none	1050	1042
403	D	abundant	abundant	none	1054	1038
404	G	moderate	moderate	none	1068	1065
405	E	none	none	none	1078	1082
406	D	abundant	abundant	none	1036	1040
409	H	slight	slight	none	1072	1080
410	H	slight	slight	none	1074	—
444A	I	none	none	none	—	—
444B	B	abundant	abundant	none	1055	1038
44C	F	none	none	—	—	—

Table III.—Showing the spore formation at different temperatures.

No.	Temp.	1 day	3 days	7 days	10 days	Remarks
375	10°C.	0	0	0	0	A large amount of budding seen.
	19	0	few	few	abundant	2-4 spores to the cell. In another
	29	0	few	few	abundant	experiment very few spores were
	36	abundant	abundant	abundant	abundant	seen
379	10	0	0	0	0	The cells show marked metachro-
	19	0	0	0	0	matism, dark granular matter at
	29	0	0	0	0	the poles and dark bands across
	36	0	0	0	0	the centre
381	10	0	0	0	0	Cells very small ; no sign of growth
	19	0	0	0	0	
	29	0	0	0	0	
	36	0	0	0	0	
396	10	0	0	0	0	Numerous dark granules seen
	19	0	0	0	0	
	29	0	0	0	0	
	36	0	0	0	0	
398	10	0	0	0	0	In first experiment no spores were
	19	0	?	few	few	seen. Not more than 2 seen in a
	29	0	few	few	few	cell.
	36	0	few	few	few	
401	10	0	0	0	0	None seen in first experiment.
	19	0	0	few	few	2 only in the cell.
	29	0	0	few	few	
	36	0	0	few	few	
403	10	0	few	few	few	Large opaque cells. Much budding
	19	few	few	few	few	
	29	few	few	few	few	
	36	0	few	few	few	
404	19	0	0	0	0	Marked metachromatism
	29	0	0	0	0	
405	16	0	0	0	0	Cells large, much budding. Vacuola-
	29	0	0	0	0	tion seen
	35	0	0	0	0	
406	16	0	?	few	few	2 spores only to a cell. None seen
	19	0	?	few	few	in first experiment
	25	0	?	few	few	
409 & 410	16	0	0	?	very few	
	29	0	0	?	very few	
	36	0	0	?	very few	
444A	16	0	0	0	0	Cells grow to an enormous length
	29	0	0	0	0	with several large dark nuclei in
	38	0	0	0	0	them.
444B	16	0	?	numerous	numerous	2-4 spores on the cell. None seen in
	29	0	?	numerous	numerous	first experiment
	38	0	?	numerous	numerous	

Table IV.—Showing growth on potato at different temperatures.

No.		0°-2°	4-5°	6-8°	10-12°	14°	20-22°	29°	35	40°	45°C.
375	3 days				0	?		good	fair	0	
	7 ,,				scanty	fair					
	14 ,,	0	0	0	scanty	fair		good	fair	0	
379	3 days		0	0	0	?		good	0		
	7 ,,		0	fair	fair	good		good	0		
	14 ,,	0	scanty	fair	good	good		good	0		
381	3 days		0	0	0	scanty		good	0		
	7 ,,		?	?	scanty	fair		good			
	14 ,,	0	fair	?	scanty	fair		good	?		
396	3 days		0	0	0	fair		fair	0		
	7 ,,		0	0	scanty	good		good			
	14 ,,	0	0	0	scanty	good		good	0		
398	3 days		0	0	0	0		fair	fair	0	
	7 ,,		0	fair		fair		good	good		
	14 ,,	0	0	fair	fair	fair		good	good	0	
401	3 days		0	scanty	?	scanty		fair	scanty	0	
	7 ,,		fair	good	good	good		good	good		
	14 ,,	0	good	good	good	good		good	good	0	
403	3 days		0	?	fair	fair		fair	fair	scanty	0
	7 ,,		0		good	good		good	good		
	14 ,,	0	scanty	?	good	good		good	good	scanty	?
404	3 days		0	0	0	scanty		fair	fair	0	
	7 ,,		0		?			fair	fair		
	14 ,,	0	0	0	scanty	scanty		fair	fair	0	
405	3 days		0	0	0	0	0	fair	fair	0	
	7 ,,		0								
	14 ,,	0	0	?	?	?	fair	good	good	0	
406	3 days		0	0	0	scanty		fair	fair	0	
	7 ,,		0								
	14 ,,	0	0	0	fair	fair		good	good	0	
409	3 days		0	fair	fair	fair	0	0	0		
and	7 ,,		fair								
410	14 ,,	0	scanty	good	good	good	good	0	0		
444A	3 days		0	0	0		scanty	0			
	7 ,,		0		0						
	14 ,,	0	0	0	scanty		fair	0			
444B	3 days	scanty	scanty	scanty	scanty	scanty		fair	0		
	7 ,,										
	14 ,,	fair	fair	fair	fair	good		good	0		

Table V.—Showing the reactions of Media after growth of various Yeasts.

No.	Type	Lactose gelatin 1 month	Litmus Milk 6 weeks.	Wort 1 month.	Glucose broth 6 weeks.	Peptone broth (*Alk.*) 1 month.
375	A	no acid fermentations	neutral reaction	acid reaction	acid	alkaline
379	E	,, ,,	,, ,,	acid	neutral	alkaline
381	F	,, ,,	,, ,,	neutral	neutral	neutral
396	I	,, ,,	,, ,,	faint acid	acid	neutral
398	G	,, ,,	,, ,,	strong acid	acid	alkaline
401	C	,, ,,	,, ,,	acid	acid	alkaline
403	D	,, ,,	,, ,,	acid	acid	alkaline
404	G	,, ,,	,, ,,	strong acid	acid	alkaline
405	E	,, ,,	,, ,,	neutral	alkaline	alkaline
406	D	,, ,,	,, ,,	acid	acid	alkaline
409 & 410	H	,, ,,	,, ,,	acid	acid	faint acid
444A	I	,, ,,	,, ,,	alkaline	neutral	alkaline
444B	B	,, ,,	,, ,,	acid	acid	neutral

Except in the case of the peptone broth, the reaction of all the media used was neutral.

General Remarks. Since the yeasts were first isolated some of them have shown certain alterations in characters. No. 401, Type C, the small round yeast, has certainly grown larger, while Nos. 409 and 410, Type H, have apparently become smaller. Many of them have acquired a greater fermenting power, and appear to form spores more readily. It was a matter of some difficulty to obtain pure cultures of some of the yeasts, particularly in No. 444. In several successive plates made from apparently pure cultures from single colonies, I found two or more varieties.

The yeasts all stain well with the usual basic aniline dyes, and they retain the Gram's stain. When stained by the Ziehl-Neelsen method, the cells do not retain the red, but there are in some of the specimens a few cells or parts of cells which remain red. This may be due to the formation of spores. No. 379 and others of Type E show a considerable amount of metachromatism, especially in the early cultures. Polar staining was observed and also a dark band across the centre.

There was little difference in the appearance of the cells on different media. In film growths in fluids there was a tendency to the formation of chains of cells having a mycelium appearance. The oidium No. 444A seemed to be distinctly smaller when grown on Leoffler's serum.

I tried several methods of observing the formation of spores. I found the moist filter paper cells method most convenient. In those yeasts in which I could not demonstrate the presence of spores there appear to be changes produced which are characterised by the appearance of metachromatism in bands and large granules and also of vacuoles.

It will be seen that some yeasts form spores more readily than others, but I was unable to differentiate the yeasts by observing the formation of spores at certain temperatures in a definite time as has been done in the case of the brewing yeasts.

The micro-photographs which are appended show the appearances of the cells and show the differences in the size and shape. They were taken by Mr. F. Simons, Technical Assistant to the Laboratory.

Conclusions. 1. Yeasts are present in all, or nearly all, of the vegetable materials in use in the tanning industry, and can be isolated from them.

2. They are present also in all the waste products, but their isolation is much more difficult owing to the presence of a large number of bacteria.

3. The yeasts in pure cultures do not produce any bad smelling gases, and do not appear to contribute to the smell of the tan liquors.

4. On the basis of morphological and cultural characters it is possible to recognise seven or eight varieties of yeasts, but I am not able to state that these forms correspond to as many good species, for after being grown for several generations in pure cultures, some of the forms which at first appeared to be distinct, gradually approached a common type.

5. In my observations spore formation seemed to take place more readily in the yeasts which had the greater power of producing alcoholic fermentation.

6. Whatever share yeasts may have in the "souring" of ordinary tan liquors, there was no clear indication of such action in any of the pure cultures studied; moreover, in old tan liquors and waste products, the yeasts did not seem to have increased in number equally with bacteria.

Investigation of an Epidemic of Fish Poisoning at Drinkwater Park.

BY

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Investigation of an Epidemic of Fish Poisoning at Drinkwater Park.

The sheet of water at Drinkwater Park is rented by an angling association.

In November, 1902, it was noticed that there was a very excessive mortality amongst the fish, hundreds of dead fish being removed from the lower reach of the lake. The epidemic continued, and in January, 1903, the association consulted Professor Delépine with a view to discovering the cause of the outbreak.

At his request we visited the place the same month.

Description of the Lake.

The lake is about five acres in extent and about six feet deep, the banks being formed of shelving earth. It is fed by a few small streams, the principal one entering at the north end, and this stream is the only one exposed to the possibility of contamination by manufacturing or other polluted effluents.

Species of Fish.

We were informed that during the three previous years the lake had been stocked with between four and five thousand fish consisting of roach (Leuciscus rutilus), dace (Leuciscus vulgaris), chub (Leuciscus cephalus), perch (Perca fluviatilis), bream (Abramis brama), and carp (Cyprinus carpio). The fish had thriven and grown in size, and up to the time of the epidemic very few dead ones had been seen.

Condition of Lake, January, 1903.

At the time of our visit the lake was very full, the mud banks being entirely submerged. The water looked fairly clean, there was no marked discolouration or scum, and no smell perceptible over the greater part of the lake; but at the upper reach, where the main stream enters, there was an unpleasant smell suggestive of sewage, and we found that the effluent from the Clough Bottom Bleach Works was passing into the stream.

This effluent was turbid, slightly yellow in colour and had an unpleasant smell, but we were informed that it did not contain any chemicals or materials used in bleaching or dyeing, but only the

last washings of the cloth. It had, we were told, up to November, 1901, passed directly into the stream, but in that month it had been diverted and only now passes into the stream during floods.

Condition of the Fish.

We noticed a considerable number of dead and dying fish, the majority of which were well fed and in good condition. Some were extensively affected with saprolegnia, but others were quite free and showed no evidence of disease.

Possible Causes of the Epidemic.

Two possible causes of the epidemic presented themselves:—

1. The fish might be suffering from some contagious disease.

2. The epidemic might be due to some poison contained in the bleach works' effluent or introduced into the lake by some other means. We carefully examined some of the dead fish and found no evidence of bacterial or protozoon infection, and in those affected with saprolegnia it appeared probable that it was a secondary infection affecting only dead and dying fish.

A chemical examination of the water and bleach works' effluent failed to detect any substance that would account for the outbreak.

Experiments.

The following experiments were made to ascertain—

A. *Whether fish would live in the lake water.*

A healthy roach was placed in a tank containing four litres of the lake water which was kept well aerated.

The fish continued well for several days, when the experiment was discontinued.

B. *Whether fish would live in the lake water to which some of the bleach works' effluent had been added.*

Two healthy fish were placed in a tank containing four litres of the lake water to which one litre of the bleach works' effluent was gradually added, efficient aeration being maintained.

In this case also the fish were not in any way affected.

C. *Whether fish would live in the bleach works' effluent.*

In this case again no ill-effects were observed.

D. *Whether a diseased fish was capable of communicating disease to a healthy one in pure water.*

A dead fish removed from the lake and affected with saprolegnia was placed in a tank containing well aerated tap water. A roach and a carp introduced into this tank continued healthy during the experiment which lasted for over a fortnight.

E. *Whether a diseased fish was able to communicate disease to a healthy one in lake water.*

A dead fish removed from the lake and affected with saprolegnia was placed in a tank containing four litres of well aerated lake water and a healthy roach and carp were placed in this tank.

The carp died in three days and the roach a day later. The blood of these fish was examined for bacteria but none were found, nor could any evidence be obtained that they had died from infectious disease. As the dead fish used in this experiment was in a state of decomposition, it was felt that too much stress should not be laid on this result, and no material was available for repeating the experiment.

The results so far obtained seemed to suggest that the epidemic was not caused by the bleach works' effluent, and probably also was not due to infectious disease.

Early in April, 1903, a sample of muddy water was sent to us which had been collected from the upper end of the lake after stirring up the water. It was found to be dark and turbid from the presence of fine black particles, and had a smell resembling bisulphide of carbon or naphtha and possibly putrid matter. On evaporating to dryness a tarry material was obtained which smelt strongly of mineral pitch. The analysis of this material by Dr. Walker Hall is given below.*

Condition of the Lake, May, 1903.

We visited the lake again at the end of May. The water was now very low and the muddy banks were exposed to view. At the north end, where the main stream enters, the sand of the exposed banks was blackened for a depth of three or four inches and in the small pools left at the side of the stream there was a scum of oily-looking material with a strong tarry smell. The deposit on the exposed banks of the stream was very evident as far as the point marked A on the map. Above this point it could be traced in places, but it had to a large extent been covered by a deposit of clean sand. From the point A to the bleach works the stream is much more rapid than below the point A, where it widens out somewhat before entering the lake.

At the bleach works the stream is culverted as far as the lower end of Prestwich Clough, and above this point is again sluggish for about 100 yards, and here again the tarry deposit was distinctly recognisable.

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* Extraction of the colouring matter by ether, hydrochloric acid and benzine yielded a yellow fluorescent solution. The acid extract gave the reactions of one of the naphthalamine series, and none of the extracts contained either phenol or naphthol.

On distillation an irritating hydrocarbon was obtained which had a low melting point. The following tests were applied at Professor Deiépine's request:—Upon evaporation of the sand to dryness and subsequent heating globules of a dark tarry substance remained. These smelt strongly of mineral pitch and were soluble in xylol and benzine, giving a reddish fluorescent solution. This reaction is characteristic of mineral pitch. Traces of iron and calcium were also found.

Still higher up the stream the same deposit could be detected by removing the surface sand and evidence of its presence was traceable as far as a short distance above the point where it is crossed by the Bury New Road. (Shown in the map as a thick, straight, black line.)

A reference to the map will show that the road here crosses the shallow valley on a raised embankment, and the whole of its surface water for about a distance of 300 yards on each side of the stream passes into it by two open channels, one on each side of the road.

At the time of our visit these channels were much discoloured and contained tarry mud. No trace of the tarry material could be found above the entrance of these channels.

We traced the stream to its source and found that no sewage or trade effluents entered it above this point.

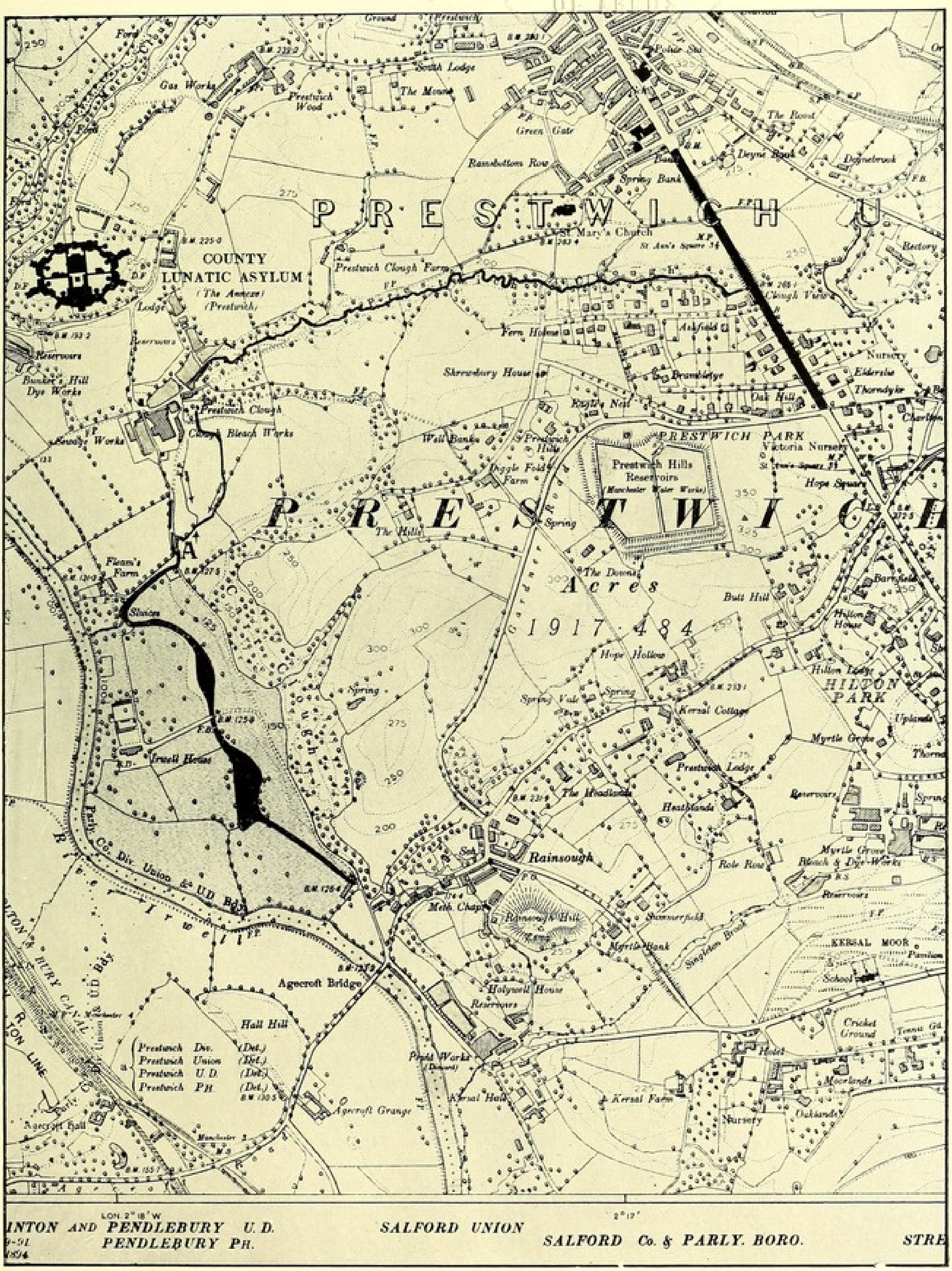
It appeared evident that the tarry material had been derived from the road, and enquiries showed that this stretch of road that drained into the stream had been remade during the winter and newly paved with granite sets laid in tar.

Experiments

Attempts were made in the Laboratory to reproduce in an experimental stream a tarry deposit similar to the one observed, and to ascertain whether the materials used in road making could impart to a pure water substances deleterious to fish life.

For this purpose a shallow V-shaped wooden trough about 12 ft. long was partly filled with silver sand and at the upper end a small stream of water was allowed to pass from a glass vessel which was supplied with tap water, the tap water being made to drip on to blocks of wood thickly coated with the freshly-prepared asphalte and solvent used in road making. The effluent, which was collected in a glass vessel, was clear and had a distinct but not very strong tarry smell. At the upper end of the trough a black deposit began at once to form and this gradually extended until the whole of the sand was coated, the appearance produced being very similar to that observed in the banks of the stream supplying the lake. A goldfish (Carassius auratus.) placed in the effluent was at once affected, rising to the surface and preserving its balance with difficulty. The opercula were at first kept closed and then opened and closed spasmodically. Death took place in 45 minutes.

A similar experiment was made with a small carp which in 10 minutes was evidently in a dying condition, but recovered in about half an hour after being placed in clean water. In water containing 30 per cent. of the effluent fish were also rapidly affected and were in a dying condition in 20 minutes, but, as before, recovered on being placed in clean water. In water containing 20 per cent. of the effluent they were visibly affected in 2 hours.



In water containing 10 per cent. of the effluent a small rudd showed toxic symptoms in 2 hours, but afterwards recovered somewhat and was still alive in 24 hours.

A similar experiment with similar results was made with a small carp.

In water containing 1 per cent. of the effluent a small rudd lived for 24 hours and showed no toxic symptoms.

In all the above experiments the water was kept well aerated during the whole of the observations.

Further experiment showed that the effluent when allowed to stand lost its toxic properties to a considerable extent. A small carp, placed in a water containing 20 per cent. of an effluent that had been prepared on the previous day was unaffected in 24 hours, and a similar result was got when a small rudd was placed in a water containing 10 per cent. of the effluent.

Conclusion.

The conclusion, therefore, is that water passing over the freshly-prepared materials used in road making is capable of producing death in fish of different species, and it appears clear that the epidemic in Drinkwater Park was due to some materials conveyed into the lake from the recently repaved Bury New Road, materials having a direct toxic action on the nerve centres. In some cases an increased redness of the gills was observable 24 hours after the conclusion of the experiment, but no deposit was ever noticed.

We must express our thanks and indebtedness to Professor Delépine for the help he gave us all through the investigation and for the valuable suggestions he made as to the method of conducting the enquiry.

On Two Aldehyde Reactions.

BY

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On Two Aldehyde Reactions.

In the course of some investigations, on which I am now engaged at the suggestion of Professor Delépine, concerning the action and behaviour of formaldehyde as a preservative in food, it became necessary that some delicate and simple method of detecting the formaldehyde should be used, which is capable at the same time of being employed as a quick method of estimation. After a survey of the various ways of determining formaldehyde I am now using the cyanide method of Romjin (1) for the stronger solutions. For the weaker solutions I employ a modification of the method given by MM. Seyewetz and Gibello in the *Bulletin de la Société Chimique de Paris* (June, 1904). These authors estimate the formaldehyde by means of sodium sulphite. Starting with a 20 per cent. solution of sodium sulphite they first neutralise 20 ccs. of it with standard H_2SO_4 using a 0·2 per cent. solution of phenolphthalein as an indicator. The quantity of H_2SO_4 required for this purpose is determined once for all. In another portion of the sulphite a known volume of the formaldehyde solution is added and a second titration with H_2SO_4 carried out. The difference in the two titrations gives a measure of the formaldehyde in the solution according to their equation

$$2Na_2SO_3 + 2HCHO + H_2SO_4 = 2(NaHSO_3HCHO) + Na_2SO_4.$$

In order to detect formaldehyde in solutions too dilute to be estimated by this method, I have modified the test by starting with sodium metabisulphite (thus carrying out a suggestion of Mr. Thos. Tyrer made publicly some time ago). A test solution is made as follows: 50 grammes of sodium metabisulphite are dissolved in 100 ccs. of water and filtered. To this strongly acid solution 50 per cent. NaOH is added until nearly neutral. 2 ccs. of the solution are now taken, made up to 50 ccs. with distilled water and 1 cc. of phenolpthalein solution added, and the whole put into a Nessler glass. Decinormal Na_2CO_3 or NaOH is added until the phenolphthalein indicates a very slight alkalinity. The original solution is then neutralised with the requisite amount of Na_2CO_3 or NaOH, and then allowed to stand for two days in a stoppered bottle. 3 ccs. of this solution added to 48 ccs. of distilled water and 1 cc. of phenolphthalein should only give the faintest possible pink colouration.

To test for formaldehyde 50 ccs. of the solution (neutral) supposed to contain formaldehyde are taken in a Nessler glass, and 2 ccs. of the reagent and 1 cc. phenolphthalein added. A pink to red colour is

produced if formaldehyde is present. A control with distilled water should always be made.

The delicacy of the test with formaldehyde is about 4 parts per million.

As might be expected, other aldehydes, *e.g.*, acetaldehyde, give the reaction, but not with the same dilution as formaldehyde.

It is important that care should be taken to use aldehyde—free alcohol in the preparation of the phenolphthalein solution, as some samples contain aldehyde in appreciable quantity. The reaction may be expressed by the equation

$$Na_2SO_3 + HCHO + H_2O = (NaHSO_3HCHO) + NaOH.$$

Coloured or turbid solutions must first be distilled.

In a search for a reaction capable of detecting quantities less than the lower limit of the above reaction, I found that the ordinary Nessler solution reacted with formaldehyde.

(2) *With Nessler Solution.*

If to a solution of formaldehyde in a Nessler glass the usual quantity of Nessler reagent (2 ccs.) is added, a reaction takes place dependent on the strength of the formaldehyde solution. If the solution is fairly strong an immediate blackish-brown precipitate is produced due to the reduction of the Nessler reagent.

In weaker solutions the reaction proceeds more slowly, a canary-yellow colouration being produced at first, then the solution becomes slightly opalescent and of a greenish-yellow colour, and finally darkens, and on allowing to stand a precipitate falls to the bottom, the colour of which depends on the concentration of the formaldehyde solution.

In a dilution of 1 part HCHO in 1,000,000 of water the change from the canary-yellow to the dark brown takes about one hour to complete. The reaction is quite apparent in a dilution of 1 part HCHO in 2,000,000, and the suspended matter settles down to the bottom in 24 hours.

This reaction is therefore of great delicacy, and can be used for detecting minute traces of HCHO.

(1) *Zeits. f. analyt. Chemie*, vol. xxxvi., pp. 18–24, 1897.

The Brain and Spinal Cord in Chronic Arsenical Poisoning.

BY

REGINALD LAWRENCE, M.D.

The Brain and Spinal Cord in Chronic Arsenical Poisoning.

THIS investigation* was undertaken at the suggestion of Professor Delépine, and was carried out in the Pathological Laboratories of Owens College. It consists of two parts, viz.:—

Part I. An examination of the brain and spinal cord of patients who died during the epidemic of Arsenical Poisoning in Salford and Manchester in 1900–1901, and who during life had well-marked symptoms of "Peripheral Neuritis." The material for this part of the investigation was obtained from the Salford Hospital, the Workhouses at Crumpsall and Withington, and from Halifax, and were collected for Professor Delépine or presented to him by Dr. Ray and Dr. Moore. Dr. Moore collected the greater part of the material and supplied information regarding the autopsies.

Part II. is an examination of the spinal cords of rats which were fed on arsenious acid or its compounds, with or without the addition of alcohol. These experiments were made by Professor Delépine, who placed at my disposal his notes upon them.

The methods employed in the histological examination deserve short mention. The material for Part I. was obtained after the interval which usually intervenes between the death of a patient and the autopsy. The material for Part II. was obtained fresh in those cases in which the animal was killed. In cases where the animal died, a period varying from two or three to sixteen hours might have elapsed between the time of death and the hardening of the specimen. The control experiments made with a view to eliminate *post-mortem* changes will be mentioned later.

The specimens were hardened in 4 per cent. formalin, for a variable time which in some cases extended to several weeks, and subsequently in alcohol of increasing strengths. They were afterwards embedded in celloidin. The sections were cut on a freezing microtome.

The sections were stained with hæmatein alone, or with hæmatein,

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*The results of this investigation formed the subject of a Dissertation presented for the degree of M.D., Victoria University. The microscopical specimens were demonstrated at a meeting at the Public Health Laboratory, on June 1st, 1904.

rubin and orange, or with borax-carmine and hæmatein, to show the structure of the cord. To examine the minute structure of the nerve cells, the sections were stained with methylene-blue or thionin-blue, the methods used differing from the methods described by Nissl in many important respects. Nissl describes two methods: The first consists in staining with an aqueous solution of fuchsin, washing in absolute alcohol and clearing in oil of cloves. This method could not be employed for staining sections embedded in celloidin. The second method described by Nissl is exceedingly complicated, it may be summed up as follows:—

1. Harden in alcohol of successively increasing strengths.

2. Stain in a 5 per cent. solution of methylene-blue, which should be heated until it begins to boil.

3. When the staining solution has cooled down wash the sections in the following mixture:—

Aniline oil	20 c.cm.
90 per cent. alcohol	200 c.cm.

This should be continued as long as the colour comes out and until the white and grey matter are clearly differentiated from each other.

4. Dry with blotting paper. Origanum oil for a short time.

5. Dry with blotting paper, and afterwards remove with benzine any origanum oil which remains.

6. Cover the section with a solution of resin in benzine evaporated down to the consistency of thin Canada balsam.

7. Pass the slide through a flame which ignites the benzine resin.

8. Cover with a cover-glass and warm until the benzine resin fills the space between the slide and the cover-glass.

The difficulties of this long process are obvious, and it was found possible to obtain good results by a much simpler method. The sections were stained in a 0·5 per cent. solution of methylene-blue or thionin-blue with or without warming, washed in the solution described above (No. 3), cleared in xylol and mounted in Canada balsam.

For the purpose of comparison, specimens were hardened in alcohol, saturated solution of perchloride of mercury or formalin 4 per cent. It was found that formalin was not inferior to the other two reagents, and preparations hardened in formalin stained excellently with methylene-blue.

Work by previous observers.

The action of arsenic on the nervous system has been the subject of a large number of experimental enquiries, and the question can scarcely be said to be decided yet. Perhaps the reason is that very

few *post-mortems* have been made till recently. *Dana* (2) says, in 1887, that he can find no record of a *post-mortem*.

The action of arsenic on frogs has been investigated by *Ringer* and *Murrell*, who conclude that arsenic affects at first and very rapidly the nerve centres. In mammals *Scolosuboff* is probably the first who has endeavoured to study experimentally the mechanism of the nervous troubles, and has come to the conclusion that they result from alteration of the nerve centres by arsenic.

In the arsenic experiments, performed under the directon of *Muerzejewsky*, *Popoff* (3) found that in animals submitted to sub-acute poisoning, there are produced very manifest lesions of the cord having the character of an acute polio-myelitis. In the more chronic cases the inflammation extends to the white substance, and produces diffuse myelitis. The nerves examined at the exit from the cord as well as in their peripheral course, show no change, even in the cases where death has supervened three months later.

Later, replying to the objection made to him, *Popoff* described in a case of poisoning in man, the same alterations which he had observed in animals—swelling, cloudiness, decolourisation and vacuolation of the nerve cells, hyperæmia and infiltration by blood of the cervical and dorsal region of the cord, in the neighbourhood of the central canal, the posterior horns and the lateral columns, and plastic exudations in the cervical enlargement. *Kreysig* (4), working under Schültze in Heidelberg, from the results of experiments on animals, says that these changes are due to the method of hardening employed, and can be met in the cord of normal animals. After poisoning by arsenic, he describes no other lesions in the nerve centres than capillary hæmorrhages in the grey matter, which are not constant. He quotes *Vulpian* (5) as having arrived at equally negative results. On a dog in which large doses of arsenic administered for ten days had brought on paralysis, *Jaeschke* only found little apoplexies in the meninges without any other indication of a central myelitis. This author appears to be the first to have attributed the motor and sensory disturbances to peripheral lesions—to a polyneuritis; and this opinion is held to-day by the greater part of those who are interested in the question. Jaeschke is of this opinion, for the following reasons:—

1. The localisation of the sensory or motor disturbances in the limits of certain nerves. In one of his cases there was a well-marked anæsthesia, limited to the region of the median nerve.

2. The marked sensory disturbances, especially the burning pains, ataxia and tenderness along the nerves.

3. Absence of the characteristic weakness in profound spinal paraplegia, viz., vesical weakness.

4. Absence of the rapid and high degree of atrophy which follows anterior polio-myelitis.

5. The comparatively rapid and complete cure, despite a high grade of paralysis.

6. The presence of herpes zoster.

7. The electrical reactions which are those of a mild form of peripheral paralysis.

8. The sensibility of the paralysed muscles.

Dana *(loc. cit.)* adds to these arguments:—

1. The existence of optic neuritis which he has observed.

2. Localised paralysis, as, for example, that of a single vocal cord, observed by Mackenzie.

3. The fact that arsenic can produce a perfect type of pseudo-tabes, like the tabes due to alcohol or diphtheria, and in the last instance we know it is due to a polyneuritis.

Lancereaux (7) relied on this proof by analogy and on the clinical picture of arsenical paralyses to range them in the group of paralyses of peripheral origin.

Naunyn (8) brought forward in a case he observed the analogy of the symptoms with those of a polyneuritis. This opinion was confirmed by the experimental researches of *Alexander* (9), who found in poisoned rabbits degenerative atrophy of nerves and muscles without alteration of the central nervous system. But at the same time he comes to this conclusion, that among animals it is as difficult to determine, by means of a chronic intoxication, the appearance of a polyneuritis as of a myelitis.

Becq (10), in his experiments, has met with the same difficulty—no modification in the structure of the cord.

It would appear from these facts that the nervous manifestations should be, in the majority of cases, attributed to peripheral changes, but no further generalisation can be made, for quite recently *Henschen* (11) has published a work in which he insists on the very clear and widespread primary medullary lesions. He describes changes in the grey matter: cells in the cervical region are scanty and present all transitions between fairly large and almost normal cells with well-defined nuclei and sharp outlines, and distinct but not numerous processes and completely atrophied cells, these latter being very numerous. They are very pale and indistinctly outlined, without nuclei but with rich pigment. Rounded cells with only a few processes are perhaps the most numerous. In the lumbar region the cord is deformed by hæmorrhage, and the ganglion cells are partially atrophied. In connection with the vessels he notices that the large ones are injected and the hyperæmia may go on to hæmorrhage. The clinical picture, he thinks, must in consequence allow a share to the cord in the morbid process.

Silberman (12) attributes the nervous troubles to altered circulatory conditions in the central nervous system; he found hæmorrhages and thrombosis in this part.

Barrs (13) and *Osler* (14) consider it a peripheral neuritis.

In the epidemic of arsenic poisoning at Le Havre a pathological examination of the cord was made in one case. The changes in the cord, viz., foci of disintegration and hyaline transformation, were pronounced by *Cornil* to be merely the result of incomplete preservation and the action of alcohol in which the cord was hardened. *Brouardel* and *Pouchet* ("Etude sur l'Arsenicisme," par Dr. G. Brouardel, 1897) could find no lesions in the cord.

Dr. Thomas Oliver, the writer of the article on Arsenic in Clifford-Allbutt's "System of Medicine," says: "Arsenical multiple neuritis in some respects resembles subacute polio-myelitis, but there is more pain, and the combination of sensory disturbances and tendency to rapid cure diminishes the resemblance. Is the lesion peripheral or central?"

Professor Delépine, struck by the inadequacy of the theory of "peripheral neuritis" and the absence of clear pathological proof, was unable to say dogmatically, in his evidence given in the early part of 1900 before the Commission appointed to enquire into the epidemic of "arsenic poisoning," whether the lesion was peripheral or central. He had examined the peripheral nerves in several cases, and in one case only had found unmistakable degeneration. The nerves in the other cases showed segmentation of the myelin sheaths and other minor changes, which, however, were not sufficient to prove that they were the seat of inflammation or degeneration due to the action of arsenic. The report of this examination of the peripheral nerves has not yet been published, but I am enabled to make the statement by Professor Delépine's kindness.

Normal Histology of Nerve Cells.

The advances in the knowledge of the intimate structure of the nerve cell have been accompanied by an increase in the number of descriptive terms which threatens to cause considerable confusion and concealment of meaning. The terms which I shall use are those which seem to me to be most widely used and open to the least objection. The word neuron is used to denote the whole cell, including its axis cylinder process and its protoplasmic processes. About the nucleus, nucleolus, the axis cylinder and the protoplasmic processes there can be no confusion. The Nissl bodies—better called the chromatophile elements—are the granules which stain with methylene-blue. The achromatic substance is that part of the cell body which does not normally stain with methylene-blue. Of these constituents of a nerve cell the chromatophile elements especially deserve a short notice.

The chromatophile elements were first described by Nissl *(loc. cit.)* in 1892. He gave the morphological characters as follows: Though varying in shape and size they present themselves in many

forms and preserve a fixed arrangement. They appear as irregular masses, etc. They are united into a network. Their parallel arrangement gives them a peculiar tigroid aspect. They are present in the cell body and protoplasmic processes, but absent from the axis cylinder process, and scanty or absent in the conical eminence from which the axis cylinder arises (see Fig. II.).

They are insoluble in mineral acids, acetic acid, warm alcohol, ether or chloroform, soluble in potassium ferro-cyanide and caustic potash. They belong to the group of nucleo-albumins. They are considered by Held (15) to be due to precipitation by the fixing agent; this, however, does not diminish the value of the pathological changes observed if this product is constant and presents itself in normal and pathological conditions in a different form constant for each.

Turner (16), at the suggestion of Dr. Mott, examined the grey matter of the spinal cord of freshly killed animals, in a solution of

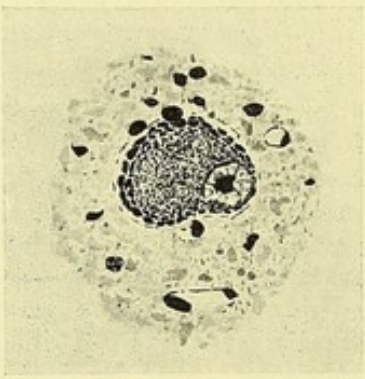

Fig. I (Case XI.).—From anterior horn of grey matter in Lumbar region of Cord. Chronic arsenical poisoning.

