

auto-suggestion by the patient's surroundings and the circumstances under which he is first seen. The surgeon is summoned by his house surgeon, perhaps in the dead of night, to see a case of "acute peritonitis probably of appendix or gastric ulcer origin." He goes to his well-known ward, where he has seen series of cases of every phase of abdominal disease but seldom or never pleurisy or pneumonia. There he finds a patient suffering from acute abdominal pain, perhaps vomiting, and the house surgeon tells him that the patient has retained an enema. The pain the patient is in, and perhaps his cries when he is being examined, disturbing the whole ward as they do at night, prevent a complete examination. It may be that the surgeon is not quite satisfied that the signs undoubtedly indicate general peritonitis, but his clinical experience at once reminds him how vague the signs of general peritonitis may be; of the case of ruptured liver or spleen in which the signs were so vague for three or four days that operation was postponed until too late; or of that case of ruptured gut which for two days was supposed to be abdominal contusions. In such circumstances it is really not surprising that patients exhibiting the signs which I have mentioned are sometimes operated on. This is an accident always to be deplored, for very frequently ether is administered and this adds bronchitis to the pre-existing lung condition, whilst an extensive abdominal incision seriously hinders coughing. The only guard against such deplorable accidents is to recognise the possibility of their occurrence, and that is my excuse for this paper and the record of these cases.

PATHOLOGY.

The pathology is one of referred pain and divides itself into three heads.

1. *Irritation in continuity* of the lower six dorsal nerves will explain the hyperæsthesia and spasm of the abdominal wall. These intercostal nerves supply the diaphragm and lower part of the pleura and here their branches, if not their trunks, will be irritated by an acute inflammatory process. The terminations of the seventh to the eleventh dorsal nerves pass to the abdominal wall and supply the skin and muscles there. If the seventh, eighth, and ninth nerves are chiefly affected the acute epigastric tenderness and spasm will closely simulate a perforated gastric ulcer or if chiefly hypochondriac and on the right side hepatitis or gall-bladder trouble. When the tenth dorsal nerve is mainly affected the pain is referred to the umbilicus, as it is in so many abdominal complaints, whilst the area of the tenth and eleventh dorsal nerves on the right side is that in which the signs of an acute appendicitis develop. Moreover, the presence of tenderness and spasm on the left side in the tenth and eleventh dorsal areas does not exclude appendicitis, for in the early stage of this affection the pain may be referred to that side. It is interesting to note that three of my six cases terminated in suppuration. This seems to indicate that the inflammation in order to produce such abdominal signs must be severe enough to penetrate to the nerve trunks themselves and to produce actual neuritis.

2. *Referred visceral pain* has been most exhaustively studied by my colleague Dr. Henry Head⁵ and it will explain referred abdominal pain from lung disease without pleurisy. The seventh, eighth, and ninth dorsal rami communicantes are those which supply the lungs. When broncho-pneumonia, phthisis, or bronchitis but not lobar pneumonia supervene, even without pleurisy, impulses may pass up to the posterior root ganglia of these nerves and there produce such changes that normal impulses passing through these ganglia from the terminal branches of the nerves in the epigastrium and upper abdominal wall are exalted into painful impressions. The spasm follows in a reflex manner from the pain. Dr. Head tells me that he has seen such cases but none of those related here are explicable on this pathology.

3. *Of the pathology of the visceral signs* to which I have referred, gastric, intestinal, rectal, and vascular, we know very little. Perhaps they are the result of a reflex in the reverse direction to that to which I have just alluded, so that irritation of the six lower intercostal nerves by a pleurisy produces disturbance in the distribution of the three splanchnic nerves which are derived from the same level of the spinal cord. On the other hand, it may be due to irritation of these nerves in continuity as they lie beneath

the pleura or pass through the diaphragm to the great sympathetic plexures and ganglia of the abdomen.

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A CASE OF RECOVERY FROM MEMBRANOUS GASTRITIS.

