is performed under light general anaesthesia, and, as a rule, it is best, though by no means always necessary, to complete the delivery with forceps.

The care and skill with which injuries are repaired are obviously important factors in the prevention of subsequent disability. The technique of repair is beyond the scope of this paper, but in passing I would endorse B. P. Watson's teaching that where a difficult or extensive repair operation is necessary it may be advisable not to carry this out during, or immediately after, the third stage of labour. If the confinement has occurred during the night in a private house and the attendant is single-handed, a better result is likely to be obtained if the operation is postponed till the following day, when the operator has the advantages of adequate assistance, an anaesthetist, fresh sterile dressings and lotions, and a more bloodless field in which to work.

After-care in the Prevention of Prolapse

Where obstetrical injury, especially of a serious nature, has been sustained much can be done by care and guidance in the convalescent period to minimize the incidence and extent of subsequent disability. In the after-care of the patients the importance of an adequate period of recumbency calls for special emphasis. In all cases the parturient should be kept in bed at least until the uterus is no longer palpable in the abdomen and the lochial discharge no longer contains fresh blood, and where more than superficial injury has been sustained a fortnight may be considered the minimum time necessary for sound healing to occur.

In this connexion the practice of getting patients up on the sixth or seventh day and discharging them on the eighth or ninth day after confinement, which many maternity hospitals are forced to adopt owing to shortage of beds, is greatly to be deplored. After the lying-in period is over the patient should be given clear and precise instructions in regard to her conduct of life generally, and in particular in regard to the amount of rest and the amount and character of exercise that is advisable during the three months which follow the confinement. I do not forget the circumstances which make it difficult for the patient of the hospital class to secure the amount of rest she should have, and it is largely because this is, as a rule, totally inadequate that the incidence of genital prolapse is relatively so much higher in this class of patient.

Massage is of value, and remedial exercises, with which the patient should be encouraged to persevere for three months, will do much to restore tone to muscles that have been over-stretched or damaged. A well-fitting corset, by giving adequate support to the abdominal organs, will also minimize the harmful effect of intra-abdominal pressure, especially in the obese patient. Bronchitis and constipation are common causes of strain requiring attention, and every means should be employed—including, for example, such a diet as Mellanby has advised—to promote the patient's general health, and in particular to improve her general muscular tone.

A pessary may occasionally arrest the tendency to prolapse of the uterus, especially if the organ is also retroverted, but the value of its employment as a routine procedure in incipient prolapse is open to question, for if it is large enough to be effective it may interfere with the normal shrinkage of the vaginal walls, and at a stage at which it might prove useful the vaginal tissues are frequently too sensitive to allow of its being worn without discomfort. If a pessary is employed the smallest size that is effective should be used, so as to interfere as little as possible with involution of the vagina, and it should be replaced at frequent intervals by smaller sizes until involution is complete.

Is Genital Prolapse always Preventable?

While it is true that in many cases of genital prolapse the conduct of the patient's previous child-bearing may invite legitimate criticism, the development of this lesion is by no means always preventable. In certain individuals, for example, the tissues are abnormally friable so that it is impossible to avoid tearing, or inelastic so that after labour the return to normal is incomplete. Another less common type of case is that in which a rigid cervix is forced down in front of the head almost to the vulva before dilatation is complete, so that over-stretching of the fascial supports around the cervix is liable to occur and the consequent development of prolapse difficult to avoid. And, lastly, there are rare occasions when it is not only advisable but essential to hasten delivery, and where conservation of the uterine supports may properly be subordinated to the more immediate interests of mother and child. Skill and good judgement in the management of labour, together with careful after-treatment where injury has been sustained, will do much to prevent the occurrence of prolapse, but the disability may develop despite scrupulous observance of all the canons of a sound obstetric practice, and criticism should not be entertained, far less expressed, in ignorance of difficulties which may have had to be faced.

ARTIFICIAL SILK KERATITIS

BY

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The keratitis about to be described occurs at a certain stage in the manufacture of artificial silk by the "viscose" process. This paper deals with the condition as it was observed in a large artificial silk factory. The factory began production in 1927, and the study of the cases started early in 1928. Full capacity for the production of artificial silk was not attained during 1928, so that greatly increased numbers of cases were met with in 1929. In all 1,598 cases were observed in these two years.

