

many of those which I have mentioned in the foregoing observations, and which are very little in use, are more certain in their operation than others which enjoy the greatest fame. For instance, the ergot of rye, which is now very generally esteemed a specific, is not by any means possessed of the same advantages, I care not how it is administered, as a solution of common salt or hippo, either alone or combined together in the form of enema. I have succeeded in rousing the action of the womb in cases of tedious labour with common salt, when used in the manner I stated in a foregoing part of this paper, in a far greater proportion of cases than with the ergot of rye, which has acquired such celebrity.

ART. III.—*Researches on Laennec's Vesicular Emphysema, with Observations on Paralysis of the Intercostal Muscles and Diaphragm, considered as a new Source of Diagnosis.*

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IN this communication I shall first consider the disease of dilatation of the air cells, and I trust to be able to establish the true principles of its diagnosis, and add some important signs to those already given by Laennec. On the symptoms and history I have but little to observe, and shall therefore only allude to them as bearing on the physical signs.

The following are the sources of the physical signs in this affection :

- 1st. The increased quantity of air within the thorax.
- 2d. The increased volume of the lung, and the resistance of the thoracic parietes.
- 3d. The displacement of the heart and abdominal viscera.
- 4th. Bronchitis of the minute tubes.
- 5th. Congestion of the lung.
- 6th. The existence of the sub-pleural vesicles of Laennec.

On percussing the chest, in a case where the disease is decided, we observe that the sound is morbidly clear. It is not, however, tympanitic, as in pneumo-thorax, but may be described as the maximum of the true pulmonary sound. In a case of extensive disease this clearness is general, but it may be partial, and merely correspond to the most affected portion of the lung. It is but little, if at all, increased on a deep inspiration, in which it differs remarkably from the sound of the healthy lung, but agrees with that of its solidified state. In fact, this character, though occurring in states of the lung so opposite as its rarefaction and solidification, is yet owing to the same cause in both, namely, the greatly diminished volume of air which can enter at an inspiration.

We may further observe, that the sound on percussion is often clear down to the lowest portion of the thorax. The natural hepatic dulness of the postero-inferior portion of the right side disappears, and unless where the heart is much enlarged, the sound of the cardiac region is remarkably clear. This will be particularly the case if the lung overlaps the pericardium to any extent, of which we can easily satisfy ourselves by means of the stethoscope.

But in almost all cases of such extensive disease, we find a complication with enlargement of the heart, the result of the long-continued and increasing obstruction to the pulmonary circulation, and this will give an increase of dulness over the organ, particularly at its right side. We then find that there is dulness from the situation of the apex of the left ventricle as far as the right side of the sternum; and as Piorry has remarked, the extent of this dulness may be found to vary according to the degree of pulmonary obstruction. This is the most common case; but in a few instances, even though the enlargement of the heart be considerable, we find in these regions a clear sound on percussion, or, at all events, a want of dulness commensurate with the heart disease, a circumstance explicable by the increased volume of the lung, which by

throwing the parietes forwards, buries the heart in the thoracic cavity. In such cases the impulse of the organ ceases to be a measure of its disease, and we are surprized at finding an hypertrophied heart, although, during life, the impulse at the side and lower sternal regions had been slight. As a general rule we may state, that where this complication exists with a distinct impulse, the sound on percussion of the cardiac region, will be dull.

But the morbid clearness of the chest is not met with in all stages of the disease ; it is only observed when the affection has arrived to an advanced degree, and may be altogether wanting in the earlier periods. A patient may have a degree of dilatation of the air cells sufficient to give decided feebleness of respiration, without any perceptible increase in the clearness of sound. Of this I saw a remarkable instance in a patient who was admitted into my wards, and who presented a group of symptoms and signs which led me to suspect the existence of an aneurism of the aorta ; his complaints had been of about five months' standing, up to which time he had enjoyed good health ; he then contracted cough, followed by severe dyspnœa on exercise, and some pain in the back and upper portion of the chest. We found that both sides sounded equally upon percussion, nor was the sound at all morbidly clear. The respiration in the right lung was puerile, while in the upper portion of the left it was exceedingly feeble. The impulse and sound of the heart, as observed below the mamma, seemed natural, but a double pulsation could be heard at the upper portion of the left side : there was no bruit de soufflet, dysphagia, or laryngeal breathing.

Here was a group of symptoms and signs, which I thought might possibly depend upon a small aneurismal tumour, compressing the left bronchus. But I made no positive diagnosis in the case. The patient sometime afterwards died with effusion into the chest ; and on dissection it was found that there was no aneurism, but that partial dilatation of the air cells

existed, affecting only the upper portion of the left lung, and that the right cavities of the heart were dilated and somewhat hypertrophied. The feebleness of respiration was clearly attributable to the dilatation of the air cells ; and the case shews, that this lesion may exist to such a degree *as to give distinct stethoscopic signs, although the sound on percussion be not perceptibly increased.* In other cases too I have found on dissection, dilatation of the air cells to some extent, although during life percussion gave no unusual results.

I now proceed to consider the remaining sources of physical signs in this disease, or those which are the principal causes of the active auscultatory phenomena. I shall, in the first place, dwell on the increased volume of the lung, and consider it first in relation to the stethoscopic signs, and next, as causing displacement of adjacent parts. And we shall inquire how far the degree of resistance afforded by the thoracic parietes tends to influence both the auscultatory signs, and those more obvious ones, which proceed from the displacement of surrounding organs.

The modifications of the sounds of respiration in this disease depend on the following causes :

1st. The increased volume of the lung.

2d. The existence of bronchitis, principally affecting the minute tubes, and often complicated with congestion of the lung.

3d. The formation of the sub-pleural vesicles.

The first of these sources of modification of the respiratory phenomena being the most important in diagnosis, I shall dwell particularly upon it.