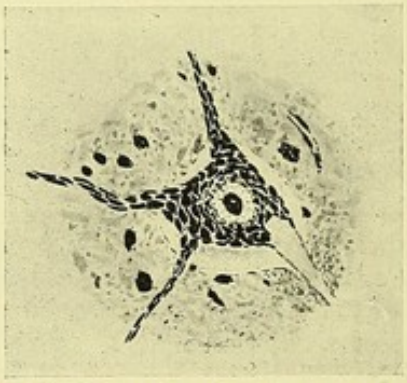

Fig. II.—Normal multipolar cells from anterior horn of grey matter.

methylene blue in normal saline, and found exactly the same appearance.

The function of the chromatophile elements is necessarily a matter of conjecture. They are supposed to form a sort of alimentary reserve—a nutrition granary. (Lugaro, Cajal and Van Gehuchten.) They are supposed to possess considerable chemical properties, giving rise according to their wear and tear to a certain amount of chemical energy.

Changes in the chromatophile elements are demonstrable by the method of staining with methylene-blue, thionin-blue or toluidin-blue. These changes are the earliest demonstrable morphological changes indicative of an altered functional state. They follow section of the prolongations of the nerve cell (shutting off the normal afferent stimuli), the action of various poisons including bacterial toxines, elevation of temperature, anæmia and excessive stimulation.

The chromatophile elements undergo disintegration and disappear

as a result of morbid processes in the cell. The constituent granules disappear and their transition is marked by diminished affinity for basic dyes, this process is called *Chromatolysis*, and cells which are the seat of this change have the appearance of having their chromatin particles distributed throughout the cell in the form of a fine powder or dust. Chromatolysis may be peripheral, peri-nuclear (see Fig. I.), or diffuse.

The achromatic substance or "unstainable substance" is generally supposed to possess a fibrillar structure. When it stains deeply with the stains which normally stain the chromatophile elements it may be interpreted as a pathological appearance. This change is said to occur in poisoning by arsenic and antimony and in Landry's paralysis.

The nucleus in the normal nerve cell is chiefly characterized by its very definite outline, its size and its nucleolus. The nucleus stains very faintly with methylene-blue while the nucleolus stains very deeply. The normal position of the nucleus is in the centre of the cell. Its excentric position is one of the most notable changes in a nerve cell which has been morbidly altered. It may even project beyond the surface of the cell or possibly be extruded. The explanation of this is that the achromatic substance which moors the nucleus in its central position becomes destroyed. Lugaro (17.)

The axis cylinder processes do not contain chromatophile elements, and these latter are scanty in the conical eminence from which it arises. In this situation chromatolysis is said to commence.

The protoplasmic processes are numerous and contain chromatophile elements in their most typical arrangement, viz., the parallel disposition. Apart from their function as conductors of impulses the protoplasmic processes immensely increase the surface of the cell, and consequently its power of absorbing nutrition.

Pigment in the central nervous system is in point of degree a characteristic of man in contradistinction to the lower animals. There are two kinds of pigment in the central nervous system: one is pale yellow and occurs in the cells of the cerebral cortex, the basal ganglia, the spinal cord, and the spinal ganglia. The other is dark brown and is present in the locus coeruleus and the substantia nigra. Neither are hæmatogenous. The former is the pigment which has come under notice in examining the brain and spinal cord in the human subject. The yellow pigment is not present at birth but appears about the sixth year and continues to increase throughout life. About the chemical composition of the pigment there are two diametrically opposed views: That it is blackened by osmic acid, but not if first treated with ether, therefore it is partly of a fatty nature. The second view is that it is not fat, but that it may undergo a fatty change and then a black reaction is obtained with osmic acid. As to the significance of this pigment Schäfer (18) considers that the presence of pigment is not a sign of decadence but rather

of activity. Possibly the date of appearance of pigment lends support to this view.

Marinesco (19) believes that the pigment results from the transformation of the chromatophile elements and that it is allied to fat. In the human cases in Part I. of this investigation the relation of pigment to chromatophile elements will be noticed.

Alterations in the pigment. The association of excess of pigment with degeneration of nerve cells has been called "Pigmentary or Yellow-Globular Degeneration." That there is a physiological increase of pigment with advancing age is not disputed, but that it is "invariably a witness of bye-gone functional activity" is not so generally accepted. Colucci is of opinion that the pigment is a product of metabolism in the cell. He thinks that all the elements on the spot are probably involved in the transformation as the appearance of the pigment is accompanied by the disappearance of the normal structure of the cell. Excess of pigment is said to be associated especially with slow dementias such as epileptic dementia and general paralysis. It has been found by Lugaro (*loc. cit.*) in experimental lead poisoning in the cells of the cerebral cortex and spinal ganglia of the dog. In diphtheria a considerable accumulation of dark pigment occurs in the nerve cells of the human spinal cord.

The occurrence of pigmentation is associated with the occurrence of chronic chromatolysis.

The progress of pigmentary degeneration cannot be described apart from the other changes which are taking place simultaneously. As the pigment increases in amount and assumes pathological proportions, a slight change takes place in its colour. Normal pigment is pale yellow in colour, while the pigment present in excess is slightly darker, its colour varying from a rich amber to an almost brown shade.

The increase in the amount of pigment is accompanied by other changes, viz., displacement of the nucleus, swelling of the cell, and the disappearance of the chromatophile elements. The cell may ultimately present the appearance of a globule filled with pigment. It is removed by way of the lymphatics either free or enclosed in leucocytes. In unstained sections the pigment may be traced along the peri-vascular lymphatics. In its progress along the lymphatics it appears to become darker in colour.

The neuroglia forms the supporting framework of the central nervous system. It consists of cells with numerous long processes which in the spinal cord run mostly on a longitudinal direction. Whether it is of mesoblastic or epiblastic origin is not decided. It is usually considered to be of mesoblastic origin, but lately, among others, Eurich (20) has endeavoured to prove its epiblastic origin. The question is raised here simply to state that its origin is disputed and dismiss it because it does not enter into the scope of the present enquiry. The neuroglia is under certain morbid conditions, capable of proliferation, and may along with migrated leucocytes perform

phagocytic functions. Leucocytic infiltration takes place owing to the presence in the tissues of the products of disintegration. The leucocytes appear first in the adventitial sheaths of the vessels.

Proliferation of the neuroglia takes place where the degeneration has affected the nerve cells only. In the later stages of a myelitis where reparative processes are in progress, proliferation of neuroglia generally predominates over the leucocytic infiltration.

Case I.

This case was undoubtedly one of so-called "peripheral neuritis" due to arsenical poisoning. The cord was examined in the cervical, dorsal and lumbar regions.

The meninges in the *cervical region* are deeply congested. There are no hæmorrhages and the walls of the vessels are normal. There is no infiltration of the meninges with leucocytes. In the lymphatics, between the fibres of white connective tissue composing the meninges there is a deposit of brownish-yellow pigment.

The white matter of the cord is apparently normal, except for the distended vessels passing through it. There are no hæmorrhages in the white matter.

The grey matter is the seat of several changes. The blood-vessels are congested, and all the capillaries are injected. The walls of the vessels show no sign of previous pathological change. There are a few hæmorrhages in the anterior horns and the neighbourhood of the central canal. Leucocytes are present in small numbers in the peri-vascular lymph spaces.

Of the large motor cells of the anterior horns a certain number, approximately one-sixth, show marked changes. The altered cells are swollen, and their outline is not distinct. Together with this there is loss of protoplasmic processes. The protoplasm of the cell body is in some cases beset with fine vacuoles.

The nucleus stains less deeply and is displaced from its normal position in the centre of the cell, towards the periphery.

The chromatophile elements in the altered cells are diminished in amount. They are present in normal form at the periphery of the cell, but in the centre of the cell and around the nucleus they have undergone chromatolysis and are scattered throughout this region in the form of fine powder.

In the most anterior part of the anterior horn are a few cells which stain very deeply with methylene-blue.

Very few cells are atrophic and denucleated.

The pigment in the degenerate cells is increased in amount, and darker in colour than normal. It may be found in the peri-vascular lymphatics, free or enclosed in cells.

The neuroglia around the degenerate cells is increased in amount and the degenerate cells are eroded by the overlying proliferous

neuroglia cells. These "scavenger" cells lie in facets in the nerve-cells.

Besides the local increase in the neuroglia around the degenerate nerve cells, there is a general increase of it throughout the white matter. Instead of the uniform distribution of neuroglia cells throughout the grey matter, they are in places present in groups of four or five and apparently proliferating.

The central canal of the cord is dilated, but the lining epithelium is intact. The central canal contains fine fibrils of fibrin with a few (not more than two or three in each section) leucocytes entangled in its meshes.

The same changes are present in the *Dorsal* and *Lumbar regions* of the cord. In the dorsal region, in addition to the above-mentioned changes, there are hæmorrhages in the meninges in the region of the ventral fissure.

In the cervical and upper dorsal region the changes are more marked in the antero-lateral group of cells.

Case II.

Patient, a female, aged 42 years, suffering from arsenical poisoning. The immediate cause of death was pneumonia. In this case the brain was examined.

The arachnoid and pia mater are altered. The arachnoid is normally non-vascular and consists of fine meshes covered with endothelium and enclosing lymph spaces. The arachnoid in this case contains small groups of red corpuscles, not enclosed in the vessel walls, showing the arachnoid to be the seat of hæmorrhages. The arachnoid also contains multi-nucleated leucocytes, some of which carry granules in their protoplasm.

The blood-vessels of the pia mater are slightly altered. Taking for examination an arteriole in the pia mater, a few leucocytes are seen migrating through the vessel wall: a few are present in the peri-vascular lymph space. The arteriole is distended with blood and some red blood corpuscles are present in small numbers outside the vessel walls.

The veins of the pia mater are distended with blood. The hæmorrhages from the veins are larger than those from the arteries.

In the cerebral cortex the capillaries are greatly distended with blood. There are small extravasations of blood around them, and leucocytes are present in the vessel walls in small numbers and in larger numbers in the adventitial lymph sheaths.

The cellular layer of the cerebral cortex, except for the dilated vessels passing through it, is normal.

The small pyramidal cells stain better than the large pyramidal cells. Some have undergone the same alterations as the large pyramidal cells.

The large pyramidal cells are more satisfactorily examined.

A large number of these are swollen and vesicular, their borders are convex, instead of concave as they normally should be. The outline of some cells are convex instead of concave, their nuclei stain faintly with hæmatin, and are situated near the periphery of the cell.

The cells in which these alterations of structure are pronounced are accompanied by leucocytes or proliferating neuroglia cells lying alongside or on top of them.

In other pyramidal cells the changes are more pronounced; the protoplasm of the cell is vacuolated and the nucleus, if present, is with difficulty distinguishable. These degenerate cells are invaded by leucocytes which erode them.

Stained with methylene-blue, the cells in which the changes are least marked show chromatolysis around the nucleus, the chromatophile elements persisting at the periphery or in the processes, where these are present. In other cells the nucleus is displaced and the chromatophile elements are entirely absent from the cell.

None of the cells stain deeply and show signs of regeneration.

The pigment in the cells is increased in amount and darker in colour than normal.

Dorsal Cord. The meninges are congested and the vessel walls normal. There is a deposit of pigment in the lymphatics of the meninges, but no infiltration with leucocytes and no proliferation of the connective tissue cells. The white matter of the cord is apparently normal. Its blood-vessels participate in the general congestion of the cord. The grey matter is the seat of the well-marked changes. The blood-vessels are engorged, especially in the region of the anterior horns and the central canal. The vessel walls are normal and there is no thrombosis.

About half of the large nerve cells of the anterior horns show more or less deviation from the normal. This deviation is manifested by swelling of the cell, giving it a convex outline, tortuosity, or complete loss of the protoplasmic processes. The chromatophile elements in many cells have undergone chromatolysis, around the nucleus which is often displaced. The chromatophile elements around the periphery are arranged in rounded blocks. There is no vascuolation.

A few cells may be found which have undergone an extreme degree of degeneration. They are denucleated, much attenuated, and contain a relatively large amount of pigment, some even giving the impression of a cell wall containing nothing but pigment.

The degenerate cells are eroded by leucocytes and the surrounding neuroglia is proliferous.

In unstained sections pigment may be found free as granules, or enclosed in leucocytes. It is seen to follow the course of the perivascular lymphatics.

The cells lining the central canal appear to be proliferating. Examinations of the cord in the *Lumbar regions* showed it to be

the seat of changes similar to those found in the dorsal region. The number of cells of the anterior horns which had undergone some retrograde changes, was, by reason of their greater number, more accurately determined. The degenerate cells, counted in several sections, averaged 50 per cent.

Case IV.

The brain and medulla of this case were examined. The part of cerebral cortex examined was from the upper end of the right ascending frontal convolution.

Brain. The meninges are normal except for a slight deposit of pigment in them. They are not infiltrated and there are no hæmorrhages. The *cellular layer* shows no change. The vessels are evident by the deep staining of the endothelial cells.

The layer of small pyramidal cells is not without evidence of change. The changes which some of the cells have undergone are change in shape due to swelling whereby the angular character of the cell is lost. Loss of processes contributes still more to the altered character of the cells. They stain faintly with nuclear stains, while in other cells which have undergone degeneration to a further extent, the nuclei are absent, the cell protoplasm has a granular appearance due to the presence in it of a number of small vacuoles. In more advanced cases they may be a large vacuole in the cell.

The changes in the large pyramidal cells are of the same character as those in the small pyramidal cells. The number of normal cells is noticeably diminished. The changes are more easily seen because of the larger size of the cells. In addition to the changes noticed in the small pyramidal cells there are changes in the chromatophile elements. In many cells the chromatophile elements are absent, while in others they are strictly limited to the periphery of the cell. The nucleus of these cells is often displaced from the centre, though it is rare to find a cell in which it actually bulges.

Except around the degenerate cells, where it is proliferating, the neuroglia is not altered.

The blood-vessels are apparently normal. Their walls are not the seat of disease.

The changes in the *medulla* closely resemble those in the cerebral cortex. In the grey matter the vessels are moderately congested. About 50 per cent. of the motor cells are altered in the sections examined. The changes which the degenerate cells have undergone are briefly "chromatolysis" around the nucleus, displacement of the nucleus from the centre of the cell and loss of processes. The neuroglia is increased.

Case V.

The brain and medulla of this case were examined.

The brain was examined in the region of the ascending frontal and ascending parietal lobules in the upper part.

The meninges are almost normal. The blood-vessels are normal and the only abnormality is a slight deposit of pigment in the lymphatics.

The cellular layer is normal.

The small pyramidal cells are slightly diminished in number; of those which are present, some are rounded and swollen and some are vacuolated.

The majority of the large pyramidal cells are altered in structure. Minor changes are present in most of the cells. The nuclei stain faintly with nuclear stains; the nuclei are excentrically placed; the chromatophile elements have undergone partial or complete chromatolysis. Where the chromatolysis is partial it occurs around the nucleus, the chromatophile elements persisting at the periphery of the cell. The cell processes are frequently absent, and if present they have variocosities on them. In other cells the changes are more pronounced. They have lost their nucleus and their shape, and a few are present which are barely recognizable as nerve cells. The pigment in the nerve cells is slightly increased. In addition to the marked diminution in the number of normal cells, the total number of nerve cells, normal and abnormal, is diminished.

A few nerve cells are large and stain deeply with methylene-blue.

The neuroglia gives evidence, by the close approximation of neuroglia cells, of proliferation. This change is marked around the degenerate nerve cells, especially in the deeper layer of the cortex (*i.e.*, the layer of large pyramidal cells).

The blood-vessels of the cortex are congested. The adventitial lymph sheaths of the vessels are dilated and contain leucocytes.

The condition of the *medulla* closely resembles that of the cerebral cortex. The blood-vessels of the meninges are well filled but show no structural change. In the meninges is a slight deposit of pigment, but no infiltration and no proliferation of the cellular elements.

In the grey matter, about half the cells show some degree of change pointing to the occurrence of degeneration. The slightest changes are peri-nuclear chromatolysis and eccentric position of the nuclei. The more pronounced changes are complete loss of nuclei and vacuolation of the cells, together with an increase in the amount of pigment, which is darker in colour than normal.

A few cells are enlarged and the chromatophile elements stain deeply.

Case VI.

In this case an examination was made of the brain, the spinal cord in the cervical and lumbar enlargements, and of the ganglion on a posterior root from the dorsal region.

Brain. The meninges are moderately infiltrated with leucocytes, but the blood-vessels are not congested.

The cellular layer of the brain is normal.

The small pyramidal cells have undergone changes in shape due to swelling and loss of processes. Many have lost their chromatophile elements as such, *i.e.*, they no longer preserve their normal arrangement in rows parallel to the periphery and do not stain deeply.

Similar but more obvious changes have taken place in the large pyramidal cells. Many are rounded and vesicular. Their processes are twisted or have varicosities on their course. Their nuclei are eccentrically situated, and in some cells stain so faintly with nuclear stains that they are with difficulty recognised. In some cases the nucleus bulges on the periphery of the cell. The protoplasm of the cells in which these changes are most marked is frequently vacuolated. The vacuoles in all cases are small.

The chromatophile elements have undergone solution around the nucleus, or throughout the whole cell.

A few cells contain in their interior an excess of pigment, but this is not a constant feature even for the cells which show signs of advanced degeneration. The neuroglia is increased generally, and especially around the cells which show signs of degeneration. The neuroglia cells and leucocytes are exerting their phagocytic function, for many of the cells are eroded at several points. Leucocytes are present in small groups in the peri-vascular sheaths, and this is constant for all the layers of the cortex and for the meninges.

The spinal cord in the cervical region shows changes very similar to those found in the brain. The blood-vessels are congested and a few small hæmorrhages have occurred into the meninges. There is a peri-vascular infiltration with leucocytes and a deposit of pigment in the lymphatics.

In the cord itself there is general congestion, and this is most marked about the anterior horns. There are no hæmorrhages and no disease of the vessels. Of the motor cells of this region about 25 per cent. vary in some respect or other from normal. The changes which are most obvious are the well marked central chromatolysis and swelling of the cells. These are accompanied by a remarkable increase in the pigment of the cell and a displacement of the nucleus. The increase in the pigment has in cases attained such proportions that some cells appear to be mere globules of pigment, *i.e.*, a cell outline containing pigment and no other recognizable structures. Free pigment is present in some positions which seem to have been the site of a nerve cell, and in unstained preparations it can be seen lying in the peri-vascular lymphatics, which are occupied by small aggregations of leucocytes.

The neuroglia cells are increased.

The lumen of the central canal is obliterated and the lining epithelial cells have proliferated.

This description applies equally to the appearance of sections of the cord through the lumbar enlargement.

The appearance of a *section of a ganglion on the posterior root* is remarkable. All the blood-vessels are intensely congested.

The connective tissue is increased in amount. It is unusually rich in cells, the connective tissue cells apparently proliferating.

The nerve cells contain a remarkably large amount of pigment, which instead of being pale yellow, is in colour a rich amber, and in one or two cells where it is present as large granules, the colour verges on brown. Pigment of the same kind is present in comparatively large amount in the lymphatics, free or enclosed in cells.

Where the pigment is in excess the chromatophile elements are diminished.

The chromatophile elements of the cells of the posterior ganglion have a reticular arrangement with thickenings at the nodes of the reticulum. The cells which are altered are subject to the changes which occur in the ganglion cells of the anterior horn. The chromatophile elements are absent from the immediate neighbourhood of the nucleus. The nucleus is displaced to the periphery of the cell and the cell protoplasm is vacuolated.

Leucocytes appear to be attacking and eroding the cells which have undergone the changes just described.

Case VII.

This case is one of so-called "peripheral neuritis" in chronic arsenical poisoning. The brain, upper part of the medulla and the lumbar cord were examined.

Brain. The meninges contain scattered granules of pigment, which, however, is rather scanty. The blood-vessels contain blood, are not over-distended, and are normal in structure. There are no hæmorrhages and no leucocytic infiltration.

The cellular layer is everywhere normal.

The small pyramidal cells are mostly normal. A few contain excess of pigment and some are eroded by leucocytes.

A large proportion of the large pyramidal cells are normal. Some, however, show signs of degeneration, viz., faintly staining nuclei rendering the outline of the nucleus abnormally indistinct and the contrast between nucleus and nucleolus less evident than normal. Some cells are swollen and rounded and have lost their processes. The pigment is increased and is present in inverse proportion to the chromatophile granules. Some free pigment is found with several neuroglia cells in the immediate vicinity which have taken it up. This points to the total disintegration of nerve cells.

In certain cases the nucleus is displaced, and though the nuclear network is preserved the body of the nucleus has not cleared as well as in normal brain cells.

The chromatophile elements are diminished around the nucleus and are present in the form of a fine powder.

The achromatic substance is faintly stained. The protoplasm of the body of the cell is vacuolated but the vacuoles are extremely small. This feature is not a very prominent one.

The blood-vessels of the cortex are more congested than those of the meninges. The peri-vascular spaces are dilated and contain multinuclear leucocytes.

The medulla is not much affected. The meninges are unchanged. The ganglion cells have undergone changes which are slight, amounting to no more than peri-nuclear chromatolysis and faint staining of the achromatic substance.

Lumbar region of the cord. The meninges are congested. White matter is normal. In the grey matter the blood-vessels are congested especially in the neighbourhood of the anterior horns. There is slight infiltration of leucocytes around the congested vessels. The walls of the blood-vessels are not altered.

The ganglion cells are in many cases unchanged. In this region the most obvious change is an increase of pigment. Except for this alteration and the slight diminution of the chromatophile elements which accompanies it, many cells would appear normal. The lymph spaces are dilated in the grey matter and especially in the region of the anterior horns and central canal.

Case VIII.

Case VIII. was a female, 30 years of age. The nervous symptoms were very marked. She was mentally weak. The spinal cord was examined in the cervical and lumbar regions.

Cervical cord. Meninges congested. The congestion has gone so far as to lead to hæmorrhage. The hæmorrhages are very minute and are situated in the neighbourhood of the anterior fissure.

The cells of the grey matter are altered in about 25 per cent. of the total number in a single section. The altered cells are swollen, and their shape is consequently altered. Their nuclei are displaced, and the chromatophile elements have undergone solution in the centre of the cell. Simultaneous with the diminution of the chromatophile elements is an increase in the pigment of the cell. The protoplasm of the body of the cell is vacuolated.

The neuroglia cells are increased in number around the degenerate nerve cells. In addition to this there is a slight general increase in the number of neuroglia cells of the grey matter.

The blood-vessels are congested. There are no hæmorrhages in the substance of the cord. Leucocytes have collected in the lymph sheaths of the vessels. The vessel walls are normal.

The central canal is dilated, and the lining epithelial cells are proliferating.

Lumbar Cord. The blood-vessels of the meninges are deeply congested, but there are no hæmorrhages. There is a slight deposit of pigment in the meninges. The white matter is normal. The blood-vessels of the grey matter are congested. The peri-vascular lymph spaces are dilated, especially in the neighbourhood of the anterior horns and the central canal. This feature is so marked that it gives the section a cribriform appearance.

As in the cervical region, the motor cells are altered in about 25 per cent. of the whole cells visible in a single section. The remainder have undergone varying degrees of change. The cells are rounded and have lost some of their protoplasmic processes. The nuclei of the cells are excentric and the chromatophile elements absent from the centre of the cell and the immediate neighbourhood of the nucleus. The pigment is increased, and present even at the base of the processes, where it is normally absent.

Of the cells regarded as normal some stain very deeply with methylene-blue. The chromatophile elements take up the stain deeply, and appear as large oblong blocks. These are rather larger than the normal aggregation of chromatophile elements. This is the only respect in which they vary from the normal. They are possibly cells which are undergoing repair.

Cells in which the other signs of degeneration are most pronounced are vacuolated. The spaces in which the degenerate cells lie are in most cases much larger than the contained cell.

The neuroglia is proliferating, and there are collections of leucocytes in the adventitial lymph sheaths of the vessels.

The central canal is dilated.

Case IX.

Patient was a male, aged 61. The cord was examined in the dorsal and lumbar regions.

Dorsal region. The blood-vessels of the meninges contain blood, but are not distended. In many of the vessels the tunica intima is separated from the tunica media, and a large space intervenes between the two layers, except at two points where the intima remains attached to the media. The vessel walls are otherwise normal; there is no infiltration or proliferation. There is a slight deposit of pigment in the lymphatics of the meninges. The pigment is in the form of dark, rounded granules.

In the white matter the axis cylinders stain normally with hæmatein. The neuroglia is normal in amount and regularly disposed. In the white matter are numerous wide spaces, in most of which a contracted capillary or arteriole lies. These spaces are most marked in the region near the central canal.

In the grey matter the blood-vessels are much the same as in the white matter, but the dilatation of the peri-vascular spaces is more marked. The nerve cells are scanty, rounded, some denucleated

and without processes. Very few appear normal. The cells when stained by methylene-blue or thionin-blue, appear to be altered. The chromatophile elements have undergone solution immediately around the nucleus where the achromatic substance is stained a diffuse blue. The nucleus is frequently displaced.

The peripheral zone contains granules which stain with methylene-blue, but the granules have not the definite parallel or stichochrome arrangement of the normal cells. The granules are arranged as rounded blocks. These blocks are less numerous than normal.

A few cells of the anterior horn stain very deeply, but are not increased in size like those undergoing regeneration as described in animals some time after section of a nerve). The pigment is slightly increased. The nucleolus is occasionally vacuolated.

Lumbar enlargement. The meninges and white matter are in the same condition as those in the dorsal region.

In the grey matter the dilated lymphatics are a very marked feature. The infiltration around the blood-vessels is slight. The cells of the grey matter are rounded, convex in outline with faintly staining nuclei. Some are overlaid with leucocytes. Changes of a degenerative character are observable in about 75 per cent. of the nerve cells.

From some cells the nucleus has completely disappeared. In the cells in which the changes have not advanced so far the signs of degeneration are peri-nuclear chromatolysis, reduction in the quantity of the chromatophile elements, increase in the amount of pigment and excentric position of the nucleus.

The neuroglia of the grey matter is not regularly disposed. In many places small groups of three or four neuroglia cells are found close together, almost in apposition. The central canal is not distinct. The lining epithelium is proliferating.

Case X.

The patient was a woman, aged 44 years. She had well-marked signs of so-called "peripheral neuritis." She was mentally deficient, and died of cardiac failure.

Brain. The blood-vessels of the meninges are moderately distended with blood. The pia arachnoid is infiltrated with leucocytes, some of which enclose pigment.

The cellular layer is normal. The blood-vessels are congested. The small pyramidal cells are not noticeably altered. Some few contain pigment, but they are normal in number and retain their normal shape.

The large pyramidal cells are not so numerous as normal. They are swollen and rounded. Their nuclei are either excentrically placed, stain faintly with nuclear stains, or entirely absent. They contain excess of pigment. The chromatophile elements are diminished or absent entirely, so that the cells stain diffusely with methylene-blue. The chromatophile elements, if present, are

situated at the periphery of the cell. The cells have lost their protoplasmic processes and are eroded by leucocytes and neuroglia cells.

The neuroglia cells are increased by proliferation around the degenerate nerve cells.

There is an increase in the number and size of the giant cells of Betz, in the deeper layers of the cerebral cortex. The blood-vessels are congested, and leucocytes have collected in the peri-vascular lymphatics.

The sections were cut from the upper end of the right ascending frontal convolution.

Medulla. The meninges are congested and slightly infiltrated with leucocytes. The lymphatics contain pigment. The structure of the blood-vessels is unaltered. There are no hæmorrhages. The motor cells show pronounced changes; they are swollen, and the chromatophile material is limited to a peripheral ring. The nucleus is displaced, and in some instances protrudes on the surface.

A few cells stain very deeply.

The blood-vessels are congested, especially in the grey matter in the neighbourhood of the nerve cells which have degenerated, and the adventitial lymph sheath contains aggregations of leucocytes.

Lumbar Cord. The meninges are as in the medulla. The white matter is normal except for the passage of congested vessels through it. The grey matter is deeply congested, and there is œdema around the vessels, but this is not so marked as in Case IX. The large motor cells are normal in about 75 per cent. of those visible in one section. The rest show central chromatolysis, with displacement of the nucleus and proliferation of the neuroglia cells immediately around. The pigment is normal in amount. The cells lining the central canal are proliferating.

Case XI.

A special interest attached to this case, which occurred in 1902 at Halifax. Apart from its general interest (the report of it evoked a leader in the *Lancet*) it has a special interest in this place because the material obtained from it was examined in the fresh state. The material examined in the preceding cases, as before mentioned, remained for some weeks in the hardening fluid before it was examined microscopically. In this case the *post-mortem* examination was made on February 6th. The spinal cord was placed at once in 4 per cent. solution of formalin, and remained in it for three days. The hardening was completed in successively increasing strengths of alcohol, and it was immediately embedded in celloidin. Examination of the sections showed that widespread changes, exactly resembling in character the changes found in the preceding cases, were present. The cord was examined in the cervical and lumbar regions. The appearances were similar, and

the following description of the lumbar region applies equally to the cervical region :—

The meninges are deeply congested, and are the seat of not inconsiderable extravasations of blood. They are not infiltrated with leucocytes. In the lymphatics are small deposits of pigment (not derived from the blood). In addition to their distension the blood-vessels are slightly altered in structure. Here and there in the large blood-vessels endothelial cells can be found which are swollen and project into the lumen of the vessel.

The white matter is not obviously altered.

The grey matter, on the contrary, is unmistakably altered and to a considerable extent. All the blood-vessels are strongly evident by reason of their congestion. The blood-vessels are not tortuous, nor is their lumen narrowed or occluded. Changes in the endothelial cells of the intima are only found in the larger vessels of the meninges. The peri-vascular spaces of the vessels are dilated, but do not contain aggregations of leucocytes. This œdema, however, is not a very obvious change, as it is in Case IX. The motor cells are diminished in number. Very few are absolutely normal in appearance. The less apparent changes are: slight diminution in the amount of the chromatophile elements. In more advanced cases the nucleus is excentric in position and the nucleolus vacuolated. The chromatophile elements form a ring at the periphery. In some of the cells the nucleus actually bulges on the surface of the cell (see Fig. 1). The pigment of the cells is increased and situated at the pole opposite to the displaced nucleus. Very few cells have retained their normal angular character. They are almost all swollen and rounded, and have lost their processes. Where the chromatophile elements have undergone solution in the centre of the cell the achromatic substance stains deeply with methylene- or thionin-blue. The cell protoplasm contains small vacuoles.

In advanced cases the cell is seen to be globular, denucleated and filled with pigment. The chromatophile elements are usually entirely absent. If they persist they are extremely scanty and exist as fine grains scattered throughout the cell body.

The neuroglia is possibly increased, but not enough to warrant a definite assertion on this point.

The cells lining the central canal are proliferating.

For control experiments the brain and spinal cord of a patient who had had no nervous disease, and whose age was 50, were examined. (It was not possible to obtain the brain and spinal cord of a patient who died as the result of accident.) They were hardened in exactly the same manner as the organs from cases of arsenical poisoning. On examination it was found that the changes found in the corresponding organs of cases of arsenical poisoning were absent. The vascular changes were absent, the cells were normal in shape,

had retained their processes, and their chromatophile elements had the normal arrangement (Fig. II.) They were consequently regarded as normal, and constantly used for reference to avoid errors of interpretations.

The part of the brain examined in all cases was the upper portion of the ascending frontal convolution and ascending parietal convolution, an area round the upper end of the fissure of Rolando, and roughly corresponding to the "leg area."

The white matter of the cord was not examined by methods specially devised to show degeneration.

Causation of Changes in the Brain and Spinal Cord.

Passing in review the former cases one cannot fail to be struck by the almost monotonous regularity with which the same changes are found. In no case were the signs of degeneration entirely absent, although the extent of the degeneration varied with each case. In addition to this, one or other feature is specially pronounced in some of the cases, *e.g.*, Case II. is characterised by widespread and advanced degeneration of the nerve cells and by extreme degree of congestion. In Case VI. the pigmentation of the nerve cells and the deep congestion of the cord are the most prominent features, the others being overshadowed but not absent. In Case VII. the changes are slight and require a more careful search to discover them. In Case VIII. there is some œdema, but it is trifling in comparison with that in Case IX.

The changes described above, which are constant to the whole series are those typical of degeneration and inflammation, it being one of the canons of Pathology that noxious influences which cause degeneration in a delicate and susceptible tissue cause proliferation in a more resistant tissue. In this connection Ziegler (21) may be aptly quoted:—"Inflammation is frequently caused by . . . . degeneration, since the more intense local degenerations are apt to be combined with exudate processes and in the later stage with hyperplastic proliferation. It is thus impossible to draw a sharp line between simple degenerations and inflammations, and accordingly the term myelitis has been applied both to processes that are characterised by inflammatory exudation and to others that begin as degenerations and only in the later stages of their course are combined with morbid exudation and proliferation." We are then justified in describing the changes found in the spinal cord as myelitis. In searching for a possible cause for this myelitis we are unfortunately met with a serious difficulty. All the patients died of general wasting or of lung complications, both of which are in themselves capable of causing degeneration in the central nervous system. But where the degenerative changes are so constant in character one is justified in asking for a cause which is common to the series.

The recognised causes of degeneration are:—

1. Marasmus or general wasting.
2. General anæmia.
3. Disuse.
4. Local disorders of circulation—ischæmia—caused by:
   (*a*) Arterio-sclerosis.
   (*b*) Hyaline degeneration of the blood-vessels.
   (*c*) Thrombosis.
   (*d*) Embolism.
   (*e*) Hæmorrhage by rupture or by diapedesis.
5. Poisons produced in the body in the course of many diseases:
   Diabetes.
   Pernicious anæmia.
   Syphilis.
   Chronic tuberculosis and many other bacterial diseases.

   Poisons introduced from without:
   Ergotin.
   Arsenic.
   Lead.
   Mercury.
   Alcohol.
6. Trauma.
7. Variations of atmospheric pressure.
8. Elevation of temperature (as shown experimentally).

All the patients were slightly or much emaciated, but in Case XI., where the changes were well marked, the patient was fairly well nourished, although he was said to have lost flesh just before his death.

General anæmia as a possible cause of degeneration is not difficult to dispose of. The blood in seven of the cases was examined by Dr. Muir. The results were:—

	Hæmoglobin.	Red cells.	White cells.
Case I.	... 112 per cent.	... 6,075,800	... 4,560
Case II.	... 111 per cent.	... 4,727,600	... 6,050
Case III.	... 93 per cent.	... 4,843,700	... 20,000
Case V.	... 112 per cent.	... 5,200,800	... 12,690
Case VI.	... 91 per cent.	... 5,017,800	... 3,690
Case VIII.	... 75 per cent.	... 4,915,100	... 4,310
Case X.	... 71 per cent.	... 4,553,500	... 25,890

It will be seen from the above table that so far from the blood being poor, in many cases it is exceptionally rich in hæmoglobin and red cells.

Disuse is not a common factor in the possible causes of degeneration in the above cases.

In examining the specimens, no local disorders of circulation are to be found. The vessels are healthy and normal in structure. In Cases I. and II. hæmorrhages are found. The causes of hæmorrhages in the spinal cord are classified by Leyden (22) according to their etiology thus: —

(*a*) Arterio-sclerosis.
(*b*) Trauma.
(*c*) Diminished pressure of air (Caisson disease).
(*d*) Spontaneous (arising from physical exertion or suppressed menstruation).
(*e*) Intoxications.

Of these the first four can be immediately dismissed. The fifth possible cause of hæmorrhage comes in group 5 of the causes of degeneration.

The group of poisons as possible causes of degeneration cannot be dismissed so summarily. It is unlikely that all the patients had either diabetes or syphilis and there is no record of symptoms of these diseases during life, nor of syphilitic lesions being found post-mortem. The brain and spinal cord are certainly not the site of local syphilitic lesions.

