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Original Articles and Clinical Cases.

ON DISTURBANCES OF SENSATION WITH ESPECIAL REFERENCE TO THE PAIN OF VISCERAL DISEASE.

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PART III.—PAIN IN DISEASES OF THE HEART AND LUNGS.

INTRODUCTION.

IN my previous papers I have dealt with referred pain and superficial tenderness from the topographical standpoint. Where a single area on the body or on the scalp was well marked out, I laid stress on that particular case, oblivious of whether it was typical of the disease which caused it or not. When an organ was associated with a large number of segmental areas, no attempt was made to show the significance of different groupings of these areas in the diseases of that organ.

The following paper treats, not of the topography of the segmental areas, but of the pain caused by diseases of various organs. Thus I lay no weight on the appearance of a single area on the body or the scalp, but attempt to show the meaning of groups of areas of superficial tenderness.

I do not presume to write a treatise on diseases of the heart and lungs, but only to show how diseases of these organs illustrate the distribution and the origin of referred pain and superficial tenderness.

This enquiry is essentially a physiological one with clinical cases in the place of laboratory experiments. Thus many of the cases I shall give as illustrations are not intended to illustrate the natural history of the disease, but simply serve as links in an investigation of the causes which lead to referred pain.

It has been the custom to speak of many of these pains as "intercostal neuralgia," "rheumatic pains," and by other names of a like kind. The referred headaches associated with these pains have been called "megrim," "cardiac headaches," and the like. I hope I shall be able to show that such words are unnecessary and act perniciously by soothing the conscience of the observer.

CHAPTER I.

Pain in Diseases of the Heart.

§ 1. *Introduction.*

The occurrence and significance of pain during the course of diseases of the heart has not been systematically dealt with in any of the text books on the subject. Most authors mention certain specific instances, such as the pains associated with aneurism, and those of angina pectoris, but no systematic attempts have been made to note the presence or the absence of pain and the character it assumes in each of the types of cardiac disease.

It is useless to select only those cases which suffer from pain, for the absence of pain is often more instructive than its presence. The presence or absence of pain should be noted in each case as part of the routine examination.

Then we must determine whether if pain be present it is local or referred. In some cases, as when it involves the neck or the arm, it is obviously referred; but in many cases it would be impossible to say, had we only the patient's description to guide us, whether it were referred or not. This is especially the case with those pains which lie in the region of the heart itself.

It, therefore, becomes necessary to examine the superficial structures of the chest, back, and arm to see if they be tender or not. For if superficial tenderness is present the pain which accompanies such tenderness is certainly referred.

Such hyperæsthesia of the skin has long been recognised as an accompaniment of affections of the heart. Thus Hilton,[1] mentions tenderness of the upper part of the chest as a symptom in diseases of the heart and great vessels, and Walshe places it amongst the occasional concomitants of cardiac disease. The first of these observers shows throughout his works how close he was to the truth with regard to referred pain and its accompanying tenderness; but he was misled by his knowledge of the gross anatomy of the peripheral nerves and by the want of that knowledge of the construction of his sympathetic system which we owe entirely to Gaskell's initiative. Walshe, with his splendid powers of observation, evidently suspected that hyperæsthesia might be a sympton of importance; for he does not dismiss it lightly, but notes that its significance is a matter for further research. Since then writers on cardiac disease have entirely neglected superficial tenderness, dismissing it with a slighting reference to hysteria under complete misapprehension of Charcot's teaching as to the nature of his hysterical points.[2]

Referred pain and superficial tenderness must remain inexplicable so long as we classify cardiac disease according to the name of the valvular defect. For the chief factor in the production of referred pain and its attendant phenomena is some alteration in the tension within the cavities of the heart secondary to the disease of the valves.

Now in some cases of valvular disease the secondary changes within the cavities of the heart are plain; but in many other cases we are ignorant of the conditions within

[1] "Rest and Pain" [4th edition], p. 258.

[2] Since Mackenzie [*Med. Chronicle*, 1892] and myself independently recognised the significance of superficial tenderness in visceral disease, I only know of the following papers bearing on the occurrence of this symptom in cardiac disease—Mackenzie "Heart Pain" [*Lancet*, Jan. 5, 1895], James [*Brit. Med. Jour.*, June 29, 1895].

the heart under which the circulation is carried on. Our knowledge of these conditions is based on our interpretation of physical signs, and it is in this very interpretation of the same physical signs that the greatest differences of opinion exist. No new physical sign or method of examination has been discovered, and yet our knowledge of the physical signs which point to mitral stenosis has advanced considerably of late. Thus it may happen in the future that the laborious association of certain symptoms with what we suppose to be some one or other condition within the heart may become valueless, owing to an increase in our knowledge of the significance of some particular association of physical signs that has as yet escaped observation.

I shall, therefore, in all cases state the conditions which determine the presence or absence of referred pain in terms of physical signs. Later, I shall devote a chapter to a consideration of what I believe to be the interpretation of these signs, and the bearing of this interpretation on the causes of referred pain. Thus if my interpretation should prove incorrect, I hope that the observed association between the presence or absence of pain and certain conjunctions of physical signs may still remain.

Every case upon which this work is founded has been taken by myself. For in such an investigation it is all important that the reader should have to deal with the errors of a palpable individual, rather than with those of impalpable ghosts. In every case from the Victoria Park Hospital for Diseases of the Chest, the physical signs were checked by other observers more skilled than myself. To Dr. Harrington Sainsbury and Dr. Colbeck, my thanks are particularly due for the trouble they took to insure the accuracy of my physical signs in case of cardiac disease.

My object here is to elucidate the conditions which cause referred pain, and I have therefore excluded all cases from these pages in which competent observers diagnosed compound lesions. For although such compound lesions form very instructive puzzles for a commentary, they can only be explained when we have settled the primary laws by a study of simple lesions.

§ 2. *Local Pain in Diseases of the Heart and Pericardium.*

The pain that is produced by diseases of the heart may be local or referred. Before passing to the characteristics and distribution of referred pain, which forms the most important portion of this chapter, I will touch shortly on some features of the local pain that occurs with certain cardiac lesions.

In examination of every patient it is necessary first to exclude referred pain, and its accompanying superficial tenderness before concluding that the pain is local. Now when the pain is situated over the arm, no difficulty can arise, for such pain in disease of the valves of the heart must be referred and not local. But when the pain lies, at any rate, partly, over the heart itself, such pain is commonly, though in most cases erroneously, supposed to be local and not referred.

Attention to the following points of difference enable us to decide whether the pain be local or referred :—

Referred Pain.	Local Pain.
1. Referred pain is not only situated on some spot on the front of the chest, but also over some spot behind at a distance from the cardiac area.	1. Local pain is confined strictly to the area of the heart or pericardium, and does not pass round the body or through to the back.
2. Referred pain is associated with more or less tenderness of the coverings of the chest and back, elicited by picking up the skin and subcutaneous tissues between the fingers.	2. Local pain is accompanied by no marked superficial tenderness. But when the intercostal spaces over the painful area are either pressed or percussed, the pain is increased, the amount of tenderness elicited depending on the force applied.
3. This superficial tenderness frequently passes round the body in a more or less horizontal band from the middle line of the back to the middle line in front. If less acute it lies over one or more spots, one of which is situated over the anterior, the other over the posterior aspect of the trunk.	3. When present, deep tenderness can only be elicited over the heart or pericardium, and is absent from the posterior aspect of the trunk.
4. Pressure as a rule relieves superficial tenderness.	4. Pressure increases deep tenderness.
5. The superficial reflexes are increased over an area of the superficial tenderness accompanying referred pain.	5. The superficial reflexes are unaltered over the area of deep tenderness accompanying local pain.
6. Referred pain is frequently accompanied by headache and superficial tenderness of the scalp, obeying rules with regard to its distribution which I laid down in my second paper.	6. Local pain is unaccompanied by that form of headache which is associated with scalp tenderness. Headache if present is a separate phenomenon, standing in no direct relation to the local pain and deep tenderness.

A.—*Local Pains in Valvular Diseases.*

It is commonly supposed that local pain is far commoner in cardiac disease than referred pain. This I believe to be a pure assumption. Probably local pain is a distinct feature of the majority of painful cardiac states, but it is usually so masked by referred pain that its presence is very difficult to determine.

But in certain cases of mitral disease, especially when the pulse is irregular, the patient complains of palpitation accompanied by pain. This pain is said to lie over the apex beat, and is unaccompanied by superficial tenderness. It seems to be a true local pain. Now this element must exist in other cases in which referred pain is also present; but the presence of this referred pain masks the local pain, and to say that a true local pain is present is but an assumption.

In the same way, in anginal attacks of the suffocative type [*vide* §7], an initial, stretching, bursting pain is felt at the epigastrium, which is undoubtedly local, and is a phenomenon quite apart from the referred pain. Local pain seems to be most often present when the heart is acting irregularly, and is peculiarly associated with a feeling of breathlessness.

Such local pain in the heart is not, however, definitely associated with deep tenderness, but simply lacks all the points which characterise a referred pain.

B.—*Pain in Pericarditis.*

When the pericardium is acutely inflamed, pain is present, which shows the most striking contrast to referred pain. Like inflammation of the pleura and peritoneum, inflammation of the pericardium produces a true local pain, with more or less marked deep tenderness.

But in the adult, inflammation of the pericardium is frequently associated with marked disease of the endocardium. Thus in adults the true phenomena due to inflammation of the pericardium are frequently masked, and all satisfactory examination rendered impossible by the coincident superficial tenderness due to the endocarditis.

Occasionally, however, the opportunity arises of examining a case in which superficial tenderness is absent. The local character of the pericardial pain and its association with tenderness on pressure and percussion (in the interspaces) are then extremely manifest. It is also interesting to see how closely the area of deep tenderness corresponds in some cases with the area over which friction is audible. Case No. 1 shows all these points distinctly, and happened to be one in which all the characteristics of the local pain were demonstrated to the satisfaction of several other observers.

Case 1.—*Illustrating the local pain and deep tenderness produced by Pericarditis.*—Nellie T. [Marylebone Infirmary, Mr. Lunn], aged 15, servant.

November 16, 1895.—The left knee began to swell and was very painful. She remained in bed two weeks as the doctor said she had rheumatic fever. Towards the end of the two weeks her heart became bad, and she was admitted December 2, 1895.

No previous attacks of rheumatism. Nothing worthy of note in the past or family history.

On admission [*December* 2, 1895].—The face was pale, with a slight underlying grey look, but no actual cyanosis. No œdema. No jaundice. The right wrist was swollen and painful, but the fingers were unaffected. The left wrist is becoming swollen.

Respiration is rapid with evident subjective dyspnœa. The alae nasi are dilating and trembling irregularly. Short, dry, hacking, semi-voluntary cough. Speech interrupted by the shortness of breath. Chest moves with every inspiration, and the respiratory rate is about 50 in the minute.

She complained of pain over the heart, and this pain was much worse when she coughed. The pain lay definitely over an area in the fourth and fifth spaces on the left side, about the size of the palm of the hand. The pain did not go through to the back or radiate round the body. There was no pain elsewhere, and no headache.

The most careful examination failed to reveal any superficial tenderness anywhere. She is a most intelligent girl, clever, and bright beyond her years.

On the other hand, there was exquisitely marked tenderness on pressure and on percussion over an area in the fourth and fifth spaces, about the size of the palm of the hand, and with its centre about in the left nipple line.

Cardiac pulsation is widely visible in the fourth and fifth spaces, and also at the epigastrium.

Cardiac dulness begins above at the upper border of the second rib, extends $1\frac{3}{4}$ in. [4·5 cm.] to the right of the middle line, and 5 in. [12·5 cm.] if not more, to the left of the middle line.

Rough to and fro friction is heard in the fourth and fifth intercostal spaces over an area corresponding very closely to that of the tenderness on precussion and pressure.

Over the apex, and as far inwards as the left sternal border, the first sound is absent, and is replaced by a soft blowing systolic murmur which is not conducted into the axilla. Over the lower part of the sternum, both first and second sounds are audible.

Pulse 124, regular, low tension, compressible with distinct tendency to dicrotism.

No capillary pulsation. Pulsation in the veins of the neck.

Beyond some few crackling sounds at the left base, no abnormal physical signs present in the lungs.

Temperature on admission 102. Fell next day to 100·2.

Urine, specific gravity 1020, acid. No albumen.

Three leeches were applied over the area of deep tenderness. The ease they gave was obvious, and, within five minutes from the time they bit, she said she no longer felt the pain, and that her breathing was easier.

In four days all friction had disappeared, but the signs of fluid in the pericardium were exceedingly marked. The cardiac dulness extended considerably more to the right, and as high as the junction of the first rib, with the sternum on either side. The deep tenderness disappeared, and never reappeared throughout the course of the case.