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MEMBRANOUS gastritis is a condition characterised by an exudation from the epithelial lining of the stomach leading to the formation of a false membrane over the whole or part of that viscus. The symptoms of the disease are extremely variable. Thirst is often distressing, occasionally vomiting is uncontrollable, and pain in the stomach may be considerable, but cases have been recorded in which no symptoms have existed. The pathognomonic sign is the vomiting of a cast of the stomach or shreds of croupous membrane, but owing to the rarity of the presence of this sign, along with the fact that this form of gastritis usually occurs as secondary to a grave disease which averts attention from the gastric symptoms, the condition is not frequently diagnosed during life. In membranous gastritis the cast consists of fibrin and blood cells only, while in phlegmonous inflammation of the stomach the necrosed mucosa is shed. This difference is not strictly adhered to in the literature on the subject.

As to the rarity of the disease authorities agree but they differ in their experiences as to the most frequent causes. Rokitsky¹ states: "Croupy inflammation of the stomach never occurs as a primary and substantive affection except in the shape of delicate flocculent exudations in the aphthous processes of children, but always, and then rarely as a sequela or degeneration of exanthematic processes, in variola, in typhus, in the absorption of pus into the circulation, and particularly in puerperal inflammation of the uterine veins." Delafield² saw a man, aged 46 years, suffering from this disease and termed the condition idiopathic, because no cause was assignable. This author recognises that membranous gastritis is usually secondary to typhus fever, pyæmia, puerperal fever, cholera, dysentery, and irritating poisons. Von Ziemssen³ suggests that the condition may arise from the membrane in diphtheria spreading down the œsophagus into the stomach, while Ziegler⁴ writes that croupous and diphtheric inflammations of the stomach are rare. They occur most frequently in typhus fever, cholera, measles, scarlet fever, small-pox, pulmonary tuberculosis, and pyæmia. In diphtheria, on the other hand, they are extremely rare. Wilks,⁵ Fox,⁶ Kundrat,⁷ Goodhart,⁸ Rotch,⁹ and several others describe the condition but add little to the above summaries. Recklinghausen¹⁰ saw one case complicating gangrenous pneumonia. Goodhart and Still record that they have seen "membrane scattered over the surface of the stomach, mostly on the top of the rugæ and accompanied by intense congestion of the mucous membrane, in a case of severe broncho-pneumonia where there was no evidence of diphtheria. The condition was associated with membranous colitis." Osler¹¹ found an extensive area of croupous inflammation represented by a thick adherent greyish-white exudate covering an area 12 by 8 centimetres in one of his series of 100 cases which died from pneumonia. Quite recently Foulerton¹² described a case of membranous gastritis from which he was able to isolate pneumococci. Bizzozero¹³ saw two cases during the epidemic of diphtheria at Milan in 1875. The presence of the membrane in the stomach was not suspected during life and caused surprise at the necropsy. Six cases of the disease are described by Smirnow,¹⁴

¹ Handbuch der Pathologischen Anatomie, Band ii.

² Delafield and Prudden: Pathological Anatomy, p. 549.

³ Von Ziemssen's Handbuch, Band vii. (1), p. 52.

⁴ Lehrbuch der Pathologischen Anatomie, 1898, Band ii., p. 503.

⁵ Wilks and Moxon: Pathological Anatomy, 1875, p. 399.

⁶ Diseases of the Stomach, 1872.

⁷ Gerhardt's Handbuch der Kinderkrankheiten, Band iv.

⁸ Diseases of Children, 1899.

⁹ Pediatrics, p. 856.

¹⁰ Virchow's Archiv, vol. xxx., p. 366.

¹¹ Transactions of the Pathological Society of Philadelphia, 1885, vol. xii., p. 188.

¹² Pathological Society of London, 1902.

¹³ Medicinisches Jahrbuch, 1877, p. 219.

¹⁴ Virchow's Archiv, Band cxiii., p. 353.

