and appreciate that which we can feel and handle, namely, the peripheral neurosis.

The third question is, What is the connection of this neurosis with the arthritis? Let me recall some of the facts. Pains about joints may precede acute gout. These pains can be demonstrated to be in nerves and not in joint. Pains, redness, and swelling round a joint which is intact—that is, not painful on being moved; best seen in thumb. Pain, redness, and swelling, with tender nerves, remaining after joint has ceased to be tender; best seen in great toe. Without disputing that the joint may have been tender, it is quite impossible to prove it. Dr. Ewart says: "*The extent of the arthritis associated with the periarticular swelling is matter of doubt.*" Sir. W. Wade's observations go far towards proving that there is much less arthritis than had hitherto been supposed—at any rate, that the *pain and tenderness* have not their seat inside, but *outside* the joint, and apparently in the nerves themselves." (Page 319. The italics are Dr. Ewart's.)

The patient's opinion is valueless in this respect; for a patient will refer the pain to the joint, when it can be demonstrated by proper examination that it is not there. All this goes to show that the essential element of the attack is to be looked for in the local nerves. But it is answered, and truly, Many years after a single attack, a uratic deposit may be found in a joint. If the attack is due to the deposit, how is it that the joint recovers before the deposit has been removed? A joint many times attacked may be found after death free from any deposit. It may have been absorbed. True; but it equally may have never been there.

The assumption that the attack is due to uratic deposit leaves unexplained why it is deposited. The neurotic theory, if it does nothing more, offers an explanation. The current view is curiously paradoxical. In an acute attack the deposit is so small that the joint quickly recovers. Yet it is acutely painful. In many chronic and subacute attacks the deposit is very copious, whereas the pain and tenderness are very small. This, then, is the paradox. In a painful disease which depends upon the deposit of urates, the greater the deposit the less the pain. It would seem that so long as the nerve is in a condition to become painful, it does not promote—perhaps retards—the deposit of urates. When by some further, at present unknown, changes it has lost the faculty of becoming painful, it does not retard, perhaps accelerates, such deposition. The inference to be drawn from all these facts is, I submit, as follows. The neurosis is the primary and essential element. If a sensory nerve is attacked we get pain and tenderness, if a vasomotor one we get redness and swelling; if the nutritive nerve of a joint, we get changes in the joint, one result of which may be a deposit of urates. I submit from this that this theory, more completely than any other, brings into line the various manifestations of gout. But it may be asked how it is that one toxin (uric acid) affects now an external, now an internal, sensory nerve; now a motor, now a vasomotor, now a trophic nerve, now an excretory one. This raises an important question, on which Dr. Ewart says (p. 143), "We still hesitate, then, to admit, without further investigation, that gout is primarily dependent upon uric acid as a cause, or that uric acid necessarily exerts any exclusive influence on the production of the phenomena of goutiness." Dr. Ewart's hesitation is shared by many. It is enhanced in my mind by the fact that different toxins have an elective affinity for different nerve tracts. Conversely we perhaps may infer that affections of different nerve tracts imply different toxins. That such may be the case in gout receives strong support from the ingenious and laborious researches of Dr. Haig.⁵ This uncompromising advocate of the uric acid theory claims to have proved that other bodies chemically allied to, but not identical with, uric acid resemble it in their action upon the human body. This, then, is a matter which, like the state of the local nerves, deserves a much more critical investigation than either have as yet received.

REFERENCES.

- ¹ Gout and Goutiness and their Treatment. ² Gout as a Peripheral Neurosis.
- ³ On some Forms of Paralysis from Peripheral Neuritis of Gouty, Alcoholic, Diphtheritic, and other Origin. ⁴ Sir Dyce Duckworth's Treatise on Gout.
- ⁵ Uric Acid as a Factor in the Causation of Disease.

OF 19 persons on whom the degree of Doctor of Medicine was conferred by the University of Geneva during 1896, no fewer than 7 were women.

ANTIPNEUMOCOCCIC SERUM.

