teen minutes after the lethal dose of sulphate of physostigma had been given, the rabbit was lying on the side, and saliva was flowing copiously from the mouth; unfrequent, laboured, and noisy respirations were occurring; the cardiac contractions were extremely feeble, and at the rate of only nine in ten seconds; and the pupils had contracted to $\frac{7}{10}$ ths $\times$ $\frac{2}{10}$ ths of an inch. In fact, the animal was at the point of death.

A marvellous change, however, was quickly produced by the administration of sulphate of atropia. Two minutes after seven-tenths of a grain of this substance had been injected under the skin, the respirations were occurring at the rate of 18 in ten seconds, while their character was nearly normal; and the cardiac contractions were strong, and at the high rate of 50 in ten seconds, the rate before the antidote was given having been only nine in ten seconds. Soon afterwards, the pupils dilated and the flow of saliva ceased; and, by and by, the animal again turned from the side, raised the body on the limbs, and then assumed a perfectly normal posture. It was shown that the dose of sulphate of physostigma from which this animal had recovered was a lethal one, by administering to it, several days afterwards, a dose of equal size, without any atropia. The usual symptoms of physostigma-poisoning were thereby produced, and death occurred in sixteen minutes.

I have said that the antagonism between atropia and physostigma was tested in dogs as well as in rabbits, and in order to illustrate the nature of this antagonism in the former animal, it may be proper to give a few details of one of my experiments. An active young Scotch terrier dog, weighing ten pounds and three ounces, received, by subcutaneous injection, three-fifths of a grain of sulphate of physostigma, dissolved in a few drops of distilled water. Before the injection, the rate per ten seconds of the cardiac impulses was 32, and that of the respirations 4, and the size of the pupils, in a full light, was $\frac{1}{10}$ ths $\times$ $\frac{1}{10}$ ths of an inch. In four minutes after the commencement of the administration, slight tremors occurred, and fibrillary twitches were present. In five minutes, a solution containing three-tenths of a grain of sulphate of atropia was injected under the skin. In two minutes thereafter, the tremors already noted had become more prominent and strong, the limbs were unable properly to support the body, saliva escaped from the mouth, and the eyeballs were unnaturally moist. In five minutes, the pupils were greatly dilated, but now the secretions of the salivary and lacrymal glands were diminished. In seven minutes, the dog lay quietly on the abdomen and thorax; and in thirteen minutes it fell over on the side. An endeavour was made to count the cardiac impulses, but, when the hand was placed over the heart, the tremors referred to became so greatly increased that it was impossible to distinguish the heart's impulse. It was not until thirty-eight minutes, that an attempt to count the heart's contractions was successful, and then it was found that the impulses occurred at the rate of 45 in ten seconds. At the same time, the respirations had a rate of 7 in ten seconds, and the pupils measured $\frac{1}{10}$ ths $\times$ $\frac{1}{10}$ ths of an inch. In forty-eight minutes, the condition of the dog had so far improved that, after some efforts, it rose on the limbs, and then lay down in a normal crouching attitude, with the head raised. Soon afterwards, it again got up and walked about the room, with only a little unsteadiness. In one hour and fifty-five minutes, the animal seemed to be perfectly well. On the following day, the dog was active, and in a perfectly normal condition. Nineteen days after the performance of this experiment, the same dog received, by subcutaneous injection, a dose of sulphate of physostigma, only one-half as large as that from which it had recovered when atropia was also given; and the result was that death was produced in twenty-two minutes.*

Gentlemen, the details of these three experiments will serve, I trust, to convince you that atropia exerts a powerful counteracting influence upon the lethal action of physostigma. I am glad to be able to state that several experiments bearing on this antagonism have been performed by Dr. Bourneville of Paris, which have led to equally satisfactory results. The experiments which I have brought under your notice by no means represent the amount of evidence that may be advanced in support of this antagonism; for results similar to those I have described were obtained in a large number of other experiments. These additional experiments, however, were not undertaken for the mere purpose of increasing the amount of this evidence.

* Full details of these, and other similar experiments, are contained in a paper by the author, in the *Transactions of the Royal Society of Edinburgh*, vol. xxvi, part III, 1870-71, pp. 529-713.

[To be continued.]

TESTIMONIAL.—Mr. Clement Hamerton of Navan, F.R.C.S.Irel., has been presented with a carriage, a purse containing £46 : 10, and a highly complimentary address, as a mode of evincing the gratification of his friends at his recovery from a severe and protracted illness.

CRITICISMS OF DR. CHAUVEAU OF LYONS ON THE DISCUSSION AT THE PATHO- LOGICAL SOCIETY ON PYÆMIA.