⁵ Herpes Zoster, Brain, 1900; Referred Visceral Pain of Heart and Lungs, Brain, 1896.

who, however, considered that only two of them were due to diphtheria, because in that number only was necrosis of the gastric mucous membrane evident; prior to the discovery of the Klebs-Löffler bacillus necrosis was considered a *sine quâ non* of the diphtheritic process and the absence of necrosis prevented the condition being termed diphtheritic. Talfourd Jones¹⁵ saw a case of exudative gastritis in 1862; his patient was a rachitic child who developed a membrane in its stomach so similar to that which normally appears in the throat in diphtheria that the diagnosis of gastric diphtheria was made. Wollstein¹⁶ reports with great detail and many references two cases, in neither of which any evidence of diphtheria bacilli in the membrane was obtainable. One of the cases was a child suffering from advanced pulmonary tuberculosis, while the other had a false membrane on the inner side of the thyroid cartilage; micrococci were found in the membranes, but no Klebs-Löffler bacilli. The only case so far recorded in which a complete cast of the stomach was vomited is that by J. Thomson.¹⁷ The diagnosis of acute pulmonary tuberculosis was confirmed at the necropsy and no evidence of other disease was found. Two cases of undoubted diphtheritic gastritis came under the care of T. Leary¹⁸; in one there was membrane in the throat and in the other upon the conjunctiva. From the stomach of the former the bacillus of diphtheria was isolated. Willett¹⁹ recorded a case of diphtheritic membranous gastritis in a child, one and a half years old, who had died from pharyngeal diphtheria. No membrane was found in the œsophagus nor did the exudation extend beyond the pylorus. W. S. Fenwick²⁰ described a case of diphtheria of the stomach in a child three years of age; there was no pharyngeal membrane but "the respiratory tract was lined by diphtheritic membrane from the larynx to the finest ramifications of the bronchial tubes. The lower lobe of the left lung was solid from broncho-pneumonia." This membrane was found to swarm with micrococci but no diphtheria bacilli were found. This author points out that the vomit ejected by his patient did not contain hydrochloric acid and Rolleston suggests that the rarity of a gastric false membrane may be due to the solvent action of the gastric secretion.

That it is rare to find a false membrane in the stomach even when it exists on many other mucous surfaces is well illustrated by the papers of Villy and of Cary and Lyon. Villy²¹ examined the gastric mucosa of a large number of patients who died from diphtheria after having had well-marked gastric symptoms. The mucous membrane was often invaded by leucocytes and hyperæmic, but in no case did he find a definite false membrane. Cary and Lyon²² review the literature of pseudo-membranous inflammation of the mucous membrane caused by the pneumococcus and they find only one case in which the membrane was seen in the stomach. Without criticising the probable accuracy of the diagnosis in the above cases they may be classified. 19 cases are recorded—eight are ascribed to diphtheria, four to pneumonia, and two as sequelæ to pulmonary tuberculosis. I cannot help thinking that the cases of membranous gastritis which are stated to have followed variola, pyæmia, &c., were phlegmonous rather than pseudo-membranous gastritis.

The patient whose case I am about to describe was a healthy-looking girl, aged three years, of a rachitic type. Her father was consumptive but no further history of the disease was obtainable. She had been rickety since she was 14 months old and was subject to the complaints which accompany that disorder. Her present illness commenced on Feb. 8th, 1902, when the child was irritable and gave her relatives the impression that she was sickening for some disease; prior to this she had had a cold for a week. Towards midnight she awoke and complained of a sharp pain in the left ear, passing down the neck on the same side, and also of a slight stomach-ache. Judging from the spells of crying the pain in the head seemed to become more intense two or three times during the hour; between midnight and 6 A.M. the patient vomited four times without, as far as one

could tell, any preceding nausea. During the following day the patient played and seemed to be well until 5 P.M., when she became fretful, and four hours later the pain in the ear returned, accompanied by vomiting. The child lay upon her left side; she was sensible and was irritable on being disturbed. Both cheeks were flushed. There was slight tenderness over the left mastoid region but not sufficient to prevent the child from lying on that side. There was no nystagmus, strabismus, or paralysis of any muscles.