Tuberculosis remains as a feature nearly common to all the series except Cases IV. and XI. Case IV. had old fibrous adhesions which were not definitely said to be tubercular. Case XI. had a thickened pleura which was calcified at one spot. There were no tubercles in the lungs or on the peritoneum. Five of the cases had widespread tubercular lesions, *i.e.*, in the lungs, on the pleura and on the peritoneum. Of the eleven cases, eight had active tuberculosis, one had healed phthisis at both apices with "signs of renewed activity," and two had lesions which might have been due to tuberculosis at a previous date, but no signs of active tuberculosis were found post-mortem. Hence, tuberculosis can scarcely be considered to be quite constant throughout the series. On the other hand, as a complication of pulmonary tuberculosis "peripheral neuritis is not common" (Osler (23)). If "peripheral neuritis" were common in tuberculosis it would assume epidemic proportions all the year round, and we may assume that whatever the rôle of tuberculosis in causing degeneration of the cells of the cord, it cannot be regarded as the cause of the nervous symptoms present in each case during life. As tuberculosis and degeneration of the cord are so constant in the series, it may be that they have a common cause. Tuberculosis is not the cause of the degeneration. In support of this theory is the fact that in ten cases there were at the time of death active pathological changes in progress in the lungs and that in five of these there was unmistakable evidence of

the spread of tuberculosis to other organs, in particular the peritoneum. This evidence of the invasion of other organs than the lungs by tubercle bacilli finds its most satisfactory explanation assuming that the resisting power of the tissues has been lowered and that tuberculosis is a "terminal infection." An attempt will be made in Part II. to show that degeneration of a similar kind can be produced experimentally in cases where it is possible to exclude tuberculosis altogether.

Of the poisons which may be introduced from without, arsenic and alcohol were common to the series. The patients all took alcohol in large quantities at the time when beer contained arsenic in considerable proportion. The action of alcohol alone, on the central nervous system is, according to Berkley (24), to produce changes in the vessels. The changes are: swelling of the endothelial cells of the intima, denucleation of some of the cells of the media, and very little change in the adventitial coat of the vessels. The vessels become tortuous and have small aneurysmal dilatations on their course. Degenerative changes in the nerve cells are always consecutive to local vascular changes. He never found degeneration except in the immediate neighbourhood of vessels that were occluded by plugs of fibrin or swollen endothelial cells. Although it is impossible in this place to exclude alcohol altogether, against alcohol as the cause of the changes may be adduced the indirect evidence that although the public continue to take alcohol in the form of beer in large quantities, yet, since public attention was directed to the contamination of beer by arsenic and the contamination removed, the cases of "peripheral neuritis" have markedly decreased.

The other causes of degeneration: Trauma, etc., were not operative in a single case. The balance of the evidence then is on the side of arsenic, but whether the toxic effect is increased by its association with alcohol cannot be definitely asserted as the result of examination of the human cases.

One point in connection with the changes caused by arsenic is worthy of discussion: What is the fate of the nerve cells which have undergone degeneration? It has been shown experimentally by Marinesco (25) and others that the nerve cell undergoes chromatolysis as the result of section of the nerve processes. The changes can be observed as early as the third day after section of the processes, and reach their height from eight to ten days later. These experiments were made on animals. The hypoglossal nerve on one side was divided or torn across in several animals. By killing the animals at varying lengths of line after division of the nerves, and examining the hypoglossal nucleus on the side of the divided nerve, the above results were arrived at. By allowing the animal to live as long as 105 days the degenerate cells in the nucleus of origin had recovered so completely that it was impossible to distinguish between the hypoglossal nuclei of the two sides.

In the human cases many of the cells had undergone changes so

profound that it seems impossible that they could recover. Where the cells have lost their nuclei, have undergone diffuse chromatolysis and are filled with pigment, it seems unlikely that they can recover, and in fact in many cases they are being attacked by phagocytic cells and are in process of being removed. Free pigment, too, exactly resembling that of nerve cells may be found in the peri-vascular lymphatics—that is probably the result of the total disintegration of nerve cells. Whether the loss of nerve cells is ever made good by proliferation of the remaining cells cannot be definitely asserted, but in the series of human cases no evidence of such proliferation was found in a single case. It is therefore reasonable to admit that the changes in the nerve cells were not secondary to change in the nerves.

The central canal of the cord is in one case dilated and contains fibrils of fibrin with one or two leucocytes in the meshes. The canal in the other cases is surrounded by several layers of cells which seem to have resulted from proliferation of the lining epithelium. The dilatation of the central canal may be due to exudation, for pathological exudations are richer in fibrin than normal lymph or cerebro-spinal fluid. The proliferation of the cells is interesting because of the close developmental relation of the cells of the epidermis to the cells of the central canal—both are epiblastic in origin. The action of arsenic on the skin is well-marked. Proliferation of the cells lining the entral canal, has been noticed by M. M. Buichi et Varnali (26) in arsenic poisoning.

Deductions.

The changes observed in the above series of cases indicate:—

1. That the degeneration of nerve cells and inflammatory reaction in the spinal cord and brain were due to a poison circulating in the blood and not to local vascular changes.

2. That the poison was not produced in the body by any pathological process, and that in all probability it was contained in the arsenical beer which had been taken by all the patients.

3. That the efficient cause of the degeneration of nerve cells was either alcohol or arsenic, probably the latter.

4. That the degeneration of the nerve cells may proceed to such an extent as to lead to their total destruction.

PART II.

Experimental Poisoning.

To supplement the observations made on the human cases a number of experiments were made on rats. The rats were kept on a low diet and were given arsenious acid in watery solution alone, or mixed with alcohol. The purpose of the experiments was to imitate as closely as possible the conditions under which arsenic was taken

by the human subjects and also to vary the amount of the constituents so as to determine if possible the relative effect of alcohol and arsenic. Two of the rats F. & G. took arsenic without alcohol.

Symptoms. It was not possible to produce by experimental poisoning symptoms of local paralysis. It is admittedly difficult in any case in animals to determine the existence of paralysis. The most noteworthy symptom was drowsiness. Just before death the animals sometimes became fierce and aggressive. They took not unkindly to beer, even when it contained arsenic. All lost weight during the time they were taking arsenic. The details of the doses and duration of experiments are given in each case.

Case A.

A rat weighing 117 grammes was given Salford arsenical beer containing $^1/_6$th grain of arsenious acid per gallon. The quantity of beer given, corresponded to what 2 gallons daily would have been to a man of average size (140 lbs.). In the course of 102 days the rat took 1372 c.c. of arsenical beer containing arsenious acid to the total amount of 0·003430 grammes or $^1/_{20}$th grain. At the end of that time the animal was kept on ordinary food and water or arsenic-free beer and then killed. The above quantity of arsenical beer was given in 93 doses, equal to about $^1/_7$th of the body weight of the animal, which would correspond to about 2 gallons of beer daily for a man of average size. The original weight of the animal was 117 grammes. The weight at death was 78·3 per cent. of the original weight.

The spinal cord was examined in the cervical region. The vessels of the meninges are normal in structure. They are congested. The white matter of the cord is normal. The vessels of the grey matter are congested. There is œdema around the larger vessels and aggregations of leucocytes in the peri-vascular lymph sheaths. The nerve cells of the grey matter stain faintly with nuclear stains. The nucleus is not often eccentric and does not react normally to staining reagents. The substance of the nucleolus is in some cells dispersed throughout the nucleus. The cells lie in spaces which are considerably larger than the contained cells. The cells are connected to the walls of these spaces by tendril-like processes giving the impression of the cells having shrunk and retained their connection at several points. The protoplasm contains numerous minute vacuoles. The chromatophile elements are present. Where chromatolysis has occurred it is diffuse. It does not occur regularly in the centre of the cell as in the human cases. A marked feature is the presence of "scavenger cells" around the altered nerve cells.

The posterior root ganglion comes in the plane of section. It is deeply congested and a few of the ganglion-cells have undergone diffuse chromatolysis.

Case B.

The rat, which originally weighed 190 grammes, took, in the course of 80 days, 0·0807000 grammes of arsenious acid. It was given Salford arsenical beer to which arsenic was added. It lived 89 days. Its weight at death was 71·5 per cent. of its original weight. The medulla and upper region of the cord were examined.

Medulla. The meninges are slightly congested and slightly infiltrated with leucocytes. The vessels of the grey matter are slightly congested. The nerve cells are altered in shape. The processes are blunt or absent. Between the processes the outline of the cells is convex. Some cells have no nuclei. Their protoplasm is vesicular, and has a "bubbly" appearance. The ragged outline of some of the cells is due to erosion by "phagocyte" cells. The neuroglia is slightly increased. There is some infiltration with leucocytes around the capillaries.

Spinal Cord. Dorsal region. The anterior horns appear attenuated. The blood-vessels of the meninges are slightly congested. There are a few small hæmorrhages. The motor cells are reduced in number. The surviving cells are shrunken and dwindled. Their outline is irregular and ragged, and the processes are absent. The protoplasm is vacuolated, and the nuclei stain faintly. The cells, especially those of the anterior cornua, are extremely wasted, and in many the greater part of the cell is occupied by a large vacuole, which pushes the chromatophile elements aside. The chromatophile elements may retain their parallel arrangement; more frequently they are dispersed throughout the protoplasm in fine grains.

The ganglion on the posterior root is the seat of intense congestion and numerous hæmorrhages. The ganglion cells are less markedly altered.

Case C.

A rat, weighing originally 181 grammes, was given bitter beer to which 10 per cent. alcohol was added and arsenious acid 7 grains to the gallon. Death occurred after 35 days from poisoning. The total arsenic taken in that time was 0·0176000. Weight at death was 67 per cent. of the original weight.

The cord was examined in the cervical region.

The meningeal vessels are normal in structure, deeply engorged with blood, but there are no hæmorrhages. There is evidence of migration of leucocytes from the vessels.

The white matter is streaked with congested vessels, the walls of which stain rather deeply with nuclear stains.

The grey matter is more deeply congested than the white. All the capillaries are closely packed with blood-corpuscles. There is no evidence of infarction or arteritis. There are several trifling hæmorrhages; a few groups of red cells are found not enclosed in

vessel walls. The motor cells are altered in shape, and their outline is ragged. Their processes are distorted, their nuclei placed excentrically and their protoplasm vacuolated. The cells of the anterior horn stain more deeply than the others. They contain large vacuoles and the protoplasm between the vacuoles retains the stain with great tenacity. The parallel arrangement of the chromatophile material is retained in the protoplasmic processes.

Some cells, especially in the antero-lateral group, are uniformly stained, and the chromatophile elements in these cells are in the form of a fine powder. The cells are vacuolated, but the vacuoles are small.

Case D.

This animal was underfed. It took 634 c.c.'s of $\frac{1}{6000}\frac{1}{0000}$ watery solution of arsenious acid administered in 64 daily doses. Total amount of arsenious acid taken was 0·0045 grammes or 0·069 grains. This would correspond to about 43 grains of arsenious acid taken in 64 days by a man of average weight (140 lbs.). During the last ten days 5 per cent. of pure ethylic alcohol was added to the solution. The animal died of poisoning at the end of 65 days.

The spinal cord was examined in the dorsal region.

The blood-vessels of the meninges contain blood, and there is slight leucocytic infiltration around them. There are no hæmorrhages.

The white matter is normal.

Grey matter. The anterior cornua seem slightly atrophied. All the cells stain faintly with hæmatein. They are distorted in shape, and their processes are lost. Some cells have no nuclei, but contain in their protoplasm dark granules which look like fragments of nuclei. The outline of the cells is frequently indistinct and eroded. The cell protoplasm is vacuolated, and the chromatophile elements are irregularly disposed in one or two large blocks between the vacuoles or scattered as fine powder throughout the cell. The grey matter is infiltrated with leucocytes.

Case E.

The same as D, only 5 per cent. of alcohol (absolute ethylic) was added to the fluid. The arsenic was given during the first 18 and last 10 days of the experiment (28 days out of 64). The total amount of arsenic administered was therefore proportionately less in this case than in D. On the other hand, the amount of alcohol given was much more considerable. The original weight of the animal was 113 grammes. At death it weighed 69 per cent of the original weight. The total weight of arsenic taken was 0·0031950 grammes (in daily doses during 65 days). Killed on the 66th day.

The meningeal vessels are filled; structure normal. There are some small hæmorrhages.

The white matter where it adjoins the grey matter is slightly infiltrated with leucocytes.

In the grey matter there are very few normal cells. The cells stain indifferently with nuclear stains, and lie in spaces much larger than the contained cell. In every section a few spaces are discoverable which are empty and have probably been occupied by nerve cells. The surviving cells are rounded, and have lost their processes. Their protoplasm is vacuolated. The nucleus is in some cases swollen (relatively to the size of the cell); in others it is irregular and shrunken. The nucleus of a nerve cell is frequently pale and indistinct in outline. The altered cells are eroded by leucocytes. In the less altered cells the chromatophile elements retain their parallel arrangement in the protoplasmic processes. With advancing degrees of degeneration the chromatophile elements are disposed as irregular blocks throughout the cell, usually persisting longer at the pole opposite to the axis cylinder, and later the chromatophile elements are visible as a fine powder, and the nucleus is situated at the periphery of the cell. The capillary vessels are dilated. Their walls are normal. Around some of the capillaries leucocytes are aggregated in small groups.

Case F.

A rat, weighing 189 grammes, took in 81 days 0·0632000 grammes of arsenic in alcohol-free beer. It lived 91 days, and death occurred from poisoning. At death it weighed 58·1 per cent. of its original weight.

The spinal cord was examined in the lower third of the cervical region and in the upper dorsal region. The condition in the two regions was exactly alike.

The meningeal vessels are congested, and there are a few small hæmorrhages.

The white matter is normal.

The nerve cells of the grey matter are well preserved. They are large, and their processes are in a good state of preservation. Their nuclei stain fairly well with nuclear stains. However, there are some cells which are attenuated, which have short blunt processes, and of which the nuclei stain faintly and are excentrically placed.

The blood-vessels are distended with blood, and there are a few hæmorrhages. There is some infiltration with leucocytes around the vessels.

Case G.

A rat weighing 168 grammes was given a $\frac{1}{10000}$ solution of arsenious acid in water. The arsenic was taken during 61 days. The animal lived 91 days and took altogether 0·0320960 grammes of arsenious acid. Died of poisoning. Weight at death 57 per cent. of original weight.

Upper part of dorsal cord examined. Meningeal vessels engorged.

There are hæmorrhages in the neighbourhood of the nerve roots. The connective tissue about the seat of hæmorrhage has undergone hyaline degeneration.

The white matter is normal. Blood-vessels congested.

The grey matter is characterized by marked vascular changes. All the vessels are distended with blood and in the neighbourhood of the grey commissure and the anterior cornua the congestion has led to hæmorrhage. The capillary walls stain deeply and the endothelial cells are swollen and numerous.

The nerve cells are small, stain faintly, have lost their nuclei and processes. The protoplasm contain large vacuoles. The processes where present appear twisted and varicose. Leucocytes are found in apposition to the degenerate nerve cells.

The outstanding feature of the experimental series is that the changes are more prominent and wide spread than in the human series. In addition, the changes are not exactly of the same type. Chromatolysis in the centre of the cell is not present, nor is the eccentric position of the nucleus so constant, unless it is pushed to the periphery by vacuoles in the centre of the cells. Vacuolation is a striking feature in the experimental series, but in the human series a large vacuole was seldom found. The circulatory changes are more prominent in the experimental than in the human series; the hæmorrhages in particular being more numerous and larger. In one case G, the endothelial cells forming the capillary walls seemed swollen and increased in number beyond possibility of error of observation. When comparing the results of experimental poisoning with those of "beer poisoning," it must be remembered that the rats took doses which translated into terms of their body weight were very large.

It is exceedingly difficult to convey by means of a description an idea of the relative degrees of pathological change. As several factors in the experiments vary, all the cases do not admit of comparison.

Cases.	Vehicle.	Days during which Arsenic administered.	Length of life days.	Total arsenic taken in grammes.	Weight at death in % or original weight.
Case A.	Salford Arsenical Beer	93	102	0·0034300	78·3%
Case B.	Beer	80	89	0·0307000	71·5%
Case C.	Beer or 10% alcohol...	35	35	0·0176000	67·0%
Case D.	Water, alcohol last 10 days	65	65	0·0045150	54·5%
Case E.	Water + 5% alcohol ...	28	64	0·0031950	69·0%
Case F.	Alcohol-free Beer	81	91	0·0632000	58·1%
Case G.	Water	61	91	0·0320960	57·0%

Case A most closely resembles the human cases. Case B resembles Case A, except that the doses of arsenic administered were much larger. The changes are very marked in Case B. There is wide-

spread degeneration and small hæmorrhages in the cord and larger ones in the posterior spinal ganglion.

Case C stands almost alone because of the short time the animal lived. The total amount of arsenic taken was more than half as much as was taken by B, and during less than half the time. The conditions found in the cord were very intense inflammation and degeneration. Some of the cells were fairly well preserved, and this probably represents an early stage of the changes found in sections from other cases. F and G are very much alike, and the condition of the spinal cord was better in F than in G and better in B where the arsenious acid was given with beer containing alcohol, although F took twice as much arsenious acid as B or G.

All the animals lost flesh to some extent. This loss was least in Case A in which also the cord was least changed. The relation of loss of weight to degenerative changes in the cord does not hold throughout the series. Though F lost weight considerably more than D, the cord in F was much less altered. Emaciation therefore does not correspond in degree to the changes in the cord. During the experiments the animals did not suffer from any inter-current disease.

Deductions.

1. Arsenious acid alone is capable of producing degenerative changes in the nerve cells of the spinal cord.

2. Ceteris paribus the changes are greater with larger doses of arsenious acid.

3. Arsenious acid does not act by producing local vascular changes.

4. The changes produced in artificial poisoning by arsenious acid are independent of any other disease.

From the human cases it was concluded that the changes were of a constant kind, varying in degree only, throughout the series; that the changes were characteristic of degeneration and inflammation; that of the possible causes of such changes only three, arsenic, alcohol and tuberculosis, were entitled to a claim to be considered the essential cause of the degeneration; that of these three tuberculosis was not quite constant, being totally absent in one case, doubtful and certainly quiescent in another, and with "slight signs" of renewed activity in the third; that on the other hand, tuberculosis might better be explained as a "terminal infection" following diminished resistance of the tissue.

The experimental animals suffered from no disease and degenerative changes were constant in them all. What was the rôle of alcohol in the experimental series, the writer was unable to determine. The changes in the blood-vessels, said to be the cause of the degeneration produced by alcohol, were absent.

Conclusions.

1. Arsenious acid causes well marked degenerative changes in the cells of the spinal cord and of the posterior roots ganglia.

2. In chronic arsenical poisoning the rôle of the alcohol is not known. It is probably not the essential cause of the degenerative changes but it may be an adjuvant.

3. The degenerative changes found in the cord during chronic arsenical poisoning are not due to inter-current disease, nor to local vascular changes.

It remains to me to express my indebtedness to Dr. Judson Bury for the loan of books. His unique knowledge of the literature of "peripheral neuritis" was unfortunately only available at the eleventh hour.

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APPENDIX.

Case I.

P.K., male, aged 40. Ill since November, 1900. Coryza, nausea, pain in the calves and feet, later swelling of the legs and abdomen. Cough, hæmoptysis in December. "Pins and needles" in hands and feet, heat and numbness, loss of power. Skin darker since December. Admitted February 6th, 1901. Present condition (February 15th, 1901): well-marked peripheral neuritis, much ascites, large right pleuritic effusion. Deeply pigmented. Moderate pyrexia. Tapped right pleura; five pints. Died February 25th, 1901. (Dr. Muir.)

Post-mortem. Rather emaciated and deeply pigmented. *Heart:* Right-sided dilatation; atheroma of the aorta. *Lungs:* No tubercle, double pneumonia (immediate cause of death, which was impending from exhaustion). *Pleura:* Numerous small tubercles with fibrinous adhesions and serous effusion, both sides. *Peritoneum:* Numerous small tubercles, much matting together of intestines, ascites. Dr. Moore considered this a direct extension from the pleura through the diaphragm, which was much affected. *Stomach:* Gastritis with submucous hæmorrhages. *Kidneys:* Congested. *Supra-renals:* Apparently normal. *Spleen:* Large, soft, congested. *Liver:* Mixture of fatty degeneration and cirrhosis; congested. *Pelvic organs:* Normal. *Cervical glands:* Deeply pigmented. *Brain:* Normal.

Case II.

C.McD., female, aged 42. History of illness since September, 1900. Nausea, pains and numbness of hands and feet. Abdomen swollen for three months. Pigmentation for six months. Admitted January 18th, 1901. Present condition (January 30th, 1901): Deeply pigmented; marked peripheral neuritis, ascites, temperature normal. Died February 12th, 1901.

Post-mortem. Rather emaciated, deeply pigmented. *Heart:* Small, with soft flabby walls. *Lungs:* Healed phthisis left apex, showing slight signs of fresh activity; pneumonia right apex, red hepatization, with immediate cause of death which was impending from exhaustion. *Pleura:* Numerous small miliary tubercles and adhesions, both sides. *Peritoneum:* Early tubercular peritonitis, much ascites. *Liver:* A mixture of fatty degeneration with slight cirrhosis, which Dr. Moore (Pathologist to the Manchester Royal Infirmary) considers very characteristic of arsenical poisoning. *Kidneys:* Chronic granular, moderate degree. *Spleen:* Slightly enlarged. *Pelvic organs:* Normal. *Supra-renal bodies:* Normal to naked eye inspection. *Cervical glands:* Deeply pigmented.

Case III.

J.H.C., male, aged 44. Cough for four months. Numbness, "pins and needles," and weakness of legs for six weeks. Admitted February 12th, 1901. Present condition (February 14th, 1901): Deeply pigmented; well-marked peripheral neuritis. Temperature raised, generally 100°–101°.

Post-mortem. Emaciated, deeply pigmented. *Heart:* normal, early atheroma of the aorta. *Lungs:* Extensive phthisis with cavitation left side, early phthisis right side. *Pleura:* Left side, thick, large serous effusions with fibrinous adhesions and scattered tubercles. A few tubercles on right side. *Peritoneum:* Ascites, some small and early tubercles. *Kidneys:* Normal. *Adrenals:* Normal in appearance; one feels hard to the touch. *Spleen:* Not remarkable. *Liver:* Small cirrhotic. *Pelvic organs:* Normal. *Cervical glands:* Deeply pigmented. Death from exhaustion.

Case IV.

Patient, a female, aged 52. Occupation, hawker. Was a beer-drinker, and used to get drunk every Saturday night. Was an inmate of Withington Workhouse.

Post-mortem. Skin slightly pigmented about mammæ; much subcutaneous fat. Old pleural adhesions; base of both lungs congested and consolidated. Chronic bronchitis; muco-pus in bronchi. Catarrhal gastritis; slight hæmorrhages in walls of stomach. Both kidneys give evidence of parenchymatous changes; right, normal in size; left, small owing to deficiency of two lobules.

Case V.

Female, aged 46. Ill with vomiting, pains and tingling of hands and feet for six weeks before admission (December 4th, 1900). Present condition (January 31st, 1901): Marked peripheral neuritis ascites. Deeply pigmented. Irregular pyrexia ranging from normal to 102°. Phthisis left apex. Died February 13th, 1901.

Post-mortem. Rather emaciated, deeply pigmented. *Heart:* small, and fairly normal. *Lungs:* Phthisis left apex, with cavitation; congested. *Pleura:* Left pleura adherent. *Peritoneum:* early tubercular peritonitis, much ascites. *Kidneys:* Fairly normal, rather firm. *Liver:* Mixture of fatty degeneration and cirrhosis. *Pelvic organs:* Normal. *Brain:* Normal. *Cervical glands:* deeply pigmented. Death from exhaustion.

Case VI.

Female, aged 53. Ill since December, 1900. Coryza, vomiting and diarrhœa, pains in hands and feet, also "pins and needles." Admitted February 13th, 1901. Present condition (February 24th, 1901): Extensive broncho-pneumonia, probably tubercular. Irregular

pyrexia, ranging from 100° to 103·6°. Well-marked peripheral neuritis and erythro-melalgia of feet. Pigmented. Died March 7th, 1901.

Post-mortem. Moderately nourished and pigmented. *Heart:* Normal. *Lungs:* Small miliary tubercles, more at bases than elsewhere. No extensive consolidation, and no cavitation. *Pleura:* Small tubercles, no effusion. *Peritoneum:* A very few small tubercles. *Kidneys:* A few small tubercles. *Adrenals:* Normal. *Liver:* Slight fatty cirrhosis. *Brain:* Normal. *Cervical glands:* pigmented. Death from exhaustion and cardiac failure.

Case VIII.

Female, aged 30. Mentally weak. Admitted October 15th, 1900, with severe peripheral neuritis. Present condition (February 11th, 1901): Wasting paralysis and contractures of the legs, trophic changes. Wasting, loss of power and trophic changes in hands. Died March 31st, 1901.

Post-mortem. Emaciated. *Heart:* Small and soft. *Lungs:* Very extensive cavitation both sides, very foul. *Pleura:* Very thick and adherent, no tubercles. *Peritoneum:* Normal. *Stomach:* chronic gastritis. *Liver:* "Nutmeg," with some fatty change.

Case IX.

Patient, male, aged 61. Much emaciated and deeply pigmented; pericardial adhesions. *Heart:* Flabby, fatty degeration; necrosis of 6th rib. *Pleura:* Adhesions. Cheesy tubercles in right apex; consolidation of lower lobe of right lung. Collapse of left lung. Nutmeg liver.

Peri-nephritis, numerous caseous purulent foci (? tubercular) in both kidneys.

Case X.

Female, aged 44. Admitted October 2nd, 1900, with well-marked peripheral neuritis, slight pleuritic effusion (left). Mentally deficient. Present condition (February 5th, 1901): Wasting and paralysis of legs. Hands wasted, loss of use, trophic changes in skin. Died suddenly, February 24th, 1901.

Post-mortem. Heart: Slightly dilated, right side thin and soft. *Lungs:* Healed phthisis both sides, with some slight signs of renewed activity. *Pleura:* Adhesions on left side. *Peritoneum:* Normal. *Liver:* Fatty. *Adrenals:* Normal. *Stomach:* Numerous small hæmorrhages on the mucous membrane. *Brain:* Normal. *Cervical glands:* Not pigmented. Death from cardiac failure.

Case XI.

T. L., male, aged 54, lurryman. Admitted January 15th, 1902. On admission temperature normal. Skin deeply pigmented. "Pins

and needles" in hands and feet. Weakness of legs. Spastic gait. Lately feet had been blistered and painful. Voice husky. Temperature normal till pneumonia developed.

Post-mortem. Deep pigmentation of skin; œdema and keratosis of feet. *Pericardium:* "Milk-spots." *Heart:* Aortic valve incompetent; valve segments thickened, rigid and shortened. Fresh "vegetations." *Pleura* thickened from old pleurisy. Calcified spot on pleura of right lung. Fresh adhesions, easily broken down. Pneumonia of right upper lobe. *Peritoneum:* No ascites, no tubercle. *Liver:* Fatty. *Kidneys:* Interstitial nephritis. *Adrenals:* Normal. *Cervical glands:* Pigmented. *Brain:* Normal. Death from pneumonia.

The Nerve Cells of the Central Nervous System in Cases of Malignant Tumours.

BY

R. LAWRENCE, M.D., Ch.B. (Vict.).

The Nerve Cells of the Central Nervous System in Cases of Malignant Tumours.

THE purpose of this enquiry was to examine the Central Nervous Systems of patients dying of malignant disease. The cases were investigated irrespective of the site of the character of the primary growth—epithelioma or carcinoma—ulcerated or covered with skin.

All the patients whose Central Nervous System was examined died in the Christie Hospital. The microscopical work was carried out at the Public Health Laboratory.

Conditions under which the patients died.

None of the patients had symptoms of organic nervous disease. Many had been bed-ridden for some time preceding death but had not had paralysis. Twenty-five of the patients died after an illness characterised by progressive wasting and weakness and increasing inability to take food. Their illnesses were otherwise uneventful. In many of these, the organs, except those invaded by new growth, appeared to be normal. The others had small patches of broncho-pneumonia, which, however, did not seem sufficient to cause death. Of the other nine patients, one (No. 25) died within twenty-eight hours of an operation and had septic pneumonia in both lungs. Three others (13, 26, 34) died of septic pneumonia. Two died of hæmorrhage from large blood-vessels eroded by new growth. One patient (No. 32) had an epithelioma of the œsophagus. She was able to take only very little food, and may be said to have died of starvation. Two of the patients (Nos. 19 and 20) had uræmic convulsions shortly before death.

The average age of the patients at the time of death was forty-nine. Many had taken large doses of morphia for some time preceding death. Had the results of the microscopical examination of the spinal cords been less constant a search would have been made for any correlation between the changes found in the spinal cords and the amount of morphia taken.

Post-mortem features of the spinal cord.

The *post-mortem* examination was made in all but the last case within twenty-four hours of the death of the patient.

The spinal cords were removed after cutting through the laminæ of the vertebræ. The cord was raised by seizing the dura-mater with forceps after dividing the nerves with a knife.

The ganglia on the posterior roots of the spinal nerves were exposed by chipping away the pedicles of the vertebral arch. They were removed carefully by dissecting along the posterior spinal nerves without touching the ganglia with forceps.

There were no signs of disease in the spinal cords. The vessels were well filled with blood owing to their relatively dependent position. (The bodies lay supine on the mortuary table during the time which intervened between the death of the patients and the *post-mortem* examination.)

Post-mortem features of the brain.

Of all the brains examined none showed any abnormality at the time of examination. There was no sign of previous disease. The blood-vessels at the occiput were distended with blood. In one case (No. 30) there was a small osteoma of the frontal bone which depressed the underlying brain substance slightly.

The spinal cords were cut into transverse sections with a razor. These were about ·3 or ·4 cm. thick. They were at once placed in alcohol or a saturated solution of mercuric chloride in normal saline solution.

From the brains a thin transverse section was removed from the upper part of the ascending frontal convolution. This was put in alcohol or the mercuric chloride solution.

Methods.

The material used was put into the fixing fluid within twenty-four hours of the death of the patient except in the case of No. 34. In this case forty hours elapsed between the death of the patient and the fixing of the tissues used. The exact procedure was:—Fix in 75 per cent. alcohol, transfer to 90 per cent. alcohol for twenty-four hours, then to absolute alcohol; change the absolute alcohol twice. Transfer to chloroform until the blocks of tissue appear translucent, then to chloroform containing some paraffin in solution. Finally, embed in paraffin melting at 54°C.

In the other method, the thin slices of spinal cord or brain were placed in a saturated solution of mercuric chloride in "normal saline" solution. They remained in this for six hours, were washed in water for twenty-four hours, and then placed in 75 per cent. alcohol coloured with a few drops of Gram's iodine solution. This iodine solution was added until the colour was permanent. The pieces were then transferred to stronger alcohol until they reached absolute alcohol. They were subsequently soaked in chloroform and embedded in paraffin.

Serial sections of the spinal cords were cut and as many as six mounted on one slide so that a large number of nerve cells were available for examination. The sections were cut with a Minot microtome, working at "two teeth" for the spinal cords and brains,

and at "three teeth" for the posterior root ganglia and the semi-lunar ganglia.

The slides used were coated with a thin layer of egg albumen solution (1 in 10) and dried in the paraffin oven.

The sections were floated on warm water to spread them out flat. They were then placed on the slides and dried in the paraffin oven for several hours. By this means they were firmly fixed to the slide.

The paraffin was dissolved off with xylol, the xylol washed off with absolute alcohol, and the slide with its adherent sections placed for a few minutes in water.

Then the slide was placed in a simple watery solution of toluidin blue until the sections were overstained. The length of time of staining, and the strength of the solution of toluidin blue are not very important, as the sections have to be decolourised to the exact point for examination.

The sections were washed with absolute alcohol until the blue colour no longer comes away in clouds in the alcohol.

They were cleared in xylol and mounted in xylol-canada-balsam. Washing in a mixture of aniline oil and absolute alcohol was omitted. This step is unnecessary, and introduces additional chances of error.

In the earlier cases the spinal cords were examined in the Cervical Dorsal and Lumbar regions. Finding no difference in the state of the nerve cells in these regions the examination of the spinal cord in the later cases was confined to the Lumbar region. The nerve cells in the anterior horns of the Lumbar region are particularly numerous.

The study of nerve cells stained by this method specially aimed at the chromatic elements of the cells. Sections so stained do not show degeneration of the white matter of the cord even if it be present; but such features as congestion, inflammatory infiltration with leucocytes, if present, are readily observed.

Characters of nerve cells.

The *large cells of the anterior horns*, stained with toluidin blue, will be described first.

They are large in size and pyramidal in shape and have several processes. In normal cords, even in very thin sections, some processes can always be found very long. In abnormal, *i.e.*, degenerated cords, the cell processes often seem to be broken off. At any rate, they cannot be traced far from the cell. Other processes, of course, pass out of the plane of the section.

The outline of nerve cells of the anterior horn is concave between the points from which the processes arise.

A convex outline indicates swelling of the cell. The position of the nucleus is central. An excentric position denotes degeneration.

The nucleus may be so far displaced from its normal central position as to bulge on the surface. This abnormal position is usually associated with other signs of degeneration.

In hardened specimens, the nerve cell lies in a space which it does not completely fill. When, however, a nerve cell is swollen and rounded, it almost completely fills the space in which it lies.

In sections stained with toluidin blue the cell protoplasm is differentiated into two parts—one which takes the stain deeply, and another which stains very faintly or not at all. The stainable or chromophilic elements consist of granules scattered throughout the unstainable protoplasm. The granules have a fairly definite shape and arrangement. The coarser granules are angular, often spindle-shaped, and lie with their long axis parallel to the outline of the nerve cell. They are more conspicuous at the periphery of the cell than around the nucleus. The smaller granules are rounded and lie nearer to the centre of the cell. The arrangement of the coarse granules gives the cell a "striped" or "tigroid" appearance.

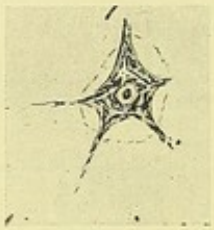


Fig. I.

These granules stain deep Prussian blue. They are absent from the axis cylinder process and the eminence from which this takes rise. They are present in the dentritic processes arising from the cell.

Pigment is present in the cells of the anterior horns. It is said to increase in amount with the age of the patient. It appears as golden-yellow granules.

The nucleus stains very faintly or not at all. The nucleolus stains uniformly a very dark blue—almost violet colour.

The *nerve cells of the posterior root ganglia* are large and round or oval in shape. The nucleus is centrally placed, and they have one cell process. As in the multi-polar cells of the spinal cord, the nucleolus stains deeply, the rest of the nucleus faintly or not at all, and the cell protoplasm consists of a stainable and an unstainable part. The stainable part appears as small granules regularly disposed throughout the protoplasm. Except at the periphery of the cells these granules are of small size. The cells have not the distinguishing "tigroid" appearance of the cells of the anterior horn of the spinal cord.

Changes in nerve cells.

The earliest changes are found in the chromophilic granules. These may break down into a fine powder which is scattered throughout the cell protoplasm, so that the cell appears to be diffusely stained. The breaking down of the chromophile granules is called chromatolysis. In extreme degrees of chromatolysis the chromophilic granules disappear from the cell body entirely. Chromatolysis is interpreted as a sign of degeneration of the nerve cell.

Other changes have been mentioned: displacement of the nucleus and swelling of the nerve cell.

The chromophilic granules are present in the dentritic processes of nerve cells. The presence here of these stained granules makes these processes visible. In degenerated nerve cells these processes cannot be traced.

Examination of a Section of Brain.

Case 24. The brain substance for examination was taken from the upper end of the ascending frontal convolution.

No macroscopic evidence of disease was found at the *post-mortem* examination.

Examined microscopically, the section showed no evidence of inflammation. The blood-vessels were normal. The cells of the superficial layer were stained blue.

The small pyramidal cells were stained blue and were angular in shape. On account of their small size, their protoplasmic contents were difficult to examine.

The larger pyramidal cells appeared normal. They were angular in shape, and their processes were well preserved. The chromophilic matter of the cell protoplasm appeared normal.

The large cells of Betz lend themselves for examination. They resemble the large cells of the anterior horns of the spinal cord, and the existence of changes in their minute structure is less open to difference of opinion than the existence of minute changes in the smaller cells of the cerebrum. These large cells of Betz were found to be normal. Their outline between the processes was concave; the nucleus was centrally placed, and the chromophilic material in the protoplasm was present in large blocks. The protoplasm between these blocks was clear and unstained. The striped or tigroid appearance of the cell was well-marked.

Examination of a Section of Spinal Cord.

Case 33 (P.M., book No. 79). Lumbar region of the spinal cord. A large number of sections were examined in series, and a large number of nerve cells were closely scrutinised with a view to finding degenerative changes.

