of chorea (*Lancet*, 1906). They found, on microscopical examination of three cases of fatal chorea, "very little evidence of embolism," although great hyperaemia was present, with thrombosed vessels in all parts of the brain.

My case is practically another of the same type, as there was neither mitral valve disease nor cerebral embolism. From what I have stated, the embolic theory as causative of chorea—be it chorea major, chorea minor (Sydenham's chorea), or the maniacal type illustrated by my case—can be no longer tenable. Rather must we recognize the difference in the severity of the choreic manifestations to be due to the presence of a toxin, and to the virulence or mildness of its action on the nerve elements of the cerebral cortex and of the lower nervous centres. In direct proportion as the psychical and the higher centres of ideation and volition or the motor centres are those most invaded, so the symptoms each case may present will be either those of mania and other disordered mental states, or those of inco-ordinate muscular contractions and movements of the body and the limbs.

The intimate association of chorea and rheumatism has been long recognized, and has of late years been more and more insisted on, and the consensus of opinion of many *clinical* observers is in favour of there being one common underlying cause for each of them. It is a well-recognized fact that the first indication of rheumatic fever may be chorea, and that myocarditis, endocarditis, or pericarditis may equally occur in either.

Sir Dyce Duckworth,⁷ in his paper read before the Medical Congress at Lisbon last June, emphasized the view that chorea should be considered as "cerebral rheumatism." He has for many years consistently maintained the intimate relationship between the two diseases, and clinical experience tends, with increased authority every year, to confirm the truth of that relationship.

In 1892, Clouston,⁸ writing upon the varieties of insanity, under the heading of "Rheumatic Insanity," describes the typical case of rheumatic chorea with acute maniacal insanity to which reference has been made above, and asks the question, "Was the rheumatism the true cause of the mental symptoms of the chorea or of both?" and asserts his view, from a *clinical* standpoint, that the poison of rheumatism was the true cause of the insanity.

It may be interesting to note, *en passant*, that this view is by no means a new one. It simply reasserts what was propounded in 1850 by M. Germain Sée.⁹ His expression is, "that chorea was due to the rheumatic diathesis acting on the brain and spinal cord." Trousseau,¹⁰ who quotes Sée's view, and acknowledges its probability, did his best to refute it and to prove that chorea was a purely functional derangement, similar to hydrophobia and tetanus. The ground upon which he based his theory was that no gross evidences of inflammation of the brain or its membranes are present in fatal cases of any of these diseases.

It is curious to see that now, in these latter days, the older views—based on clinical observation—have again gained ground, and that the keen and exhaustive researches of scientific minds, combined with highly skilled bacteriological dexterity and acumen, have shown that the pathological conditions of these so-called functional nervous diseases are really due to microbic infection and toxic influences. The first ray of light which illumined this difficult subject—after which so many thoughtful physicians were groping in the dark and feeling after the truth, if possibly they might find it—was the discovery by Wassermann, in 1899, of a diplococcus which he had isolated from the cerebro-spinal fluid and inflammatory exudations of a patient who had died of acute rheumatism and chorea. With this diplococcus he had experimentally inoculated rabbits and produced in them polyarthritis.

Drs. Poynton and Paine, in 1900, confirmed the observations of Wassermann, and found the diplococcus in a case of chorea. In 1905 they reported another case of fatal chorea, in which they demonstrated its presence. Isolating the diplococcus from the cerebro-spinal fluid, they had produced by inoculation arthritis, endocarditis, and pericarditis in a rabbit.

In a valuable paper¹¹ Dr. Beattie accepts the theory of a bacterial origin of rheumatic fever, and deals with the

objections raised against it, while he calls the organism *Micrococcus rheumaticus*.

It is not my intention to follow further this most interesting branch of the subject, but would refer to the lucid and convincing contribution on the pathology of chorea which Dr. F. J. Poynton and Dr. Gordon M. Holmes published in the *Lancet*, October 13th, 1906, and from which I have already freely quoted above. These observers found the diplococcus in the brain and its membranes, and also in the diseased valves of the heart, in 3 fatal cases of chorea and rheumatism, and they assume that the presence of micro-organisms goes far to prove that these inflammatory pathological changes are due to a toxin derived from these micro-organisms, and that chorea is caused by the action of bacterial poisons on the brain.

"The conclusions they come to, in their research on this subject, in the first place support the clinical evidence that chorea is a manifestation of acute rheumatism; and secondly, that the demonstration of diplococci in each of their three cases is a striking confirmation of the assertion that the *Diplococcus rheumaticus* is the infective agent in acute rheumatism."