In regard to the respiratory system a detailed examination of the lungs did not reveal any signs of disease. The condition of the circulatory system was as follows. The apex beat of the heart was behind the fifth rib one-third of an inch internal to the nipple line. The heart was beating rapidly (128 per minute) but the sounds were clear and did not suggest any cardiac lesion. The pulse was regular, of normal volume, and about 100 mm. Hg pressure. The temperature was 97.8° F. The abdomen was rather full and of a rachitic type. There was no tenderness or resistance in any part. The spleen could not be felt below the costal margin nor did percussion lead to the conclusion that there was any enlargement of that organ. The lips were of good colour and the tongue was moist and thinly but evenly coated. The breath was slightly foul. The tonsils and fauces were hyperæmic but no trace of a membrane was visible in any part of the throat. The vomit consisted of glairy mucus, free from bile and blood. The bowels were slightly costive but with that exception the motions were normal. In regard to the nervous system the knee-jerks could not be obtained on either side. Although all the superficial and deep reflexes were examined no other alteration from the normal was found. The urine contained a trace of albumin. The blood was examined on Feb. 10th with the following results: red cells, 4,480,000; and white cells, 8700 per cubic millimetre. A differential count showed: polymorphonuclear cells, 43.1 per cent.; lymphocytes, 48.5 per cent.; mononuclear cells (large), 7.9 per cent.; and eosinophile cells, 0.5 per cent.

The child complained of great thirst and since vomiting followed even small doses of water by the mouth rectal feeding was adopted. On Feb. 10th the temperature and pulse remained unaltered. Vomiting occurred repeatedly during the day but the thirst seemed to have been relieved by the rectal injections. In the evening the child again complained bitterly of thirst and was given a few teaspoonfuls of water, shortly after which she cried out with gastric pain and then vomited a piece of membrane, the nature of which will be described later. Occasional vomiting continued during the night, accompanied by slight abdominal pain. About 3.30 on the following afternoon, after considerable pain and retching, another piece of membrane was vomited and this along with the former piece seemed to be a complete cast of the stomach. The vomiting from this time onward became less frequent, while the condition in other respects remained unaltered. On the 12th, with a pulse of 128 and a temperature varying between 97° and 98° F., the patient gave the impression of improving, the tongue being slightly cleaner and the vomiting less frequent. Knee-jerks could not be obtained. On Feb. 14th the pulse was 132 and the temperature was 97.4°. The patient slept well during the night but was drowsy in the morning. A trace of albumin was present in the urine. During the day the child expressed a desire for food. On the 16th the pulse was 116. The child had not vomited for 24 hours. The heart sounds were weaker and the rhythm rather embryonic. The desire for food was more marked. On the 17th the pulse was 112 but very weak. One ounce of peptonised milk mixed with three times that quantity of water was given by the mouth and was retained. The patient was improving on the 18th. Milk and beef-juice were digested. From this time onwards the patient steadily improved and *pari passu* with larger quantities of food being taken by the mouth the rectal injections were diminished in volume and frequency. The right knee-jerk returned on March 6th and the left on the day following. The child has been perfectly well for several months.

Diagnosis.—That the case was one of membranous gastritis was placed beyond doubt by the shape and nature of the cast vomited. The cause of the exudation had to be considered. The temperature was subnormal, the pulse was rapid, there was slight albuminuria accompanied by the loss of knee-jerks, and a normal number of white cells in the blood. These are all compatible with diphtheria. A swab of the back of the throat was taken on Feb. 10th but no

¹⁵ Brit. Med. Jour., 1889, vol. i., p. 880.

¹⁶ Archives of Pediatrics, vol. ix., p. 489.