§ 3. *Referred Pain in Diseases of the Aortic Valves.*

I have begun with the pain which appears with lesions of the aortic valves, because from its position over the upper part of the chest and arm, at a distance from the heart, it is most obviously referred. Again, the association of the presence or absence of pain with certain conjunctions of physical signs is striking. And, lastly, the interpretation of these signs is less open to dispute than is the case with lesions of other valves.

Pain of the character I am about to describe, occurs most typically in young people who, as a sequel to acute rheumatism, present the following typical signs: a diastolic murmur over the sternum, conducted more or less towards the apex beat, accompanied by a systolic murmur over the upper part of the chest, conducted into the great vessels, and absence of the aortic second sound. The first sound is present over the apex beat or in the axilla, and no mitral systolic murmur is audible at the angle of the left scapula.

But sometimes an attack of acute rheumatism leaves behind it a loud systolic murmur, heard over the aortic area, followed by a perfect second sound and no diastolic murmur of any kind. The pulse is peculiar neither for the strong stroke and low tension, characteristic of regurgitation, nor for the small stroke and maintained tension which accompanies marked stenosis. Such cases present no marked symptoms, and pain is absent. The lesion of the aortic valves, though acoustically patent, is probably physiologically of little importance.

Pain is also a frequent symptom in those cases of aortic regurgitation which arise without a history of acute rheumatism in men of middle age and laborious occupation. But here again the aortic second sound is absent. No mitral systolic murmur must be audible, and the pulse must be collapsing in character.

When present the pain tends to be situated over the upper three or four intercostal spaces of the left half of the chest, in the upper interscapular region on the left side of the back, and on the inner side of the left arm as far as the elbow. Pain is also sometimes present in the left infra-clavicular region, and over the left side of the neck. This pain is frequently present on the left side alone, but if it is severe it spreads according to the laws I laid down in my first paper, and involves the same parts on the right side of the chest and back, and the inner side of the right arm. In such cases the pain appears later on the right side than on the left, and disappears from the right side earlier than from the left half of the chest.

When severe the pain is said to shoot and dart, but when less severe it is stationary over certain points. Thus the pain on the inner side of the arm to the elbow, when severe, is said as a rule, to shoot upwards from the elbow to the shoulder, but when less severe to be situated over the inner condyle of the humerus.

If the pain in the chest is severe the patient not uncommonly complains of headache, "like neuralgia," situated in the forehead, "over the nose and eyes." This headache comes and goes with the pain in the chest. It is

more marked when the pains in the chest and arm are severe. It is more marked on the left side of the forehead than the right, and is sometimes confined to the left side of the head, in cases where the pain is confined to the left side of the chest and arm. The eyes are said to ache when the headache is present, and not uncommonly the patient becomes unable to read, owing to the pain in the eyes, even when no error of refraction is present. If, however, latent hypermetropia exists, this hypermetropic defect may become manifest under the influence of the headache.

When the sharpness of the pain has somewhat abated, the patient not uncommonly complains that the chest feels sore. On examination, certain areas of the skin of the chest and arm are found to be tender, just as with every other referred pain of sufficient severity and duration. This tenderness of the superficial structures lies mainly over the second, third, and fourth interspaces in front, the inner aspect of the left arm, and an area of the back, situated roughly between the level of the first dorsal and the sixth dorsal spines. The area involved comprises the second, third, and fourth dorsal segmental zones to a greater or less extent. The third and fourth cervical areas are also frequently affected and the pain is then said to extend above the clavicle.

In the same way the headache is associated with distinct tenderness of the superficial structures of the forehead, occupying mainly the frontal, mid-orbital, and fronto-temporal segmental areas of the scalp.

To illustrate these points I give in detail the case of a young girl with marked double aortic murmurs, retention of the faint sound in the left axilla, and no systolic murmur heard at the angle of the left scapula. [Case No. 5, table i., p. 166.] The pain was never very severe, excepting after some particularly indiscreet exertion, to which these aortic cases are peculiarly prone. The tenderness represented [fig. 1] is that usually present when she was not confined to her bed.

Case 5.—*To illustrate the non-paroxysmal referred pain produced by lesions of the aortic valves.*—Louisa I. (Marylebone Infirmary, Dr. Lunn), aged 19, servant.

At the age of 13 she had rheumatic fever, during which the right wrist and left ankle swelled and became painful. She was more or less ill for ten months. About a year afterwards was suddenly taken bad with a pain in the upper part of the left side of the chest and in the neck. In 1892, at the age of 16, she went to service. She then suffered from attacks of shortness of breath, especially after exertion. In 1893 pain became a marked feature, and she was admitted to the infirmary. She much improved, and was discharged, but was re-admitted in 1894. She again improved, and took another situation, but the pain became so bad she was re-admitted in October, 1895.

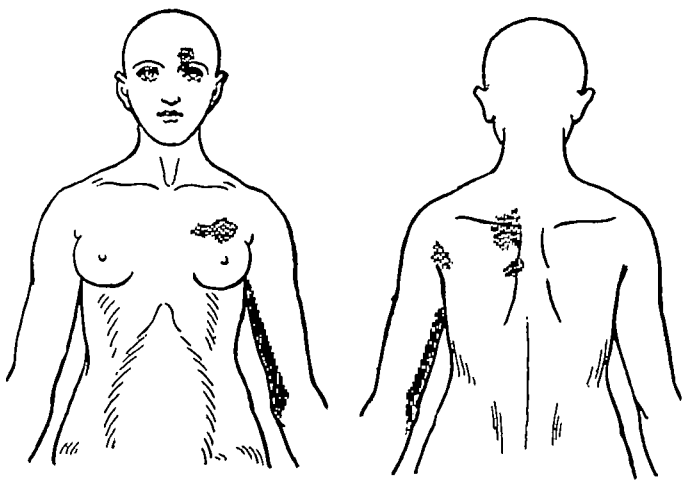

FIG. 1.

To show the areas of superficial tenderness in a Case of Aortic Obstruction and Regurgitation [Case No. 5.]
The area mainly affected is that of the 2nd dorsal segment.
Slight scalp tenderness was present on the left side of the forehead.

December, 1895.—Her condition was as follows :—Very bright, small, slight girl of 19. The greater part of her face is pale, with an irregular underlying vascular mottling. On her cheeks is an irregular pink, mottled flush, which comes and goes. No true anæmia. No œdema. No jaundice. No clubbing of fingers. No nodules. No wasting.

She has no feeling of faintness except with the attacks of pain. When not in bed the heart beats very fast on any exertion, and this seems to bring on the pain. Not short of breath when in bed, but when up and about she has a tight feeling in her chest as if she was going to be choked.

When kept strictly in bed she is free from pain and headache, and all superficial tenderness is absent. When up she is an exceedingly active worker. Pain then appears over the second

left intercostal space, close to the anterior axillary fold, which passes through to the back, close to the vertebral border of the spine of the left scapula. A variable amount of superficial tenderness is present, mainly over the area of the second dorsal segment (fig. 1).

She has occasional headache over the eyes, with slight tenderness of the forehead on the left side (fig. 1).

The pulse, when she is up, is about 100, regular, of good stroke, but typically collapsing. Marked pulsation of the arteries of the neck.

Marked capillary pulsation.

No pulsation of the veins of the neck.

Heart's apex beat in fifth space four inches (10·5 cm.), from the middle line (just outside nipple line). It is heaving. No thrill.

Cardiac dulness beginning at third rib, extends to right edge of sternum, and four and three-quarters inches (12 cm.) to the left (about one inch outside the left nipple line).

Over the whole front of the chest, extending to the mid-axillary line, a loud systolic murmur is heard, followed by a diastolic murmur, forming the typical to-and-fro aortic murmurs. Over the aortic area neither the first nor the second sounds are heard. Over the actual apex beat the first sound is just audible as a dull booming, but on travelling outwards into the left axilla the first sound becomes quite distinct, and is long and booming, in spite of the fact that the aortic systolic murmur is still faintly heard. At the angle of the left scapula a booming first sound is distinctly audible, unaccompanied by any murmur. The pulmonary second sound is audible, but is not accentuated.

Respiration 24, quiet, regular. No cough.

No abnormal physical signs in lungs.

Tongue clean, moist. Appetite good. No pain after food. Bowels irregular.

Urine normal. No albumen.

Vision $\frac{6}{6}$. Jäger 1. No hypermetropia.

Up till April, 1896, the physical signs and main features of the case have remained unaltered.

A marked feature of the pain which appears in aortic disease is the tendency it shows to sudden exacerbations. The pain rapidly spreads, the patient feels exceedingly ill, and, if the spread of the pain is sufficiently rapid and wide, the attack may resemble true primary angina pectoris. Although no hard-and-fast line separates these attacks of increase of pain from the violent anginal attacks which occur in some patients with aortic disease, I shall deal with the whole subject of paroxysmal pain and angina pectoris in a later section [§ 7, p. 202].

All cases of aortic regurgitation, or of regurgitation and stenosis, are liable to suffer from occasional referred pain and tenderness so long as the apical first sound is good and no mitral systolic murmur is heard at the angle of the left scapula. That is to say that so long as the mitral valve holds, pain is liable to appear. If, however, the first sound at the apex and in the axilla is replaced by a systolic murmur conducted to the angle of the left scapula, pain, such as we have learnt to recognise as the possible accompaniment of aortic disease, is absent.

If a patient has long suffered from referred pain and tenderness over the upper dorsal segments, and the mitral valve then fails as a late feature of the disease, it might be said that the pain ceased because the patient's faculties were too low to originate or to appreciate pain.

I have, therefore, collected a series of cases from my notes in which the apical first sound was abolished and replaced by a systolic murmur, conducted to the angle of the left scapula, as a primary feature of the disease. In these cases the mitral regurgitation was not a secondary matter due to heart failure. I have arranged them in a table so that they be compared with a similar table of those cases in which the first sound at the apex was intact, and no systolic murmur was audible at the angle of the left scapula. The absence of referred pain and tenderness in the former group, and its presence in the latter, is striking.

Thus the conclusions to which we have arrived in this section may be summed up as follows :—

(1) When an aortic diastolic murmur, with or without an aortic systolic murmur, is present, the aortic second sound abolished, and, when the pulse shows marked collapsing characteristics, referred pain is liable to form a symptom of the disease.

(2) If in addition to these signs the first sound is absent both over the apex and in the axilla, and a systolic murmur is heard at the angle of the left scapula, referred pain of cardiac origin will be absent. If this condition arises owing to cardiac failure, the pain which was previously present will disappear, and those cases in which this conjunction of

TABLE I.—DISEASE OF THE AORTIC VALVES, WITH A GOOD

No.	Sex & Age.	Apex Beat.	Thrill.	AORTIC.			
				1st.	2nd.	Syst.	Diast.
2	F. 28	$\frac{6}{\text{Axilla}}$	O	O	O	X	X
3	F. 21	$\frac{5}{1\frac{1}{2}\text{ Extl. N.L.}}$	O	?	O	X	X
4	F. 18	$\frac{5}{2\text{ Extl. N.L.}}$	O	O	O	X	X
5	F. 20	$\frac{5}{\text{Just Extl. N.L.}}$	O	O	O	X	X
6	F. 50	$\frac{5}{1\frac{1}{2}\text{ Extl. N.L.}}$	O	O	O	X	X
7	M. 40	$\frac{5}{1\text{ Extl. N.L.}}$	O	O	O	O	X
8	M. 38	$\frac{6}{1\text{ Extl. N.L.}}$	O	O	O	X	X

TABLE II.—DISEASE OF THE AORTIC VALVES, WITH A MITRAL

No.	Sex & Age.	Apex Beat.	Apical Thrill.	AORTIC.			
				1st.	2nd.	Syst.	Diast.
9	M. 26	O	O	O	O	X	X
10	M. 17	$\frac{6}{2\text{ Extl. N.L.}}$	Systolic	O	O	X	X
11	F. 42	$\frac{7}{\text{Ant. axilla}}$	Systolic	O	O	X	X
12	M. 35	$\frac{6}{\text{Ant. axilla}}$	O	O	O	X	X
13	M. 43	$\frac{5}{\text{Nipple line}}$	O	O	O	X	X
14	F. 15	$\frac{5}{1\text{ Extl. N.L.}}$	O	O	O	X	X
15	F. 18	$\frac{6}{2\frac{1}{2}\text{ Extl. N.L.}}$	O	O	O	X	X
16	M. 45	$\frac{6}{2\text{ Extl. N.L.}}$	O	O	O	X	X
17	M. 38	$\frac{5}{1\text{ Extl. N.L.}}$	O	O	O	X	X
18	M. 44	$\frac{5}{1\text{ Extl. N.L.}}$	O	O	O	X	X
19	M. 50	$\frac{5}{1\text{ Extl. N.L.}}$	O	O	O	X	X
20	F. 30	$\frac{5}{1\text{ Extl. N.L.}}$	O	O	O	X	O

In the third column the figure above the line represents the intercostal space in represents in inches the distance outside the nipple line. Thus $\frac{6}{2\text{ N.L.}}$ = apex beat in

In the column devoted to the pulse, *regurgit* means that the pulse showed the *Post-mortem Examinations.* Case No. 9.—Aortic valves incompetent; mitral valve valves incompetent; mitral valve admitted five fingers; left ventricle much hyper- three fingers with ease; left ventricle hypertrophied and dilated. Case No. 18.—Aortic cone, 1·55 in.]; left ventricle dilated and hypertrophied.

