INFECTION OF HAEMOTHORAX BY ANAEROBIC GAS-PRODUCING BACILLI.

BY

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(A Report to the Medical Research Committee.)

WHEN a man has been wounded in the chest, the two chief questions which arise in the minds of those who have the care of his case are whether his life will be endangered by further leakage of blood and air into the pleural cavity, or by the development of an infection.

Such figures as have been collected up to the present show that about one-fourth of all cases of haemothorax from gunshot wounds of the chest in this Flanders campaign are infected. In consequence of this frequency of sepsis, the principle of early exploration as a test for bacteriological infection of a haemothorax is adopted in all our military hospitals. But experience of the treatment of chest wounds in France is continually emphasizing the need for still greater vigilance in the early detection of sepsis, because there exists one particular group of infected cases which, though common, is liable to errors in diagnosis and treatment on account of its peculiar nature. A paper was written by us in description of this particular clinical group eighteen months ago, but owing to various circumstances it was not published at the time. A year's further observations, and in particular what was seen with the recent casualties from the fighting on the Somme, have convinced us that the need still exists for a more or less formal account of what may happen in the course of these chest wounds, for this particular infection seems to have been fully as common this autumn as ever it was before in France.

The group with which this paper deals is that due to the growth in a haemothorax of certain anaerobic bacilli, which may produce gas in the pleural cavity in so rapid an abundance that the physical signs of what is really a case of serious infection may be such as to lead the surgeon to the opinion that he is dealing only with a simple non-infected pneumo haemothorax that is being aggravated by a progressive leak of air from the lungs. To act on this opinion and leave the patient to rest and morphine, though the correct treatment for the early stages of a simple air haemothorax, is merely to await the death of a man whose life might have been saved by timely recognition of the sepsis and immediate rib resection.

This form of infection with gas production is frequent. Out of the first 500 specimens of haemothorax fluid examined by one of us in the ordinary routine of laboratory work, 195 were found to be septic; and of these, 87, or 43.6 per cent. of the total of septic cases, were proved to be infected with such anaerobic bacilli.

After a latent period of varying duration, the gas and the poisons produced by the bacilli may develop in many instances with fulminating rapidity amid the haemothorax, so that a case which on the second or third day following the wound was regarded only with suspicion of sepsis has often been seen to pass in the next forty-eight hours into a state of the gravest danger. Even if the patient's life is not immediately imperilled by a developing toxæmia, there is still a disadvantage in every hour's delay in detecting the infection, because the increasing volume of gas compresses the lung and enlarges the empyema cavity, so that subsequent convalescence after rib resection is likely to endure for a longer time than would have been necessary had the pleural cavity been evacuated on an earlier date.

For both reasons, therefore, whether to save life or to lessen the period of invalidism, it is of the utmost importance that every endeavour be made by medical officers to recognize this type of infection at the earliest possible moment, and to deal with it without delay.

GENERAL STATISTICS.

During the year 1915-1916, in a consecutive series of 145 examples of septic haemothorax of which clinical

notes were taken by us, the infection was bacteriologically identified in 101 cases. The details are as follows:

Organisms Found:	No. of Cases.	Total Deaths.	Total Deaths from Septic Haemothorax of Hopeless Gravity and without other Complications.
Pneumococcus, <i>B. influenzae</i> , and <i>M. tetragenus</i> —that is, from respiratory tract	17	4	2
Streptococcus and staphylococcus	38	11	4
Anaerobic gas bacilli (alone = 23, with other organisms = 19)	42	14	4
Diphtheroid bacillus, <i>B. coli</i> , etc.	4	0	0
	101	29	10

From these figures it appears that the haemothorax was infected from the lacerated lung in rather less than 20 per cent. of the total; and that when infection was introduced from without, from the soiled skin or by shreds of clothing, the anaerobic bacilli were present in about one half of such cases of external infection.

Some statistics given elsewhere by one of us (in a paper with Sir John Rose Bradford, *British Journal of Surgery*, iii, p. 249, 1915) showed that one-fourth of the cases of haemothorax seen at certain base hospitals during the first year of the war were infected. From this it follows that approximately 10 per cent. of all cases of haemothorax seen at this level on the L. of C. contain anaerobic gas-producing bacilli. These are, or would become, for the most part, gravely ill cases; but with proper recognition of the sepsis and timely rib resection the mortality among instances of such infection is shown by the last column in the table printed above to be very similar to that for the other types of infected haemothorax, namely about 10 to 15 per cent. This reduced mortality is the total of deaths, four in number, which were due simply to the infection of the haemothorax, and which appeared to be hopeless according to the present methods of treatment. The other ten deaths, which were put aside for the purposes of attaining this figure, included three in which there was some error in the surgery employed, and six in which the empyema was drained for various reasons at too late a date to give much chance of recovery.

It must be admitted that the distinction which is made in the analysis of these numbers rests on a personal judgement gained by fuller experience of those which might have been saved by earlier operation, and of those which seemed hopeless, so that it is not one which yields an exact figure of mortality. But the results actually obtained in another series of these gas bacillus infections, which was collected subsequently to that quoted above, proved that their death-rate at base hospitals should not exceed 15 per cent. Most of the cases recover rapidly, even though they may have seemed to have been desperately ill before the empyema was drained.