¹⁷ Ibid., vol. xii., p. 286.

¹⁸ Journal of the Boston Society of Medical Science, 1897, No. 16, p. 8.

¹⁹ Transactions of the Pathological Society of London, 1892, vol. xliii., p. 62.

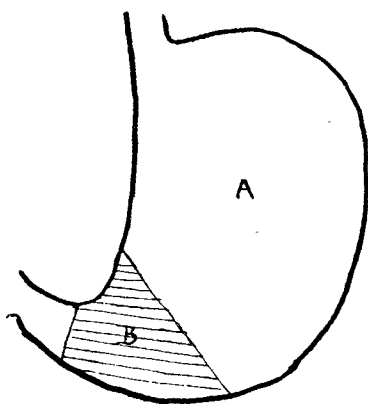
²⁰ Ibid., 1894, vol. xlv., p. 60.

²¹ Medical Chronicle, 1899, p. 391.

²² Transactions of the Association of American Physicians, 1901, p. 379.

suspicious bacilli were found either in a stained film or by culture on blood serum. I regret that no cultures or inoculations were made from the vomited membrane. Microscopically I failed to find in it any bacilli which resembled Klebs-Löffler bacilli. In toxæmia of diphtheritic origin it is not rare for the heart's action to be altered in rate and rhythm without the temperature of the body being greatly elevated. The temporary albuminuria and loss of knee-jerks make one doubt whether croupous gastritis would have been the correct diagnosis. The subnormal temperature along with an absence of leucocytosis both make it improbable that the membrane was of pneumonic origin. The examination of the blood did not add to the data upon which diagnosis was based. Although a marked leucocytosis as a rule accompanies diphtheria it is not invariably the case. The differential count of the white cells is not far from normal; the child was three and a half years old, at which age normally the polymorphonuclear and mononuclear elements of the blood are present in equal numbers. There is a tendency in rachitis, as in all conditions of malnutrition, for the lymphocytes to be relatively increased, and this may have been responsible for the high percentage of mononuclear cells. It is scarcely necessary to add that full investigation as to the possibility of the gastritis being due to an irritant introduced through the mouth was made, with a negative result. The piece of membrane vomited on Feb. 10th was of a shape and size that suggested a cast of the greater part of the stomach, including the cardiac opening; the pyloric end was missing, but the tissue ejected on the following day completed the cast (Fig. 1). The thickness of the membrane varied in different

FIG. 1.



A was vomited on Feb. 10th. B was vomited on Feb. 11th.

parts, but in no place did this exceed one millimetre. It was of a pinkish-grey colour coursed by red streaks; when examined in water it assumed a rugous aspect (Fig. 2). Microscopically it consisted of a fine network of fibrin, the meshes of which contained large numbers of white and red blood-corpuscles, the former being in excess. Epithelial elements were totally absent (Fig. 3). Sections stained with Löffler's blue and by Gram's method show the presence of some number of micrococci, mostly arranged in pairs, but only very few are of the shape that suggests the diplococci of pneumonia. No bacilli resembling diphtheria bacilli have been seen in the membrane. The micro-organisms are most numerous on the side which was in contact with the gastric mucosa. The outer side of the cast is ragged; the fibrin has been dissolved by the gastric secretion which probably led to the separation of the exudation from the epithelium. It is of interest to note that the false membrane covering the pyloric end was the last to be shed, as one would have expected from the fact that the glands at that end of the stomach do not secrete so much hydrochloric acid.

Treatment.—Until the cast of the stomach was vomited the diagnosis of exudative gastritis was not considered, but the continued vomiting necessitated the adoption of rectal feeding. Although the possibility of the diphtheritic origin of the disease was raised immediately upon the ejection of the membrane, nevertheless it was decided not to use diphtheria antitoxin. The advantage of the use of antitoxin on the fourth day of the disease is questionable, and in a case which has already shed its membrane without that

membrane possessing evidence of diphtheria it did not seem justifiable. This decision was fortunate, for if an injection of antitoxin had been given it would not have been easy to fully realise that the recovery was purely *post hoc* and not *propter hoc*. An attempt was made to maintain strength by

FIG. 2.



