flattening of the iliac bones, more marked on the right side. When worked out, the amount compares favourably with the doctor's direct measurements.

Between Iliac Crests.	Pelvis.	Skiagram.
Standard	28 cm.	32.5 cm.
Mrs. I.	23 cm.	26.8 cm.

At the level of the antero-inferior spines the bones are distinctly narrower than normal.

FIG. 6.—Drawing from skiagram—female pelvis showing deformity of iliac bones and normal cavity.

Internal.—On comparison with the "standard," the inlet appears normal, and when worked out it is as follows:

	Antero-posterior.		Transverse.	
	Pelvis.	Skiagram.	Pelvis.	Skiagram.
Standard	11.5 cm.	13.5 cm.	13.6 cm.	15.9 cm.
Mrs. I.	11.5 cm.	13.6 cm.	13.6 cm.	16.0 cm.

This case demonstrates clearly the advantages of this method of pelvimetry.

Fig. 7 is a case of normal full term pregnancy with a normal delivery, x-rayed at eight and a half months, and showing the fetal head engaging the pelvis.

FIG. 7.—Drawing from skiagram—fetal head engaging pelvis.

Conclusion.

In conclusion I would like to emphasize the following:

1. The necessity of a "standard" plate for pelvic measurements.
2. The patient must be radiographed in the same position as the "standard" pelvis, having the same point of focus, the x-ray tube at the same angle and at the same distance from the sensitive plate.
3. The accuracy of diagnosis in showing to advantage the variety of pelvic contraction, and the ease with which the various pelvic diameters can be worked out.
4. The minimum amount of discomfort to the patient compared to other methods of pelvimetry.
5. The absence of any ill effect to the fetus.

FROM October next the University of Maryland will admit women students to the medical department. Women have been admitted to the dental and pharmacy departments for some time.

THE PART PLAYED BY CONCOMITANT INFECTION WITH ANAEROBIC ORGANISMS (OTHER THAN *B. TETANI*) IN THE CAUSATION OF TETANUS.

BY

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(From the Laboratories of the Lister Institute.)

I. INTRODUCTION.

THE problem of tetanus is bacteriological, and susceptible of solution only by bacteriological inquiry. While it is a platitude thus to state that the important factor in the causation of tetanus is reproduction of *B. tetani* in the living tissues or in immediate proximity thereto, it is almost necessary at the present time to emphasize this factor of infection as opposed to the factor of intoxication. The tendency on the part of investigators to focus all attention upon the process of intoxication and upon its mechanism has led to the disease being regarded from too limited a view point—that of the pharmacologist and physiologist.

The result has been that the prophylaxis of the disease has been so far limited to the employment of tetanus antitoxin, and to those general surgical measures designed for the limitation of sepsis. Admittedly tetanus antitoxin has reduced to a very remarkable extent the incidence of tetanus, but one feels that, as the immunity conferred by the prophylactic use of this agent is transient, lasting apparently only seven to ten days, and as even its repeated employment at seven-day intervals for a period of four weeks has not absolutely eliminated the disease, other influences in its causation, notably those relating to infection, are worthy of special attention.

The general surgical methods directed to the limitation of wound infection have also done much to assist this reduction in the numbers of cases of tetanus. It must be clearly appreciated, however, that even the most efficient antiseptic is unable to sterilize an infected wound, for the conditions obtaining in a wound, notably the presence of living tissue, are such that the value of any reagent which can be incorporated in a dressing is much reduced in respect of its bactericidal and antiseptic properties. So far, no substance has been obtained which does not exert a deleterious influence upon the tissues at the same time as it exerts a restraining influence upon the growth of bacteria in the wound exudate.

The ideal method of dealing with war wounds, which are all infected to a greater or less degree, is excision of the wound area. This, however, is not always practicable, and even when practicable a certain number of cases are bound to remain considerably infected—though the mass infection is greatly reduced—owing to the conditions under which military surgery must be practised in the field.

In what Direction can the Prophylaxis of Tetanus be Improved?

