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Post-Abortal and Puerperal Gas Gangrene*

A REPORT OF THIRTY CASES.

BY

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SINCE the publication of Little's¹ classical article in 1905, examples of post-abortal and puerperal infection due to *clostridium Welchii* have been reported sporadically by American, German, English, French and Australian investigators.

In 1928 Toombs and Michelson² were able to discover 41 cases in the literature, and Toombs, describing a further example in 1932,³ was then able to raise the recorded number to 56. Since that date accounts of at least 28 additional cases have appeared in the American, German, Polish and Australian literature.

It has been the experience of comparatively few investigators, however, personally to encounter a large series of grave and fatal cases. Lehmann in 1926 recorded 10 such in a total of 15,⁴ and with Brütt⁵ in the following year was able to add at least four more. Sleeman⁶ in 1927 described the eighth fatal case he had seen. Heim⁷ in 1933 reported 10, of which eight died. Other writers have met with fewer of these desperate types.

It is my intention to record a series of 30 cases of post-abortal and puerperal gas gangrene clinically observed at the Women's

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Hospital, Melbourne, between April 1933 and February 1935. During that time the author was medical superintendent of the hospital, and each case reported was personally followed by me from, or shortly after, its admission until death or discharge. In 1934 there were indeed two further post-abortal cases, from each of which *cl. Welchii* and streptococci were isolated from the blood before death, but as both were admitted during the writer's vacation they are excluded from this series.

Of the 30 cases, 22 were associated with abortion (post-abortal), and eight with labour at term or near-term (puerperal). Twenty-seven were grave, and 19 fatal. This occurrence, within 23 months, of what I believe to be the largest single series yet published, presents an economic and medical problem of the greatest gravity.

Incidence.

Examination of the records of the Women's Hospital prior to 1933 would indicate, beyond all possibility of increasing diagnostic proficiency, or improved methods of bacteriological investigation, that the frequency of post-abortal and puerperal gas gangrene has never previously approached that of the two years here reviewed.

The following table, compiled from the reports of the Government's pathologist upon all cases of abortal sepsis that came to inquest in the city of Melbourne during the five years 1930 to 1934, is illuminating.

INCIDENCE (Abortal Cases).

City of Melbourne, 1930-1934.

Year	Inquests on deaths from abortal sepsis.	Deaths due to Sepsis other than gas infection.		Deaths due to gas infection	Percentage of deaths due to gas infection
		Without general peritonitis	With general peritonitis		
1930	16	8	7	1	6.2%
1931	22	15	5	2	9.1%
1932	23	18	4	1	4.3%
1933	24	10	10	4	16.6%
1934	30	13	7	10	33.3%
Total	115	64	33	18	Aver., 15.6%

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Women's Hospital, 1933-1934.

Year	Cases admitted suffering from abortion	Frankly infected on arrival	Deaths due to sepsis	Deaths due to gas infection	Percentage of deaths due to gas infection
1933	1484	332	25	4	16%
1934	1565	346	25	12	48%
Total	3049	678	50	16	Aver., 32%

In those cases here included as gas infection, in which complete bacteriological confirmation was lacking, the combination of history, clinical features and classical post-mortem findings left no doubt in the mind of an experienced pathologist as to the actual cause of death.

The figures for the Women's Hospital for 1933 and 1934 run almost parallel. In those two years 3,049 women were admitted suffering from abortion, 678 being already frankly infected on arrival. In each year 25 deaths resulted from sepsis, infection with gas bacillus accounting for four of these in 1933 and for 12 in 1934. The explanation of this remarkable increase is not easy to find.

Heim in 1933 reported a similar increase in case incidence at the Leipzig clinic. Of 10 undoubted examples of gas infection encountered during 10 years, seven had occurred in the preceding 11 months. Heim concluded that increasing economic pressure, combined with the exercise of a primitive technique of abortion by unqualified persons, provided the correct explanation. Although ample evidence of the second factor exists in our series, the greatest incidence by no means coincided with the national and local time of greatest economic pressure.

Of our 30 cases 26 were apparently city subjects, and 20 were admitted between September and February, that is, during spring and summer. It is of interest that in 1934 four cases were admitted within four days in January, and five within 16 days in November.

Bacteriology.

In 42 instances, representing 24 of the 30 cases, Gram-positive, gas-forming, anaerobic bacilli were cultured from the blood, uterine cavity, lochia, urine or peritoneal cavity during life. In 22 patients the organism was the *cl. Welchii*, and in two the *vibron septique*.

In six women ante-mortem bacteriological evidence of infection was not obtained. In two of these (Nos. 27 and 30) *cl. Welchii* was cultured from the tissues after death, and in two (Nos. 25 and 28) smears at autopsy revealed organisms with the morphological characteristics of *cl. Welchii*. In the remainder (Nos. 15 and 17) the diagnosis of gas infection rested on combined clinical and macroscopic pathological evidence; and in one (No. 15), the subsequent investigation of a practically identical case (No. 16) made confirmation almost irrefutable.

Blood-Cultures.

From 26 of the 30 patients—six of the eight puerperal, and 20 of the 22 post-abortal—blood cultures were taken during life.

In four of the puerperal class these were positive for *cl. Welchii*, in one instance in association with an anaerobic streptococcus. In the two patients from whom organisms were not recovered the cultures had been inadvertently discarded at the time of the patient's death, only 11 hours and 16 hours respectively after beginning incubation.

In all, 20 post-abortal patients positive blood-cultures were obtained, in 15 for *cl. Welchii* either alone or together with other organisms, in two for *vibron septique*, in two for an aerobic streptococcus and in one for *staphylococcus albus*.

Blood culture was performed in 18 patients once, in five twice, in two thrice and in one four times; 28 of the 38 cultures were positive.

(i) *Clostridium Welchii*.

In a total of 19 cases, *cl. Welchii* was recovered from the blood once in 16 and on two occasions in three cases.

In seven of the 19, or 37 per cent, the *cl. Welchii* was part of a mixed blood infection, the additional invaders being an aerobic streptococcus in three, an anaerobic streptococcus in two, aerobic streptococcus and *bacillus coli communis* in one, and an anaerobic streptococcus and *staphylococcus aureus* in one. If to these be added the three cases in which the streptococcus or *staphylococcus albus* alone was grown from the blood, but in which *cl. Welchii* was cultured from the uterus, the incidence of mixed infection with *cl. Welchii* becomes 10 in 22 cases, or 45 per cent.

(ii) *Vibron septique*.

In two cases *vibron septique* was recovered from the blood, in neither instance accompanied by other organisms.

Intra-uterine Cultures.

In eight post-abortal cases cultures were taken directly from the uterine cavity in seven, and from gauze removed therefrom in one. The time of culture was that of curettage in five, at a later date in two, and immediately following hysterectomy in one.

In all instances *cl. Welchii* was recovered, on one occasion in association with *staphylococcus albus*, and once with an aerobic streptococcus and Gram-negative bacillus.

Peritoneal Cultures.

These were taken from one puerperal and four post-abortal patients. In the puerperal and two post-abortal cases *cl. Welchii* was grown, in the latter being associated with an aerobic streptococcus in one, and with an aerobic streptococcus and Gram-negative bacillus in the other.

Vibron septique was isolated from one woman, and an aerobic streptococcus from another.

Lochial Cultures.

These were taken from four post-abortal cases. Three were returned positive, each for *cl. Welchii*, in one instance in association with other organisms.

Urine Cultures.

Urine cultures were taken from two post-abortal cases only. From each *cl. Welchii* was recovered, in one in association with an aerobic streptococcus and a Gram-negative bacillus.

Culture Media.

In every instance cultures were made aerobically and anaerobically in plain broth and cooked meat medium, respectively. For blood-cultures three to five cubic centimetres were injected into broth, and two or three cubic centimetres into the cooked meat medium.

Of greater value than cooked meat medium for the early detection of these organisms is anaerobic tryptic broth, which may allow of a recovery of *cl. Welchii* within 10 hours, or even less. In an infection so swiftly fatal, early diagnosis is of the first importance.

Identification of the Organisms.

(a) *Cl. Welchii*. In all cases the organisms recovered gave the morphological and cultural characteristics of *cl. Welchii*, including invariably the production of a stormy clot in milk. In nine patients—in six from the organisms recovered from the blood, and in three from those recovered from the uterus—

further confirmation was obtained by the production of a toxin from the organisms under investigation and, following its intravenous injection into mice, its neutralization by the specific anti-toxin.

(b) *Vibron septique*. In each of the three cultures taken from the two examples of this rare infection the organisms recovered showed the morphological and cultural characteristics of vibron septique, and in all final confirmation was obtained by the production of the bacterial toxin with, following its injection into animals, its complete neutralization by the specific antitoxin.

In the first case seen the strain was virulent, but vibron septique antitoxin protected guinea-pigs against as many as 20 minimal lethal doses. *Cl. Welchii* antitoxin did not give any protection.

Smears.

(i) *Lochia*. Organisms with the morphological characteristics of *cl. Welchii*, together with streptococci and Gram-negative bacilli, were present in a lochial smear from a woman in whom these organisms were cultured from uterus, peritoneal cavity and urine.

(ii) *Muscle*. In one case of metastatic gas gangrene organisms with the morphological characteristics of *cl. Welchii* were recovered from the deep muscles of the left buttock after needling.

Table I gives the synopsis of the bacteriological cultural investigations during life.

PRESENTATION OF CASES.

This will be entirely from the clinical standpoint, and under the following headings:

Post-abortal Series.

- (i) Infection with *cl. Welchii*.
- (ii) Infection with *vibron septique*.

Puerperal Series.

Infection with *cl. Welchii*.

Post-abortal Series.

There were 22 post-abortal cases, with 13 deaths, a mortality of 59 per cent.

The average age was 27.5 years, the youngest being 18, and the oldest 40.

Five women were nulliparous, the average parity of the remainder being just below four. In 17 pregnancy had advanced

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 TABLE I.
 BACTERIOLOGY (ANTE-MORTEM CULTURES).

Post-abortal series	Case No.	Blood-cultures				Intra-uterine cultures	Urine cultures	Lochial cultures	Peritoneal cultures
		First	Second	Third	Fourth				
I. Group A	1	Cl. W.							
	2	Cl. W.*							
	3	Cl. W.*	Cl. W.						
	4	Staph. alb.	Staph. alb.			Cl. W.			
	5	Cl. W.*	—			Cl. W.*	Cl. W.	—	Cl. W. Strep.
	6	Strep.							
	7	Cl. W.				Cl. W.			
	8	Cl. W.				Cl. W.			Cl. W. Strep.
	9	Strep.				Gm-ve bac.	2 { Cl. W. Strep. Gm-ve bac.		Gm-ve bac.
Group B	10	Cl. W.	—						
	11	Cl. W.							
	12	An. strep.							
	13	Staph. aur.							
	14	Cl. W.*	Strep.	—					
Group C	15	Cl. W.	—						
	16								
Group D	17								
	18	Cl. W.							
	19	Cl. W.							
	20	Strep.							
II.	21	Vib. sept.*							Vib. sept.*
	22	Vib. sept.*							
Puerperal series	23	Cl. W.	Cl. W.	—					Cl. W.
	24	Cl. W.	An. strep.						
	25								
	26	Cl. W.							
	27	—							
	28								
	29	Gl. W.*	Cl. W.	—	—				
	30	—							

INDEX TO TABLE;

Cl. W. ... Clostridium Welchii.
 Vib. sept. ... Vibrio septique.
 Strep. ... Aerobic streptococcus.
 An. strep. ... Anaerobic streptococcus.
 Staph. alb. ... Staphylococcus albus.
 Staph. aur. ... Staphylococcus aureus.
 B.c.c. ... Bacillus coli communis.
 Gm-ve bac. ... Gram-negative bacillus.

* Indicates final confirmation by specific toxin-antitoxin neutralization in animals.

to between six weeks and three months, and in five to between 14 weeks and five months.

Introduction of Infection.

A history of mechanical interference was obtained in 13 cases. One of these had had cervical plugging performed by a doctor to induce the abortion of a retained macerated foetus. The remaining 12 admitted attempts at criminal abortion, eight by forcible douching with Higginson's syringe using solutions of soap, Epsom's salts or lysol; three with instruments (including a crochet needle); and one with both an instrument and Higginson's syringe.

Interference in 11 cases had occurred between eight hours and five days before admission to hospital, the average being $2\frac{1}{2}$ days. In two (Nos. 22, 7) it had apparently last been instituted six weeks and two and a half months before, respectively; and in both symptomatology was absent until two days before admission. No woman who had used a Higginson's syringe admitted its employment on any prior occasion for rectal injections, and in one instance the purchase was new.

Seven women denied attempts at interference, and in two attempts were not made to elicit such information.

I. INFECTION WITH CLOSTRIDIUM WELCHII.

There were 20 post-abortal cases conclusively or presumptively infected with this organism. They were divisible into four main groups:

- A.—Cases with marked jaundice and gross blood destruction.
- B.—Cases with jaundice, but lacking serological or urinary evidence of blood destruction.
- C.—Metastatic gas gangrene.
- D.—Miscellaneous and presumptive cases.