The membrane floated out in water showing the rugous appearance.

FIG. 3.



Section of cast prepared with a nuclear stain showing altered blood corpuscles in a fibrin network. The upper surface was the inner side of the cast.

giving a rational rectal diet. The form of nutrient selected consisted of 60 cubic centimetres (two ounces) of ox serum, 60 cubic centimetres of milk, and eight grammes (two drachms) of raw starch every four hours. Three grammes of chloretone were dissolved in every litre of serum. This had

a triple effect: it preserved the serum, it acted as a sedative to the mucous membrane of the rectum, and it also caused the child to sleep for an hour or two after the administration of the injection, thus facilitating its retention and hence its assimilation. 360 cubic centimetres of serum, 360 cubic centimetres of milk, and 48 grammes of carbohydrate were introduced during the 24 hours, which diet possesses an energy value of over 500 calories, which is not much short of three-quarters of what is necessary for a child three and a half years of age. When the pulse became feeble and the heart sounds assumed an embryonic type a few minims of tincture of *nux vomica* and of *digitalis* were added to the nutrient enemata. The efficiency of a rational rectal diet containing materials which we know can be absorbed by the large intestine was proved by the very slight loss in weight of the patient in spite of no food being given by the mouth for 10 days.

To Dr. H. D. Rolleston I am indebted for kindly criticism in writing out the case and for assistance in the recent literature of the subject. Mr. Robert MacQueen was good enough to take daily notes of the case and to draw two of the diagrams, and without his able assistance it would have been impossible for me to have recorded the case.

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PURPURA FULMINANS FOLLOWING SCARLET FEVER.

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THE diagnosis of the exceedingly rare condition from which the patient whose case is narrated below suffered is not by any means clear. It seems to me to correspond more closely to the condition which is described by Henoch under the name of purpura fulminans than to any other with which I am acquainted. It is noticeable, too, that one of the two cases related by him occurred after scarlet fever, as did also one of two cases related by Ström and Arctander. Now, hæmorrhagic scarlet fever, as described in some works, is of great rarity in this country at the present time; it is doubtful, indeed, if extensive cutaneous and subcutaneous hæmorrhages ever occur in the early stages of the disease; whether the cases related in those works, in fact, were not actually cases of hæmorrhagic diphtheria. Hæmorrhages into the skin and subcutaneous tissue are met with not very infrequently in association with obstinate suppurative lesions after scarlet fever, such as chronic suppurative lymphadenitis, suppurations in joints, and suppurations in and about the temporal bone. No such lesion obtained in this case. The child had a very severe attack of scarlet fever which did not present any unusual features and convalescence appeared to be established. He still had some ulceration of the soft palate, it is true, but the other symptoms had either passed off or had much abated, the temperature was settling down, and he was bright and comfortable. At this point cutaneous hæmorrhages in enormous abundance took place, accompanied by hæmatemesis, bloody stools, and oozing from the gums. In 36 hours from the appearance of the first hæmorrhages he was dead. The chief feature of the post-mortem examination was the state of the kidneys. These were transformed almost entirely into fat, a little blood-clot being found in the calices. This blood-clot, too, must have been very recent, as none was found lower down the urinary tract, even the urine in the bladder being free from blood. The kidney was a very different one from that associated with any variety of scarlet fever and no sign or symptom of nephritis occurred during life, except that albumin in small quantity was present in the urine for one day soon after admission. The change would appear to have been far more of the nature of an acute degeneration than of an inflammation, the homogeneity of the condition being very different from the sectional infiltrations seen in either the septic or glomerular type of scarlatinal kidney. No renal phenomena were noted by Henoch in the necropsies of his cases and Ström and Arctander do not state whether post-mortem examinations were made in their cases.