My case does not exactly add support to the views just stated, in so far as no micro-organism was discovered by Dr. O'Sullivan in his careful bacteriological examination of the brain and spinal cord. I have, however, felt it my duty to record it, as a contribution to the literature and clinical history of this most interesting subject, since it is only by the accumulated records of cases of chorea, in which a full and reliable pathological report is made by capable bacteriologists, that a sure foundation can be laid and a true pathology erected.

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ON THE VALUE OF AN ABNORMAL RISE IN THE AVERAGE LEVEL OF THE DIAPHRAGM AS A GUIDE TO THE VOLUME OF THE BLOOD IN ACTIVE CIRCULATION.*

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THE object of this paper is to bring forward clinical evidence in favour of the following propositions:

1. When, from any cause, the total volume of the blood in circulation is materially diminished, the total bulk of the intrathoracic viscera is correspondingly diminished by the relative emptiness of the thoracic blood vessels, especially those of the lungs. This diminution in the bulk of the intrathoracic contents shows itself by an elevation of the diaphragm, which has to be maintained at a higher average level than the normal in order to adjust the cubic content of the thorax to the altered volume of its contained viscera.

2. That such a rise of the diaphragm may frequently be detected clinically, and, when present, is of value in diagnosis, and affords important indication for treatment.

NORMAL POSITION OF THE DIAPHRAGM.

The position of the diaphragm can be recognized clinically by ascertaining the upper level of the gastric resonance in the left nipple line, or of the upper border of the liver in the right nipple line. In both cases percussion is best done with the patient recumbent, and in the case of the gastric resonance the recumbent posture is essential to accuracy, because in the erect posture the gaseous contents of the stomach, if scanty, may rise up into the arch of the diaphragm, and the dullness of the liquid contents of the stomach be continuous with the dullness of the left lobe of the liver and the heart.

Of the two methods named, the percussion of the gastric resonance is easier and more reliable for the following reasons:

1. There is much variation in the upper level of the liver dullness of healthy individuals, dependent upon

* A paper read before the Birmingham Branch of the British Medical Association.

the degree to which the lungs have been expanded by exercise or athletics, etc.

2. The respiratory movements of the lower edge of the lung render exact accuracy of record as to its level impossible.

3. The level of the diaphragm on the right side is more apt to be interfered with by ill-defined pathological conditions of the lung than is the case with the level of the central tendon which underlies the heart.

Gastric Resonance.

For the study of the gastric resonance it is necessary, as before mentioned, for the subject to be lying on his

Upper Level of the Liver Dullness.

Although the percussion of the upper border of the liver is of less value than that of the stomach, where exact observations are desired upon the level of the diaphragm, it nevertheless gives us important information as to the condition of the diaphragm, for its elevation above the normal level may be very great when the diaphragm is high. Thus in one patient (Case XI) there was distinct relative liver dullness up to the middle of the third rib in the right nipple line.

The percussion of the upper border of the liver dullness is also of considerable value, because it often gives an indication as to the amount of arching of the diaphragm

Fig. 1.—Case I: Normal chest.

Fig. 2.—Case II: Collapse of lung. Diaphragm high on left side only.

Fig. 3.—Case III: Collapse of lung. Very high left diaphragm.

back, so that any gas in the stomach may come to the front and give a contrast with the dullness of the left lobe of the liver and the heart, which lie above it.

The determination of the area over which the gastric resonance is obtainable is greatly assisted by listening to the area over which "reverberation" can be heard when some continuous sound is communicated to the air in the stomach. This is simply done by means of a short rough bone rod, which is pressed over the stomach area and scratched regularly with the finger-nail. The echo, similar to the bell sound in pneumothorax, is clearly heard over the whole gastric area, but is sharply limited to it.

As a rule the results obtained by percussion and by

present, if the distance between the level of the absolute dullness and that of the relative liver dullness be carefully observed, and the changes during respiration noted. Thus a wide area of relative dullness suggests an abnormally arched diaphragm, and a sudden change from full lung resonance to absolute liver dullness suggests a flatter diaphragm, as in emphysema.

This abnormal wideness of the area of relative dullness indicating a highly-arched diaphragm is well shown in some of the following diagrams—namely, Figs. 6, 10, 19.

Observation of the Lower Edge of the Liver.

The careful mapping out of the lower edge of the liver dullness is also of importance in the study of the level of

Fig. 4.—Case IV: Gastric ulcer. March 7th. Before limitation of food.

Fig. 5.—Case IV: March 13th. After 6 days of rectal feeding.

Fig. 6.—Case IV: March 15th. More food given; heart larger.

noting the area of gastric reverberation coincide very closely with each other.