One of the first circumstances which strikes the observer in this affection, is the want of accordance between the inspiratory efforts, and the sound of pulmonary expansion ; the first being evidently excessive, and the latter extremely feeble. When he employs percussion he will at once discover that the cause of this feebleness cannot be any solidification of the lung, as the

sound is either natural, or clearer than natural, and under these circumstances he must seek for some other cause to explain the phenomenon. It appears to me, that in the increased volume of the lung he will find the cause of this important sign: for the organ being in a permanent state of enlargement, *the dilatation of the chest can be but little added to by the inspiratory effort, and hence the sound of respiration becomes proportionably feeble.* Hence this feebleness of respiration, coinciding with clearness of the chest and increase of the inspiratory efforts, becomes the most important physical sign of the disease in question. Other causes, however, have been enumerated, particularly the thickening of the mucous membrane, the result of that chronic bronchitis which so constantly attends this affection. Thus Laennec has stated, that in the dry catarrh, which so commonly produces this disease, the mucous membrane of the minuter tubes is often extremely thickened, which, to certain degree, explains the feebleness of respiration, and also the fact, that when we compress the lungs taken from an emphysematous patient, we find greater difficulty in reducing them to their state of flaccidity than if they were in their ordinary condition. In fact, *cæteris paribus*, the sound of respiration is directly as the facility of the entrance of the air, and any mechanical obstruction, whether in the trachea, the larger or the more minute bronchial tubes, will cause a corresponding feebleness of the respiratory murmur. This has been long known; I remember seeing an interesting example of this in a patient whose chest I was requested to examine previous to the performance of tracheotomy. The history of the case was such as might warrant the supposition of the existence of a pulmonary emphysema. On percussion the chest sounded everywhere extremely clear, but the vesicular murmur was feeble, notwithstanding the violent efforts of the patient; yet on the trachea being opened, it became at once loud, even to puerility, and continued with this character for some time after the operation. But without

denying that this thickening has an effect, I cannot help thinking, that we must also attribute much to the increased volume of the lung for the following reasons :

First. In cases of ordinary bronchitis, even when the minute tubes are engaged, this remarkable disproportion between the inspiratory efforts and sound of expansion is either not observed, or occurs in a much smaller degree. In these cases we hear either a mixture of the vesicular murmur with various rales, or observe that the murmur is almost masked by the rales ; but in both cases the phenomena indicate full expansion and contraction of the lung, and their intensity can be remarkably modified by the efforts of the patient. Yet in cases of dilatation of the cells this is not observed, and the phenomena are but little modified whether the patient breathes in his ordinary manner, or makes an increased effort at inspiration. In the former case, the air cells may be considered as unaffected, and on the obstruction which results from the thickening of the bronchial membrane, or the presence of secretion in the tubes being overcome, the lung expands, and this expansion is evident to the auscultator.

Secondly. I have observed that in chronic cases of dilatation of the cells, the sign of feebleness of respiration is but little affected by the increase or diminution of the bronchitis, at least as far as we can judge of the latter by the physical signs and constitutional symptoms. Thus, it not unfrequently happens, that such patients are attacked with exacerbations of the bronchial irritation, which may subside under treatment, but during their continuance the physical signs are less an increase of the feebleness of respiration than of the various rales ; and on the other hand, when they subside, that feebleness is scarcely, if at all, diminished : in fact, the sign of feebleness is but little affected by the increase or diminution of the bronchitis, a circumstance quite in accordance with my view of its cause, namely, the diminished quantity of air that enters the affected portion of the lung.

I have already stated, that the feebleness of respiration in this affection is owing to the increased volume of the lung, by which the amount of the inspiration is diminished; for if the lung be thus hypertrophied, so as to press strongly on the chest, and preserve that cavity distended, even after expiration, it is obvious, that on the next inspiration the volume of air entering will be minus the expanding of the lung from its own distending force.

Let us suppose that the area of the healthy chest, after expiration, to be equal to 10, and the maximum of its expansion to be equal to 15, it is plain, that if from the disease the lung acquires a volume in rest equal to $12\frac{1}{2}$, the inspiration would be diminished by one-half; hence a cause of feebleness of respiration, as part of the inspiratory effort is supplied by the expansion of the lung, which results from its being kept compressed in the state of rest.

It is obvious, however, that the physical signs of the pulmonary compression must vary according to the rigidity of the thoracic walls. If we take two cases of Laennec's emphysema, and suppose that in one the chest yields *pari passu* with the enlargement of the lung, while in another it is rigid and unyielding, it is plain that the physical condition of the lung, and of course the physical signs of its actions, must be different. If the feebleness of respiration depend upon the compression of the lung, it should follow, that if in any case the chest yielded easily and fully to the pulmonary enlargement, we might have great and extensive dilatation of the cells, without the sign which is supposed to be characteristic, so that the feebleness of respiration would seem more a measure of compression of the lung, than a direct sign of dilatation of the cells. Of these views the following case is strongly illustrative, and I place the more value on it as the patient has been at different periods under my observation.

A young man of feeble muscular development, and considerably below the middle size, entered the Meath Hospital,

labouring under the usual symptoms of Laennec's emphysema ; the chest was enormously enlarged on both sides, but the principal yielding seemed to have taken place in the upper and anterior portions ; the circumference at the mammary regions being three feet and an inch, an increase of at least seven inches above its natural development. The sternum and clavicles were arched, the scapular regions nearly horizontal, and the development of both sides equal. Yet, in this case, the characteristic signs existed only in the supero-anterior portion of the right side, while over the rest of the thorax the respiration could be heard loudly, and after the individual had been treated for bronchitis it was pure. In this case the symptoms had lasted for upwards of five years, and after the second year the enlargement of the chest became so manifest as to excite the attention of all the patient's friends.

Here there was a case in which the yielding of the chest was more remarkable than any we had ever witnessed, and yet over the greater portion of the thorax the respiration was anything but feeble ; and it is a most curious and interesting fact, that with the absence of the signs there was also absence of the symptoms of compression. There was no evidence of disease of the heart ; there had never been œdema ; the jugular veins were not distended ; the liver was not depressed ; and the patient, so far from being embarrassed by exercise, was always better after walking a considerable number of miles. A short time before entering the hospital he performed a journey of forty miles on foot in the course of a single day. His only inconvenience was the recurrence of bronchitic attacks, but when these were absent his general health was excellent.

It might here be inquired, what was then the cause of the feebleness of respiration in the anterior portion of the right lung. I think that in all probability there was here rupture of the air cells, and that in this condition we have a cause, in addition to that of compression of the lung, for the ordinary feebleness of respiration.

We shall now consider some of the other physical signs, which result from the enlargement of the lung, and which, like the preceding, vary with the amount of resistance of the thoracic walls.

SIGNS CONNECTED WITH THE INTERCOSTAL MUSCLES AND DIAPHRAGM.