The experiments to be described indicate one direction in which improvement can confidently be hoped for and the measures which they suggest, while they in all probability will result in the limitation of the incidence of gas gangrene, will also form a valuable adjuvant to the prophylactic measures at present in use for the prevention of tetanus.

The reason for conducting the experiments under consideration is indicated in the results obtained by Vaillard and Vincent and by other investigators, who demonstrated that tetanus spores deprived of toxin by washing and heating, when injected into animals, do not frequently determine the development of the disease. If, however, an irritant foreign body were introduced, or a solution of an irritant chemical were injected along with such spores, tetanus almost invariably supervened. Similar experiments, in which growths of staphylococci or other aerobic micro-organisms were used as the irritant, gave variable results.

It may be stated broadly, then, that anything which causes devitalization of tissue will enhance the infectivity of *B. tetani*. The devitalizing factors that play a notable part in war wounds are (1) trauma and the presence of

foreign bodies in the tissues, (2) concomitant infection with other organisms.

The first of these is, when possible, dealt with by the surgical measure of excision of the wound area, which procedure also to a great extent limits infection with all organisms. But upon the nature of the concomitant infection may largely depend the subsequent development of tetanus. Anything which tends to bring about necrosis, and especially necrosis of muscle, will greatly enhance the development of the tetanus bacillus in wounds.

Is there any Known Infection Common in War Wounds that Leads Especially to Devitalization of Muscle?

While any infection by staphylococcus, streptococcus, or the colon bacillus, etc., may to some extent lead to this, there are two organisms which are particularly suspect—namely, *B. welchii* and *Vibrio septique*. As both of these develop diffusible toxins, and as the toxin of *B. welchii* is known—in consequence of the brilliant work of Major Carrol Bull, U.S.A.M.C.—to be definitely “myotropic,” it seemed of special value to determine the influence of the toxins of *B. welchii* upon the development of tetanus. An investigation of this kind is all the more imperative in that an antitoxin to *B. welchii* is available, while the prevalence of this organism in wounds, and its capacity for rapid growth, make it a peculiarly dangerous constituent of wound flora. It seems not improbable, too, that this suggested symbiotic relationship between *B. welchii* and *B. tetani* may play some part in the causation of cases of delayed tetanus. It is conceivable that when wounds have healed and a slight trauma, received later, has resulted in the development of acute tetanus, there has been a reproduction of *B. welchii* or *Vibrio septique* locally which has exerted a trigger effect in the development of resting tetanus spores lying in the wound area. In such instances it is improbable, unless the trauma be fairly severe, that *B. tetani* develops without some assistance of the kind indicated. In an animal susceptible to tetanus the degree of infection by *B. welchii* required to light up tetanus need not be marked, so that it may well escape clinical observation.

Could we combat the necrotizing influence of *B. welchii*, and of other similar organisms, during the early stages of wound treatment much might be done to assist in the healing process, and, what is equally important, a healthier scar with fewer necrotic islets within it would be obtained. In order to investigate the symbiotic relationship suggested in the preceding paragraph the following experiments were undertaken.

II. EXAMINATION OF THE INFLUENCE OF THE TOXIN OF *B. WELCHII* UPON THE DEVELOPMENT OF TETANUS SPORES IN VIVO.

Reagents Employed.

In all series of the experiments the tetanus spores were prepared thus:

Tubes of Douglas broth, enriched by the addition of fresh sterile rabbit kidney and incubated to ensure sterility, were inoculated each with one of the definitive serological strains of *B. tetani*. The tubes were incubated for three weeks at 37° C., by which time the maximum sporulation had taken place. The growths were then filtered through sterile wool, centrifuged, and the supernatant fluid pipetted off. The deposit was suspended in saline and the tubes again centrifuged. This process having been repeated twice, sufficient saline was added to make the spore content of each suspension equal to 2,000 million spores per cubic centimetre, and the suspension was then heated to 80° C. for thirty minutes.

The toxin of *B. welchii* employed was of such potency that it caused the death of one guinea-pig of 250 grams weight out of three that were inoculated, when administered intramuscularly in a dose of 1 c.cm.; and only produced slight oedema when injected by the same route in a dose of 0.2 c.cm. diluted in 1 c.cm. of saline.