Apical First Sound and no Mitral Systolic Murmur.

Mitral. 1st.	Mitral. 2nd.	Mitral. Syst.	Pulm. 2nd.	Pulse.	Veins of Neck.	Liver.	Referred Pain and Superficial Tenderness.
X	X	0	X	80 reg. regurgit.	0	+	Sudden attacks of pain. Tenderness Left Cerv. 3 to Dorsal 5.
X	X	0	+	80 reg. regurgit.	0	0	Left shoulder and arm to elbow. Tenderness C_4 to D_2. Mainly Left.
X	X	0	X	80 reg. regurgit.	0	0	Anginal attacks, *vide* § 7, p. 202.
X	X	0	X	100 reg. regurgit.	0	0	Left shoulder and arm to elbow. Left D_2 mainly, *vide* p. 162.
X	X	0	X	84 reg. regurgit.	0	0	L. neck, shoulder and arm to elbow. Tenderness Left C_4 and D_2.
X	X	0	X	100 reg. regurgit.	0	0	Sudden attacks on exertion. Widespread tenderness L. side.
X	X	0	X	100 reg. regurgit.	0	0	Left arm to elbow.

Systolic Murmur, abolishing the Apical First Sound.

Mitral. 1st.	Mitral. 2nd.	Mitral. Systolic.	Pulm. 2nd.	Pulse.	Veins of Neck.	Liver.	Referred Pain and Superficial Tenderness.
0	?	Conducted to angle of scap.	X	100 reg. regurgit.	0	0	No pain. No tenderness.
0	Feeble	Conducted to angle of scap.	+	108 irreg. regurgit.	0	0	No pain. No tenderness.
0	Feeble	Conducted to angle of scap.	X	100 reg. regurgit.	Pulsation	+	No pain. No tenderness.
0	0	Heard at angle of scap.	X	96 reg. regurgit.	0	0	No pain. No tenderness.
0	0	Heard to mid. axilla	X	92 reg. regurgit.	0	0	No pain. No tenderness.
0	X	Heard all over the back	+	120 reg. regurgit.	0	0	No pain. No tenderness.
0	2	Heard all over the back	+	96 reg. regurgit.	0	0	No pain. No tenderness.
0	X	Conducted to angle of scap.	X	80 reg. regurgit.	0	0	No pain. No tenderness.
0	X	Conducted to posterior axilla	X	68 reg. regurgit.	0	0	No pain. No tenderness.
0	X	Conducted to angle of scap.	X	84 reg. regurgit.	0	0	No pain. No tenderness.
0	0	Conducted to angle of scap.	X	80 reg. regurgit.	0	0	No pain. No tenderness.
0	X	Heard all over the back	+	88 irreg. low tension	Pulsation	0	No pain. No tenderness.

which the maximum pulsation of the apex is seen and felt. The figure below the line th space, 2 in. outside the nipple line.

ow tension collapsing character typical of marked aortic regurgitation.
dmitted five fingers; left ventricle hypertrophied and dilated. Case No. 10.—Aortic rophied and dilated. Case No. 11.—Aortic valves incompetent; mitral valve admitted alves incompetent; mitral valve admitted five fingers [diameter, measured with the

physical signs exists from the beginning will not suffer from referred pain.

(3) The pain, when present, is mainly over the upper part of the chest and back, on the left side and in the left arm. It is accompanied by superficial tenderness over the second, third, and fourth dorsal, and sometimes the third and fourth cervical segmental areas.

(4) Headache over the frontal region is liable to be present. This is accompanied by tenderness over the frontal mid-orbital and fronto-temporal segmental areas on the scalp.

§ 4. *Pain produced by Aneurisms of the Aorta.*

The pain produced by an aneurism of the aorta is usually said to be due to: (1) pressure upon the walls of the chest and the vertebra, or (2) pressure upon nerve cords or roots. Now there is no doubt that these factors play a part in the production of the pain, especially when the aneurism has reached a great size. But in the case of smaller aneurisms, such pressure on surrounding structures plays a comparatively small part in the production of the pain. And yet pain is comparatively more common with the smaller aneurisms than with those of great size.

Now a careful examination shows that the pain is accompanied by superficial tenderness in a considerable proportion of cases of aneurism of moderate size. Moreover, this tenderness marks out areas on the surface of the chest and arm, which are to a great extent the same as those I have described above as concomitant phenomena of the pain of aortic disease. Thus this pain, accompanied as it is by superficial tenderness, is a simple referred visceral pain. For there can be no question of pressure on bones or nerves when the same pain arises as a sequel to aortic disease. And yet both aneurisms and aortic disease may cause pain accompanied by superficial tenderness over almost similar areas. We cannot wonder at this when we remember that etiologically the first part of the aorta is as much part of the heart as the ventricle.

The pain produced by aneurisms of the aorta must therefore always be considered under the following three groups: —

(1) True referred pain accompanied by superficial tenderness of the chest, neck, and arm.

(2) Referred sensations due to pressure on nerve trunks.

(3) Local pain produced by pressure on such structures as the vertebræ and ribs.

The commonest situation for the pain is down the inner side of the arm. Sometimes it only extends to the inner condyle of the humerus, but occasionally and not infrequently it extends over the ulnar side of the forearm to the little finger.

It is more common in the left arm than in the right. I cannot help thinking that if the pulsation of the aneurism appears to the right of the sternum, the pain will radiate down the right arm, whilst if it is visible to the left of the sternum, the left arm will be affected. I have not, however, at present sufficient material to make certain of this statement.

The pain is usually of a dull aching character, relieved by pressure or by rubbing. Thus patients who suffer from this pain frequently make the following characteristic movements: The left arm is somewhat adducted so that the elbow lies over the cardiac area of the chest. The right palm is placed over the left arm, so that the fingers reach over to that aspect of the left arm which is now outwards.[1]

The right hand is then rubbed up and down over the outer part of the left arm.

Occasionally the pain is of a shooting character, and in that case it usually is said to shoot up from the elbow to the chest and back. If it extends to the little finger it shoots up from that finger to the elbow.

The pain sometimes assumes a paroxysmal character, and secondary anginal attacks may be developed. These are considered in § 7.

The pain is accompanied or followed by more or less marked superficial tenderness over the inner side of the arm, and sometimes over the ulnar side of the forearm to the little finger. When carefully marked out these areas

[1] Owing to the rotation of the left arm which accompanies the adduction this portion which now lies outermost is in reality the inner aspect of the arm.

correspond either to the maxima or to the full extent of the second and first dorsal segmental areas.

Now I pointed out in my first paper (p. 33) that the second dorsal area had a limb on the anterior surface of the chest, over the second space and third rib, and a dorsal limb, characterised by a spot close to the vertebral border of the spine of the scapula. In the same way the first dorsal area has a small portion on the back, at the level of the seventh cervical and first dorsal spine. Thus, if the whole of both these areas are represented, pain and tenderness will be present in the second space in front, and over the first and second dorsal spines, and the inter-vertebral groove to the left or the right of them.

Now in many cases this pain has been mistaken for a local pain, and has been ascribed to erosion of the chest in front, and of the vertebræ behind. But examination of the chest shows no signs of an aneurism of sufficient size to cause erosion of both the anterior and posterior walls of the chest. Moreover, the presence of the superficial tenderness shows that these spots are nothing more than the anterior and posterior limbs of segmental areas, of which the lateral limbs are to be found on the inner side of the arm.

But, it might be objected, that this pain down the inner side of the arm is of itself nothing but the expression of pressure on nerve trunks. But if this were so, it is difficult to see why it should appear in diseases of the aortic valves, following acute rheumatism (Case No. 5, p. 162). Again, pressure upon nerve trunks produces numbness and tingling, but not superficial tenderness, as anyone can learn who will compress his ulnar nerve and observe the result.

Moreover, the distribution of the hyperæsthesia is not that of any peripheral nerve or cord of the brachial plexus. Such a case as No. 21 (p. 171) is entirely incompatible with the supposition that this pain down the arm, to which I now refer, could be due to pressure upon nerve roots.

A most interesting motor phenomenon is occasionally associated with pain and superficial tenderness of the areas on the inner aspect of the arm and forearm. The patient complains that the hand feels stiff and numb. The grasp of

the hand is undoubtedly weaker when the pain is present than when it is absent. Thus I have seen a patient, in whom this pain came on owing to the effort of sitting up to eat his dinner, drop his fork from his left hand. When carefully tested, the movements of the fingers of the left hand were clumsy. There was no absolute paralysis, and no profound paresis that could be localised in any particular muscle, but yet all the fine movements of the left hand were badly performed. Movements of the thumb do not seem to

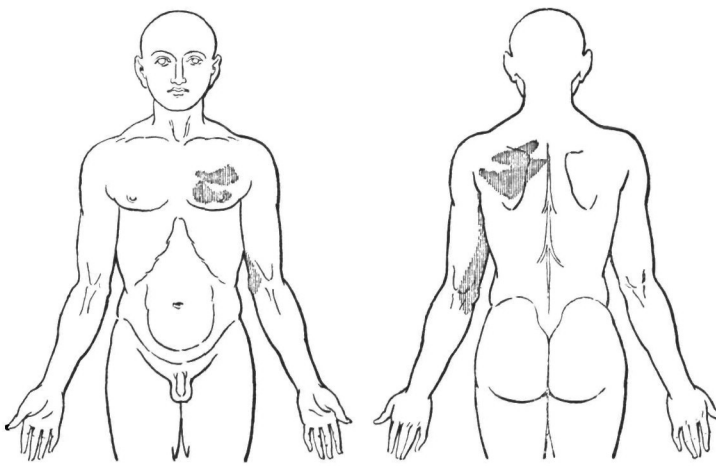

FIG. 2.

To show the superficial tenderness present in a Case of Aneurism of the Ascending Portion of the Aorta [Case No. 21].
The areas mainly affected belong to the 2nd and 3rd dorsal segments.

suffer to the same extent as fine movements of the fingers. This is a very transitory phenomenon, and soon disappears when the pain has gone. The interest of this motor condition lies in the fact that the first dorsal root with the eighth cervical supplies the small muscles of the hand. Thus an extreme disturbance on the sensory side, within the first dorsal segment, seems to cause some motor weakness within the area supplied by the first dorsal and eighth cervical root.

Case 21.—*To illustrate referred pain produced by aneurism of the ascending aorta.*—Charles T. (V.P.H., Dr. Eustace Smith). Aged 31, carpenter. Admitted, December 8, 1893.

An extremely strong healthy man till seven years ago, when he had a "hard chancre." Was treated with mercury. No secondary rash or other sequelæ.

Two years ago began to suffer from pain in the chest. He is a carpenter, and the pain came on particularly after using the heavy plane. September 22.—The pain became so bad he gave up work.

On admission.—He is a very finely built man of six feet. Face of a uniform pinkish colour, with no flush. No anæmia, jaundice, or œdema. Well nourished.

He is conscious of the pulsation of the swelling. No palpitation or fainting.

He states that three weeks before admission he suffered from violent pain in the left arm, which began in the second phalanx of the little finger and travelled upwards to the inner side of the elbow. The pain then extended as an intense aching over the whole of the ulnar half of the left forearm and in the little finger. Occasionally it darted and shot, and sometimes darted from the inner aspect of the elbow to the joint between the first and second phalanges of the little finger. This pain disappeared a week before admission, and has given place to the pain from which he now suffers. Pain is now present over the inner side of the arm as low as the fold of the elbow. Its point of greatest intensity is situated one inch (2.5 cm.) above the inner condyle of the humerus.

Some distinct superficial tenderness is present over an area extending upwards from the internal condyle of the humerus over the inner aspect of the left arm.

Visible pulsation in the first and second intercostal spaces. In the second space not only pulsation but a distinct systolic thrill can be felt.

The apex beat can be felt in the fifth space in the left nipple line. Impulse is feeble.

There is no bulging of the chest wall over the left front, but the percussion note is high pitched, and there is increased resistance over the upper part of the chest on the left side, extending from the first intercostal space to the fourth rib, and laterally from the left sternal border almost to the anterior axillary fold.

The cardiac dulness extends from the nipple line on the left to the mid-sternum on the right. Above, it merges into the dulness over the aneurism.

A loud systolic murmur is heard over the upper part of the chest on the left side, with its maximum in the second intercostal space. It is followed by an accentuated second sound.