It will be observed that the wound in every single one of the fourteen clinical examples of anaerobic infection, which are quoted later in this paper, was caused either by a fragment of high explosive shell or by shrapnel ball. Undoubtedly there is a greater liability for anaerobic bacilli to be carried in by shell fragments than by rifle bullets, but the preponderance is by no means so exclusive as these cases would suggest.

PATHOLOGICAL PROCESSES.

Certain details of the pathological changes which occur in the development of a septic haemothorax require description first in order that the clinical features may be better understood.

Jagged missiles which enter the chest often carry in shreds of dirty clothing around them. Most of these infected shreds are left close to the entrance wound; the remainder may adhere, especially to a shell fragment, and if such be the case infection must develop around the latter in its final resting place. This resting place is generally either in the structures of the thoracic wall or within the hollow of the pleural cavity. Pieces of shell, and more rarely bullets, may occasionally be embedded in the lung itself; but as a rule these missiles have enough

momentum to force their way through the non-resistant pulmonary tissue, after which they may fail to penetrate the bony framework of the chest, and so drop back into the pleural cavity.

The wound made through the lung is generally a lacerated track surrounded by a considerable amount of haemorrhagic extravasation. From these torn tissues and from the wound in the thoracic wall, where ribs are often splintered, fibrin ferment will be liberated in sufficient quantity to coagulate rapidly all the fibrinogen in the blood that is poured out from the wound track in the lung or chest wall. But the continued movements to which the effused blood is subjected within a breathing chest interfere with its coagulation and prevent its setting into a massive jelly. The fibrin is partly "whipped" out and deposited on the pleural surfaces; irregular lumps of more complete blood clot may form like mush ice in the haemothorax, especially in the recesses of the pleural sac and along the vertebral groove at the back when the patient is recumbent. The rest of the defibrinated blood remains as a fluid, a fluid which resembles blood in depth of colour more or less closely according to the number of red corpuscles that have escaped entanglement in the primary clot and are floating freely in it.

The pleural surfaces react to the irritation by the blood, and a serous effusion containing lymphocytes and endothelial cells, with a few polymorphonuclear leucocytes, is added to the haemothorax. Often the fibrinogen of this effusion also is clotted by the surplus of ferment from the haemothorax blood, and it reinforces the deposit of clot laid down on the pleura, so that adhesions of some tenacity are soon developed at the boundaries of the haemothorax, and thus separate the collection of fluid and clot from the rest of the pleural sac.

We are dealing in this paper exclusively with infections by anaërobic bacilli which are introduced chiefly from without, and therefore respiratory tract infections that may develop in the neighbourhood of the lacerated lung can be neglected. An infection from without may be introduced, broadly speaking, in two ways.

I. Generalized Form.

The infection may be disseminated throughout the fluid haemothorax from the very commencement, as occurs most conspicuously where the external wound track remains open and septic, so that organisms are sucked in with the to-and-fro currents produced by respiration.

II. Localized Form.

The septic focus may be one which is buried in a mass of blood clot—for example, a shell fragment with tattered clothing around it lying at the bottom of the pleural sac. Ultimately the organisms escape through the agglomerated clot, and are spread by respiratory movements through the pool of serum above.

The anaërobic organisms, presumably on account of their feeble powers of growth at first in freshly shed blood, often appear to grow slowly from an implantation which is enveloped by blood clot as described in (II). During this time they produce toxic substances which are absorbed into the circulation from the tissues around the septic blood clot, causing a general febrile reaction, while the same substances diffuse into the fluid haemothorax and excite from the pleural membranes an increase of serous exudate, to which polymorphonuclear cells are now added in greater numbers.

The growth of the bacilli within the septic clot is generally accompanied by the evolution of foul gas, which at first may remain imprisoned in the mass of clot as a small pneumothorax localized within the general haemothorax. Ultimately this gas bursts the clot asunder, and so scatters the infection through a wider field, or the bacilli make their own way directly through the obstructing clot into the fluid haemothorax.

Condition (II) is then thrown into the ordinary form of condition (I), with the free pullulation of bacilli throughout all the haemothorax fluid. An intense inflammatory pleurisy follows, and there is an abundant discharge of fluid effusion and of pus cells into the pool. Gas formation sometimes increases the volume of the empyema even more rapidly than does the fluid exudate, and this gas now lies free above the upper level of the fluid. Hour by

hour the lung collapses more and more beneath the growing pressure; but the early adhesions at the boundary of the haemothorax usually hold fast and tether the upper part of the lung so that its collapse is not symmetrical and complete, and the gas may fail to reach a mobile position at the apex of the pleural sac. On account of this inability of the lung on the side of the haemothorax to collapse completely away from the side of the chest, the increasing volume of the pyo-pneumothorax causes an extra displacement of the mediastinum and the heart to the opposite side.