By J. W. WASHBOURN, M.D., F.R.C.P.,

Physician to the London Fever Hospital; Assistant Physician to Guy's Hospital.

In the year 1891 the researches of G. and F. Klemperer, Bonome, Foà and Carbone, Emmerich and Fowitzky, proved that the blood serum of rabbits immunised to the pneumococcus possessed the property of protecting other rabbits against infection with this micro-organism.

G. and F. Klemperer, Foà and Carbone, Foà and Scabia, and Janson have since treated cases of pneumonia in the human subject with the serum of immunised rabbits; while Andeoud has used for the same purpose the blood serum of patients convalescent from pneumonia. In these cases the quantity of serum used was of necessity small, and no exact determination of its strength was made.

To obtain a sufficient quantity of serum it is necessary to immunise large animals, such as the horse; and, to estimate its strength, it is necessary to use more reliable methods of cultivating the pneumococcus than have hitherto been employed.

In this paper I propose to give an account of the immunisation of a pony, to describe the method I have adopted for estimating the protective power of the serum, and to report two cases of pneumonia treated with the serum.

IMMUNISATION OF PONIES.

In January, 1895, a pony was placed at my disposal by Professor Ruffer, then Director of the British Institute of Preventive Medicine. I take this opportunity of thanking the Council of the Institute for generously providing me with most ample opportunities for carrying out this work. The expenses of the purely scientific part of the investigation were defrayed by a grant from the Royal Society, but the greater expenses incurred in purchasing and keeping the ponies were borne by the Institute. I would also heartily thank Professor Ruffer for most valuable advice, and Dr. Bulloch for the great assistance he has rendered in carrying out the necessary manipulations. To both I am deeply indebted for freely giving me the results of their large experience in the immunisation of horses.

The pony was first injected with broth cultivations of the pneumococcus which had been heated to 60° C. for one hour, and subsequently with living cultivations. In October, after being under treatment for nine months, it was suddenly seized with intestinal obstruction, and died after a few days' illness. This was especially unfortunate, as a fairly high degree of immunity had been attained, and the blood already possessed some protective power.

The immunisation of a second pony was commenced in November, 1895. The pony was first injected with broth cultivations heated to 60° C. for one hour, then with living agar cultivations, and lastly with living broth cultivations. The injections were made into the subcutaneous tissue of the shoulder.

The temperature chart has unfortunately been mislaid and the record of the temperature has thus been lost. In a few instances a note of the temperature was also made in the report.

November 21st, 1895; 70 c.cm. broth cultivation heated to 60° C. for one hour. The temperature rose next morning to 103.5°, but soon subsided.

November 30th; 170 c.cm. broth cultivation heated to 60° C. for one hour. No pyrexia and no local reaction.

December 7th; a small loopful of a living agar cultivation. No local reaction.

December 13th; the whole of a living agar cultivation. No local reaction.

December 19th; two living agar cultivations. No local reaction.

January 4th, 1896; 6 living agar cultivations. A swelling appeared at the spot of injection and lasted five days.

January 13th; 50 c.cm. living broth cultivation. A swelling of the size of an orange appeared at the spot of injection and lasted a few days.

January 21st; 82 c.cm. living broth cultivation. A large

swelling appeared at the spot of inoculation, and lasted nine days.

February 7th; 150 c.cm. living broth cultivation. A diffuse swelling appeared, but soon subsided. The temperature reached 104.2° F.

February 21st; 100 c.cm. living broth cultivation injected into both sides of the neck. A swelling appeared on both sides, more marked on right side. This subsided in a few days. The temperature reached 103° F.

March 3rd; 112 c.cm. living broth cultivation injected into both sides of neck. The animal appeared ill for twenty-four hours, and there was some swelling at the spot of injection.

March 14th; 150 c.cm. broth cultivation injected into both sides of the neck. Some swelling occurred on both sides, but soon disappeared. Animal ill for twenty-four hours.