Symptoms, Signs, and Treatment

The earliest symptom was blurring of vision, with the appearance of haloes round lights. It was, too, the most important symptom, because it stamped the affection as a corneal one from the first. In a comparatively small number of cases recovery took place without the development of further symptoms, and, there being no slit-lamp available, the only objective sign was conjunctival injection, which was never very severe; workers would then say that they had been threatened with an attack, but it passed off. In the great majority, however, the condition progressed, and the blurring was followed in from half an hour to several hours by a feeling of grittiness under the lids, photophobia, lachrymation, often blepharospasm, frequently drooping of the lids without spasm, dull aching at the back of the eyes, and headache. The lachrymation tended to be profuse, and, where there was blepharospasm, the tears gushed out when the lids were separated, the separation sometimes requiring considerable force.

The pathology was as one might easily surmise from the symptomatology; in those cases which went on to irritation, fluorescein staining showed more or less desquamation of the corneal epithelium on the centre of the cornea. The destruction of tissue was essentially superficial. The keratitis was always accompanied by circumcorneal injection and by conjunctival injection of all degrees. The breath of those who were affected was

pungent, the odour being curiously suggestive of onions. That this may not have been without significance is suggested by the fact that at one stage of manufacture there is produced a substance which will be described later—thio-formaldehyde.

The manner of onset of the symptoms and its place in the industrial week are of such importance that they will be fully described later. The affection was transient, the worst cases clearing up in less than four days, and most of them in less than three; no corneal scarring was found.

In treating these patients removal from the spinning room or acid house was almost always necessary; the most welcomed active treatment was the application of a weak cocaine ointment, such as 0.5 per cent. in ung. zinci. This was found to be at least as effective as any of the many lines of treatment suggested by other writers.

Problem of Aetiology

In order to discuss the aetiology of this condition it is necessary to understand a little about the process by which artificial silk is made; but as the workers affected are in only two of the departments—the spinning room and the acid house—these departments only will be described in detail.

PROCESS OF MANUFACTURE

Wood-pulp is delivered at the factory in the form of sheets, not unlike coarse cardboard. These are soaked in a strong solution of caustic soda, and the excess is then separated off by pressure. The pulp is macerated in grinding machines into "crumbs," which are treated with carbon bisulphide to form cellulose xanthate. The latter is first soaked in 18 per cent. and then dissolved in 4 per cent. lye; as viscose it is now ready for the spinning room. Here it is forced through nozzles into an acid bath containing 10 per cent. sulphuric acid and various salts. Viscose consists of 7 to 8 per cent. of cellulose, 7 to 8 per cent. of lye, and water; when it comes in contact with the acid it is coagulated, and in this reaction H_2S , CS_2 , and other gases are given off (the amount of CS_2 , however, is negligible so far as injury to health is concerned). The spinner seizes the coagulated viscose as it emerges from the nozzle and "spins in" by guiding the strand over a pulley, whence it passes into a spinning box, where it is spun into "cakes." When the "cakes" are fully spun they are removed by "cake-changers," who place them in drying ovens. After drying the thread goes through further processes of bleaching, etc., which, interesting as they are, do not concern us.

THE SPINNING ROOM

The spinning machines are arranged in rows, and occupy practically the entire floor space of the spinning room. A diagrammatic sketch will be sufficient to illustrate a machine.

Diagram of spinning machine.

The machine consists of the following parts:

1. A narrow trough of water in which the spinner dips his hands to get rid of the chemicals.
2. A wider trough—the spinning bath—containing 10 per cent. sulphuric acid.
3. A series of viscose pipes, which are bent over to be immersed in the spinning bath. At the end of each pipe is a nozzle, which is finely perforated so that the coagulated thread of viscose will contain a number of fibres corresponding to the number of perforations. The thickness of the silk thread is thus determined.
4. A vertical back-plate, in front of which, near the top, is a rotating shaft on which are mounted at intervals corresponding to the nozzles a series of glass pulleys or gaudets. The threads pass over these gaudets and then vertically downwards to
5. A series of spinning boxes, each containing a rapidly rotating "pot," in which the silk is spun into a "cake."

The spinner works in front of the machine. The acid bath is below waist level and the gaudets are at about face level. The boxes are on the same level as the bath. The "cake-changers" work at the back of the machines. The temperature of this room is usually about 70° F., and the air is artificially kept at a humidity of about 80 per cent. Owing to the large proportion of water in the viscose, the spinning bath contents become diluted and therefore have to be reconditioned; for this reason the acid is sent to the acid house.

THE ACID HOUSE

Here the acid is received into large tanks. A proportion of it is heated to 70° C., and by evaporation becomes concentrated. Fans operate to draw off the fumes from this acid. The concentrated acid is then mixed with the rest, further salts are added, and the solution is returned to the spinning room.