By J. BURDON SANDERSON, M.D., F.R.S.,
Professor of Practical Physiology in University College.

I HAVE to thank the Editor of the BRITISH MEDICAL JOURNAL for having called my attention to a recently published criticism, by my friend Dr. Chauveau of Lyons, of my communication to the Pathological Society last April on the subject of pyæmia. The criticism in question forms an Appendix (entitled "*Le poison pyohémique à la Société Pathologique de Londres*") to a course of lectures on the Physiology of Infective Liquids, which has been published during the last three months in the *Revue Scientifique*. In these lectures the author has embodied the results of a lengthened and most important experimental inquiry, which in its general bearing somewhat resembles that in which my colleague Dr. Klein and I were engaged last winter. M. Chauveau's purpose is to demonstrate the close relation which exists between the virulent (or, as I prefer to call them, the infective) diseases and ordinary inflammation. He founds this *rapprochement* mainly on the resemblance between the irritant properties of inflammatory products, and those of the specific morbid poisons; and asserts that ordinary pus induces inflammation in any living tissue with which it is brought into relation, in the same way that a virus reproduces the disease from which it originated under similar conditions. Those of M. Chauveau's experiments which have to do with recent—i.e., living—inflammatory products are in close relation with ours. But, in addition to these, he has made others in that older field of inquiry which concerns the toxic action of pus in various degrees of putridity.

Knowing as I do, by personal intercourse with M. Chauveau, the extreme accuracy of his method of working, and regarding myself as in some measure his pupil (for there are few men from whom I have learnt more pathology), I felt perfectly certain, as soon as I found that we were on the same ground and looking in the same direction, that, if there were disagreement between us, it could only arise from the imperfect manner in which the facts had been presented on one side or the other—in this case on my side.

At the Pathological Society, I founded what I had to say on an entirely new experiment, which I then regarded, and still regard, as a fundamental one. It is to this experiment, or rather to my interpretation of it, that M. Chauveau objects. I shall have no difficulty in showing that his objection arises from a misconception. He describes it in terms which (with the important exception of the words I have put in italics) are correct: "If a pyæmic liquid, introduced into the peritoneal cavity of a guinea-pig, be left there for a couple of days, *during which it does not determine any intense symptom in the animal*, the toxic power of that liquid increases to such a degree that, when taken from the first animal and transported to a second, it manifests the most pernicious activity, and produces symptoms which are very rapidly fatal." This, M. Chauveau adds, was demonstrated to the Society in a "dog, into the abdominal cavity of which six drops of a pyæmic liquid which had resided two days in the peritoneal cavity of a guinea-pig had been injected."

M. Chauveau expresses no doubt as to the strict accuracy of the facts, but thinks I have misunderstood them, and proceeds to recite an experiment of his own, which appears to him to furnish the key to mine. It is as follows. An old horse was sent to the veterinary school with a seton, the discharge from which was extremely fetid; the animal, however, was in good health; pulse, 32; temperature in rectum, 99.7 deg. Pus was collected from the seton, diluted with twice as much distilled water, and strained. Of this liquid, 15 minims were injected subcutaneously on the right side into the neck of the same animal from which it had been taken. In twenty-four hours, the pulse had increased to 45 and the temperature to 101.8 deg. On the fourth day the animal died. There was an enormous diffuse swelling around the seat of injection, due to "œdematous gelatiniform infiltration" of the subcutaneous tissue. The swelling was gangrenous at the centre, and exhibited elsewhere patches of vascular engorgement or extravasation. There were no internal lesions. Thus, to quote M. Chauveau's own commentary on the facts, "a few drops of the same pus, which when contained in a pyogenic cavity occasions neither local irritation nor any appreciable general disturbance, when injected into the cellular tissue of the same animal destroys it in less than four days; and the inflammation thereby produced is of so violent a character that the circulation stops, hæmorrhagic infarctus are formed, the tissues die, and the animal