March 27th; 200 c.cm. broth cultivation injected into both sides of the neck. Marked swelling occurred on both sides, and lasted two days.

On April 3rd the near fore leg became hot, swollen, and tender, and in a few days an abscess formed and burst. The pus contained streptococci. Probably the infection was caused by a slight grazing of the skin against the wall of the stable. The animal became very ill, and we thought it would die. Owing to the unremitting attention of Dr. Bulloch, the abscess gradually healed, and in three weeks the animal was convalescent.

On August 25th, 1896, five months after the last injection, the animal was bled, and the serum was found to possess marked protective properties.

The bleeding was deferred so long partly on account of the inflammation in the leg and partly from an experience gained during the immunisation of the first pony. After the injection of 100 c.cm. of a living broth cultivation, an abscess formed at the spot of inoculation, which only healed after three weeks. A week later, that is to say a month after the inoculation, blood was removed, and some of the serum was injected into the peritoneal cavity of a rabbit. The animal died of pneumococcal infection, showing that the serum contained living pneumococci. A fortnight later more blood was removed, and the serum injected into the peritoneal cavity of a rabbit. No ill effects ensued, so that by this time the cocci had disappeared from the blood.

The pneumococcus which was used in immunising the pony was from a different source to that used in testing the serum. The cultivations were always made directly from the blood of an infected rabbit, and were used after twelve to eighteen hours' incubation at 37.5° C.

The effects observed after inoculating the pony were similar to those described after inoculation with cultivations of the streptococcus, that is, swelling at the site of inoculation, pyrexia, *malaise*, and loss of appetite. With small doses neither local nor general symptoms occurred.

ESTIMATION OF STRENGTH OF SERUM.

In testing the strength of the serum it was necessary to obtain a pneumococcus of a known virulence, and to employ a method of cultivation which would preserve the virulence unaltered.

After many trials, I have used for the latter purpose the surface of agar streaked with sterile rabbit's blood. This method had been used by me previously, and had been independently described by E. Fraenkel and Reiche.¹ Careful experiments have since been made by Dr. J. W. Eyre and myself upon this method. As our results will shortly be published in the *Journal of Pathology and Bacteriology*, it is unnecessary for me to enter here into details. It will suffice to say that great care is requisite in making the agar, which is never heated above 100° C.; and that the reaction is of great importance. The agar is neutralised with caustic soda, rosolic acid being used as an indicator. As soon as the neutral point is reached 4 c.cm. of normal caustic soda solution is added to each litre of agar. Upon such a medium the pneumococcus will preserve its initial virulence for as long as nine weeks, provided the tubes are carefully capped and kept in the incubator at a temperature of 37.5° C.

In all the experiments a loopful of the cultivation was made into an emulsion with a known quantity of sterile broth, and by dilution any fraction of the contents of the loop could be obtained. The loop was made in such a way that its contents did not vary, and the same loop was used throughout. It contained about 0.5 mg. of the cultivation. Cultivations twenty-four hours old were always used, and thus the loop contained either only living cocci, or at any

rate the same proportion of living and dead cocci in each experiment.

The pneumococcus was obtained from the blood of a mouse which had died after inoculation with the sputum from a case of pneumonia. After 9 passages through rabbits, with intermediate cultivation on blood agar, it attained a fixed virulence, which was not increased by further passages. The virulence was carefully determined by a number of experiments. When introduced into the peritoneal cavity of rabbits 0.000001 of the loop invariably caused death from general septicæmia, and the blood was found immediately after death to be teeming with cocci. Sometimes 0.000001 of the loop was fatal, but as a rule this dose was innocuous. The dose which was fatal when introduced into the peritoneal cavity of rabbits was also fatal to mice when injected into the subcutaneous tissue. Rabbits die in about thirty-six hours, and mice in three to seven days.