The next result of the increased volume of the lung, which we now consider, is its effect in displacing the more yielding parts of the thorax. These may be considered to be the mediastinum, the intercostal muscles, and the diaphragm; and we shall find, that although the mediastinum yields in cases of the disease occurring in a single lung, yet that the muscular expansions exhibit a great power of resistance, and in many cases do not yield, even after the chest has been much enlarged. In this respect we observe a remarkable difference between this disease and empyema, in which the yielding of the muscular expansions forms one of the most important signs.

When we examine the intercostal spaces in this affection, even after great dilatation of the chest has occurred, we see them, so far from being obliterated, deeply marked, and the muscular fibres acting powerfully, so as to elevate the ribs, and assist in the imperfect inspiration. I have never seen an exception to this, and the rule applies to every intercostal space; and as a point of difference between the two diseases of accumulation, empyema, and Laennec's emphysema, it is of the greatest interest. In the second part of this paper I shall point out the causes of this difference, which have not been hitherto understood.

But the same remarks cannot be made with respect to the diaphragm, which, in certain cases, yields before the enlarged lung, so as greatly to increase the cavity of the chest downwards. This circumstance may be taken as a most important distinguishing mark in cases of this disease, which may be divided into those with, and those without, diaphragmatic displacement.

From the position of the muscle, and its inferior mechanical support, we should expect, *a priori*, that it should yield more to mechanical pressure than the intercostals. And such I have ascertained to be the fact, as while I have often seen displacement of the diaphragm, in no case did I find that the intercostals were similarly affected.

Between the two cases of Laennec's emphysema, with and without this displacement, I have observed some striking differences as to symptoms and signs. Of those in which the diaphragm is not affected, we have an excellent example in the case which I have described of great yielding of the thoracic walls. Here the signs of pressure on the lung were much less distinct, and there existed no indication of hepatic displacement, the epigastrium so far from being tumid, being actually collapsed. But in the case with displacement of the diaphragm we observe that there is much more distress in breathing; that the epigastrium is full and resisting, and that the heart is pushed down sometimes so far as to be on a level with the ninth, or even tenth intercostal space.

Under these circumstances the postero-inferior portion of the chest, and the regions of the liver and heart anteriorly, give a perfectly clear sound, which is explicable by the displacement of these viscera, and also by the condition of the lung; and the respiratory phenomena may be heard down to the very last rib posteriorly, and even for two inches below the ensiform cartilage.

When these patients are stripped, and lying on the back, a remarkable character of respiration may be observed. We see the thorax powerfully elevated upwards, and the abdomen as powerfully protruded downwards; but there is this remarkable difference from forced respiration in the healthy state, that the abdominal protrusion does not begin so high, and while the umbilical and hypogastric regions move upwards and forwards, the epigastrium and upper portions of both hypochondria remain comparatively motionless, while the corresponding ribs

are drawn in. This is explicable by the new position of the diaphragm ; it has descended, and carried the abdominal viscera before it ; and its contraction takes effect at a point lower in proportion to its displacement.

That this displacement is a purely mechanical result, and not analogous to that in empyema, shall be shewn hereafter. It varies so remarkably with the volume of the lung, that I have seen the heart, after the subsidence of a bronchitic attack, mount from the tenth to the eighth intercostal space.

On the subject in general, we want some accurate dissections. I regret that my experience is but limited, but I shall state it. It would appear that much will depend on whether the disease predominates in the upper or lower lobes ; if in the latter, the shape of the lung is altered, and I have found in this way, that from the great enlargement of the cells, and the formation of sub-pleural vesicles, the lower surface, from being concave, had become flattened, or even convex. Under these circumstances the diaphragm must of course yield.

In a patient who died in the Meath Hospital, the following appearances were found : the liver was in its natural situation, but the left ala of the diaphragm was pushed far down, so as to become convex towards the abdomen. But a source of fallacy exists in this case, and in all dissections made to clear up this point, it must be borne in mind that the diaphragm may have yielded *post portem*, merely from the pressure which during life it had been able to resist.

Thus we arrive at a division of the cases of Laennec's Emphysema into two classes, namely, those with, and those without diaphragmatic displacement. The distinction between these cases will be best understood by comparing them by pairs of opposite characters.

WITHOUT DISPLACEMENT OF
THE DIAPHRAGM.

1. The shoulders greatly elevated, and the upper part of the chest convex.

2. The sound on percussion of the upper portions morbidly clear, of the lower but little altered.

3. The stethoscopic signs manifest in the upper portions.

4. The epigastrium collapsed, and the heart and liver in their natural situations.

5. The distress in breathing much less, except during an exacerbation of bronchitis.

WITH DISPLACEMENT OF THE
DIAPHRAGM.

1. The shoulders not affected, the upper part of the chest flat, and the convexity manifest only in the lower part of the thorax.

2. The sound on percussion of the upper portions not affected, of the lower morbidly clear.

3. These signs predominating in the lower lobes, and audible below the usual level of the diaphragm.

4. The epigastrium full and resisting; the right hypochondrium dull on percussion, and the heart displaced downwards.

5. The dyspnoea much more permanent, and less affected by treatment calculated to relieve bronchitis.

I need hardly remark, that we cannot observe these differences in all cases, inasmuch as the whole lung may be engaged so as to present the phenomena of enlargement in every direction; still the distinction may be often made. The greater dyspnoea and liability to morbus cordis, when the disease predominates in the lower lobes, is traceable to the fact that the patient is deprived, in a great measure, of the powerful assistance of the diaphragm in inspiration.

SIGNS FROM THE DISPLACEMENT OF THE MEDIASTINUM.

In considering these signs we find, that although they may exist so as to be demonstrable during life, yet that they are less remarkable than those in empyema. In certain cases where

the disease is confined to one lung, the morbid signs extend across the mesian line to a distance proportioned to the extent of the disease; and as in empyema we have dulness and absence of respiration extending across the mesian line *from disease of one pleura*, so in the dilatation of the air cells we have the morbid clearness and characteristic respiration, under the same circumstances; and if any thing was wanting to complete the analogy, it is, that the displacement of the mediastinum can be observed to vary with the state of disease in either case.

Thus, when the dilatation of the cells is confined altogether, or nearly so, to one lung, percussion gives a peculiarly clear sound over the affected side; and if the disease has displaced the mediastinum this clearness will be found across the whole sternum, and it may be for an inch or so beyond it. This line, which is well defined, having been passed, we then observe the natural pulmonary sound, which an experienced ear will have no difficulty in distinguishing from that of the diseased lung. If the observer now applies the stethoscope over the affected side, and carries the instrument across the chest, he will find that the peculiar phenomena of respiration do not disappear until he passes the sterno-costal articulations of the opposite side, where, like the clearness on percussion, they suddenly cease, and are replaced by the natural respiratory murmur.