Apart from these local changes within the pleural sac on the side of the septic haemothorax, there are three directions in which the spreading results of the infection may appear.

1. In the opposite pleural sac, causing a pleurisy which is usually dry and rarely leads to the development of effusion and empyema. This contralateral pleurisy is a very frequent accompaniment of a septic haemothorax, especially when the latter has been neglected for some days.

2. In the pericardial sac, a path of spread which is much less common than that into the contralateral pleural sac, though when the infection takes this course, a purulent effusion usually develops and is quickly fatal. None the less, there is unequivocal evidence that a neighbouring infection may excite a dry pericarditis from which there is recovery as complete and as straightforward as in the case of a contralateral dry pleurisy.

3. In the blood stream, producing general toxæmia or even septicæmia. This may develop with rapid fatality when the focus of infection lies in vascular tissue, as when a shell fragment is embedded in the lung. In such a case there is hæmolysis of the circulating blood, the serum acquires a deep yellow tint from the changes undergone by the freed blood pigment and a jaundiced colour spreads over the face and body of the patient as a signal of the toxic state that is threatening his life.

The results of sepsis may appear along this third path when the infection is a pure one by anaërobic bacilli only. But our observations suggest that these bacilli do not easily spread from a haemothorax into the pericardial sac or the opposite pleura; and the infections described under headings 1 and 2 are probably in most of the cases due to an associated contamination with streptococci, which are the active invaders of the outlying regions beyond the haemothorax.

THE RESULTS OBTAINED BY EXPLORATORY PUNCTURE.

Before narrating the clinical features which are associated with an infected haemothorax of this nature, it is best to describe the varieties of samples that may be met with on exploration of the chest, because it will be continually necessary to refer to these in the course of the clinical description. The exploring needle is the sole means for obtaining the final evidence for a strict diagnosis. Cases which clinically present the appearance of being septic may be found by repeated punctures to be completely sterile; and on the other hand, cases which seem almost innocent, and beyond suspicion so far as rise in temperature is concerned, may turn out then and there to be heavily and dangerously infected. Therefore the needle should be used whenever doubt arises.

The contents of the pleural cavity, when infection is present in a haemothorax, often are not homogeneous. The needle may chance to enter an area of reactive serous effusion, or of haemothorax fluid, or blood clot; and any one of the samples so drawn off may at first be free from organisms, though a couple of days later bacilli may be abundant everywhere and their presence be at once made evident by the malodorous smell attached to the fluid as the consequence of their growth.

The following are the varieties of samples that may be met with in exploration of a haemothorax infected with anaërobic:

- (a) A "blood" with an offensive odour, and of an intense purple colour, which is darker and more transparent than that of venous blood. This fluid contains dissolved hæmoglobin from hæmolysis of the red corpuscles, and consequently it will throw up a crimson froth in the barrel of the syringe. The crimson-purple colour is in itself almost characteristic of an abundantly growing infection by anaërobic bacilli; but the foul smell is the chief criterion,

and it may be accepted as sufficient proof of infection. From such a sample, on standing, a large flocculent deposit of reddish-brown pus may fall down, but the eye cannot recognize the presence of this pus in the freshly drawn sample. In some of these cases the needle may at first find only offensive gas under pressure, and fail to withdraw any fluid.

(b) A fluid that is obviously loaded with pus, reddish-pink or deep buff in colour, and either slightly or not at all offensive.

(c) A red fluid like that of an ordinary sterile haemothorax and inoffensive. Microscopic examination finds in films from this fluid a moderate increase in the number of polymorphonuclear cells, and bacilli appear on culture.

(d) A yellow serous fluid with the usual fine gelatinous secondary clot on standing, and the same findings under the microscope as in (c).

The offensive odour is the only criterion of infection that can be accepted at once without further microscopic study.

A purulent appearance may in very rare cases be given by a traumatic leakage of chyle, so that this naked-eye appearance is not completely reliable.

Otherwise all specimens should be examined under the microscope, and cultures made if no bacilli can be seen in direct films. The most rapid incubation is that within the chest itself. If a case is clinically suspicious, but the first sample receives a negative report from the bacteriologist, a second and even a third exploration should be made on the following days and in different places.

The delay in bacteriological proof of infection in a case which on clinical grounds appears certainly to be septic is a striking feature of this particular group. Such delay is never met with in infection by streptococci and staphylococci. It occurs occasionally with infection by the influenza bacillus or pneumococci, when it may easily be ascribed to a slow passage of these organisms from injured areas within the lung out into the surrounding haemothorax. But it is quite frequent with the anaërobic bacilli, although there can be no doubt that the infection in such cases was introduced from without at the moment when the wound was received.

Thus out of one continuous series of septic cases:

Thirty were found to be due to streptococci and staphylococci, and the proof of the infection in every one of these cases was found at the first exploration.

Forty-five were infected by anaërobic bacilli, and nineteen of these were infected only by members of this group. Of the latter, six were reported on the first sample to be absolutely sterile, and four others yielded proof of infection only by culture. Hence 50 per cent. of these pure infections by anaërobic bacilli yielded on first exploration a fluid that showed no organisms in a direct and careful film examination. These first samples were taken on days varying from the fourth to the eighth after the wound had been received, and the real proof of bacteriological infection was obtained from one to five days after the first sample that was reported to be sterile.