Possible Causal Factors

The keratitis affected the spinners and the "cake-changers" in about equal proportions. It occurred, uncommonly, in the acid house too. It has been attributed to various things, but one or all of three different causes have been generally favoured:

1. The thread in its passage from bath to spinning box vibrates rapidly, and a fine spray of acid is thus given off; these droplets are held by some to be the main cause.
2. The vapours from the bath are blamed by others.
3. The irritant has been said to reach the eyes of the men when they wipe away perspiration with their fingers.

Strebel,¹ in describing the condition as keratitis punctata superficialis traumatica, blamed H_2S , which, he thought, attacked the conjunctiva and the cornea either directly or via the lachrymo-nasal route through inhalation (this method affecting only very susceptible workers), and droplets. He constructed masks consisting of feuзал-glass goggles and double treillage containing cotton-wool impregnated with powdered charcoal and zinc oxide, to absorb and neutralize the gases. He found these satisfactory when ventilation was bad; he also recommended the erection of hoods to intercept the spray.

Eva Klein,² in a study of over 200 cases, gave the order of symptoms as: first, sudden severe pain in the globe of the eye; secondly, lachrymation; and thirdly, blepharospasm. Objectively, the interpalpebral conjunctiva was described as intensely red, sometimes showing punctiform haemorrhages. The cornea took on a grey appearance, and, if one were lucky enough to obtain a proper view, showed pin-head elevations of the epithelium. She noted that there were no complications such as iritis, etc., and for treatment recommended atropine and butyn. Further, she mentioned that only a proportion of workers were susceptible. Having visited a "perfectly ventilated" factory at Strasbourg, where keratitis still occurred, she formed the conclusion that this proved that droplets were more responsible than vapours. In support of this conclusion she referred to experiments in which a solution made to resemble the spinning bath was used in an atomizer and rabbits' eyes were exposed to the spray. Keratitis followed—not unnaturally.

Colrat³ divided his cases into two types. In the first and commonest there were superficial burns of the conjunctiva, and sometimes of the cornea, caused by caustic vapours. In the second there were more serious burns, caused by the direct projection on the eyes and adnexa of products of the process. He subdivided the first group into about 400 mild cases without corneal lesions and 200 with corneal lesions; he thought that ventilation was of prime importance, and that chronic blepharitis and lachrymal obstruction were predisposing factors. In our series the second type mentioned by Colrat has not been included, as these cases are regarded as frank accidents under the description of "viscose splashes." These splashes produced typical caustic burns, involving always the conjunctiva as well as the cornea.

Further Important Points

At this factory important points were noted which do not seem to have attracted attention elsewhere.

First, about two-thirds of the men appeared to be immune. Searching examinations were made in the hope of being able to discover predisposing factors, but nothing was found to stamp one man as being more likely than another to be a victim. Chronic blepharitis and lachrymal obstruction are not common among male workers, and certainly were not factors in predisposition in this series. Errors of refraction had no influence. Weather conditions, variations in the temperature, and alterations in the humidity of the spinning room had no relationship to the number of cases.

The method of onset of symptoms was specially studied. The table shows very clearly that there was a peak period in each week, in which the vast majority of cases occurred. The working day was of twenty-four hours, divided into three shifts of eight hours on and sixteen off. Production commenced at 10 p.m. on Sundays and continued till midday on Saturdays. From the table, then, it will be

Table showing on which day of the week symptoms appeared in 755 consecutive cases (January–August, 1929)

Monday	Tuesday	Wednesday	Thursday	Friday	Saturday
14	98	264	278	94	7

seen that almost invariably at least two eight-hour exposures were necessary to produce symptoms, and, very curiously, even in a bad week, few cases occurred at the end of the week. This suggests that, given sufficient cause present, three or four exposures will produce keratitis in susceptible workers. An important qualification must be mentioned here. During a strike overtime was worked, the shifts then being twelve hours. In that week keratitis was earlier and much more severe.

The other important point in connexion with the manner of onset is this. Symptoms very frequently commenced not during exposure but after leaving work. Many workers even went home and to sleep without symptoms and found blurring present on awakening. From these observations two facts stand out clearly:

1. In the great majority of cases two or three exposures of eight hours each were necessary to produce symptoms.
2. There is a latent period of varying length between exposure and symptoms.

Clinical Picture

Now considered as a whole artificial silk keratitis presents the following features of particular interest:

1. Oedema or irregular swelling of the corneal epithelium, evidenced by blurring of vision, haloes, etc., and actually described by Klein as pin-head elevations of the epithelium.
2. Desquamation of the epithelium.
3. Rapid and complete repair.
4. In mild cases an interruption of the process, followed by a return to normal without desquamation.
5. The condition follows repeated exposures of eight hours each, separated by periods of sixteen hours—that is, less than twenty-four hours.
6. A latent period precedes the onset of symptoms.