succumbs to poisoning caused by the absorption of the putrid products engendered by this gangrenous phlegmon". The experiment is of great interest, and, as Chauveau says, well worthy of the attention of the surgeon, but it is too widely different from ours to admit of comparison with it. To make this difference evident, all that is necessary is to refer to the professed purpose for which it was made. M. Chauveau's object was, to use his own expression, to investigate the action of "pus putride"—i. e., of stinking pus. Our object, on the contrary, was not to inquire into the properties of putrid liquids, but of the products of inflammation; and for this reason, the most fastidious care was used to avoid the introduction of the element of putridity at any stage of our experimental procedure, by preserving our liquids from the possibility of septic contamination. In the first inoculation, the "pyæmic liquid" (as M. Chauveau calls it) used was simply the product of an acute inflammation, produced by the subcutaneous injection of a chemical irritant. This liquid was introduced into the peritoneum of a guinea-pig in extremely small quantity, and produced there an intense peritonitis. It was the product of this peritonitis which exhibited that extraordinary virulence, which I sought to illustrate to the Society by the experiment shown. In describing the results, I applied to them the term *intensification*. To this term M. Chauveau objects, and reasonably enough, for he supposes that in using it I meant to imply that in the case of the guinea-pig which received the first product and yielded the second, the former was *intensified* by mere residence in the uninfamed peritoneum of a healthy animal. I need scarcely say that this was not what I meant. In using the word *intensification*, I meant to apply it, not to the liquids, but to the *process* which produced it; for obviously the infective liquid finally obtained is not the same with that which was first injected, but simply the product of the inflammation excited by it—i. e., of the peritonitis. Its virulence, its infective activity, depends not on the mode in which the inflammation originated, but on the actual intensity it had acquired during the forty-eight hours of its duration.

There is one other point in M. Chauveau's paper to which I would ask space to refer. He objects strongly to my statement "that the ordinary bacteria of putrefaction do not in themselves possess a toxic action, and that liquids containing them can be injected into the circulation of a living animal without producing any effect". The statement, as it stands, is perfectly correct. Bacteria of putrefactive origin, if cultivated in liquids which are themselves innocuous, can be injected into the veins without producing any disturbance. It was suggested that in all probability the same thing would hold good of the bacteria of infective origin; but it was not stated either that the organisms of an infective liquid would, if they could be separated from it, be innocuous or inactive, or that the liquid without its organisms would retain its activity. As a result of these considerations, the opinion was expressed that, although these living particles are probably the carriers of infection, there is as yet no sufficient ground for supposing that their existence is the essential condition of infectiveness of certain products of inflammation.

A CASE OF RESECTION OF THE KNEE FOR BONY ANKYLOSIS, WITH THE LIMB IN FAULTY POSITION.

By JAMES E. ADAMS, F.R.C.S.,
Senior Assistant-Surgeon to the London Hospital.

CASES of bony ankylosis of the knee-joint with the bones at right angles to one another, are fortunately of somewhat rare occurrence; but it is evident, from the examination of specimens in the museums, that even among the few cases one is likely to meet with, there will be considerable variety in form and degree of union between the bones. It is therefore not sufficient to determine that any case is one of true bony ankylosis, but we must ascertain, as far as possible, what extent of the surfaces of the articular ends of the bones are in contact, how much new bone has been thrown out, etc., before determining how to proceed.

Dr. Little, in his article in Holmes' *System of Surgery*, does not, apparently, allude to any cases except such as can be relieved by subcutaneous chiselling, for he says, "that it (the subcutaneous section) cannot fail to become a standard operation, and is, in every respect, preferable to the operation of resection, when performed for mere ankylosis."

Now, the point I wish to bring forward is this; that there are certain cases of knee ankylosis which can easily be cured by the operation described by Dr. Little, and others in which anything short of an excision of a wedge-shaped piece is wholly useless.

Drawing No. 1* is intended to show a case of slight ankylosis with the anterior portions of the articular surfaces entire and separated by a wide

Fig. 1.

interval, and No. 2 represents such a case as mine, where all trace of the articular layers is destroyed, and the cancellous texture of one bone is continuous with that of the other, over the entire extent of the articular surface.

N. S., aged 14, a pale but fairly nourished boy, was admitted into the London Hospital in April last, with his right leg fixed at a right angle, the skin at the point of flexure marked by numerous cicatrices on all sides, some, especially those in the popliteal space, puckered in and adherent to the bones. There was absolutely no pain or tenderness; there was the most complete rigidity (under chloroform); the adjacent portions of the bones were expanded and rounded, so as to leave no interval or depression between them; the position of the patella could not with certainty be made out. The disorganisation of the joint occurred when he was one year and a half old, consequently the limb had been wholly useless for twelve years and a half; notwithstanding this, there was not much wasting and no appreciable shortening. On May 8th the operation was undertaken; a semicircular flap was raised from the front as in ordinary excision of the knee, and the original point of union carefully sought for, but no indication of it was found. A cut was then made with the saw in the direction of line 1 (Fig. 2), extending not quite through

Fig. 2.

the bone; this was joined by a second cut (line 2) meeting it about half an inch in front of the posterior surface; the piece between them was then removed, and the remaining portion broken by flexing the limb. The object of this was to avoid any possibility of wounding the popliteal artery, which, it was surmised, might be lying closer to the bones than natural (supposing the posterior ligament to be destroyed); moreover, it was expected that there might be some new bone thrown out at

* Both diagrams are intended to represent vertical sections through the bones.