The sudden occurrence of these widespread hæmorrhages

suggests a very acute toxæmia or blood infection due to the elaboration of some toxic substance or the supervention of some fresh organism, but what the nature of such body may have been and what determined its appearance at this period of the disease are speculations beyond the range of legitimate theorisation. A conceivable criticism is that these abundant hæmorrhages were but an advanced stage of the punctiform hæmorrhagic rash that occasionally follows the administration of animal sera, for diphtheria antitoxic serum had been given to this child in error. That this should be so is in the highest degree improbable, for not only has such an occurrence never been noted after the many thousand administrations that have come within one's purview, but this particular serum was used for injection into many children in the hospital without any analogous phenomena ensuing.

The clinical and pathological features of the case may be briefly summarised as follows:

A boy, aged three and a half years, was admitted into the Grove Fever Hospital on May 16th, 1902, certified to be suffering from diphtheria and on this supposition was given an injection of serum containing 24,000 units of diphtheria antitoxin. On examination, however, his trunk and limbs were found to be covered with a coarse, blotchy, punctiform erythematous rash; the throat was much reddened and very oedematous; the tonsils were coated with slimy exudation; the nasal mucous membrane was turgid and covered with muco-purulent discharge; the submaxillary lymphatic glands were considerably enlarged; and the tongue was dry and clothed with fur. Culture of the tonsillar exudation presented an abundant growth of streptococci. He was very ill till the 21st, when the temperature, which had been continuous at 103° F., fell to 99°, and there was a general abatement of the symptoms. On that evening he became more feverish (the temperature being 102.4°), and on the next day an urticarial rash appeared on the trunk, due doubtless to the previous injection of serum. Double otitis media, diarrhoea with green stools, and free general desquamation ensued. On the 25th a further eruption, a macular, purplish-red one, was noticed about the knees, the ankles, and the elbows. By the 29th he was much better; the glandular swellings had almost disappeared and there was no rash or diarrhoea. Bilateral otorrhoea, however, remained, and there was a little rhinitis, whilst an ulcer of the size of a threepenny-piece was found at the junction of the soft palate and the right anterior pillar of the fauces, though the tonsils were free from exudation and the tissues of the throat were but little swollen. From this time he improved rapidly till June 4th, notwithstanding that the temperature still fluctuated between 101° and 99°. On that day a vast number of pin-point hæmorrhages were seen in the skin of both sides of the trunk, whilst the next morning the trunk and limbs were literally covered with these points, the upper extremities being so densely set with them as to appear almost uniformly purple. There were but few on the face, except on the inner sides of the eyelids where they were about the size of grape-stones. Blood oozed from the gums, but there were no subcutaneous or subconjunctival hæmorrhages. His general condition was markedly worse. He vomited half a pint of red blood twice during the day and towards the evening he passed about that quantity of brightish-red blood per rectum. He died soon afterwards.

Necropsy.—At the post-mortem examination, which was performed 30 hours after death, the cutaneous hæmorrhages were abundantly apparent and blood-clot encrusted the lips, the nose, and the anus. Under the visceral pericardium and pleuræ a few punctiform hæmorrhages were seen, similar points being present under the endocardium of the left ventricle. The pleuræ each contained a few drachms of clear, pink fluid and the trachea and bronchi similarly-tinted frothy mucus. The stomach showed many medium-sized, sub-mucous hæmorrhages, and both it and the intestines, which latter were deeply cyanosed, contained chocolate-coloured fluid; crimson, viscous blood filled the rectum. Beyond thickening and ulceration of the tissues of the throat nothing else remarkable was seen till the urinary system was examined. Both kidneys were a little enlarged, but their capsules stripped readily. On section the parenchyma was found to be transformed almost entirely into fat, which stained readily with osmic acid but not at all with iodine solution. There was no differentiation into cortex and medulla and a few faint pink streaks alone suggested the direction of the tubules. Several of the calices contained a