I must state here, that although we should expect *a priori* that these signs always exist in the advanced stages of the disease, when confined to one lung, yet that I have only verified them in a few instances, and that additional observations will be necessary to ascertain their exact value or constancy. I have little doubt, however, from the analogy of the disease in question with empyema, that they will be found to occur in all cases of confirmed dilatation of the cells, when the disease occupies but a single lung.

But although in both instances the mediastinum be displaced, yet in the disease before us the change is seldom seen

in so striking a manner as in empyema. One reason for this may be the fact, that in most cases of decided dilatation of the cells, the disease exists in both lungs, while double empyema is one of the rarest of diseases. Another will be admitted when we recollect, that the inflammatory action of pleuritis, by softening the serous membranes, will render them more likely to yield in that disease than in Laennec's emphysema, where no such action exists.

The heart, of course, will follow the displaced mediastinum, and its position vary with the affected lung and the amount of disease. My experience, however, leads to the conclusion, that in this affection lateral displacement of the heart is rarely seen to any remarkable degree, another circumstance of difference between this affection and empyema, and to be explained by the preceding considerations. This remark, however, does not apply so much to the displacement downwards, which, as I have shewn, may occur to a very great extent. Under these circumstances the præcordial region is clear on percussion, and the impulse of the heart may be altogether wanting in its natural position, but occur as low down as the tenth rib, and between the costal cartilages and mesian line.

It is now admitted that most of the patients affected with this disease die with symptoms of morbus cordis and general dropsy, and it is not difficult to understand why disease of the heart should be so common a complication. The cause of this seems to reside almost altogether in the great enlargement of the lung, which must have a deleterious effect upon the heart in the following respects.

First, as I have already shewn by its interference with the process of inspiration. The experiments of modern physiologists have shewn the great influence which is exercised by the respiratory process on the venous circulation; but in the disease before us we find the chest in a state of permanent dilatation, to which the inspiratory effort can add but little, the manifest consequences of which must be an accumulation of

blood at the right side of the heart, and consequent disease of its pulmonary cavities. The vena cava becomes loaded, the hepatic veins engorged, and the liver consecutively engaged. Under these circumstances the muscular parietes of the heart become hypertrophied, and active aneurism of the auricle and ventricle is produced.

Secondly, it seems more than probable that the same pressure which has distended the chest and displaced the diaphragm must act directly in impeding the circulation through the pulmonary artery and its ramifications, and thus we see an additional cause for the production of hypertrophy of the right cavities of the heart.

Lastly, we must recollect that the heart itself is under the influence of anormal pressure. It is removed from its natural situation, and to a certain degree deprived of its natural protection by the bony and elastic parietes of the chest, and is compressed between the distended lung, on the one hand, and the distended abdomen on the other. Under these circumstances its actions of dilatation and contraction must be materially interfered with, the auricles will experience a powerful impediment in filling the ventricles; and if these cavities have an active power of dilatation, this must also be materially impeded. Thus, many circumstances concur to derange the pulmonary, cardiac, venous, and hepatic circulation. And we can only wonder at the powers of nature in prolonging life under such a complication of evils. In the great majority of cases such patients die with symptoms of what is commonly called hydrothorax, to the disappointment of the practitioner, who prescribes according to the rules of the nosological writers, and a *post mortem* examination reveals the causes of his failure, and the error of his teachers.

SIGNS FROM THE EXISTENCE OF BRONCHITIS.

On the subject of the signs manifestly proceeding from bronchial irritation I have to remark, that there is not one of

them which can be considered as pathognomonic of the complication with dilated or ruptured air cells, inasmuch as we may find them all in cases where no such affection exists. None of them are constant; and when they do occur, scarcely differ from what is observed in simple bronchitis: we may have all varieties of the sonorous, sibilous, mucous, and muco-crepitating rales in this affection, and the occurrence and mode of combination of the phenomena are infinitely various. The two most common are, the dry sibilous, and a diffuse mucous rale. Laennec has stated that there is one form of rale which is pathognomonic of the interlobular emphysema, although it may also occur in the simple dilatation; this he calls the *dry crepitating rale with large bubbles*, and describes it as conveying the impression of air entering and distending lungs which had been dried, and of which the cells had been unequally dilated. He compares it to the sound produced by blowing into a dry bladder, and states farther, that it is similar to that observed in common sub-cutaneous emphysema when we press the stethoscope on the affected portion. Now, without at all calling in question the extraordinary tact of Laennec, I would say, that this is a sign, which, if it does exist, must be so easily confounded with other phenomena, such as those proceeding from bronchitis, that an ordinary observer would not be safe in founding a diagnosis on its supposed existence. I have never been able to satisfy myself that I had recognized it, and have even found the interlobular emphysema in the lungs of persons, in whom during life I was not able to distinguish the rales from those of simple catarrh. He states, however, that the phenomenon is not common, and when it exists is of short duration, and observed in points of only small extent. On this subject further observations are necessary.

I shall lastly allude to the sign of the rubbing sound, or *frottement*, which has been described by Laennec as an indication of those sub-pleural air vesicles which occur in the interlobular emphysema, and which, according to him, when oc-

currence with the other symptoms of dilated cells, may be looked on as diagnostic of the lesion in question. But this point of diagnosis requires still further investigation; and indeed it seems difficult to understand how the existence of an air vesicle could give rise to the rubbing sound. We know that in the healthy condition of the internal surfaces of serous membranes, the friction of their opposite faces is so diminished by their smoothness, and their being lubricated by the serous exhalation, that no perceptible sound accompanies their motions. It is only when the surfaces are rendered dry by an arrest of secretion, or roughened by the effusion of lymph, that their motions produce sounds perceptible to the ear. Now, even where extensive vesicles exist, we commonly find that the serous surface, as far as smoothness and lubrication are concerned, continues in its natural state; and I cannot help agreeing with Meriadec Laennec, that the sign of frottement is to be looked on more as an indication of slight pleurisy than of these sub-pleural vesicles. I have never observed this phenomenon unless in cases where the serous surface was roughened; and as it is admitted, both by the above author and by M. Reynaud, that the sound in pleurisy is undistinguishable from that described by Laennec in this disease, we have, I think, sufficient reasons for extreme caution in the diagnosis of sub-pleural vesicles from the existence of the sign in question.