There is thus a clinical picture remarkably like that of photophthalmia, which is characterized by a chromatolysis with oedema and swelling of the cytoplasm, formation of inclusion bodies in the nucleus, and, after rupture of the nuclear membrane, death of the cell. Describing this abiotic lesion Duke-Elder⁴ says:

"... after mild exposures the process may stop at any stage and the cells return to normal in appearance and staining reaction. Two features of the process of resolution are characteristic: first, the rapidity of the recovery of the

traumatized cells and of the proliferation of fresh cells to replace those exfoliated, and, second, the comparative absence of mitotic activity in the process of repair";

and, a little earlier,

"... the effect of repeated exposures with intermissions is equal to their equivalent as a single long exposure, always provided that the intervals do not exceed twenty-four hours, beyond which time the factor of physiological repair comes into play. The reaction comes on after a latent period, the length of which varies with the intensity of the exposure, but averages six to eight hours. The latent period is of the nature of a photochemical induction, a practically universal feature in photochemical reactions whereby a change does not assume at once a constant value, but increases, at first slowly, before a constant level is attained."

Corneal Sensitization

The similarity is striking. Is it possible that in the emanations from the viscose-acid process there is some substance which sensitizes the corneal epithelium in some workers so that it becomes liable to photochemical changes produced by ordinary daylight or artificial light? The chief chemist at the works told me of an interesting occurrence at a factory in Breslau. During a very severe winter some seven or eight years ago, when the ground was covered with snow, there was a very severe outbreak of keratitis among those men who cycled to work. When buses were provided to bring them to work the incidence fell by about 50 per cent.—possibly due to the interception by the windows of the active ultra-violet rays reflected from the snow.

It will now be seen that discussion of the actual aetiology of the keratitis is of the greatest importance. The "droplets" theory was ruled out early in this investigation because the men themselves said that acid splashes were everyday occurrences and produced only temporary irritation, which passed off in a few moments, and because in the acid house there were no droplets. A very severe outbreak occurred in the acid house when some men were engaged in breaking up the concrete floor of an acid tank, which had been found to be leaking. The acid had soaked into the floor, and the fumes were released by the breaking up. The "contact-with-fingers" theory seemed very unlikely, and proved later to be quite erroneous.

A gaseous cause was decided as being the most probable, but H_2S was not thought to be a likely cause, because

Graph showing weekly numbers of cases of artificial silk keratitis from July 10th, 1928, to June 14th, 1930.

there were no reports of its producing this characteristic lesion in laboratories and in other factories where it was encountered in quantities; nor did any other gas, which the chemists thought might occur, seem to be the cause. Therefore late in 1929, at the height of the incidence of keratitis, thoughts were turned to the ventilation of the spinning room. The engineers insisted that this was perfect, since it produced fifteen changes of air each hour. It was pointed out to them that this did not mean that everywhere was there ventilation at that rate, and that the method was, indeed, very faulty, in that fresh air was introduced at ground level and at roof level, while extraction was only through the spinning boxes, at waist

level. It was suggested that if the induction pipes on ground level were made to act as extraction pipes there would then be a uniformly downward system of ventilation. This suggestion was adopted, and when put in operation produced a most dramatic drop in the number of cases of keratitis, as is strikingly shown in the graph. Moreover, from this time keratitis became a rarity. A few outbreaks have taken place from time to time, but, with one very important exception, they were traced to the closing down for one reason or another of one or more of the extraction fans. This seems to be conclusive proof that the cause is gaseous, or at any rate an emanation from some volatile substance.

Thio-formaldehyde

The exception just mentioned was in December, 1935. There was an outbreak of keratitis which could not be attributed to any fault in the extraction system in the spinning room. On inquiry it was found that, owing to a coal shortage, the acid house evaporating temperature was very much lowered and the fans over the tanks were stopped. This at once suggested that normally the evaporation in the acid house served also to drive off excess of the causal substance which was produced in the spinning room. During the coal shortage this did not

take place, and so the acid returned to the spinning room already charged with the substance to such an extent as to render even the new ventilation insufficient. On asking whether there might be some volatile substance which failed to be driven off at this time, the reply was given that such a substance was present—namely, thio-formaldehyde, which is formed from CS_2 in the presence of nascent hydrogen. It is thought that this reaction takes place readily in the spinning bath. Thio-formaldehyde is a little-known substance, having a curious odour suggestive of stale cabbage. Might not this, plus H_2S , give rise to the "oniony" breath referred to early in this paper?

Experiments are being undertaken to ascertain whether this substance has any relation to keratitis, and, if so, in what way it acts. Should interesting results be obtained they will be published in due course.

