

Gram negative.

Does not form spores.

Sluggishly motile.

Does not coagulate milk.

Pathogenic to white mice : 2 drops of a broth culture intraperitoneally killed in 48 hours. Organism recovered in pure culture from heart blood.

Forms acid and gas in glucose, galactose, levulose, maltose, and mannite quickly, and in dulcitol and salicin more slowly.

Does not form acid or gas in lactose, saccharose, dextrin, adonite, or inulin.

Grows well on potato.

Litmus milk shows at first slight acidity, later becoming alkaline.

Indol is formed in peptone water.

Voges and Proskauer's reaction negative.

The organism is evidently not *B. coli communis*. It was strongly agglutinated by the patient's own serum, over 1 in 1,400, but not by typhoid or paratyphoid A or B serum.

The patient's serum did not agglutinate *B. typhosus*, para A or B.

The organism differs from the group containing *B. suispestifer*, *B. enteritidis* and paratyphoid B in forming indol and fermenting salicin, and from the group containing *B. coli communis*, *B. neapolitanus*, and *B. acidilactici* in failing to ferment lactose. A good many organisms, as yet unnamed, have been described during the last few years as occurring in the urine. This is another.

There are three suppositions to be considered.

The patient's urine originally contained

(1) both *B. coli* and the unnamed bacillus;

(2) only *B. coli*;

(3) only the unnamed bacillus.

If (1) was the case, then *B. coli* had died out (a) owing to the presence of the unnamed bacillus, (b) owing to the vaccine employed; (c) for some other reason.

If (2) was the case, then a new infection of the urine with the unknown bacillus occurred and *B. coli* died out from the effect either of the new bacillus or of the vaccine.

If (3) was the case, then the original diagnosis of *B. coli* in the urine was incorrect.

Neither of the first two suppositions appears to us to be probable, and we are inclined to believe that (3) was the real state of affairs, that the diagnosis of *B. coli* in the urine was made on insufficient grounds. Further, it is quite possible that the patient's original illness was due to infection with this bacillus and not to paratyphoid A at all, considering the fact that he had no agglutinins in his serum for para A about four months after the beginning of his illness, whereas it agglutinated his own bacillus in dilutions of over 1 in 1,400.

The objects of this note are (1) to add another unnamed bacillus to the number already recorded as living and multiplying on occasion in the bladder for considerable periods; (2) to draw

attention more closely to the fact that when a urine is found to be swarming with bacilli it does not follow that they are either *B. coli communis* or belong to the typhoid group.

Nothing short of a complete bacteriological examination in a laboratory can determine precisely what the infecting organism is or is not, and, until the determination is made, treatment with a vaccine should not be considered for the reason that strains of bacilli of the colon-typhoid groups differ biologically so much that a vaccine which is not prepared from the infective strain in all probability will be useless. It does not follow that a vaccine will be effective even if prepared from the autogenous strain.

ADDENDUM TO AUTO-HÆMIC OR AUTO-SERUM THERAPY.

By D. N. SEN, L.M.S., *Pathologist*,

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THE following few lines are intended to form an addendum to my article "Auto-Hæmic or Auto-Serum Therapy" published in the March number of this journal. Since its publication I have received numerous enquiries about the subject from medical men all over India which have been replied to individually. I have also read with great pleasure the letter on the subject by Dr. R. K. Sen in the June issue of the Gazette.

The article "Auto-Hæmic or Auto-Serum Therapy" appeared to be complex and technical but really it is not so. The following details will elucidate the matter.

The treatment of patients is generally carried out by any of the following agents:—(1) normal serum, (2) defibrinated blood, or (3) hæmolysed blood of the patient. The preparation of the first two agents is quite a simple matter and can be carried out by the bedside of the patient who receives the injection within a few minutes of the withdrawal of blood. The method of hæmolysing blood is a bit complex, but with a little care and practice it is not at all tedious. Any of the above agents may be used according to the choice and convenience of the practitioner in the treatment of diseases with equally good result.

The preparation of—(1) Normal serum. It is obtained by the coagulation of the patient's blood. For this purpose 5 c.c. or 10 c.c. of venous blood is taken aseptically by a syringe. It is poured into a sterilised test tube plugged with cotton-wool or a sterilised one ounce glass stoppered phial and allowed to coagulate. The clear serum stands out of the clot and is used as a therapeutic agent. From every 4 c.c. of blood about 1 c.c. of clear serum is derived in 15 to 20 minutes' time.

(2) Defibrinated blood is prepared by withdrawing blood in the way above described and transferring it to a sterile glass flask.