It might be supposed that the permanence of the sign and the absence of pain would prove diagnostic marks, but the truth is, that even these circumstances will not be sufficient. Thus I have seen cases in which the frottement of pleuritis continued for a month with scarcely any alteration, and in which, after the first week, the patient felt no pain, and only complained of the rubbing sensation produced during respiration.

Before leaving this subject I shall describe another sign which promises to be of some importance in diagnosis. It

is founded on the difficulty of expiration which occurs in this disease, a difficulty by some attributed to the obstruction of the minute bronchial tubes, and more lately by Magendie to the diminished elasticity of the lung itself.

I have at present under my care, a patient aged upwards of sixteen years, who has been subject to cough and dyspnoea from infancy. The right side is enlarged, and very convex anteriorly, the sternum somewhat arched, and the clavicle elevated. Over this side the sound is morbidly clear on percussion, and the clearness extends across the sternum; yet on applying the stethoscope during ordinary respiration, nothing is heard but a muco-crepitating rale, occasionally combined with Laennec's *rale crepitant à grosses bulles*; these signs are audible during inspiration, while expiration is marked by a dry, prolonged wheeze. On a forced inspiration, however, a distinct sound of pure pulmonary expansion follows the rales above-mentioned.

From these observations I concluded that the case was one of Laennec's emphysema, which had not yet arrived at its most extreme stage, inasmuch as that by a forced inspiration the lung could be still considerably distended. It then struck me that by making the patient perform a number of forced inspirations rapidly, the lung might be so far distended with air as to prevent the occurrence of any natural sound of pulmonary expansion for a time, and that thus we might obtain a direct proof of the difficulty of expiration. This experiment I put into effect, and found that after four or five inspirations, rapidly performed, the respiratory murmur altogether disappeared, nothing being heard but the crepitating rales, and even these in a diminished degree. The patient was now allowed to rest and to breathe naturally for a certain number of times, when on the experiment being repeated, the first inspiration was distinctly followed by the murmur, which, however, diminished at each successive effort, until at length it became extinct as before.

The results of this experiment are easily explained by referring to the difficulty of expiration, proceeding from either or both of the causes already alluded to. In fact, the repetition of the inspiratory efforts caused such an accumulation of air in the diseased portion of the lung, as ultimately to nearly prevent its further expansion, and thus hinder the sound of the respiratory murmur. But on the cessation of these efforts, the air was gradually evacuated, and the lung restored to its original condition. If this sign be found constant, it will be a most valuable addition to our means of detecting the emphysema of Laennec, but the frequent repetition of the experiment must be avoided.

TREATMENT OF DILATATION OF THE CELLS.

But as the end of all diagnosis is treatment, we may now inquire whether or how far this is a curable disease.

Can we expect, after the disease is established, that the dilated air cells can ever resume their natural condition? Now we find that some patients have laboured under this disease, or its causes, from infancy, while in others it is brought on by bronchitis at a late period of their lives, and after many years of previous health. In the first case, it seems scarcely possible that any effort of medical skill can restore the lung to its original condition, and all that we can hope for is to palliate the symptoms. But in cases of a comparatively recent origin, to give up all hope of cure seems scarcely in accordance with our knowledge of analogous affections. We may consider the pathological condition of the air cells in the same point of view that we look upon chronic dilatations of other hollow organs, such as those of the stomach, colon, bladder, and heart. In these cases we commonly observe the two following circumstances to occur: first, that the cause of the dilatation is some mechanical obstruction to the exit of their natural contents; and next, that if this obstruction be long continued,

what was first a mere dilatation or extension of the organ becomes a combination of this with an organic alteration of the parietes, which is, in most cases, an increase in their thickness and strength. Hence the hypertrophy of the tunics of the stomach when the pylorus is affected; of the bladder when the urethra or prostate are diseased; of the colon in stricture of the rectum; and of the right cavities of the heart in affections of the lung. This change from mere dilatation to increase of growth seems to be a condition very unfavourable for cure, and the chances of its production may be stated to be directly as the length of time the obstruction is allowed to continue; for we know that in the earlier periods of these mechanical dilatations, the removal of the obstruction is often followed by the return of the cavity to its natural dimensions. Applying these considerations to the case of dilatation of the air cells, it seems not impossible that in the earlier periods the removal of the obstruction would be followed by a subsidence of the disease; for when we inquire into the causes of the affection, we find these to be principally obstructions to the free exit of the contents of the cavities; the viscid mucus and the turgescence of the bronchial tubes being to the air cells what pulmonary obstruction is to the heart, or urethral to the bladder; and the distention in these cases being perfectly analogous.

We may then admit that where actual change of structure has not occurred, a cure, or a great alleviation of the disease, is not impossible. Our next inquiry is, whether there is evidence of such ever occurring. On this question Laennec speaks doubtfully. After alluding to the combination of extravasation of air with dilatation of the air cells, he observes, that it is of slight consequence as compared with the latter affection, as we can hope for its removal by absorption as in other similar cases, whilst we cannot well see in what manner either nature or art can remedy the other morbid derangement. "At the same time," he continues, "I do not think we are

justified in considering this affection as altogether incurable. In several instances I have fancied that I discovered the traces of cicatrization of ruptures of the pulmonary tissue of the kind above described. In the case of subjects affected with asthma I have several times, during the fits, detected a crepitous ronchus with large bubbles, which ronchus entirely disappeared afterwards; and it is quite intelligible, that if we can diminish the intensity of the cause which keeps up the habitual distention of the cells, we may in the end hope, that these will be actually lessened in volume." The same author, when describing the treatment of dry catarrh by alkalies, states, that many persons who had already emphysema of the lungs, and either incessant dyspnœa, or very frequent fits of asthma, have been restored by this treatment to a state of health so comfortable, that they hardly exhibited any signs of disease.