The antitoxin of *B. welchii* used was the product of the Rockefeller Institute of Experimental Medicine, and was marked as “Horse 2,” and contained 1,400 units of antitoxin per cubic centimetre.

A preliminary series of experiments was carried out (1) to determine approximately how many washed and heated tetanus spores could be injected into the guinea-pig without causing the development of tetanus, and (2) to determine what dosage of the toxin of *B. welchii* available invariably produced the disease (tetanus) when injected along with a constant number of tetanus spores, but was

by itself unable to cause more than slight oedema, demonstrable by palpation.

These preliminary experiments showed that if care were taken 1,000 million washed and heated spores could be injected in a volume of 1 c.cm. into the gastrocnemius of the guinea-pig without causing tetanus. It was therefore decided to employ this number of spores in all subsequent experiments, as such a number excluded the possibility of any inoculum consisting entirely of non-viable spores.

When this number of spores was injected along with 0.2 c.cm. of the toxin of *B. welchii*, the whole inoculum having a volume of 1 c.cm., acute tetanus invariably developed on the second or third day after inoculation. This experiment was carried out using three series of three animals each.

1. Animals *a*, *b*, and *c* were injected with 1,000 million Type 1 tetanus spores plus 0.2 c.cm. of *B. welchii* toxin, the volume of the inoculum being 1 c.cm. and the inoculation made into the right gastrocnemius.

2. Animals *d*, *e*, and *f* received the same inoculation, but Type 2 spores were used instead of Type 1 spores.

3. Animals *g*, *h*, and *i* received the same material, but Type 3 spores were used in this instance.

All nine animals died of acute tetanus between the second and third days after inoculation. Control experiments, in which 1 c.cm. of normal horse serum was inoculated into the left gastrocnemius before the inoculation was made on the right side, showed that normal horse serum did not prevent the fatal issue.

When a smaller dose of the toxin of *B. welchii*—less than 0.2 c.cm.—was employed, especially if it were diluted, the results were variable. It was therefore decided to employ in future experiments 0.2 c.cm. of the toxin. Having thus obtained an experimental method which gave constant results, I proceeded to determine whether passive immunization with *B. welchii* antitoxin would prevent the development of tetanus in animals inoculated with mixtures of tetanus spores and *B. welchii* toxin. The following series of experiments was therefore carried out. Details are given of the experimental methods employed in each series.

Experiment I.

Guinea-pig I was inoculated in the right gastrocnemius with 1,000 million spores Type 1 (U.S.A.) tetanus in 1 c.cm. saline. For the first five days the animal was well; on the sixth and seventh days it limped slightly (? local tetanus); on the eighth day there was no obvious limp, and on the remaining six days during which the animal was under observation it remained well.

Guinea-pig II was inoculated with 1,000 million spores Type 1 tetanus plus 0.2 c.cm. *B. welchii* toxin (volume 1 c.cm.). The following day there were signs of local tetanus; on the second day there was definite local tetanus; and on the third day the animal died.

Guinea-pig III also received 1,000 million spores Type 1, plus 0.2 c.cm. *B. welchii* toxin (volume 1 c.cm.), in the right gastrocnemius. One hour later 1,400 units of *B. welchii* antitoxin were injected into the left gastrocnemius. The animal showed no sign of tetanus, and remained well during the period it was under observation.

Experiment II.

Guinea-pig I A was inoculated in the right gastrocnemius with 1,000 million Type 2 tetanus spores in 1 c.cm. saline. It remained well for the first five days; on the sixth there was a slight limp (? local tetanus); on the seventh day there was definite local tetanus; on the eighth general tetanus, and the animal was killed.

Guinea-pig II A was inoculated with 1,000 million Type 2 tetanus spores, plus 0.2 c.cm. *B. welchii* toxin (volume 1 c.cm.). On the first day the animal appeared to be well; on the second it was moribund, and was killed.