Over the apex the first sound is weak, and is followed by a very soft systolic murmur, which disappears in the left axilla, but increases in intensity along the left border of the sternum to reach its maximum over the area of pulsation. Thus this murmur at the apex seems to be the conducted aneurismal murmur.

No diastolic murmur heard anywhere.

Pulse 80, regular. Artery large and full between the beats. The pulses seem to be equal.

No dyspnœa. No cough. Voice good. Larynx normal.

No abnormal signs in the lungs.

Tongue clean, appetite good. Bowels opened daily.

Liver not enlarged.

Urine 1020, acid. No albumen.

Pupils equal; react to light and accommodation. Both pupils dilate to shade.

December 13.—The pain extended down the ulnar side of the forearm to the little finger for a few hours.

December 14.—It is now represented by pain over the inner condyle, with definite superficial tenderness of inner aspect of arm.

The pulsation is decidedly more marked.

He was put strictly to bed, and the pulsation distinctly diminished, so that by March 5, 1894, the pulsating area in the second space could be covered by the top of the thumb.

Pain was practically absent throughout this period.

March 7.—He again began to complain of pain down the inner aspect of the arm, and marked superficial tenderness was present over the second dorsal segmental area. The pulsation is now visible over an area of three inches by one and a half inches (7·5 cm. by 3·75 cm.).

March 31.—The pulsating area has again increased, and has returned to the size on admission. The pulsation is accompanied by a marked systolic thrill, and the closure of the aorta semilunar valves can be felt over the pulsating area as a sharp slap. Apex beat and cardiac dulness as before.

Over the whole left side of the chest, as low as the fifth rib, a greatly accentuated second sound is audible. The systolic murmur is more widely heard over the upper part of the chest. No murmur or second sound audible in the back. Over the apex beat the first and second sounds are good, and are not obscured by the systolic murmur.

He now complains of pain—(1) over a spot just internal to the nipple in the third space. He places a single finger over this spot, and says that when the pain gets bad it enlarges. This pain passes through to a point in the back in the interscapular space, at a level of about the fourth dorsal spine. He says it is exactly as if someone were cutting him with a knife.

He also has some pain over the second rib, extending through to a point behind, close to the vertebral border of the spine of the scapula.

(2) He also has a dull aching "numb" feeling over the inner aspect of the left arm and ulnar aspect of the left forearm, to the ulnar side of the wrist, ulnar border of the hand, and to the head of the basal phalanx of the little finger. He is exceedingly precise in his description of the limits of this pain. When this

pain is bad he has loss of power in the fingers of the left hand, and very slightly in the thumb. The fingers feel "silly and useless." He could not grip his fork at dinner.

There is absolutely no loss of sensation to touch, pain, heat, or cold, but there is marked and definite superficial tenderness (hyper-algesia) over the areas in fig. 2.

Pulses equal. Pupils dilate to shade, and are equal. Larynx normal.

But with aneurism of the aorta, pain makes its appearance over other regions than these. Pain is sometimes situated in the left shoulder joint and in the supra-clavicular fossa and back of the neck. This pain may be accompanied by superficial tenderness over the third and fourth cervical areas as in the following case (No. 22). Now it is quite impossible that this pain above the clavicle could be due to pressure on nerve cords or roots, and yet, in this case, we see the pain and tenderness over the second and third dorsal merging imperceptibly into that above the clavicle.

Case 22.—*To illustrate the pain and tenderness over the cervical areas produced by aneurism of the aorta.*—James C. (V.P.H., Dr. Thorowgood), aged 43, sawdust dealer.

I first saw him December 11, 1893. He had suffered from pain in the back for six months. Six weeks before admission a cough came on, and the pain became much worse. He denies gonorrhœa and syphilis. Married, but wife has never been pregnant. No general illness. Not intemperate (two to three pints of beer daily).

On admission he complained of pain above and outside the left nipple, darting through to a point just below the spine of the scapula behind. He also had pain down the inner side of his arm to the elbow. Beautifully marked superficial tenderness was present over these spots.

The physical signs were those of aneurism of the aorta (see Part II. of this work, BRAIN, 1894, p. 371).

The right pulse was stronger than the left.

The pupils were normal and equal. Larynx unaffected.

He left the hospital free from pain, but on returning to work the pain and tenderness returned. But now the tenderness had spread to the fourth cervical area in the supra-clavicular fossa (*vide* fig. 16, p. 371, BRAIN, 1894).

Since this time he has frequently attempted to return to his somewhat heavy work, and each time the pain returned.

March 7, 1896.—The pain is distinctly increasing somewhat. It is still confined entirely to the left side of the chest, shooting through from the scapula to the upper spaces in front, from the

back of the neck to the left shoulder, and down the inner side of the arm to the elbow.

There is beautifully marked superficial tenderness as in fig. 3.

He complains of headache over the left half of the forehead, and this headache is accompanied by scalp tenderness, as in fig. 3.

No fainting. Occasionally feels faint and has to sit down. No marked palpitation. Becomes very short of breath on exertion.

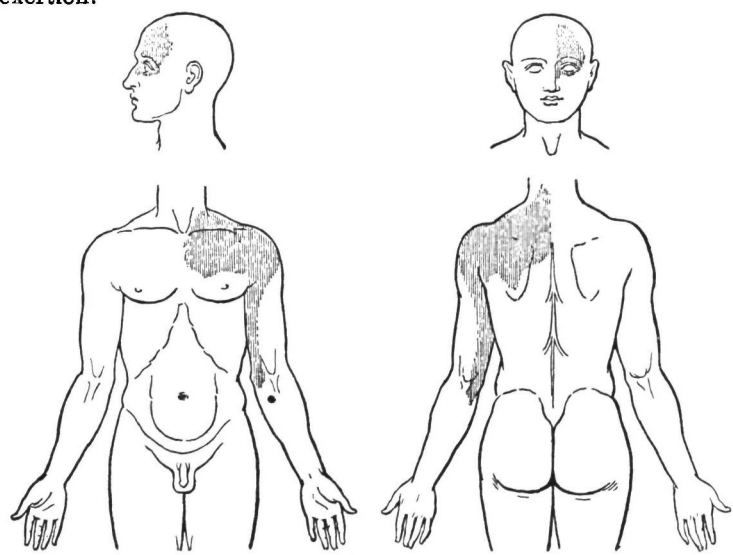

FIG. 3.

To show the superficial tenderness present in a Case of Aneurism of the Aorta [Case No. 22].

The areas mainly affected are those of the 4th cervical and 2nd and 3rd dorsal segments.

Tenderness of the scalp was present on the left side only, over the frontal, mid-orbital, and fronto-temporal areas.

Pulse 64; left pulse decidedly weaker than the right, all up the arm. No pulsation in the neck.

Cardiac pulsation in fifth space and left nipple line ($4\frac{3}{4}$ in. from middle line of sternum). Not heaving. No other pulsation visible or palpable over the chest.

Slight dulness or diminution of resonance over the upper part of the sternum. Cardiac dulness extends from left nipple line to right edge of sternum.

Over the upper part of the sternum and in the second and third right interspaces, a loud accentuated second sound is audible. This second sound is doubtfully heard to the left of the

second dorsal spine behind. A loud systolic murmur replaces the first sound over the aortic area. It is conducted down the left edge of the sternum. At the apex the first and second sounds are good. No murmur heard in the left axilla or at the angle of the left scapula. No tracheal tugging. Larynx normal. Pupils normal.

The same dulness at the right base to the angle of the right scapula, with absent breath-sounds and vocal resonance. These signs have remained unaltered since December, 1893.

This brings me to a point of much theoretical interest and importance. Some cases of aneurism, I believe mainly those which involve the arch of the aorta, complain of pain in the throat. This pain is described as "a kind of cramped feeling," "a sort of painful stiffness," and is situated in the space between the anterior border of the sterno-mastoid and the middle line on one or both sides. This pain is accompanied by superficial tenderness over an area which I have called the inferior laryngeal, an area which is shared by the structures of the larynx proper (fig. 4). Case No. 23 shewed this pain and superficial tenderness to a marked degree. I believe it is particularly liable to occur in those cases in which either the cervical sympathetic (shown by the pupil) or the recurrent laryngeal, or both nerves are affected, that is to say, where there are signs pointing to the arch of the aorta as the situation of the aneurism.

Case No. 23.—*To illustrate pain and tenderness over the areas of the front of the neck in aneurism of the aortic arch.*—Jane R. (Marylebone Infirmary, Mr. Lunn), aged 53 years, married, charwoman. Admitted November 17, 1895.

October, 1894.—She had a cough and some pain in the back. This did not disappear, and in January, 1895, she went to the Brompton Hospital and was admitted. Thence she was transferred to the Middlesex Hospital, where she remained till her admission to the Infirmary. Throughout this time she has suffered from more or less pain. When she got up the pain grew worse; when she lay in bed it was easier.

Menstruation ceased twelve years ago. Married at 21. Never pregnant. No miscarriages. Never any discharge from the vagina.

Temperate. No history of acute rheumatism or any illness except, possibly, typhoid fever.

On Admission.—She is a well-built, bright, cheerful looking woman of 52. Looks somewhat older. No anæmia. No jaundice. No œdema. Is not wasted.

She says that her heart beats very fast on exertion, but never seems to stop or beat irregularly. This rapid beating is not accompanied by any pain. No shortness of breath whilst in bed. She can lie flat at night.

She has no pain now, and there are no signs of any definite superficial tenderness. No headache.

Pulse 68, regular. Rather small stroke but of well maintained tension. The arterial wall is a little hard. The right pulse is if anything a little stronger than the left.

Apex beat cannot be definitely localised. No thrill. No obvious pulsation seen or felt over the upper part of the chest.

Cardiac dulness begins in the third space, extends $3\frac{1}{4}$ in. (8 cm.) to the left (left nipple line), and to the middle of the sternum on the right. No dulness elsewhere over the chest.

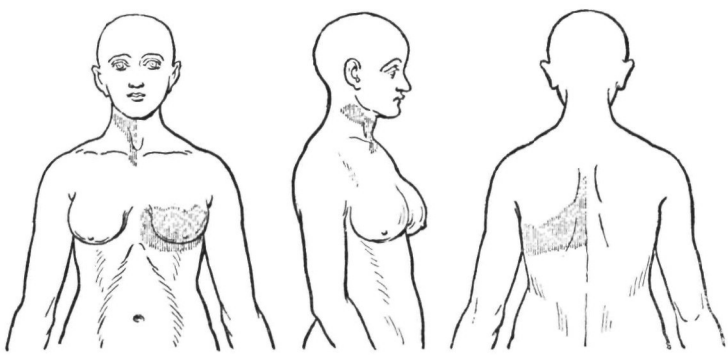

FIG. 4.

To illustrate the pain and tenderness over the Inferior Laryngeal Area of the Neck sometimes present in Aneurism of the Arch of the Aorta [Case No. 23].

Superficial tenderness was also present in this case over the 5th and 6th dorsal areas on the left side of the chest and back.

Over the upper part of, and especially to the right of the sternum, an accentuated slapping second sound is heard. Over almost the same area a soft blurring systolic murmur is audible which is conducted slightly towards the apex. The first sound over the aortic area is feeble. Over the apex the systolic murmur is just audible, and the first and second sounds are good. Neither the second sound nor the systolic murmur are heard anywhere in the back.

No cough now. No marked abnormality in the physical signs of the lungs.

Liver not enlarged.

The voice is a little hoarse, no tracheal tugging. The left vocal cord does not move so well on phonation as the right.

The left pupil under ordinary circumstances is almost 3 mm. in diameter, and is equal to the right. It practically does not react to light or dilate to shade. With a strong light the difference between the pupils becomes very marked. The left pupil dilates somewhat, but not to the extent of the right, under the influence of cocaine. Both pupils react well to accommodation. There is no marked difference in the size of the two palpebral fissures or in the projection of the eyes.

Urine normal.

She remained absolutely in the above condition till the afternoon of January 14, 1896. She then suddenly began to scream with the pain in the head and abdomen. She complained of pain across the upper part of the abdomen on the left side, and over the back below the left shoulder blade. Intense widespread superficial tenderness of chest and back, more marked on the left side. Very marked tenderness over the frontal, mid-orbital, fronto-temporal and temporal areas of the scalp.

Pulse 64, with the same doubtful difference between the two sides.

There is now a distinct fulness over that part of the first and second intercostal spaces on the right side, which lies on each side of the second rib cartilage; thus the second rib cartilage has the appearance of being bulged. A systolic impulse and at times a systolic thrill is felt over this area, followed by a shock corresponding to the second sound.

A loud systolic murmur is heard over the second right rib cartilage, followed by a loud bell-like second sound.

The cardiac signs as before. No sign of dilatation of the left ventricle.

Nothing heard elsewhere in the chest.

The left pupil is markedly smaller than the right, and does not dilate at all to shade.