By these methods the normal level of the upper limit of the gastric resonance is found to be in the sixth left interspace. It sometimes in health reaches the upper border of the sixth rib, but, as a rule, when it reaches the fifth interspace it must be considered pathological. Only once or twice have I found well-marked gastric resonance in the fifth interspace in a person who was apparently in perfect health, and under circumstances which pointed to its being the normal level for the individual concerned.

the diaphragm, because as the diaphragm rises, the rise of the lower border of the liver dullness is often more marked than the upper. It is not uncommon to find in such cases a total disappearance of the absolute dullness of the liver, internal to the right nipple line, accompanied by a marked rise in the lower edge of the liver, sometimes to the extent of 2 in. or more above the costal arch. This change is well shown in Figs. 5, 10, 17, 19. It seems as if the liver slips back into the hollow of the diaphragm, and its lower anterior edge is thus drawn up and back so much that it barely comes in contact with the ribs anteriorly at all; and the intestinal or gastric resonance meets the lung

resonance over this area. This strange change in the shape of the absolute dullness of the liver will sometimes develop in a week or so, and then as speedily give way to the normal relationship of the liver dullness to the costal arch, when the conditions causing the ascent of the diaphragm subside (Case IV).

SCOPE OF SUBJECT.

Though this paper does not deal with cases in which the level of the diaphragm is altered by some obvious physical cause, such as the pressure of distended abdominal viscera, or the traction of a collapsed or fibrosed

CASE III (EXTREME ELEVATION).

There was complete non-expansion of the left lung (Fig. 3). The right lung crossed over, and its left border could be detected just internal to the left nipple line (2½ in. from the sternal edge) on expiration, and during full inspiration it crossed to beyond the nipple line (more than 4 in. from the left edge of the sternum) in the second, third, and fourth interspaces. This shows the extreme shrinkage of the left lung. The diaphragm also was correspondingly high, as judged by the level of the gastric resonance.

The expansion of the right lung pushing down the heart prevented any great rise of the diaphragm in the nipple line, and it was only at the fifth interspace; but in the axilla there was full gastric resonance to the posterior axillary line in the

Fig. 7.—Case IV: March 19th.
Food increased.

Fig. 8.—Case IV: March 29th.
Transient cardiac dilatation.

Fig. 9.—Case IV: March 30th.
Practically normal.

lung, nevertheless, it may be well to say a few words as to the changes which take place when the left lung shrinks from healing tuberculosis, or remains unexpanded after compression by extensive pleurisy. In these cases we know that the diaphragm is drawn up by the lessened size of the left lung, and as the changes which take place in the level of the gastric resonance are precisely those which occur in the cases dealt with in this paper this may be taken as evidence in favour of the points raised.

I give the notes and figures of two such cases, one moderate and one extreme.

CASE II (MODERATE ELEVATION).

The heart is drawn up a little to the left by the rise

sixth space, 8 in. from the middle line. Its lower border was in the eighth space.

HIGH DIAPHRAGM IN ENTERIC FEVER.

Although, in common with all those who carefully percuss the cardiac dullness, I have long been conscious that there is occasionally abnormal resonance below the cardiac area, I did not give the subject special attention till the following case brought it forcibly under my notice:

A man suffering from an extremely severe attack of enteric fever presented very great asthenia but no complications of any sort until the end of the third week. He then became decidedly feebler, although the temperature was not specially high, and the condition of the abdomen was satisfactory. He

Fig. 10.—Case v: Gastric ulcer. January 20th.
After 3 days' rectal feeding.

Fig. 11.—Case v: January 29th.
More food; diaphragm lower.

Fig. 12.—Case v: February 10th.
Diaphragm lower.

of the diaphragm, but there is a marked rise in the gastric resonance (Fig. 2), not only in the left nipple line, but much more so in the axilla. Instead of there being no gastric resonance in the axilla, as in the normal chest, in this case the resonance reached the fourth interspace in the nipple line and extended nearly to the anterior axillary line, 4 in. from the sternum. In the fifth space it reached 5½ in., and in the sixth space there was full gastric resonance and reverberation to the mid-axillary line, 6½ in. from the sternum; the lower border was at the eighth rib.

This condition developed on the rapid absorption of pleuritic effusion, and in eleven days the upper border of the gastric resonance had returned to the normal level, and there was none in, or external to, the anterior axillary line.

had had no diarrhoea. This asthenia was suggestive of the virulence of the poison he was absorbing, but as his condition became worse it was evident that some other cause was at work.

The abdomen from being moderately full became morbidly retracted, and his face and limbs gave the appearance of extreme emaciation. This rapid change in his appearance was evidently due to lack of liquids in the body. He was taking three pints of milk daily, besides some water. I tried the effect of a subcutaneous saline injection of half a pint or more; it was rapidly absorbed, but as rapidly excreted by the kidneys, and the patient's condition was in no way improved. There was a peculiar expiratory type of dyspnoea, which I have now learned to associate with this condition, and also a decided

elevation of the diaphragm, the gastric resonance reaching well into the fifth space, and the liver being decidedly above its usual level in the right nipple line. He died within two or three days of these symptoms becoming noticeable.