Guinea-pig III A received 1,000 million Type 2 tetanus spores, plus 0.2 c.cm. *B. welchii* toxin (volume 1 c.cm.), in the right gastrocnemius. Two hours later 1,400 units of *B. welchii* antitoxin were injected into the left gastrocnemius. For the first five days the animal was well; on the sixth and seventh days it limped slightly (? local tetanus); on the eighth day the limp was less marked, and on the ninth day the animal was well, and remained well during the time it was under observation.

Experiment III.

Guinea-pig I B was inoculated with 1,000 million Type 3 tetanus spores in 1 c.cm. saline, in the right gastrocnemius. Guinea-pig II B received 1,000 million spores Type 3, plus 0.2 c.cm. *B. welchii* toxin (volume 1 c.cm.). Guinea-pig III B was inoculated in the right gastrocnemius with 1,000 million spores Type 3 plus 0.2 c.cm. *B. welchii* toxin (volume 1 c.cm.), and received 1,400 units *B. welchii* antitoxin in the left gastrocnemius. Guinea-pig I B remained well; Guinea-pig II B died of tetanus on the third day; and Guinea-pig III B, beyond exhibiting slight stiffness of the inoculated limb from the sixth to the fourteenth day after inoculation, remained well.

The deductions to be drawn from these three experiments are self-evident. They indicate that an antitoxin for *B. welchii* and tetanus antitoxin should be employed together for the routine serum prophylaxis of tetanus. The evidence is unequivocal that the antitoxin of *B. welchii*, in addition to neutralizing its toxin, has the advantage that it completely protects (in the case of the guinea-pig) against the development of tetanus spores in tissue which has been exposed to the devitalizing effect of that toxin. It is highly probable that the same protecting value would be found in the case of man.

Attention is called to the death of the control animal, I A, in Experiment II. The death of this animal, while in no way detracting from the validity of the experiments or of the deductions to be drawn from them, indicates that the development of tetanus depends upon a number of different factors. It is not improbable that in this instance a certain degree of trauma, when the inoculation was made, was responsible for the development of the infection. It is to be specially noted, however, that while the test animal, II A, died on the second day after inoculation, the control animal did not die until the eighth day. Moreover the test animal died of acute, almost fulminating, tetanus, while the course of the disease in the control animal was much less acute. Experiment II is therefore exceptionally instructive, for, while it shows that the danger of infection with *B. tetani* and notably of early development of the disease owing to symbiosis with *B. welchii* could be considerably mitigated, if not completely eliminated, by the use of a combined serum for prophylaxis, it also calls attention to the fact that while such a reagent would probably be of great value, surgical measures could not be permitted to be less carefully applied because of its introduction.

I call attention to this as, if a combined prophylactic serum were to be introduced, it might lead to the development of a feeling of false security, and too optimistic a view might be taken regarding the probable value of such a combined serum. As a natural sequence its unmerited condemnation would ensue if the results obtained did not completely bear out expectations.

III. INFLUENCE OF THE TOXIN OF *VIBRION SEPTIQUE* ON THE DEVELOPMENT OF TETANUS SPORES IN VIVO.

Vibrion septique is another of the organisms which are commonly found in wounds, and develop diffusible toxic products. While it is not known whether the toxin of this organism is a particularly active necrotizing substance or only has marked oedema-producing properties, it seemed of importance nevertheless to determine the possible influence which it might exert upon the development of tetanus.

As the properties of this toxin, then, are at present less well defined than are those of the toxin of *B. welchii*, the experiments to be described in this section cannot be so clearly interpreted as are those dealt with in section II of the present communication. This difficulty is enhanced by the fact that at the time of writing specific *Vibrion septique* antitoxic serum was not available, so that protection experiments could not be undertaken.

I here wish to record my thanks to Miss Robertson of the Lister Institute of Preventive Medicine for placing at my disposal a quantity of the toxin of *Vibrion septique*.

The methods employed and the results obtained in making this investigation were as follows:

Experiment IV.

Guinea-pig A was inoculated in the right gastrocnemius with 0.5 c.cm. *Vibrion septique* toxin plus 0.75 c.cm. saline. On the first day there was slight oedema and stiffness of leg. On the second day the oedema was palpable, but not extensive; there was still stiffness of leg. On the third day the oedema was almost gone and the leg less stiff. On the fourth day the animal had almost recovered, and on the fifth day it was well, and remained so during the period of the experiment.