From this point onwards the pain and superficial tenderness lay mainly within the fifth, sixth, and seventh dorsal areas on the left side. (Fig. 4.) The headache and scalp tenderness were more temporal than before.

March 11.—She still has the pain and tenderness in the left side, but she has just begun to complain of a fresh pain on the right side of the throat. She places her right hand flat over the right side of the space between the anterior borders of the sterno-mastoids, the fingers almost touching the lobule of her ear, and the ulnar border of the hand parallel to the lower border of the jaw. She complains that it is an aching pain like the pain on the left side of the back.

There is more exquisitely marked superficial tenderness over the right inferior laryngeal area (*vide* fig. 4). On the left side of the trunk is seen the tenderness which has been present ever since the attack on January 14.

Occasional slight cough. No alteration in the voice. No marked difference in the movements of the two vocal cords. No

intra-laryngeal cause for the pain discoverable. Cords a normal colour.

Pulse of small stroke. The right pulse is slightly stronger than the left.

There is slight but distinct dulness in the first and second spaces on the right side. Over this area a distinct systolic thrill can be felt. This dulness joins with the upper limit of cardiac dulness across the sternum.

The same systolic murmur and markedly accentuated second sound are audible over the upper part of the right chest.

The left pupil acts to accommodation, but does not dilate in the least to shade.

She became freer of pain, but slight pain and superficial tenderness were still present over the right infra-laryngeal area on April 13, 1896.

The pain of aneurism usually improves greatly under rest and treatment, and all pain and tenderness may disappear after a time. But these cases are notoriously disappointing. Physician and patient are full of hope, when, without obvious cause, or perhaps in consequence of some increased exertion, the pain suddenly returns, not uncommonly with great violence. Suppose the pain and tenderness lay originally over the upper areas of the chest and down the inner side of the arm; with a sudden return of this kind, the pain and tenderness may no longer lie over the upper part of the chest, but may occupy areas under the heart, in the epigastrium and below the shoulder blade (case 23). The tenderness, which previously lay within the fourth cervical and first and second dorsal segments, may now occupy the fifth, sixth or seventh dorsal.

Now we cannot suppose that a new aneurism has suddenly sprung up four inches lower down in the region of the diaphragm, which is above the level of the new outburst of pain. Moreover, the superficial tenderness shows that the pain is referred and not local. Therefore I think we must assume that that portion of the aorta which causes referred pain over the first and second dorsal segments and over the inferior laryngeal area, lies close to that part which causes referred pain over the fifth, sixth and seventh dorsal segments. Now this distribution of pain and tenderness seems to me peculiarly liable to be present in those aneurisms which involve the cervical sympathetic or the recurrent laryngeal,

that is to say, aneurisms of the arch. I shall show reason to believe that the aorta beyond the position of the ductus arteriosus refers into the mid-dorsal region, whilst the aortic bulb and arch refer on to the higher dorsal and inferior laryngeal areas.

Some aneurisms are painless from the beginning or become so in course of time, and many observers have commented on the absence of pain in aneurisms sufficiently large to cause marked symptoms of pressure on the trachea and bronchi. I give as an instance of such a condition case No. 24, where, though there was a history of pain, no referred pain or tenderness was present during the ten weeks he was under observation.

The explanation of this absence of referred pain and superficial tenderness seems to me to be as follows:— The walls of such aneurisms are composed of matted connective tissue, and all true aortic wall has long ago disappeared. The aorta is markedly atheromatous and it is these atheromatous patches which yield to the pressure within. Connective tissue becomes thickened around the yielding patch on the outer side of the aortic wall, and thus the aneurism gradually grows, not by a true distension of the aortic wall, but by the stretching of a wall composed of connective tissue. Thus the wall of the organ, the integrity of which is necessary for the production of referred pain, is no longer present, or is only present in such a condition that the nerves are long past transmitting afferent impulses to the central sympathetic system. Thus such an aneurism may cause local pain by pressure on the structures within the chest, or pain and anæsthesia in the course of nerve cords or roots, but will not cause referred visceral pain accompanied by superficial tenderness.

Case No. 24.—To show how an aneurism that causes marked pressure symptoms may be painless.—William M. (V. P. H. Dr. Eustace Smith) age 53, an engine fitter.

In 1892 he began to suffer from constant pain in the right axilla. This gradually died away. In February, 1893, after all pain had died away, he began to suffer from shortness of breath which gradually became constant. He then began to cough, and

during the summer of 1893 noticed that his expectoration was occasionally streaked with blood.

At the age of 23 he says he had syphilis. No secondary symptoms. Has drunk to excess occasionally. No gout. No rheumatism.

On Admission (*February* 7, 1894).—Medium-sized man of 53. Face pale and blotchy, with dilated vessels on the cheeks but no true flush. Lips and ears a little blue. Fingers and toes much clubbed. Lower part of the back œdematous.

He complains of palpitation, but only on exertion. It did not cause pain. Short of breath even when in bed; he cannot sleep except when propped up.

He complains of a pain on exertion situated over the epigastrium. He has no other pain. No pain in the arm or upper part of the chest. There is no superficial tenderness anywhere, not even over the situation of the epigastric pain. No headache. No scalp tenderness.

Pulse 88, regular, fair stroke and good tension. Arterial wall distinctly hard. Pulses equal. No capillary pulsation. No venous pulsation in the neck.

General heaving impulse in fourth and fifth spaces. No definite apex beat. No pulsation visible elsewhere. No thrill.

Cardiac dulness much diminished. No dulness over the upper part of the chest.

Over the upper part of the sternum and to either side of it is heard a low-pitched systolic murmur. This murmur is conducted down the sternum, but is not heard at the apex of the heart. It is heard in the left interscapular fossa and over the spine with greater intensity than in front. The aortic second sound is heard but is not increased. Pulmonary second sound short and sharp. At the apex first sound is booming, second sound good.

Respiration about 30. Distinct dyspnœa. Alæ nasi dilating and sterno-mastoids acting. Harsh, dry, tearing cough, more troublesome at night. He brings up a small quantity of tough, aerated mucous expectoration.

Chest somewhat rigid. Movements very poor at both bases, but about equal on the two sides. Hyper-resonant note all over the chest. Breath sounds and vocal resonance good at apices but weak over both bases. Moist râles over both bases.

Tongue red, fairly clean and moist, no pain after food now. Bowels confined.

Liver can just be felt. Spleen not felt.

Urine, sp. gr. 1025, acid. No albumen. No sugar.

He had a very irregular temperature, rising sometimes to 102° F. (39° C.), but frequently not reaching 100 as the highest point in the day. He began to spit up small round masses of muco-purulent material in considerable quantity, and signs first of consolidation and then of excavation began to appear over the right lung. By April 15 the whole right side was implicated, and he died on April 18.

Throughout the whole of this time he suffered from no pain, except that at the epigastrium. No referred headache or tenderness.

Post-mortem.—A sacculated aneurism was found about the size of a large orange. It began below, about ¾ in. (2 cm.) above the aortic valves, and involved the greater part of the transverse arch of the aorta, including the origin of the innominate and left carotid arteries. The right branch of the pulmonary artery was extremely flattened and seemed to be running in the substance which formed the wall of the aneurism. Its lumen was almost completely occluded. The left branch of the pulmonary artery was stretched over the front of the aneurism, and its lumen was diminished but not occluded. The aneurism was filled with laminated clot and its walls showed no recognisable remains of the normal aortic lining. They were formed of tough more or less uniform connective tissue. Where not affected by the aneurism the aorta was intensely degenerated. The wall contains much calcareous matter, and in places the coats are infiltrated with blood. Right ventricle much dilated and hypertrophied. Left ventricle small, normal. Aortic valves slightly atheromatous but competent. Mitral valves normal.

Right lung contained one large ragged walled cavity filled with blood-stained stinking grumous fluid. The greater part of the lung was gangrenous, apparently from occlusion of the right pulmonary artery. Left lung normal.

To sum up the conclusions to which we have arrived in this section :—

(1) Aneurism of the aorta may cause referred pain, associated with superficial tenderness over the following areas: —(*a*) Third and fourth cervical and first, second and third, and occasionally fourth, dorsal segmental areas usually on the left side but occasionally on the right side. (*b*) Over the inferior laryngeal area on one or both sides. (*c*) Over the areas from the sixth to the eighth dorsal. These areas appear when we have reason to suppose the aneurism has spread beyond the point where the ductus arteriosus enters the aorta.

(2) Aneurism of the aorta may cause local pain from pressure on structures in the chest other than nerves.

(3) Aneurism of the aorta may cause pain, and occasionally, though rarely, anæsthesia from pressure on peripheral nerves or roots. Such pain is not associated with superficial tenderness over segmental areas. Contrary to the

usual opinion this is the least common cause of pain in aneurism.

(4) Aneurisms which arise in a highly diseased aorta (case 24) or such as are bounded by matted connective tissue only, cause only local and no true referred visceral pain.

§ 5. *Referred Pain in Disease of the Mitral Valves.*

There are no lesions about which greater difference of opinion exists than those of the mitral valve. For at one time the presence of a systolic murmur over the apex in organic cardiac disease at once led to the diagnosis of mitral regurgitation. That the murmur is due to some regurgitation through the mitral orifice is highly probable, but we want to know whether it is the main lesion or not. Broadbent and Graham Steell[1] have long taught that this murmur alone tells us very little with regard to the condition of the mitral orifice, and that the important fact is the relation of the murmur to the first sound of the heart. Thus the only sign of marked mitral stenosis may be a systolic murmur accompanied by a sharp first sound at the apex. In such a case the murmur is that of regurgitation, but the important pathological factor in the physics of the circulation is stenosis of the mitral orifice.

In the course of this research I soon found that it was quite impossible to classify my observations on the referred pain that occurred with lesions of the mitral valves according to the name of the supposed valvular lesion. For in any one case competent observers would disagree in how far regurgitation or stenosis were the main factor. Now the presence or absence of referred pain depends on the conditions within the cavities of the heart. As these conditions can only be inferred from a knowledge of the conditions of the valves, such fundamental differences of opinion amongst experts formed a severe hindrance to me in the earlier stages of this work. With regard, however, to the actual physical signs, I found no such difference of opinion; the difference lay rather in the interpretation of these signs.

[1] Broadbent, *American Journal of Med. Sciences*, No. 181, n.s., Jan., 1886. Graham Steell, *Med. Chronicle*, May, 1888; Sept., 1895.

Throughout this section I shall therefore speak as little as possible of mitral stenosis or mitral regurgitation, but shall attempt to show that, with lesions of the mitral valve, the presence of certain conjunctions of physical signs is associated with referred pain, whilst when certain other physical signs are present, pain is absent. My ideas of the significance of these physical signs are relegated to a special theoretical chapter (chap. ii.).

By adopting this plan of speaking in terms of physical signs I hope that my observations will stand the test of time, though my ideas of the significance of these signs may be falsified by future research. I am certain that if this plan had been adopted by all writers on the subject of mitral disease, much of the present confusion would have been avoided.

In this section I shall only treat of those cases in which the right heart has not failed. For, as soon as the liver begins to be enlarged, pain may make its appearance which is not directly due to the heart. Such cases are reserved for § 6. Thus the auscultatory signs which most concern us here are the mitral diastolic, præsystolic, and systolic murmurs, and the conditions of the apical first and second sounds. Of the diastolic and præsystolic murmurs nothing need be said, but a few words are necessary about the systolic apex murmur.

A systolic murmur heard at the apex may weaken or abolish the first sound, so that the first sound may be absent over the apex, in the axilla, and at the angle of the left scapula. On the other hand, the same murmur may be accompanied by a short, sharp, first sound, such as is usually heard in conjunction with a præsystolic mitral murmur. As Steell[1] has pointed out, a systolic murmur of this kind, associated with a sharp, first sound, is usually not conducted into the axilla, or heard at the angle of the left scapula. But he recognises that this conduction or non-conduction to the angle of the left scapula is an unsafe guide. Thus the real point of importance is not the conduction of the murmur, but the fact that it is associated with a sharp

[1] *Med. Chronicle*, May, 1888, and Sept., 1895.

first sound, either at the apex or in the axilla. That these observations of Broadbent and Steell are of vital physiological importance, and not simply of academic interest, is borne out by the value of these signs as a basis for the classification of a physiological condition such as referred pain.

(*a*) Whenever the signs of old endocardial mischief consist solely of a systolic murmur heard at the apex, and no first sound is heard either at the apex or in the axilla, or at the angle of the left scapular, referred pain is absent as a marked feature of the case.

I carefully exclude from this statement all cases where a systolic murmur is heard owing to the failure of a heart that has hypertrophied from other causes than endocarditis (*e.g.*, chronic Bright's disease). I also exclude those cases of acute rheumatism in which a systolic murmur appears during the attack. Table III., p. 186, shows a series of cases in which these conditions were fulfilled. They exhibited neither referred pain, referred headache, nor superficial tenderness of the chest or scalp throughout a period of observation extending from a minimum of six weeks up to a year.