A. *Cases with marked jaundice and gross blood destruction.*

This is the largest group, and comprises the classical post-abortal gas gangrene to which the majority of lethal cases in the literature belong.

Hand in hand with ease in diagnosis runs therapeutic hopelessness. Failing immediate and urgent treatment, and almost invariably despite it, death ensues within a few hours or a few days. The established picture is unmistakable and unforgettable. A typical course is as follows:

Within two or three days of an abortion showing some of the general and local signs of sepsis, a woman develops jaundice

which becomes most marked on the face and body, least marked on the legs. This rapidly deepens, and is soon accompanied by slight dusky cyanosis of the fingers and toes. Within a few hours the skin has darkened almost to a bronze or mahogany, and the conjunctivae may be of the shade of chocolate.

Catheterization removes an ounce or two, or possibly but a few cubic centimetres, of a strongly acid, portwine-coloured urine containing haemoglobin, methaemoglobin and fragments of destroyed red cells; the blood-serum is the colour of burgundy with the same products of haemolysis.

The pulse-rate, which was 110 to 120 on admission, has gradually quickened to 140 and the pulse is of low tension; it presently becomes soft and running, heralding complete peripheral circulatory failure with ice-cold, sweating extremities, deepening cyanosis, increasing thirst, rapid shallow respirations and terminal restlessness. The patient, who is now completely prostrate, may appear physically more dead than alive, but a usual and striking feature is her maintenance of a clear consciousness to the last.

This syndrome, which can develop with amazing speed, I believe to be pathognomonic of infection by *cl. Welchii* or an anaerobic bacillus of its group. I have not yet seen it simulated even remotely by infection due to other organisms or by chemical poisoning. Its course may be retarded or modified, but only exceptionally arrested by therapy.

There were nine patients in this group, of which eight were fatal, three within 12 hours of reaching hospital. Ante-mortem blood-cultures were positive for *cl. Welchii* in five, for *cl. Welchii* and aerobic streptococcus in two, for staphylococcus albus in one, and for aerobic streptococcus in one. In this last, a fatal physometra with generalized blood infection clinically, *cl. Welchii* and streptococcus were recovered from the urine, peritoneal cavity and uterine contents.

The staphylococcus albus was twice grown from the blood and once from the uterus of the sole survivor, but *cl. Welchii* predominated in the uterine culture, the strain being strongly pathogenic to white mice and guinea-pigs.

Certain clinical features require elaboration.

1. *Jaundice*. In all patients this was early in onset and rapid in development, but in only four did it attain the deep bronzing with underlying cyanosis which is the result of combined gross red-cell destruction, marked methaemoglobinaemia and peripheral circulatory failure.

In the remainder it was fairly marked in one and deep in four, of whom one recovered.

In four women, in whom it developed *in situ*, the exact time of onset of the jaundice could be determined. In three it appeared on an average of 40 hours after the first symptom of illness and just under six and a half days before death; each of these had received urgent local, general and serological therapy from the start. The fourth and most fulminating example was admitted to hospital 12 hours after interference with a Higginson's syringe; she developed bronze icterus under observation, and died untreated 3 hours 40 minutes after reaching the ward.

2. *Cyanosis*. This was absent in the one woman who recovered. In the remainder it followed jaundice, and ranged from a slight duskiness of the extremities to the deeper and more generalized shade seen in the bronzed cases and in the very terminal stages of all.

3. *Haemoglobinaemia*. The blood-serum in every case was burgundy-coloured, and biochemical and spectroscopic examination revealed the presence of free oxyhaemoglobin and methaemoglobin.

The severe and extremely rapid anaemia accompanying haemolysis tends to be masked by the developing ictero-cyanosis, and red-cell counts alone can accurately measure it. These were not performed in our series, but Fraenkel and Lehmann have noted the red-cell count fall by over two million in six hours, and in one case reported by Sleeman the reading on admission was only 680,000.

In three of our group haemoglobin estimations (Tallquist) shortly after admission read 50 per cent, 50 per cent and 35 per cent respectively, but with large quantities of free blood-pigment in the serum these give an inadequate impression of the true oxygen-carrying depletion of the blood.

Two women showed evidence of capillary damage and haemolysis at the sites of hypodermic injection in the arms, developing blue-black areas of subcutaneous bruising from two to four centimetres in diameter. In another a blue-black, ice-cold area of multiple petechiae, simulating gangrene, appeared at the tip of the nose; this sign has been reported by Sleeman.

4. *Haemoglobinuria*. The urine was invariably scanty, strongly acid and contained albumin. In seven cases it was of a port-wine colour, spectroscopic and biochemical examination

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revealing oxyhaemoglobin and methaemoglobin. Fragments of haemolysed red cells, but only occasionally an intact red corpuscle, were seen microscopically.

In one fatal case urine was not obtained for examination, while in another it contained urobilin but not any blood pigment. In the sole case of survival, evidence of haemolysis had disappeared from the urine and serum within 36 hours.

5. *Renal failure.* A constant feature of the fatal cases was renal inhibition, and in those surviving some days it contributed to the fatal issue.

In three patients, of whom complete urinary charts were available, the total excretion was 34.5 ounces for seven and a half days, 11 ounces for five days, and 14 ounces for six days, respectively.

In the first of these, who developed terminal general peritonitis, the combined lesions raised the blood-urea from 140 to 396 milligrams per 100 cubic centimetres in five days. In the last, who became clinically uraemic, the reading reached 185 milligrams per 100 cubic centimetres.

In two women the blood-urea on admission read 79 and 151 respectively; the former died within 12 hours.

6. *Placental tissue.* The condition of three patients was so desperate on admission that uterine interference was not justifiable. But placental tissue from the remainder was examined macroscopically after curettage in four, after removal of a portion from the cervical canal in one, and following hysterectomy in one.

In all it showed certain features characteristic of invasion by organisms of the *cl. Welchii* class.

This typical placental tissue is of a dirty greyish or pinkish-grey colour, soft, friable and spongy in consistence, and shows various degrees of liquefaction so that more or less it tends to adhere in paste-like fashion to uterine wall and curette. It exudes a slightly pungent, mousy, cadaveric odour, which in later stages may become foul. Its removal by blunt curettage is usually attended by little haemorrhage, but after a short interval uterine amyotonia is liable to express itself in a steady oozing.

In only one instance, after hysterectomy, was gas discovered in the placenta and decidua; but exhaustive examination for it had not been made in all previous specimens.

In five cases intra-uterine cultures were taken; all were returned positive for *cl. Welchii*.

7. *Ante-mortem gas.* In the case following hysterectomy gas was demonstrable in the uterine musculature. In no other member of the group was it detected in the tissues during life.

8. *Pulse-rate and tension.* The pulse-rate on admission ranged from 80 to 160, with an average of 115.

The pulse tension was almost invariably low. The highest recorded systolic blood-pressure was 120; this patient died in 12 hours. In five other cases in which it was estimated, at or within a few hours of admission, the highest systolic reading was 95, and the average 71; the diastolic pressure, when measurable, was below 54. In one case the pulse was both impalpable and inaudible on admission (No. 6).

9. *Temperature.* This did not present any particular feature. The average at admission was 100°F; the average maximal in hospital, 101.2°F.

10. *Tongue.* This was dry and furred on admission in six patients, moist and furred in three.

11. *Course of the disease.* In the fatal cases the average duration of life was just over five days from the first symptom of illness, and three days 16 hours after admission to hospital.

The longest survival period was nine and a half days from the first symptom of illness, and seven and a half days after admission; the shortest, 16 hours after interference and three hours and forty minutes after admission.

In six women who had attempted abortion with a Higginson's syringe, death occurred on an average six days later.

CASE HISTORIES.

Table II is a summary of the salient features of the whole post-abortal and puerperal series.

Group A consists of cases 1 to 9, of whom number 4 alone survived. Further details of cases 5 and 9, representative examples who received entirely dissimilar local treatment, will here be given.

CASE NO. 5.

D. S., aged 23 years, 3-para, whose last menstrual period had been two months previously, was admitted on 12th November, 1934, two days after attempted abortion with a wax taper and Higginson's syringe. This had been followed by shivers, vomiting, abdominal pains, slight vaginal haemorrhage, and, more recently, diarrhoea.

On admission the patient was sallow, with moist furred tongue, pulse-rate of 90, temperature 103°F., and slight lower abdominal tenderness. The

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retroverted uterus was the size of an eight weeks' pregnancy, the os was open and offensive blood-clot lay in the vagina; the fornices were clear.

Six hours later she was slightly jaundiced, pulse-rate 108. The blood was cultured and 40,000 international units of *cl. Welchii* antitoxin were given intramuscularly.

Fourteen hours after admission her whole body was bronzed, her conjunctivae the colour of chocolate, pulse-rate 126. Blue-black ecchymoses two centimetres in diameter marked the sites of hypodermic pituitrin injections in the arms. Four ounces of strongly acid, port-wine-coloured urine was withdrawn by a catheter.

Immediate blunt curettage under ethylene and oxygen anaesthesia removed soft necrotic placental tissue of 'typical' variety; glycerine gauze was left in the uterus for 12 hours.

Curettage was at once followed by the intravenous administration of 60,000 units of *cl. Welchii* antitoxin with 30 ounces of 10 per cent glucose in normal saline, and followed in two hours by sub-mammary one per cent sodium bicarbonate solution, 40 ounces.

November 11th, 1934.—The bronze colour was actually deeper, with definite cyanosis of the fingers, the systolic blood-pressure was 76, haemoglobin 50 per cent, pulse-rate 116 to 120.

Two ounces of urine were excreted during the day; this and the blood-serum were a burgundy colour with oxyhaemoglobin and methaemoglobin. An intra-uterine glycerine injection was accompanied by some small explosions of gas bubbles from the uterine cavity.

Sodium bicarbonate was included in all the drinks administered, two drachms to 20 ounces, and sodium citrate grains 60 to 120 was prescribed two hourly.

Further therapy included 40,000 units of *cl. Welchii* antitoxin, 15 ounces of glucose-saline and two drachms of sodium bicarbonate, intravenously; blood transfusion, 22 ounces; and two intramuscular injections, separated by 12 hours, of 40,000 units of *cl. Welchii* antitoxin.

November 14th, 1934.—Patient developed rapid, grunting respirations with signs of congestion at bases of both lungs, necessitating the hourly use of a carbogen tent, and injections of atropine and coramine alternately every two hours.

Bronzing unchanged, cyanosis of fingers and lips marked. Pulse-rate 132, blood-pressure 94/50, haemoglobin 65 per cent.

Urine now alkaline, colour the same, 7.5 ounces excreted. Blood-urea 140 milligrams per 100 cubic centimetres.

Abdomen distended and slightly tender; needling in three places removed only a drop of watery fluid containing a few polymorphs but organisms were absent. Right labium majus oedematous; brownish vaginal discharge, slightly necrotic-smelling.

80,000 units of *cl. Welchii* antitoxin given intramuscularly in two injections separated by 13 hours; 40 ounces of submammary saline administered.

November 15th, 1934.—Clinically improved although colour unchanged.

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Nails cyanosed and slate-grey, fingers dusky bronze, palms have violet pink erythema.

Pulse good volume 112, blood-pressure 104/76, haemoglobin 65 per cent. Serum shows less blood pigment, but urine unchanged and only three ounces excreted.

Pulmonary signs now largely confined to right lower lobe. Back oedematous to mid-dorsal level, with little demonstrable pitting. Vulva oedematous and unhealthy looking; lochia smell necrotic. Uterus the size of a six weeks' pregnancy, not tender, os almost closed; intra-uterine glycerine expelled greyish, necrotic-smelling discharge.

Therapy included four-hourly rectal salines containing brandy; bi-daily submammary salines 40 ounces; one intramuscular injection of 10 cubic centimetres of 10 per cent calcium gluconate; and 80,000 units of *cl. Welchii* antitoxin intramuscularly in two injections separated by 14 hours.

November 16th, 1934.—Oedema slowly increasing; all extremities, except left forearm, slightly involved. Abdomen uniformly distended, with slight general tenderness; no shifting dullness. Infrequent twitching of face and hands.

Congestion both lung bases; cough occasionally expels a grey plaque of sputum.

Urine browner, but still contains blood pigment; excretion four ounces; *cl. Welchii* recovered on culture. Blood-urea 252 milligrams per 100 cubic centimetres.

Vulva very oedematous, exudes necrotic odour. Intra-uterine injection of glycerine and acriflavine.

Further therapy included blood transfusion 20 ounces, and intramuscular antitoxin 40,000 units.

November 17th, 1934.—Colour mahogany, cyanosis absent. Increased oedema and severe abdominal pains. Very tender, distended, ascitic type of abdomen. Tongue dry, with prominent red papillae; breath renal. Pulse 120, blood-pressure 112/64, haemoglobin 85 per cent. Methaemoglobin still in serum; urine almost beef-tea colour, necrotic-smelling, 5 ounces excreted.