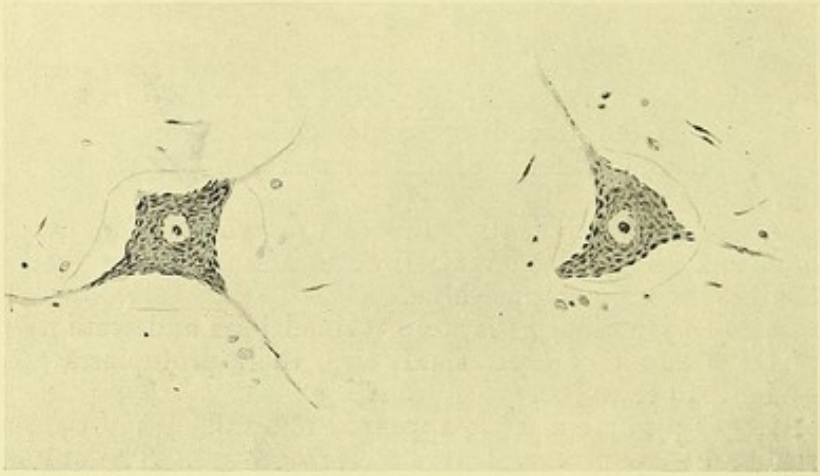
The general features of the cord are normal. The meninges are normal. There is no congestion of the blood-vessels, no inflam-

matory exudate from them, and no œdema. The gray matter of the cord contains many large cells. These stand out prominently by reason of this staining. The nuclei of other cells—neuroglia cells, epithelial cells lining the central canal, and endothelial cells of capillaries—retain the blue stain, but the other structures are decolourised.

The nerve cells have their normal angular shape, with long nerve processes and a concave outline between the processes. They are not swollen, and do not fill up the space in which they lie.

Their nuclei are centrally placed.

The chromophilic granules are normal—large, angular, and well-defined. They give the nerve cells a striped appearance. There is no sign of chromatolysis.



Case 30 (76).

Fig. II.—Hardened in corrosive sublimate. Stained with toluidin blue. Two nerve cells normal in appearance.

1. Angular outline.
2. Long dendritic processes.
3. Centrally situated nucleus.
4. Chromatophile granules.
5. Nerve cell does not completely fill the space in which it lies.
6. Neuroglia cells and nuclei of endothelial cells of capillary vessels

The dentritic processes are stained for some little distance from the cell.

There are no aggregation of leucocytes and no signs of proliferation of neuroglic cells around the nerve cells.

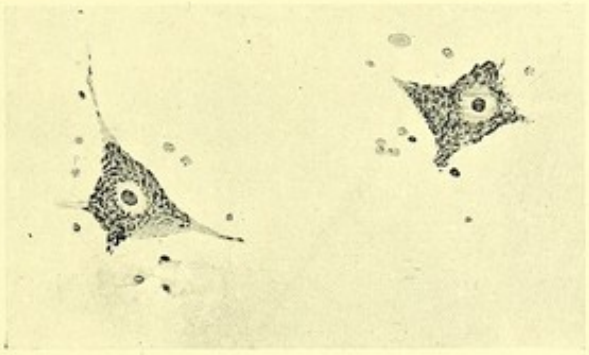
In the large number of cells examined, one was found which had a rounded outline, less than the usual number of processes, an excentrically-placed nucleus, and chromatolysis of its granules. The chromatolysis was not extreme. The granules at the periphery of

the cell were clear and distinct, but around the nucleus they had disappeared, and the protoplasm stained uniformly a pale blue colour.

The semi-lunar ganglia and the ganglia on the posterior roots of the spinal nerves were examined. In a single section of these a large number of nerve cells are available for examination. They were found to be normal. Their chromophilic granules were distinct and their nuclei centrally situated.

An earlier case (No. 20), re-examined for comparison with the later cases. Cord at the region of the lumbar enlargement. Meninges normal. No congestion of blood-vessels and no signs of inflammation.

The cells of the anterior horn were stained blue. They were



A B

Case 31 (77).

Fig. III.—Corrosive sublimate; toluidin blue.

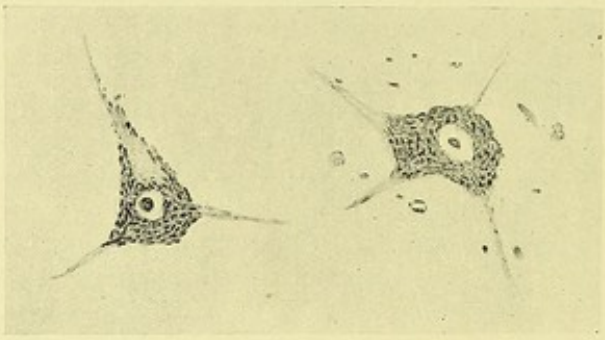
A. A perfectly normal nerve cell.

B. The chromophilic material not quite so clear as normal.

angular in shape, not swollen, had well-marked dentritic processes and a centrally-situated nucleus. The cell protoplasm was occupied by blue-stained granules of large size giving the cell the usual tigroid appearance. The protoplasm between the granules was clear and unstained.

The cells did not fill up the spaces in which they lay. In the supporting tissue around, there was no change. The condition of this spinal cord exactly resembles the condition of the spinal cords later in the series.

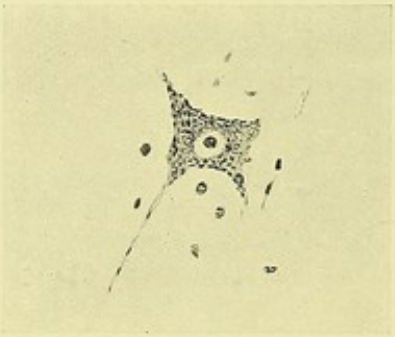
To describe in detail more cases would be to multiply the above descriptions. Instead, illustrations are given of the nerve cells from four consecutive cases. The nerve cells were examined with a Reichert No. 7A objective and a Zeiss No. 2 eye-piece.



A B

Case 32 (78).

Fig. IV.—Corrosive sublimate; toluidin blue. Two nerve cells. In *A* there is an absence of chromophilic material from the axis cylinder process.



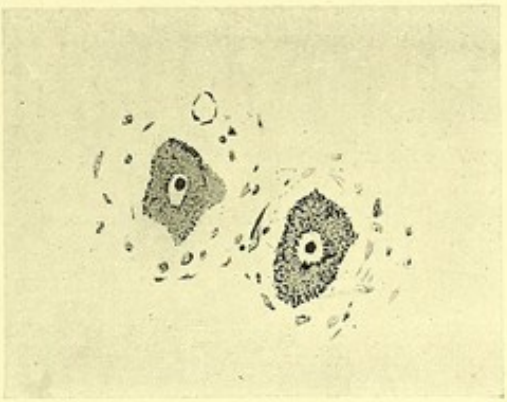
Case 32 (78).

Fig. V.—Corrosive sublimate; toluidin blue. Normal nerve cell.

Case 33 (79)

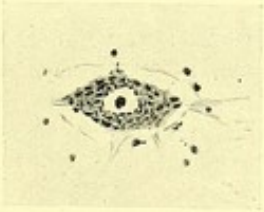
Fig. VI.—Corrosive sublimate; toluidin blue. Degenerated nerve cell.

1. Nucleus displaced
2. Chromatolysis
3. Swelling of the nerve cell
4. Processes are not well-marked



Case 33 (79).

Fig. VII.—Corrosive sublimate; toluidin blue. Normal nerve cells from the spinal ganglia.



Case 33 (79).

Fig. VIII.—Corrosive sublimate; toluidin blue. Normal nerve cell.

A B

Case 34 (80).

Fig. IX.—Corrosive sublimate; toluidin blue.

A. Marked degenerative changes: rounded outline, loss of processes, excentric nucleus, chromatolysis.

B. Normal shape. Chromophilic granules not clear owing to diffuse staining of the "unstainable" protoplasm.

Effects of Post mortem changes.

Case 34 (80). More than forty hours elapsed between the death of this patient and the time when the spinal cord was placed in the fixing fluid.

General features were normal.

The nerve cells examined were numerous. They were angular and had long and well-marked dentritic processes. Their nuclei were centrally placed, but were faintly tinged with violet.

The chromophile granules were stained blue, but were not so clear and sharply defined as normally they are. In addition, the "unstainable" protoplasm had taken on the stain, though much less deeply than the chromophilic granules. This feature of the staining gave the cells a blurred appearance. (Fig. IX. B.) This is the only departure from the normal. The other features of normal nerve cells—shape, long processes, and centrally placed nucleus—were present.

Almost all of a large number of cells examined answered to this description.

One was found in which the nucleus was displaced from the centre to the periphery, and the centre of the cell stained diffusely while the granules at the periphery stained clearly. (Fig. IX. A.)

The sections of the semi-lunar ganglia and the posterior root ganglia showed that the small chromophilic granules had almost entirely disappeared, and the cells had stained diffusely a pale blue colour.

In some of the cells there was distinct displacement of the nucleus to the periphery.

Evidences of secondary degeneration.

Cases 25 and 26 were examined by Marchi's method. The cords were first placed in 4 per cent. formalin until they were hard enough to allow very thin slices to be cut from them. These thin slices were placed in Marchi's fluid for a week, washed in water, and finally placed in 75 per cent. alcohol. They were transferred to increasing strengths of alcohol until they reached absolute alcohol. From alcohol they were transferred to cedar oil and then to paraffin. Sections were cut in paraffin.

No degeneration was found in the white matter of the cords.

Conclusions.

Almost all the cells of the central nervous system are normal in cases dying of malignant disease.

The few abnormal cells found are probably not more numerous than those found in any case after death. (It is said that in every case a few degenerated cells will be found if the spinal cord be examined by this method of staining.—Ewing: *Medical Record*, April 9th, 1898.)

Appendix.

APPENDIX: SUMMARY OF CASES EXAMINED.

1. (P.M. book, No. 2.) J.F.D., female: age 47. Carcinoma of left breast—recurrence after operation. Secondary deposits in liver; jaundice. Brain and spinal cord examined.
2. (4.) M.W., male: aged 58. Epithelioma of œsophagus. No pneumonia. Spinal cord examined.
3. (6.) W.S., male: age 44. Epithelioma of tongue. Secondary deposits in cervical glands. Broncho-pneumonia of right lung. Brain and spinal cord examined.
4. (7.) W.P.G., male: age 60. Epithelioma of lip. Secondary deposits in cervical glands. No pneumonia. Spinal cord examined.
5. (11.) J.P., female: age 51. Carcinoma of breast—recurrent. Secondary deposits in almost all organs of body. Brain examined.
6. (17.) R.M., male: age 53. Epithelioma of skin of neck; metastases in cervical glands and lungs. Broncho-pneumonia. Brain examined.
7. (22.) W.F., male: age 70. Carcinoma of rectum. Metastases in lymphatic glands and liver. Brain examined.
8. (25.) L.C., female: age 48. Carcinoma of breast. Secondary deposits in lymphatic glands, brain, liver and lungs. Brain and spinal cord examined.
9. (26.) A.O., male: age 61. Epithelioma of larynx. Broncho-pneumonia. Brain and spinal cord examined.
10. (29.) C.C., male: age 45. Epithelioma of floor of mouth. Deposits in cervical glands. Broncho-pneumonia. Brain and spinal cord examined.
11. (33.) J.M., male: age 32. Carcinoma of rectum. Metastases in lymphatic glands, lungs, liver and skin. Brain and spinal cord examined.
12. (34.) A.H., female: age 63. Carcinoma of breast. Metastases in glands, pleuræ and diaphragm. Brain and spinal cord examined.
13. (35.) W.K., male: age 56. Epithelioma of tongue. Metastases in cervical glands. Death from pneumonia. Brain and spinal cord examined.
14. (38.) E.W., male: age 47. Epithelioma of scrotum. Deposits in inguinal glands. Death from hæmorrhage. Spinal cord examined.
15. (39.) A.W., female: age 64. Epithelioma of clitoris. Brain and spinal cord examined.

16. (40.) E.G., male: age 48. Endothelioma of neck. Tuberculosis of lungs, intestines and liver. Brain and spinal cord examined.
17. (41.) J.P., male: age 48. Epithelioma of larynx. Spinal cord examined.
18. (45.) H.B., female: age 56. Carcinoma of breast. Secondary deposits in glands, lungs, adrenals and kidney. No pneumonia. Brain examined.
19. (46.) H.Y., female: age 40. Carcinoma of uterus. Broncho-pneumonia. Uræmia. Brain and spinal cord examined.
20. (50.) J.T., male: age 60. Epithelioma of floor of mouth. Secondary growths in glands, liver and bones. Brain and spinal cord examined.
21. (51.) W.M., male: age 34. Epithelioma of tongue. Deposits in cervical glands. Death from hæmorrhage. Spinal cord examined.
22. (52.) A.W., female: age 70. Carcinoma of breast. Secondary growths in lungs. Tuberculous pleurisy and peritonitis. Brain and spinal cord examined.
23. (54.) M.H., female: age 39. Carcinoma of breast. Secondary growths in glands, lungs, heart, adrenals and ovaries. Brain examined.
24. (60.) T.C., male: age 67. Carcinoma of rectum. Congestion of lungs at bases. Brain and spinal cord examined.
25. (70.) E.S., female: age 40. Carcinoma of cervix uteri. Secondary growths in pelvic and abdominal glands, liver, left kidney and lungs. Suppurative nephritis of right kidney. Broncho-pneumonia. Spinal cord examined.
26. (71.) M.H., female: age 45. Epihelioma of the œsophagus. Secondary deposits in the glands of the anterior mediastinum. Septic pneumonia of left lung. Spinal cord examined.
27. (72.) F.McM., female: age 27. Carcinoma of cervix uteri. Metastases in pelvic abdominal mesenteric and deep inguinal glands and in liver. Pneumonia of right lung. Spinal cord examined.
28. (74.) E.H., female: age 58. Carcinoma of left breast. Metastases in pectoral muscles, axillary glands and adrenals. Spinal cord examined.
29. (75.) J.W., male: age 40. Epithelioma of lip, with enlarged cervical glands. Died within 48 hours of operation for removal of glands. Broncho-pneumonia of both lungs. Spinal cord examined.
30. (76.) D.C., female: age 59. Carcinoma of cervix uteri. Secondary deposits in pelvic and lumbar glands. Hydronephrosis of right kidney. Uræmia. No broncho-pneumonia. Spinal cord examined.

31. (77.) M.L.C., female: age 52. Carcinoma of right breast—recurrence after operation. Metastases in skin, axillary glands, pleura, lungs, pericardium, liver, supra-renal glands and bone. Brain and spinal cord examined.
32. (78.) C.D., female: age —. Epithelioma of pharynx. No metastases. Broncho-pneumonia of middle lobe of right lung. Spinal cord examined.
33. (79.) M.A., female: age 50. Carcinoma of right breast; widely ulcerated; perforated chest wall; spread to right lung, pericardium, and left lung. Fatty degeneration and enlargement of the liver. Spinal cord examined.
34. (80.) J.R., male: age 61. Epithelioma of floor of mouth. No metastases. Broncho-pneumonia of lower lobe of right lung. Spinal cord examined.

Address on Some Points Concerning Human Intestinal Parasites.

BASED UPON THE RESEARCHES OF THE AUTHOR.

BY

EDOARDO PERRONCITO,

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Address on Some Points Concerning Human Intestinal Parasites.

BASED UPON THE RESEARCHES OF THE AUTHOR.

GENTLEMEN,—

I wish, in the first instance, to express to you how deeply I appreciate the honour which the Council of the Victoria University of Manchester has done me by inviting me to be present at the inauguration of the new Public Health Institute, erected in this celebrated town, and by conferring upon me the Degree of Doctor of Science. I have also much pleasure in addressing you upon one of my most favourite studies. Before beginning I desire to offer to this illustrious University and to my friend Professor Delépine my best wishes for the success of this Institute of Public Health.

Animal parasites are of special interest to England, whose immense possessions over the seas, endowed as they are with natural beauties of all kinds, offer in many instances conditions suitable for the development of many parasites which are the cause of numerous diseases.

Introduction.

There was a time when all diseases were supposed to be due to worms, and Varrone held that in marshy districts there lived minute animals which, when introduced into the organism, caused severe and often fatal diseases.

Modern researches have shown that these infinitely small, and often invisible* organisms or microbes, as they are now called, are not all animal, but that many of them belong to the vegetable kingdom.

Animal parasites belong to the Protozoa, Worms, Arachnides and Insects. These parasites have many biological and bio-chemical characters in common.

Effects of temperature upon animal parasites.

Owing to the properties of the essential constituent of animal parasites, viz., protoplasma, they do not resist a temperature exceed-

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*Delivered on the occasion of the opening of the Public Health Laboratory, and of the conferring upon the Lecturer of the Degree of Doctor of Science, *Honoris Causa*, of the University of Manchester.

*Microbes may, generally speaking, be divided into ultra-visible (or ultra-microscopic) and visible microbes.

ing 48° to 50° C. for more than five or ten minutes; this I was able to show by a long series of experiments begun in 1874 and continued till the year 1878. Most of these experiments have been referred to by my regretted colleague Spencer Cobbold in his works.

Animal parasites are killed by freezing. The eggs of insects may, as had already been shown by Reaumur, resist temperatures of −25° to −30°C., but animal parasites in their more advanced stages of development, for instance, cysticerci, larvæ of intestinal worms, etc., do not survive the freezing temperature.

These facts have important bearing upon hygiene, for cold is not only suitable for the preservation of food, but also for the destruction of many animal parasites.

Effects of fermentation and putrefaction.

A moderate amount of fermentation or decomposition of food (or of the contents of the intestine) may favour or even be necessary to the development of intestinal parasites, but prolonged putrefaction is fatal to them. This fact is of importance in connection with the changes taking place in cesspools or in sewage treated in various manners and used for the irrigation of land in the neighbourhood of large towns.

In this address I will deal only with those intestinal parasites which I have studied specially.

PROTOZOA.

Lamblia intestinalis (Blanchard, 1888).

In *children* and *adults*, as well as in various animals such as *rats*, *mice*, *rabbits*, and even *dogs*, one finds frequently a kind of Lamblia, also known under the names of *Cercomonas intestinalis* (Lambl, 1856), *Dimorphus Muris* (Grassi, 1879), *Megastoma entericum* (Grassi, 1882). This parasite is one of the *Flagellata* most frequently observed.

The Lamblia intestinalis is pyriform in shape, with a curved anterior extremity; it is provided with a deep groove looking like a sucker or mouth.

A median depression extends from the anterior to the posterior extremity or tail of this protozoon. Three or four pairs of flagella spring from the borders of the median depression and a last pair is attached to the caudal extremity. The length of this parasite is variable, usually 14μ to 15μ, it may reach 20μ and more. (Fig. 1.)

The Lamblia is usually found attached by its mouth or sucker to the epithelial cells of the duodenum or other parts of the small intestine. The posterior part of the parasite remains free and motile.

I have observed that when these parasites are numerous their presence is associated with intestinal troubles such as disorders of digestion, pains in the hypochondriac regions, constipation alter-

nating with diarrhœa, in the latter case the stools are pale and fœtid.

If the fluid fæcal matters are examined immediately after they have been evacuated the parasites are found in variable numbers and motile; their movements are very rapid when the material is kept warm. It is difficult to see the flagella distinctly, even when the preparation is stained with iodine dissolved in a watery solution of iodide of potassium. When the fæcal matters are soft, and sometimes even when they are fluid, one finds a variable number of peculiar translucent, spherical, or oval corpuscles, measuring from 10 or 12 to 17μ in diameter, besides the motile forms already described and their immobile chitinous skeletons.

These corpuscles are provided with an external membrane through which the contents, composed of flagellated cells, can be

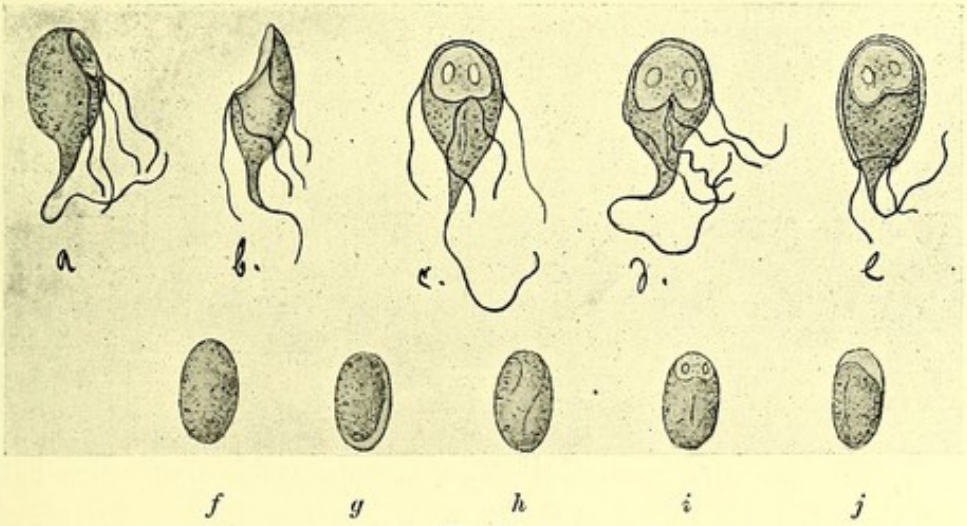

Fig. 1.

a, *b*, *c*, *d*, *e*, Lamblia intestinalis in different positions.
f, *g*, *h*, *i*, *j*, Encapsulated forms.]

seen. During the period of constipation these corpuscles are the only form of the parasite found in the fæces. By lengthy researches I have been able to show that these corpuscles are encysted or encapsulated Lamblias (Megastomas). My observations,* communicated to the Royal Academy of Medicine of Turin (13th May, 1887), have been confirmed since by Grassi and by others: they have proved that Flagellata found in the intestine of man and various animals become encysted and acquire the characters of durable spores which become the means of propagating the disease. The presence of perfect Megastomas in the fluid stools and their replace-

* E. Perroncito. Incapsulamento del Megastoma intestinale. Giornale della R. Academia di Medicina di Torino, 1887.

ment by the encysted form in the solid stools indicates the relation between the two forms of the parasite. When one studies the Megastomas of rodents (Mus decumanus, Mus rattus, etc.), it is possible to determine the parts of the intestine where the evolution of the parasite takes place. It is in the large intestine that, under ordinary conditions, the parasite becomes encysted; by the time it leaves the body in the fæces the cyst has generally an oval shape and is provided with a distinct translucent membrane through which flagella, nuclei, and other elements can be seen. When I described for the first time the cystic form of Megastomas in man I had already observed that this gradual encystment was taking place in the large intestine. The formation of cysts can only be completed when the contents of the intestine are not evacuated rapidly, *i.e.*, when there is constipation. When there is diarrhœa the parasites are eliminated in earlier stages of development.

Along with the Megastomas present in diarrhœal stools obtained either direct from the intestine or after evacuation during life, I have noticed the common cercomonas, very active and provided with two or more flagella. It has appeared to me that certain cysts resembling those of the Megastomas belonged to these parasites; they differ from the Megastoma cysts by their more irregular form and the greater convexity of one of their sides.

I have demonstrated experimentally that the encapsulated Megastomas are means of infection by taking four white mice (Mus Musculus) and mixing with the food of two of them human fæces containing encapsulated Lamblias, the two other mice being kept as controls. After seven or eight days the first two mice had already in their fæces a fairly large number of encysted Megastomas identical to those found in the human fæces. The two control mice, which were kept under observation for a considerable time, remained free from these parasites.

I have observed the transmission of the Lamblia intestinalis to the rabbit, and have frequently noticed among these animals cases of death for which no other cause could be found than the presence of this parasite. A rabbit that had died from this disease was sent lately to my laboratory; it came from a locality where other fatal cases had occurred. In this animal the lungs were in a state of diffuse emphysema, the stomach was distended with dry hard material consisting of bran and hay, the intestine, and specially the colon and rectum, were distended with gas. The droppings found in the rectum and colon were hard, dry, firmly adherent to the intestinal walls. The duodenum contained a large number of Megastomas similar to those found in man; some of these were attached to the intestinal epithelium by their "mouth." These parasites were found in large quantities along the whole length of the duodenum. In the large intestine the fæcal matter had taken, owing to prolonged constipation, the character of coproliths, and there the parasites had undergone typical encystment. I have also had the opportunity of

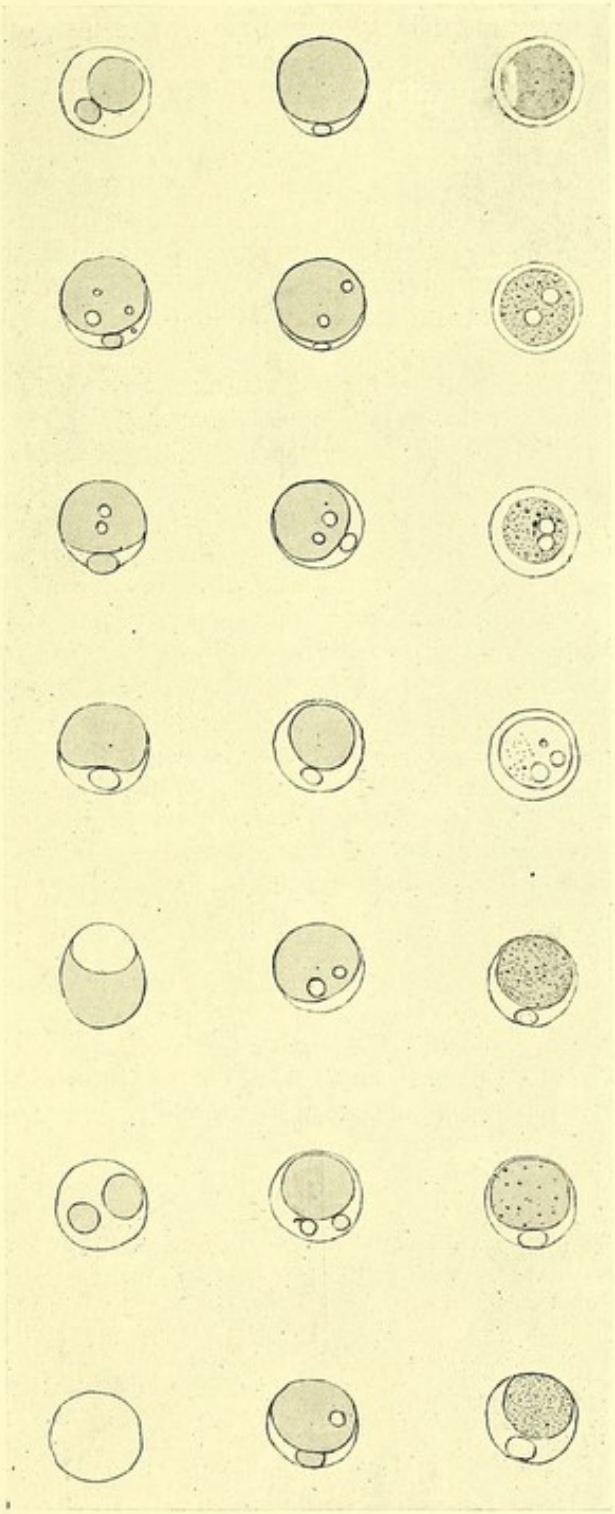

Fig. 2.
Coccidium jalinum.

observing Megastomas in large numbers in the fluid contents of the stomach, and in the hæmorrhagic catarrhal exudations present in the intestines of a dog which had exhibited epileptic and paraplegic symptoms.

Coccidium jalinum (n. Sp.)

I have observed this parasite several times in individuals suffering from enteritis; it has a peculiar translucent white or yellowish-white colour; it is sometimes reddish; its shape is spherical, measuring from 3 to 10 or 14 μ in diameter; it is provided with a nucleus.

Fig. 3.
Sarcocystis Miescheri.

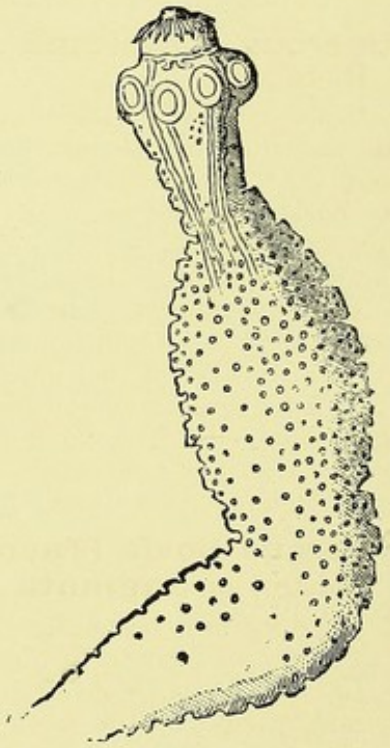

Fig. 4.
Cysticercus cellulosae, observed on the warm stage, during life.

This Coccidium is often surrounded by a granular barely visible halo, as shown in the figures illustrating my communication to the Société de Biologie de Paris, in 1899, on the occasion of its 50th anniversary (see also the last edition of my book, *I parassiti dell uomo e degli animali utili, Milano*, 1902, p. 138). This parasite has been observed by Dr. Prospero Demateis and by Dr. Borini in persons affected with severe chronic enteritis, and reduced by chronic diarrhœa and dysentery to a cachectic state.

It appeared to me that this parasite, which I have placed among

the Coccidia, might be a stage in the development of Miescher's or Rainey's tubes. *(Sarcocystis Miescheri* (Ray Lankester), *Synchytrium Mieschcrianum* (Kühn)). (Fig. 3.)

I have found it in great number in the colon of pigs, the flesh of which was also infected with Miescher's corpuscles. This view is supported by the fact that the Sporozoites contained in the corpuscles of Miescher when made to develop outside the tissues assume a form resembling that of the Coccidium jalinum. Further study is needed in order to settle this question.

CESTODA.

Cysticercus cellulosae (Taenia Solium) (Linnaeus) Rudolphi).

Owing to the application of hygienic measures, the two forms of this parasite have become less common than they were both in man and in animals. Almost everywhere meat is inspected and measly pork is destroyed or submitted to various processes such as cooking or salting.

The mature deutoscolex (Cysticercus) is killed in five minutes by exposure to a temperature of 48° to 50°C.; in less than 24 hours by the action of a weak solution of chloride of sodium. The parasite dies naturally in meat in from 30 to 40 days. The preservation of meat by refrigeration is therefore a source of security when sufficiently prolonged. The danger of infection through the eggs of the parasite is increased when, as sometimes occurs, there is more than one Tænia Solium in the intestines of one individual.

Cysticercus bovis (Taenia Mediocanellata (Küchenmeister) or **Saginata** (Göze).

This tapeworm is becoming proportionally more frequent than the Tænia Solium on account of the difficulty of seeing its cysticercus in beef. The *cysticercus bovis* is smaller and more transparent than the cysticercus cellulosæ, and is therefore more liable to escape notice. It is sometimes killed by a temperature of 44° to 45°C., and always by a five minutes' exposure to 48° to 50°C. It does not resist freezing, salting, or pickling for a period of one month.

I have been able to confirm the observations made by Dr. Oliver in India (1869), showing that after getting into the human intestine the cysticercus bovis developes into a mature Tænia Saginata in from 54 to 56 days.

Echinococcus polymorphus (Taenia Echinococcus) (v. Siebold).

This parasite occurs in almost every country, as tænia in the dog and some other carnivora, as hydatid cyst in man and several animals. Very little has been added of late to our knowledge of Tænias, but several interesting facts have been observed in connection with the deutoscolici.

With regard to the *Acephalo-cysts*, contrary to the opinion held formerly, that they were capable of developing into tænias in the dog, it is now recognised that they are incapable of perpetuating the species. In conjunction with Dr. Griglio, I have made a number of experiments showing that acephalo-cysts are incapable of producing tænias. Dévé, working in Blanchard's laboratory, and myself, in Turin, have simultaneously demonstrated that the deutoscolici or echinococcus heads are capable of producing cysts. This phenomenon is observed more specially when the *Echinococcus multilocularis* is used. Echinococcus infection must eventually yield to the preventive measures indicated by the advances of comparative pathology.

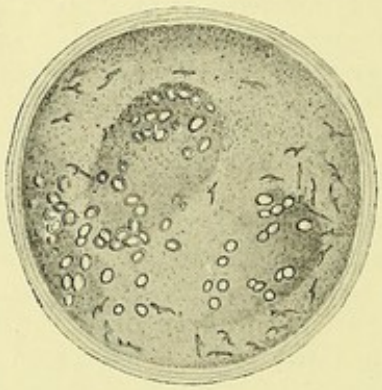

Fig. 5.

Echinococcus cyst resulting from the development of a deutoscolex.

Tænia nana (von Siebold) Hymenolepis nana (Leuckart).

This tænia was observed for the first time by Bilharz in Egypt, in 1851; seen afterwards by Spooner (1872) in the United States of America, by Blanchard (1885) in Belgrade, by Wernicke in the Argentine Republic; the same parasite has been observed in various parts of Italy, in Lombardy, by Grassi, Visconti, and Segré, and also by Comini; in Catania and Sicily by Grassi and Calandruccio, in Piedmont by myself, by Dr. Airoldi (1888) and by Dr. Vaccino (1903).

The presence of this Tænia is often associated with nervous symptoms which assume an eliptiform character. It is a small tænia which has many characters in common with the *Tænia murina* found in mice, of which it is probably only a variety as Grassi's experiments seem to show.

Tænia cucumerina (Bloch) Dipylidium C. (Leuckart), Tænia canina (Linnæus),

very common in the dog, is of great rarity in man. Krabbe has observed a few cases of this form of infection in Denmark. Blan-

chard also refers to several human cases observed in Germany by Leuckart, Salzmann, Schmidt; in Switzerland by Schoch-Bolley; in Scandinavia by Krabbe, and by Friis; Cobbold has observed one case in Scotland, and Martin one in France; Neumann also mentions a few cases.

Bothriocephalus latus (Bremser, 1817) Dibothrium latum (Rudolphi, 1810).

During the last twenty years a number of scientific observers have made a special study of the development of this worm. Among their researches I may specially mention those of the late Dr. Ernesto Parona, who found fully-developed larvæ of this cestode in the muscles of fish living in the lakes of Geneva, Lugano, Como, Varese and others. I have observed the occurrence of the Bothriocephalus latus in men, dogs and cats that had never been out of Piedmont, and I have also found larvæ of this worm in fish obtained from Piedmontese rivers.

TREMATODA.

I will only allude to a few of my observations relating to Trematoda. In 1867–8, in conjunction with my regretted " maître " Professor Rivolta, I made observations on the *erratic distomata* occurring in the lungs of cattle. These observations have since been extended by Baeltz in Japan, who has observed the same kind of occurrence in man.

I have also seen in the liver of a frog a *cercaria* without tail. The action of chloride of sodium upon free cercaria developed in Molluscs has been the subject of another investigation. I need only mention here my work regarding the treatment of distomatosis in sheep by the administration of the ethereal extract of male fern, and my study upon the development of the distoma hepaticum from the escape of the embryo from the egg to the penetration of the free larvæ into the *Limnæa truncatula* and *L. peregrina.*

NEMATODA.

Ascaris lumbricoides (Linnæus).

The Ascaris lumbricoides is generally found in the small intestine, from which it may wander upwards, through the stomach, into the œsophagus, and occasionally from there into the mouth and even the lachrymal duct.

This worm may also penetrate into the common bile duct or the pancreatic duct or descend into the cæcum and colon.

Dr. Demateis, in 1900, found that the intestine of ascarides obtained from the human intestine contained pathogenic microbes, and that by means of its buccal apparatus the worm could inoculate the intestinal mucosa with these microbes. This view has also been supported by Guiart.

Metchnikoff has attracted attention to the important causal relation between round worms and appendicitis. In 1902 Triboulet published a case of appendicitis due to the Ascaris Lumbricoides. Guiart has recorded four cases of appendicitis, two of which were recurrent and associated with the presence of round worms; in one of these cases he found eggs of the Trichocephalus, in the other those of the Ascaris lumbricoides. One of these cases was treated by Santonin, and the other by Thymol, and they both recovered. The successful result of this mode of treatment supports the view that the disease was of parasitic nature. Cases of appendicitis attributable to the presence of the Trichocephalus or Ascaris have also been observed by Schiller.

Weinberg has published a case of appendicitis occurring in a chimpanzee received at the Institut Pasteur from the French Congo: "The small intestine of this chimpanzee was actually stuffed with lumbrics; the cæcum alone contained three of them."

Beiguerel, Davaine, Natale observed the passage of a great number of Ascarides through a perforated appendix into the peritoneal cavity.

Brun has found an ascaris in a retro-appendicular abscess following a perforating appendicitis.

Cartaz published a case in which the appendix was completely obliterated by an ascaris, the cephalic end of which was free in the cæcum.

Töth was of opinion that these worms are capable of producing certain forms of anæmia and chlorosis, occuring more specially in girls.

Ascaris megalocephala (Cloquet).

In *foals* there are examples of perforation of the small intestine produced by *Ascaris megalocephala*, the perforation being followed by fatal peritonitis.

It is evident from what precedes that Ascarides are capable of producing more or less serious intestinal or even general disorders.

Oxyuris vermicularis (Bremser, 1819), Ascaris vermicularis (Linnæus, 1767).