Such cases, however, suffer from palpitation on exertion, and this palpitation is accompanied by shortness of breath and occasionally by a pain which is said to be situated over the apex of the heart. This pain does not go through to the back or round the body like a referred pain, and is unaccompanied by headache or by superficial tenderness of the trunk or scalp. It is apparently a true local pain.

(*b*) The second group of cases consists of those in which a mitral diastolic murmur is audible together with a systolic murmur. The systolic murmur is widely heard and conducted to the angle of the left scapula. No first sound is audible either at the apex or in the axilla, or at the angle of the left scapula.

Such physical signs are not associated with referred pain connected with the heart. As, however, the later stages of mitral stenosis fall into this group, referred pain may be

TABLE III.—MITRAL SYSTOLIC MURMURS AT THE APEX, OBSCURING

No.	Sex & Age.	Apex Beat.	Apical Thrill.	APEX.		
				1st.	2nd.	Systolic.
25	F. 17	5 / Just Extl. N.L.	Systolic	O	O	Heard widely over back
26	F. 14	5 / 1 Extl. N.L.	O	O	X	Conducted to angle of scap.
27	F. 12	5 / ½ Extl. N.L.	Systolic	O	X	Heard widely over back
28	F. 21	5 / In N.L.	O	O	X	Heard widely over back
29	F. 23	5 / ½ Extl. N.L.	O	O	2	Heard widely over back
30	M. 16	5 / In N.L.	Systolic	O	2	Conducted to angle of scap.

TABLE IV.—MITRAL DIASTOLIC ACCOMPANIED BY SYSTOLIC MURMUR AT

No.	Sex & Age.	Apex Beat.	Apical Thrill.	APEX.		
				1st.	2nd.	Systolic.
31	M. 23	5 / In N.L.	Diastolic	O	2	Conducted to angle of scap.
32	F. 19	5 / In N.L.	O	O	X	Conducted to angle of scap.
33	M. 42	6 / 1 Extl. N.L.	O	O	2	Conducted to angle of scap.
34	M. 18	5 / 1 Extl. N.L.	Diastolic	O	2	Conducted to angle of scap.
35	F. 17	6 / 1 Extl. N.L.	Systolic	O	O	Conducted to angle of scap.
36	F. 18	6 / ½ Extl. N.L.	O	O	O	Conducted to angle of scap.

The abbreviations are the same as on Tables I. and II.

In the column devoted to the Pulse *Maintained*, means that although the pulse- "Virtual Tension"]. On the other hand, *low tension* means that the pulse is com-

In the column devoted to the 2nd sound, 2 means that this sound is re-duplicated;

In the column devoted to the 1st sound at the apex, + means that this sound is

THE FIRST SOUND AT THE APEX, AND IN THE LEFT AXILLA.

Diast.	Presystolic.	Pulm. 2nd Sound	Pulse.	Veins of Neck.	Liver	Lungs.	Referred Pain and Tenderness.
0	0	+	100 reg. low tension	0	0	Crackles at bases	No pain. No tenderness.
0	0	+	84 reg. low tension	Slight pulsation	0	R. base crackles	No pain. No tenderness.
0	0	+	120 reg. low tension	Slight pulsation	0	Crackles at bases	No pain. No tenderness.
0	0	+	116 reg. low tension	Slight pulsation	0	0	No pain. No tenderness.
0	0	+	84 reg. low tension	0	0	0	No pain. No tenderness.
0	0	2+	66 reg. low tension	Slight pulsation	0	0	No pain. No tenderness.

THE APEX, OBSCURING THE FIRST SOUND AT THE APEX AND IN THE AXILLA.

Diast.	Presystolic.	Pulm. 2nd Sound	Pulse.	Veins of Neck.	Liver	Lungs.	Referred Pain and Tenderness.
X	0	+	74 irreg. maintained	0	0	0	No pain. No tenderness.
X	0	+	120 reg.	0	0	0	No pain. No tenderness.
X	0	+	60 irreg.	0	0	Crackles both bases	No pain. No tenderness.
X	0	+	88 reg. maintained	0	0	L. base	No pain. No tenderness.
X	0	+	76 reg.	0	0	Crackles L. base	No pain. No tenderness.
X	0	2	52 irreg.	0	0	0	No pain. No tenderness.

stroke is small, the tension, such as it is, is maintained between the beats [Broadbent's
pressible, and whatever the initial stroke the tension is low between the beats.
2+ means that the 2nd sound is re-duplicated, but one element is accentuated.
short, sharp, and high-pitched, as is typically found in marked mitral stenosis.

TABLE V.—SYSTOLIC MURMURS HEARD AT THE APEX

No.	Sex & Age.	Apex Beat.	Apical Thrill.	APEX.		
				1st.	2nd.	Systolic.
37	M. 21	5 / Intl. N.L.	O	+	X	Not conducted
38	F. 36	5 / Extl. N.L.	O	+	X	Not conducted
39	F. 22	5 / ½ Extl. N.L.	Presystolic	+	X	Conducted
40	F. 29	5 / Intl. N.L.	Diastolic	+	X	Not conducted
41	F. 33	5 / Intl. N.L.	O	+	X	Not conducted
42	F. 17	5 / 1 Extl. N.L.	Diastolic	+	2	Conducted
43	M. 42	5 / Intl. N.L.	O	+	X	Not conducted
44	F. 38	5 / Intl. N.L.	Systolic	+	X	Not conducted
45	F. 42	5 / In N.L.	Systolic	+	X	Not conducted
46	F. 36	5 / 1 Extl. N.L.	Diastolic	+	?	Not conducted

TABLE VI.—MITRAL DIASTOLIC OR PRESYSTOLIC MURMUR. NO

No.	Sex & Age.	Apex Beat.	Apical Thrill.	APEX.		
				1st.	2nd.	Systolic.
47	F. 24	5 / N.L.	O	+	X	O
48	M. 27	5 / N.L.	O	+	X	O
49	M. 30	5 / N.L.	Diastolic	+	X	O
50	M. 30	5 / Intl. N.L.	O	+	X	O
51	M. 37	5 / Intl. N.L.	Presystolic	+	X	O
52	F. 49	5 / N.L.	Presystolic	+	X	O
53	F. 50	5 / N.L.	O	+	X	O

The abbreviations are the same as on Tables I. and II.

In the column devoted to the Pulse *Maintained*, means that although the pulse- " Virtual Tension "]. On the other hand, *low tension* means that the pulse is com-

In the column devoted to the 2nd sound, 2 means that this sound is re-duplicated;

In the column devoted to the 1st sound at the apex, + means that this sound is

Post Mortem Examinations. Case No. 43.—Mitral valve much stenosed [diameter Mitral valve much stenosed; converted into flattened funnel [diameter with cone

TOGETHER WITH A SHORT, SHARP, FIRST SOUND.

Diast.	Presys-tolic.	Pulm. 2nd Sound	Pulse.	Veins of Neck.	Liver	Lungs.	Referred Pain and Tenderness.
X	?	+	76 reg. maintained	O	O	O	Stomach, back, tmpls. L. D_6 & D_7
O	O	+	100 reg. maintained	Pulsation	O	O	Stomach & back. D_5 to D_8 L. & R.
O	X	+	100 reg. maintained	O	O	Crackles both bases	Stomach & back. L. D_6 and D_7.
X	X	+	64 reg. maintained	O	O	Crackles both bases	Stomach & back. D_6 and D_7 R. & L.
O	O	+	76 reg. maintained	O	O	O	L. breast & back. L. D_5 to D_8.
X	O	+	88 irreg. maintained	Pulsation	O	Crackles both bases	L. breast & back. L. D_6.
	O	+	88 irreg. maintained	O	O	Crackles both bases	Stomach & back. L. D_6 and D_7.
O	O	+	100 reg. maintained	O	O	Crackles both bases	L. breast & back. L. D_5, D_6, D_7.
X	O	+	76 reg. maintained	O	O	O	L. breast & back. L. D_6.
X	X	+	80 irreg. maintained	Slight pulsation	O	Crackles both bases	Stomach & back. L. D_5, D_6 D_7.

MITRAL SYSTOLIC. FIRST SOUND SHORT AND SHARP AT APEX.

Diast.	Presys-tolic.	Pulm. 2nd Sound	Pulse.	Veins of Neck.	Liver	Lungs.	Referred Pain and Tenderness.
O	X	+	70 reg. maintained	O	O	O	On exertion under L. breast & back.
X	X	2+	60 irreg. maintained	O	O	O	On exertion under L. breast & back.
X	X	+	54 reg. maintained	O	O	O	On exertion only.
O	X	+	80 irreg. maintained	O	O	O	On exertion.
O	X	+	80 irreg. maintained	O	O	Crackles at bases	O
O	X	+	84 irreg. maintained	O	O	O	O
X	X	+	60 irreg. maintained	O	O	O	O

stroke is small, the tension, such as it is, is maintained between the beats [Broadbent's pressible, and whatever the initial stroke the tension is low between the beats.
2+ means that the 2nd sound is re-duplicated, but one element is accentuated.
short, sharp, and high-pitched, as is typically found in marked mitral stenosis.
measured with cone 0·8 in.]. Left ventricle somewhat hypertrophied. Case No. 51.—0·58 in.]. Left ventricle not hypertrophied.

present from enlargement of the liver, or affection of other organs than the heart. I have therefore not included such cases of cardiac failure in the short tabular arrangement (Table IV.) that I have drawn up to illustrate this statement.

(*c*) When a mitral systolic murmur is heard together with a sharp first sound at the apex or in the axilla, referred pain tends to appear as a symptom of the disease. Such a systolic murmur is usually not conducted into the axilla or heard at the angle of the left scapula, but this rule is frequently broken. It is, however, rarely if ever heard all over the back, and when heard at the angle of the left scapula is not infrequently accompanied by the first sound. This class of case may occasionally be characterised only by the above-mentioned signs, but usually a presystolic or diastolic murmur, and occasionally both murmurs are heard in addition to the systolic apical murmur. [Table V.]

By the statement that referred pain tends to appear in this class of case, I do not mean that it is continuously present. Thus, when the patient is at rest it is usually absent; but any undue exertion or any deterioration in the general condition will be signalised by the appearance of referred pain and headache. Moreover, a complaint of more or less pain is a feature in the history of such cases if they are watched over a considerable period of time.

(*d*.) A fourth group of cases of mitral disease consists of those in which no systolic mitral murmur is present. Either a presystolic, a diastolic, or both murmurs may be present, and the first sound has the typical short, sharp character usual in stenosis. The second sound is usually heard at the apex, and such cases thus belong to what Broadbent calls the first stage of mitral stenosis. [Table VI.]

In cases of this kind referred pain is not usually present. It may make its appearance as the sequel of some unusual act of exertion, or on first getting up after a considerable rest in bed; but nevertheless, referred pain is the exception rather than the rule during the time these signs are actually present.

I have so far spoken of referred pain in disease of the mitral valve without mentioning its position or characteristics. In such lesions as cause pain, it is situated within an area bounded above by a horizontal line at the level of the nipples and below by the line from the umbilicus to the costal border, and thence straight to the centre of the back. Not that the pain occupies the whole of this area at any time. It is mostly situated in the fifth space below the heart, at the epigastrium, and in the back just below or just internal to the angle of the scapula. It is situated mainly on the left side of the chest and back, sometimes entirely on the left side. The pain is not usually sharp and darting, but is, as a rule, dull and aching in character. It never becomes paroxysmal, or shows a tendency to spread suddenly and widely like the pain of aortic disease. It disables the patient less, but at the same time is more constant than the pain of aortic disease.

This pain is accompanied by superficial tenderness of greater or less extent over the sixth, seventh, and eighth dorsal segmental areas. The ninth dorsal also appears to be occasionally affected, but never without one or more of the above-mentioned areas. I therefore feel inclined to think that the implication of the ninth dorsal is due to spread downwards.

This tenderness is always worse on the left side, and the tenderness on the left side is the first to appear and the last to disappear.

The headache is situated in the temples and on the vertex, and more or less superficial tenderness is usually present over the temporal, vertical, parietal, segmental areas of the scalp.

Case 43, *to illustrate referred pain produced by lesions of the mitral valve.*—Peter H., aged 42. In 1882 he had rheumatic fever in which his feet and ankles were swollen and painful.

In 1892, one evening, after coming from work he spat a little blood. A month later he again spat a little blood after coming from work. He had suffered from pain in the chest for some time, and at last was compelled to go to a doctor (at the end of 1892), who said his heart was affected. He has been under the

doctor on and off ever since. He drank somewhat heavily till 1886, when he married; since then has been sober.