Clinical examples of this delay will be quoted later. The essential fact is that infection by anaërobes often tends to remain localized for a relatively long time in some part of a haemothorax, probably within clot. Hence early exploration of the chest may in one place strike a bubble of gas and foul blood as in (a), while in other places it may discover only such fluids as (c) or (d), fluids which after the lapse of a few days would in the same places be developed respectively into (a) or (b).

BACTERIOLOGICAL OBSERVATIONS.

The method of examination in the laboratory of pleural fluids taken from cases of haemothorax has been uniform throughout and has resolved itself into two stages, namely:

A. The immediate microscope examination of the fluid or of its centrifuged deposit.

B. The preparation from it of both aerobic and anaërobic cultures.

A.—The Direct Microscopic Examination.

Air-dried and flame-fixed films made on microscope slides are rapidly stained with a drop of Loeffler's alkaline methylene blue, and examined in the wet stain through a superimposed cover-glass. This method affords immediate and valuable evidence of the nature both of the cellular

exudate and of the infecting micro-organisms. A second film yielding supplementary information is stained by Gram's method, while the first is being examined.

In fluids which are frankly purulent or which throw down a purulent deposit on centrifuging, it is a rule to find organisms after a brief search through these two films. The only microbe that is likely to escape detection by this simple routine is the influenza bacillus, which can be demonstrated by staining in weak carbol-fuchsin for half an hour.

We have found that, with reasonable patience in searching, the wet methylene-blue film and the Gram film together are sufficient to show the presence of organisms in 90 per cent. of all fluids subsequently proved by cultures to be infected, and to give an approximate indication of their nature. The accompanying table gives briefly a list of the organisms likely to be met with in an infected haemothorax fluid:

Table of the More Common Organisms Found in Infected Haemothorax Fluids.

Cocci.

Gram-negative:

Micrococcus catarrhalis.

Gram-positive:

1. Streptococci.	4. <i>Micrococcus tetragenus</i> .
2. Staphylococci.	5. Sarcinae.
3. Pneumococcus.	

Bacilli.

Gram-negative:

1. *B. coli* and coliform organisms including *B. proteus* and *B. pneumoniae*.
2. *B. pyocyaneus*.
3. *B. influenzae*.

Gram-positive:

- (a) Aerobic—
 1. *Subtilis-mycoides* group.
 2. A diphtheroid bacillus.
- (b) Anaërobic—
 1. *B. perfringens*.
 2. *B. sporogenes*.
 3. *B. von Hübner* IX and others.
 4. A small diphtheroid.

The cocci can be neglected in the present discussion, for no real anaërobes were found in this group, and none are capable of forming gas. Among the Gram-negative organisms in the bacillary group it is only the coli-like bacilli that are gas producers; but none of these were found by themselves—that is, in the absence of an anaërobo—to give rise to gas inside a haemothorax cavity.

There remain only the Gram-positive bacilli for consideration.

Diphtheroids.

The stout aerobic diphtheroid bacillus, which closely resembles the bacillus of Hoffman, is but rarely seen in films, and as a rule it becomes obvious only in cultures. It is always associated with large numbers of other microbes, and may be looked upon merely as evidence of gross contamination of the pleural contents. The anaërobic diphtheroid is a small short granular bacillus growing in dense clumps, and so far we have succeeded in growing it only in liquid media. Its exact nature is so far undetermined. It does not produce gas, and would appear to be relatively harmless.

Aërobes.

It is with organisms of the *subtilis-mycoides* group that confusion may arise in diagnosis of anaërobo infections by the film method, for there are no morphological features which distinguish organisms of this type from the real anaërobes. They are stout Gram-positive, anthrax-like bacilli, which may give streptobacillary and filamentous forms and may also form spores, all of which features are common to one or other of the anaërobes. Eleven out of 195 septic cases showed sporing anaërobes of this type; in four of these the infection, which might have been an accidental contamination, was pure with a relatively mild clinical course; in five the combination of aerobic and anaërobo existed, while in the remaining two a streptococcus was found in addition to the sporing aerobic. The chance of confusing aerobic with anaërobo is therefore small, and one may take it that the presence of stout anthrax-like Gram-positive bacilli in a film is very strong presumptive evidence of anaërobic infection.

Anaërobes.

A short thick bacillus occurring singly or in pairs with occasional short chains is likely to be *B. perfringens*.

Streptobacillary and filamentous forms are common to a number of anaërobic forms, so that these morphological features are as a rule of little value as a means of differentiation.

The presence of sporing forms in a direct film bespeaks *B. sporogenes* or aerobic spore-formers. Out of eighteen infected haemothorax fluids proved to contain *B. perfringens* on culture, only one answered the spore test by heating to 80° for twenty minutes. In the bulk of cases, then, *B. perfringens* is present only in the vegetative and not in the sporing form.