The question as to the curability of Laennec's emphysema has been scarcely agitated in medical circles; and Dr. Osborne deserves great credit for bringing this subject forward in his lately published brochure on the pathology and treatment of dropsy, in which he states his conviction, that this disease is at all events susceptible of great amelioration, on the ground that in certain cases he observed the feebleness of respiration, and morbid clearness of sound, to subside, or become greatly diminished, after treatment calculated to remove the obstruction, and diminish the frequency and violence of cough. On this subject I can only bring forward the observations of a few cases, but which, as far as they go, are of great importance in elucidating the question. In the patient, to whose case I have already alluded as illustrative of the diagnosis from mediastinal displacement, I found after certain treatment, calculated to relieve bronchial irritation and diminish cough, that coincident with great relief of symptoms, the following changes in the physical signs took place: first, that the morbid clearness of the affected side,

though not removed, was diminished, and that it terminated at the mesian line in place of extending, as before, beyond the opposite side of the sternum. Secondly, that the rales became more humid and larger, and the vesicular respiration was manifestly increased. And thirdly, that the stethoscopic phenomena, like those of percussion, ceased to be heard beyond the mesian line of the sternum, when they had been before audible, and that in this situation they were replaced by the healthy murmur of the opposite lung. These alterations in the signs, so characteristic of diminution in the obstruction and volume of the affected lung, were accompanied by the most marked improvement in the symptoms; the cough, dyspnœa, and acceleration of breathing being wonderfully diminished, and the condition of the patient in every respect improved.

The treatment pursued was the employment of local bleeding and counter-irritation, with the exhibition of the tartar emetic for several days, followed by sedative and demulcent remedies.

That in this case the volume of the affected lung was reduced by treatment, there can be no doubt; and when we connect the results of the case with those obtained by Dr. Osborne, and with the observations of Laennec on the treatment of dry catarrh, we have decided evidence in favour of the possibility of the diminution of the disease, and are consequently justified in considering it as not altogether incurable. In another instance I have seen the heart, which was so much displaced downwards as to pulsate at the cartilage of the tenth rib, after a few days of treatment, remount towards the thorax, and correspond to the eighth intercostal space.

Some important questions here arise. Is the mere diminution or even removal of the obstruction all that is necessary for the restoration of the lung to its natural condition, or may there not be some other morbid state to be overcome before we can bring about so fortunate a result? Does a paralysis or

atony of the circular fibres of the more minute tubes exist? Or, as Magendie has suggested, is the natural elasticity of the lung destroyed or injured? It seems not improbable but that both these circumstances may occur, the muscular structure being paralyzed, as we see in the case of the bladder or the intestinal tube, and the longitudinal fibres losing their elasticity from the persistence of chronic irritation, just as the elastic coat of arteries loses its property when chronic disease affects these vessels.

It is plain that farther observations are necessary to clear up these points; and I shall merely remark, that after the use of treatment calculated to remove congestion, inflammation, or other obstruction of the minuter tubes; after the adoption of the means which Laennec has pointed out for the relief of the dry catarrh; and lastly, after using all means which could moderate the cough, or render it less frequent, we might then have recourse to measures calculated to stimulate the contractile tissues of the lung. As yet we are not in possession of means capable of restoring elasticity to such tissues as the longitudinal fibres of the lung, or the middle coat of the arteries; but we do know of remedies capable of stimulating muscular fibre to resume its vital contractility, at least of that portion of the muscular system which is supplied by the cerebro-spinal nerves. It has been suggested to me by my friend and pupil, Mr. Martin, that in the exhibition of strychnine this object might be attained. This practice would be well worthy of trial, for if, as there is reason to believe, the pulmonary branch of the vagus is a nerve of motion to the lung, we might expect that the stimulation exercised by the remedy on the cerebro-spinal centres would have a beneficial effect in paralysis of the bronchial muscles.

PART II.

ON PARALYSIS OF THE INTERCOSTALS AND DIAPHRAGM CONSIDERED
AS A NEW SOURCE OF DIAGNOSIS.

The observations which I have to offer on this subject were communicated to the medical section of the British Association, at the late meeting in Dublin, and inserted in an abridged form in the report of its proceedings, which appeared in this Journal. Since that period I have had additional opportunities of verifying the statements then made, which I now consider as established, and of no little importance in diagnosis and general pathology.

Without entering on the question, as to whether the organic diseases of the thoracic viscera are followed *in all cases* by some alteration in the volume of these organs, we may divide the cases of thoracic diseases into two classes :

First, Those in which there is no manifest alteration ; and,

Second, Those in which there is a manifest alteration of volume.

It is obvious that in the latter case two subdivisions must be made, as there may be on the one hand an increase, and on the other, a diminution of volume.

Now this division applies more to the cases than to the diseases themselves ; for a similar disease may occur in one patient with, and in another without, alteration of volume ; and in the same patient we may have, at different periods of the affection, either an augmentation or a diminution of volume.

These enlargements are of two kinds. There may be an actual increase of volume in the parenchyma of the organ, as we see in hypertrophy of the lung or heart ; or the serous coverings of these organs may be distended by the products of disease, so as to displace the surrounding parts, and thus give rise to most important physical signs. To these affections then, when occurring to such a degree as to cause obvious changes in the form of the surrounding parts, we may give

the name of "*diseases of accumulation*;" and we shall find that the principles of their diagnosis are, to a certain degree, similar.

But another, and still more important division is to be founded on the effect of disease, in diminishing or increasing the quantity of air within the thorax. In the great majority of instances, the amount of air is diminished; but in a few it is increased. Now if we take empyema, on the one hand, and dilatation of the air cells and pneumo-thorax on the other, we find that these *diseases of accumulation* may occur with a diminution, or with an increase in the quantity of contained air: so that their diagnosis depends, first, on the evidences of accumulation; and next, on the physical properties of the accumulated matter. In empyema, there is accumulation, and pressure from a non-elastic fluid; while in Laennec's emphysema, and in pneumo-thorax there is also accumulation, but from an elastic medium; hence we arrive at the first step in the diagnosis of these lesions. In empyema we have, in addition to all the evidences of displacement of the lung, the side, the mediastinum, and diaphragm, proofs of a *diminution of the quantity of air*, which may amount almost to its total absence from the affected side, *the sound on percussion being dull*. In the other affections we have also displacement, which, as far as the non-muscular portions of the chest are concerned, is similar to that in empyema, but there is evidence that the air has not only not been diminished, but that it is increased, the sound on percussion being clear, or morbidly clear.

When we compare the chests of two individuals, the one affected with empyema, and the other with this dilatation of the cells; we observe that in both there is evidence of accumulation, the side being distended, and the mediastinum displaced. But when we investigate this point more closely, we find some interesting points of difference between the results of these diseases on the thoracic parietes, *particularly with reference to their muscular portions*.