In testing the serum measured quantities were mixed with 10 times the minimum fatal dose of the cultivation. The mixture, immediately after being made, was injected into the peritoneal cavity of a rabbit. The smallest quantity of serum which protects the animal under these circumstances I propose to call a unit. For instance, if 0.03 c.cm. of the serum mixed with 10 times the fatal dose of the cultivation protects, then, each cubic centimetre of the serum contains approximately 33 units.

As 0.000001 of a loop generally represented the minimum fatal dose—0.000001 loop was the quantity mixed with the serum. But as 0.0000001 loop was sometimes fatal two control rabbits were used in each series of experiments. The one control was inoculated with 0.000001 loop and the other with 0.0000001 loop. In all the experiments the animal inoculated with 0.000001 loop died, and in most the animal inoculated with 0.0000001 loop survived. If the latter animal died the experiment was rejected, because the animals injected with the serum had been inoculated with 100 times instead of with 10 times the minimum fatal dose.

The following series of experiments made with the serum removed on August 25th, 1896, show that 0.03 c.cm. protects against 10 times the minimum fatal dose of the cultivation, while smaller quantities have no effect. Consequently each cubic centimetre of the serum contains 33 units.

I should state that Dr. Eyre and myself have tested in the same way the sera from horses not immunised to the pneumococcus, and from other animals, but in no case have we observed any protective properties. It therefore follows that the serum of the immunised pony is specific in its character. Our results will be shortly published in the paper I have already referred to.

TABLE I.

Rabbit A	2,900 g.	0.0000001 loop	Not affected.
" B	2,980 g.	0.000001 loop	Died in 36 hours.
" C	2,950 g.	0.000001 loop + 1 c.cm. serum	Not affected.
" D	1,500 g.	0.000001 loop + 0.5 c.cm. serum	"

TABLE II.

Rabbit E	1,600 g.	0.0000001 loop	Not affected.
" F	1,400 g.	0.000001 loop	Died in 60 hours.
" G	1,700 g.	0.000001 loop + 0.5 c.cm. serum	Not affected.
" H	1,500 g.	0.000001 loop + 0.25 c.cm. serum	"
" I	1,700 g.	0.000001 loop + 0.12 c.cm. serum	"

TABLE III.

Rabbit J	2,100 g.	0.0000001 loop	Not affected.
" K	2,300 g.	0.000001 loop	Died in 36 hours.
" L	2,100 g.	0.000001 loop + 0.25 c.cm. serum	Not affected.
" M	2,050 g.	0.000001 loop + 0.12 c.cm. serum	"
" N	2,200 g.	0.000001 loop + 0.06 c.cm. serum	"

TABLE IV.

Rabbit O	3,000 g.	0.0000001 loop	Died in 36 hours.
" P	3,600 g.	0.000001 loop	Died in 60 hours.
" Q	3,000 g.	0.000001 loop + 0.03 c.cm. serum	Not affected.
" R	1,600 g.	0.000001 loop + 0.015 c.cm. serum	Died in 36 hours.

NOTE.—In this case 0.0000001 loop was the minimum fatal dose.

In the following tables mice were used instead of rabbits, and the injections were made into the subcutaneous tissue. It will be seen that the minimum fatal dose is the same as for rabbits, and that the same quantity of serum protects:

TABLE V.

Mouse A	17 g.	0.0000001 loop	Not affected.
" B	18 g.	0.000001 loop	Died in 6 days.
" C	17 g.	0.000001 loop + 0.03 c.cm. serum	Not affected.

TABLE VI.

Mouse D	13 g.	0.0000001 loop	Not affected.
" E	13 g.	0.000001 loop	Died in 7 days.
" F	17 g.	0.000001 loop + 0.03 c.cm. serum	Not affected.
" G	15 g.	0.000001 loop + 0.015 c.cm. serum	Died in 8 days.

TABLE VII.

Mouse H	17 g.	0.0000001 loop	Not affected.
" I	17 g.	0.000001 loop	Died in 6 days
" J	20 g.	0.000001 loop + 0.03 c.cm. serum	Not affected.

Before using the serum for the treatment of pneumonia in the human subject, it was necessary to ascertain, first, whether it would protect animals when injected after infection, and secondly, whether large quantities produced any ill effects.