Post mortem the diaphragm was at the third rib on the left and at the fourth rib on the right side; the heart was small,

Fig. 13.—Case VI: Haematemesis.

its lower border at the fourth interspace, and all the organs dry and bloodless.

In thinking over the cause of death, I was forced to the conclusion that death was practically due to starvation, owing to the interference with intestinal absorption,

case, because I am careful to guard against such malnutrition.

HIGH DIAPHRAGM IN LIMITATION OF FOOD.

If the above suggestion were accurate and a diminution in the intake of nutritive material would, if carried to excess, cause a diminution in the total volume of the blood, and show itself by a shrinking of the thoracic contents, the same phenomenon of high diaphragm ought to be found where semi-starvation is adopted as a therapeutic measure; for instance in the treatment of gastric ulcer.

For many years I have noticed that as a rule in cases of gastric ulcer there is a marked rise in the diaphragm after some days of strict limitation of food, as where the patient is fed only by nutrient enemata.

The amount and the duration of the rise varies much in different cases. In some the gastric resonance will, after a few days of rectal feeding, rise into the fifth interspace, or even into the fourth, and as rapidly return to the normal level on the resumption of full feeding. In others the rise may take place more slowly, attaining its maximum height after the cessation of rectal feeding, and only returning to the normal level after the patient has been on ordinary diet for some time.

The accompanying table of measurements (p. 982) shows an unusually rapid rise and fall of the diaphragm in such a case.

HIGH DIAPHRAGM AFTER SEVERE HAEMORRHAGE.

Closely allied to these cases are those where, after a sudden severe haemorrhage, the diaphragm may be

Fig. 14.—Case VII: Anaemia. High diaphragm and dilated heart.

Fig. 15.—Case VIII: Anaemia. February 15th. Diaphragm very high in axilla.

Fig. 16.—Case VIII: March 29th. Nearly normal.

which resulted from inflammatory changes in the mesenteric lymphatics, and my explanation of the case was as follows:

This defective absorption from the intestines, coupled with the tissue waste due to the fever, would presumably in course of time lessen the proteids in the body to such a degree that the total volume of the blood would have to diminish in order to prevent it from becoming too dilute. This diminution at last became so extreme that the proper nutrition of brain and heart was no longer possible, and the patient died. The explanation of the elevation of the diaphragm which appeared to be most probable was that the diminution in volume of the circulating blood caused such a lessening in the size of the heart and such emptiness of the pulmonary artery and great vessels, that the volume of the heart and lungs was so materially diminished that the diaphragm had to rise so as to diminish the cubic content of the thorax to a corresponding extent.

I have had some confirmation of this explanation of the preceding case, for I have on several occasions seen the same symptoms appearing in severe cases of enteric fever. When I have detected the peculiar sighing breathing and the elevation of the diaphragm, associated with signs of lack of fluid in the body, I have been able at once to remove the dangerous symptoms by the administration of peptones and of milk sugar, the absorption of which takes place through the portal vein and not through the intestinal lymphatics.

It is now some years since I have seen such a

found to be an interspace or more above the normal level.

High diaphragm, if looked for, can very often be found in such cases as are blanched by severe haemorrhage, and the gradual return of the diaphragm to the normal level can be observed as the patient recovers (Fig. 13).

Fig. 17.—Case IX: Neurasthenia.

HIGH DIAPHRAGM IN ANAEMIA.

In anaemia, from causes other than loss of blood, the same phenomenon of high diaphragm, though by no means constant, is very frequently noticed. In a series

Details as to the Rise and Fall of the Diaphragm in Case IV.

Date.		Stomach Level.	Upper Border of Liver Dullness.			Lower Border of Liver.	
			Absolute.	Relative.	Width of Relative Dullness.	Situation.	Distance from Costal Arch.
March 7	Fed entirely by the rectum	6th rib (U.B.)	5th rib (L.B.)	4th rib (L.B.)	1½ in.	—	1½ in.
„ 13		5th rib (U.B.)	5th rib (Mid.)	4th rib (Mid.)	—	—	2 in.
„ 14		4th rib (L.B.)	—	—	—	—	—
„ 15	1 oz. per hour by mouth	4th rib (L.B.)	5th rib (L.B.)	4th rib (U.B.)	2 in.	7th rib (L.B.)	2½ to 3 in.
„ 18	Low diet; enemata stopped	6th rib (L.B.)	5th space	5th rib (L.B.)	1½ in.	8th rib (Mid.)	1½ in.
„ 19		—	—	—	—	—	—
„ 23	—	6th rib (L.B.)	6th space	5th rib (Mid.)	¾ in.	9th rib	At C.A.
„ 26	Fish diet	—	—	—	—	—	—
„ 30	—	6th rib (L.B.)	6th rib (Mid.)	5th rib (L.B.)	—	9th rib (L.B.)	Below C.A.