The resistance of the thread worm to the most rational forms of treatment is well known. In children affected with this worm one observes that from time to time there is a considerable increase in their number. Hundreds and even thousands of thread worms may at times be discharged from the bowel with a single evacuation. These worms, when not paralysed by cold or some other cause, are very active.

They cause at times considerable malaise and pruritis of the anus, vulva, or vagina.

Either as a result of appropriate treatment, or from no apparent cause, these worms seem to disappear entirely for variable lengths

of time, after which they reappear. How can these relapses following apparent cures be explained? Some observations which I have recently made tend to show that the oxyuris larvæ undergo development in the cæcum, and more specially in the appendix vermiformis, where they are capable of producing a form of appendicitis.

A well-nourished rabbit which I had under observation died of appendicitis, and I found mixed with the contents of the appendix a large number of oxyurides in various degrees of development. In the thickness of the wall of the appendix there were numerous small white patches situated between the serous and muscular coats, the muscularis was partly destroyed, the mucosa was covered with clear mucus containing a variable number of cells, in that mucus there was an extraordinay number of larvæ of the *Oxyuris ambigua* in the first stage of development, and, in addition, there was a fully-developed male oxyuris.

These motile larvæ, by attacking the mucous membrane with their buccal papillæ, had caused ecchymoses and desquamation of the epithelium—in fact, a state of inflammation which had caused the walls of the appendix to become at least four times thicker than normal.

In the rest of the cæcum (fixed portion) one noticed a large number of male and female oxyurides in various stages of development, but already quite visible to the naked eye.

This observation indicates, I think, that the early stages of development of the larvæ occur in the terminal portions of the appendix, the contents of which are comparatively still and composed of partly-digested vegetable matter mixed with leucocytes and mucus the secretion of which is considerably increased by the irritation caused by the larvæ.

These facts support the view which has been advanced by Metchnikoff, Schiller, Still, Ramstedt, Oppe, Bruno Galli-valiero, and Martin regarding the share taken by the larvæ of the Oxyuris vermicularis in the production of appendicitis in the human subject.

It is true that Leuckart, Grassi, and Calandruccio have shown that the ingestion of adult oxyurides is followed in from 15 to 20 days by infection, but this does not explain the occurrence of the very large number of thread worms found in the intestine of infected children and animals. In order to account for this number of worms it is necessary to admit that they are capable of multiplying without leaving the intestine. As the eggs of this worm are very seldom found in the fæces it is reasonable to suppose that the impregnated females migrate to the cæcum from time to time to deposit there a variable number of ripe eggs, and that the embryos issuing from these eggs penetrate into the appendix to pass there the first part of their life. When capable of a more indepen-

dent existence the young worms would then pass into the cæcum and colon and mix with the intestinal contents; they would there become sexually mature and capable of perpetuating their kind.

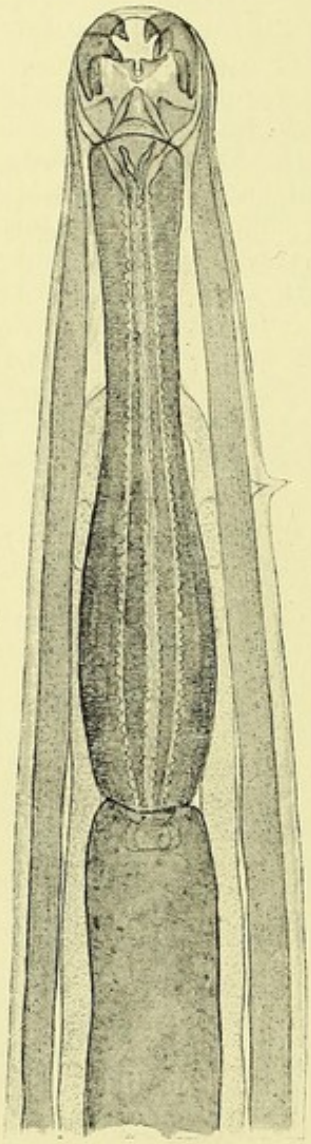

Fig 6.
Uncinaria duodenalis.

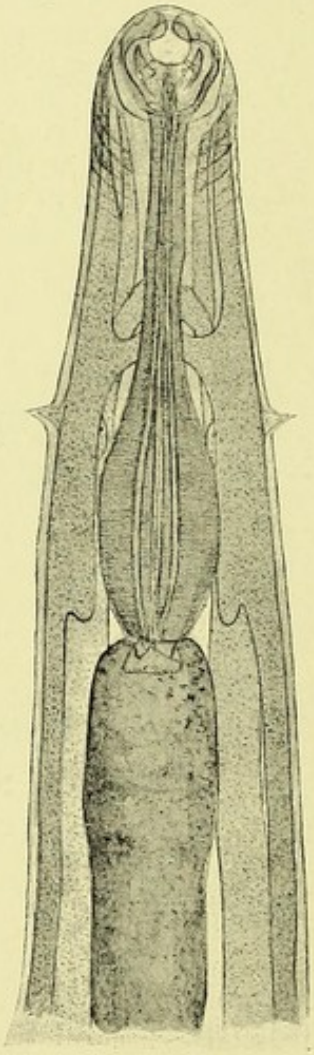

Fig. 7.
Uncinaria Americana.

The mature female worms which escape from the intestine with the fæces become the means of spreading the infection. Certain localities where external conditions are favourable to the preservation of eggs, embryos, or larvæ, may thus become infected.

Ankylostoma duodenale (Dubini, 1838-43), Uncinaria duodenalis (Raillet, 1885). (Fig. 6.)

This worm was formerly supposed to affect only peasants and brickmakers working in damp districts. In 1880 I found that it was the cause of the severe epidemic which affected miners engaged in the boring of the St. Gothard Tunnel, and also that it was the same worm that had caused the disease which for hundreds of years had produced great mortality among the miners working at Schemnitz, in Hungary, and in other places. Ankylostomiasis has been observed in Africa, where it is the cause of the Egyptian chlorosis, in Brazil, in France (St. Etienne and Valenciennes), in Russia, Germany, and Belgium. In England several centres of infection have recently been recognised (see papers and reports by A. E. Boycott and J. S. Haldane with regard to Cornish mines, and of Dr. Oliver, Newcastle-on-Tyne). In the United States of America Stiles and others have observed infection produced by the ordinary Uncinaria, and also by a variety called Americana, which is not provided with teeth. (Fig. 7.)

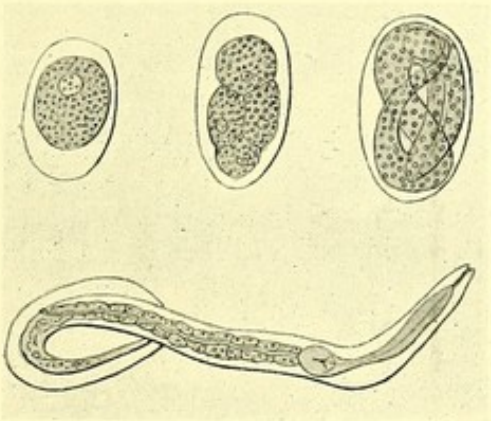

Fig. 8.
Eggs of Ankylostoma duodenale in various stages of development.

In 1884 Dr. Lütz, of St. Paolo (Brazil), sent me a number of Ankylostomata of the same kind as those described later by Stiles under the name of Uncinaria Americana.

The fecundated eggs are eliminated with the fæces, and when they find warm and damp surroundings they undergo segmentation and produce free embryos. The free embryo developes more or less rapidly in mud rich in organic detritus, and the larva reaches the stage of larval maturity, *i.e.*, it becomes encysted or encapsulated. During this period of their existence the larvæ take up mineral salts, part of which are deposited in their capsule. When in that state the larvæ, mixed with other dust, may be carried by the wind, or in some other way they find their way into water where thousands

of them may at times accumulate causing the water to become highly infectious.

If one introduces into some water a number of mature larvæ, it is found that in a short time they run together, forming here and there small opalescent clumps more or less clearly visible to the naked eye. It is easy to understand how a miner becomes infected by drinking such water. This mode of infection has actually been observed at the St. Gothard.

Up to the year 1880 clinicians were under the impression that the anæmia of miners was practically incurable, as no drug had been found that would invariably kill the worm in the human intestine. In that year, in conjunction with my assistant, Dr. Carita, I succeeded in breeding larvæ of Ankylostomata and Anguillulæ and obtaining millions of them, upon which it was

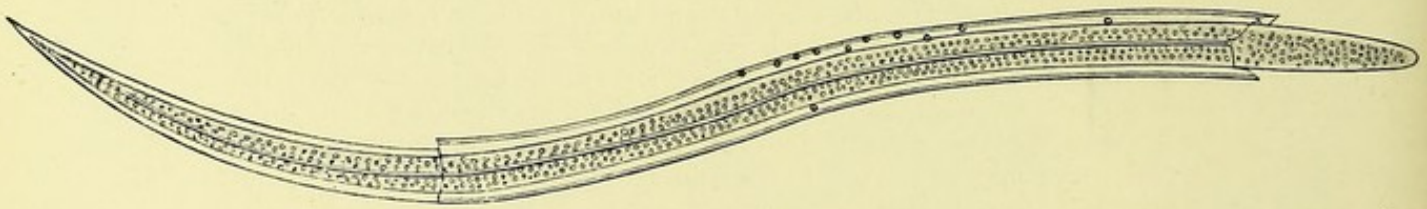

Fig. 9. Larva of Ankylostoma duodenale, with the capsule broken at both ends.

possible to try the action of various chemical substances. I was thus able to show that the larvæ were killed by the following substances:

Carbolic acid 1 per cent. solution, in less than 5 minutes.

Hydrochloric and Sulphuric acid 5 per cent. solution in 20 to 30 minutes.

Chloride of sodium, solutions of various strengths, in a time proportional to the strength of the solution.

Thymol, saturated solution, in 5 to 10 minutes.

Ethereal extract of male fern, in 5 to 10 minutes.

These facts have been utilised in the treatment and prevention of Ankylostomiasis. Thus, under the care of Drs. Schillinger and Töth, miners' Anæmia has disappeared from Schemnitz. These discoveries have also made it possible for Dr. Volante Guiseppe to eliminate the danger connected with Ankylostomiasis during the boring of the Simplon Tunnel.

Treatment by the ethereal extract of male fern and by thymol has been found efficacious in practice by many eminent clinicians, among whom I may mention Bozzolo, Parona, de Renzi and Maragliano.

Rhabdonema strongyloides (Leuckart), Strongyloides intestinalis (Bavay) (Grassi), Anguillula intestinalis and Stercoralis.

This worm is frequently a commensal of the Ankylostoma, but it may be found alone, and occur in such large numbers as to produce a more or less severe anæmia. I had, in 1880, the opportunity to observe cases of this form of infection in anæmic miners employed at the St. Gothard Tunnel and in other places.

This parasite lives usually in the duodenum and the jejunum, and less frequently in the ileum. The head and anterior parts of the worm penetrate through the epithelium, and after undermining it regain the surface by passing again through the epithelial layer. It thus produces lesions which may become serious by opening channels through which infection by virulent bacteria present in the intestine may take place.

The *Rhabdonema strongyloides* is dimorphobiotic (heterogonic). The *Anguillula stercoralis* of Normand and Bavay or *Pseudorhabditis stercoralis* (Perroncito), which was thought by Normand to be the cause of the dysentery observed in Cochin China, is the oviparous and viviparous offspring of the Rhabdonema strongyloides; it undergoes development during its passage through the intestine, from which it escapes with the fæces and reaches larval sexual maturity while in a state of free existence.

The larvæ eliminated from the intestine either become encapsulated or reach a state of sexual maturity under favourable circumstances. The development of the sexual organs may take place either while the worm is still in the fæces or when it has passed into some other medium; the new generation of larvæ produced by these Anguillulæ may reach maturation or become encysted. Water is easily infested by these larvæ, and may be the means of infecting new human hosts. On reaching the intestine they give rise again to the typical Rhabdonema strongyloides.

The relations of the Anguillula or Rhabdonemas to the intestinal mucous membrane account for the frequent failure of anthalminthic treatment. The worm being protected by the epithelium and a thick layer of mucus, is not easily reached by drugs which would otherwise readily kill it.

Trichocephalus dispar (Rudolphi, 1801; T. Hominis, Schrank, 1788).

The whip worm lives usually in the cæcum, rarely in other parts of the intestine. Its cephalic extremity penetrates the epithelial layer, and after undermining it regains the surface by perforating the same layer in the opposite direction.

Metchnikoff recognises a form of appendicitis attributable to this worm, and Girard has described, in 1901, two cases of this kind. In a case of appendicitis operated upon by himself Guinard has observed living *Trichocephalus dispar*.

Kermisson and Dilpritz have found this worm in 18 out of 22 cases. and subsequently Metchnikoff has found it in 12 out of 17 cases of appendicitis. I have already, when speaking of the Ascaris lumbricoides, mentioned the four cases described by Guiart, in two of which eggs of the Ascaris and Trichocephalus respectively were found. The same author believes that the whip worm is a factor of some importance in the production of typhoid fever and other enteric diseases. Schiller gives a series of five cases of appendicitis in which the following worms were found:—

Ascaris lumbricoides	in 1 case.
Trichocephalus dispar	,, 1 ,,
Ascaris and Trichocephalus...	,, 1 ,,
Oxyuris vermicularis	,, 2 ,,

It seems possible that the lesions produced by the Trichocephalus facilitates the penetration of microbes capable of producing septic and other forms of infection.

Raillet holds this view, and supports it by some observations in the dog.

Trichina spiralis (Richard Owen),

The Trichina spiralis was seen for the first time by Peacock (1828) and by Hilton (1832) at Guy's Hospital, London. It was again observed at St. Bartholomew's Hospital by Wormald and by Sir James Paget in 1835, and it was from specimens collected by the last observer that the worm was first described and named by Owen.

This worm has been admirably studied by Zenker, Leuckart, Virchow, Luschka, Cobbold, and other eminent observers, and it may be said that our knowledge concerning it is practically complete. The most recent work deals with the differential diagnosis of certain muscular lesions produced by Trichinæ, Rainey's tubes, young cysticerci, and actinomycetes when these parasites have died and undergone calcification. This differential diagnosis depends on careful micrscopical examination. Spencer Cobbold, as far back as 1880, insisted upon the importance of microscopical inspection of meat, and this method has gained in importance *pari passu* with the improvements which have taken place in latter years in the sanitary supervision of abattoirs all through the world.

During the years 1876–1877 I demonstrated that Trichinæ, like other worms, died when exposed to a temperature of 48° to 50°C. In 1879 I showed that in trichinous meat imported from America the trichinæ were usually dead at the time when the meat was placed on the market. It is well, however, to remember that since then the time of transit has been considerably reduced, and that it would not be wise to neglect the usual measures by which infected foods are excluded from our markets.

Ordinary cooking is sufficient to kill cysticerci, trichinæ and other worms liable to infect meat.

As far back as 1876 I have published the results of experiments upon the effects of cooking on animal parasites present in meat. I have referred to these experiments and others in my book on parasites *(loc. cit.)*. A few of them may be briefly mentioned here. Pieces of various kinds of meat were immersed in boiling water for various lengths of time, and the temperature reached by the central parts of these pieces of meat observed. The following table gives some of the results obtained: —

	Material tested.	Size or weight.	Duration of exposure.	Temperatures observed. centigrade scale.
1	Piece of Veal.....	9½cm. × 7cm.	— 10′	53°
			— 20′	63°, 65°, 67°
2	Piece of Beef (leg)...	8cm. × 10cm.	— 20′	47°
			— 35′	66°, 67°
3	Ham.....	6 kilos.	2 hours	46°, 55°, 58°, 62°, 64, 67°
4	Ham.....	7 „	4 hours	51°, 58°°, 59°, 61°, 67°
5	Ham.....	7 „	3 hours 25′	67°, 73°, 74°, 75°
6	Ham.....	8 „	3 hours 25′	62°, 74°, 78°, 84°
7	Calf's Tongue Ox Tongue „	Ordinary size Largest	1 hour 20′ 1 hour 54′ 2 hours 30′	58°, 63° 88° 90°
8	"Salame di testa"..	diam. 9cm. weight 2·16 kilos..	2 hours	83°
9	Sausage	3 × 10cm. weight 120 grms...	17′	64°, 65°, 66°

These experiments show that meat cooked in this way could not have transmitted the Trichina spiralis, the Tænia solium or the Tænia mediocanellata.

Epidemics of Trichinosis, so frequent in Germany from 1860 to 1870, have now become quite exceptional. Some isolated cases of trichinosis occur now and again, as if to remind us of the necessity of not neglecting the inspection of meat.

Leuckart's experiments upon the influence of cold upon Trichinæ should be repeated on a large scale in order to establish more fully the importance of refrigeration as a protective method.

I am inclined, on the basis of my experiments upon the action of various drugs, to believe that it would be possible to treat satisfactorily patients in the early stages of trichiniasis, *i.e.*, at the time when they show the first symptoms of intestinal infection.

INSECTA.

The presence in the human intestine of larvæ of various kinds of insects is a question which has attracted attention of late. In the horse and other animals the larvæ of the *Gastrophilus equi, pecorum, nasalis, hæmorrhoidalis* are frequently found.

With the collaboration of my late assistant, Dr. Bosso, I found in 1895 that sulphide of carbon was a true specific against these parasites.

APPENDIX.

Report of the Advisory Committee

on the Building and Opening of the

New Laboratory at York Place

and

Directors' Report

for the

Session 1904-5

TABLE OF CONTENTS.

Victoria University of Manchester.

PUBLIC HEALTH LABORATORY,

YORK PLACE.

Advisory Committee.

W. J. CROSSLEY, Esq., *Chairman.*

S. DELÉPINE, M.B., B.Sc., Professor of Comparative Pathology and Bacteriology, Director of the Laboratory.

HAROLD B. DIXON, M.A., F.R.S., Professor of Chemistry.

EDWARD DONNER, B.A., Member of the Council.

J. DRESCHFELD, M.D., F.R.C.P., Professor of Medicine.

ALFRED HAWORTH, B.A., Member of the Council.

D. B. HEWITT, M.D., Chairman of the Public Health Committee of the County of Cheshire.

The Right Hon. Sir J. T. HIBBERT, K.C.B., LL.D., Chairman of the Lancashire County Council.

ALFRED HOPKINSON, K.C., LL.D., Vice-Chancellor of the Victoria University of Manchester.

W. H. JOHNSON, Esq.

Alderman McDOUGALL, J.P., Vice-Chairman of the Sanitary Committee, Manchester.

T. de COURCY MEADE, M.Inst.C.E., Manchester City Surveyor.

J. DIXON MANN, M.D., F.R.C.P., Professor of Forensic Medicine.

JAMES NIVEN, M.A., M.B., Medical Officer of Health for Manchester.

ARTHUR RANSOME, M.D., F.R.S.

Sir HENRY E. ROSCOE, LL.D., D.Sc., F.R.S.

ARTHUR SCHUSTER, Ph.D., F.R.S., Professor of Physics.

EDWARD SERGEANT, M.R.C.S., D.P.H., F.C.S., Medical Officer of Health for the County Palatine of Lancaster.

Alderman WALTON SMITH, Chairman of the Manchester Health Committee and of the Port Sanitary Committee.

C. H. TATTERSALL, M.R.C.S., D.P.H., Medical Officer of Health for Salford; and Lecturer on Public Health.

Alderman JOSEPH THOMPSON, Treasurer of the University.

FRANCIS VACHER, F.R.C.S., M.R.C.P., F.C.S., Medical Officer of Health for the County of Chester.

F. E. WEISS, D.Sc., Professor of Botany.

PUBLIC HEALTH LABORATORY.

REPORT OF THE ADVISORY COMMITTEE.

General Statement.

In issuing for the first time a report upon the Public Health Laboratory, the Committee think it desirable to give a short account of the origin of this institution.

Under the Public Health Act, 1875, the whole of England was divided into sanitary districts, which were further defined by the Local Government Act, 1894. Every District Council had to appoint from time to time a Medical Officer of Health. After the 1st of January, 1892, no such appointment could be made in any county, district, or combination of districts, with a population of 50,000 or upwards, unless the Officer, in addition to the usual registrable medical qualifications, was also registered as the holder of a diploma in Sanitary Science, Public Health or State Medicine, under Section 21 of the Medical Act, 1886.

Since the year 1889, the Victoria University had granted a Diploma to medical men who wished to devote themselves to the Public Health Service and had attended special laboratory courses in the Pathological and Chemical Departments; but it was not till the 1892 regulations came into force that the teaching of Public Health was fully organised at the Owens College. At that time the examinations for the Diploma (then known as Diploma in Sanitary Science) became also more stringent, and more importance was attached to Pathology and Bacteriology than had been done before. Bacteriology had made rapid progress during the previous fourteen years; it had yielded results of the greatest importance in the field of preventive medicine, and it had become evident that bacteriological research was destined to be of great assistance in the work of sanitary authorities. These facts were brought before the Council of Owens College by the newly appointed Professor of Pathology; but the Pathological Laboratory was at that time very inadequate, consisting of three rooms only, two of which were daily occupied by students, and the third, which was also the Professor's room, was the only one available for delicate research work. The staff was also very small, consisting of the Professor and of only one Assistant who gave to the College half of his time. Notwithstanding these difficulties several important investigations bearing upon Disinfection, Tuberculosis, Asiatic Cholera and Cancer were carried out in the Department during the years 1892, 1893 and 1894.

In the year 1894 the Pathological Department was transferred to

the new Medical School Buildings, where ample accommodation had been made for it. It then became possible to devote a well-fitted laboratory to the teaching of Bacteriology and Pathology in relation to Public Health, and, shortly after this, two small rooms were set apart for the conduct of bacteriological investigations for public authorities.

The Council recognised that it was one of the duties of a University to take an active interest in scientific work which could be of benefit to the public, apart from teaching, more specially when such work was not already undertaken outside. Unfortunately the College had at its disposal no fund that could be devoted to the promotion of such work; it was therefore decided that Authorities requiring the assistance of the Pathological Department should pay fees towards the expenses of investigations carried out for them. Although for some years this source of income was inadequate, still as the benefits of the scheme have become more widely known, the income has steadily increased. Apart from finance, however, the arrangement has worked well from the beginning, for the Authorities were able to obtain from the University the assistance they required without incurring unnecessary expenditure, and the Bacteriological Laboratory was provided with a large supply of material and information valuable to research workers and to students of public Health.

The movement started in Manchester in 1892 was based upon a distinct want, as is shown by the subsequent adoption of a system similar to that established at Owens College by many Authorities in the kingdom. It is a matter of satisfaction to know that we have been able during the last ten years to assist by our advice many University and Municipal Authorities wishing to organise laboratories of the same kind as our own.

Notwithstanding the fact that the course of study has greatly increased in length and cost, the changes previously described have led to an increase in the number of students coming to the College Laboratories to study Public Health and to undertake various researches.

Removal of the Public Health Work from the College Buildings to Laboratories in Stanley Grove.

In 1901, the growth of the Bacteriological Section of the Pathological Department necessitated the removal of the Bacteriological Laboratory from the Medical School, and the erection of new Laboratories in Stanley Grove. These new Laboratories included research rooms and class-rooms for the study of Pathology, Bacteriology and Chemistry in relation to Public Health, and also rooms suitable for the conduct of investigations for Public Authorities. The teaching of Sanitary Chemistry and of Bacteriology remained respectively under the direction of the Professor of Chemistry and of the Professor of Pathology, the latter acting as Director of the Laboratory.

By the new arrangements it became possible for students to find under one roof the means of studying systematically and practically the branches of science bearing upon Public Health, and to have the opportunity of witnessing the application of various scientific methods to the solution of many problems bearing upon the prevention of disease.

Advisory Committee.

In 1902, the Council appointed an Advisory Committee, consisting of members of the Council, of the Senate and of the Public Authorities most interested in the work, the duties of this Committee being:—

1. To consider and report from time to time as to any development of the work of the Department, and especially as regards further provision for research, and as regards the relations of Local Authorities to the Laboratory.

2. To consult with the Director as to subjects relating to the Department which he may desire to bring forward.

3. To make reports periodically as to the work of the Department with a view to publication if the Council thinks fit.

Removal of the Public Health Laboratory to York Place.

The removal of the Manchester Royal Infirmary made it soon necessary to remove the Public Health Laboratories from Stanley Grove. This new displacement, though causing a considerable expenditure and interfering with the work of the staff for more than a year, had the advantage of providing the opportunity for the designing of laboratories better adapted to the study of the causes and prevention of disease and to the conduct of Pathological, Bacteriological and Chemical investigations bearing upon Public Health.

The present site in York Place was selected on account of its close proximity to the new Infirmary, a proximity which should be of mutual advantage to both institutions.

Division of the Chair of Pathology.

The various changes which had taken place during the previous ten years had caused the Pathological Department to assume much larger proportions than had originally been anticipated; and yet the Professor of Pathology felt that further developments were still needed, more especially in connection with the teaching of Morbid Anatomy in the Pathological Department of the Royal Infirmary. In 1902, he applied to the Council for the appointment of a Lecturer who would take charge of the teaching of Morbid Anatomy to students preparing for medical degrees, expressing, at the same time, the hope that, as soon as adequate endowments should make it possible, the Professorship of Pathology, as it existed then, should be divided,

one of the Professors being responsible for the teaching of Pathology and Morbid Anatomy to students preparing for medical and surgical degrees, the other taking charge of Pathology applied to Preventive Medicine and Public Health.

On the advice of a Committee appointed to consider this matter the Council adopted the latter course in 1904. The then Professor of Pathology was appointed Procter Professor of Comparative Pathology and Bacteriology as well as Director of the new Public Health Laboratory, and a new Professor of Pathology and Morbid Anatomy was placed in charge of the Pathological Department in the Medical School. These improvements have more than doubled the financial liabilities originally connected with the Pathological Department, but it is hoped that compensation will be found in the increased usefulness of the University, and that Public Authorities, and all those interested in the prevention of disease, will speedily relieve the University from this additional financial burden. Substantial help has already been given in this direction by the city of Manchester and by the Chairman of the Committee, who has presented a sum of £2,000 towards the erection of the new Laboratory.

The progress which has taken place during the last fourteen years is indicated in the following summary:—

SUMMARY SHOWING THE NUMBER OF REPORTS ISSUED AND THE TOTAL AMOUNT OF FEES RECEIVED, 1895—1905.

Year.	No. of Reports.	Total Receipts. £ s. d.	Average Number of Reports per Week.
1895–6 ...	300 ...	74 5 8 ...	3
1897 ...	1,504 ...	446 8 1 ...	28
1898 ...	2,402 ...	786 1 7 ...	46
1899 ...	3,003 ...	874 4 2 ...	57
1900 ...	4,652 ...	1,682 15 3 ...	89
1901 ...	5,220 ...	1,887 0 8 ...	100
1902 ...	5,165 ...	2,064 5 5 ...	99
1903 ...	5,773 ...	2,051 0 11 ...	111
1904 ...	5,777 ...	2,108 1 10 ...	111
1905 ($\frac{1}{2}$ year)	3,790 ...	1,278 2 0 ...	145

The monthly records for the last three years show that the work is still progressing:—

AVERAGE NUMBER OF SPECIMENS RECEIVED DAILY DURING THE YEARS 1903, 1904, AND 1905.

	1903.	1904.	1905.	Average for three years. 1903–4–5.
January	14·8	15·0	18·3	*16·0*
February ...	15·8	17·3	22·8	*18·8*
March	15·3	20·0	24·7	*20·0*
April	13·5	15·0	21·6	*16·7*
May	14·3	11·4	22·0	*15·9*
June	12·0	12·0	16·0	*13·3*
July	15·2	12·7	17·0	*15·0*
August	11·6	11·3	18·0	*13·6*
September ...	21·4	15·9	20·9	*19·4*
October	19·5	19·1	24·1	*20·9*
November ...	18·5	18·7	25·8	*21·0*
December ...	17·6	20·5	19·6	*19·2*

Since 1892 the following gentlemen have acted as Scientific Assistants in the Laboratory:—

1894–1895.—J. Richmond (M.A., M.B. Oxon., D.P.H. Cantab.), now Medical Officer of Health for Handsworth and Perry Bar, near Birmingham (occasional assistant).

1895–1905.—E. J. Sidebotham (M.A., M.B. Cantab.), now Senior Assistant and Lecturer on Practical Bacteriology in the Public Health Laboratory.

1897–1898.—A. E. Brindley (B.Sc. Vict., M.D. London, D.P.H. Vict.), now Medical Officer of Health for the County Borough of Bury.

1898–1905.—J. R. Carver (M.D., D.P.H. Cantab.), now Senior Assistant in the Public Health Laboratory.

1899–1901.—F. J. H. Coutts (M.D., D.P.H. Vict.), now Medical Officer of Health for the County Borough of Blackpool.

1899.—A. Greenwood (M.D. Vict., D.S.M.), now Medical Officer of Health for the County Borough of Blackburn.

1900–1905.—A Sellers (M.D. Edin., D.P.H. Vict.), now Senior Assistant in the Public Health Laboratory and Assistant Lecturer in Comparative Pathology.

1904–1905.—Gilbert Fowler (D.Sc., Vict.), Senior Chemical Assistant in the Public Health Laboratory and Lecturer in Bacteriological Chemistry.

1904–1905.—W. B. Ramsden (B.Sc., M.B., Ch.B. Vict.), Chemical Assistant in the Public Health Laboratory, and Lecturer in Sanitary Chemistry.

The following gentlemen should also be mentioned amongst those who gave temporary assistance in periods of stress:—

1895–1900.—F. C. Moore, (M.D., M.Sc. Vict.), Lecturer in Hygiene, Municipal Technical School.
1896–1897.—Francis Villy (B.A., M.D. Cantab.).
1897–1898.—J. H. Ray (M.B., M.Ch. Vict.).
1902.—W. J. S. Bythell (B.A., Cantab., M.D. Vict.).
1902.—R. W. Marsden (M.D., D.P.H. Vict.), late Superintendent, Monsall Fever Hospital.
1902.—T. A. Rothwell (M.D., D.P.H. Vict.), Medical Officer of Health for Hale.
1902–1903.—I. Walker Hall (M.D. Vict.), late Assistant Lecturer in Pathology; Professor of Pathology, University College, Bristol.

It is a matter of satisfaction to know that all these gentlemen have received the whole of their bacteriological training in our laboratory. They have fully justified the high opinion which we had formed of them by their subsequent work, and, in several cases, by the important appointments which they have obtained.

A list of over 370 students who have studied Bacteriology in this Department since 1892 is given in Appendix III. Among them will be found many Medical Officers of Health who hold important appointments.

OPENING OF THE PUBLIC HEALTH LABORATORY.

The new Laboratory was opened on January 27th of this year by Mr. W. J. Crossley. Earl Spencer, the Chancellor of the University, presided over the ceremony, and was supported by the Vice-Chancellor and the Lord Mayor of Manchester. Among those present were Lord Stanley of Alderley, the Right Hon. Sir James Fergusson, Bart., Sir W. Mather, Sir Bosdin T. Leech, Mr. Edward Donner, Alderman Stephens, Mayor of Salford, Alderman Joseph Thompson, Alderman McDougall, the Dean of Manchester, Mr. Cosmo Melvill, Mr. Neville Clegg, Dr. Arthur Ransome, Councillor Fildes, and a large number of representatives of Local Authorities. Many English and foreign men of science were also present: The Vice-Chancellor of the University of Liverpool; Sir Wm. S. Church, Bart., President of the College of Physicians; Professor Woodhead, of Cambridge; Professor R. Ross and Professor Hope, of Liverpool; Professor Trevelyan, Professor Hartley and Professor Grünbaum, of Leeds; Sir James Russell and Dr. D. Noël Paton, of Edinburgh; Colonel J. Lane Notter, Dr. Charles J. Martin and Dr. Charles Slater, of London; Professor Calmette, representing the Pasteur Institutes of Paris and Lille; Professor Salomonsen, of Copenhagen; Professor Perroncito, representing the Minister of Public Instruction, Italy, and the University of Turin; and many other representatives of Manchester and other Universities. Lord Lister, the late Sir John Burdon Sanderson, Sir James Crichton

Browne and other scientific men who were unable to be present, expressed by letter their warm approval of this extension of the University work.

After the ceremony of opening, Honorary Degrees were conferred by the Chancellor upon Professor Calmette, Professor Perroncito and Professor Salomonsen in the Whitworth Hall.

The following speeches were delivered:—

Opening Ceremony at the New Laboratory.

In presenting the keys to Mr. W. J. Crossley, the Director said: My Lord and Chancellor, My Lord Mayor, Ladies and Gentlemen,

There is a special fitness in our asking Mr. Crossley to open a Laboratory devoted to the study of the causes and prevention of Disease. Everyone knows how for years he has striven to supply the best possible means for the treatment and prevention of consumption, and how much this district is indebted to him for his princely gift of a sanatorium second to none in the country. When we asked him several years ago to become the Chairman of our Advisory Committee we knew of his sympathy for our work, and of the benefits which we would derive from his experience and advice. Quite recently he gave us further proof of his good-will by assisting us materially when we were confronted by financial difficulties due to the displacement of the laboratory. It is therefore with special pleasure that we see him come here to-day to open the new Public Health Laboratory of the University.

The building which he is to open is not fully equipped yet; 9 months ago the site upon which it now stands was occupied by four houses, and the old laboratory was still standing in Stanley Grove. Since last April we have had to pull down the old laboratory, replace the old material in the new building, and we have done our best "to carry on our work as usual during the alterations." Before this year is over we hope our laboratory will be properly furnished and equipped.

It may be interesting to recall that up to 1902 Public Health had no special abode in Owens College. The teaching of medical men wishing to enter the Public Health service was conducted in the Pathological and Chemical Laboratories. About 1895 two small rooms were set aside in the Pathological Laboratory for investigations carried out for Public Health Authorities. In 1901 this work had grown to such an extent that it was necessary to find new accommodation for Public Health Work outside the Medical School, and a house situated in Stanley Grove was adapted for the purpose. At the same time the College created a separate Department for the study of Pathology, Bacteriology, and Chemistry as applied to Public Health and for the conduct of investigations for Public Authorities. Finally the removal of the Royal Infirmary to Stanley Grove made it neces-

sary to rebuild the Laboratory on the present site. The development of this department was slow at first, from 1891 to 1893 we were doing pioneer work, there was no other laboratory in the kingdom in close connection with local sanitary authorities, and few people realised the use which bacteriology could be to the public. But from 1895 to the present day the progress has been rapid.

It was greatly due to the interest which Dr. Ward, the late Professer Leech, Professor Dixon, Mr. Donner, and Alderman Joseph Thompson took in the matter, that any progress was made at first. The voluntary assistance given in the early stages by Dr. E. J. Sidebotham was also of great value as well as that of the successive members of the staff who willingly undertook onerous duties without regard to adequate remuneration. Outside the College, we also received encouragement, support and advice, in succession from Dr. C. Porter, Dr. Niven, Dr. Vacher, Dr. Tattersall, Dr. Sergeant and several others, among whom I may specially mention Dr. A. Ransome. I wish also to add a word to express my personal sense of the obligation we are under to the Vice-Chancellor for his continued efforts to promote the scheme. The great services rendered by you, sir, have already been mentioned.

It is only natural that we should remember at the present time the share taken by all these gentlemen in the development of an institution which it is earnestly hoped will prove of still greater use in the future than it has been in the past.

Mr. Crossley, in asking you to declare the Laboratory open we beg you to accept in remembrance of this day a key of the Laboratory.

We feel confident that whenever you visit us you will find this Laboratory full of earnest workers engaged in studies worthy of the University, and having for object the prevention and relief of suffering.

Our work has already received encouragement from the interest taken in it by the distinguished men of science who have done us the honour of coming from various parts of Great Britain and of the Continent to take part in this ceremony.

Mr. W. J. Crossley, in declaring the laboratory open, said that if he needed a text for his address he would choose the time-honoured saying: "Prevention is better than cure," not that the investigations conducted at the laboratory were not largely for the purpose of curing, but that the great aim of public health work was to bring about such sanitary conditions as might prevent what Prof. Delépine called the "accident of disease." We were invited to look upon it as a bad accident, worse perhaps than the breaking of a limb, if we should encounter such an army of hostile bacilli as might suffice to overcome the opposition of our health conditions, and carrying the citadel by assault, produce in us the disease for which they were the duly accredited agents. This, too, before we were conscious that we had been in battle at all. This was an accident, but under proper

conditions it should be an avoidable accident. It was the duty of our officers of public health to keep these foes in check, to watch and record their movements, temper or doings; to issue warnings and instructions how such foes were to be met. Surely it was a tragic thing that we, "the lords of creation," should be unable to see our most dangerous enemies who struck before we knew of their presence. To look for these enemies and to find them was no easy task, and involved much study. Our protectors, our scouts, must be taught, and in the Public Health Laboratory they were offered every facility for learning the methods of warfare. Without such institutions and such equipment they could do no more than a workman without tools. Hence the duty of making ample provision both for teaching and for research in the New Laboratory. It was a strange thing that in this country, in which we spent so vast sums on armaments against visible foes who might never attack us, our Exchequer could spare so little to assist the work of institutions such as that. On the Continent Governments liberally supported work done in this field, but in this country nearly everything had to be done by voluntary effort, even our County Councils were unable to contribute towards building funds, being restricted to payments for investigations ordered. He thought it would be agreed that the director of such an institution as the Public Health Laboratory should not be weighted down with financial troubles, but should be able without considering every shilling, to keep abreast of the times with the best apparatus and the best professional assistance.