THERAPEUTIC EFFECT OF SERUM.

In the following experiments the serum was injected into the peritoneal cavity of rabbits at different periods after inoculation. The tables show that a single dose of 66 units serum protects when injected five or six hours after inoculation, but that the same quantity injected as late as eight or twelve hours only retards the fatal event. It will be observed that the controls died in twenty-four to twenty-six hours, and therefore recovery ensued when the treatment was begun in the first quarter of the disease.

TABLE VIII.

Rabbit S	1,500 g.	0.0000001 loop	Died in 24 hours.
" T	1,500 g.	0.000001 loop	Died in 24 hours.
" U	1,600 g.	0.000001 loop. Six hours later, 2 c.cm. serum (66 units)	Alive 6 weeks later
" V	1,600 g.	0.000001 loop. Eight hours later, 2 c.cm. serum (66 units)	Died in 4 days.

TABLE IX.

Rabbit W	2,750 g.	0.000001 loop	Died in 24 hours.
" X	2,400 g.	0.000001 loop. Twelve hours later, 2 c.cm. serum (66 units)	Died in 48 hours.

TABLE X.

Rabbit Y	2,350 g.	0.000001 loop	Died in 26 hours.
" Z"	2,500 g.	0.000001 loop. Five hours later, 2 c.cm. serum (66 units)	Alive two months later.

INNOCUOUSNESS OF SERUM.

The following experiments show that large doses of the serum produce no ill effects on rabbits:

Rabbit 1: 5 c.cm. serum injected into the peritoneal cavity. No rise of temperature ensued, and the animal neither lost weight nor appetite.

Rabbit 2: 10 c.cm. serum injected into the peritoneal cavity. No rise of temperature ensued, and the animal neither lost weight nor appetite.

NATURE OF SERUM.

From the foregoing experiments it will be seen that the serum possesses the power of protecting animals against infection when injected either at the same time or subsequent to inoculation. It is impossible to say whether it possesses antitoxic powers, because hitherto no satisfactory toxin has been obtained from cultivations of the pneumococcus.

CASES OF PNEUMONIA TREATED WITH SERUM.

The following are brief notes of two cases of pneumonia treated with the serum. One case was under the care of Dr. Pye-Smith, to whom I am indebted for permission to publish the notes. Other cases have been treated, and will shortly be published by the physicians in charge.

From such a limited material it would be rash to draw conclusions, although in Case II the treatment appeared to have a distinctly beneficial effect. I am induced to publish the results of my investigations at this early period, as I hope that the British Institute of Preventive Medicine will shortly have a sufficient quantity of the serum to supply to those who wish to use it in the treatment of pneumonia.

CASE I. *Pneumonia of Left Lower Lobe: Treatment with Serum: Recovery.*—R. F., 29, stevedore, admitted into Guy's Hospital on November 24th, 1896, under the care of Dr. Pye-Smith.

Previous History.—Six months ago was in bed for a week with cough. Has otherwise enjoyed good health. Admits that he drinks somewhat heavily.

History of Present Attack.—Was suddenly taken ill on November 18th, six days before admission, with pain in the chest, and shivering.

November 24th. **Condition on Admission.**—The skin is hot and dry, the tongue furred and tremulous, the breathing is rapid, and there is cough with rusty expectoration. Pulse 108, temperature 103.6°, respirations 33. The urine contains a trace of albumen, and the chlorides are slightly diminished. The chest is resonant in front, and cardiac dullness absent; rhonchi audible all over the front. At the back the right side is resonant, and loud rhonchi are present. On the left side a pleuritic rub is audible in the posterior axillary region, and there is impaired resonance from the middle of the scapula to the base with bronchophony, tubular breathing, and fine crepitations.