Explanation.—U.B., Upper border. L.B., Lower border. Mid., Middle. C.A., Costal arch.

of consecutive observations upon cases of anaemia and chlorosis I found a well-marked rise of the diaphragm—that is, to the fifth interspace, or higher—in about one-third of the cases; but I was not able to determine the exact reason for the difference in this respect without a fuller investigation into the blood changes present in each case than the time at my disposal permitted.

In true chlorosis it is supposed that the volume of the

that in some of the cases, at all events, the displacement is simply due to the rise in the diaphragm.

HIGH DIAPHRAGM IN NEURASTHENIA.

In many other diseases a high diaphragm may occasionally be met with, but in cases of neurasthenia it is of such relatively frequent occurrence as to raise the question whether in this condition some of the chemical

Fig. 18.—Case x: High diaphragm in sudden cardiac failure.

Fig. 19.—Case xi: Pericarditis. November 20th. Very high right diaphragm.

Fig. 20.—Case xi. December 8th. Normal again.

blood is, if anything, increased; and if this be proved to be so, the observation of the level of the diaphragm may prove of diagnostic value in these cases.

The reason for the rise of the diaphragm in these cases cannot be definitely stated. It may be the result of deficiency of proteids, or it may be due to deficiency of iron, or some other necessary constituent of the blood acting in the same way as deficiency of food acts.

This hypothesis may be expressed in general terms by saying that the total volume of the blood diminishes because there is a deficiency of some one or more of the essential constituents of the blood, and therefore the total volume of the blood in circulation must be lessened in order to keep the percentage of that substance at its irreducible minimum.

In these cases of anaemia the area of cardiac dullness is, as a rule, larger than in the cases previously mentioned. This is due to the frequent presence of dilatation of the right ventricle. As it involves the conus arteriosus of the right ventricle and enlarges the heart upwards and to the left, it increases the area of the cardiac dullness to the left of the sternum, sometimes to a very considerable extent.

In connexion with these changes in anaemia there is one result of high diaphragm which may have been misinterpreted in the past—namely, the displacement upwards of the apex beat. In papers I have written and seen upon this subject this upward and outward displacement of the apex of the heart in anaemia has been explained by changes in the heart itself. I am now sure

or organic constituents of the blood are not deficient in amount.

A case of a somewhat similar nature is that of a labouring man who was sent to me as a possible malingering, because he looked comparatively hale and hearty, but nevertheless complained of great lassitude and inability to work. His colour was good; there was no complaint except of a little atonic dyspepsia. Physical examination showed nothing except a high diaphragm, the gastric resonance reaching to the fourth left interspace and the absolute dullness of the liver being absent internal to the right nipple line (see Fig. 17). There were some symptoms which suggested the possibility of deficiency of thyroid secretion, and treatment by thyroid extract entirely cured him.

COMMENTARY.

We must now discuss the explanation of the phenomena described and illustrated by the diagrams given.

Is it right to conclude that elevation of the gastric resonance on the left side and of the liver dullness on the right side implies certainly an elevation of the diaphragm? As the phenomena described, though less marked, are still present in the erect as well as in the recumbent attitude, and as the rise in the gastric resonance can usually be noticed far back in the axilla as well as in front, an affirmative answer must be given. The diaphragm is high in such cases.

Granted, then, that the diaphragm is high in the cases of which we have been speaking, what is the explanation of its rise in this marked manner?

It must either be drawn up by suction from above, or

pushed up by pressure from below; for we know of no power inherent in itself whereby the diaphragm could rise and compress the thoracic viscera.

But of these two forces it is easy to eliminate pressure from below as a cause of the elevation of the diaphragm. In all these cases the abdomen is empty or normal in appearance, and there is certainly no increase of intra-abdominal pressure.

Then, again, it cannot be due to some peculiar action of the stomach, because the rise is bilateral, involving the liver as well as the stomach. Moreover a glance at the diagrams will show that the lower level of the stomach is often raised as well as the upper border, and I have never in these cases noticed any prominence of the stomach in the epigastrium such as is always noticeable when the stomach is sufficiently distended and tense to exert pressure on the thoracic viscera.

Moreover, the pitch of the reverberation note as heard over the stomach is not of the high and varying character noticeable where the organ is distended and undergoing contraction.

We must therefore conclude that the diaphragm is drawn up as the result of some change in the intrathoracic pressure.

What intrathoracic force can do this?

Now although there is a danger of error in reasoning when a process of exclusion is used, we may, I think, resort to it in this instance.