December 19, 1894.—Medium-sized man of 42; looks about his age. Face dusky, with a blue-red flush on both cheeks. No cyanosis of extremities, no œdema, no jaundice, no obvious dyspnœa. Says he becomes short of breath if he exerts himself much. Lies on his right side in bed. He complains of pain on

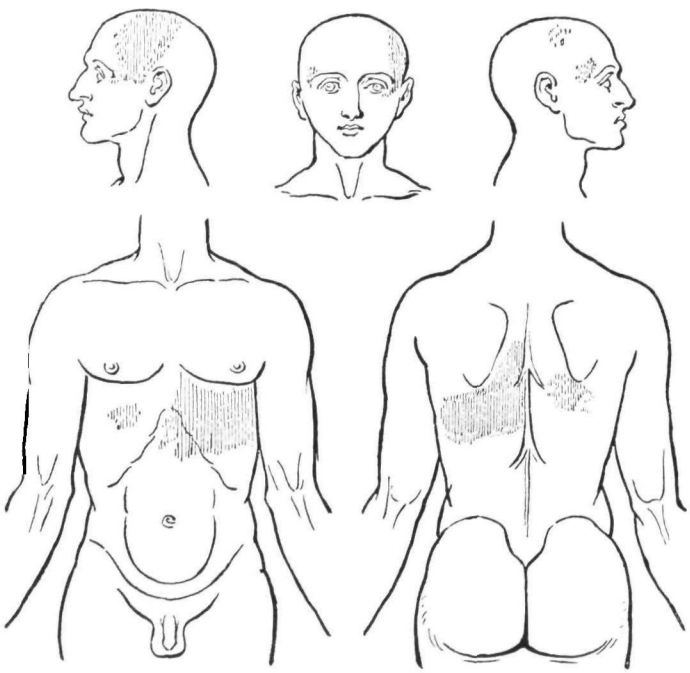

FIG. 5.

To show the extent of the superficial tenderness in a Case of Mitral Stenosis [Case No. 43], characterised by the following signs—a sharp first sound at the apex, followed by a systolic murmur not conducted to the angle of the left scapula.

The areas mainly affected on the trunk are the 6th and 7th dorsal on the left side. On the scalp the temporal and vertical areas are affected, mainly on the left side.

the left side of the chest in the fifth and sixth interspaces, and in the back close to the angle of the scapula. There is distinct superficial tenderness over both points.

Pulse 80, irregular both in rate and force. The irregularity in force is more marked than the irregularity in rhythm. Arterial wall not hard.

No pulsation of veins in the neck. Heart's apex beat seen and felt in fifth space 4½ inches from the middle line (11·5 cm.), just outside the left nipple line. Impulse weak and irregular; no thrill.

Cardiac dulness begins above at the third space, extends about 4½ inches (11·5 cm.) to the left and to the mid-sternum on the right.

Over the apex, the first sound is distinct, but sharp and thin; but further to the left in the axilla the first sound is distinctly sharp and high-pitched. The second sound is repudiated. A systolic murmur is audible over the fifth and sixth spaces from the left edge of the sternum to the line of the anterior axillary fold. Its maximum intensity lies over the apex beat, and no murmur is audible in the mid-axilla or at the angle of the left scapula. *Over the left second and third spaces* the second sound is accentuated; *over the right second and third spaces* the first sound is weak, the second sound somewhat slapping.

Respiration 24, *regular*. Alæ nasi and accessory muscles of inspiration are not working. No cough heard; no expectoration.

There are no abnormal signs in the lungs beyond those of slight general emphysema and a few moist crackling sounds at both bases.

Tongue clean, not furred. Appetite good, no vomiting; no pyrosis or eructation. Bowels opened daily. Urine 1020 acid. No albumen.

January 3.—He complained of pain in the epigastrium and below the angle of the left scapula. Marked band of superficial tenderness marking out the seventh dorsal area. Headache over the left temple with superficial tenderness over the left temporal area. Pulse 88, very irregular. Tongue clear, no vomiting. Physical signs as below.

January 4.—Exactly the same pains, headache and tenderness.

January 5.—The pain and headache are more marked, but are still practically confined to the left side.

The superficial tenderness both of the trunk and scarp are now very marked (fig. 5), and slight spots of tenderness have begun to appear on the right side. Tongue clean, moist. The pain is somewhat increased after taking food, but it is always there whether he takes food or not, and all food of every kind increases it. No vomiting; bowels still opened daily.

Pulse very irregular and of small stroke. No pulsation of veins in neck. Cardiac impulse felt diffusely in fifth space with its maximum above in his nipple line. Some epigastric pulsation. Suspicion of an occasional diastolic thirst.

Cardiac dulness beginning above in third space, extends about four inches to the left and about half-inch to the right of middle line of chest.

Over apex, first sound accentuated and sharp, second feeble. Systolic murmur not conducted into axilla or heard at angle of

left scapula. *Over left second and third spaces* second sound less markedly accentuated than before; *over right second and third spaces* first sound heard, second weak. Signs in lungs as before.

January 9.—The pain and tenderness became more accentuated, but occupied the same area as in fig. 5, except that the tenderness is more marked over the right side than in that figure.

January 15.—He continued in this condition till the evening of January 14, when the liver enlarged and signs of fluid appeared in the abdomen. To-day the pain is more marked on the right side than the left. He points to the right half of the abdomen from the costal margin to the iliac fossa and says he has pain in the right loins. The superficial tenderness extends from roughly the level of the seventh rib on the right side down to a point drawn from about the fourth lumbar spine forwards to a point about three inches below the umbilicus (seventh, eighth, ninth and tenth dorsal segmental areas). On his left side more or less tenderness is present over the same areas, but it is less well marked.

He now has tenderness of the scalp on both sides over the temporal, vertical, parietal areas, and over the right occipital area. This scalp tenderness is somewhat more marked on the right side than on the left.

Cardiac impulse very feeble. Cardiac dulness now extends $2\frac{1}{2}$ inches (6·5 cm.) to the right of the middle line of the chest and $4\frac{1}{2}$ inches to the left.

Over the apex, first sound weak, second reduplicated. No murmur of any kind can be heard. *Over pulmonary area*, second sound is scarcely heard. *Over aortic area*, sounds are extremely feeble.

Pulse about 76, very irregular, and with very small beat. Pulsation in the veins of the neck. Liver distinctly felt below the right costal margin. Signs of some fluid in the abdomen. Considerable quantity of æreated mucous expectoration, some of which is, however, browner than the rest. Cannot lie down in bed. Some crackling râles at both bases. No dulness.

Died January 17, 1895. The *post-mortem* revealed the following condition in the heart:—Right auricle somewhat dilated; right ventricle considerably dilated; left auricle much dilated, wall tough and fibrous; muscular substance diminished, excepting round the base of the appendix where it is hypertrophied; wall of left ventricle hypertrophied, but not greatly so; tricuspid valve dilated, smooth, no vegetations. From the auricular aspect the mitral valve appears as a long buttonhole slit with the edges in apposition throughout. It is very rigid. Fine bead-like vegetations on the free surface of the valve. The diameter of the valve measured with the cone is 0·8 inches (2 cm.). From the ventricular aspect the valve appears as a thickened, continuous ring of smaller diameter than from the auricular aspect. No marked change in aortic or pulmonary semi-lunar valves.

No micro-organisms discovered in the vegetations on the valve. Lungs congested. No infarcts, no consolidation. Liver, 58 ozs.; typical nutmeg.

Now the position of this referred pain and tenderness corresponds with that in affections of the stomach. We are thus brought face to face with the difficulty that the same areas may be affected in painful states due to lesions of the mitral valve and disturbances of the stomach. It might quite justly be urged that the presence of this pain and tenderness was due solely to a concomitant gastric disturbance; for troubles of digestion are not infrequent in these cases, and lend support to this objection.

I am unable to definitely prove my contention that this pain and tenderness is referred from the heart or disprove the objection that they are due to the stomach, but I think that a consideration of the following points tends to show that, in the majority of these cases, this pain and tenderness is of cardiac, rather than of gastric origin.

(1) True gastric disturbances are more common when the tricuspid valve fails and stagnation takes place in the system of the inferior vena cava. But it is exactly such a condition in which the left-sided pain and tenderness I have described is absent.

(2) In most cases where the first sound is abolished by a systolic murmur that is widely conducted referred pain is absent. But such cases not infrequently exhibit troubles of digestion.

Moreover, if the left ventricle fails, the first sound which was previously short and sharp may disappear, and a widespread systolic murmur be heard; at the same time, if a presystolic murmur was previously audible this murmur disappears. Under such conditions referred pain and tenderness may disappear to reappear again when the first sound returns under treatment. To illustrate this point I give the following case in which I twice watched this disappearance and reappearance of the first sound at the apex:—

Case 54.—*To illustrate the disappearance of the referred pain, with alteration of the physical signs, and its reappearance when the physical signs improved again.*—Rachel R. (V.P.H., Dr. Heron), aged 38, housewife.

First attack of St. Vitus's dance at the age of 13, second attack at 17. In 1875, at the age of 19, had an attack of acute rheumatism.

Since that time her heart used to beat quickly when she hurried, but she did her housework, and bore seven living children without complication.

1890.—She got a bad cough, and became very short of breath. Admitted to V.P.H. (Dr. Smith); went out well.

1892.—She became pregnant, and at the end of the fifth month came into V.P.H. again, with failure of the heart. Child born August, 1893, and three weeks afterwards again came into the hospital. From this time my observations on this case begin. She had evidently suffered for years from an ordinary type of mitral stenosis, and the least diminution in her general health would cause the left ventricle to fail.

September 4, 1894.—She had been admitted three weeks before with a systolic murmur at the apex, abolishing the first sound; the apical second sound was weak and reduplicated, and the pulmonary second sound was accentuated and single. There was no œdema of any part. She recovered in her usual manner.

August 30.—Menstruation began, and was accompanied by a good deal of pain in the lower abdomen and back. September 2. She was allowed up for half the day.

To-day there is a very marked change in her condition. She is taking no digitalis, so that this source of error is avoided, but menstruation still continues. Ears and lips are somewhat blue. No jaundice. No œdema anywhere. She has no pain, no headache, and no superficial tendency anywhere, but she complains that she feels faint and sick. She lies propped up in bed, but there are no marked objective signs of dyspnœa. Pulse small, and very irregular. Very slight pulsation of the veins in the neck. Apex beat in sixth space, five inches (12·5 cm) from mid-sternum. No thrill. Epigastric pulsation. Cardiac dulness begins above at the third rib, extends one and a half inches (4 cm.) to the right, and five and a half inches (14 cm.) to the left of the mid-sternum.

Over the apex, a loud systolic murmur is heard conducted into the axilla, but not heard at the angle of the scapula. Outside the nipple line and in the axilla the first sound is very feeble, but as we pass inwards there is a small area in the fourth and fifth spaces, extending for about one and a half inches (4 cm.) to the left of the left sternal edge, over which the first sound is short and sharp. The second sound is reduplicated. *Over the left second and third spaces* the second sound is accentuated, and not reduplicated. *Over the lower right sternal border* first sound is fairly well heard, and second sound is accentuated and single. Beyond a few crackling sounds at both bases, which were always audible in this case, there were no abnormal signs in the lungs. Tongue clean and moist. Nausea, but no vomiting. Complete loss of appetite. Bowels opened. Liver not enlarged. Urine

thirty ounces in twenty-four hours; no albumen. She was kept strictly to bed, and given tinct. strophanthus ♏v., with extr. convallaria gr. v., every six hours. Menstruation ceased on September 9.

September 18.—She has greatly improved. The feeling of faintness, sinking, and giddiness is passing away. She has begun to take food well. Pulse now 88, irregular in force and rate, but more so in force than rate. Tension now maintained. Apex beat four inches from middle line, and cardiac dulness extends one and a quarter inches to the right and four and a quarter inches (11·25 cm.) to the left of the middle line of the sternum.

Over lower part of left back and in left axilla a loud, sharp, accentuated first sound is audible, but no murmur. *Over the apex* the first sound is still audible, but is obscured by a short systolic murmur. At the line of the anterior axillary fold both the sharp first sound, so well heard in the axilla, and the systolic, heard over the apex, are audible together. Second sound over the apex reduplicated. *Over the left second and third spaces* second sound accentuated and single.

By September 15 the pulse was nearly regular, and a typical short, sharp, accentuated first sound was now heard over the whole area from the left sternal border to the angle of the left scapula behind. Over the apex beat it was accompanied by a short systolic murmur. No murmurs within the ventricular diastole heard anywhere.

September 18.—All the faint, sinking feeling, the feeling of sickness, and the shortness of breath have been absent for some time, and have not reappeared. To-day pain has begun to appear again, and she is complaining of pain over the lower ribs under both breasts and under the scapula, more marked on the left side than the right. There is definite superficial tenderness over portions of the seventh and eighth dorsal areas on both sides. Tongue clean. Appetite good. Bowels opened daily. Pulse 90, somewhat irregular in rate. Tension maintained. The cardiac signs are as on September 13, excepting that a præsystolic murmur of short duration now precedes the short, sharp, accentuated first sound. The systolic murmur is heard over the front of the chest only, and is not conducted into the axilla or to the angle of the left scapula. At the angle of the scapula the first sound is distinctly heard. From this point onwards a præsystolic or diastolic murmur was audible, in addition to the local systolic murmur.