November 25th. During the night patient was slightly delirious. From 6 o'clock yesterday evening the temperature steadily fell, and reached 100.8° at 6 o'clock this morning. At 10 A.M. to-day it had again risen to 103.2°. The physical signs have not altered, except that impaired resonance at the left base is now replaced by complete dullness. At 2 P.M. the temperature had risen to 104.2°. At 2.20 P.M., 660 units antipneumococcic serum (20 c.cm.) injected. At 4 P.M. the temperature had fallen to 101.6°, but at 6 P.M. had again risen to 103.4°.

November 26th. During the night the temperature fell, and was 100.2° at midnight. At 10 A.M. to-day it was 103.2°. Pulse 116, respirations 40. Physical signs and general condition unaltered. At 10 P.M., 660 units serum injected. Temperature 101.2°.

November 27th. Is much better to-day. At 10 A.M., temperature 99.2°, pulse 88, respirations 28. There is no pain, the cough has almost gone, and the sputum is no longer rusty. The pleuritic rub has disappeared. There is still dullness at the left base, and the tubular breathing is replaced by rales.

From this time the patient rapidly convalesced, the physical signs gradually cleared, and by December 7th had completely disappeared.

On December 10th an urticarial rash appeared. On December 17th the patient was discharged from the hospital.

Remarks.—The injection of the serum was followed on both

occasions by a fall of temperature, and on the day after the second injection the crisis occurred. Inasmuch as this was on the ninth day of the disease, it is difficult to say whether the result was due to the treatment or not. The attack was a fairly severe one, but the pulse was good before the serum treatment was adopted.

Chart 1.

CASE II. Double Pneumonia: Treatment with Serum: Recovery.—A. H., 25, law writer; admitted December 10th, 1896, into Guy's Hospital under care of Dr. Washbourn.

Previous History.—Was in the army, and was stationed in India for five years. Returned to England last month. Has been a heavy drinker.

History of Present Illness.—Was seized with a rigor at 4 P.M. on day before admission. Was very sick during the night.

December 10th. *Condition on Admission.*—Temperature 101.4°, respirations 44, pulse 140; tongue furred; skin hot and dry; rapid shallow breathing; pain in right side of chest; harsh vesicular murmur at right base, but no other physical signs in lungs; first cardiac sound rather blurred, and impulse to be felt in line with nipple and half an inch external; urine contains trace of albumen. A mixture containing acetate of ammonia ordered every six hours. At 10 P.M. temperature 103.8°; patient delirious, attempting to get out of bed; $\frac{1}{4}$ gr. morphine injected.

December 11th. Coughing up a little rusty sputum. On right side of chest behind there is dulness at the base, extending up to angle of scapula. Tubular breathing, bronchophony, *râles*, and a pleuritic rub audible over dull area. On the left side behind there is diminished resonance at the base extending up to angle of scapula. Small area of tubular breathing, and bronchophony just below angle of scapula. The apex beat still displaced outwards. Strychnine added to mixture, and two ounces of brandy ordered daily. At 1 P.M. very restless and delirious, $\frac{1}{4}$ gr. morphine injected. At 4 P.M. the dulness on right side had increased upwards for 2 inches. At 5 P.M. 660 units antipneumococcic serum injected (20 c.cm.).

December 12th. Has passed a quiet night. This morning better; the physical signs are unaltered. Temperature 102.6°, pulse 112, respirations 60. At 11.30 A.M., 660 units

serum injected. During the day patient was restless, and $\frac{1}{4}$ gr. morphine was injected. At 6 P.M. the temperature rose to 104.5°. At 11 P.M. patient was delirious; $\frac{1}{4}$ gr. morphine again injected; brandy increased to $\frac{3}{4}$ iv.

December 13th. Temperature 100.4°, respirations 33, pulse 104. On the right side, the dulness has decreased by 2 inches; tubular breathing faint. On the left side, the tubular breathing replaced by fine *râles*. At 10.15 P.M. 660 units serum injected. Brandy omitted.

December 14th. Passed a quiet night, and is much better, this morning. Temperature 99°, pulse 92, respirations 36. On right side dulness replaced by impaired resonance, and tubular breathing by *râles*. Left side has completely cleared.