The thorax contains practically only four things whose variation could materially alter the volume of the thoracic contents: the lung tissue, including the bronchi; the air in the lungs and bronchi; the heart and blood vessels; and the blood.

Now some of these variable factors are easily excluded. (1) There can be no abnormal suction exerted by the air in the lungs, as there is no mechanical interference with the entrance or exit of the air from the thorax. (2) The actual bulk of the lung tissue is not changed; the lungs in the cases spoken of are healthy.

The agency causing elevation of the diaphragm must, therefore, be either a variation in the cardiac and vascular tissue, or in the degree of distension of the heart and blood vessels in the thorax (including those of the lungs) with blood. As the cardiac tissue is practically constant in bulk from day to day, the factor we are searching for must consist of a lessening in the amount of blood contained in the lungs, heart, and great vessels in the thorax.

When we consider how large a proportion of the lung tissue consists of capillary vessels, how much blood the veins of the thorax and the heart itself are capable of holding, it is clear that a decided diminution in the volume of blood circulating in the thorax might cause a distinct lessening in the thoracic contents, and the space thus left vacant would have either to be filled up by air or by a corresponding lessening of the cubic content of the thorax by means of a rise in the level of the diaphragm. There is, therefore, physiological justification for the theory that the rise in the diaphragm is caused by a lessening in the amount of blood in the thorax.

These theoretical considerations are in accord with the facts of the cases mentioned, for in them there were conditions leading to malnutrition of the body, and presumably also lessened power of regenerating the blood.

One theoretical consideration which follows upon the clinical facts here brought forward, if the explanation given be correct, is that there must be a limit below which the specific gravity of the blood (or the percentage composition of its various constituents) is not allowed to fall. Where there is a lessened intake of food, when the impoverishment of the blood reaches this minimum limit, it can go no further without damage to the organism, and consequently if the lessened intake of food continues the total volume of the blood must be lessened, in order that the minimum limit be not passed.

It is possible that this law of an irreducible minimum may hold for many of the constituents of the blood, and that the high diaphragm of neurasthenia is due to the deficiency in the blood of some chemical substance, or some internal secretion. The case I have already referred to of the labourer who suffered from neurasthenic symptoms, and had a high diaphragm and was cured by the administration of thyroid gland, seems to point towards such a possibility.

HIGH DIAPHRAGM FROM HEART WEAKNESS.

Further evidence can, however, be adduced in favour of this proposition that a lessening of the amount of blood in circulation through the lungs and heart causes a recognizable diminution in their total bulk, and is followed by a rise in the diaphragm.

The volume of the blood circulating through the lungs is not dependent solely upon the total volume of the blood in the body; it is also dependent upon the strength of the heart.

A decided weakening of the heart as a whole might reasonably be expected to diminish the volume of blood circulating through the lungs. If a diminution of the total volume of the blood in circulation causes such emptiness of the lungs and vessels that the diaphragm has to rise so as to adjust the size of the thorax to the diminished bulk of its contents, then this same phenomenon would also occur if a weakening of the heart muscle results in an automatic diminution in the amount of blood in circulation in order to accommodate, as far as possible, the burden to the heart's power of bearing that burden. There is good clinical evidence that this takes place, for marked cardiac asthenia is extremely frequently accompanied by a rise in the level of the diaphragm.

This phenomenon of high diaphragm would not, however, be expected in cases in which cardiac weakness is accompanied by fullness of the veins, such as cases of failing compensation. In cases of valvular disease, nevertheless, the level of the diaphragm ought to be watched, as it may give very important indications.

Such a sudden weakening of the heart with a fall in the strength of the pulse is a well-known sign of the onset of pericarditis, and when this takes place in a young patient whose fibrous tissues have not yet become unyielding a well-marked rise in the level of the diaphragm will be found to be present.

This rise is especially noticeable where failure of the heart muscle from myocarditis or pericarditis occurs as a complication of rheumatic fever, for the rheumatic poison causes a softening of fibrous tissues, allowing the displacement of organs to occur more readily than if rheumatism were not present.

I give an illustration of an extreme case of this type:

The gastric resonance reached as high as the lower border of the fourth rib on the left, and the relative liver dullness reached the upper border of the third rib in the right nipple line, the lower edge of the liver dullness being at the lower edge of the sixth rib (Case XI, Figs. 19 and 20). When the heart recovered, some eighteen days later, the gastric resonance and the liver dullness had both returned to the normal level.

A similar well-marked case was one in which sudden myocardial weakness occurred in a case of influenza complicated with acute colitis.