100 cubic centimetres of 50 per cent glucose injected intravenously, and two cubic centimetres of salyrgan intramuscularly.

Abdominal needling revealed straw-coloured fluid; vast numbers of organisms with the morphology of *cl. Welchii* and streptococci were in the centrifuged deposit.

Immediate bilateral iliac, muscle-splitting laparotomy under local anaesthesia was performed with the patient in bed; considerable quantities of brown-yellow, slightly faecal-smelling fluid containing lymph escaped; cultures later grew streptococci and *cl. Welchii*. 80,000 units of *cl. Welchii* antitoxin were injected by catheter to the upper abdominal areas affected, and tubes drained Douglas's pouch, the lower abdomen and both loins. Post-operatively 40,000 units of antitoxin were injected intramuscularly. Pulse-rate 100 in evening.

November 18th, 1934.—Occasional vomiting. Tongue dry, renal type. Pulse-rate 116, blood-pressure 92/64.

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Abdomen draining freely; six ounces of urine excreted, dark brown, offensively necrotic-smelling, and forming one-sixth deposit on standing.

Fairly frequent twitching and bouts of air hunger. 100 cubic centimetres of 50 per cent glucose given intravenously.

Increasing vomiting, becoming of small intestine type. Stomach wash-out and intravenous injection of 21 ounces of three per cent saline in 10 per cent glucose given; little improvement. High ileostomy performed under local anaesthesia without moving patient. Ileal lavage given and 20 ounces of saline left behind. 40,000 units of antitoxin given intramuscularly.

November 19th, 1934.—Condition worse. Semi-conscious and stuporose. Blood-cultures of 14th November negative. Methaemoglobin in serum and urine; three ounces of latter excreted.

Saline 20 ounces with one ounce of brandy given hourly by ileal tube and four-hourly by rectum.

Colour a mixture of yellow, mahogany and an underlying greyish hue. Further vomiting was shortly followed by death.

The total serum dosage over eight days had been 620,000 units; 100,000 intravenously, 440,000 intramuscularly and 80,000 intraperitoneally. The total urinary output had been 34.5 ounces.

At autopsy, 19½ hours after death, the abdomen was distended, the vulva grossly oedematous and the lower extremities moderately so; gas was not palpable in the external tissues.

The heart was small and firm, with a little turbid fluid in the pericardial sac, and recent haemorrhages on the basal pericardium. The lungs were congested and oedematous, with a little turbid fluid in each pleural cavity and small haemorrhages on the visceral pleura. The air passages were congested. Both liver and spleen were dark red, friable and soft. The kidneys were enlarged, swollen and friable, the capsules peeling easily, the cortex red, the medulla pigmented, almost black, the general markings blurred. The bladder was oedematous and contained blood-stained urine; its mucosa was red and necrotic. The pancreas and suprarenals were congested and soft. The peritoneal cavity contained purulent fluid with fibrin and some gas, numerous soft adhesions uniting coils of small intestine.

The uterus was enlarged, dark and soft, with an area of necrosis at the fundus. The uterus had not been perforated, neither was there any identifiable wound therein. Its cavity was four inches long and contained foul-smelling necrotic debris. The cervix was necrotic and open.

Microscopy.

Heart. Considerable toxic spoiling and pigmentation. Muscle outlines and striations not well marked, staining poor. Very occasional Gram-positive bacillus seen.

Spleen. Toxic spoiling of varying intensity throughout; congestion, marked pigmentation and some blood extravasation. Scattered Gram-positive bacilli, some in vessels, others in pulp.

Liver. Universal extreme degeneration, with loss of gross lobular outline

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and cell structure. Extensive disappearance of cellular tissue, leaving in parts only a reticular framework. Greatest damage in region of central veins.

Normal parenchyma was not visible; cell outlines and orientation lost, cell substance granular and poorly staining, nuclei almost entirely non-visible. Pigmentation and congestion marked.

Large aggregations of Gram-positive bacilli and Gram-positive cocci in portal veins and between hepatic cells; degeneration more marked about these aggregations. Definite gas bubbles could not be seen.

Kidney. Enormous general congestion, with marked pigmentation and blood extravasation through parenchyma.

Extensive toxic spoiling and degeneration, chiefly of the tubular system. Cells granular, swollen, poorly staining, and in places showing various degrees of disintegration and disorientation. Tubules choked with cellular *débris* and red blood-cells.

Glomeruli far less affected than tubules, but show separation of fibres by non-staining material, and increased lobulation of tufts.

Scattered Gram-positive bacilli and occasional Gram-positive cocci through kidney substance.

Uterus. Cavity lined with pyogenic membrane containing degenerated blood, large clusters of Gram-positive bacilli, streptococci and Gram-positive cocci. Polymorphonuclear infiltration where this abuts on muscle.

The muscle next to the uterine cavity was completely degenerated, with loss of outline, structureless appearance and poor staining; proceeding inwards fatty degeneration, cloudy swelling, then almost normal muscle was encountered. In stained sections a large amount of muscle appeared quite normal, and most of it showed slight toxic spoiling only. Fibrin in vessels.

Large groups of Gram-positive bacilli scattered through muscle, unaccompanied by other organisms or by cellular reaction, and commonly congregated at the edges of spaces. Fewer bacilli in vessels.

This case demonstrates the complete failure of thorough serological and general therapy in the absence of removal of the primary focus.

CASE No. 9.

J. P., aged 26 years, 1-para, whose last menstrual period had been nine weeks before, was admitted on 2nd February, 1935, with the provisional diagnosis of inevitable abortion. Eight hours previously she had syringed soap and water into the uterus, and two hours later had experienced slight vaginal haemorrhage, severe lower abdominal pain, shivers and sweats.

On admission her tongue was dry and furred, pulse-rate 80, temperature 97°F., uterus the size of a 20 weeks' gestation with membranes bulging through the os, blood-clot in the vagina.

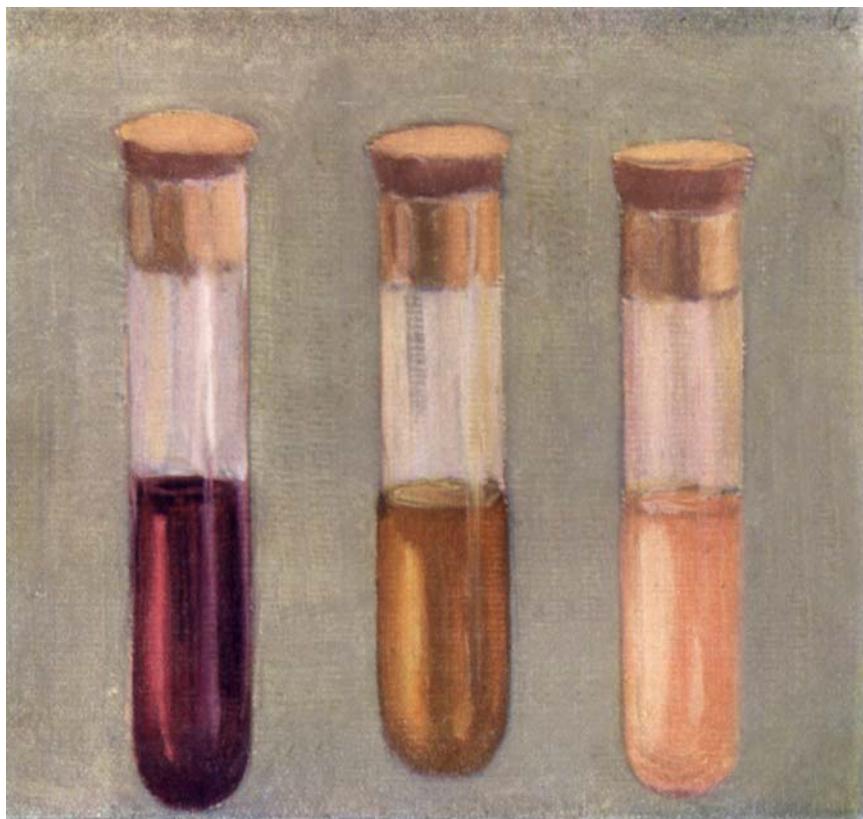
February, 3rd, 1935.—Twelve hours after admission the patient aborted; the foetus was not kept.

Three hours after abortion the skin and conjunctivae were definitely jaundiced, tongue moist and completely furred, pulse-rate 116 to 120. The lochia were red and not noticeably offensive.

Four hours after abortion jaundice was deeper, pulse-rate 120. There was



Urine of case of "classical" post-abortal
gas gangrene.



Serum of case of
"classical" post-abortal
gas gangrene.

Same, after 72 hours'
treatment.

Normal serum, as
control.

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tenderness low down in the iliac fossae; the uterus was the size of a 12 to 14 weeks' pregnancy, tense and very tender, with a feeling of bulging at its left cornu and also further out in the right broad ligament; crepitus was absent. In view of the rapid development of the syndrome, and the local signs, physometra was diagnosed and hysterectomy decided on.

60,000 units of *cl. Welchii* antitoxin were now given intravenously with 20 ounces of 10 per cent glucose in normal saline, and 20 units of insulin. The pulse-rate, now 136, steadily increased until just before operation it was 150.

Nine and a half hours after abortion hysterectomy was rapidly performed under ether anaesthesia. The vagina was first cleansed with ether soap, water and mercuric biniode solution, then dried and painted with five per cent iodine. After inserting a five per cent iodine gauze pack into the uterus, the cervix was closed with a number three catgut suture. The vagina was then repainted, and packed with gauze soaked in acriflavine solution 1 in 1000.

Laparotomy revealed three to four ounces of brown blood-stained free fluid in peritoneal cavity; smear was negative, but culture later grew *cl. Welchii*, streptococci and Gram-negative bacilli. The uterus, the size of a 14 weeks' pregnancy, was of even contour and dark brown to grey-red in colour, darkening to red-black at the right end of the fundus. The right broad ligament, Fallopian tube and ovary were very swollen and almost blue-black, as though thrombosed; crepitus was absent. The uterine vessels were large, blue and distended to the calibre of the index finger; the uterus itself, and to a less extent the contiguous pelvic tissues, appeared soft, boggy and friable.

The right broad ligament with adnexa, and the whole uterus, except a very small outer shell of dilated cervical lip, were removed. One vaginal and one lower abdominal drainage tube were inserted. During the operation a blood transfusion, 20 ounces, was given.

The removed uterus was grossly infected; grey-red necrotic placental *débris* filled its cavity and was not sharply divisible from the underlying necrosing muscle. The muscle was nowhere of a normal pink or firm to touch, and became progressively involved as the endometrium was approached, the affected areas varying from dull grey to blue-black. The vessels in the uterine wall were greatly distended, and bulged with black thrombi. Fine crepitus was palpable through the organ, and fine bubbles could be expressed from the cut muscle surface, chiefly next the endometrium. Broth culture from the uterine cavity later grew *cl. Welchii*, streptococci and Gram-negative bacilli. A portion of the uterine muscle later subjected to microscopy revealed degeneration but no organisms. The whole right broad ligament was swollen and blackened due to contained haemorrhage and its vessels were distended with blood-clot; the Fallopian tube appeared normal; and the ovary was enlarged, swollen, discoloured with surface bluing, and on section showed scattered haemorrhages the size of a pin's head through its stroma.

After the operation the patient's condition was satisfactory, pulse-rate 144. Sodium bicarbonate two drachms to the pint was included in all drinks, and 60 grains of sodium citrate given two hourly. 30,000 units of *cl. Welchii* antitoxin were injected intramuscularly.

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February 4th, 1935.—Condition excellent. Pulse-rate 90, satisfactory volume. Blood-pressure 95/54. Jaundice quite gone.

Later in the day some abdominal pains and pulse-rate 120. Two ounces of very acid, grey-brown urine obtained by catheter.

Removal of vaginal drainage tube released two small explosions of gas; smears from tube revealed apparent *cl. Welchii*, streptococci and Gram-negative bacilli.

Two injections of 40,000 units of *cl. Welchii* antitoxin were given intramuscularly, separated by 12 hours, the latter being accompanied by 60 cubic centimetres of puerperal streptococcal antiserum.

February 5th, 1935.—Pulse-rate 132, blood-pressure 114/68, temperature 99.8°F. Pale, haemoglobin 35 per cent.

Abdominal drainage tube shortened; no discharge. Three ounces of brownish, blood-stained urine containing methaemoglobin excreted; albumin one-sixth deposit on standing. Blood urea 136 milligrams per 100 cubic centimetres.

Patient drinking well. Intravenous 10 per cent glucose saline, 30 ounces, containing 8 grams of sodium bicarbonate, given. Pulse-rate later fell to 92.

Two intramuscular injections of 40,000 units of *cl. Welchii* antitoxin given, separated by five hours.

February 6th, 1935.—No change. Pulse-rate 100, blood-pressure 124/70, temperature 98.4°F. Taking copious fluids. Urine culture of 3rd February positive for *cl. Welchii*, streptococcus and Gram-negative bacillus. Present excretion 1.5 ounces of alkaline, dirty brown, slightly offensive urine; culture again revealed the same organisms.