It will be evident from these facts that the examination of morphological features in a film is never sufficient for the identification of these stout Gram-positive bacilli, and that the method must be supplemented by cultural tests.

B. Cultural Examination.

Cultures have been made from every specimen of haemothorax fluid sent to the laboratory. Implantations of varying amounts were made into plain broth, and on to agar either in tubes or in plates. These were found to be sufficient to grow any aerobic organisms met with, for the richness of the original sample in protein and in haemoglobin content make it unnecessary to use any special media containing these substances.

Anaërobic cultures were made by inoculating freshly sterilized milk under paraffin oil.

The investigation of the anaërobic forms in haemothorax fluids was carried just sufficiently far enough to come to a determination of the main features of the organisms, for it was impossible in the routine of work to track each anaërobe down separately. The bulk of the infections were multiple—that is, there existed together several anaërobic, or aerobic and anaërobic, the combination of *B. perfringens* and a streptococcus being the most frequent.

The following simple scheme was found to give information which placed the organisms met with in certain more or less clearly defined groups.

I. The *B. perfringens* Group.

If one accepts the definition of *B. perfringens* as being a stout Gram-positive non-motile anaërobic bacillus, which gives stormy fermentation of milk, and which does not form spores in milk, then the definition groups together a number of organisms which have been described from time to time by different authors under different names. The question of the kinship and identity of these organisms cannot be here discussed. The group comprises:

1. *B. aerogenes capsulatus* of Welch and Nuttall; synonym, *B. welchii*.
2. *B. perfringens* of Veillon and Zuber.
3. *B. enteritidis sporogenes* of Klein. This was probably a mixed culture of a sugar-splitting non-motile bacillus of the *perfringens* type, together with a motile proteoclast akin to *B. sporogenes*.
4. Bacillus of articular rheumatism of Achalmé.
5. *B. pleuropneumoniae emphysematosa* of Fraenkel.
6. *B. saccharobutylicus immobilis* of Schattenfroh and Grassberger.

The primary milk culture inoculated with a pipetteful of haemothorax fluid is examined after twenty-four and forty-eight hours' incubation. The development after twenty-four hours of stormy fermentation, indicated by strong coagulation followed by explosive disruption of the casein clot, is taken as evidence of *B. perfringens*. There is no other anaërobe that produces this effect with such vigour under suitable conditions. In the case of an indefinite or delayed reaction in the primary culture, one or two subcultures into milk may serve to dispel any doubt. Out of 87 specimens of haemothorax fluid proved to be infected with anaërobic forms, 71 specimens, or 81.6 per cent., gave this typical milk reaction, and were thus shown to contain *B. perfringens*.

The organism produces no digestion of the casein clot formed from ordinary milk. If naked-eye signs of digestion are present, then some other organism, aerobic or anaërobic, is present in the cultures. It never spores in milk, and, indeed, most cultures in milk die in about a week or ten days. A subculture on alkaline minced meat gives spores after several days' incubation, and the medium becomes bright red in colour. There is never any blackening of the meat.

II. The *B. sporogenes* Group.

A pipetteful from one of the original milk cultures taken after three or four days' incubation is heated to 80° for twenty minutes. By this means all vegetative organisms are killed, including *B. perfringens* and all the aerobes except those that spore. The organisms that spore in milk survive and are transferred to a second milk culture. A slow digestion and a clearing of the milk from the cream surface downwards is due to *B. sporogenes* or some near relative of this organism. *B. sporogenes* gives rapid digestion and blackening in meat cultures.

This is the same motile microbe that has been described by recent English writers as *B. oedematis maligni*. It was actually found in 14 out of the 87 anaërobic infected cases, in 9 of which it was associated with *B. perfringens*. Its real incidence was probably much greater, because the investigation of a haemothorax often ceased on finding the stormy fermentation of the primary milk cultures.

III. The Third or Miscellaneous Group.

This group comprises the so-called *B. von Hibler IX*, which was found once in combination with *B. perfringens* and *B. sporogenes*; *B. aëroferditus* of Weinberg, found once only; and nine other organisms which were not identified and could not be grouped in either the *perfringens* or *sporogenes* category.

We were not able to recognize any particular clinical features as being characteristic of one or other of these forms of bacillary infection, doubtless because the infections within the chest usually contained more than one member of the group.

An investigation of the biochemical reactions of a number of pure strains of *B. perfringens* and *B. sporogenes* in culture media has been made by us in conjunction with Captain C. G. Wolf, R.A.M.C., and this has given some indication of the effects produced by these organisms individually in a haemothorax fluid. *B. perfringens* is characteristically a carbohydrate splitter. The gas it produces in a haemothorax cavity is derived chiefly from the glucose present in the original extravasated blood and in the superadded serous effusion. The amount of gas produced by a strain of *B. perfringens* from any given fermentable sugar varies considerably from time to time under artificial cultivation.