December 15th. There was a rise of temperature to 102.8° during night, but it has fallen to 99.4° this morning. Patient is now convalescent.

From this time he rapidly recovered. On December 17th, the heart sounds were normal, and the apex beat had returned to normal position.

On December 18th the physical signs in the chest had completely cleared. An urticarial rash appeared on the chest and limbs, and lasted two days.

Chart 2.

Remarks.—The patient, though young, was a bad subject for pneumonia on account of his intemperate habits. The attack was a severe one, and both lungs were affected. There was a good deal of delirium; the pulse was 140, and the heart showed signs of dilatation on the second day of the disease. The serum appears to have exerted a beneficial effect upon the disease. On the day after the first injection the pulse-rate had fallen from 132 to 112. The process in the left lower lobe never advanced after the treatment was begun. It will be observed that the serum was injected at an early stage, namely, on the third day of the disease. In both cases an urticarial rash was observed during convalescence similar to the rashes occurring after injection with antitoxic and antistreptococci sera.

ADMINISTRATION OF SERUM.

The serum should be injected into the subcutaneous tis-

sue, and strict aseptic precautions should be taken. The skin should be well washed with an antiseptic, and the syringe should be boiled immediately before use. As to the dose, 20 c.cm (660 units) should be injected, and it would probably be well to inject more frequently than in the cases above recorded. I would suggest that the injection be made twice a day until the patient is convalescent. It is important to commence the treatment as early in the disease as possible.

REFERENCE.

¹ *Journal of Pathology and Bacteriology*, April, 1895; and *Zeits. f. klin. Med.*, 1894.

THE HUNTERIAN LECTURES

ON

THE PATHOLOGY AND SURGERY OF
INTUSSUSCEPTION.

Delivered at the Royal College of Surgeons of England.

By D'ARCY POWER, M.A., M.B. OXON., F.R.C.S. ENG.,

Hunterian Professor of Surgery and Pathology; Surgeon to the Victoria Hospital for Children, Chelsea.

LECTURE III.—(Concluded).

MORIBUND INTESTINE.

WHEN the surgeon has opened the abdomen and finds that the intussusception is greatly congested and has lost its gloss he must not too hastily assume that it is dead, yet it is often a matter of no slight difficulty to decide whether or not the bowel is capable of recovery. A piece of intestine which is only congested will bleed if it be pricked, even though it has lost its lustre and if it be gently stroked until its vessels are emptied the blood will be seen to pass along the vessels again as soon as the pressure is taken off them. A piece of intestine in such a condition must be handled very tenderly. It is unnecessary to remove it, and the patient is often so collapsed as to render any prolonged operation impossible, even if enterectomy were advisable. The wisest thing to do in such cases is to wrap a layer or two of gauze round the injured intestine as soon as the invagination has been released. One end of the gauze is left hanging out of the abdominal wound, the intestine is laid inside the peritoneal cavity, and the incision is lightly closed with temporary silk sutures. If the bowel ruptures the intestinal contents may then find their way out of the abdominal cavity, whilst if it recovers the gauze can be removed and the wound will close by granulations.

b. IRREDUCIBLE INTUSSUSCEPTION.

We have hitherto considered the operation of abdominal section for the relief of comparatively simple cases of intussusception in which the ingenuity of the surgeon is not taxed unduly, for the intussusception is reducible. The really serious, and by far the most interesting, cases are those in which the intussusception is found to be irreducible after the abdomen has been opened.

Intussusceptions become irreducible under many different conditions. In the first place the intestine may be healthy, but it is kept invaginated by more or less extensive adhesions; by peculiarities of structure, such as twists in its axis or the forcing of one piece of intestine upon another; by great swelling of the intussusceptum, or by the presence of large tumours in the intussuscepted bowel. Secondly, an intussusception may become irreducible because the bowel is so gangrenous or ulcerated that the surgeon dare not exercise traction upon the tumour, nor dare he leave the injured intestine within the peritoneal cavity.