Guinea-pig B was inoculated with 0.25 c.cm. *Vibrion septique* toxin, plus 0.25 c.cm. saline, plus 1,000 million tetanus spores, Type 1. On the first day there was slight oedema and stiffness of leg. On the second day oedema palpable but not extensive; still stiffness of leg. On the third day the animal was recovering, and on the fourth it had almost recovered. On the fifth day it was well, and remained so.

Although the oedema resulting from the injection of 0.25 c.cm. of this toxin was quite as marked as, if not more marked than, that produced by 0.2 c.cm. of the toxin of *B. welchii*, it is remarkable that tetanus did not

develop. I therefore decided to make the test much more stringent, employing 1 c.cm. of the toxin plus 1,000 million tetanus spores. The result of an experiment employing this technique is shown below.

Experiment V.

Animal C was inoculated in the right gastrocnemius with 1 c.cm. *Vibrion septique* toxin plus 0.5 c.cm. saline. On the first day there was marked oedema over the whole of the injected limb; on the second the oedema was more marked and extending over the abdominal wall, soft on palpation. The next day the oedema was reduced and the animal lively; limb stiff. On the fourth day the oedema was disappearing; leg still stiff. On the fifth day the animal had recovered, and remained well till the conclusion of the experiment.

Animal D received 1 c.cm. *Vibrion septique* toxin, plus 1,000 million Type 1 tetanus spores. On the first day there was marked oedema over the whole of the injected limb; on the second, oedema as in animal C. On the third day the oedema was reduced and the animal lively, but limb stiff. Fourth day, oedema almost gone; leg still stiff. On the fifth day the animal was well, and remained so.

A slight stiffness of the limb remained in both animals until the completion of the experiment, which was considered to have occurred on the fourteenth day. The animals have remained well, and the stiffness is slowly disappearing. This experiment is striking in that although the oedema produced in the animals was much more marked than that resulting from the injection of 0.2 c.cm. of the toxin of *B. welchii* employed in the previous series of experiments, no development of tetanus occurred.

This result was scarcely expected, and the experiment was therefore repeated.

Experiment VI.

Animal E was inoculated with 1,000 million Type 1 tetanus spores suspended in 1.5 c.cm. of saline. Animal F with 1 c.cm. of *Vibrion septique* toxin plus 1,000 million Type 1 tetanus spores. These animals both remained well, so corroborating the findings indicated in Experiment V.

As the disturbance produced by *Vibrion septique* toxin—oedema production—was so pronounced, it seemed remarkable that tetanus did not develop. The experiment was repeated a second time, using spores of a representative Type 3 tetanus bacillus. The findings of this experiment are as follows:

Experiment VII.

Animal G was inoculated in the right gastrocnemius with 1,000 million Type 3 tetanus spores suspended in 1.5 c.cm. saline. It remained well. Animal H was inoculated with 1,000 million Type 3 tetanus spores plus 1 c.cm. *Vibrion septique* (volume 1.5 c.cm.). There was marked oedema on the first day, but the animal was active. On the second day there was general tetanus, and the animal was killed.

As specific antitoxin for *Vibrion septique* was not available it was decided not to complete the present series of experiments using Type 2 spores, for the results shown in Experiment VII indicate that *Vibrion septique* may, like *B. welchii*, play an ancillary part in the causation of tetanus.

The negative results obtained on three occasions when Type 1 spores were used indicate, however, that the toxin of *Vibrion septique* is probably a less constant factor in stimulating the growth of *B. tetani* in the tissues than that of *B. welchii*. In view, however, of its frequent occurrence and in view of the fact that it sporulates more readily than does *B. welchii*, its capacity for doing harm is possibly extended over a more prolonged period than is the case with *B. welchii*. It would seem, therefore, advisable that antibodies to the toxin of *Vibrion septique* should also be included in serum used for the prophylaxis of tetanus.