Then defibrination is effected by whipping it with a glass rod or shaking with glass beads for about ten minutes and the resulting defibrinated blood is ready for injection. If it be for intravenous injection, it should be filtered through a layer of sterile gauze after defibrination. For hypodermic injection this precaution of filtering is not necessary.

From the above it is clear that during the preparation of normal serum or defibrinated blood they are not subjected to any laboratory process. The "certain laboratory processes" mentioned in the beginning of my former article refer to dilution, lysis and thermolysis. These laboratory processes are resorted to only in the case of hæmolysing the patient's blood and will be appreciated when I deal with the process of hæmolysis.

(3) Hæmolysed blood for carrying out hæmolysis of the patient's blood successfully it is necessary to have (i) a steriliser and (ii) an incubator. The possession of the above is a distinct advantage as contamination rarely occurs. But the serum may be prepared with ordinary appliances in the most successful if attention to asepsis is paid. Moreover, the technique is very simple, and I have made out the following method for myself.

The technique—(a) to 10 c.c. of freshly sterilised distilled water (kept in a sterilised test tube plugged with sterilised cotton) add $\frac{1}{2}$ c.c. of the patient's blood drawn aseptically from a vein in front of the elbow joint by a sterilised syringe.

(b) Thoroughly mix the blood with the sterilised distilled water by gently shaking the test tube.

(c) Then put the test tube in an incubator or thermos-flask at a temperature of 42°C. for 24 hours. (A thermos-flask may serve the purpose of an incubator.)

After 24 hours we find that the hæmolysis of the patient's blood is complete. The supernatant fluid is transparent but red in colour and the stroma settles to the bottom of the test tube. This supernatant fluid is the hæmolysed blood or auto-serum of the patient which is used as a remedial agent. Before injecting the serum, it is absolutely necessary to ascertain whether the fluid is sterile. For this purpose, take a drop of the fluid with a sterilised platinum or ordinary needle and spread it over a slide. Fix it by heat and stain by methylene blue or any other stain. Then look under an oil immersion lens in the microscope for any organism. If there be no organism, the fluid is quite sterile and is ready for injection. If there be any organism, the serum should be rejected and never be used. The process, then, should be repeated until a sterile serum is obtained.

From the above, it is clear that the blood is first diluted and then hæmolysed by distilled water. It is also thermolysed, *i.e.*, hæmolysed

and solution of the chemical constituents of the blood are accelerated and effected by the heat of the incubator or thermos-flask. The heating of the solution to a temperature of 42°C. accelerates hæmolysis and simultaneously helps to split up the highly complex constituents of the blood into simpler substances by the liberated ferments obtained by hæmolysis which were previously inactive by being intracellular. It will thus be seen that by subjecting the patient's blood to the laboratory process of dilution, lysis and thermolysis, a serum is obtained, which is not a mere solution of blood corpuscles (hæmolysed blood) but also of chemical substances quite different from those found in the normal blood.

No chemicals or preservatives are added to the fluid either during or after the preparation of the serum. The serum can be preserved in sealed bulbs in a cool place for future use for about a month without the addition of any chemicals whatsoever.

In one operation, *i.e.*, by mixing $\frac{1}{2}$ c.c. of blood with 10 c.c. of sterilised distilled water, I get about 7 c.c. of serum rejecting 3 c.c. of turbid stroma which is quite sufficient for 3 injections.

Directions for use—Injections are given twice a week. Intravenous injections are always given by me. They are less painful and more efficacious. I never use the serum obtained by one operation for more than two injections. The use of freshly prepared serum gives better results.

Dose of hæmolysed blood—1st injection, 1 c.c.; 2nd, 2 c.c.; 3rd, 3 c.c.; 4th, 4 c.c.; 5th and subsequent injections, 5 c.c.

The above dose holds good for normal serum, but for defibrinated blood is double the quantity.

With reference to Dr. R. K. Sen's note of using "a vastly simpler and easier method of auto-hæmic therapy" which consists in "withdrawing 20 c.c. of the patient's venous blood and injecting the same hypodermically while still warm and liquid in influenzal-pneumonia cases," I may say that the method of injecting "whole and unaltered" blood is not much in vogue in auto-hæmic therapy. Moreover, difficulty is sometimes experienced in injecting the whole blood as clotting takes place. The other method mentioned by him is the injection of serum taken from a blister produced on the patient's body by a blistering agent. This is indeed a well established practice. But its disadvantages are that the patient refuses to undergo such a painful method repeatedly and that care is needed for the after-treatment of the wounds produced by the blisters. When other methods fail it may be tried.

Now it is evident that the preparation of auto-serum, be it normal serum, defibrinated blood, or hæmolysed blood, is on the whole a simple matter and the supposed difficulty of preparing

it must not form an obstacle to its general employment.