Under favourable conditions, growth in milk—that is, in a lactose-containing medium—will result in the formation of an amount of gas which may be more than twice the volume of the original culture medium, and will actually develop in twelve hours' incubation a pressure of two atmospheres, or more than 60 feet of water in the containing vessel. So high a pressure as this is never reached within the pleural cavity of a living man. The highest that we have actually measured was +15 to 20 cm., that is about 30 cm. of water above the normal intrathoracic pressure, or an increase of one-thirtieth of an atmosphere. The pressure may be so great as to drive back the piston of a glass exploring syringe and force it clean out of the barrel. The gas burns with a pale blue flame, and consists of a mixture of hydrogen and carbon dioxide, the proportions varying at different periods of the fermentation, but being approximately as two to one. Neither methane nor nitrogen are found in it.

The odour of the gas is due to the presence of traces of sulphuretted hydrogen and of certain volatile bodies produced in the degradation of the protein molecule. *B. perfringens* by itself is a feeble protein splitter in cultures, and never produces sulphuretted hydrogen or any offensive odour under these conditions. The smell results from a proteolytic destruction that in itself is not associated with the liberation of much gas, and hence one may meet with a most offensive and deeply haemolyzed fluid in cases where there is but little free gas (see Case iv).

In cultures outside the body these odours are met with only during the growth of protein-splitting organisms such as *B. sporogenes*, or where *B. perfringens* is mixed with other organisms. But with an infected haemothorax the gas is found to be malodorous even when bacteriological examination reports only the presence of *B. perfringens*. The explanation of this smell may be either that it proves the haemothorax in all such cases to be infected, in addition to *B. perfringens*, with some other anaërobic bacillus

that was not recognized in the rapid method of examination employed; or that *B. perfringens* can exert within the chest a proteolytic action of a nature which cannot be reproduced under the conditions of outside cultivation.

Whatever be the explanation of this difficulty, the essential fact remains that the gas is produced chiefly from carbohydrates, and, so far as that origin is concerned, is odourless, though it may be tainted by other substances derived from a bacterial proteolysis that does not liberate much gas. *The smell and the gas are separate results of an anaërobic infection of a haemothorax, and they may appear separately.*

Sulphuretted hydrogen is an extremely poisonous substance to man and it is very soluble, but it appears improbable that this malodorous body is ever produced in sufficient quantity to cause a fatal intoxication. The harmful effect of such gas as is evolved is chiefly mechanical.

The retardation of development of anaërobic after implantation in a haemothorax is such a striking feature clinically that it continually excites the hope that a fuller knowledge of the processes concerned in this resistance to the general dissemination of the bacilli may give the desired means to arrest once and for all the spread of infection from the original focus.

Laboratory experiments show the following facts:

1. Attempts to grow *B. perfringens* either in fresh whole blood, or in serum, or in old sterile haemothorax fluid, give only negative results, or meet with very scanty success.
2. If fresh serum be altered by the previous addition of trypsin to it, a medium is produced which is much more favourable to growth.
3. Blood serum obtained from the body at varying intervals after death is a medium in which *B. perfringens* will grow vigorously and almost without fail.

Therefore it follows that the cultures fail with fresh blood either because there is some body in it which inhibits growth, or because the food substance necessary for development is not presented in an accessible form. The general question has been discussed by Sir Almroth Wright,¹ in an attempted explanation of the "avalanche" growth that bursts forth when the necessary conditions are established.

Measurements made by Captain Wolf show that sterile haemothorax fluid of ten or twelve days' standing is slightly more alkaline than normal blood, and that there may be no acidosis in a fluid in which *perfringens* growth is proceeding vigorously. The nature of the facultative change is still unknown. In so much as a man, when his haemothorax is found to be sterile in its fluid part, may exhibit the clinical features of infection on the third or fourth day, it is certain that the bacilli must be growing very actively in some sheltered focus and liberating digestive products of sepsis. Presumably these products diffuse into the general pond of the fluid and alter its properties in such a way that the bacilli are enabled to follow and establish themselves abundantly.

GENERAL CLINICAL FEATURES OF THESE INFECTIONS.

The clinical features of these septic cases may be classified under three main headings:

1. Those indicating the general toxic action on the patient of the septic substances produced.
2. Those caused by inflammation of the pleural cavity.
3. The special physical signs within the chest.

Usually within three or four days after having received a wound in the chest which does not carry deep infection with it, the injured man has passed into a condition of relative ease. His dyspnoea becomes progressively less urgent; the temperature approaches normal by a smooth curve of decline, and the pulse falls to below 90. He sleeps well and his appetite returns. In every respect he is rapidly regaining the appearance of a healthy man, and each day marks an obvious step forward in that direction.

But when sepsis is present in a haemothorax the patient does not mend, and day by day each feature of uneasiness suffers increase. Sleep is broken and uncertain; the appetite fails; pain and tenderness in the affected side of the chest are complained of, and the breathing becomes more dyspnoeic; the cheeks may develop a hectic flush,

and an obvious icteric tint appear in the conjunctivae. Together with these changes the face assumes a slightly anxious expression.

Restlessness, amounting to actual light delirium, by day as well as night, is noted in the most serious infections, of which it is often a very early and suggestive feature. Such delirium has no relationship to pericarditis.