The heart weakness showed itself by a sudden fainting attack, during which the heart-rate fell to 40, and even to as low as 30 for a short time, if the pulse-rate were to be trusted. The cardiac asthenia was so extreme, that for a fortnight or more the patient dare not raise his arms for fear of bringing on a fainting attack. During this time the diaphragm rose steadily. In spite of the fact that the patient was a young man, with voluminous lungs, the gastric resonance on the fourth day of the illness reached into the fifth left intercostal space, and by the twelfth day reached the middle of the fifth rib in the left nipple line. Similarly, the absolute liver dullness by the twelfth day had risen from the sixth interspace to the lower border of the fifth rib. Accompanying this rise in the diaphragm there was a marked diminution in the cardiac dullness; in fact, it almost entirely disappeared, there being on the twelfth day of the illness no absolute dullness, and only a little relative dullness in the fourth space, internal to the nipple line but not reaching to the sternum.

Here, then, we had marked cardiac asthenia associated with a rise in the diaphragm and diminution in the size of the cardiac dullness.

Within five weeks of this date the gastric resonance was back at the normal level, and there were 4 in. of cardiac dullness in the fourth interspace instead of under 2 in., as on the date above mentioned, and the heart was much stronger. The patient made a perfect recovery, although appendicitis supervened on the acute colitis.

CLINICAL SIGNIFICANCE OF HIGH DIAPHRAGM.

As regards the clinical significance of high diaphragm, the question has of necessity been dealt with in the above discussion of the phenomenon. It is an important danger signal in some cases of severe enteric fever. It is a useful guide as to the extent to which rectal feeding may be

pushed. It must also be borne in mind because in cases of suspected rupture of a gastric ulcer the absence of a liver dullness cannot, as is now well recognized, be relied on as an indication of free gas in the peritoneal cavity.

In heart disease also the level of the diaphragm is a useful guide as to the condition of the circulation. If the weakness of the heart muscle be the prime factor, we shall expect a high diaphragm, unless, as in advanced age, the tissues be too rigid to yield easily; if, on the other hand, some obstruction to the circulation, such as valvular defect, be the chief cause of failure, there will be excess of blood in the veins of the thorax, and a normal or a low diaphragm would be found.

Thus we have in the height of the diaphragm, as shown by the upper level of the gastric resonance, an indication for treatment and a help in the diagnosis and prognosis of disease, with which we can ill afford to dispense.

CASE IV.—*Gastric Ulcer.*

Kate G., aged 38, admitted to hospital March 6th, 1906 (Figs. 4, 5, 6, 7, 8, and 9). There was a history of dyspepsia of many years' standing, and an attack of haematemesis six years ago. Present illness dates back to an attack of influenza three weeks ago; ten days ago an attack of vomiting was followed four days later by haematemesis, half a pint of blood being brought up.

On admission, much pallor. Pain after food and epigastric tenderness were noted. Examination of the abdomen showed that the diaphragm was rather high, the gastric resonance reaching up to the upper border of the sixth rib, and the lower border of the liver being $1\frac{1}{2}$ in. above the costal arch. There was $1\frac{1}{2}$ in. of relative liver dullness in the right nipple line. She was fed entirely by the rectum for a week, and then the food was gradually increased.

The rise of the diaphragm, and its return to normal, are well shown in figures No. 4, 5, 6, 7, 8, 9, and also in the table given. With regard to Fig. 8, the transient dilatation upwards of the heart is of the type usually seen in anaemia.

CASE V.—*Gastric Ulcer with Severe Loss of Blood.*

Nelly Y., aged 20, admitted January 18th, was kept on nutrient enemata only till January 20th, then allowed one pint of milk a day as well. This diet was increased gradually till by February 10th she was taking a full fish diet.

The stomach resonance and the liver dullness (Figs. 10 to 12) continued to rise till January 29th, when they stood at the upper border of the fifth left and fourth right ribs respectively. At this date she had been for two days on an ordinary bread and milk diet. By February 10th there was still some elevation of the diaphragm, but the relationships of the organs were more normal.

CASE VI.—*Haematemesis.*

Louisa C., aged 33, admitted with very severe haematemesis. She was so blanched that the foot of her bed had to be kept elevated to prevent faintness.

The figure (13) shows the marked rise in the level of the gastric resonance, and of the lower edge of the liver; also the broadening of the relative liver dullness. In this case there was full intestinal resonance for 2 in. above the costal arch in the right nipple line.

CASE VII.—*Severe Anaemia.*

Cissy G., aged 31. The gastric resonance reached well into the fourth left interspace, and in the fifth interspace there was well-marked gastric resonance throughout the whole interspace from the sternum to the anterior axillary line (Fig. 14). The heart was displaced upwards, and, owing to the anaemic dilatation of the right ventricle, there was a larger area of dullness than in the cases of gastric ulcer without marked anaemia. There was some cardiac impulse in the fourth interspace, but the maximum impulse was in the third left interspace external to the nipple line (3 in. from the sternum). In the second left interspace there was relative dullness and pulsation for 2 in. from the sternum.