Blood transfusion, 23 ounces, given. Diuretin grains 10, and sodium sulphate grains 120, prescribed thrice daily. Abdominal wound clean and healing; tube removed. Drowsy later in day; epistaxis during night.

February 7th, 1935.—Pulse-rate 108, blood-pressure 128/80, temperature 98°F. Renal failure more complete. Nausea and hiccough. Only 7 drachms of slightly acid urine excreted in 24 hours. Patient pale; no demonstrable oedema. Haemoglobin 55 per cent. Blood-serum a normal straw colour. Blood-urea 185 milligrams per 100 cubic centimetres.

Magnesium sulphate 4 drachms given, and the following solutions injected intravenously: 100 cubic centimetres of 50 per cent glucose, 20 cubic centimetres of 10 per cent calcium gluconate, and 30 ounces of 10 per cent glucose in normal saline.

Blood-culture of 3rd February returned positive for streptococcus. Later in the day the patient was somnolent and lethargic. Slight cough, with râles at right base.

February 8th, 1935.—Brighter. Pulse-rate 104, temperature 97.2°F. Tongue fairly moist. Patient has rather swollen appearance without demonstrable oedema. Signs of congestion at both lung bases. Taking fluids well. Urine excreted, 5.5 ounces, rather offensive and dirty brown, with deposit and albumin; no casts. Slight, non-offensive, red-serous vaginal discharge. Occasional twitchings of face and hands. Intravenous 10 per cent glucose-

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saline, 40 ounces, with 20 cubic centimetres of 10 per cent calcium gluconate, given.

Two hours after intravenous therapy violent twitching of the arms occurred for a few minutes; 30 minutes later patient suddenly collapsed and died.

At no time had the temperature exceeded 99.8°F., and for the three days preceding death it had ranged from 97.2°F. to 98.6°F., with a pulse-rate from 100 to 110. The total urinary excretion over six days had been 14 ounces, and the total dosage of *cl. Welchii* antitoxin 330,000 units, of which 60,000 had been given intravenously.

At autopsy the external tissues were free of gas. The heart was relaxed and flabby, and its cavities full of frothy blood. The lungs were engorged with frothy fluid, and the bases somewhat collapsed. The hepatic substance was friable and the vessels contained frothy blood; the spleen was crepitant and swollen. The kidneys were large, swollen and friable, with broad, smooth cortices and readily peeling capsules. The operation area was clean and healing, and there was no peritonitis.

This case demonstrates the hopelessness of established renal failure, and also admits of three criticisms of treatment, viz:

1. The early cessation of serum therapy despite a pathological urine.
2. The leaving behind of a small portion of cervical lip; this might well have been the focus which maintained fatal infection.
3. Delayed and insufficient alkali therapy (*see under Treatment*).

B.—Cases with Jaundice, but lacking Serological or Urinary evidence of Blood destruction.

The five members of this group reveal a range in clinical type and severity. The one feature common to all was jaundice, but in none did it approach the bronze hue seen in the previous group. In one patient it was slight, in one definite, and in three fairly marked. In four it developed, and with considerable speed, only after admission to hospital. Disappearance followed local and specific therapy in every instance.

In three (Nos. 11, 12, 14) jaundice was the sole symptom suggesting the diagnosis, the underlying clinical picture being that of fairly severe post-abortal sepsis such as is not uncommonly seen with streptococcal infection. These three cases were all examples of multiple blood invasion, and in only one (No. 14) were measures instituted against *cl. Welchii* before the diagnosis had been established by recovery of the organisms from the blood.

The remaining two cases were those of pure *cl. Welchii* infections, in which clinical recognition was early and therapy immediate. Before bacteriological confirmation had been obtained each patient received blood transfusion, 120,000 inter-

national units of *cl. Welchii* antitoxin by intravenous and intramuscular routes, and uterine curettage under gas-and-oxygen anaesthesia.

In one patient (No. 13), diagnosis rested upon a combination of jaundice which was early in onset and rapid in development, with a corresponding quickening of the pulse-rate. The later removal of soft, necrotic placental tissue of the typical variety was confirmatory.

The other case (No. 10) was a classical example of fulminating *cl. Welchii* septicaemia in a woman who had been in hospital for three days with a septic inevitable abortion. Following insidious collapse she rapidly developed peripheral circulatory failure with running pulse, subnormal temperature, progressive jaundice, cyanosis and dyspnoea, and within three hours a fatal issue appeared imminent. Her subsequent recovery was dramatic, and her general symptomatology provides a stepping stone between the cases of group A and those next to be described (group C).

The one fatality in the group (No. 14) was a patient with a dual blood infection who succumbed to streptococcal pneumonia 12 days after she had apparently completely overcome her *cl. Welchii* invasion.

Only those of Nos. 10 and 14 will here be recounted.

CASE NO. 10.

E. R., aged 30 years, 7-para, whose last period had been 18 weeks previously, was admitted on 29th November, 1933, with the diagnosis of septic inevitable abortion. She had attempted to induce abortion with Higginson's syringe and soapy water two days before.

On admission she was slightly flushed, with moist, furred tongue, pulse-rate 130, temperature 101.4°F. The uterus was the size of a 16 weeks' pregnancy, not tender; the os was open and the vagina contained bloody discharge. Next morning the pulse-rate was 96, temperature normal.

During the third night after admission (1st December) the patient aborted a foetus; this was not kept. A rigor and temporary tachycardia of 132 followed the abortion; the pulse-rate then settled to 104.

December 2nd, 1933.—Eight hours after abortion patient's condition was noted to go off suddenly; she became mentally drowsy, with dusky colour and rapid pulse-rate. Thirty minutes later the resident doctor noticed she was slightly jaundiced, took a blood-culture and gave 10,000 units of *cl. Welchii* antitoxin intramuscularly.

One hour later the woman was desperately ill, with clouded consciousness, definite jaundice of skin and conjunctivae, and running pulse of 152. Her temperature was 97.2°F.; her extremities ice-cold and clammy; lips, ears and

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finger-tips cyanosed; tongue dry and furred; face grey; forehead sweating; respirations rapid and shallow. There was not any abdominal rigidity, but tenderness over uterus and liver. Pelvic examination was not made.

40,000 units of *cl. Welchii* antitoxin in 30 ounces of 10 per cent glucose-in normal saline were immediately administered intravenously, and 20,000 units injected intramuscularly. Fluids containing glucose were given every 15 minutes, and half an ounce of brandy four-hourly.

Seven hours after intravenous therapy the volume of the pulse was slightly better, rate 130; but the general condition was still poor and jaundice marked. Blood transfusion, 22 ounces was given.

December 3rd, 1933.—General condition much improved. Mentally clearer, jaundice less, pulse fair volume, 128.

30,000 units *cl. Welchii* antitoxin in 20 ounces of 10 per cent glucose-saline given intravenously, and 20,000 units intramuscularly. Blood-culture of 2nd December positive for *cl. Welchii*. Blunt curettage performed under nitrous oxide and oxygen anaesthesia; much typical necrotic and liquefying placental tissue removed; glycerin gauze left in uterus 12 hours. Rectal 10 per cent glucose in saline, 20 ounces, with brandy one ounce, given after curettage.

December 4th, 1933.—Condition very satisfactory. Pulse good volume, rate 112, jaundice gone. Urine clear.

20,000 units *cl. Welchii* antitoxin in 20 ounces of 10 per cent glucose-saline given intravenously, and 20,000 units intramuscularly.

December 5th, 1933.—Pulse-rate 96, temperature, 98.8°F. Intravenous and intramuscular serum, 20,000 units of each, administered as before. Intra-uterine injection of glycerine given; return almost clear. Intramuscular serum alone was continued daily for the next seven days in quantities ranging from 20,000 to 32,000 units. Blood culture taken on the fifth day after curettage was negative. Convalescence was complicated by two bouts of serum dermatitis and adenitis, and the patient was discharged cured on 5th January, 1934.

The total serum dosage was 382,000 units, of which 110,000 had been given intravenously.

In the light of more recent experience, curettage and pelvic examination in this case would have been performed as early as possible after the initial intravenous therapy.

CASE No. 14.

V. R., aged 33 years, 2-para, was admitted on 8th July, 1934, with a septic incomplete abortion; interference denied.

She was ill, with pulse-rate 124, temperature 102.2°F., tender uterus the size of a four weeks' pregnancy, os slightly open, urine clear.

On account of slight jaundice of skin and conjunctivae her uterus was immediately curetted, and she was given 40,000 units of *cl. Welchii* antitoxin intravenously and 30,000 units intramuscularly. Blood-culture preceded curettage.

On 9th July, 1934, jaundice was deeper, and 50,000 units of serum were injected intravenously and 40,000 units intramuscularly; in a further six

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hours, owing to still deeper jaundice, another 40,000 units of antitoxin with 20 cubic centimetres of 10 per cent calcium gluconate, were given intravenously, and 30,000 units intramuscularly. All intravenous therapy was accompanied by 20 to 30 ounces of 10 per cent glucose-saline.

July 10th, 1934.—*Cl. Welchii* present in culture from intra-uterine gauze.

July 11th, 1934.—Pulse-rate 80, temperature 98°F. Jaundice less. Blood culture of 9th July positive for *cl. Welchii* and anaerobic streptococci. 30,000 units of *cl. Welchii* antitoxin given intramuscularly.

July 12th, 1934.—Acute onset of severe abdominal pains and development of signs of general peritonitis. Immediate laparotomy under gas-and-oxygen anaesthesia, tubes inserted to drain both loins, both iliac fossae and the pouch of Douglas. Much foul, dark, semi-purulent fluid released; culture later grew aerobic streptococci. The operation was followed by 30,000 units of *cl. Welchii* antitoxin in 30 ounces of 10 per cent glucose-saline intravenously, and 40,000 units intramuscularly.

July 13th, 1934.—Blood transfusion, 20 ounces, with 10 ounces of glucose-saline given.

July 14th, 1934.—Condition very good; free abdominal drainage.

Improvement continued, the jaundice disappeared, and for the next three days the patient was nonfebrile, with pulse-rate from 68 to 92. The drainage tubes were removed.

Temperature then began to ascend slightly, and on 20th July the patient developed pain and clinical signs in the left side of the chest. Needling on 23rd July did not reveal anything, but on 26th July she had extensive signs in both lungs, and was coughing up the foul sputum of pulmonary abscess. Pyrexia persisted; local signs, distressed breathing and general toxæmia steadily increasing, the patient died on 28th July.

At autopsy, 39 hours later, there was extensive consolidation of the right lung: the left lung was collapsed and coated with lymph, and purulent fluid was in the pleural cavity. The heart, liver, spleen and kidneys were toxic; there was not any gas in the external tissues, solid viscera or blood. There were adhesions and a small amount of gas between the small intestines. The uterus had lymph on its posterior surface, and its cavity was three inches long and fairly clean.

C.—Metastatic Gas Gangrene.

There were two examples of this rare type. In each the course was distressing and catastrophic, and recalled the fulminating surgical gas gangrene of war-time.

In one instance four days after incomplete abortion, and in the other 32 hours after curettage for that condition, the patient developed deep skeletal muscle pain which soon became excruciating and was accompanied by increasing restlessness and an overwhelming toxæmia. In swift succession there followed collapse, peripheral circulatory failure, and death.

One woman survived 10 hours and the other four hours after

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the onset of muscle pain. Jaundice, haemoglobinaemia and haemoglobinuria were not at any time present.

This desperate syndrome, of apparently metastatic origin, would appear to be chiefly due, as in surgical gas gangrene, to the effects of the *cl. Welchii* myotoxin (cytolysin). In marked contrast is the classical picture already described (group A), in which the signs are predominantly dictated by the bacterial haemotoxin (haemolysin).

In the first case encountered the true cause was not suspected during life, but its dramatic features left so deep an impression that 13 months later the second case was provisionally and correctly diagnosed upon hearing the history, and before the patient had been seen.

In the second example gas was detected in the tissues during life, deep in the muscles of the left buttock.

CASE No. 15.

S. C. aged 22 years, 4-para, was admitted on 8th April, 1933, with an incomplete abortion for which her uterus was curetted 20 hours later. At that time her pulse-rate was 80 and her temperature had not exceeded 98.6°F. A few hours after curettage she had one rigor, and complained of pain in the chest; examination was negative. Pulse-rate was then 126, temperature 103.5°F. Twenty-four hours after curettage patient felt quite well, with pulse-rate 104, temperature 102.8°F. Thirty-two hours after curettage patient complained of cramp-like pains in both thighs; aspirin mixture was given. One hour later the pains were more severe and the woman looked pale and very ill, with hot, dryish tongue, and rather poor volume pulse of 140. The pain made her extremely restless; morphia and aspirin mixture was given. Local signs were confined to a suggestion of fullness in the upper part of the right thigh, which was exquisitely tender on deep pressure equally on the anterior, medial and posterior aspects. There was neither thrombus nor palpable crepitus, neither colour nor circulatory change visible.