The tongue may be tremulous and lightly coated with fur, though usually it shows so little change that one is often justified in the conjecture that a particular case of septic haemothorax is caused by anaërobic because the tongue is so unexpectedly clean. Other forms of infection produce either a heavy fur, or, as especially with the streptococci, a dry brown surface.

The temperature is always raised, but sometimes to so slight a degree that on this count alone it would appear idle to suspect sepsis. Indeed, the temperature is apt to be a most misleading guide if too exclusive reliance is placed upon it in searching for evidence of infection.

Increase of the pulse-rate is of greater importance. Tachycardia above 110 is rarely seen with a simple haemothorax in a patient who is free from other complications. Case VII illustrates the value of a rapid pulse-rate in raising the suspicion of sepsis in a case with very mild pyrexia. A low pulse-rate and slight fever are never seen combined in a case of anaërobic infection. On the other hand, severe infection and high fever may coexist with a pulse-rate not exceeding 90. (Cp. Case XII.)

The development of *jaundice* should be looked for with the closest attention. A sterile haemothorax may cause a slight conjunctival icterus, but nothing more. Real jaundice, especially if associated with epistaxis, has, without exception, in our experience been an index of a particularly severe type of infection by anaërobic; and not an hour should be lost in dealing with the patient when once this yellow tint has appeared.

Vomiting never occurs. Severe diarrhoea, of the offensive and uncontrollable type that may appear in any subject of old foul wounds, may be noted in patients with empyemata that have existed for many days without recognition, but it is not an early feature.

The *pleural inflammation* within the chest on the side of the haemothorax is shown by an increase in the size of the effusion and by local tenderness and pain. In most cases this pain is mild and needs inquiry to reveal it; but in a few it produces continual distress, and may even lead to a referred tenderness over the hypochondrium and abdominal rigidity, so that the surgeon is tempted to an exploratory laparotomy on the supposition that the missile has passed from the chest into the abdomen.

In cases where there is practically no haemothorax, and the infection is spreading from a focus around a splintered rib, coarse pleural friction will be heard on the side of the developing empyema. But usually it is fruitless to search on the injured side for pleural friction as a sign indicative of sepsis. On the opposite side a coarse pleurisy often develops from spread of the infection across the chest, and its recognition should always emphasize the need for exploration of the haemothorax.

Oedema of the contralateral base of the lung is also a feature suggestive of sepsis; but contralateral massive collapse may occur in either sterile or septic cases equally, and is chiefly the expression of a thick purulent secretion encumbering the airway of the bronchial passages.

Pericarditis develops in a small proportion of these infections, and is usually fatal before the effusion has attained a large size. But friction sounds heard over the cardiac area are not in themselves to be interpreted as being of invariably bad prognosis. They may be caused by a dry pericarditis, which slowly disappears in the same way as does the dry contralateral pleurisy; or they may simply be the trivial and transitory sounds that are occasionally produced from leakage of air from the lung into the mediastinal tissues. Speaking generally, there is no special form of pericarditis associated with the anaërobic infection of the haemothorax. That which develops is similar to what is found in other forms of infection, and as a matter of fact it is generally due to streptococci or other organisms that were associated with the anaërobic bacilli in the haemothorax. The case of gaseous pneumo-haemopericardium, which was so successfully dealt with by Major Little Jones,² was a rarity of which we have never seen an example in over two hundred autopsies on chest wounds at base hospitals. Occasionally frothing bubbles of gas

have been observed in a semipurulent pericardial effusion associated with anaerobic septic haemothorax; but the gas was not abundant, and there was no reason to believe that it was not entirely a *post-mortem* production. No example of a large pyo-pneumopericardium has been found by us at autopsy.

The special physical signs on the side of the haemothorax are simply those caused by the *development of gas* within it. This gas may be loculated within the haemothorax, or it may collect freely above the fluid in the pleural sac. In the latter case the signs are indistinguishable from those of an ordinary exogenous or air pneumo-haemothorax; but the gas may be formed so abundantly that the heart suffers continually greater displacement, and the side of the chest may sometimes be bulged out under the internal pressure. (Cp. Case III.) In aspirating a traumatic air pneumothorax, we have never found a pressure greater than that of the atmosphere; but in these inflammatory gas collections the pressure may be as high as +15 or +20 cm. of water. The fluid below the gas is mobile, and its horizontal level of percussion dullness shifts when the patient is raised from the lying to a sitting posture. The anvil ("bell") sound is often yielded by the coin test.

The loculated collection of gas is almost in itself a diagnostic feature. Percussion reveals a resonant area, often with a low-pitched note that is almost "cracked-pot" in character; within the boundaries of the area of dullness that demarcate the haemothorax. The level of the latter does not change with posture, for the collection is not freely mobile. The gas producing the note is usually loculated at the back, somewhere near the angle of the scapula. It is extremely rare to meet with a similar collection of ordinary air that has escaped from a lacerated lung into a haemothorax and remained imprisoned so low down. Consequently, whenever this peculiar percussion note is met with in a haemothorax, where the cardiac displacement seems unusually great in proportion to the area of dullness and the physical signs are difficult to interpret, gas infection should be suspected, and tested for by exploration at the lower limit of the resonant area.