Commenting on the results obtained in the two series of experiments described in the present communication, it is to be noted that Vaillard and Vincent foreshadowed the possibility of its being demonstrated that certain organisms might play a more important part in stimulating the growth of *B. tetani* in vivo than would others.¹

CONCLUSIONS.

1. There is good ground for believing that the ancillary part played by *B. welchii* in the causation of tetanus is clearly defined. The capacity of this organism for doing harm in the connexion under consideration can be almost eliminated by the use of the antitoxin for *B. welchii*.

2. The capacity shown by the toxin of *Vibrion septique* for stimulating the growth of tetanus spores in vivo is more

variable than is that of *B. welchii*. Experiment VII indicates, however, that it too may play a part in the causation of tetanus.

3. It follows from conclusions 1 and 2 that antibodies to the toxins of *B. tetani*, *B. welchii*, and *Vibrio septicus* should be included in all serum employed for the prophylaxis of tetanus.

4. While such a polyvalent serum promises to reduce still further the incidence of tetanus it would be too optimistic to assume that it would absolutely eliminate that disease, for infections other than those dealt with in this communication may also play a part in stimulating the growth of *B. tetani* in wounds. One of these, *B. oedematis*, I propose to investigate immediately.

REFERENCE.

¹ *Annales de l'Institut Pasteur*, January 1st, 1891.

ANEURYSM OF THE THIRD PART OF THE LEFT SUBCLAVIAN ARTERY, SUCCESSFULLY TREATED BY LIGATURE OF THE SECOND PART.

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IDIOPATHIC aneurysm of the subclavian artery is not common, and until recent years its treatment has been most unsatisfactory. Ligature of the first part of the artery is an operation of considerable difficulty, owing to the anatomical relations of the vessel and its depth from the surface, and in pre-antiseptic times was invariably fatal.

The second part, behind the scalenus anticus, has apparently not often been chosen for ligature; but it is far more accessible than the first part, and the operation is not especially difficult. In the case recorded below, I started the operation with the idea of ligaturing the first part, but found the second part could be more easily reached, and there was no difficulty in ligaturing the vessel. The result of the operation was completely satisfactory.

The patient, a farm labourer aged 50, was admitted into the East Suffolk Hospital on February 27th, 1918, complaining of severe pain in the left arm, which kept him awake at night. There was a pulsating oval tumour extending above the clavicle into the posterior triangle, rather smaller than a hen's egg. The left radial pulse was distinctly smaller than the right. The systolic pressure in the right brachial artery was 230 mm., in the left 195. The patient was kept in bed, given potassium iodide, and morphine at night.

Operation.

On March 8th, 1918, an incision was made along the posterior border of the sterno-mastoid, commencing at about its centre, and extending over the head of the clavicle for an equal distance on to the thorax; it was about six inches in length or rather less. The posterior edge of the sterno-mastoid was defined, the muscle drawn inwards, glands, etc., removed, and the internal jugular vein exposed. The attachments of the sterno-mastoid to the upper border of the clavicle, and of the pectoralis major to the lower border, were divided, and the inner half of the bone cleared with a periosteal elevator, the clavicle was divided with a saw, disarticulated at the sterno-clavicular attachment, and the inner half removed. The sub-clavius muscle was taken away and a large suprascapular vein; the subclavian vein was then exposed and cleaned. After removal of some lymphatic glands the scalenus anticus with the phrenic nerve was defined. It was evident that the aneurysm commenced at the outer edge of the scalenus anticus, and was confined to the third part of the artery. The first part had not yet come into view. The phrenic nerve was drawn inwards, the vagus, internal jugular, and internal carotid were also retracted out of the way, and the scalenus anticus muscle divided a short distance above its attachment to the rib.

The second part of the subclavian artery was now fully exposed to view. It appeared to be quite healthy and normal, and there was no difficulty in passing a ligature round it. Two silk ligatures of No. 4 silk were tied sufficiently tightly to occlude the vessel without rupturing the coats. Pulsation in the aneurysm and radial artery

immediately ceased. The wound was closed after suturing the divided sterno-mastoid and pectoral muscles, and a small drainage tube left in the centre of the wound. This was removed after forty-eight hours.