Three hours after the first onset of pain the patient collapsed. Her pulse was now running and almost imperceptible, temperature 96°F; her appearance was one of profound shock, with icy, clammy forehead and extremities, marked dyspnoea and cyanosis. General examination did not reveal any causative cardiac, pulmonary, abdominal or cerebral lesion; there were a few basal pulmonary crepitations only. The thigh signs were unchanged. Brandy, morphia, atropine and strychnine were given without effect. Death occurred just under four hours from the onset of pain.

At autopsy, 15 hours later, the whole body was swollen and crepitant with gas. The right thigh and to a less extent the left thigh and both upper extremities were markedly swollen, and discoloured, deeply, blue-black; while blebs containing fluid and gas were on the skin. The abdomen was greatly distended.

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The liver was friable and honeycombed with gas; the spleen swollen and crepitant with gas; the kidneys friable, with gas bubbles in the cortex; and the uterus was slightly enlarged, foul and inflamed in the cavity, and infiltrated with pus at the fundus.

CASE No. 16.

O. W., aged 38, 7-para, was admitted on 9th May, 1934, four days after a 14 weeks' abortion. She had had shivers and vomiting for the past 12 hours, and pains in the hips for two hours. On admission she looked ill and still complained of much pain in the hips; pulse-rate 108, temperature 102°F, tongue furred. A causative lesion for the pain was not discovered. Uterus size of a 10 week's pregnancy, with open os and bloody discharge. Morphia grain $\frac{1}{4}$ was injected hypodermically.

Six and a half hours after admission (4.30 a.m.) the doctor was called urgently to patient. The pain in her buttock had grown excruciating, and she had collapsed. Her appearance was one of grave shock, with great restlessness and anxious expression; she complained of agonizing pain in the left buttock. Her pulse was running and almost imperceptible, colour pale and cyanosed, extremities and forehead cold and clammy. The left buttock was swollen and non-fluctuant, not greatly tender, and blue and tense in the deep tissues; deep crepitus was elicited near the sacrum.

Morphia grain $\frac{1}{4}$ was immediately injected, blood-culture taken, 40,000 units of *cl. Welchii* serum injected intramuscularly and 60,000 units given with 20 ounces of 10 per cent glucose-saline and two cubic centimetres of coramine intravenously. Needling of the left buttock did not release any gas; but a smear revealed thousands of Gram-positive bacilli resembling *cl. Welchii*.

After a temporary improvement the pulse again became imperceptible, the patient dyspnoeic, and death occurred 10 hours after onset of the pain and eight hours after admission to hospital. *Cl. Welchii* were recovered a few hours later from the ante-mortem blood-culture.

At autopsy, 28 hours after death, the body was greatly swollen with gas, and blebs were on the skin. The cardiac muscle was friable and crepitant with gas, the liver friable and honeycombed with gas, and the spleen swollen, soft and crepitant with gas. The kidneys were large, swollen and friable, their surfaces granular, their capsules adherent. The uterus was enlarged, cavity $5\frac{1}{2}$ inches long, with placental site at fundus.

D.—*Miscellaneous and Presumptive Cases.*

This group includes one presumptive and three definite cases.

The presumptive case (No. 17) was admitted with clinical features indicating streptococcal post-abortal infection, and when seen in the terminal stages three days later was moribund, comatose and dyspnoeic, with running pulse, cold extremities and universal blotchy cyanosis. Bacteriological investigations were not performed, but at autopsy 17 hours later, in addition

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to bilateral patches of basal bronchopneumonia and small pericardial haemorrhages pointing to a streptococcal origin, the liver was friable and completely honeycombed with gas, and the placenta was soft, greyish and necrotic. A proven case with closely similar clinical features was reported by Teissier, Rivalier and Thurel.⁸

In the definite cases (Nos. 18, 19, 20) the diagnosis was entirely bacteriological in two, and largely clinical in the third, in which a collapsed appearance and increasing pulse-rate out of proportion to abortal loss of blood, suggested the possibility which intra-uterine culture confirmed. Jaundice was not present in any case in this group.

CASE NO. 19.

This is an example of endometritis and mechanical bacteraemia in which clinical signs of *cl. Welchii* infection were absent.

S.S., aged 29 years, 2-para, whose last menstrual period had been on 7th August, 1934, was admitted on 30th September, two days after having inserted a crochet needle into the uterus. This had been followed by pains in the abdomen and legs, vaginal bleeding, and latterly vomiting and shivers.

She looked ill, with dry furred tongue, pulse-rate 114, temperature 104°F., and systolic bruit at all cardiac areas. There was lower abdominal tenderness, the uterus was tender and the size of an eight weeks' pregnancy; the os was open, the right fornix tender. Blood-cultures were taken.

Twelve hours later the patient's condition was excellent, pulse-rate 90, blood-pressure 100/60, temperature 99.2°F., urine clear. But a marked growth of *cl. Welchii* and aerobic streptococci had occurred in the blood-culture media. In the absence of local or general clinical signs to suggest *cl. Welchii* infection, antitoxin therapy was deliberately withheld. Lochial culture was taken and later grew *cl. Welchii*.

After the first 24 hours in hospital the temperature only once exceeded 98.6°F., and the pulse-rate never rose above 88. The uterus was curetted six days after admission, and she was discharged four days later.

II. INFECTION WITH VIBRION SEPTIQUE.

So far as I can discover from the literature, these two cases are the first examples of post-abortal infection reported in which the organism recovered from the blood during life has been positively identified as the vibron septique.

In one suggested example recorded by Fraenkel⁹ in 1923 the description is not adequate for positive identification, and from the more recent classification by Zeissler it is probable that the organism was *cl. oedematiens*.

No classical syndrome emerges from the dissimilar features presented by the two cases.

The first patient was admitted in a desperate condition with septicaemia and general peritonitis following intra-uterine manipulations three days before. At immediate operation gas was found to be widespread through the injected abdominal muscles and escaped with dark blood from the peritoneal cavity, while the uterus was perforated and necrotic. Death occurred in 14 hours.

In the second patient, who aborted of a macerated foetus, and was moderately ill, there were not any distinctive features of diagnostic value, and the recognition was entirely bacteriological.

CASE No. 21.

E. Y., aged 28 years, 4-para, whose last menstrual period had been on 8th August, 1933, was admitted on 29th January, 1934. She had been bleeding for some weeks, and three days before, following dilatation of the cervix, her doctor had extracted a partially macerated foetal limb; he had then plugged the cervix and vagina with swabs, and had given injections of pituitrin. The plugging was removed in 24 hours. On the day prior to admission there had been severe vaginal haemorrhage, and a blood transfusion had been given.

On admission the patient was pale, desperately ill, and losing fairly freely *per vaginam*. Her tongue was dry and furred, lips covered with sordes, pulse-rate 140, temperature 100°F. Her expression was anxious, her respirations rapid. There was not any jaundice. The abdomen was distended, with generalized exquisite tenderness and rigidity. Vaginally there was free, foul, bloody discharge, the cervix was patulous, but tenderness embarrassed examination. Blood-culture was taken, and morphia grain 1/6 injected hypodermically, 25,000 units of mixed *cl. Welchii* and *vibron septique* antitoxin (Parke-Davis) and 60 cubic centimetres of puerperal streptococcal antiserum in 24 ounces of 10 per cent glucose-saline were injected intravenously, and another 15,000 units of mixed *cl. Welchii* and *vibron septique* antitoxin were given intramuscularly.

At immediate laparotomy the abdominal muscles were red, injected and crepitant with gas, and quantities of foul, dark, fluid blood and gas escaped under pressure from the peritoneal cavity. Drainage tubes were passed into lower part of the abdomen and both flanks. Owing to considerable vaginal haemorrhage not controllable from above, it was necessary to pack the uterus and vaginal vault with gauze; examination then revealed a large tear in the wall of the posterior cervix and apparently gross fundal perforation; the site of the bleeding was not clearly definable.

At operation the patient was given 40,000 units of mixed *cl. Welchii* antitoxin intramuscularly; and one hour later a blood transfusion, 15 ounces, and a further 60,000 units of mixed *cl. Welchii* antitoxin intravenously with five ounces of glucose-saline.

Three hours after the operation patient's condition had improved, but two hours later she began to vomit, and from then progress was steadily

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downwards. 40 ounces of saline with two cubic centimetres of coramine were given submammary, and three hours later 20 ounces of 10 per cent glucose-saline intravenously. Death occurred 13 hours after operation.

During life *vibron septique* was obtained in pure culture from both blood and peritoneal fluid.

At autopsy, nine hours after death, the cadaver was very pale, and the skeletal muscles generally were swollen and crepitant with gas.

The liver was pale and enlarged, the spleen soft and crepitant with gas, the kidneys pale and friable. The upper part of the abdomen was almost completely walled off from the lower by adhesions. The lower abdominal cavity was full of dark, foul blood, blood-stained fluid and degenerated tissue débris. The uterus was dark, reddish-grey and completely necrotic, with entire absence of a fundus; the side walls were disintegrating, and there was a large tear from the posterior part of the cervix into the pouch of Douglas.

In both cases of *vibron septique* infection Parke-Davis serum containing *vibron septique* antitoxin was used; in each this was initially fortuitous, as at that time it was felt wiser to use a polyvalent serum until identification was complete. Very shortly afterwards the serum in almost universal use was that from the Commonwealth serum laboratories, which does not contain any antitoxin against *vibron septique*; Burroughs and Wellcome serum, which has been employed occasionally, is also a purely *cl. Welchii* antiserum.

PUERPAL SERIES.

Infection with Cl. Welchii.

There were eight puerpal cases, six of them fatal, a mortality of 75 per cent. The average age was 30 years; the youngest being aged 20 and the oldest aged 40.

Three women were primigravida, the average parity of the remainder being just below three. Five were at term, and three within two to three weeks of term. Two mothers died undelivered, two had stillborn babies, and four were delivered of well-developed living children.

Ante-mortem blood-cultures grew *cl. Welchii* in three, and *cl. Welchii* and anaerobic streptococcus in one, the organisms in this latter case being also cultured from the peritoneal cavity. Two patients with pure *cl. Welchii* infection recovered.

Of the remainder, *cl. Welchii* was cultured from the uterus and heart blood after death in two (Nos. 27, 30), and was simply morphologically identified by smears therefrom in two (Nos. 25, 28).

Introduction of Infection.

The widespread incidence of *cl. Welchii* is well known. At the Women's Hospital pathogenic strains have been cultured from the recta of patients; from the floor-dust of the mortuary, pathological laboratory and labour ward; and from the hands of

medical attendants after washing and drying, less often after thorough scrubbing, and in one instance after thorough scrubbing and putting on wet sterilized gloves.

These circumstances, added to the common faecal contamination of the perineum which occurs after enemata and is often unavoidable during labour, provide ample opportunity for the near presence of *cl. Welchii* at times of ante-partum, intra-partum and post-partum interference.

In the present series favourable predisposing and initiating factors for the invasion of the uterine cavity by *cl. Welchii* were outstandingly evident. In only one case, that of elective Caesarean section upon a woman who had had a single vaginal examination two days before, were the cause and source of the fatal infection difficult to understand. In only two cases was there no definite evidence of intra-uterine interference.

The following table gives the apparent aetiological factors in chronological order :

CASE No.	CONTRIBUTORY AND PREDISPOSING FACTORS	FORM OF ACTIVE INTERFERENCE
23	? Vaginal douche two days before labour. Dry labour lasting four days. Macerated foetus.	Head-on-perineum forceps. Manual removal of placenta.
24	Trial labour. Membranes ruptured 26½ hours at operation.	Classical Caesarean section.
25	Placenta praevia; ante-partum haemorrhage. Baby, ? dead 12 hours before maternal death.	Vaginal plugging.
26	Albuminuric toxæmia ; stillbirth.	Induction of labour with rectal tube.
27	Eclampsia; post partum haemorrhage.	Classical Caesarean section. Post-operative intra-uterine plugging.
28	Albuminuric toxæmia. Baby ? dead three hours before maternal death.	Induction of labour with rectal tube.
29	Pyelitis of pregnancy ; disseminated sclerosis.	Mid-forceps. Episiotomy.
30	Nil ; except vaginal examination two days before.	Classical Caesarean section.

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Wrigley¹⁰ has contended that for severe maternal infection to occur the following conditions must be fulfilled in the great majority of cases:

- "(i) The organisms must be introduced into the uterus, by intra-uterine manipulations.
- (ii) The child must be dead at the time of introduction of infection, that is, at the time of the intra-uterine manipulations.
- (iii) The child must remain dead *in utero* for some hours following the initial intra-uterine manipulations.
- (iv) The original manipulations must have damaged the uterus or birth canal."

The necessity of conditions (ii) and (iii) is definitely refuted by three of our fatal cases (Nos. 24, 27, 30), in each of whom the mother was delivered of a well-developed living child. Toombs reported a similar case in 1932.

Clinical Features.