But there is one fallacy to be guarded against. The diaphragm on the wounded side often takes up an abnormally high position as an immediate result after the injury of some paralysis of the respiratory movement. It may be sealed up in this position by adhesions and remain elevated despite the increase of intrapleural pressure. Under such circumstances percussion over the base of the chest behind may elicit a tympanitic note from the raised abdominal viscera, on the right side as well as on the left. Therefore the preliminary diagnosis of gas should be accepted only when this abnormal note is found behind at a level above that of the ninth rib.

It is needless to discuss the differential diagnosis from pneumonia or other chest complications. The physical signs often fail to make the diagnosis secure. Safety for the patient is gained by the discarding of these dubious distinctions, by promptness to suspect infection, and by reliance on the exploring needle. In difficult cases an x-ray examination of the chest by fluoroscopic screen with the patient in a sitting posture will often lead to the solution of the problem. When mobile gas and fluid are present in the pleural cavity, the fluid will appear as a dark pool with an upper horizontal level. The surface of the free fluid shows oscillations caused by the heart-beat, and will be violently agitated if the patient coughs. Its level changes in relation to the chest wall when the patient is tilted first to one side and then to the other. Such an appearance is diagnostic of a collection of free fluid below gas or air.

REFERENCES.

¹ *Lancet*, January 6th, 1917, p. 1. ² *British Journal of Surgery*, vol. iv, July, 1916, p. 103.

(To be continued.)

A COMMITTEE of superintendents of public instruction and other officials concerned with education in the United States has recently been formed to further the passage by the legislatures throughout the country of a bill, drafted by Dr. Dudley A. Sargent, the physical education expert at Harvard, for the promotion of physical training in the public schools. The committee believes that "the time has come for the adoption of a definite plan for the preparation of American boys and girls for the pursuits of peace or the vicissitudes of war."

CLINICAL OBSERVATIONS ON DYSENTERY.

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The clinical symptoms of dysentery vary greatly, and rarely do they actually conform with the usual textbook description.

The simple acute bacillary type and the simple acute amoebic type are clinical entities and no difficulty should arise in distinguishing between them clinically. It is impossible to distinguish between the various types of bacillary dysentery without the aid of the bacteriologist. Clinically they must remain in the common group.

Another type of dysentery that has come under my observation during the present war is what might be called an acute mixed type of dysentery. The clinical symptoms are at first those of the acute bacillary type, which after varying intervals merge into those of the acute or subacute amoebic type. This type of dysentery is most important from the point of view of treatment. It is to be regretted that in this note no mention can be made of the bacteriological and microscopical examination of the stools. This should be carried out as a routine measure in every case whenever possible, but the circumstances under which I have observed and treated the majority of my cases have rendered this impossible.

Incidence and Causation.

From several years' experience of active service conditions on the North-East Frontier of India, and during the present war in Egypt, Anzac, and Mesopotamia, I cannot help coming to the conclusion that in the bacillary type the main source of infection is the common house-fly.

It is a remarkable coincidence that during the first few weeks or months in any campaign in which I have taken part the prevailing type of dysentery has been the bacillary type.

During the Abor expedition on the North-East Frontier of India, while the troops were collecting at the base and were engaged in making roads for the advance, in fact were more or less stationary, and the climate was such that flies could live and breed, then the prevailing type was the bacillary type; but when the troops pushed on, had fatiguing marches to do day after day, as the season also advanced and the weather became colder and wetter and the heavy rains seriously damaged the quality of the rations, then the amoebic type of dysentery made its appearance.

Again, during the expedition into the Mishmi country on the North-East Frontier of Assam, as long as the weather was hot and flies were abundant, and as long as the troops were engaged in cutting roads through the dense jungle, and progress was necessarily slow, then the bacillary type prevailed; but as soon as the weather became colder and wetter, and the flies disappeared and the troops pushed on to the hills, then did amoebic dysentery make its appearance. Flies are by no means a heritage of modern civilization, for in this dense jungle area of over sixty square miles, which was practically devoid of any human habitations, flies were as abundant as in an Indian bazaar in the middle of the hot weather.

Deficient sanitary arrangements attract flies and also encourage their propagation, and all that is required with such conditions is a favourable climate, and dysentery is bound to make its appearance.

At the beginning of any campaign, when large bodies of men congregate in limited areas, and before the sanitary details have had time to become properly organized, it is then, as it were, that the seed for future dysentery becomes sown, and it is only with the greatest difficulty and with the most thorough measures that the coming outbreak can be arrested or checked.

Military exigencies must always in every campaign be the first consideration, and a typical example of this occurred during the landing at Anzac on April 25th, 1915, and the following few days. Fighting men had to be landed at all costs and as rapidly as possible. All other branches of the army were during this critical time of secondary importance, and it was only when the position had been captured and consolidated that sanitary and other details could be landed.

During these first few days, when every one was in