CASE VIII.—*Anaemia.*

Ada F., aged 17. Admitted to hospital February 15th with anaemia and severe chilblains. On admission the area of cardiac dullness was small, there being no absolute dullness, and the gastric resonance was very high—namely, up to the middle of the fourth rib in the left nipple line. There was also a great increase towards the axilla, for gastric resonance and reverberation were clearly obtainable in the fourth interspace near to the mid axillary line (Fig. 15). In the course of ten days the diaphragm was lower, the gastric resonance reaching only to the fifth rib, and only extending into the axilla in the sixth and seventh interspaces. After being in hospital six weeks the stomach, though still up to the fifth rib, did not reach into the axilla at all, and the cardiac dullness was larger, showing some anaemic dilatation.

CASE XI.—*Rheumatic Pericarditis.*

Emily E., aged 15, was admitted on October 29th, very ill with rheumatism and pericarditis.

The diaphragm was very high, the gastric resonance reaching to the fourth rib, and the relative dullness of the liver was

at the third rib in the right nipple line. The lower edge of the liver only reached to the lower border of the sixth rib. Three weeks later, on November 20th, the condition had improved, but the diaphragm was still high, as shown in Fig. 19. Within another three weeks the diaphragm was nearly back to its normal level (see Fig. 20).

There was very considerable dilatation of the right ventricle upwards to the left in this case.

THE POSSIBILITY OF ACTINOMYCOTIC INFECTION BY THE VAGINA.

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IN the course of a study of actinomycosis in man Ruhrah¹ classified 628 cases according to the original sites of infection. His series included 132 abdominal cases, all of which he considered had been infected from the digestive tract. He gives no instance of primary vaginal infection. If a doubtful case published by Zemann² is excluded, I have discovered two cases of this nature in the literature. They were published respectively by Grainger Stewart and Muir,³ and by Giordano.⁴

I. J., single, a kitchen maid, was admitted to St. George's Hospital on October 24th, 1904.

History.

She was recovering from an illness characterized by acute abdominal pain, nausea, vomiting, constipation, and painful micturition which dated back nine days. There was a history of two similar attacks seventeen months and eleven months previously which confined her to bed for three weeks on each occasion. Dysmenorrhoea had been present for two months before admission, otherwise the menses had been normal. She had been somewhat anaemic for the previous four years. The family history was good.

Condition on Admission.

The patient was well nourished, her tongue suggested dyspepsia, and tenderness was present on deep palpation in the right iliac region. A mass was felt in the pelvis on bimanual examination. There was no vaginal discharge. The thorax and the urine presented no abnormality.

After-history.

On November 3rd the uterine appendages were removed. There was a right pyosalpinx, and the left ovary had become a bag of pus. The appendix, which showed signs of old inflammation only, was also excised. The patient was relieved by the operation, notwithstanding a persistent faecal discharge, and she went to a convalescent home on December 9th. Here she began to complain of pain on the right side, and the temperature rose to 103°. On January 17th the abdomen was explored without result. On January 31st it was noticed that there was a fluctuating swelling over the right lower ribs. This was incised, and a pint and a half of pus, coming apparently from a liver abscess, was evacuated. Subsequently a rib was resected over the liver, and the wound, which had become oedematous, was scraped. An abscess, which pointed 2 in. higher up, and which contained 2 oz. of pus, was also opened, and the discharge from the wound over the liver became faecal. The patient lived till June 14th, gangrene of the right leg being the immediate cause of death.

Necropsy.

I found chronic peritonitis in the pelvis and in the hypogastric region. The caecum and ascending colon formed part of the posterior abdominal wall, having their anterior surfaces only in relation to the peritoneum. Several abscesses were found in the muscles and fasciae on the right side. There was a somewhat diffuse retrocaecal abscess and an abscess the size of a large marble 1 in. above the iliac crest. The ilium was smooth, and no connexion could be traced between the bone and the lower abscess, or between one abscess and the other. A third abscess was found between the posterior surface of the right lobe of the liver and the diaphragm, reaching as far down as the suprarenal gland. It contained inspissated pus, and the contiguous surface of the liver was eroded. There was a burrowing hepatic abscess the size of a large orange close to the surface of the liver and to the last-mentioned abscess. It was made up of a large number of small cavities which gave it a fenestrated appearance on section, and a resemblance to a sponge containing pus. The liver was enlarged and fatty, and it weighed 5 lb. 10 oz. The spleen was large and soft, and the kidneys were cloudy. The mesenteric glands were enlarged and caseous. The organs showed no waxy change to the iodine test. The alimentary canal was normal except in the caecal region. The pleurae were normal. The right lower extremity was gangrenous, and there was a clot in the common femoral vein.