These, as in the post-abortal series, were protean. But the diagnosis in general was more difficult than in the post-abortal type, the symptomatology tending to be less arresting until almost the very end, while such contributing factors as toxæmia of pregnancy, post-operative depression and anaemia after haemorrhage helped to cloud rather than clear the issue.

The following variations in clinical type were encountered:

1. In only one patient of the series (No. 26) was jaundice present, and here its fairly rapid deepening, in association with an increasing pulse-rate, and following upon ample aetiological factors, allowed of early diagnosis and successful therapy.

2. In two cases after Caesarean section (Nos. 24, 27), in one performed over 26 hours after rupture of the membranes, and in the other succeeded by intra-uterine plugging for postpartum haemorrhage, the ante-mortem diagnosis was only presumptive, resting upon a combination of adequate aetiology with the unexplained syndrome of increasing post-operative tachycardia and pallor, the patient having a continued feeling of well-being despite growing clinical gravity, and a final onset of peripheral circulatory failure.

The third patient to follow Caesarean section (No. 30), developed a similar type of syndrome, but the absence of apparent causative factors allayed suspicion until too late.

In all three the serum therapy instituted was quite inadequate.

3. Two cases of the series (Nos. 25, 28) were fulminating,

and in their sudden onset of pain, followed by collapse and rapid course to death resembled the disastrous metastatic cases of the post-abortal series.

The pain, which was apparently of uterine origin, began in one instance 11 hours after the removal of vaginal plugging for *placenta praevia*, and death ensued in five hours; in the other it commenced nine hours after the extraction of rectal tubes which had been *in utero* for two days, and death occurred in three hours.

In the former case, the diagnosis of physometra was made only at autopsy; in the latter the condition was strongly suspected at the time of death, but actual confirmation had to await post-mortem examination.

4. In the remaining two cases of the series (Nos. 23, 29) recognition was initially or entirely bacteriological. One of these patients recovered.

CASE HISTORIES.

CASE NO. 24.

H. D., aged 25 years, primigravida, came into labour on 9th October, 1933, six days after her expected date. The presentation was a vertex in the left posterior position, and the head was not fixed at the onset of labour.

After 24 hours labour, with the maximal diameter of the head still above the pelvic brim, the membranes ruptured and the cervix was found to be fully dilated.

October 11th, 1933.—25 hours later there had not been any advance; the maternal pulse-rate was 124, temperature 100°F.; foetal heart-rate 160 to 170. Pelvic examination showed condition unchanged since the previous day. Twenty-six and a half hours after rupture of the membranes classical Caesarean section was performed. There was a definitely offensive odour on opening the uterus. The child was a living female, eight pounds five ounces in weight.

October 12th, 1933.—Pulse-rate 150, temperature 103°F. Patient feels well. Some soft abdominal distension. Blood-culture taken, and 10,000 units of *cl. Welchii* antitoxin given intramuscularly.

October 13th, 1933.—Condition much the same. Pulse-rate 146 to 152, temperature 100° to 103.4°F. Feels well, taking fluids freely. Soft abdominal distension, not tender. 10,000 units of *cl. Welchii* antitoxin given intramuscularly.

October 14th, 1933.—Still feels well but looks sick. Tongue furred. Pulse-rate has never fallen below 140, temperature now 99°F. Occasional vomiting; bowels open with enema. Blood-culture so far negative.

October 15th, 1933.—Very much worse, but is mentally clear and is definite about feeling well. Pulse running and soon imperceptible, colour greyish. Death at 11 a.m.

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Blood-culture was returned positive for *cl. Welchii* 12 hours after patient's death.

At autopsy the body was pale and very distended, with frothy black fluid exuding from mouth and nostrils. The heart was toxic and friable, the lungs showed terminal congestion. The liver, spleen and kidneys were all soft, friable and contained gas. There was some semi-purulent fluid along the uterine scar, but no general peritonitis. Smears from spleen and liver showed thousands of Gram-positive bacilli resembling *cl. Welchii*.

CASE No. 27.

I. M., aged 27 years, 2-para, was admitted two weeks before term, 27th December, 1933, with ante-partum eclampsia. She had had renal trouble with her first confinement.

One eclamptic fit had preceded admission, and one accompanied examination. Her pulse-rate was 92, blood-pressure 190/134, urine formed a solid deposit of albumin on boiling, and there was oedema of ankles, vulva and eyelids. Fluids and sedatives were immediately applied and the bowels washed out. Eighteen hours after admission, as the patient's condition had become less satisfactory and further fits had occurred, classical Caesarean section was performed. The child was a living male, weight six pounds four ounces. There was some tendency to post-partum haemorrhage, but the uterus was contracted and bloodless at conclusion of operation.

December 28th, 1933.—Four and a half hours after operation severe post-partum haemorrhage occurred, necessitating plugging of uterus with gauze soaked in glycerine; this was done with full antiseptic and aseptic precautions. Seven hours after plugging, the patient's condition was satisfactory, pulse-rate 108. She felt well but was inclined to be restless; morphia grain 1/6 was given. Ten hours after plugging her condition had become bad. Her face was pale, breathing laboured, pulse-rate 150 and poor volume; and in another five hours blood transfusion, 20 ounces, was given. Nineteen hours after plugging, the pulse-rate was 150 to 160, and quantities of brown fluid were vomited; gastric lavage with Rehfus's tube relieved the latter. One hour later the patient stated that she felt very well, but her pulse was running and she looked desperately ill. Blood-culture was now taken; 25,000 units of *cl. Welchii* antitoxin were injected with 15 ounces of 10 per cent glucose-saline intravenously, and 15,000 units of *cl. Welchii* antitoxin given intramuscularly.

December 29th, 1933.—12.30 a.m. (22½ hours after plugging), 40 ounces of saline given subpectorally.—9.30 a.m. Semi-comatose. Urinary excretion for last 24 hours, seven ounces.—12.10 p.m. 40 ounces of 10 per cent glucose-saline with 15 units of insulin, and theocin grains 10, given intravenously. Magnesium sulphate four drachms by Rehfus's tube. Blood-pressure 70/?; blood-urea 105 milligrams per 100 cubic centimetres.—2.15 p.m. Urinary excretion of last eight hours has been 10 ounces. Blood-culture still negative. The patient died.

At autopsy, 20 hours after death, the body was greatly swollen with gas, and blebs marked the skin. The heart was relaxed and contained frothy

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blood. The lungs showed basal congestion. The liver was crepitant with gas, the spleen friable and crepitant. The renal cortices were pale, the medulla streaky, the capsules tending to adhere. The uterus was clean and there was no suppuration in the wound. Cultures from uterus and heart's blood grew *cl. Welchii*.

CASE NO. 25.

R. F., aged 35, 3-para, was admitted on 21st October, 1933, two weeks before term, believed to be in beginning labour. The presentation was a vertex in the left anterior position, the head unfixated, and the cervix dilated to admit one finger. Some hours later pains ceased.

Twenty-five and twenty-six hours after admission the patient passed small clots of blood, and on the second occasion vaginal examination revealed a right posterior marginal placenta praevia; the cervix was little further dilated, the membranes intact. Twenty-seven hours after admission fairly free haemorrhage necessitated plugging of the vagina with swabs wrung tightly out of mercuric biniodide solution 1 in 4000. Ten hours later the patient was having pains, and the plugging was removed. The cervix was dilated to the size of three fingers, and on rupture of the membranes the head came fairly well down. Further haemorrhage did not occur. Four hours later marked the last occasion on which the foetal heart was heard. Nine and a half hours after removal of plugging, pains had ceased and slight vaginal haemorrhage had commenced. Half-hourly hypodermic injections of pituitrin, minims four, were begun. Pains recommenced after the fourth injection, and soon became increasingly frequent and strong. Within two hours they were so severe and frequent that uterine contractions were almost tonic, and the pulse-rate was 104. Thirteen and a half hours after removal of plugging the patient presented signs of grave shock. She was in great pain with anxious expression and marked restlessness; her pulse-rate was 120 and soft, her colour grey. The uterus was tender and very tense; vaginal examination revealed full dilatation and taking-up of the cervix, with a large foetal caput despite absence of disproportion and good engagement of the head in the pelvic brim.

There was no definite evidence of, or cause for, uterine rupture, and the patient looked more grey and toxic than a case of concealed accidental haemorrhage. Effortless and frequent vomiting presently set in.

Morphia grain $\frac{1}{4}$ hypodermically did not relieve the pain, and a further grain $\frac{1}{6}$ was given 30 minutes later. Submammary and rectal salines, the latter containing brandy, were administered.

Fourteen and a half hours after removal of the plugging, the woman was *in extremis*, with Hippocratic facies, thready pulse of 160, and peripheral circulatory failure. The abdomen was tense, and at this stage fine gas crepitus was elicited in the region of the transverse colon, but in the presence of colonic distension its significance was at the moment lost. A further injection of morphia grain $\frac{1}{6}$ was given, with little effect. The woman died 26 hours after insertion of the vaginal plugging.

At autopsy, 9 $\frac{1}{2}$ hours later, the body was greatly distended, the subcutaneous tissues filled with gas; dark frothy fluid exuded from mouth and

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nose. The cardiac muscle was toxic and friable, the cavities full of frothy blood, the coronary arteries and valves normal. The lungs showed some terminal basal congestion. The liver was honeycombed with gas; the spleen soft and crepitant with gas; the kidneys rather friable, with readily peeling capsules and fine gas bubbles in the cortex; the stomach and intestines normal. The uterus was distended, tense, and contained a full-time foetus; gas was present in bubbles beneath the peritoneal coat, in the uterine muscle, and in quantity in the cavity of the uterus. The placenta lay in the right of the lower uterine segment.

The foetus was undergoing maceration, gaseous blebs being scattered over the surface of the body. There was a large caput succedaneum, containing gas only. The child's viscera was distended with gas. Smears from the foetal blebs, and from maternal heart-blood and spleen revealed numberless Gram-positive bacilli resembling *cl. Welchii*.

CASE NO. 28.

A. S., aged 40, 7-para, was admitted on 10th September, 1934, almost at term, with albuminuric toxæmia. She had marked oedema of ankles, shortness of breath, systolic blood-pressure 152, and oliguria with albumin 12 grams to the litre. She was given fluids and the bowels were well evacuated.

September 18th, 1934.—Since the toxæmia persisted, induction of labour was indicated. The cervix was dilated and one rectal tube was inserted between the membranes and the uterine wall. Medicinal stimulation followed.

September 20th, 1934.—Pains absent. Rectal tube removed from uterus. Nine hours later labour had apparently commenced. Her pulse-rate was 92, temperature 100.2°F. The presentation was a vertex without disproportion, and the foetal heart was not heard. A pelvic examination was not made. Ten hours after the removal of the rectal tube, the pains were strong, maternal pulse-rate 90. One hour later the patient was complaining considerably of the severity of the pains. Eleven and a half hours after removal of the rectal tube the woman was extremely restless, anxious, and throwing herself about the bed with pain. Her pulse-rate was 96, her tongue furred and rather dry, and she had a fear and premonition of impending death. The uterus was contracting only occasionally, the foetal head was entering the pelvic brim and there was not any disproportion. A cause was not evident for the patient's distress, and an oral sedative was given, with little effect. Very shortly afterwards her colour became grey suddenly, her pulse was found to be soft and running, and breathlessness grew marked. Very quickly peripheral circulatory failure became complete, the pulse imperceptible, unconsciousness supervened, and in a few minutes death had occurred. It had been three hours since the onset of pains. Examination immediately post-mortem did not reveal any evidence of gas in the tissues, and the urine removed from the bladder was free of blood-pigment.

At autopsy, 12 hours later, the macroscopic findings were those of classical generalized gas infection, with physometra and foetal involvement, as in the case last described (No. 25).

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TABLE III.
CASE SYMPTOMATOLOGY (*Cl. Welchii* cases)

I. Group features.

	Group	Number of Cases	Predominant local lesions	Group features		Mortality
				Positive	Negative	
Post-abortal series	A	9	Endometritis. Physometra (Peritonitis)	Jaundice. Methaemoglobin serum urine	No pain.	89%
	B	5	Endometritis.	Jaundice.	No pain. No methaemoglobin serum urine	20%
	C	2	Endometritis. Skeletal muscle involvement.	Pain (Skeletal muscle). Early collapse.	No jaundice. No methaemoglobin serum urine	100%
	D	4	Endometritis.	Tachycardia. Or bacteriological diagnosis.	No pain. No jaundice. No methaemoglobin serum urine	25%
Puerperal series	1	1	Endometritis.	Jaundice.	No pain. No methaemoglobin serum urine	-
	2	3	Endometritis.	General symptoms only.	No pain. No jaundice. No methaemoglobin serum urine	100%
	3	2	Endometritis. Physometra.	Pain (uterine muscle). Early collapse.	No jaundice. No methaemoglobin serum urine	100%
	4	2	Endometritis. (Peritonitis.)	Bacteriological diagnosis. Abdominal pain.	No jaundice. No methaemoglobin serum urine	50%

II. General symptomatology. (Common to well-established and fatal cases.)

1.	Rapid, low-tension pulse. Pallor. General feeling of well-being.
2.	Subnormal temperature. Cold, clammy extremities. Peripheral cyanosis. Terminal restlessness and dyspnoea. Clear consciousness.