This leads to a most interesting subject for investigation, namely, the effect of these *diseases of accumulation* on the muscular or more vital portions of the thoracic walls. I shall examine the state of the intercostal muscles and diaphragm in the two diseases which I have selected for comparison.

The peculiar smoothness of the side in empyema has been long described as a pathognomonic sign of the disease. It proceeds, as every one knows, from a yielding of the intercostal muscles, so that the spaces become obliterated, and thus the smoothness is produced. Further we find, as I have shewn in a former paper, that in like manner the diaphragm yields until it may even become concave towards the chest, and convex towards the abdomen; pushing before it the viscera which lie in the upper portion of that cavity.

But these phenomena are by no means so marked in the dilatation of the air cells, in which, as I have shewn in the preceding part of this paper, the disease may exist to a great amount, and the chest be extremely dilated, without any one of the appearances above mentioned; the intercostal spaces continue, in all cases, well and deeply marked; and in one class of cases the diaphragm remains unaffected, even though the pressure be so great as to change the form of the chest.

Let us now inquire why it is that this remarkable difference exists. By examining the circumstances of either case we may arrive at the explanation.

In empyema, *there is a combination of vital and mechanical causes.* We have *inflammation followed by pressure*, and pressure from a liquid.

In the dilatation of the cells we have only pressure, and this from an elastic fluid.

Now in this circumstance of inflammation,—of the pleuritis, which causes the effusion in empyema, and *which continues to act long after the effusion has set in*, it appears to me that we have the explanation of the dilated state of the intercostals, and the yielding of the diaphragm.

When a tissue such as mucous or serous membrane is inflamed, we find that certain effects are produced on the muscular expansions or the masses with which they are closely connected; their functions suffer, and we observe, first, an increase of innervation, as shewn by pain and spasms; and next, a paralysis more or less complete. The same circumstances occur when the inflammation is seated in *the muscular structures themselves*, or in the *cerebro-spinal centre from which they derive their innervation*. In all these cases, whether of contiguous inflammation, of actual disease of the muscular fibre itself, or of inflammation of the brain or spinal marrow, we have produced, first, a *plus*, and afterwards a *minus* state of innervation. When the latter condition supervenes, the muscular fibres lose their contractility; and if the organ be a tube surrounded by fibres, it dilates; or if an expansion similar to the intercostals or diaphragm, it yields easily to pressure.*

Now the true explanation of the protrusion of the intercostals and diaphragm will be found to be, that they are affected with this paralysis following inflammation of a contiguous structure, that their contractile powers are lost, and that hence they yield easily to a pressure which, in their healthy state, (as we have seen in the vesicular emphysema,) they could effectually resist.

But we must examine into the evidence of this theory of displacement of the thoracic muscles in empyema.

The first point of evidence is obvious when we reflect on the general effect of irritation on muscular fibre. Now in the case before us we may observe, that the phenomena are in accordance with this admitted effect. In the first stage of pleuritis we have great pain; difficulty of respiration; hurried breath

* Here I would refer to the researches of Abercrombie on Ileus, which have demonstrated, that the contracted portions of the tube are healthy, and that the morbid appearances are confined to the dilated parts; the loss of power being the true cause of the constipation.

ing; pain increased on a deep inspiration; and all this *without protrusion of the intercostal spaces or diaphragm*, but rather with a spasmodic state of these expansions, conditions which accurately correspond to the plus state of innervation which occurs in the first stage of muscular irritation.

But in the more advanced periods, the reverse of all this occurs. The pain ceases, the dyspnœa greatly diminishes, the breathing becomes slower, the diseased side is comparatively motionless, while the healthy one is acting with great power, *and the intercostal spaces and diaphragm yield*; the first causing the characteristic smoothness of the side, and the next, the depression of the abdominal viscera. I need hardly remark, that these circumstances correspond with the minus condition of innervation, or paralysis of the muscular fibres.

The next and most important evidence is the fact, that mere pressure seems insufficient for the phenomenon in question. If the theory which I have given be true, it should follow, that in other diseases of accumulation, where inflammation of the pleura was not present, but where there was merely pressure, this muscular protrusion should either not occur, or be much less marked. Now such may be observed to be the fact. Let us take Laennec's emphysema, hydrothorax, and enlargement of the liver as examples; in all of which there is evidence of pressure from within. Thus, in Laennec's emphysema, we have already studied the great enlargement of the chest, and the displacement of the mediastinum and heart, and have seen, *that even when the diaphragm is flattened, (as occurs in a certain class of cases,) its innervation is not destroyed*. In hepatic enlargement we may see, also, evidences of pressure from the great tilting out of the side, and the state of the lung. While in hydrothorax, the pressure is demonstrated by the diminished volume of the lung, which, though a muscular organ, cannot avail itself of its powers in resisting pressure from without.

But notwithstanding all this pressure it will be found, that

in all cases of emphysema and enlargement of the liver, and in many, at least, of hydrothorax, the intercostal spaces do not yield; a fact which may be constantly verified in dilatation of the air cells, and in hepatic enlargement, and which I *have lately observed in three cases of symptomatic hydrothorax*, in which, although the effusion amounted to several pints, and the corresponding lung was reduced in volume, neither the intercostals nor diaphragm were affected. It is not many years since I pointed out the value of the absence of intercostal dilatation in distinguishing an enlarged from a displaced liver. In all these cases we may have great displacement of the side or thoracic viscera; yet there is merely pressure, and though the ribs are dilated, the intercostal spaces preserve their relative positions.

The last point of evidence is the fact, that in certain cases of empyema I have observed a *sudden* yielding of the diaphragm, which, up to a certain period, had preserved its natural position. This yielding was as extensive as it was sudden, *and in one case was not accompanied by evidences of increase of effusion*. How much more easily we can explain this interesting fact, on the supposition which I have adopted, than on the idea of gradual pressure on a vitally resisting medium.

I trust that I have now established that the protrusion of the intercostal spaces and the depression of the diaphragm in empyema, result principally from a paralyzed state of these expansions; and without denying the influence of pressure, that this latter seems secondary to the inflammatory action in causing the yielding of the muscles by a paralysis. If this doctrine be true, it furnishes some important additions to diagnosis: it will give a new point of difference between the signs of hydrothorax and empyema, and perhaps furnish a measure of the intensity of inflammation in any given case of the latter disease. On the value of this point in prognosis, and in determining for or against operation, I need not here descant.