The Vice-Chancellor, speaking on behalf of the University, complimented Professor Delépine, to whose devoted labour, he said, the existence of the Laboratory was due. Professor Delépine had been able to excite warm interest in his work amongst those who were engaged in the work of local administration. The work of combating disease, of preventing disease, and of advancing all kinds of knowledge which would aid in the cure as well as in the prevention of disease, and in teaching a band of men who would be the Public Health Officers of the future, if it was to be done efficiently in a great community, must be on the lines of the Laboratory, and must have the co-operation not only of the University and its teachers, but also of the local authorities around them.

The Lord Mayor of Manchester, in moving a vote of thanks to Mr. Crossley, said that the Public Health Laboratory had done a very large amount of work for the Manchester Corporation, more particularly in connection with fevers and consumption and the control of the milk supply. As a matter of fact nearly half of the total public health work done by the Laboratory had been done for the Manchester Corporation. As a result of the work it had been possible to take preventive action with much greater precision, and so save both suffering and expense. Important work had also been done for the City in connection with the public water supply, and with the

air of sewers. One great advantage which Manchester possessed in the Public Health Laboratory was the security that should cholera or plague assail this country, they could rely upon Professor Delépine to give exact and timely information as to the character of the disease. It would be an advantage in practical prevention of infection if Professor Delépine were free to devote more time and money to research both personally and by the direction of others.

Dr. A. Ransome, F.R.S., said:

I will not detain you long, though there is much that might be said on the value of Preventive Medicine. "The Health of the People is the Supreme law," not only for Statesmen and Lawgivers, but for the whole community. The Common Health comes even before the Common Wealth, which indeed would be impossible of attainment without it—"*Sanitas sanitatum, omnia sanitas.*"

There is no Department of Medical Science more worthy of study and support than that of Public Health.

High praise and many honours have been showered upon the Department of Surgery for its recent magnificent successes, and it has well deserved them. I would not say one word in disparagement of the great skill and courage of its professors; but it should be remembered that its greatest triumphs have been rendered possible only by means of the knowledge that has been given them by bacteriologists. Moreover, when we come to the subject of life-saving, we may say, in paraphrase, that "if Surgery has saved its thousands, Preventive Medicine has saved its tens of thousands." In proof of this statement you have only to look at the enormous diminution that has taken place of late in all kinds of preventable disease, especially in the mortality from such microbic diseases as Consumption and all belonging to the Zymotic class. Many millions have thus been saved.

You have to-day provided the University and, through it, the whole of this great community, with the means of adequately teaching the Principles of Public Health and, under the guidance of Professor Delépine, one of the first Pathologists of the day, you have furnished the weapons for meeting and staving off many diseases. You may be sure that they will be valiantly wielded, and that, under his drilling, there will soon be trained a small army of men devoted to the work of saving, not destroying, human lives.

I earnestly pray for God's blessing upon their noble work.

Dr. A. Calmette, Directeur de l'Institut Pasteur de Lille, delivered the following speech:—

Je suis doublement heureux de m'associer à cette fête de la science que vous célébrez aujourd'hui: d'abord parce que je puis exprimer à votre glorieuse Université toute ma reconnaissance pour le degré honorifique de docteur ès-sciences qu'elle veut bien me conférer en même temps qu' à d'autres savants illustres. Ensuite et surtout parce que j'ai reçu de l'Institut Pasteur de Paris et de l'Institut Pasteur

de Lille l'agréable mission d'apporter au Professor Delépine et au Comité d'organisation de ce nouveau laboratoire d'hygiène publique, l'expression sincère de notre estime, avec nos meilleurs voeux de prospérité.

Vous êtes tous déjà bien convaincus par expérience de la grande utilité de cette œuvre. Vous avez pu vous rendre compte des immenses services qu'elle vous rend pour la lutte sociale contre les maladies infecteuses les plus meurtrières telles que la tuberculose, la diphtérie, la fièvre typhoide.

Il est évident que vous pouvez en attendre des services beaucoup plus importants encore dans l'avenir, à la seule condition que vous assuriez son existence. Or, à ce sujet, jéprouve le besoin de vous faire part d'une réflexion qui m'est venue à l'esprit lorsque j'appris que le laboratoire n'avait aucune ressource fixe, aucune subvention annuelle, et qu'il était exclusivement, pour employer vos propres expressions, *self-supporting*.

Permettez-moi de vous dire que je vois là un très grand inconvénient : plus ce laboratoire vous rendra de services, plus il contribuera au developpement de l'hygiène, plus ses ressources diminueront, et le jour ou il aurait presque entièrement fait disparaitre de Manchester les maladies contagieuses, c'est lui qui devrait mourir si vous ne lui apportez pas de larges souscriptions comme tribut de votre reconnaissance !

Vous avez tout intérêt, certes, à lui apporter ces souscriptions et à les faire très généreuses, parce qu'elles permettront au Professeur Delépine d'entreprendre des recherches nouvelles en vue de contribuer aux progrès de la science de l'hygiène. Le Professeur Delépine a toutes les qualités nécessaires pour donner un éclat particulier à ces recherches et pour illustrer ces nouveaux laboratoires. Son passé scientifique est la pour vous l'attester. Son nom, connu et estimé de tout le monde savant, est désormais inséparable de cet Institut : il serait aussi difficile de les séparer qu'il est difficile de séparer la chaleur de la lumière. Aussi est-ce de tout coeur que je lui apporte mes félicitations les plus cordiales et que, tant au nom de l'Institut Pasteur de Paris qu'en celui de Lille, je souhaite à ses travaux tout le succès dont il est digne !

The Degree Ceremony at the Whitworth Hall.

Before conferring Honorary Degrees the Chancellor, Earl Spencer, expressed the pleasure it gave him to be present. For the last two years he had been prevented from taking part in the proceedings of the University of which he had the honour to be Chancellor. He then referred to the events which had led recently to the establishment of three Universities in place of the old federal Victoria University. For the first time he stood among them as Chancellor of the University of Manchester. He had the honour to be Chancellor

of the Victoria University before the reconstruction took place, and he had thought it his duty then to place his resignation in the hands of the Vice-Chancellor and his colleagues. And, he said, he regarded it as a mark of high distinction and a great honour that he should have been asked to retain the office of Chancellor in the Victoria University of Manchester. He felt this all the more because when the discussions about the change took place he differed from many of those who were acting with him. He wished now to say most earnestly and distinctly that he would always do his utmost to uphold and support the University with all the influence which he might possess. He was convinced that the University would fulfil its lofty aims and duties, and that it would be a centre of light and learning to the nation, and especially to those round it, that it would foster civilisation, improve commerce, and give to all who came within its influence the best equipment—a high and intelligent education. He believed that in Manchester those who were at the head of society and commerce, proud of their University, would come forward with the enthusiasm and with the liberality which had been so remarkable among them, to cherish and foster the noble institution which the wisdom and generosity of their ancestors had set up amongst them.

The Chancellor then referred to the splendid work of Professor Delépine and his co-adjutors, who had been pioneers in public health work not only in Lancashire but in the nation at large. Fifteen years ago little or nothing was done by public authorities with regard to bacteriological research, but thanks to the University and to Professor Delépine, a great advance had been made in this respect. He was glad to know that other municipalities besides Manchester were now awakening to the importance of this means of prevention of disease, and that another weapon would be put in the hands of local authorities in combating the attacks of disease. He spoke of the wide field covered by the study of public health, overcrowding, insanitary buildings, water supply, drainage, temperance, and many other subjects where a great deal yet remained to be done by Parliament and by public bodies. But they could not overlook the great progress that had recently been made, and which had placed at the services of the poor, facilities which could before only have been commanded by the rich. He knew what enormous benefit the Laboratories which they had opened would confer on local authorities, and how much illness would be prevented as a result of the researches there.

Dr. C. J. Salomonsen, Professor of Pathology and Director of the State Serum Institute, Copenhagen, delivered the following address after the Chancellor had conferred the Degrees:—

In the name of Professor Perroncito and Dr. Calmette, my two renowned colleagues, and in my own I most respectfully thank the Victoria University, who has to-day conferred on us the Degree of

Doctor of Science. We highly appreciate the great honour shown us by this famous University—famous not only for its having so largely contributed to the advancement of science and to the raising of the intellectual standard of English youth, but famous also for its special stamp as to origin, programme and growth.

It was perhaps fortunate for this town that the Parliament assembled in the year of 1640 did not pay attention to the claims of the northern parts of England, and shut their ears to these often quoted words about "the many ripe and hopeful wits who were utterly lost for want of education." For the failure of this first attempt to erect a University in Manchester made it possible a long time afterwards to start the movement, which, 120 years ago, was inaugurated by the paper of Thomas Barnes "A plan for Improvement and Extension of liberal education in Manchester." It is true that his new and original programme, "connection between liberal science and commercial industry," has never and nowhere been accomplished, but it certainly has influenced the men, who during the past century fought for promoting liberal education in Manchester, tried to suit the Owens College to the peculiarities of this town and this district, and thus contributed to give the Victoria University its individual feature.

Between the two Right Reverend old and classical sisters in Oxford and Cambridge, who in a happy manner, unite the splendour of former days with the glories of present time, the Victoria University is emerging as a modern, unsectarian, young, and charming English woman, fostered and cherished by the industrious, the rich, the freeborn citizens of Manchester, eager to break down deep-rooted prejudice, zealous to open new strata of the population to the influence of University life; ardent and youthful!

Rapid growth is a prerogative of youth, and the Highest School of Manchester has grown rapidly from the year when the Owens College—the real nucleus of this University—was founded. The short history of the Institute whose new building we opened to-day, symbolises this rapid growth. We congratulate the University of Manchester on the consummation of this splendid Public Health Laboratory, and especially we congratulate you, Professor Delépine, on this result of your earnest work during fourteen years. We are convinced that the remembrance of your admirable perseverance and your scientific enthusiasm will always live, in this Laboratory, as a latent source of energy for further progress, and we cannot express our hearty thanks for the great honour shown us to-day in a better way than by these words.

May the new Public Health Laboratory gloriously inscribe its name in the annals of English science, and may it support the Victoria University in its noble task of always adding fresh lustre to Manchester, to Lancashire, to England!

Sir William Church commented upon the rapid rise and progress

of State medicine, and the increased knowledge which has recently been acquired as to the true causes of such diseases as anthrax, cholera, tuberculosis, typhoid fever, and malaria. Increased attention has also been paid of late years, he said, to the circumstances under which our trades and manufactures—more especially those which are prejudicial to the health of the workers—are conducted, as well as to the conditions under which the masses of the people live. Numerous problems were therefore involved in the question of public health, or State medicine, and for the right investigation of these problems special laboratories were required, in order that there might be proper scientific means of investigation, and that in connection with them accurate and true results might be obtained. "State medicine," he added, "is, in my opinion, the highest branch of our art, and it is with very great satisfaction that everyone has seen that the newer universities have accorded to it in their curricula a most honoured position."

Professor WOODHEAD, of Cambridge, delivered an interesting address on the aims and recent developments of Bacteriology, and wished success to the new Laboratory, of which he spoke as being an important addition to the resources of the Manchester University.

The following letter was then presented to the Chancellor by Professor PERRONCITO:—

ROYAL UNIVERSITY OF TURIN.

LETTER FROM HIS EXCELLENCY, THE MINISTER OF PUBLIC INSTRUCTION, ITALY.

TORINO,
27 gennais, 1905.

Ricevo in questo momento da S. E. il Ministro della P. I. la lettera che integralmente qui Le trascrivo.

"Prego la S.V. di voler invitare il Chiar. Prof. Edoardo Perroncito di codesta Universita a rappresentami alla solennita della inaugurazione del laboratorio della publica igiene, solennita che avra lusgo nella 'Victoria University' a Manchester.

"E avro caro se Egli al fiorente liteneo inglese e agl' insegnanti e agli studenti di esso, porgera, in mio nome, un affettuoso saluto di simpatia e di ammerazione.

Il Ministro fir. ORLANDO.

Colgo quest' occasione per porgere alla S.V. Chiar. gli atti della mia distinta ed affettuosa osservanza.

Il Rettore,
G. CHIRONI.

Al Chiar. Sig. Prof. Dott. Comm. Edoardo Perroncito
presso l'Universita di Manchester.

ROYAL UNIVERSITY OF TURIN.

LETTER FROM HIS EXCELLENCY, THE MINISTER OF PUBLIC INSTRUCTION.

27 January, 1905.

I have just received from His Excellency the Minister of Public Instruction the letter of which I send you a literal transcript.

"Pray be good enough to invite Prof. Edoardo Perroncito of your University to represent me at the ceremony of the opening of the Public Health Laboratory, which ceremony will take place at the Victoria University of Manchester.

"I should wish him to present in my name an affectionate greeting of sympathy and admiration to that flourishing English Institution to its Professor and students.

"(Signed by the minister) ORLANDO.

I take this opportunity of expressing my own personal esteem and regard for you.

G. CHIRONI,
Rector.

To Professor E. Perroncito.

LETTER FROM LORD LISTER, F.R.S., D.C.L., LL.D.

12, Park Crescent,
Portland Place,
January 25th, 1905.

My dear Professor Delépine,

Had the state of my health permitted, I should have had peculiar pleasure in attending the opening of your Public Health Laboratory. For I need hardly say that I feel deep interest in this enterprise, calculated as it is to do immense service to Manchester and its neighbourhood, and also to bring additional renown to the Victoria University.

Let me congratulate you to whom it is largely due on your great achievement.

I cordially hope that generous benefactors will be forthcoming to provide the funds still required for the complete development of the Institution.

Believe me,
Very sincerely yours,
LISTER.

LETTER FROM ADOLPHUS WILLIAM WARD, ESQ., Litt.D., LL.D., Master of Peterhouse College, Cambridge, late Principal of Owens College and Vice-Chancellor of the Victoria University.

Falmouth,
January 7th, 1905.

I am obliged to deny myself the pleasure of attending the opening of the Public Health Laboratories on the 27th, but I should like to send you a word of congratulation on the achievement which this ceremony seems to signify, as well as on the prospect of still greater and more beneficient usefulness which it opens. I always thought the College could not do better than connect its research directly with a purpose of such public utility, and now that it has become the University of Manchester this responsibility seems to come to it more directly than ever. May your work continue to prosper.

A. W. WARD.

After the ceremony Professor PERRONCITO delivered a lecture on "Intestinal Parasites," at the Public Health Laboratory. This lecture is published in the 1st Volume of the Archives of the Laboratory.

BUILDING OF THE NEW UNIVERSITY LABORATORY (YORK PLACE).

The last report presented by the Director to the Advisory Committee referred to the year 1903. The report for the year 1904 should have been submitted as usual in the early part of this year, but it was found desirable to make the Laboratory's financial year correspond with the academic year, and to make it extend from June to June, instead of from December to December as heretofore. In future therefore the report of the Director will be presented to the Committee either at the end of the Summer Session or at the beginning of the Winter Session as may be found most convenient. To prevent any confusion in the accounts, the statement prepared at the beginning of this year and relating to the year 1904 has been kept separate from that for the first half of this year.

Since the last report was presented the Public Health Laboratory has been transferred from Stanley Grove to the present site. An account of the steps taken up to the month of February, 1904, will be found in the report for the year 1903, pp. 24 to 30.

On March 8th, 1904, the Advisory Committee approved of the plans designed by the Director and which were subsequently carried out, and recommended the Council to accept Messrs. William Southern and Sons' tender, the estimate of the cost of the building, without fittings, amounting to £6,124. On March 16th, 1904, the contractors began to clear the site, and on April 14th the laying of the foundations commenced. The building

operations proceeded rapidly under the immediate supervision of the Director, who has drawn the attention of the Committee to the energy, care and rapidity with which Mr. Wm. Southern carried out his contract. On October 14th, 1904, *i.e.*, exactly six months after the beginning of the building operations, Public Health classes were held in the new Laboratories, and on October 31st, 1904, municipal research work was entirely transferred to the new buildings.

During the preceding six months the work of the Laboratory had been conducted under extraordinary difficulties. Part of the Laboratory at Stanley Grove had to be pulled down to provide building material for the new Laboratories, and the members of the staff were obliged to conduct their investigations in small outbuildings or sheds, until even these were pulled down, and it became necessary for them to transfer their work to the Pathological Department during the summer recess. It was also necessary to take for six months a small house in York Place to store up books, apparatus and furniture. Notwithstanding all these disturbances bacteriological investigations went on without interruption, but it was found advisable to discontinue some important work which could be deferred without serious inconvenience to Authorities. The members of the staff submitted cheerfully to all this trouble, and deserve much credit for the admirable way in which they discharged their respective duties under these trying conditions.

The capital account balance-sheet shows that the debt incurred by the University in respect of the new Laboratory is at present £2,415. 18s. 6d. (to which must be added £350 which will become due on the land (at the end of next year). The debt on the Stanley Grove Laboratory, which was £2,536. 5s. 3d. at the time of the removal, must also be kept in mind. These three sums make the *total debt £5,302. 3s. 9d. at the present date.* Of the £8,556. 8s. 7d. available for the purpose of building and equipping the new Laboratory Mr. Crossley has given £2,000 towards building, and the Council have allotted £400 out of the Manchester Corporation grant towards the equipment of the Laboratories. A sum of £20 has also been received from the Lancashire Medical Veterinary Association for the same purpose.*

Description of the New Laboratory.

The new Laboratory consists of a main building 150 ft. in length and 60 ft. in width, with a caretaker's cottage and out-buildings attached to it. The building stands on its own grounds, measuring 5,831$\frac{7}{8}$ square yards, extending between High Street and York Place. The space available round the building will gradually be utilised in carrying out experiments upon purification of water, soil bacteria and their influence upon plants and animals, the erection of annexes to some of the experimental laboratories, and various other purposes.

* A sum of £6000 was received from the Royal Infirmary in compensation for the displacement of the Stanley Grove Laboratory. Of this sum, £1000 was contributed towards expenses caused by certain requirements of the Royal Infirmary.

The front of the Laboratory runs along York Place and has a north exposition, and the main entrance is at the east end of the building. The front elevation was designed by Professor Capper. The ground floor is divided by a straight passage extending from the east to the west end of the building. On the northern side of the passage are all the laboratories requiring a north light for microscopical work. Given in order from east to west the laboratories are devoted to the following purposes:—

1. Clinical Pathology (22 ft. × 27 ft.), to be used chiefly for work done for the Royal Infirmary.
2. Practical Bacteriology (35 ft. × 27 ft.), to accommodate 24 students.
3. Research Laboratory (29 ft. × 27 ft.), sub-divided into four small private research rooms (12 ft. × 6 ft.), a common work room, micro-photographic and dark rooms.
4. A small research room (12 ft. × 6 ft.) for work of a dangerously infectious kind; this room is easily isolated from all the others.
5. Municipal Investigations Laboratory (30 ft. × 27 ft.), with places for seven workers. Attached to this room is an Incubating Chamber.
6. Preparation room (22 ft. × 30 ft.), with working benches for four laboratory servants. This room gives access to the Refrigerating Chamber, and to a cellar used for storing purposes.
7. On the south side of the building and communicating with the preparation room is the Engine room (25 ft. × 14 flt. 6 in.), where centrifugal, shaking, grinding, &c. machines are kept. This room is also used as a store room.

The following laboratories are on the south side of the central passage:—

8. Dissecting room with dark room (21 ft. × 17 ft. 6 in.).
9. Chemical store room (20 ft. × 10 ft.).
10. Balance room (12 ft. × 6 ft. 9 in.).
11. Sanitary Chemistry laboratory (52 ft. 6 in. × 17 ft. 6 in.), with accommodation for 18 students.

On the first floor, beginning at the east end and north side of the building, there are the following rooms:—

12. Lecture room (27 ft. × 22 ft.).
13. Apparatus room (22 ft. × 15 ft.), connected with the museum.
14. Museum (79 ft. × 22 ft.) for the display of specimens illustrating the mode of production of preventable human and animal diseases, and the exhibition of specimens, plans, drawings, &c., relating to public health matters.
15. Museum preparation and store room (31 ft. × 22 ft.).

On the south side of the building, beginning at the west end of the first floor, the following rooms are situated:—

16. Staff's common room (25 ft. × 17 ft.).
17. Director's laboratory (27 ft. × 18 ft.).
18. Library (67 ft. × 18 ft.).
19. Director's office (13 ft. 6 in. × 18 ft.).
20. Secretary's office (12 ft. × 18 ft.).
21. Documents' room (6 ft. × 18 ft.).

At the west end of the building there are yards and outbuildings, including a cubical room measuring 1,000 cubic feet, and fitted up for the purpose of testing various methods of disinfection.

The whole building is heated by hot water and lighted by electricity, and all the laboratories are fitted with an abundant supply of gas and water. The floors of the first storey are covered with linoleum; those on the ground floor are concreted and covered with asphalt. As far as possible all the angles between the walls, floors, and ceilings are rounded to facilitate cleaning. On the ground floor the laboratory sinks discharge into channels made in the floor and lined with asphalt; these channels are covered with movable iron plates, they discharge externally into movable earthenware receptacles, which act at the same time as seals and intercepting traps; these discharge into ordinary gullies. The tables in the rooms where bacteriological work is conducted are made of glass and iron. The metal supports allow the floors to be easily cleaned. Each room is ventilated independently by shafts opening externally above the ridge of the roof, acting as outlets, and openings in the windows acting as inlets, the natural system of ventilation being adopted all through the building. In all the rooms ample provision has been made for the admission of natural light. The objects which the director had in view in designing these laboratories were—1st, To provide the permanent and occasional workers with a healthy abode where they could carry out work which is at times dangerous without unnecessary risk; 2nd, To prevent the teaching work from interfering with the municipal work and *vice versa;* 3rd, To group the rooms in such a way as to facilitate the work of each section and save time as much as possible; 4th, To avoid any unnecessary expenditure on building and fittings whenever this was possible with impairing efficiency.

W. J. Crossley,

Dec. 1st, 1905. *Chairman of the Advisory Committee.*

Work Done in the Public Health Laboratory

DURING THE YEARS 1904 AND 1905 (FIRST HALF).

DIRECTOR'S REPORT.

This work may be considered under the following heads:—

I.—INVESTIGATIONS CARRIED OUT FOR PUBLIC AUTHORITIES, ETC.
II.—TEACHING WORK.
III.—RESEARCH.

I—INVESTIGATIONS FOR PUBLIC AUTHORITIES.

STAFF.

Director: SHERIDAN DELÉPINE, M.B. (Edin.), M.Sc. (Vict.), B.Sc. (Lausanne).

Bacteriological Section: E. J. SIDEBOTHAM, M.A. (Camb.), M.B., M.R.C.S.; J. R. CARVER, M.D. (Camb.), D.P.H. (Camb.), ARTHUR SELLERS, M.D. (Edin.), D.P.H. (Vict.).

Chemical Section: G. J. FOWLER, D.Sc. (Vict.); W. B. RAMSDEN, M.B. (Vict.), B.Sc. (Vict.).

Secretary: E. C. ILIFF.

Technical Assistant: F. SIMONS.

INVESTIGATIONS FOR SANITARY AUTHORITIES.

Prevention of disease saves suffering and loss of life to a much greater extent than the best methods of treatment, for treatment can only repair, often incompletely, the damages caused by disease. Nothing can be more gratifying to the physician or surgeon, as well as to his patients, than the successful application of a good treatment, but it is quite obvious that if a disease is preventable it should not occur at all. The highest aim of medicine is the prevention of disease. Infection is responsible for a very large proportion of the total number of cases of disease, and early diagnosis of a case of infectious disease is one of the most important means of prevention. By bacterial methods the diagnosis of many diseases such as Diphtheria, Typhoid Fever, Tuberculosis, Anthrax, Cholera, Plague,

etc., can generally be obtained at an early stage of the disease, and before the usual symptoms allow an absolutely certain diagnosis to be made. Bacteriological methods are not infallible, but they yield a very high percentage of accurate results. For instance, while it is difficult in the early stages of Typhoid Fever to establish on the basis of ordinary symptoms, an early and reliable diagnosis in more than 50 or 60 per cent. of the cases, by bacteriological methods it is possible to make an accurate diagnosis in 90 to 95 per cent. of the cases. In addition to this there are many obscure cases of disease, the nature of which can be found during life only by careful bacteriological investigation.

A knowledge of the actual cause of a case of illness is not only useful for the purpose of applying preventive measures in connection with that case, and of protecting in time other individuals, but it also adds considerably to the utility of statistical returns. An exact knowledge of the distribution of disease allows the authorities to concentrate their preventive measures where they are needed, and to avoid the waste of money and energy resulting from the application of preventive methods where they are not needed.

If, however, bacteriological examinations are to be of use to authorities, and to the public generally, even the simplest of them must be conducted with considerable care and a sense of serious responsibility which can be found only in well-trained bacteriologists whose medical training has made them familiar with disease.

The time may come when an army of medical bacteriologists will be available who will deal with morbid products in the same way as chemical public analysts deal with various substances which have to be analysed chemically. But at the present time experts of this kind are not available outside well-organised laboratories, and it is for this reason that the University has undertaken this work, which is one of public utility. Moreover, there are numerous investigations the practical applications of which are not immediately obvious, but which form the basis of further advances; the expense of these investigations cannot be covered by fees, and can only be undertaken in endowed laboratories.

In the field of Sanitary Chemistry there are many very important questions bearing upon public health, and which require lengthy investigations before they can be dealt with by the Public Analyst in his routine work. To provide for the study of such questions a suitable Chemical Laboratory has been added to the Bacteriological Laboratory, and two very competent chemists have been invited to co-ordinate their work with that of the bacteriologists. Great care has been taken, however, to prevent any clashing between the work done in the laboratory and that done outside. We have confined our work to the study of *the causes and prevention of disease*, and more specially those diseases which are produced by microbes, by larger parasites, or by noxious influences of any kind connected with food, air, water, or occupation. We have not attempted to manufacture

certain bacterial products used in the treatment of disease, and which are at the present time prepared by well-endowed institutions and great commercial firms.

In connection with our examinations we have collected since 1895 a large mass of facts relating to each case of disease, and these facts have been gradually classified with the view of extending our knowledge of the conditions favouring the occurrence and spread of several diseases prevalent in this district. This information, which is not included in official routine reports, has frequently been communicated privately to Medical Officers of Health. A detailed account of the organisation of this part of our work and of some of the results obtained will be given in our next report.

In the course of the last few years cases of the following human and animal diseases have been investigated in the Laboratory:— Diphtheria, Typhoid Fever, Tuberculosis (human, bovine, equine, porcine, avian), Pseudo Tuberculosis, Pneumonia, Typhus, Asiatic Cholera, Plague, Malta Fever, Malaria, Aleppo Boil, Hydrophobia, Anthrax, Tetanus, Malignant Œdema, Septicæmia, Bacterial Food Poisoning by Milk, Cheese, Meat, etc., Gonorrhœa, Cancrum Oris, Tumours (including Cancer), Actinomycosis, Botryomycosis, Glanders, Pleuro-Pneumonia, Fowl Cholera, Psorospermosis, Echinococcosis, Strongylosis, Bilharziosis, Myasis, etc., etc.

Various products have been examined for evidence of noxious properties.

Water, air, sewage, milk, cheese, butter, ice-cream, beer, wine, meat, bread, preserved foods, food preservatives, etc.

We have also tested and improved various methods of Sterilisation by steam, Chemical Disinfection, Filtration, Treatment of Sewage, etc.

During the last year several special investigations have been carried out, these are mentioned under the head of Research.

The routine work done during the course of the year 1904 and 1905, up to June 24, is summed up in the following tables:—

REPORTS ISSUED DURING 1904.

C.B., County Borough; M.B., Municipal Borough; U.D., Urban District; R.D., Rural District.

N.B.—The number of specimens yielding positive results is given under the figure showing the total number of specimens examined.

Place.	Diphtheria.	Typhoid.	Sputum.	Milk,	Various.	Total.
Stockport, C.B.	62 15	39 12	20 7	—	—	121
Manchester, C.B.	1253 419	678 285	659 243	524 60	42	3156
Eccles, M.B.	24 3	38 14	35 11	—	1	98
Withington, U.D.	64 13	50 19	41 14	1 0	1	156
Salford, C.B.	175 53	130 45	67 21	23 3	3	398
Stretford, U.D.	23 9	8 5	7 5	—	—	38
Moss Side, U.D.	27 5	16 6	25 3	—	—	68
Crewe, M.B.	23 5	5 0	30 8	—	—	58
Alderley Edge, U.D.	1 0	1 0	—	—	—	2
Blackburn, C.B.	1	121 84	—	16 3	13	151
Haydock, U.D.	1 0	—	3 1	—	—	4
Newton-in-Makerfield, U.D.	8 2	10 5	8 4	—	2	28
Oldham, C.B.	33 11	64 48	—	2 0	—	99
Darwen, M.B.	7 2	2 0	—	—	—	9
Bury, C.B.	50 11	17 7	30 5	—	5	102
Chadderton, U.D.	2 0	10 7	3 0	—	1	16
Rawtenstall, M.B.	7 1	8 4	—	—	—	15
Prestwich, U.D.	8 1	4 1	—	—	—	12
Urmston, U.D.	4 1	1 1	1 0	—	—	6
Bucklow, R.D.	4 0	1 1	1 1	—	—	6
Knutsford, U.D.	—	2 0	—	—	—	2
Winsford, U.D.	—	2 0	—	—	—	2
Northwich, U.D. Northwich, R.D.	40 17	5 1	—	—	1	46

Place.	Diphtheria.	Typhoid.	Sputum.	Milk.	Various.	Total.
Congleton, M.B.	2 / 0	2 / 0	2 / 0	—	—	6
Middleton, M.B.	3 / 0	5 / 2	7 / 3	—	—	15
Accrington, M.B.	1 / 1	—	—	—	—	1
Royton, U.D.....	1 / 0	—	—	—	—	1
Altrincham, U.D.....	6 / 2	1	—	—	—	7
Nantwich, R.D.....	1 / 0	—	—	—	—	1
Ashton-on-Mersey, U.D.	1 / 0	5 / 3	5 / 1	—	—	11
Failsworth, U.D.	1 / 1	3 / 0	—	—	—	4
Levenshulme, U.D. ...	14 / 3	5 / 2	7 / 2	—	8	34
Wilmslow, U.D.	—	8 / 5	1 / 1	—	—	9
Mossley, M.B.	—	1 / 0	—	—	—	1
Heywood, M.B.....	15 / 6	8 / 3	—	—	—	23
Blackpool, M.B.	75 / 33	95 / 32	—	4 / 0	5	179
Rochdale, C.B.	8 / 0	6 / 2	14 / 6	—	2	30
Heaton Norris, U.D....	13 / 1	5 / 0	2 / 1	—	—	20
Marple, U.D.....	1 / 0	—	—	—	—	1
Farnworth, U.D.	—	3 / 3	2 / 0	—	—	5
Cheadle and Gatley, U.D.	7 / 1	4 / 2	—	—	—	11
Lancashire County Council.....	40 / 11	—	—	—	6	46
Hale, U.D.....	4 / 2	—	1 / 1	—	—	5
Abram, U.D.....	—	1 / 1	—	—	1	2
Cheshire County Council.....	—	—	—	—	8	8
Ashton-under-Lyne, M.B.	—	—		—	17	20
Swinton and Pendlebury, U.D.....	20 / 3	11 / 6	1	—	—	35
Bredbury and Romiley, U.D.	1 / 1	3 / 0	—	—	—	4

Place.	Diphtheria.	Typhoid.	Sputum.	Milk.	Various.	Total.
Manchester Port Sanitary	—	—	—	—	—	—
Bowdon, U.D.	1/1	—	1/0	—	2	4
Adlington, U.D.	—	—	—	—	—	—
Lymm, U.D.	1/1	2/2	—	—	5	8
Barton-upon-Irwell, R.D.	1/0	1/1	—	—	—	2
Little Lever, U.D.	—	1/0	1/0	—	1	3
Runcorn, R.D.	4/1	—	—	—	—	4
Denton, U.D.	—	2/0	1/0	2/0	—	5
Sale, U.D.	7/2	1/0	4/1	—	—	12
Nantwich, U.D.	—	2/0	—	—	—	2
Ladywell Sanitorium	275/83	—	—	—	1	276
Ulverston, U.D.	44/13	—	—	—	—	44
Burton-on-Trent, C.B.	—	—	—	66/11	—	66
Huddersfield, C.B.	—	—	—	2/0	—	2
Derby, C.B.	3/1	—	11/4	—	—	14
Crompton, U.D.	1/0	—	—	—	—	—
Various	15/7	20/7	115/41	2/0	173	326
	2383/742	1407/580	1111/387	642/77	298	5840

GROSS RESULTS OF SOME OF THE CURRENT BACTERIOLOGICAL EXAMINATIONS.

	Total.	Positive.	Positive Per Cent.
Diphtheria	2383	742	31·1
Typhoid	1407	580	41·2
Tuberculosis (Sputum)	1111	387	34·7
,, (Milk)	642	77	11·9

REPORTS ISSUED DURING THE FIRST AND SECOND QUARTERS OF THE YEAR 1905.

C.B., County Borough; M.B., Municipal Borough; U.D., Urban District; R.D., Rural District.

N.B.—The number of specimens yielding positive results is given under the figure showing the total number of specimens examined.

Place.	Diphtheria.	Typhoid.	Sputum.	Milk.	Various.	Total.
Stockport, C.B.	27 2	11 3	20 9	—	3	61
Manchester, C.B.	699 233	415 164	287 115	439 38	26	1866
Eccles, M.B.	15 2	10 2	12 1	—	1	38
Withington, U.D.	35 10	27 9	33 11	—	—	95
Salford, C.B.	71 26	45 12	33 14	—	1	150
Stretford, U.D.	6 1	9 4	2 0	—	—	17
Crewe, M.B.	10 5	2 0	22 12	—	1	35
Alderley Edge, U.D....	21 5	2 1	1 0	—	—	24
Blackburn, C.B.....	48 9	54 11	86 24	10 2	8	206
Haydock, U.D.	2 2	—	2 1	—	—	4
Newton-in-Makerfield, U.D.	5 1	2 0	7 5	—	—	14
Oldham, C.B.	13 3	44 21	—	—	1	58
Darwen, M.B.	2 0	2 1	3 1	—	—	7
Bury, C.B.	11 4	7 2	7 4	—	1	26
Chadderton, U.D.	4 2	5 2	2 0	—	—	11
Rawtenstall, M.B.	2 0	5 1	—	—	—	7
Prestwich, U.D.	9 4	2 1	—	—	—	11
Urmston, U.D.	2 1	—	6 2	—	1	9
Bucklow, R.D.	6 3	3 0	2 1	—	—	11
Knutsford, U.D.....	1 0	—	—	—	—	1
Middleton, U.D.....	—	—	—	—	27	27
Winsford, U.D.	1 0	1 0	—	—	—	2
Northwich, U.D.	8 5	1 0	—	—	—	9
Congleton, M.B.	1 1	1 0	1 1	—	—	3
Middleton, M.B.	—	2 0	2 0	6 2	—	10

Place.	Diphtheria.	Typhoid.	Sputum.	Milk.	Various.	Total.
Royton, U.D.....	1/0	—	—	—	—	1
Altrincham, U.D.	3/0	1/0	—	—	—	4
Nantwich, R.D.... ...	3/1	—	—	—	—	3
Ashton-on-Mersey, U.D.	2/1	—	2/2	—	—	4
Failsworth, U.D. ...	—	5/2	—	—	—	5
Levenshulme, U.D. ...	5/3	1/0	13/8	—	—	19
Wilmslow, U.D. ...	7/2	—	5/3	—	—	12
Heywood, M.B.... ...	12/8	2/0	—	—	—	14
Hollingworth, U.D. ...	—	—	—	—	—	—
Blackpool, M.B. ...	56/27	28/11	2/0	4/0	—	90
Rochdale, C.B. ...	1/0	9/3	8/3	1	—	19
Withnell, U.D. ...	—	2/0	—	—	—	2
Heaton Norris, U.D....	—	1/0	—	—	—	1
Marple, U.D.	—	—	2/1	—	—	2
Farnworth, U.D. ...	—	1/0	—	—	—	1
Cheadle and Gatley, U.D.	1/0	—	—	—	—	1
Lancashire County Council	—	—	—	—	4	4
Hale, U.D.	10/3	1/0	4/2	—	—	15
Cheshire County Council	—	—	—	—	1	1
Swinton & Pendlebury	8/1	4/0	—	—	—	12
Bredbury & Romiley	1/0	1/1	—	—	—	2
Bowdon, U.D.	1/1	—	—	—	—	1
Lymm, U.D.	9/6	—	—	—	2	11
Sale, U.D.	3/0	—	4/2	—	—	7
Nantwich, U.D.... ...	1/0	—	—	—	—	1
Northwich, R.D. ...	7/4	—	—	—	—	7
Ulverston, U.D. ...	38/8	—	—	—	1	39
Ladywell Sanatorium	285/89	—	—	—	—	285
Bristol, C.B.	—	—	—	2/0	—	2
Bolton, C.B.	—	—	—	—	16	16

Place.	Diphtheria.	Typhoid.	Sputum.	Milk.	Various.	Total.
Burton-on-Trent, C.B.	—	—	—	47 / 4	—	47
Derby, C.B.	47 / 17	1 / 0	6 / 4	—	—	54
Baguley Sanatorium	4 / 1	2 / 1	—	—	—	6
Garstang, R.D.	—	—	—	—	1	1
Walton-le-Dale, U.D.	—	—	—	—	1	1
Chorlton Guardians...	—	—	—	3	1	4
Royal Infirmary, Manchester ...	—	—	—	—	10	10
Various	8 / 0	1 / 1	58 / 25	—	104	171
	1512 / 491	710 / 253	632 / 251	513 / 46	210	3577

GROSS RESULTS OF SOME OF THE CURRENT BACTERIOLOGICAL EXAMINATIONS

For the Half-year ending December, 1904, to June, 1905.