It has already been pointed out that slight and newly-formed adhesions do not necessarily render an intussusception irreducible, even by such a simple method of treatment as irrigation, though their presence is of unfavourable augury, because increased force is necessary to effect reduction. The adhesions formed in an ordinary case of intussusception which has been allowed to run its course unchecked may be very extensive and very dense. I found in ex-

perimental invaginations produced in cats that slight adhesions were formed between the intussusceptum and the returning layer of the intestine as early as twelve hours after the operation, and that the adhesions were formed earlier and were more rapidly converted into scar tissue in the enteric than in the ileo-cæcal invaginations. Clinical observations show how widely different is the tendency to the formation of adhesions in different persons. Mr. Marsh³⁰ performed abdominal section on a child about fifteen hours after the onset of symptoms, only to find that the intussusceptum had become so universally adherent to its sheath that it was impossible to effect a reduction. A similar experience befel Mr. Makins³¹ in a child aged 4 years, who had suffered from symptoms of intussusception for forty-eight hours. The serous coats of the bowel were so firmly united to each other that they could only be separated with difficulty when the bowel had been laid open longitudinally after its removal from the body. On the other hand, the formation of adhesions is sometimes delayed for an indefinite length of time, as in the cases seen by Dr. Lettsom,³² in which the symptoms of intussusception were of three months' duration, and by Dr. Leichtenstern,³³ where they had lasted eleven months, and yet the *post-mortem* examination showed that hardly any adhesions were present. Dr. Carver,³⁴ too, met with a remarkable case of the same kind, in which a boy aged 2 years and 9 months suffered from symptoms of intussusception for seven weeks. He had fecal vomiting for twenty-five days, and the bowel protruded at the anus for a fortnight, yet the intussusception was easily reduced after the abdomen had been opened, as there were hardly any adhesions. The adhesions are formed more readily along the concave than along the convex side of the intussusception, and their extent, at any rate in the ileo-cæcal forms, depends, I assume, upon the varying relations of the mesenteric folds which were described in my second lecture. They are not necessarily associated with suppurative processes, and, in spite of the cases I have just quoted, they are more often found in chronic than in acute cases.

Gangrenous and ulcerating intussusceptions form the second great group of irreducible intussusceptions of the bowel—a group which can be further subdivided into those associated with peritonitis and those in which there is no peritonitis though the belly may be full of stinking gas. I have already occupied some part of your time with a consideration of gangrenous intussusception, so that I shall not deal further with its pathology or causation, except to say that it is only produced with the greatest difficulty in experimental invaginations upon animals. I have quite failed to produce it in cats and rabbits, even when the vessels going to the neck of the tumour have been securely tied.

The treatment of an irreducible intussusception is fraught with danger, whether it be irreducible from mechanical obstacles, on account of extensive adhesions, or because of gangrene. The prognosis in these cases was formerly considered desperate, and with a few rare exceptions the patients were left to die even by those advanced surgeons who had dared to do a laparotomy. But the advance of abdominal surgery has led to a recognition of the extraordinary readiness with which wounds of the intestine repair when their edges are kept in proper apposition, and we now have good hope that our efforts will be crowned with success even in the most unsatisfactory conditions.

I. SIMPLE IRREDUCIBLE CASES.

In chronic cases, when the end of the intussusceptum is gangrenous; when the intussusception is short and when it is irreducible from adhesions landed to its neck from the presence of a polypoid tumour, or from a bulbous enlargement of the end of the intussusceptum, the method recommended by Barker,³⁵ Jessett,³⁶ and Greig Smith³⁷ may give excellent results. The broad principle underlying each modification of the operation is to ensure the union of the intussusciens with the intussusceptum by inserting a continuous suture at the neck of the tumour, then to slit open the intussusciens, remove so much of the intussusceptum as lies dead or free, stop the bleeding, and sew up the wound in the sheath. It is sometimes impossible to remove the intussusceptum after it has