The patient made an uninterrupted recovery. The left arm and hand continued to be quite warm and free from pain. There was never any cyanosis or oedema. Muscular movements and sensation were unaffected. He left the hospital on March 28th, within three weeks of the date of operation.

I saw the patient again a month later—April 28th. He looked and felt perfectly well. All movements of head and arm were normal. He was quite free from pain and was advised to return to work. The radial pulse was still absent; the only trace of the aneurysm was a very small soft tumour above the clavicle resembling a lipoma. The systolic pressure in the right brachial artery was then 165 mm.

The operation has apparently been completely successful.

I am indebted to Colonel A. Carless for his kindness in giving suggestions as to the line of incision and *modus operandi*, and to my colleague, Mr. A. Y. Pringle, and Mr. Arthur Woo, the house-surgeon, for assistance during the operation.

Memoranda:

MEDICAL, SURGICAL, OBSTETRICAL.

A CASE OF CEREBRAL TOXAEMIA— ? "BOTULISM."

THE following notes relate to a patient recently under my care. In the light of the interest lately aroused in the subject of botulism, and in consideration of the "epidemic" character of that disease, a report of this case, which occurred in Glasgow, may be suggestive epidemiologically. So far as I am aware, no case has, at this time, been recorded further north than Leeds.

Clinical Features.

J. G., male, aged 35, was admitted on May 6th, notified as typhus. His illness began on May 1st. The history, given by the relatives, was unsatisfactory, but headache, sickness, constipation, stupor, and delirium were the prominent features. The latter two appeared early, persisted throughout, and, as the disease progressed, tended to deepen.

On admission: coma, pyrexia (101–108° F.), sudamina or toxic erythema, dry brown flaccid tongue, bucco-pharyngeal hyposecretion, dysphagia, palatal paralysis, divergent strabismus, slightly dilated pupils reacting sluggishly to light, and retention of urine. Cerebral excitement was a prominent feature, producing muscular tremors greatly aggravated by interference. Forcible raising of the arms or separation of the legs revealed a spastic tone of the musculature. Movements constant, irregular, quick, choreoid (as in choreic diplegia), involuntary—although sufficiently purposive to necessitate restraint of the arms to prevent interference with catheterization. Sensation appeared hyperacute, as even slight digital pressure induced exaggerated collateral muscular response. Knee-jerks exaggerated, Kernig and Babinski present, ankle clonus indefinite. Heart: rate 120–144, sounds indefinite. Lungs: bronchopneumonic catarrh. Trace of albumin in urine. Abdominal examination negative.

Although, clinically, the case in no way resembled typhoid fever, the Widal reaction showed complete agglutination of *B. typhosus* in a dilution of 1 in 50 in an hour and a half.

Whereas, on admission, there appeared to be photophobia, next day paresis of both levatores palpebrae superioris was evident, and on the day before death there was bilateral ptosis with slightly contracted, fixed pupils. Bilateral facial paralysis was suspected, but could not be established on account of the profound stupor. Percussion over the chin and attempts at drinking produced a curious clonus of the masticatory muscles. The electrical reactions were not examined. Sixty hours after admission death occurred peacefully as from a gradual paralysis of the respiratory centre.

Post-mortem Findings.

Skin.—Over the shoulders, upper arms, and sides of thorax there were numerous small, circular, brownish spots, of variable size, some showing minute vesicular elevation. Over both suprascapular regions and backwards over the shoulders there was a copious eruption of sudamina.

Chest.—The left lung was intensely hyperaemic and oedematous. The right lung had an ill-defined area of pneumonic consolidation in the lower lobe posteriorly, the upper part of the lobe and the upper lobes being hyperaemic and oedematous.

Heart.—The wall of the left ventricle showed cloudy swelling, and the cavity was dilated; valvular structures normal.

Abdomen.—Spleen enlarged, very hyperaemic, but fairly firm; kidneys hyperaemic; liver not enlarged, but on section some biliary staining and cloudy swelling. All over the cardiac end of the stomach the mucous membrane was hyperaemic, but