	Total.	Positive.	Per cent.
Diphtheria	1512	491	32·4
Typhoid Fever	710	253	35·6
Tuberculosis (human, pulmonary) ...	632	251	39·7
Tuberculosis (bovine, milk)	513	46	8·9

II.—TEACHING WORK.

Vice-Chancellor:

ALFRED HOPKINSON, K.C., LL.D., M.A., B.C.L.

Registrar:

E. FIDDES, M.A.

Director:

SHERIDAN DELÉPINE, M.B., B.Sc., M.Sc.

Teaching Staff:

Public Health	Lecturer, C. H. TATTERSALL, M.R.C.S., D.P.H.
Public Health Administration..	Lecturer, JAMES NIVEN, M.A., M.B. (Cambridge).
Comparative Pathology and Bacteriology.	Procter Professor, A. SHERIDAN DELÉPINE, M.B., C.M. (Edinburgh), B.Sc. (Lausanne), M.Sc. (Manchester). Assistant Lecturer, A. SELLERS, M.D. (Edinburgh), D.P.H. (Manchester). Lecturer in Practical Bacteriology, E. J. SIDEBOTHAM, M.A., M.B. (Cambridge). Lecturer in Bacteriological Chemistry, G. J. FOWLER, D.Sc. (Manchester).
Chemistry	Professor HAROLD B. DIXON, M.A. (Oxford), M.Sc. (Manchester), F.R.S., Director of Chemical Laboratories. Professor W. H. PERKIN, Ph.D. (Munich), F.R.S. Assistant Lecturer and Demonstrator, D. L. CHAPMAN, B.A. (Oxford).
Physics and Meteorology ...	Professor A. SCHUSTER, Ph.D. (Heidelberg), Sc.D. (Cambridge), M.Sc. (Manchester) F.R.S., Director of Physical Laboratories. Lecturer in Physics, CHARLES H. LEES, Sc.D. (Manchester). Assistant Lecturer in Physics and Lecturer in Meteorology, G. C. SIMPSON, M.Sc. (Manchester).
Biology — *Zoology*	Professor S. J. HICKSON, M.A. (Cambridge), D.Sc. (London), F.R.S.
Biology	Professor F. E. WEISS, M.Sc. (Manchester), D.Sc. (London),
Infectious Diseases	Clinical Lecturer, A. K. GORDON, B.A., M.B., B.C. (Cambridge).
Veterinary Hygiene ...	... Lecturer, WILLIAM WOODS, F.R.C.V.S. (England).
Veterinary Inspection	A. HOLBURN, M.R.C.V.S. (England)) For G. W. BRITTLEBANK, M.R.C.V.S. (Eng.) / M'nchest'r

Diploma in Public Health Committee.

The Vice-Chancellor.
Professor Delépine.
Professor Dixon.
Professor Perkin.
Professor Schuster.
Professor Stirling.
Professor Weiss.
Dr. Niven.
Dr. Tattersall.

The External Examiner in Public Health.

Dr. J. Lane Notter.

Diploma in Veterinary State Medicine Committee.

The Vice-Chancellor.
Professor Delépine.
Professor Dixon.
Professor Hickson.
Professor Perkin.
Professor Schuster.
Professor Stirling.
Dr. Niven.
Dr. Tattersall.
Mr. Woods.

The External Examiner in Veterinary Medicine.

Sir J. McFadyean.

The organisation of the Public Health teaching is quite distinct from that of the investigation work of the Laboratory. As in other University Departments, the Public Health Department is under the control of the Senate, which has appointed two standing committees, viz.:—The Public Health Committee and the Diploma in Veterinary State Medicine Committee (see p. 32), the function of which is to report to the Senate upon special questions.

The following courses have been given at the Laboratory during the last session:—

1. Courses for medical men preparing for the Public Health Service.

(*a*) Comparative Pathology and Bacteriology (including food inspection).

(*b*) Sanitary Chemistry.

(*c*) Advanced courses in Public Health, including Sanitary Administration.

2. Courses for chemists intending to become Public Analysts, Agricultural Chemists, &c.

(*a*) Practical Bacteriology—part of Course 1 (*a*).

(*b*) Bacteriological Chemistry.

3. Courses for Veterinary Surgeons who desire to become Veterinary Inspectors under public Authorities.

(*a*) Comparative Pathology and Bacteriology (including food inspection). See 1 (*a*).

(*b*) Sanitary Chemistry. See 1 (*b*).

(*c*) Veterinary Hygiene.

4. Courses for Medical men, Chemists, Veterinary Surgeons, &c., who wish to undertake *special* researches bearing upon causes or prevention of human or animal diseases. These students often join one or more of the above courses, and, in addition receive personal tuition previous to undertaking research work proper.

5. Post graduate courses in *Clinical Pathology* for Medical men wishing to study some of the recent advances in Microscopical, Bacteriological, and Chemical methods of diagnosis.

6. A special course of *Dental Bacteriology* was given during the Session 1904–5 pending the rearrangement of the courses in the Dental Department.

During the Session 1904–5 the number of students attending these various courses has been as follows:—

1. Public Health course	17
2. Bacteriology for Chemical students	8
3. Veterinary State Medicine courses	5
4. Research students	6
5. Clinical Pathology	25
6. Dental Bacteriology	8
	69

One of these students began Research work after entering for Public Health course, thus causing a double entry, so that the actual number of students was 68. Of these 68 students 25 attended a practical course given at Burnley. The giving of extra mural courses has thrown so much work upon the staff, that it has been found desirable to discontinue them for the present. Arrangements have been made for the teaching of Dental Bacteriology in the Medical School, so that in the future Dental students will not attend at the Laboratory unless they enter as Research students. *The number of students attending courses which will continue to be given at the Laboratory* was therefore 68 minus 33; that is 35 during the past academic session (see p. 38).

In previous reports the entries have been given from January to January; in this report they are given from June to June. The following table gives the entries for the three previous academic years for comparison with the year 1904–5. The reduction in the number of Research Students during the Session 1904–5 was due to the moving of the Laboratory.

Comparison of Entries for the Sessions 1902 to 1905.

	Session 1901-2 Winter	Session 1902-3 Winter	Session 1903-4 Winter	Session 1904-5 Winter
Practical Bacteriology	5	10	17	19
Practical Pathology and Microscopy.	6	13	15	13
Sanitary Chemistry	5	13	16	13
Laboratory Practice	2	0	3	1
Veterinary Hygiene	—	—	—	5
Dental Bacteriology	—	—	—	8
Research*	0	3	8	4
Advanced Public Health Lectures ...	—	—	33	—
Clinical Pathology (extramural) Burnley	—	—	20	25

* The number of research students working at any time in the laboratory is not indicated by the number of entries, because entries are sometimes made for more than one session, other entries are made for shorter periods.

	Summer	Summer	Summer	Summer
Practical Bacteriology	10 ...	9 ...	4 ...	6
Sanitary Chemistry	1 ...	5 ...	— ...	—
Laboratory practice	0 ...	3 ...	— ...	—
Research*	0 ...	3 ...	3 ...	1
Bacteriological Chemistry	— ...	— ...	— ...	2
Clinical Pathology (extramural) Wigan	— ...	— ...	22 ...	—
Totals for the whole Academic year	29 ...	59 ...	141 ...	97
Entries for postgraduate courses not usually given	— ...	0 ...	75 ...	33
Entries of Regular Students preparing for diplomas, or degrees, or doing research work	29 ...	59 ...	66 ...	64

Of the 68 students taking courses during the year:—

16 were medical graduates of the Edinburgh University.
9 „ „ „ „ Victoria „
9 „ „ „ „ Aberdeen „
3 „ „ „ „ Glasgow „
2 „ „ „ „ Ireland „
1 was a medical graduate of the Cambridge „
1 „ „ „ „ Brussels „
1 „ „ „ „ Bombay „
3 were members of the Royal College of Surgeons of England.
3 „ „ „ „ Veterinary Surgeons.
3 „ fellows „ „ „ „
1 was a fellow of the Institute of Chemistry.
7 were chemical students.
8 were dental students.
1 was a medical student.

68 (excluding double entries).

Of these students 43 worked in the Laboratory.
„ „ 25 attended practical courses held in Burnley.

These students came from the following places:—

Manchester	21
Burnley	20
Nelson	4
Blackburn	3
Briersfield	2
Bolton	3
Oldham	2
Dukinfield	2
Ramsbottom	1
Stockport	1
Bury	1
Cheadle	1
Preston	1
Colne	1
Bombay	1
Ashton-under-Lyne	1
Darwen	1
Sheffield	1
Wigan	1
Total	68

(Owing to the alteration in the mode of reckoning the year, the following entries do not appear in the report for the year 1903 nor in the present report for the year 1904–5.

During that period of six months the students attending the various courses were as follows:—

Public Health courses	8
Bacteriology for Chemical students	1
Research	1
Clinical Pathology (extramural course, Wigan 1)	22
Total	32

By deducting 22 students attending extramural courses there remain 10 regular students (see also p. 40).

DEGREES AND DIPLOMAS OBTAINED BY STUDENTS WHO HAVE WORKED IN THE LABORATORY BETWEEN JANUARY 1904, AND JULY 1905.

D.P.H.—The following 17 candidates have obtained the *Diploma in Public Health* of the University:—

E. M. Ashcroft, J. R. Hutchinson, C. S. O'Neill, C. C. Webb, E. B. Leech, Reginald Lawrence, W. C. Rigby, O. H. Chapman, James Howard, Harold Thorp, W. A. Newall (old student), Isobel Tate, F. D. Blandy, B. K. Goldsmith, A. H. Radcliffe, G. G. Buckley, C. W. Crawshaw.

D.V.S.M.—The following 2 candidates received the *Diploma in Veterinary State Medicine* of the University:—

J. S. Lloyd and J. M. Stirling.

M.D.—The following 7 candidates received the *Degree of M.D.* of the University:—

M. B. Arnold, A. C. Clarke (commended), A. W. Latham (gold medal), Thomas Tierney, J. J. Butterworth, W. W. Clemesha, H. W. Russell (gold medal). (Dr. Arnold and Dr. Tierney did only part of their Dissertation work in the Laboratory.)

The following 4 candidates have obtained the *Diploma in Public Health* of other examining Boards:—

D.P.H., Cambridge.—A. B. Arnold, M.D., Vict.; Harold Kerr, M.B., Ch.B., Edin.

D.P.H., Conj. Board, London.—H. Holt, M.B., Ch.B., Vict.

D.P.H., Durham.—J. M. Ferguson, L.R.C.P.

Advanced Course of Public Health Lectures.

This course was given during the year 1904 by well-known experts, who came from various parts of the country at our invitation, to deliver one lecture each on subjects upon which they are well recognised authorities. The course was attended by Medical Officers of Health, Lecturers in the Public Health Department, and advanced students, 33 in all. Each lecture was fully illustrated by experiments, demonstration of methods, specimens, photographs, diagrams etc. After lecture the Laboratory was opened to members of the class, and members of the Laboratory staff exhibited or demonstrated specimens, apparatus, experiments, or methods of interest to Medical Officers of Health.

The following outline of subjects suitable for lecture was prepared by the Director for the guidance of lecturers:—

1. Climate in relation to disease (including Geographical distribution, Meteorology and methods of observation).
2. Family and race in relation to disease (Ethnology in relation to Etiology).
3. Food and drink in relation to disease (including preparation, preservation, and analysis).
4. Air in relation to disease (including ventilation, methods of analysis).
5. Water in relation to disease (including collection, storage, distribution, analysis, etc.).
6. Sewage and other refuse in relation to disease (including collection, storage, distribution, analysis, etc.).

7. Soil in relation to disease (including Geology in relation to disease).
8. Habitation in relation to disease (private dwellings, workshops, schools, hospitals, etc.).
9. Clothing in relation to disease.
10. Social habits in relation to disease.
11. Occupations in relation to disease.
12. Parasites in relation to disease (including parasitological Zoology and Botany).
13. Spread of infectious diseases (Epidemiology).
14. Diseases prevalent at various periods of the history of man. History of the development of preventive methods.

The subjects falling under these various groups may in many cases be treated from one of the following points of view:—

1. Causation and modes of spread of disease (Epidemiology and Etiology including Diagnosis) (Human and animal diseases).
2. Prevention and control of disease (Human and animal diseases).
3. Hygiene of the individual, of congeries of individuals (human or animal).
4. Law and administration.
5. Surveying, Engineering, Architecture.

The lectures delivered during the year 1904 will be found in the first volume of the Archives of the Laboratory.

III.—RESEARCH WORK.

The research work carried out in the Laboratory during the last $1\frac{1}{2}$ years falls under two categories:—

1. Researches carried out by the members of the staff in connection with investigations for Public Authorities but apart from the routine reporting.

2. Researches carried out by students or graduates who desire to learn methods of research or to conduct some original investigations.

I. Researches by Members of the Staff.

Our obligations towards Public Authorities and the absence of any Research Endowment, do not permit the members of the staff to select their field of research. Many questions which appeal strongly to us on account of their importance must necessarily be left aside so long as the duties which we have undertaken claim all the time which it would be our desire to devote to independent research. Fortunately our work for Public Authorities does not consist only in the application of well-established methods, but in addition to this routine work, the scientific value of which is chiefly of a statistical value, we have frequently to study questions which require for their solution lengthy and sometimes difficult researches bearing on facts about which little is known. Although these investigations have always a practical aim they are none the less of a purely scientific character, the only disadvantage connected with this form of research being a want of continuity.

The reorganisation of our work in the new Laboratory, and the increased facilities which the Laboratory affords us, will, I hope, allow me to make arrangements by which each member of the staff will in the near future have enough time to undertake continuous investigations independently of the ordinary routine work.

Some of the research work carried out during the last eighteen months by Dr. Sidebotham, Dr. Carver, Dr. Sellers, Dr. Fowler, Dr. Ramsden and myself may be mentioned here to show that, notwithstanding the difficulties under which we have laboured, some useful scientific work has been undertaken:—

1. Influence of preserved foods on the causation of certain bacterial diseases (for the Manchester Corporation).

2. Bacteriology of sewer air (for the Manchester Corporation).

3. Rôle of flies in the dissemination of certain pathogenic bacteria (Manchester Corporation).

4. Relative importance of imported food, home-grown fodder, manufactured manures, infection of the soil, in the causation of Anthrax (County Council of Cheshire).

5. Persistence of tubercle bacilli in dairy products manufactured with the milk of tuberculous cows (County Council of Cheshire).

6. Pollution of the River Lune and its dangers (County Council of Lancashire.)

7. Possible dangers of infection from the use of inferior bedding wool (flock) (County Council of Lancashire).

8. Causes of failure of sterilisation of milk by steam (Ashton-under-Lyne).

9. Influence of trade effluents upon the pollution of streams (Carlisle Rural District Council and other authorities).

10. A comparative study of the bacteriology and chemistry of various waters.

11. Standardising of disinfectants and relative value of various chemical disinfectants (Laboratory investigation).

12. Comparative study of various methods used for estimating the quantity of formaldehyde in food (by Dr. Ramsden, Research Fellow in Sanitary Chemistry), etc.

Some of these researches have been completed, others are still proceeding. An account of some of them will be found among the scientific papers published in connection with this report.

II. Researches by Laboratory Students.

The nature of the work done by various research students may be briefly summarised as follows:—

John W. Wells, M.D. (Edin.), Influence of cod liver oil, Sesame oil, petroleum, cod liver oil emulsion, and simple feeding upon tuberculosis.

R. Prosser White, M.D. (Edin.), Ætiology of coryza.

R. Lawrence ,M.D. (Vict.), Changes occurring in the central nervous system of patients affected with malignant tumours.

A. W. Latham, M.B. (Vict.), The changes occurring in organs in connection with invasion by secondary tumours (M.D. thesis, gold medal, 1904).

J. J. Butterworth (M.B.), The action of arsenic on cardiac and skeletal muscle and fat (M.D. thesis, 1904).

H. W. Russell (M.B.), Tuberculous infection of the lungs and bronchial glands in guinea pigs (M.D. thesis, gold medal, 1905).

A. C. Clarke (M.B.), Lesions produced in lymphatic glands by varieties of the tubercle bacillus (M.D. thesis, commended, 1905).

M. B. Arnold (M.B.), Diphtheria toxin and antitoxin (only partly prepared in the Bacteriological Laboratory (M.D. thesis, 1905).

Some of these contributions are published in the Archives of the Laboratory.

A selected list of scientific communications, based upon work done in the Bacteriological Laboratory since 1892 is given in Appendix II.

APPENDIX I.

AUTHORITIES CONNECTED WITH THE PUBLIC HEALTH LABORATORY, 1892 to 1905.

Places which have made temporary arrangement or required occasional assistance only and Public Institutions are indicated by italics.

No. of Reference	Date of Admission	Name of Place	Authority	Population 1901	Medical Officer of Health, Chairman of Committee under Public Authority, Superintendent or Secretary of Public Institutions.
54	1900	Abram	U. D.	6,306	Dr. E. Hannah
18	1898	Accrington	M. B.	43,095	Dr. A. Greenhalgh
64	1902	Adlington	U. D.	4,523	Dr. F. J. A. Mayes
9	1897	Alderley Edge	U. D.	2,856	Dr. G. W. Dowling
31	1898	Altrincham	U. D.	16,831	Dr. A. Golland
57	1901	Ashton-under-Lyne	M. B.	43,890	Dr. W. H. Hughes
33	1899	Ashton-upon-Mersey	U. D.	5,563	Dr. C. J. Renshaw
	1905	*Atherton*	U. D.	16,211	Dr. J. Marsh
50	1900	Audenshaw	U. D.	7,217	Dr. F. W. Allkin
	1903	*Baguley Sanatorium*			Dr. B. Rhodes
	1899	*Barnsley (Yorks.)*	M. B.	41,083	Dr. F. J. Sadler
66	1902	Barton-upon-Irwell	R. D.	8,068	Dr. A. E. Berry
10	1897	Blackburn	C. B.	127,527	Dr. A. Greenwood
42	1899	Blackpool	M. B.	47,346	Dr. F. J. H. Coutts
93	1897	*Bollington*	U. D.	5,244	Dr. D. W. Main
75	1900	Bolton	C. B.	168,205	Dr. J. E. Gould
	1901	*Bootle*	C. B.	58,558	Dr. T. W. N. Barlow
63	1901	Bowdon	U. D.	2,788	Dr. M. Duggan
61	1901	Bredbury and Romiley ...	U. D.	7,197	Dr. F. Cant
97	1899	*Bristol*	C. B.	328,842	Dr. D. S. Davies
21	1898	Bucklow	R. D.	19,885	Dr. T. W. H. Garstang
	1898	*Burnley*	C. B.	97,044	Dr. T. Dean
91	1900	Burton-upon-Trent	M. B.	50,386	Dr. J. M. Cowie
15	1897	Bury	C. B.	58,028	Dr. A. E. Brindley
58	1901	Bury Rural	R. D.	8,088	Dr. J. W. Cook
	1903	*Bury Dispensary*			Mr. F. A. Woodcock *(Secy.)*
	1905	*Carlisle*	R. D.	17,832	Dr. J. MacDonald
16	1898	Chadderton	U. D.	24,892	Dr. H. Ashton
51	1900	Cheadle and Gatley	U. D.	10,807	Dr. J. H. Godson
55	1900	Cheshire County Council ...	C. C.	601,970	
		Sanitary Committee ...	—	—	Dr. F. Vacher
	1094	Diseases of Animals Acts' Committee	—	—	Colonel Hamersley
	1900	*Chester Water Co.*			Mr. Wm. S. Moss (*Secy.*)
	1904	*Chorlton Guardians*			Mr. D. S. Bloomfield (*Clerk*)
26	1898	Congleton	M. B.	10,706	Dr. P. M. Davidson
8	1897	Crewe	M. B.	42,075	Dr. A. J. Laird
14	1897	Darwen	M. B.	38,211	Dr. F. G. Haworth
69	1902	Denton	U. D.	14.934	Dr. F. W. Allkin

98	1899	Derby	C. B.	105,785	Dr. W. J. Howarth
95	1898	*Doncaster*	M. B.	28,924	Dr. D. L. Anderson
44	1899	Dukinfield	U. D.	18,929	Dr. R. S. Park
3	1896	Eccles	M. B.	34,369	Dr. W. M. Hamilton
35	1899	Failsworth	U. D.	14,152	Dr. G. S. Leslie
	1902	*Faringdon (Berks)*	R. D.	11,626	Dr. F. E. Streeten
49	1899	Farnworth	U. D.	25,927	Dr. A. Kershaw
	1900	*Fylde Water Board*			Mr. C. Arthur *(Supt.)*
		Garstang		10,436	Dr. F. Fisher
	1902	*Grasmere (Westmorland)* ...	U. D.	781	Dr. R. M. Craven
	1898	*Guisborough (Yorks)*	U. D.	5,645	Dr. W. W. Stainthorpe
53	1900	Hale	U. D.	4,562	Dr. T. A. Rothwell
	1903	*Halifax*	C. B.	104,933	Dr. J. T. Neech
11	1897	Haydock	U. D.	8,575	Dr. T. E. Hayward
47	1899	Heaton Norris	U. D.	9,474	Dr. F. W. Jordan
40	1899	Heywood	M. B.	25,461	Dr. H. H. I. Hitchon
41	1899	Hollingworth	U. D.	2,447	Dr. W. E. S. Burnett.
90	1902	*Huddersfield*	C. B.	95,008	Dr. S. G. Moore
39	1899	Hyde	M. B.	32,768	Dr. J. Bennett
	1901	*Ilkeston (Derby)*	M. B.	25,383	Dr. J. J. Tobin
22	1898	Knutsford	U. D.	5,172	Dr. T. W. H. Garstang
	1903	Ladywell Sanatorium			Dr. J. W. Mullen
30	1898	Lancaster	M. B.	40,329	Dr. G. R. Parker
52	1900	Lancashire County Council ...	C. C.	1,827,436	
		Sanitary Committee ...	—	—	Dr. E. Sergeant
	1905	Diseases of Animals Acts' Committee	—	—	Mr. J. P. Muspratt *(Dep. Clerk)*
	1899	*Leeds*	C. B.	428,953	Dr. J. S. Cameron
	1902	*Leigh*	M. B.	40,001	Dr. J. King
36	1899	Levenshulme	U. D.	11,485	Dr. H. E. Edlin
67	1902	Little Lever	U. D.	5,119	Dr. J. S. Pickford
	1896	*Liverpool*	C. B.	702,235	Dr. E. W. Hope
65	1902	Lymm	U. D.	4,707	Dr. D. C. M. Lunt
2	(1892) 1896	Manchester Sanitary Committee..	C. B.	543,969	Dr. J. Niven
	,,	Manchester Rivers Committee ...			Sir Bosdin T. Leach
	,,	Manchester Ventilation of Sewers Committee			Councillor Dearden
	,,	Manchester Markets Committee			Alderman McCabe
62	1901	Manchester Port Sanitary ...			Dr. A. M. N. Pringle
		Manchester Royal Infirmary ...			Mr. W. L. Saunders (*Secy.*)
		Manchester Southern Hospital ...			Mr. G. W. Fox (*Secy.*)
48	1899	Marple	U. D.	5,595	Dr. H. Burton
27	1898	Middleton	M. B.	25,178	Dr. W. Graham
23	1898	Middlewich	U. D.	4,669	Dr. T. W. H. Garstang
56	1901	Milnrow...	U. D.	8,241	Dr. J. Chadwick

38	1899	Mossley	M. B.	13,452	Dr. J. Healey
7	1897	Moss Side	U. D.	26,677	Dr. S. H. Owen
71	1903	Nantwich	U. D.	7,722	Dr. J. D. Munroe
32	1898	Nantwich	R. D.	23,196	Dr. R. T. Turner
	1901	*Nelson*	M. B.	38,816	Dr. A. P. Millar
91	1900	*Newark-upon-Trent*	M. B.	14,985	Dr. C. Wills
	1905	*Newquay Water Co.*			Mr. G. G. Bullmore *(Secy.)*
12	1897	Newton-in-Makerfield	U. D.	16,699	Dr. H. E. Watkins
25	1898	Northwich	U. D.	17,609	Dr. H. E. Gough
73	1904	Northwich	R. D.	22,071	Dr. H. E. Gough
	1899	*Odiham (Hants.)*	U. D.		Dr. Reid
13	1897	Oldham	C. B.	137,238	Dr. J. B Wilkinson
	1902	*Oldham Infirmary*			Mr. E. L. Blake *(Secy.)*
34	1899	Oswaldtwistle	U. D.	14.200	Dr. E. Haworth
		Pendlebury Children's Hospital			Mr. H. J. Eason *(Secy.)*
74	1905	Poulton-le-Fylde	U. D.	2,223	Dr. J. Anderson
	1899	*Preston*	C. B.	112,983	Dr. H. O. Pilkington
18	1898	Prestwich	U. D.	12,835	Dr. F. Stephenson
43	1899	*Ramsbottom*	U. D.	15,920	Dr. W. Deans
17	1898	Rawtenstall	M. B.	31,052	Dr. J. E. Helm
45	1899	Rochdale	C. B.	83,112	Dr. J. Henry
		Royal Albert Asylum			Dr. A. R. Douglas
29	1898	Royton	U. D.	14,881	Dr. R. Young
68	1902	Runcorn	R. D.	23,224	Dr. J. Adams
70	1902	Sale	U. D.	12,088	Dr. O. Withers
5	(*1892*) 1896	Salford	C. B.	220,956	Dr. C. H. Tattersall
	1902	*Salford Coroner*			Mr. Arthur Holmes
	1903	*Selby*	U. D.	7,786	Dr. T. B. Stedman
96	1898	*Sheffield*	C. B.	408,994	Dr. H. Scurfield
1	1895	Stockport	C. B.	92,832	Dr. Meredith Young
6	1897	Stretford	U. D.	30,346	Dr. W. J. Heslop
60	1901	Swinton and Pendlebury ...	U. D.	27,001	Dr. S. Hosegood
19	1898	Todmorden	U. D.	25,419	Dr. C. W. Thorp
72	1904	Ulverston	R. D.	17,716	Dr. G. H. Patterson
20	1898	Urmston	U. D.	6,591	Dr. G. E. Fryer
	1902	*Uttoxeter (Staffs.)*	U. D.	5,135	Dr. H. Herbert
	1905	*Walton-le-Dale*	U. D.	11,271	Dr. R. Trimble
59	1899	*Whittingham Asylnm*			Dr. F. Percival
37	1898	Wilmslow	U. D.	7,361	Dr. T. A. Somerville
24	1898	Winsford	U. D.	10,382	Dr. T. W. H. Garstang
4	1896	Withington	U. D.	36,201	Dr. T. C. Railton
46	1899	Withnell	U. D.	3,349	Dr. Henry Case
		Manchester Markets			Mr. Alfred Holborn (*Inspector*)
		Manchester Police			Mr. Robert Peacock
	1898	*Saltburn-by-the-Sea*	U. D.	2,578	Dr. W. W. Stanthorpe

APPENDIX II.

A Selection of Original Publications by Workers in the Bacteriological Laboratory bearing upon Causes and Prevention of Disease. (1892 to 1905.)

A.—DISSERTATIONS.

1. M. B. Arnold.
   A Study of *Diphtheria* Toxin and Antitoxins and of the pharmacological action of the preservatives added to commercial antitoxins. (Dissertation for the M.D. Degree, 1905) (partly worked out in Bacteriological Laboratory).
2. James Charles Buckley.
   Laryngeal *Tuberculosis*. (Dissertation for the M.D. Degree, 1894.)
3. J. J. Butterworth.
   The *Action of Arsenic* on Cardiac and Skeletal Muscles and Fat. (Dissertation for the M.D. Degree, 1903.)
4. W. J. S. Bythell.
   Empyœma in Children. (Dissertation for the M.D. Degree, 1902.) *Gold Medal.* (*Med. Chron.*, 1902.)
5. J. R. Carver.
   The *Typhoid Bacillus* and its presence in the Excreta. (Dissertation for the M.D. Degree, Cambridge. *Med. Chron.*, 1898.)
6. A. C. Clarke.
   Research upon the Lesions produced in the Lymphatic Glands by *different varieties of the Tubercle Bacillus.* (Dissertation for the M.D. Degree, 1905.) *Commended.*
7. Harold Coates.
   Dissemination of *Tuberculosis* by means of Infected Dwellings. (Dissertation for the M.D. Degree, 1902.) *Commended. Transactions* of the British Congress on Tuberculosis, 1901.
8. John Smalley Dockray.
   Experimental *Calcification* (more specially in relation to Hydrargyrism) (Dissertation for the M.D. Degree, 1898.)
9. W. F. Jackson.
   The Action of *Arsenic* on the Kidney (1902).
10. John Mountford Johnson.
    Disinfection by means of Vapours of some members of the Phenol series. (Dissertation for the M.D. Degree, 1897.)
11. T. N. Kelynack.
    Pathology of the *Vermiform Appendix.* (Dissertation for the M.D. Degree, 1893.) *Gold Medal.*

12. W. J. Kerr.
The Effects of *Alcohol* on the Liver. (Dissertation for the M.D. Degree, 1895.) *Commended.*
W. J. Kerr.
The Effects of *Alcohol* on the Liver. (*Med. Chron.*, 1895–6.)

13. A. W. Latham.
The Changes occurring in Organs in connection with Invasion by *Secondary Tumours.* (Dissertation for the M.D. Degree, 1904.) *Gold Medal.*

14. Reginald Lawrence.
The Brain and Spinal Chord in *Chronic Arsenical Poisoning.* (Dissertation for the M.D. Degree, 1902.) *Commended.*

15. H. H. MacNabb.
Bacteriology of *Serpent Ulcers of the Cornea.* (Dissertation for the M.D. Degree, 1902.)

16. R. W. Marsden.
The Joint Affections in *Scarlatina.* (Dissertation for the M.D. Degree, 1896.)

17. F. Craven Moore.
Scrofulous Lymphadenitis. (Dissertation for the M.D. Degree.) *Gold Medal*, 1898. (*Journ. of Path. and Bact.*, 1899.)

18. J. C. Muir.
Blood and Bone-marrow Lesions in *Arsenical Poisoning.* (Dissertation for the M.D. Degree, Cambridge, 1901. *Journ. of Path. and Bact.*, 1901.)

19. W A. Newall.
The Serum Treatment and Sero-diagnosis of *Typhoid Fever.* (Dissertation for the M.D. Degree, 1900.)

20. Harry Osborne.
Suppuration in Children. (Dissertation for the M.D. Degree, 1902.) *Commended.*

21. Knowles Renshaw.
Nasal *Tuberculosis.* (Dissertation for the M.D. Degree, Cambridge, 1899. *Journ. of Path. and Bact.*, 1901.)

22. Frank Radcliffe.
Spread of *Tuberculosis* in Guinea-pigs. (Dissertation for the M.D. Degree, 1897.)

23. Thomas Andrew Rothwell.
Aspergillosis. (Dissertation for the M.D. Degree, 1899. *Commended.*

24. H. W. Russell.
The Relative Time of *Infection of the Lungs and Bronchial Glands* in Guinea-pigs inoculated with *Tuberculous* material. (Dissertation for the M.D. Degree, 1905.) *Gold Medal.*

25 J. Simcock.
Chronic Wasting of Children due to *Improper Feeding*. (Dissertation for the M.D. Degree, 1893.)

26 Francis Villy.
The Bone Marrow of *Cancer* Patients. (Dissertation for the M.D. Degree, Cambridge. *Journ. of Path. and Bact.*, 1896.)

B.—OTHER PUBLICATIONS.

27 William Milligan, M.D., C.M.
Tuberculous Disease of the Mucous Membrane of the Middle Ear and its Adnexa; on Experimental Investigations. (*Brit. Med. Journal*, 1895.)
Tuberculous Disease of the Middle Ear. (*Med. Chron.*, 1895–6.)

28 Thomas Harris, M.D., F.R.C.P.
On some cases of Chronic *Non-tuberculous Pneumonia* and the Clinical value of inoculation experiments of Guinea-pigs in the diagnosis of *Pulmonary Tuberculosis*. (*Brit. Med. Journal*, 1896.)

29 Arnold W. Lea., M.D., B.S., F.R.C.S.
Two cases of *Puerperal Septicæmia* due to *Streptococcic Infection*. (*Brit. Med. Journal*, 1899).

30 F. Craven Moore, M.D., M.Sc.
Hepatic Tuberculosis. (*Med. Chron.*, 1899.)
Pathological Aspects of *Acute Pancreatitis*. (*Med. Chron.*, 1899.)

31 Francis Villy, M.D.
An uncommon form of *Tumour* of the Thyroid Body. (*Journ. of Path. and Bact.*, 1896.)

32 F. J. H. Coutts, M.D., and A. Sellers, M.D.
Pseudo-Tubercle Bacilli in Milk. (*Public Health*, 1901.)

33 J. R. Carver, M.D,, D.P.H.
The Characters of *Yeasts occurring in Tanning Material*, etc, (*Archives of the Public Health Laboratory*. Vol. I.)

34 E. J. Sidebotham, M.A., M.B., and Arthur Sellers, M.D., D.P.H.
On an *Epidemic of Fish Poisoning*. (*Archives of the Public Health Laboratory*. Vol. I.)

35 G. J. Fowler, D.Sc., F.I.C.
The Application of Chemical Analysis to the Study of *Biological Processes of Sewage Purification*. (*Archives of the Public Health Laboratory*. Vol. I.)

36 W. B. Ramsden, B.Sc., M.B., Ch.B.
Two *Aldehyde Reactions*. (*Memoirs and Proceedings of the Manchester Literary and Philosophical Society*. Vol. 49, 1905.) (*Archives of the Public Health Laboratory*. Vol. I.)

37 Reginald Lawrence, M.D., D.P.H.
Changes occurring in the *Central Nervous System* of patients affected with *Malignant Tumours*. (*Archives of the Public Health Laboratory*. Vol. I.)

38 S. Delépine and P. R. Cooper, M.B., Ch.B., B.Sc., F.R.C.S.
A few facts concerning *Psorospermosis.* (*Brit. Med. Journal*, 1893.)

39 Sir T. Lauder Brunton, M.D., F.R.C.P., LL.D., F.R.S., and S. Delépine.
Report upon some of the Changes produced on *Liver Cells* by the *Action of some Organic and Inorganic Compounds.* (Proc. Royal Society, 55, 1894.)

40 S. Delépine and Arthur Ransome, M.D., F.R.S.
A Report on the *Disinfection of Tubercle-infected Houses.* (*Brit. Med. Journal*, 1893—95.)

41 S. Delépine and Arthur Ransome, M.D., F.R.S.
On the Influence of certain Natural Agents on the Virulence of the *Tubercle Bacillus.* (Proc. Royal Society, 56, 1894.)

42 S. Delépine and J. Richmond, M.A., M.B., M.R.C.S.
Variability of the "Comma Bacillus" and the *Bacteriological Diagnosis of Cholera.* (*Journ. of Path. and Bact.*, 1895.)