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SYNOPSIS OF GENERAL FEATURES.

The following features of the post-abortal and puerperal series as a whole have not been previously indicated.

Pulse-rate on Admission.

In abortal cases this ranged from 80 to 160, the average being 118 in fatal cases and 119 in those which recovered. In puerperal cases it lay between 84 and 144, with an average of 101; in six of the eight patients it was below 100.

Temperature.

The average on admission was 100.8°F. in abortal and 99.4°F. in puerperal cases; the average maximum in hospital was 102.5°F. in abortal and 102.6°F. in puerperal cases. It is of interest that the average maximum in abortal patients was 101.5°F. in those patients who died, and 103.8°F. in those who recovered; and in puerperal patients 102.2°F. in those who died, and 103.9°F. in those who recovered.

Rigors.

These preceded admission in 14 of 22 abortal cases (63 per cent), and in one puerperal (pyelitic) case. In women from whom *cl. Welchii* or *vibrio septique* was recovered from the blood during life, rigors preceded admission in six of 14 examples of pure infection (43 per cent) and in six of seven examples of mixed infection (86 per cent).

Rigors followed admission in seven abortal cases and one puerperal case, five being mixed infections; three of the latter died.

Vomiting.

This preceded admission in 14 of 22 abortal cases (63 per cent) but not in any puerperal case. Vomiting and rigors together preceded admission in 11 cases (50 per cent) which included all six abortal examples of mixed blood infection. Vomiting occurred after admission in five abortal and four puerperal cases, of which one of each was an example of mixed infection; eight of the nine were fatal.

Diarrhoea.

This was present before admission in two mixed abortal cases, one of whom died; and after admission in two fatal abortal cases.

Influence of Mixed Infection.

Although in one case (No. 14) it was certain that streptococci were the cause of death after the *cl. Welchii* invasion had apparently been successfully overcome, the mortality of our series as a whole does not show any evidence of an increased gravity due to mixed infection.

In 24 cases from which *cl. Welchii* or vibrion septique was recovered from the uterine cavity or blood-stream during life, the mortality of the 14 pure infections was 57 per cent and of the 10 mixed infections 50 per cent.

Considering only those patients from whom the organisms were cultured from the blood, there were again 14 pure cases with a mortality of 57 per cent, and seven mixed cases with a mortality of 57 per cent.

Duration of Life in Fatal Cases.

In the 13 fatal abortion cases death occurred on an average just over eight days after the last interference or suspected date of infection, and four days and eight hours after admission to hospital.

In five of the six puerperal cases death occurred on an average two days eight hours after interference; in one instance the time was only 26 hours. The remaining patient, who had a mixed infection, lived for 68 days.

Placental Tissue of Typical Variety. (Vide group A.)

This was present in eight of 22 abortal cases (36 per cent).

Ante-mortem Gas.

This was detected in one puerperal and four abortal cases (16.6 per cent).

In the former, gas crepitus was elicited in the upper abdomen just before death, but its significance was not realized at the time. In the latter gas was present in the uterus at operation and later in the vagina in one (No. 9); in the abdominal muscles and peritoneal cavity at operation in one (No. 21); in the gluteal muscles in one (No. 16); and was expelled from the uterine cavity during glycerine injection in one (No. 5).

Ante-mortem gas in extra-pelvic tissues has been noted by Adams¹¹, Collins¹², Gemmill (quoted by Toombs²), Lehmann, Matthews¹³ and Theobald¹⁴.

Uterine Perforation.

This was present in two cases (Nos. 8, 21).

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Case Admission.

The following was the chronological order of the admission of the cases:

1933 (8 cases): 15, 23, 1, 24, 25, 10, 26, 27.

1934 (20 cases): 17, 21, 11, 22, 2, 3, 4, 16, 12, 13, 14, 18, 28, 29, 19, 5, 30, 6, 7, 8.

1935 (2 cases): 20, 9.

CLINICAL DISCUSSION.

It must be admitted that in the present state of our knowledge and experience the early detection of post-abortal and puerperal infection due to *cl. Welchii* and *vibron septique* is usually difficult and often impossible. The preceding cases demonstrate to an extent little indicated in the literature and certainly not appreciated generally, the wide variation possible in clinical manifestations. Several factors contribute to this.

Firstly, there is the well-known variability in toxin-producing capacity of different strains of *cl. Welchii* (Gordon¹⁵, Lash¹⁶, Medical Research Committee¹⁷).

Secondly, pathogenic strains produce varying effects on the human organism according to the stage of pregnancy, the degree and site of bacterial invasion, the blood supply of the infected tissues and—doubtless in close association with these conditions—dependent on whether the bacterial haemotoxin or bacterial myotoxin chiefly dictates the clinical picture.

Although post-abortal and puerperal infections tend in general to differ in type, clinical effects cannot be accurately anticipated.

At one end of the scale is classical post-abortal gas gangrene, with its rapid development of anaemia, bronze icterus, methaemoglobinaemia and methaemoglobinuria, and due predominantly to the effects of the bacterial haemotoxin; this syndrome was present in 45 per cent of our post-abortal cases.

At the opposite end of the scale are puerperal physometra with its uterine pain and cataclysmic collapse, and metastatic muscle gas gangrene, both of which are surely dictated by the bacterial myotoxin.

In intermediate position, but tending toward the latter group, lie the majority of puerperal cases in which the effects of the bacterial haemotoxin are considerably in abeyance. In seven of our eight puerperal subjects, and in the greater number of fatal puerperal cases in the literature, jaundice, haemoglobinaemia and haemoglobinuria have been absent throughout.

What is the explanation of this? I would suggest the following possibility: During early pregnancy the uterine blood vessels have a relatively small calibre, and venous thrombosis as a result of endothelial damage by the *cl. welchii* toxin is particularly likely. The extent and rapidity with which this may occur were demonstrated in the uterus and adnexa removed at operation from case No. 9. In the uterus engorged with blood-clot the organisms find a rich and undisturbed pabulum in which growth and haemolysis proceed apace.

In the later weeks of pregnancy, on the other hand, the uterine muscle is greatly hypertrophied and the calibre of its vessels is enormously increased. A more massive muscle infection and a greater rate of toxic and organismal absorption are now possible. But in keeping with the increased blood supply and the diminished frictional resistance in the large venous channels, thrombosis, and with it haemolysis, are much less liable to occur. It is possible that in some cases of overwhelming infection thrombosis may be forestalled by death.

In skeletal muscle infection, as Toombs suggested, the relatively poorer vascular and lymphatic supply apparently accounts for the comparative rarity of general haemolytic phenomena.

Finally, to the *cl. Welchii* toxin as a whole may be attributed that state of collapse and peripheral circulatory failure which is the terminal feature of fatal cases and the result, as Topley and Wilson¹⁸ have pointed out, of the deleterious action of the toxin on the suprarenal glands.

Diagnosis.

This involves not only identification of the bacterial cause, but an estimation of the underlying anatomo-pathological lesion. From the standpoint of successful therapy the latter is of fundamental importance.

With regard to the former I reiterate these outstanding points of clinical value.

I. In any pregnant, post-abortal or puerperal woman who is the presumptive or conceivable subject of uterine infection, the development of any one or more of the following signs or groups of signs should arouse immediate suspicion of infection due to *cl. Welchii* or an organism of its class:

(a) Jaundice. The rapid development of jaundice, though characteristic, is not invariable.

Jaundice occasionally arises in streptococcal and less often in other organismal infections, but is then usually late, slow in

development and does not attain the depth seen with *cl. Welchii* invasion.

In lead-poisoning with jaundice the pulse-rate is slow, basophilic stippling is almost invariable, and there is not any rapid clinical progression.

When deepening jaundice is accompanied by free blood pigment in serum and urine *cl. Welchii* infection is certain and the prognosis already desperate. Haemolysis is not seen in the absence of jaundice.

Jaundice was present in 63.6 per cent of our abortal and 12.5 per cent of our puerperal cases.

(b) Increasing pulse-rate out of proportion to an evident pathological lesion, and usually attended by a feeling of well-being. The patient is characteristically pale and her blood-pressure is low.

(c) Increasing and excruciating pain of uterine, skeletal muscle or indeterminate origin, accompanied by shock and not adequately explained by the known lesions present.

Pain was a feature in only eight cases of our series (26.6 per cent); in two cases of puerperal physometra and two of metastatic gas gangrene, which fall into the group just described; and in four cases of general peritonitis.

(d) Sudden collapse for which general examination does not reveal any obvious cardiac, pulmonary, cerebral or intra-abdominal cause.

2. Only occasionally can one expect to detect gas in the tissues during life. Heim has suggested the possibility of the X-ray detection of physometra.

3. Factors liable to mask diagnosis are mixed infection, chiefly to be seen in post-abortal cases; and in puerperal women such predisposing and contributory conditions as toxæmia of pregnancy, post-haemorrhagic anaemia, exhaustion from dystocia and post-operative shock.

4. Blood cultures, both aerobic and anaerobic, should be taken as early as possible in all cases of abortal and puerperal sepsis.

CLASSIFICATION.

In attempting to estimate from clinical features the nature and extent of the causative lesion the following classification, taken largely from Nürnberger¹⁹ and Heim, is of value.

Four grades of post-abortal and puerperal gas gangrene may be recognized, any one or combination of which may be present in the same subject.

I. *Local gas gangrene.* Here infection is limited to the uterine contents and superficial layers of the decidua.

The majority of these cases are mild, but occasionally severe infection occurs, with haemolytic phenomena^{5, 7, 19}. A moderately sick case in our series was No. 20; a grave example with blood destruction, No. 4.

With early and careful cleansing of the uterine cavity and adequate specific therapy the prognosis is usually good.

It is important to realize that in the occasional case with haemolysis the clinical picture becomes indistinguishable from that of the next group.

II. *Physometra or gas gangrene of the uterine musculature.*

These cases are all desperate, and a fatal issue is to be expected. It is indeed difficult to understand how, once the uterine musculature has become involved, recovery can occur without early extirpation of the uterus.

In abortal cases the syndrome produced is that of classical gas gangrene with its gross haemolysis and rapid downhill course. Blood culture is positive in the great majority.

The detection of the causative physometra is, however, a matter of extreme difficulty. Brütt and Lehmann⁵ state that the uterus is characteristically enlarged, very sensitive to pressure, and frequently elastic; gas crepitus, which they elicited in two of nine examples, is pathognomonic. In addition they hold that evidence of peritonitis is constant and usually expressed in marked meteorism, loss of muscle tone in the lower abdomen, and paresis of the intestine. Heim⁷ concurs with the latter view and states that, if there are neither symptoms of peritonitis nor evidence of it at abdominal paracentesis, the infection is probably limited to uterine contents and superficial layer of the decidua.

Cases 5 and 9 of our series were examples of post-abortal physometra, proved at autopsy and at operation respectively. In the first, helpful local signs were confined to uterine tenderness and slight lower abdominal tenderness; this woman developed general peritonitis four days later. In the second, the uterus was tense and markedly tender, there was palpable bulging in both lateral fornices, and both iliac fossae were tender; at operation there was blood-stained fluid in the peritoneal cavity, and gas was present in decidua and uterine muscle; crepitus had not been elicited clinically.

Uterine and abdominal tenderness are confined neither to

physometra nor anaerobic infections generally; and the former sign was present in our mildest case (No. 19).

In puerperal physometra, of which there were two examples in our series (Nos. 25, 28), haemolysis appears to be in abeyance, and the picture becomes one of excruciating pain, followed by rapid collapse and death. I do not know of any record of recovery in a proven case.

III. *Peritonitis.*

Peritonitis most commonly arises by lymphatic spread from the uterine cavity or muscle; much less often it is due to uterine perforation.

In the lymphatic form peritonitis is typically pelvic and clinically unobtrusive. The exudate is haemorrhagic, soon becomes brown, and is full of organisms; in mixed infection it is offensive.

Signs of general spread are an increasing pulse-rate and dyspnoea in association with a poor general appearance and increasing abdominal distension. This was a terminal event in two of our cases (Nos. 5, 23). Brütt and Lehmann believe that peritonitis without contemporary physometra is rare.

IV. *Bacteraemia.*

Bacteraemia may be associated with infection of the decidua, uterine muscle or peritoneum. Bacteria enter the blood-stream transiently, continuously or in groups, and may there produce free toxin or metastatic infection. Many of the organisms are destroyed in the blood and ingested by leucocytes: some pass through the kidneys and may be cultured from the urine.

It must be emphasized that the mere recovery of organisms from the blood-stream, as is not uncommonly possible in entirely non-suspect cases following abortion or curettage, by no means constitutes clinical infection. An example of transient bacteraemia associated with interference and clinically unimportant was No. 19 in our series.

With the continual or repeated passage of bacteria into the circulation clinical signs are soon manifest, and may be essentially haemolytic or simply those of generalized blood infection.