In conclusion, I wish to thank those who have encouraged and helped me in the writing of this paper, and particularly Sir Stewart Duke-Elder, who kindly read the notes and advised publication, and Dr. Berkson, who translated the foreign writings.

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INFECTIVE HEPATITIS WITH A FATAL RECRUDESCENCE

BY

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(With Special Plate)

Clinically the syndromes of catarrhal jaundice and of toxic hepatitis merge into one another. Pathologically the underlying changes in catarrhal jaundice have not been clearly defined. Recent reviewers of the subject suggest that the clinical syndrome of catarrhal jaundice represents at least three different pathological states. An obstructive jaundice due to blockage of the common bile duct by duodenal catarrh, a scattered liver necrosis, and, thirdly, an obstructive jaundice from cholangitis have all been suggested by pathological examination.

In view of the lack of unanimity as to the pathological state of the liver in the clinical syndrome of catarrhal jaundice the following case is worthy of record. In brief, it is the history of a disease in a young girl, which began apparently as a catarrhal jaundice, and passed on to a condition of portal obstruction. After a period of 300 days she had recovered clinically save for the evidence of portal obstruction. Two and one-third years later a similar illness commenced and continued with jaundice and intestinal upsets for forty-seven days, at which time a laparotomy was performed. Subsequent to operation she died from septicaemia. The post-mortem examination revealed old cirrhosis of liver and also recent hepatitis.

Clinical Record

A young girl, a jam packer by trade, was first seen at the age of 12 as a medical out-patient on March 22nd, 1932, when she gave the following account of her illness. Two weeks previously she had begun to get attacks of epigastric pain a short time after meals. These attacks lasted for seven days, when there developed a slight yellowness of her skin. The jaundice rapidly became worse. Associated with its onset there was one attack of vomiting and some nausea. She was admitted to the medical ward on the same day (fifteenth day of illness) and was described as a cheerful, well-nourished, very jaundiced girl. Her temperature was

99.4° F., and the pulse rate was 100. There were no abnormal physical signs in the heart, lungs, or nervous system. The liver edge was felt $1\frac{1}{2}$ inches below the right costal margin in the mid-clavicular line. The urine was acid in reaction, with a specific gravity of 1030; it contained no albumin or sugar, but both bile salts and pigments were present in considerable quantities. She was treated with a fat-free diet and with small doses of calomel and magnesium sulphate by mouth. The patient remained in hospital until April 12th, during which time the following notes were made.

March 29th (22nd day).—A bacteriological examination of the faeces showed the presence of non-haemolytic and *viridans* streptococci in moderate numbers. No non-lactose fermenting organisms were isolated.

April 2nd (26th day).—A fat analysis of the stools showed: neutral fats, 0.4 per cent.; fatty acids, 1.8 per cent.; and soaps, 8.3 per cent. The jaundice was now much less intense, and the stools were dark in colour.

November 16th (254th day).—She was again seen in the out-patient department, when she was slightly jaundiced. The liver edge was still easily palpable and the spleen easily felt for the first time. From November, 1932, until April, 1935, she was in good health, and stated that her jaundice had completely cleared by the end of 1932 (300th day).

April 21st, 1935.—The jaundice returned with malaise but no abdominal pain. There was a little diarrhoea, the stools were pale, and the urine was very dark in colour. She was readmitted to the wards on April 29th with a temperature of 99.4°, a pulse rate of 120, and a respiratory rate of 24. She was markedly jaundiced, but well nourished, and she had no itching of the skin. There were no abnormal physical signs in the heart, lungs, or nervous system, but there was slight furring of the tongue. The spleen now reached the umbilicus, and the liver edge was felt two inches below the costal margin in the mid-clavicular line. There was some fullness in the left loin. The urine was alkaline in reaction, the specific gravity being 1015; it contained no sugar, a trace of albumin, and some bile salts, but no bile pigments. She was again treated with a fat-free diet, glucose drinks, and calomel and magnesium sulphate by mouth. The progress of her second illness is shown by the following notes.

May 4th (14th day).—The blood examination showed: haemoglobin, 85 per cent.; erythrocyte diameter, 7.4μ ; leucocytes, 6,000—polymorph cells, 70 per cent.; lymphocytes, 22 per cent.; large mononuclears, 7 per cent.; basophil cells, 1 per cent.; and reticulocytes, less than 0.5 per cent.

May 8th (18th day).—Fat analysis of the faeces showed: neutral fats, 4.2 per cent.; fatty acids, 2.4 per cent.; soaps, 5 per cent. The red cell fragility was within normal