Let us next inquire how far the existence of paralysis of the inspiratory muscles may act in inducing that contraction of the

side which Laennec first described as a consequence of the absorption of inflammatory effusions into the pleura. For this result he has given two causes, namely, the binding down of the lung by a fibro-cartilaginous membrane, and its diminished volume, the consequence of pressure and disease. But we may safely add to these causes the weakened state of these muscles which so powerfully expand the chest; the intercostals no longer elevating the ribs, and the corresponding ala of the diaphragm not being capable of contraction, it would follow, that even though the lung was not bound down by fibro-cartilaginous membrane, or atrophied by pressure or disease, it would not regain its volume, as the effusion disappeared. The great source of its restoration is injured or removed, and it remains in a diminished volume because it is not expanded by the inspiratory efforts. Laennec himself has admitted, that the existence of a fibro-cartilaginous membrane is not necessary for producing contraction, and I can verify this by the observation of several cases; and may add, that in the cases of recovery which I have observed, I constantly found that the lower the form of inflammation, the more the disease approached to hydrothorax, the less did contraction result on the absorption of the fluid.

I cannot help pointing out some other applications of this principle as subjects for future investigation. We find in the different forms of thoracic disease, three cases, in which muscular fibre is affected by inflammatory disease of another structure. These are *pleuritis*, of which I have already spoken, *bronchitis*, and *pericarditis*: in all these the suffering of the muscular tissue in the earlier stages has been recognized; but the effects of inflammation in a more advanced condition, and which must in many cases be present, have been quite neglected.

In bronchitis we may inquire how far the paralysis of the circular muscles of Reissessen accounts for that accumulation in the tubes, so commonly seen in bad catarrhal fevers. In such

cases we constantly see patients dying *without general prostration*, but on the contrary, with a singular degree of muscular strength, as far as the system of animal life is concerned, and with loss of power only of those muscles of organic life which have been injured in the way pointed out. We may by emetics again and again relieve the chest, and in the interim neglect no means with which we are acquainted to remove congestion or inflammation, and yet a fatal termination supervene to this melancholy, and to the thinking physician most humbling scene. We should inquire how far the existence of this state should lead us to modify our treatment, and seek for some agent which would stimulate the bronchial muscles to contract. Further, we may understand more clearly how it is, that after the first stage of bronchitis or that of muscular spasm has passed by, that further antiphlogistic treatment is so often followed by copious accumulation in the tubes, and also get an additional explanation of the efficacy of stimulants, both general and specific, in the advanced stages, or those in which a paralysis of the circular muscles in all probability exists.

It might be inquired how far this condition may assist in producing dilatation of the tubes. All authors seem agreed that the explanation by Laennec of the mode of production of this affection was too mechanical, and that something more is wanting on this point of pathology.

Lastly, in the case of pericarditis, it satisfactorily explains the fatal termination of the disease, and clears up the singular fact, hitherto unexplained, that death should occur from the inflammation of so limited a structure as the pericardium, while so extensive a sac as the pleura may be universally diseased, with preservation of life. If with these views we examine the symptoms of the first and second stages of pericarditis, it is not difficult to trace the analogy between this disease and pleuritis or enteritis. In the first stage, the violent palpitations, and the hardness of pulse, point out the state of muscular excitement, similar to what occurs in the early periods of pleu-

ritis or enteritis ; and in the second the faintings, the weakness of pulse, the coldness, the feeble action of the heart, and obstruction of the circulation, are the indices of the muscular paralysis, of the same state of the muscles of the heart, as those of the intercostals in empyema, or the circular muscles in ileus.

Assuming that a semi-paralyzed state of the heart exists in advanced pericarditis, may we not further have an explanation of the common percussion of active aneurism to the disease. In the weakened state of the organ it yields to the pressure of the blood, and by degrees its cavities distend. On the hydrostatic principle the force of this distention must be every moment increasing, just as we see, in aneurisms of the aorta, that the larger the sac, and the more fluid the blood, the more violent will the impulse be, and of course the more rapid the extension of disease. Now, let us suppose the inflammatory process to cease, the muscular fibres of the heart recover their tone, but from the increased size of the cavities they have an increased duty to perform : they have to force blood into vessels which have not been proportionally dilated, and which of course resist ; and then, from the well known law in physiology, we have increase of growth : to dilatation is added hypertrophy, and the active aneurism is established. This view of the supervention of active aneurism to pericarditis is still suggestive ; further observations are necessary to clear up the point, but it is one of great importance, and I am the more anxious on the subject, inasmuch as the explanation which I have given differs materially from that of Andral,* who affirms that muscular hypertrophy takes place from the first, and omits the possibility of an intervening paralytic condition of the heart. I do not suppose that my explanation will overturn his, for the disease may occur under both circumstances ; let it suffice that I claim to add to the observations of such an author.

In concluding this paper, I beg to express a hope that the

* Clinique Medicale. Maladies du Cœur.

profession will receive these views and observations with indulgence. Some apology seems due for the length of the communication, and for adding to a record of actual observations a discussion on views which may be considered more or less hypothetical. But it was necessary to connect the papers on the diagnosis of vesicular emphysema, and on the paralysis of the respiratory muscles, as they are mutually illustrative, and embody principles of diagnosis hitherto unapplied, and common to the diseases of which they treat.

Observations on the Effects of Cold on the Human Body, and on a Mode of measuring Refrigeration. By JONATHAN OSBORNE, M. D., President of the King and Queen's College of Physicians, &c. &c.

[Read before the Medical Section of the British Association, August 14, 1835.]

It would be an interesting observation to ascertain by a tabular view the proportion of diseases produced by cold, as compared with other agents. In order to obtain some idea of the probable results of such a table, I have caused the patients at present in Sir Patrick Dun's Hospital to be examined, and it appears that of fifty-seven (the entire number) thirty-four can be distinctly referred to cold contracted in the following manner: in twelve from wet clothes, five from damp feet, three from bathing, and fourteen cold air when heated. The investigation of so powerful a cause of disease, and of an agent no less important as a means of cure, will, I trust, not appear unworthy of the attention of the section. I beg, however, to premise, that in the following observations, so far from taking a complete view of the subject, I design only to throw out a few hints intended to point out some new directions in which future researches may be carried on with advantage.

The temperature of the blood being about 98°, the same