43 S. Delépine and E. J. Sidebotham, M.A., M.B.
On the *Sero-Diagnosis of Typhoid Fever.* (*Lancet*, 1896.)

44 J. Dixon Mann, M.D., F.R.C.P., and S. Delépine.
A case of *Tumour* of the Pons. (Brain, 1898.)

45 C. H. Tattersall, M.R.C.S., and S. Delépine.
Special Report on an Epidemic of *Arsenical Poisoning* from Beer, in 1900. (Salford, 1901.)

46 S. Delépine and J. H. Johnston, M.Sc.
The influence of Bacterial Multiplication, of Dilution, of Sedimentation, and of other factors on the *Natural Purification of Running Water.* (*Journl. of State Medicine*, 1901.)

47 W. J. Howarth, M.D., and S. Delépine.
Report on the Outbreak of *Food Poisoning* at Derby. (Derby, 1902.)

48 S. Delépine and Colin Campbell, M.R.C.S.
Treatment of Phthisis by Intratracheal Injections of Izal. (Trans. Brit. Congress on Tuberculosis, 1901.)

49 S. Delépine.
Psorospermosis as a possible cause of *Epithelial Tumours.* (Trans. 7th Congress of Hygiene, London, 1892.)

50 „ *Protozoa and Carcinoma.* (*Brit. Med. Journ.*, 1892.)

51 „ *Tuberculous Infection* through the Alimentary Canal. Address to the Medico-Ethical Society, Manchester, 1892. (*Med. Chron.*, 1895.)

52 „ On the value of *Experimental Tuberculosis* in Diagnosis. (*Brit. Med. Journ.*, 1893.)

53 „ On the Value of Bacteriological Diagnosis of *Asiatic Cholera.* (*Brit. Med. Journ.*, 1894.)

S. DELEPINE.

54 „ Spread of *Tuberculosis* through Lymphatics. (*Brit. Med. Journ.*, 1894 ; *Med. Chron.*, 1894.)

55 „ On the *disinfection of rooms* infected with Tuberculous Products. (*Med. Chron.*, 1894.)

56 „ Prevalence of *Tuberculosis* in the Domesticated Animals.(*Med. Chron.*, 1895.)

57 „ Report on the *Disinfecting and Antiseptic Properties of Izal.* (*Med. Chron.*, 1895.)

58 „ Bacteriological *Diagnosis of Diphtheria.* (*Med. Chron.*, 1895.)

59 „ *Anti-diphtheritic Serum*, with remarks upon *Artificial Immunity.* (*Med. Chron.*, 1895.)

60 „ Staining of *Tubercle Bacilli in Sections.* (*Med. Chron.*, 1896.)

61 „ Desirability of *Legislation* in connection with *Tuberculosis* of Living Domesticated Animals, and more specially of Cattle. (*Journ. of State Medicine*, 1896.)

62 „ Therapeutic Use of *Röntgen's Rays* (actions on Bacteria). (*Brit. Med. Journ.*, 1896.)

63 „ Sero-Diagnostic of *Typhoid Fever.* (*Med. Chron.*, 1896.)

64 „ On the value of different Bacteriological Methods of Diagnosis of *Typhoid Fever.* (*Brit. Med. Journ.*, 1896–97.)

65 „ The Technique of Serum Diagnosis, with special reference to *Typhoid Fever.* (*Brit. Med. Journ.*, 1897.)

66 „ The detection of *Pathogenic Properties of Milk.* (*Journ. of Comp. Path.*, 1897.)

67 „ Some Experiments on *Sterilization by Steam.* (*Journ. of State Medicine*, 1897.)

68 „ The *Bacteriological Diagnosis* of certain Infectious Diseases in connection with Public Health work. (*Lancet*, 1898.)

69 „ Some of the *Ways in which Milk becomes Pathogenic.* (*Brit. Med. Journ.*, 1898.)

70 „ *Disinfection.* (*Journ. of Sanitary Institute*, 1898.)

71 „ Bacteriological Survey of *Surface Water Supplies.* (*Journ. of State Medicine*, 1898.)

72 „ Tuberculosis and the *Milk Supply*, with some general remarks on the Dangers of Bad Milk. (*Lancet*, 1898.)

73 „ Some Experiments on the *Disinfection of Rooms* by Gaseous Formaldehyde. (*Journ. of State Medicine*, 1898.)

74 „ Note on a method used for separating the Bacillus Typhosus from Contaminated Soil. (*Public Health*, 1898.)

75 „ Effects of Pollution of Water Supplies. (*Med. Chron.*, 1899.)

76 „ Article on *Actinomycosis.* (*Encyclopædia Medica*; William Green and Sons, 1899.)

S. DELEPINE.

77 „ Some Remarks on the Effects of the *Pollution of Water Supplies.* (*Med. Chron.*, 1899.)

78 „ The *Prevention of Tuberculosis.* (*Public Health*, 1899.)

79 „ *Prevention of Tuberculosis in Cattle*; some Economic Aspects of the Question (2 papers). (*The Veterinarian*, 1899.)

80 „ Experiments on *Disinfection* by Rapid Currents of Saturated Steam (2 parts). (*Journ. of State Medicine*, 1900.)

81 „ *Bacteriological Diagnosis* of Disease. (*Lancet*, 1900.)

82 „ The *Application of the Tuberculin Test* to the Diagnosis of Tuberculosis in Cattle. (*Brit. Med. Journ.*, 1900.)

83 „ Some practical notes on the *Diagnosis of Human Plague.* (*Brit. Med. Journ.*, 1900.)

84 „ The *Detection of Arsenic* in Beer and Brewing Materials. (*Brit. Med. Journ.*, 1901.)

85 „ How can the Tuberculin Test be utilised for *stamping out Bovine Tuberculosis.* (*Lancet*, 1901.)

86 „ *Communicability of Human Tuberculosis to Cattle.* (*Brit. Med. Journ.*, 1901.)

87 „ Report on *Beer, Brewing, and other Materials.* Salford. (1900 and 1901.)

88 „ *Splenic Fever.* (*Encyclopædia Medica, XI.*; Green and Sons, 1902.)

89 „ *Concretions.* (*Quain's Dict.*, 1902.)

90 „ *Arsenic in Modern Life.* Address to the Sanitary Congress, 1902.

91 „ The Bearing of Outbreaks of *Food Poisoning* upon the Etiology of *Epidemic Diarrhœa* (Epidemiological Society). (Journal of Hygiene III. pp. 68—94.)

92 „ Report upon an alleged *Effluvium Nuisance* attributed to the use of yeast in a *Tannery*, and upon an Outbreak of *Diphtheria.* (Report to the Runcorn Rural District Council, 1903.)

93 „ Civilisation and *Health Dangers in Food.* (Lecture at the Royal Institution of Great Britain, 1903.)

94 „ *Protection* of Upland Gathering Grounds and *Filtration* compared. (Association of British Waterworks' Engineers, Bolton, 1903.)

95 „ Memorandum on *Anthrax*, presented to the Diseases of Animals Acts Committee of the County Council of Cheshire, 1904.

96 „ Report on Samples of *Water, Soils, and Mud* collected from the Estuary of the River Lune. (Public Health Committee of the County Council of Lancashire, 1904.)

97 „ The Causes of the increase of *Agricultural Anthrax* in Great Britain. (*Public Health*, 1905.)

And various other Reports to Public Authorities, some of which are in course of publication.

APPENDIX III.

List of Graduates and Undergraduates who have taken Public Health or post graduate courses, or have worked in the Bacteriological Laboratory from 1892 to 1905.

(Public Health and other appointments in a Public Service held in 1905 are indicated in black type. V = Veterinary courses. P.G. = Post graduate courses).

* Indicates that particulars could not be obtained from official sources in 1905. The names of those who were deceased in 1905 are in italics.

Years of attendance.	
1903, 1904,	ADAMS, JOSEPH, 1, Bewsey Road, Warrington, M.B., C.M. EDIN. **M.O.H. Runcorn R.D.C.**
1905 V.	ADAMSON, JOHN, 89, Bradshawgate, Bolton, M.R.C.V.S.
1904 P.G.	AINSCOW, JAMES, 224, Manchester Road, Ince, Wigan, M.R.C.S., L.R.C.P. LOND.
1895	AINSWORTH, HUGH, **Capt. I.M.S. Bengal,** late Asst. Medical Officer Monsall Fever Hospital, M.B. VICT., M.R.C.S., L.R.C.P.
1895	ALDRIDGE, A. H., Heatherlea, Torphins, Aberdeenshire, M.R.C.S., L.R.C.P. LOND.
1898	ALLAN, C. McA., Island House, Longton, Staffs., M.A. ABER., M.D., CH.M. EDIN.
1896-7	ALLKIN, F. W., 136, Ashton Road, Denton, Manchester, M.B., C.M. EDIN. **M.O.H. Denton and Audenshaw.**
1891-2	ANDERSON, A. J., Health Office, City Hall, Cape Town, South Africa, M.B. OXON., D.P.H. CAMB. **M.O.H. Cape Town,** late M.O.H. Blackpool.
1891-2	*ANDERSON, HENRY, M.A., M.D., R.U.I., 9, Beswick Street, Ancoats, Manchester.
1902	ANDERTON, W. B., Elsinore, Raynham Avenue, Didsbury, Manchester, M.B. LOND. **Demonstrator of Morbid Anatomy, Vict. Univ., Pathological Registrar Royal Infirmary,** Manchester.
1904 P.G.	ANGIOR, F. L., Standishgate House, Wigan, L.S.A.
1898-9	ARDERN, EDWARD, Urmston, Manchester, M.SC. VICT. **Chemist to Manchester Corporation Sewage Works.**
1900 1903—1904	ARNOLD, M. B., "Elmfield," Victoria Road, Whalley Range, Manchester, M.D. VICT., D.P.H. CAMB., **First Asst. Medical Officer Manchester Fever Hospital.**
1902-3	ASHCROFT, E. M., Public Health Department, Ford Street, Derby, M.B., CH.B., D.P.H. VICT. **Assistant to M.O.H. Derby.**

1896-7 ASHE, C. S., Northenden Road, Sale, M.B., CH.B., VICT

1895 ASHWORTH, J. H., Fernholm, 385, Cheetham Hill Road, Manchester, M.B., CH.B., D.P.H. VICT.

1892 P.G. 1894-5 ASHWORTH, J. W., Thorne Bank, Heaton Moor, Stockport, L.R.C.P., M.R.C.S.

1898 ATKINSON, J. R., Mirion House, Crewe, M.B., C.M. EDIN, D.P.H. GLASG.

1892 P.G. *BAILEY, ANTHONY, L.R.C.P., L.R.C.S., Openshaw, Manchester.

1900-1, 1902 BAILEY, J. G., Hayesleigh, Yelverton, M.B., C.M. EDIN.

1901—1903 BAXTER, A., 39, Sudell Road, Darwen, M.D. ABERD.

1893 BAYER, H. M., L.R.C.P., L.R.C.S. EDIN., L.F.R.S. GLAS.

1904 P.G. BECKITT, J. C., The Lindens, Leigh, Lancs., M.R.C.S., L.R.C.P. LOND.

1900, 1901 BENNETT, JAMES, Lynton Grange, Padgate, near Warrington, M.R.C.S. L.R.C.P., D.P.H. LOND.

1900, 1902 BENNETT, JOHN, Stockley Place, Chapel Street, Hyde, M.B., B.CH., R.U.I. **M.O.H. Hyde.**

1898-9 BENNETT R. A., 16, Diamond Street, Saltburn-by-Sea, Yorks., M.B. LOND.

1904 P.G. BENSON, M., 35, Dicconson Street, Wigan, M.D. BRUX., M.R.C.S. ENG., L.R.C.P. LOND.

1903-4 P.G. 1905 P.G. BIRD, WILLIAM, Lily Cottage, Briersfield, Lancs., M.B., C.M. EDIN.

1896-7 BISHOP, E. S., 189, High Street, Manchester, F.R.C.S. ENG., L.R.C.P. EDIN.

1904 P.G. BLAIR, JOHN, Bidston House, Up. Dicconson Street, Wigan, M.D., R.U.I.

1904 BLANDY, F. D., The Lingards, Anson Road, Victoria Park, Manchester, M.D. LOND., M.R.C.S., L.RC.P., D.P.H. VICT.

1894 BOOTH, GEORGE, Holywell House, Chesterfield, M.D. DURHAM, M.R.C.S., L.R.C.P., Vice-Chairman Health Comm., Chesterfield.

1904 P.G. BOYD, J. McA., Drumcroon, Park Road, Wigan, B.A., M.B., B.CH. R.U.I.

1904 P.G. BRADY, C. M., Sandville, New Market Street, Wigan, L.R.C.S.I., L.A.H. DUB.

1894 BRAZIL, W. H., 365, Blackburn Road, Bolton-le-Moors, Lancs., M.D. LOND., D.P.H. CAMB., B.SC. VICT.

1897-8 BRIGHTMORE, H. S., Limefield, Broughton Lane, Manchester, M.B., CH.B. VICT.

1894, 1896-7 1897-8 BRINDLEY, A. E., The Elms, Walmersley Road, Bury, M.D. LOND., B.SC. VICT., D.P.H. VICT. **M.O.H. Bury, late Asst. in Public Health Laboratory,** and Deputy M.O.H. Salford.

1892 P.G. BROADBENT, G. H., 8, Ardwick Green, Manchester, L.R.C.P.I., L.M., M.R.C.S. ENG.

1894 P.G. BROWN, E. V., Surrey Lodge, 2, Birch Lane, Longsight, Manchester, M.D. LOND.

1892 P.G. *BROWN, R. C.

1904 P.G. 1905 V. BROWN, J., 28, Standishgate, Wigan, M.R.C.V.S.

1904-5, 1905 BUCKLEY, G. G., Norwood, Oldham, M.B., CH.B. VICT., D.P.H. VICT. **Assistant M.O.H. Oldham.**

1893-4 BUCKLEY, J. G., 11, Goldsmith Street, Nottingham, M.D. VICT.

1905 BUCKLEY, S. M., Dental Student,

1898 BURGESS, A. H,, 23, St. John Street, Manchester, M.B., CH.B. VICT., M.SC., F.R.C.S. ENG. Asst. Surgeon Royal Infirmary and Christie Hospital, Manchester.

1894 BURNETT, W. E. S., Fairlie, Bowdon, L.R.C.P., L.R.C.S. EDIN. **Certifying Factory Surgeon, M.O.H. Mottram and Hollingworth U.D.C. and Tintwhistle R.D.C.**

1902 BUTCHER, W. J., Southview, Westhoughton, Lancs., M.R.C.S., L.R.C.P. LOND., D.P.H. VICT. **Asst. M.O.H. Westhoughton.**

1904 BUTLER, WM., Health Office, Stockport, Meat Inspector, Stockport.

1896-7 *BUTTERWORTH, H. P., L.R.C.P., L.R.C.S. EDIN., D.P.H. VICT.

1898, 1899 1901-2 BUTTERWORTH, J. J., 13, Moorfield Road, Pendleton, Manchester, M.D. VICT., D.P.H. VICT. **Asst. M.O.H. Salford, Lecturer on Hygiene and Physiology at the Royal Techninal Institute, Salford.**

1894 P.G. BUTTERWORTH, SAMUEL, **Major R.A.M.C.,** L.R.C.P. EDIN., M.R.C.S. ENG.

1897-8, 1900-1 BYTHELL, W. J. S., Woodcliffe, Prestwich, Manchester, M.D. VICT., B.A. CAMB. **Occasional Assistant in the Public Health Laboratory.**

1905 P.G. CALLAM, ALEX., 187, Colne Road, Burnley, M.B., CH.B. CH.B. ABERD.

1905 CALLAN, THOMAS, Massie Street Cheadle (Chemistry Student).

1902 CALLAND, T. B., M.B., CH.B. GLASG.

1903-4 P.G. 1905 P.G. CARTER, J. H., Church Street, Burnley, Lancs., F.R.C.V.S., F.R.S.E. **Veter. Insp. Board of Agric. and Fisheries, and County Palatine of Lancashire.**

1895, 1896, 1897 CARVER, J. R., The Meadows, Alderley Edge, M.D. CAMB., D.P.H. **Senior Asst. Public Health Laboratory.**

1905 P.G. CHADWICK, JOSHUA, 123, Oxford Road, Burnley, L.R.C.P., L.R.C.S. EDIN., L.F.P.S. GLAS.

1903-4 P.G. CHADWICK, HITCHON, 43, Oxford Road, Burnley, L.R.C.P., L.R.C.S. EDIN., L.F.P.S. GLAS.

1904-5 CHAPMAN, O. H., Ferncliffe, St. Catherine's, Lincoln, M.D., C.M. EDIN. D.P.H. VICT.

1897-8 CHORLTON, H. D., 97a, Oxford Road, Manchester, M.R.C.V.S. LOND.

1004 P.G. CHRONNELL, JAMES, Ash Cottage, Hindley, Lancs., M.R.C.S. ENG., L.R.C.P.I. **M.O.H. Hindley U.D.C.,** Medical Superintendent Hindley Fever Hospital.

1903, 1904 CLARKE, A. C., Roman Place, Higher Broughton, Manchester, M.D. VICT.

1895 CLEGG, J. G., 22, St. John Street, Manchester, M.D. LOND., F.R.C.S. ENG., L.R.C.P. LOND., late Asst. Demonstrator of Pathology, Owens College.

1903-4, 1905 P.G. CLEGG, J. W., Rosegrove, Burnley, L.S.A.

1899, 1900 CLEMENTS, J. A., Lytham House, Farnley, Leeds, M.B., B.CH., R.U.I., L.R.C.D.E., L.R.C.P.E. EDIN.

1905 CLEMENTS, R. W., **Captain R.A.M.C.,** Upper Chorlton Road, Manchester. B.A., M.B., CH.B.

1903 CLEMESHA, W. W., **Captain I.M.S. Bengal,** M.D. VICT., D.P.H. VICT., **Deputy Sanitary Commissioner Northern Bengal Circle.**

1897-8, 1899, 1900 COATES, HAROLD, Hornsey Municipal Office, Southwood Lane, Highgate, London, N., M.D. VICT., D.P.H. VICT. **M.O.H. Hornsey,** Med. Super. Hornsey Isol. Hospital, and Med. Officer Hornsey Education Auth., late M.O.H. Burton-upon-Trent, and Senior Asst. M.O.H., Manchester.

1894 COLLINSON, F. W., 32, Wickley Square, Preston, M.D. EDIN., F.R.C.S. EDIN.

1897 COOKE, J. A., Tue Brook Villa, Green Lane, Liverpool. M.R.C.S., L.R.C.P. LOND., D.P.H. EDIN. AND GLASG.

1902-3 COOPER, E. R., Thornecombe, St. Annes Road, St. Annes-on-Sea, M.B., CH.B. VICT.

1896 COOPER, H. G., Foye, Manchester Road, Altrincham, M.A., M.B., B.C. CAMB.

1893, 1894, 1895-6 COOPER, P. R., Glenthorn, The Downs, Bowdon, M.D. LOND., F.R.C.S. ENG., M.B. CH.B. VICT., B.SC. (HONOURS) LOND. **Late Asst. Demonstrator in Pathology Owens College.**

1904-5 CORE, DONALD, Groombridge House, Withington.

1891-2, 1899 1901 COUTTS, F. J. H., Public Health Office, Blackpool, M.D. VICT., D.P.H. VICT. F.C.S. **M.O.H. Blackpool,** Med. Super. Inf. Dis. Hospital, Blackpool, **late Asst. in Pub. Health Lab., Owens College,** Deputy M.O.H. Salford.

1903 COWAP, J. C. (Chemical Student).

1897-8 *COWIE, JOHN, M.B. Aber. Died May 5th, 1902.*

1894 CRAWFORD, J. H., 46, Ash Grove, Bradford, Yorks., M.D. EDIN., M.R.C.S., L.R.C.P., D.P.H. VICT.

1904, 1904 CRAWSHAW, C. W., Barwood Mount, Ramsbottom, M.B., CH.B. VICT., D.P.H. VICT.

1892 P.G., 1899 CRAWSHAW, SAMUEL, Trafalgar Square, Ashton-under-Lyne, M.B., CH.B. VICT., M.R.C.S. ENG.

1892, 1894 CROCKER, J. H., Petersham, Richmond, Surrey, M.D. VICT., D.P.H. VICT. **M.O.H. Richmond, London,** late M.O.H. Manchester Port Sanitary, and Eccles.

1904-5 CROMPTON H. J., County Asylum, Prestwich, M.D. VICT.

1903-4 P.G. CRUMP, T. G., 66, Bank Parade, Burnley, B.A. CAMB., M.B., B.C., M.R.C.S., L.R.C.P.

1897 CUNLIFFE, T. V., 76, Greengate Street, Oldham, M.D. LOND., M.R.C.S. ENG., L.R.C.P. LOND.

1896 *DAINE, H. S., 9, Spring Bank, Preston, **Lecturer at the Harris Institute, Preston.**

1896-7 *DAVIES, W. E., M.B., Ch.B. Vict., D.P.H. Vict. Died 1902.*

1894 *DICK. JOHN.

1905 P.G. DIXON, H. A., 141, Oxford Road, Burnley, M.R.C.S. LOND.

1899 DIXON, J. L. B., Bulli, New South Wales, M.D. VICT., D.P.H. VICT., late Asst. M.O. Brisbane.

1896, 1897, 1898 DOCKRAY, J. S., 4, High Street, Bishop's Stortford, M.D. VICT., B.SC. LOND. VICT.

1893 DOWZER, J. J. M., Fern Acre, Eccles, L.R.C.P.I., L.R.C.S.I., L.M., D.P.H. VICT., M.R.C.S., L.R.C.P.

1898, 1899 DREAPER, R. H., Wells, King's Co., New Brunswick, Canada, L.R.C.P.I., L.R.C.S.I., L.M., D.P.H.

1892 DREAPER, W. G., Park Villa, Grosmont, Yorks., M.R.C.S. ENG., L.R.C.P. LOND., D.P.H. VICT. **Certifying Factory Surgeon.**

1894 *DRUCE, E., **Teacher in Agricultural School, Preston.**

1903-4 P.G. DUN, H. W., 125, Netherfield Road, Nelson, M.B., C.M. EDIN.

1898, 1899, 1900 *DUNCAN, A. W., 42, Trevelyan Street, Eccles, F.C.S. **Public Analyst.**

1892 P.G. DUNN, J. E., 25*a*, Winckley Square, Preston, L.R.C.P. EDIN. M.R.C.S. ENG.

1896 DYSON, WILLIAM, Alison House, Broseley, Salop, M.B., CH.B. VICT.

1892 P.G. EDLIN, H. E., Middlemore House, Stockport Road, Levenshulme, M.R.C.S. ENG., L.R.C.P. LOND. **M.O.H. Levenshulme.**

1903-4 P.G. EDMONDSON, HERBERT, Fernhill, Burnley, B.A., M.B., B.C. CAMB.

1892 P.G. EDWARDS, JOHN, 171, Cheetham Hill Road, Manchester, M.R.C.S. ENG., L.R.C.P. EDIN. **Prin. M.O., H.M. Prison, Manchester.**

1897, 1898, 1899 EDWARDS, N. F., Broseley, Salop, M.B., CH.B. VICT.

1894 *EDWARDS, WILLIAM, Agricultural School, Preston.

1897-8 *ENTWISTLE, A. L., Analytical Chemist.

1905 P.G. FALCONER, ALEX., Tyrconnel House, Earby, nr. Colne, M.B., C.M. ABERD.

1905 V. FAULKNER, EDWIN, 6, Penrhyn Terrace, Stretford Road, Old Trafford, M.R.C.V.S. **Veter. Insp. Stretford,** late Veter. Insp. of Meat, etc., Manchester, Veter. Insp. to Board of Agric. and Fisheries and Moss Side Urban District.

1896-7 FENN, R. M., 147, Cheetham Hill Road, Manchester, M.B., C.M. EDIN.

1895 *FERGUSON, HOWARD.

1903-4 P.G. 1905 P.G. FERGUSON, J. M., 72, Colne Road, Burnley, L.R.C.P., L.R.C.S. EDIN., D.P.H. DURHAM.

1905 P.G. FINDLAY, ANDREW, 20, Carr Road, Nelson, M.B., C.M. GLASG.

1898-9 FINNEY, A. E., Barwood, Wilmslow, M.D. VICT., **late Demonstrator in Pathology, Owens College, Hon. Asst. Pathologist Cancer Hospital,** Manchester.

1893-4 FLETCHER, J. H., Bedford House, Ince, nr. Wigan, M.R.C.S., L.R.C.P. LOND.

1903 FORBES, DUNCAN, **M.O.H. Cambridge,** M.D. EDIN., B.SC. (PUBLIC HEALTH), EDIN., D.P.H. CAMB. late Asst. M.O.H. and Deputy Port M.O. Leith, Asst. M.O.H. Manchester.

1903 FOSTER, B. LEN., c/o Messrs, F. C. Calvert & Co., Bradford, Manchester, B.A. CAMB.

1895-6 FOWLER, G. J., Broad Oak, Urmston, Manchester, D.SC. VICT., F.I.C. **Lecturer in Bacteriological Chemistry and Senior Chem. Asst. Public Health Laboratory, Consulting Chemist to Manchester Sewage Works.**

1892 *FRASER, W. M., M.B., C.M., D.P.H. VICT.

1895, 1903-4 P.G. 1905 P.G. GARDNER, JAMES, Thorn Hill, Burnley, M.B., C.M. GLAS., late M.O.H. Royton.

1900 GARNER, CHAS., Watford Villa, Irlam Road, Flixton, M.D. VICT.

1904 GARNETT, J. H., Springfield, Ramsbottom, B.SC. VICT., Chemical Student.

1894 GARRETT, P. G., Earl Shilton, nr. Hinckley, M.B., B.S. DURHAM, D.P.H. VICT.

1896, 1897 GARSTANG, T. W. H., Edge Mount, Altrincham, M.A. OXON., M.R.C.S. ENG. L.S.A., D.P.H. VICT. **M.O.H. Biddulph, Knutsford, Middlewich, Winsford U.D.C. and Bucklow R.D.C.** late M.O.H. Northwich R.D.C.

1894 GARTH, JOHN, 1, Avenue Terrace, Preston, L.R.C.P.I., L.M. **M.O.H. Fulwood.**

1900-1 GAULT, A. H., 8, North Terrace, Adelaide, South Australia, M.D. LOND. **M.O.H. Mitcham.**

1899, 1900 * GILL, J. A., Analytical Chemist.

1905 P.G. GLASHAN, A. C., 114, Westgate, Burnley, M.B., CH.B. ABER.

1896, 1897 GODSON, J. H., Linden House, Cheadle, M.B., B.C. CAMB., D.P.H. CAMB. **M.O.H. Cheadle and Gatley.**

1903-4 GOLDSMITH, B. K., **Asst. to M.O.H. Manchester,** late Asst. Medical Officer Fever Hospital, Monsall, Manchester, M.B., CH.B. EDIN., D.P.H. VICT.

1894 *GORDON, JAMES.

1903 GORDON, REGINALD, Everest House, Great Crosby, Lancs., M.B., CH.B. VICT.

1892 P.G. GOULDEN, J. H. O., 8, Greek Street, Stockport, L.F.P.S. GLAS., L.M.

1892 P.G. GOWLAND, G. R., 118, Wilmslow Road, Rusholme, L.R.C.P., L.R.C.S. EDIN. Late M.O.H. Warsop.

1904 P.G. GRAHAM, C. R., Netherby House, Wigan, M.R.C.S. ENG., L.R.C.P. EDIN.

1904 GRAY, W. C., St. John's Lodge, Blackburn, M.B., CH.B. ABER. **Lieut. Indian Medical Service.**

1893 GREENHALGH, ARTHUR, 1, Brunswick Terrace, Blackburn Road, Accrington, M.B., CH.B., D.P.H. VICT. **M.O.H. Accrington.** Superintendent Smallpox Hospital.

1893 GREENHALGH, HANDEL, 155, Rochdale Road, Bury, L.F.P.S. GLAS., L.M.

1895, 1896, 1899, 1900 — GREENWOOD, ALFRED, Health Office, 51, Ainsworth Street, Blackburn, M.D. VICT., D.P.H. **M.O.H. Blackburn,** M.O. Educ. Auth., Lecturer on School Hyg., Technical School, Blackburn, **late Asst. Public Health Laboratory,** late Asst. M.O.H. Stockport and M.O.H. Crewe.

1898-9 — GREGORY, THOS., 6, Bury Old Road, Cheetham Hill, Manchester, M.R.C.S., L.R.C.P. LOND.

1903-4 P.G. 1905 P.G. — *GRIFFIN, S. M., Bank Parade, Burnley, L.R.C.P., L.R.C.S. EDIN.

1900 — GRUNER, O. C., Director of the Yorkshire Pathological Laboratory, Honorary Pathologist Leeds Public Dispensary, Leeds, M.B. LOND.

1897-8 1898 — HALL, J. P., 7, Eaton Road, West Derby, Liverpool, M.B., B.S. LOND., M.B., CH.B. VICT., L.R.C.P., M.R.C.S.

1902-3 — HALL, I. WALKER, **Pathologist to the Bristol Royal Infirmary, Professor of Pathology, University College,** Bristol, late Asst. Lecturer in Pathology Victoria University.

1892, 1895 — HAMILTON, D. L., Frohenlog, Dolgelly, North Wales. L.R.C.P. EDIN., F.R.C.S. EDIN.

1900 — HAMILTON, W. M., Pendleton Lodge, Patricroft, M.D., M.CH., D.P.H. DUBLIN. **M.O.H. Eccles.** J.P.

1896-7 — HARING, N. C., 112, Wilmslow Road, Rusholme, M.B. LOND., B.SC. VICT.

1895-6 1896-7 — HARRIS, THOMAS, 325, Oxford Street, Manchester, M.D. LOND., F.R.C.P. LOND., M.R.C.S. ENG.

1896-7 — *HARRISON, —, County Council Farm, Preston.

1902-3 — HARWOOD, H. M., London, M.B., B.C. CAMB.

1899 — HAWORTH, F. G., Vale House, Darwen, M.B., C.M. GLAS., D.P.H. CAMB. **M.O.H. Darwen,** Med. Supt. Inf. Dis. Hospital, M.O. Educ. Auth. Darwen.

1904-5 — HAWORTH, H. D., 166, Scotland Road, Nelson, M.B., CH.B. VICT.

1905 — HEALD, O. W., Dental Student.

1905 — HEAP, HARRI, 3, Thursby Square, Burnley, Chemical Student.

1904 P.G. — HEYES, J. P., 1, Bridgewater Terrace, Wigan, F.R.C.V.S.

1902-3 — HEYWOOD, C. C., Earnscliffe, Alderley Edge, M.A., M.B., B.C. CAMB., late Lecturer on Physiol. and Hyg., Salford Royal Tech. Inst.

1899 — HEYWOOD, T. W., Lower Bank, Darwen, L.R.C.P.I., M.R.C.S. ENG., D.P.H. J.P. **Certifying Factory Surgeon.**

1899, 1900, 1901 — HILL, E. F., 1, High Street, C.-on-M., Manchester, M.B., CH.B. VICT., M.R.C.S., L.R.C.P.

1905 — HINSON, H., Dental Student.

1903-4 P.G. — HODGES, A. E., 11, Yorkshire Street, Burnley, L.R.C.P., L.R.C.S. EDIN.

1903-4 P.G. 1905 P.G. — HODGSON, JOHN, 20, Gannow Lane, Burnley, L.R.C.P., L.R.C.S. EDIN.

1896-7 1903 — HOLBURN,, ALFRED, City Abattoirs, Water Street, Manchester, M.R.C.V.S. **Chief Veterinary Inspector, Manchester, Examiner and Meat Insp. Roy. San. Inst., Lond., Lect. Meat Insp. Tech. Schools, Manchester.**

1900-1 HOLMES, W. S., 31, Wellington Street, Bradford, Manchester, M.B., CH.B. VICT.

1903, 1904 HOLT, HENRY, 98, Wellington Road, Heaton Chapel, M.B., CH.B. VICT., D.P.H. LOND. **Deputy to M.O.H. Stockport.**

1898, 1899 HOLT, J. J. H., The Lodge, Middleton, M.B., CH.B. VICT., D.P.H. DUBLIN.

1897 HOLT, R. C., 60, Bank Parade, Burnley, M.D. DURH., F.R.C.S. EDIN., M.R.C.P., L.R.C.P.

1894, 1903-4-5 P.G. HOLT, THOMAS, 36, Colne Road, Burnley, M.B., C.M. ABERD., D.P.H. GLASG. **M.O.H. Burnley.**

1905 HOPKINSON, S. M., Dental Student.

1896-7 HORROCKS, OSWALD, Nagda Muttra State Rly., Sewai, Madhopun, Rajputana. F.R.C.S. EDIN., L.R.C.P., L.R.C.S. EDIN.

1898-9 *HORSFALL, J., 4, Grange Avenue, Rawtenstall, F.C.S. **Consulting Chemist,** Chem. Master Tech. Even. Schools, Rawtenstall.

1905 HOUGHTON, E., Dental Student.

1903, 1904 HOWARD, JAMES, Royal Infirmary, Manchester, L.R.C.P., M.R.C.S. ENG.

1896 HOWARTH, W. J., Public Health Office, Ford Street, Derby, M.D. VICT., D.P.H. VICT. **M.O.H. Derby,** Med. Supt. Isol. Hospital, M.O. Educ. Auth., late M.O.H. Bury.

1900 HOWLES, FRED, 38, Glenhue Road, Blackheath, London, M.SC. VICT.

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1902, 1903, 1904 HUTCHINSON, J. R., Eye and Ear Infirmary, Liverpool, M.B., CH.B. VICT., D.P.H. VICT.

1899, 1900 *HYDE, ELLIS, Analytical Chemist.

1895-6, 1897-8 INGRAM, J. W., Stockport Road, Ardwick, Manchester, M.R.C.V.S.

1892 IRVING, L. A., **Lieut.-Colonel A.M.S.** (retired), M.R.C.P., L.R.C.S. IRELAND, D.P.H. VICT.

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1895, 1896 JOHNSON, J. M., 31, Ball Haye Street, Leek, Staffs., M.D. VICT. **M.O.H. Leek U.D.C., Factory Surgeon.**

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1897-8 JOHNSTON, J. H., Public Office, Hampton Middlesex, M.SC., F.I.C., **Chemist and Bacteriologist Hampton U.D.C.,** late Asst. Chem. and Bact. to Worcestershire C.C.

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1894 JONES, HERBERT, Hereford, L.R.C.S.I., L.M., D.P.H. CAMB. **M.O.H, Hereford combined District and Bromyard U.D.**, Med. Supt. Hereford Isol. Hospital, Hon. Bact. Herefordshire Gen. Hospital, **Certifying Factory Surgeon,** late M.O.H. Rhondda U.D. and Crewe.

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1898 NICHOLS, F. P., **Lieut.-Colonel R.A.M.C.,** B.A., CAMB., M.B. CAMB.

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1892 P.G. *O'GORMAN, R. P., L.R.C.P.I., L.M., L.R.C.S.I.

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1900, 1901 OSBORNE, HARRY, Eston, R.S.O., Yorks., M.D. VICT., M.R.C.S., L.R.C.P.

1904, 1905 PARK, J. R. S., 183, King Street, Dukinfield, L.R.C.P., L.R.C.S. EDIN. **M.O.H. Dukinfield.**

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1894 1895-7 PLATT, J. E., 191, High Street, Oxford Road, Manchester, M.D. LOND., M.S. LOND., F.R.C.S.

1894 PORTER, CHARLES, Municipal Buildings, Johannesburg, M.D. DUB., D.P.H. CAMB. **M.O.H. Johannesburg, M.O. Johannesburg and Rand Plague Committee,** late Asst. M.O.H. E. Kent, M.O.H. Stockport, and M.O.H. Shropshire.

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1897-8, 1904-5 RAMSDEN, W. B., 287, Wilmslow Road, Fallowfield, Manchester, M.B. CH.B. VICT., B.SC. VICT. (HONOURS). **Chemical Asst. Public Health Laboratory, Victoria University, Research Fellow in Sanitary Chemistry.**

1895-6, 1897-8 1900-1 RAY, J. H., 11, St. John Street, Manchester, M.B. VICT., F.R.C.S. ENG. **Late Medical Officer Salford School Board.**

1897-8, 1898-9 RENSHAW, J. A. K., 11, St. John Street, Manchester, M.B., B.C. CAMB., M.A., D.P.H. CAMB.

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1902, 1903 RODGERS, F. M., Lancashire County Asylum, Winwick, Warrington, M.B. CH.B. VICT., D.P.H. VICT.

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1896-7 *SCOTT, H. A., M.B., Ch.B. Vict., D.P.H. Died August 24th, 1900.*

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1904 P.G. *STEVENS, G. H., M.R.C.S., L.R.C.P.

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