In our series, cases No. 10, 5, 21 and 16 represent pictures of clinical septicaemia in association with endometritis, with physometra, with physometra and general peritonitis, and with metastatic gas gangrene, in that order.

TREATMENT.

A. *Prophylaxis.*

In abortal cases this is particularly difficult. That all examples of septic abortion may be regarded as suspect scarcely justifies their routine prophylactic injection with *cl. Welchii* antitoxin. But one important weapon of attack which has not yet been employed is a widespread publicity campaign exposing the grave dangers accompanying all attempts at criminal abortion.

In puerperal cases prophylaxis entails the avoidance before, during, and after delivery, of all unnecessary trauma, gross manipulations and intra-uterine interference; more especially in women with long-standing rupture of the membranes, signs of incipient sepsis, foetal death, or a general condition undermined by shock, cold, fatigue, haemorrhage, toxæmia of pregnancy or constitutional disease.

The most potent factors in assisting invasion in the puerperal class are vaginal plugging, tubal induction, destructive operations, forcible attempts at delivery, and intra-uterine manipulations, particularly when performed in the presence of foetal death, long-standing rupture of the membranes, or in a patient already fatigued, toxæmic, or shocked from haemorrhage.

Any combination of the above predisposing and initiating factors should suggest the advisability of a prophylactic intramuscular injection of 20,000 to 40,000 international units of *cl. Welchii* antitoxin following, or if necessary before, delivery. In the presence of an offensive or necrotic-smelling liquor or vaginal discharge the larger dose should be given forthwith, and if the patient's general condition is not completely satisfactory two to three hours after delivery, its repetition must be considered.

B. *Therapy.*

Success in treatment depends upon the practical application of three cardinal principles: early diagnosis, early elimination of the primary focus, and immediate and massive specific and general therapy.

Once again the importance of an early clinical diagnosis must be emphasized. Smears from lochia, cervix or uterine cavity may be presumptive but are not bacteriologically final, while the cultural investigations necessary for positive identification of organisms of the *cl. Welchii* class involve a delay of eight to 12 hours at least, and often longer. How fatal such delay may be was shown in puerperal case No. 24.

In the most serious members of our series who recovered

diagnosis was without exception initially clinical, and therapy had already been instituted some hours when bacteriological confirmation was received. (Cases No. 10, 26, 4, 13, 20.)

I. *Elimination of the primary focus.*

(i) In the majority of abortal cases (endometrial infection) this involves immediate and careful uterine curettage under gas and oxygen anaesthesia. Lack of adequate care in the procedure may change endometritis into physometra.

It is our practice post-operatively lightly to pack the uterine cavity with gauze soaked in glycerine, owing to the tendency to delayed oozing from the raw surfaces; this gauze is removed in six hours. Intra-uterine glycerine therapy is thereafter continued daily until involution is satisfactory and uterine discharge has ceased.

(ii) In cases of abortal physometra the correct local treatment is immediate total hysterectomy, together with removal of the adnexa should these appear congested, swollen or discoloured.

Once the uterine musculature has been invaded death is inevitable if the organ remains *in situ*; a striking example of the futility of general and specific therapy, however intensive, when the primary focus has not been adequately dealt with, was case No. 5.

The problem lies, as already stressed, in the detection of the physometra. In the presence of the classical haemolytic picture, and local signs as laid down by Brütt and Lehmann, the decision to operate is not difficult. But what of those patients with the same classical picture but lacking adequate local signs?

I have come to the conclusion that in these also the correct procedure is hysterectomy; when organisms are early recovered from the blood, or haemolysis is obviously advancing, or signs of general toxæmia are steadily increasing the indication is absolute. Desperate though the remedy is, it offers the only chance. No mortality could be more disastrous than that which has attended our most strenuous efforts to save these patients with less radical measures. Brütt and Lehmann have saved four out of nine, and Heim two out of 10 cases of physometra by immediate hysterectomy.

The operation should be preceded by the administration of adequate fluid, serological therapy, and blood transfusion, and by thorough cleansing of the vagina following the insertion

into the uterus of an antiseptic gauze, and closure of the cervix. Total removal of the uterus, with vaginal and abdominal drainage, should be performed with the utmost speed, preferably under gas and oxygen inhalation; spinal anaesthesia which necessitates for some hours post-operatively a posture unfavourable to an infective case, is contra-indicated.

(iii) In cases in which laparotomy and drainage are performed for peritonitis the decision as to whether the uterus should be removed or not becomes one of nice distinction. Brütt and Lehmann hold that if the uterus appears externally intact it should be left; Heim, that as external normality does not necessarily mean muscular normality, hysterectomy is the safer procedure.

If, as Brütt and Lehmann assert, peritonitis is rare without contemporary physometra, then, if the patient is a suitable operative risk, laparotomy for peritonitis should automatically include hysterectomy; in the presence of the classical haemolytic syndrome, as previously stated, I certainly believe the patient will die without it. In cases in which peritonitic symptoms alone have dictated immediate laparotomy and drainage, the patient is usually too ill to withstand further operative procedures.

(iv) The application of the principle of focal elimination to puerperal cases is more difficult.

As Wrigley¹⁰ points out, in the presence of a dead foetus delivery should be at the earliest moment and in the gentlest possible manner. When clinical evidence of infection develops with the child *in utero* the same principle holds, but too often clinical evidence is synchronous with collapse and a rapid down-hill course.

In the puerperium daily intra-uterine glycerine therapy may be instituted, but is of doubtful value. The question of more radical local measures in this type of case must await the thorough trial of early and adequate general and specific therapy.

2. Specific and General Therapy.

(i) *Specific serum therapy.* This constitutes, next to elimination of the primary focus, our most potent method of attack. The value of the *cl. Welchii* antitoxin is unquestioned, and it may be taken that, in its absence, any case of post-abortal or puerperal gas gangrene with signs of severe toxic absorption or severe organismal invasion will prove rapidly fatal.

One cannot expect serum to save life if a persistent or spreading focus, such as physometra, is left *in situ*, if infection has

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passed into the general peritoneal cavity, if other organisms are the chief contributors to a fatal result, if the kidneys and liver are already irreparably damaged by toxins, or if its employment is not along scientific lines. The commonest errors in administration are delay in initiation, inadequate dosage and too early cessation.

Coincident with diagnosis 40,000 international units of *cl. Welchii* antitoxin should be given intramuscularly as a routine, followed as soon as possible by 40,000 to 60,000 units intravenously in 20 ounces of normal saline or 10 per cent glucose in normal saline.

Intramuscular injections of 40,000 units should be repeated every 12 hours for at least three days in serious cases, after which daily injections are usually sufficient, and may be continued for a week or more as necessary.

Intravenous therapy in 40,000 to 60,000 unit doses is best repeated daily for two to three days, or even longer in grave cases; in many of less gravity response is so rapid that after the first 24 hours intramuscular therapy alone is sufficient.

Serious cases usually require 200,000 to 600,000 units by combined routes over the course of a few days.

So long as jaundice, haemoglobinaemia, haemoglobinuria or rapid pulse-rate persist, or organisms are recoverable from the blood, urine or uterine cavity, specific therapy must be continued. In one of our cases (No. 4) deep jaundice persisted for several days after all other evidences of *cl. Welchii* infection had disappeared. It is advisable to err on the side of safety and to discontinue serum only when clinical indications for its usage have been absent for at least two to three days.

(ii) *Hydrotherapy*. Copious oral fluids containing glucose are administered from the start according to a definite plan. It becomes one nurse's duty to see that the patient's fluid intake is four to six ounces every 15 minutes. Quantities should be charted.

Oral fluids are invariably supplemented by intravenous 10 per cent glucose in normal saline in quantities of 20 to 30 ounces, the first injection being given immediately following diagnosis. These injections, which are useful vehicles for serum therapy, are repeated at 12, 18 or 24 hour intervals, according to the patient's condition. Insulin, one unit per two grams of glucose, may with advantage accompany them. Submammary or intra-axillary, and rectal salines are commonly called for in addition.

(iii) *Blood transfusion.* This is an indispensable factor to success in patients with marked haemolysis, in whom it should be instituted early and as a routine. Repeated transfusions may be necessary to combat the rapidly progressing anaemia.

In serious cases, even in the absence of evident haemolysis, blood transfusion appears to be of definite value in lessening toxæmia.

In two puerperal and 10 post-abortal cases of our series, of whom eight of the latter showed evidence of haemolysis, blood transfusion was performed. In seven patients the blood was transfused once, four twice and one thrice; the average quantity of blood given to women receiving a single transfusion was 21 ounces, to those receiving multiple transfusions, $15\frac{1}{2}$ ounces per injection.

(iv) *Administration of alkalis.* *Cl. Welchii* during growth creates an acid medium¹⁷, and its toxin is most stable at a hydrogen-ion concentration well on the acid side of neutrality²⁰, [and Walbum.]²¹

Clinically the former is expressed early in the strongly acid urine excreted, and later in the thirst and dyspnoea accompanying acidosis.

In a patient with severe haemolysis one of the gravest dangers to be faced, should the patient survive some days, is renal failure. In addition to the known effects of *cl. Welchii* toxin on the convoluted tubules, an important contributory factor in producing anuria must surely be the mechanical blockage resulting from the precipitation of acid haematin in the renal tubules as free haemoglobin is excreted in an acid filtrate. For these several reasons early and intensive alkaline therapy is essential, and becomes an absolute necessity in cases with severe haemolysis. Sodium bicarbonate is the most satisfactory salt for general administration, and is included in all drinks in quantities of two to four drachms, and, when tolerated, even up to eight drachms to 20 ounces. It can with advantage be given intravenously at the start, 15 to 20 ounces of a four per cent solution in normal saline¹⁷, which should be repeated if lesser measures do not keep the urine alkaline. In lower concentrations it is usefully included in submammary and rectal injections.

(v) *Treatment of renal failure.* Urinary suppression must be anticipated by the institution, from the moment of diagnosis, of intensive fluid, antitoxic and alkaline therapy. Once established, it appears to defy all treatment.

Oliguria should be combated by the free exhibition of saline

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TABLE IV.

COMBINED ANATOMO-PATHOLOGICAL, CLINICAL, AND TREATMENT TABLE.

Anatomo-pathological type	Clinical findings			Treatment		General	
	Local signs	SYMPTOMATOLOGY		LOCAL			
		Abortal cases	Puerperal cases	Abortal cases	Puerperal cases		
LOCAL GAS GANGRENE (endometritis and infection of uterine contents).	Necrotic placental tissue. Recovery of Cl. Welchii from uterine cavity.	Commonly absent. Or blood destruction, or general symptomatology as below.		Curettage. Glycerine therapy.	Delivery as early and as gently as possible. Glycerine therapy.		
PHYSOMETRA (infection of uterine muscle).	Uterine crepitus (rare), tenderness, enlargement, irregularity.	i. Jaundice, dusky cyanosis, port-wine urine, colour of burgundy serum. Grades between i. and ii. ii. Muscle pain, pallor, collapse. iii. General symptoms of blood infection; typically with rapid, low-tension pulse, pallor and general feeling of well-being.		Total abdominal hysterectomy.	? Hysterectomy.	Same principles apply in all types:— i. ANTI-SERUM. ii. HYDRO-THERAPY.	
PERITONITIS (commonly associated with physometra).	Lower abdominal tenderness and distension. Haemorrhagic or brown exudate, laden with organisms; offensive when mixed infection.	May be associated with any one or combination of the above clinical features.		Laparotomy and drainage. (Hysterectomy.)		iii. BLOOD TRANS-FUSION. iv. ALKALI THERAPY.	
BACTERIAEMIA (may be associated with any one or more of preceding types).	Metastatic infection. (rare).			—			

aperients; by intravenous injections of 10 per cent glucose in normal saline and, later, of 50 per cent glucose solution, preferably suitably buffered; and by the application to the loins of hot plastines, cupping or medical diathermy. When these measures have failed we have employed blood transfusion (No. 5) rather in the hope that its combined colloid and salt content might initiate a successful hydraemic plethora when purely crystalloid solutions had failed.

Calcium gluconate, 20 cubic centimetres of a 10 per cent solution intravenously and, as a last resort, oral diuretin, and salyrgan have been administered, without success.

Decapsulation of the kidneys, equally unsuccessful, has been reported by Lehmann.⁴

CONCLUSIONS.

1. A series of 30 cases of post-abortal and puerperal gas gangrene has been described, from 22 of which *clostridium Welchii* and from two of which *vibron septique* were recovered from the tissues during life. The mortality was 63 per cent.

2. The considerable frequency of this dread infection, which is protean in its manifestations, and of recently increased incidence in Melbourne, is indicated.

3. The fundamental importance of early diagnosis is emphasized. The clinical features of the various types have been outlined, systematized, and, where this is thought to be of value, classified in an attempt to achieve clarity and assist early recognition. An explanation is offered of certain divergencies of the clinical picture in early and late pregnancy.

4. The principles underlying successful treatment are early diagnosis, early elimination of the primary focus, and immediate and massive serological and general therapy. A synopsis of treatment on these lines is presented.

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