Regarding the application of Wright's Opsonic Index method as a means of determining the time and dose of injection of auto-serum, it may be remarked that it becomes purely a complicated and academical subject rather than of practical use. Moreover, authorities differ regarding the value of the Opsonic Index.

The subject of auto-hæmic therapy affords an ample field for further investigation. Numerous observers have reported very favourable results following the administration of normal serum, defibrinated blood, hæmolysed blood, serum obtained from blisters, and pleuritic or ascitic fluid of the patient in the treatment of various diseases such as eczema, asthma, chorea, leprosy, neuralgia, ascites, influenza, typhoid fever, pneumonia, pleurisy, gonorrhœal arthritis, erysipelas, pellagra, headache, neuritis and metabolic diseases. Other investigators, on the other hand, have stated that they never observed the slightest influence of auto-serum injection in the treatment of typhoid fever, pneumonia, and psoriasis. But the consensus of opinion is that the auto-hæmic treatment has been well tested in functional and metabolic disorders and has proved of much value; whereas, in bacterial diseases, how far it is efficacious has not yet been fully determined.

OSMOSIS THROUGH THE SKIN.

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I do not know if there is any literature on the subject, and this is my excuse for publishing the results of a few observations made on the subject during the treatment of certain diseases.

On the 2nd September, 1916, a patient was brought to the hospital with general anasarca.

The case was diagnosed as one of Bright's disease. He was given the usual treatment of purgatives without effect. The tension of the fluid in the skin was very great, so much so that fluid appeared to be on the point of bursting through the skin of some parts of the body, such as the scrotum and about the ankle joint. This tension of the fluid in the skin suggested to me osmosis as a means of treatment. This was carried out by hypertonic saline baths. The brief history of the case and its progress is as follows:—

The patient was a boy of 14, duration of swelling one month. The accumulation of the fluid in the skin was very great. The puffy eyelids almost obstructed the vision. There was hydrothorax and ascites. The swelling of the thighs and scrotum prevented the patient from walking. Urine, which was scanty, showed on boiling a thick cloud of albumen, filling half the length of the urine in the test tube. For the first three days the patient was given magnesium sulphate

and tincture digitalis with no effect. From the 5th September, 1916, saline baths at the body temperature (dr. ii to a pint) were given for two hours twice a day. The patient was put on milk diet and given tinct. ferri. perchlor., all other medicines were stopped. By the 8th September, there was marked reduction of the anasarca over the knees, and the skin at many places became wrinkled. On the 9th September, the patient developed erysipelas in the right leg, which subsided by the 14th September. The baths were continued all along and the anasarca and the ascites were reducing. On the 16th September, the patient developed dysentery (the usual scanty stools of mucous and fæcal matter and not copious watery discharges) which continued more or less till the patient left the hospital. By the 18th September, the anasarca had subsided, with the exception of a little puffiness of the eyelids and swelling of the feet. Urine, on boiling, showed no precipitate, but only a cloudiness. There were a few tube-casts and the urine was free. From 19th to 23rd, when the baths were altogether stopped, the patient was given a bath only once a day, owing to the cloudy weather. The patient left the hospital on the 26th September, when all anasarca and ascites had disappeared, a slight puffiness of the eyelids and œdema of the feet still remaining. Examination of the urine showed no tube-casts but just a little milkiness on boiling. Nitric acid test was negative. Urine was quite free.

It is interesting to note that, with the subsidence of the anasarca, there was a reduction in the quantity of albumin and an increase in the flow of urine.

The patient reported at the hospital on the 21st October, 1916. He was still anæmic, with slight puffiness of the face, and no œdema of the feet. Urine was free; albumin, just a cloud, but no tube-casts. He reported again on the 10th December, 1916. Albumin the same as the last time; no tube-casts and no œdema. The patient, on both the occasions, refused to undergo a further course of treatment.

At about the same time when the above case was being treated, a patient, who used to attend the outdoor for abscesses in the right elephantoid leg, was admitted in the hospital on the 21st September, 1916, and saline baths tried on him. The patient, an adult male of 45, had the disease for three years. On admission, he had three sinuses which were almost healed up. The leg was too heavy for him and he had to crawl. He was given saline baths, the strength of which were gradually increased from 3 drachms to the pint up to 2 ounces and 3 drachms to the pint. From 23rd September to 17th November, 1916, the treatment was almost continuous, with a few interruptions when, owing to fever, baths could not be given. The leg used to swell up after every bath until the strength was increased to oz. ii and dr. iii to pint 1. The œdema then