September 22.—She went home, engaged in house work, &c., and on September 27 came back in the same condition as on September 4. No sign of a præsystolic murmur; the apical first sound is entirely abolished at apex, in axilla, and at angle of left scapula. All the sinking faintness had come back, and she had orthopnœa as before; but all pain and superficial tenderness were absent.

(3) The referred pain and tenderness I have described may make their appearance quite apart from food, when the patient first gets up after a long stay in bed, or after some special exertion. This is best seen in those cases where pain and tenderness are not a marked feature. Case No. 47 showed this point well.

(4) If the pain is present it may be aggravated by food, but it may entirely disappear by rest in bed, or by other treatment directed towards the heart, although the diet remains quite unchanged.

(5) I think that the pain over the shoulder joint and in the left side of the neck (accompanied by superficial tenderness over the fourth cervical segmental area) which in gastric disturbances is so commonly associated with pain and tenderness within the areas we are considering is, as a rule, absent in lesions of the mitral valve. Thus, although the pain under the heart at the epigastrium, and in the region of the shoulder-blade is shared by both disease of the stomach and certain lesions of the mitral valve, the pain and tenderness over the shoulder-joint is not common to these two diseases.

The value of these considerations can only be gauged by repeated observations of cases of mitral disease; but, so far as my experience goes at present, I cannot help believing that this referred pain is due, under the conditions I have described, to the cardiac condition, and not to the stomach.

§ 6. *Referred Pain due to Enlargement of the Liver produced by Failure of the Right Side of the Heart.*

As a rule, no referred pain can be definitely associated with the right side of the heart. For tricuspid regurgitation is apparently unaccompanied by referred pain and tricuspid stenosis is of such rarity that no one physician can obtain sufficient cases for observation.

But when the tricuspid valve yields, an exceedingly important focus of referred pain not uncommonly makes its appearance in consequence of the distension of the liver. All cases do not show this referred pain, but it most often appears when the liver enlarges for the first time, especially if this enlargement takes place with considerable rapidity.

If the liver remains enlarged the pain may entirely disappear in a few days, and if we have reason to think that the liver is hard and fibrotic, or, if enlargement has frequently occurred before, it may be completely painless.

When the liver suddenly enlarges for the first time, the patient complains of pain over the right hypochondrium, iliac fossa, and in his right loin.

If pressure is made over the enlarged liver the patient complains of pain, and the liver is therefore said to be tender. Now it is quite probable that the liver itself is really tender, but in the large number of cases the supposed tenderness of the liver is found to be in reality superficial. Thus the tenderness is well marked in the right loin and when tested in the usual way the superficial structures of the abdominal wall and back are found to be tender. If well developed, the eighth, ninth, and tenth dorsal segmental areas can be marked out. The important fact in such cases is the presence of the tenth dorsal area. For the presence of the eight and ninth dorsal areas on the right side may be due to the occurrence of doubling from the left side, and may be simply a sign that the pain and tenderness from some cardiac lesion on the left side has become very intense. But no cardiac lesion causes primary tenderness between the level of the umbilical line, and, therefore, the presence of the tenth dorsal area which lies below this line, points in cardiac cases directly to the liver.

The same area is also usually present on the left side of the abdomen and back, but the tenderness is less intense, more spotty in distribution, appears later, and disappears earlier than that on the right side.

With the appearance of this tenth dorsal area on the body tenderness usually appears over the occipital region of the scalp. But as the eighth and ninth dorsal areas are also present on the right side of the scalp we usually find that the headaches and tenderness of the scalp are distributed over the vertical parietal and occipital areas.

An exceeding good instance of the referred pain and superficial tenderness due to secondary enlargement of the liver is given in fig. 6.

The tenderness is, however, usually somewhat wider spread when the liver enlarges.

Case 55.—Sarah S. (Victoria Park, Dr. Eustace Smith), aged 25. Single.

July 1893.—Acute rheumatism. Remained in bed a month and when she got up found that her breath was very short.

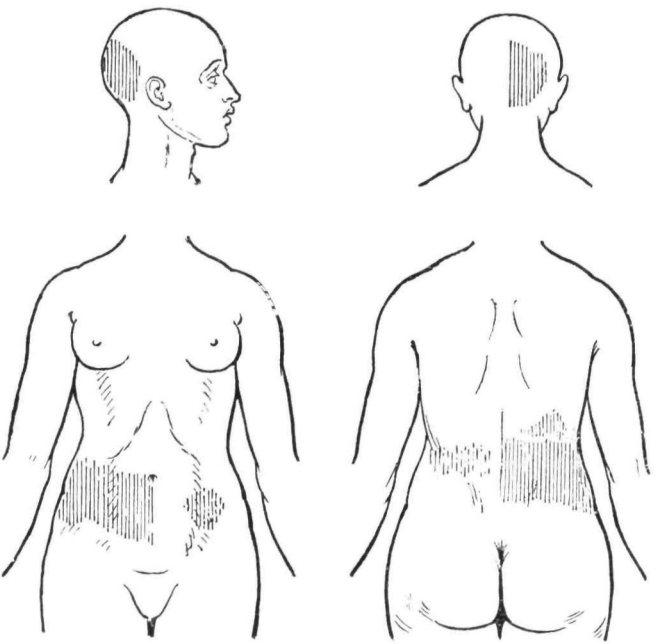

FIG. 6.

To illustrate the pain and tenderness over the 10th dorsal area, produced by Acute Enlargement of the Liver, in consequence of Cardiac Failure [Case No. 55].

Note the tenderness over the occipital region of the scalp.

September, 1893.—Admitted to V.P. Hospital with mitral stenosis and regurgitation.

Apex beat diffuse and forcible. Diastolic apical thrill. Upper limit, cardiac dulness third rib. Right limit, right edge of sternum. Left limit, nipple line. At apex first sound is loud and followed by a systolic murmur conducted as far as the left axilla. Second sound heard at the apex and followed by a short diastolic murmur. The pulmonary second sound was reduplicated. Aortic second sound normal.

Pulse small, irregular, easily compressible.

Edge of liver just felt.

She much improved, and was discharged on December 13, 1893.

As soon as she was discharged she tried to walk about the house, within a few days began to vomit. Much pain in the abdomen and headache.

Readmitted December 29, 1893. Condition as follows :—

Skin of face of a slightly yellowish tinge with a red spot on each cheek. Conjunctivæ not yellow. Ears and lips a little blue. No swelling of ankles. Distinct clubbing of the fingers. Knee-jerks normal. Plantar reflexes brisk.

She cannot lie flat in bed. Very short of breath on the least exertion. No fainting, but frequently feels faint.

She complains of pain in the abdomen below the navel and in the loins ; she also has pain "across the shoulders and over the thorax below the level of the nipples." Much superficial tenderness.

Headache over the occipital region and over both temples associated with superficial tenderness of the scalp.

Pulse about 84, irregular both in force and rhythm, small stroke, compressible.

Distinct pulsation, but no marked distension of the veins of the neck.

Apex beat in fifth space in the nipple line. Pulsation also seen in third and fourth spaces internal to nipple line. Upper limit of cardiac dulness third rib. Right limit, cardiac dulness right edge of sternum. Left limit, cardiac dulness outside the nipple line. Heart's action very irregular. With the short beats a systolic murmur only is heard, but when the diastola is prolonged a soft diastolic murmur is heard. The first sound is feeble at the apex, but can be heard independently of the systolic murmur. The systolic murmur is conducted to the angle of the left scapula. No murmur heard over aortic area. First sound feeble over the right edge of the sternum.

Liver a good 2 inches (5 cm.) below the border of the ribs.

Suspicion of fluid in the abdomen.

Respiration 22. Slight cough ; worse at night.

Some crackling sounds heard at the bases of both lungs and at apex of right lung.

Tongue red and a little furred. Bowels opened daily. She frequently vomited undigested food.

She has not menstruated for five months. Usually lasts two days. No pain.

January 9.—The feet are swollen to-day and there are distinct signs of fluid in the abdomen.

The liver edge can now be felt just above the level of the umbilicus.

She complains of pain in the abdomen below the umbilicus and occipital headache.

Marked superficial tenderness of the abdominal wall over the area in fig. 6, with definite tenderness of the occipital region of the scalp.

The liver continued to enlarge and the pulse became very irregular and small. She died on January 20, 1894.

On January 6 the temperature began to rise and oscillated irregularly between 100° and 98° F. (37·8° and 36·7° C.)

The urine was acid, sp. gr. 1025, and contained an amount of albumen which varied from about $\frac{1}{12}$ to $\frac{1}{10}$.

Post-mortem. — No pericarditis. Right side of the heart dilated. Tricuspid orifice admitted four fingers. Pulmonary valves otherwise normal. Left ventricle hypertrophied. Some thickening of the edges of the mitral valve which was somewhat stenosed. The valve was incompetent. Around its base on the auricular surface was a small layer of bead-like vegetations. The aortic valves showed fresh vegetations, which contained streptococci in considerable numbers.

No peritonitis. Fluid in the abdomen.

Liver weighed three pounds six ounces. Congested and fibrotic.

Kidneys congested, but otherwise normal.

Spleen small and congested with small fresh infarct in upper part.

Left lung healthy except for congestion at the base. Right lung congested. In middle lobe was a small infarct with some collapse around it.

Pelvic organs normal. Ovaries showed no signs of recent menstruation.

§ 7. *Paroxysmal Pain of Cardiac Origin.*

Writers on cardiac diseases express the most diverse opinions in their attempts to define the disease "angina pectoris," but are practically unanimous with regard to the symptoms of an "anginal" attack. Given the symptoms in any one case, the one group of observers refuse to diagnose angina pectoris if the physical signs of cardiac disease are present; whilst, at the opposite pole, are those who speak of angina pectoris whenever the symptoms are present, regardless of the age of the patient or the physical signs of disease in the heart.

But if differences of opinion are found between the various authorities during the life of the patient, still greater differences manifest themselves at the autopsy. Some cling to one anatomical lesion, some to another, and every case

that does not show a particular lesion is spoken of as false, or pseudo-angina pectoris.

From the point of view of the nomenclature of disease it is of obvious utility to confine the name angina pectoris to cases of a certain clinical type (*e.g.*, the classical cases in elderly men), or, to those which show some simple anatomical lesion (*e.g.*, occlusion of the coronary arteries.)

But throughout this study I am not engaged in a description of diseases, but in a physiological investigation of pain by the help of cases of disease. In this attempt to unravel the origin of pain it is necessary to look to physiological causes, and not to those descriptive characteristics which would enable a naturalist to separate one species from another. For the same anatomical lesion may produce different physiological effects, and the same physiological effects depend upon many different anatomical lesions. Thus, for my purpose, it would be a grave mistake to confine this section to a study of only one clinical class of paroxysmal cardiac pain. And yet I should be the last to attempt to bring all the cases here considered under the heading of a single disease.

In those cases, therefore, in which I am obliged to call the case by the name of some disease, I shall speak of primary angina pectoris where the symptoms are present without gross physical signs within the heart, and secondary angina pectoris where physical signs exist pointing to lesions of the aorta, or its valves.

Now, the symptoms of an anginal attack may be grouped under three main headings.

(1) A feeling of suffocation, accompanied by a pain at the epigastrium. This pain is variously described as "pressing pain," "bursting pain," "like being tied in with a cord," "as if the heart was too big."

(2) A sense of impending death. The face is said to become cold, the jaws stiff, and then this cold, "deathly" feeling spreads over the body.

(3) Violent radiating pains mostly over the upper part of the chest, and in one or both arms. These are accompanied by marked and wide-spread superficial tenderness, and are thus truly referred.

In any individual case these three symptoms may be present to a very varying degree. Thus my first illustration (Case 56) is chosen because of the complete development of these three groups of symptoms in the attacks. Yet for the purposes of classification of disease this man was probably a case of angina pectoris secondary to aneurism of the aorta.

Case 56.—*To illustrate the complete phenomena of an anginal attack in a man with aneurism of the aorta.* William D. (V.P.H. Dr. Eustace Smith), aged 38, railway porter.

At the end of 1892 he was out of sorts, and found he could not lift such heavy weights as before. One morning in January, 1893, he had a feeling of suffocation and giddiness, and his heart began to beat very fast. He felt faint and had to sit down. The doctor said he had "valvular disease." Throughout the next six months he had frequent attacks, in which he had a sudden feeling of suffocation with a choking sensation in the throat, and accompanied by a pain in the epigastrium as if a cord was pressing it in. In June, 1894, his attacks altered in character, and became like those seen in the hospital. These attacks came on particularly during his work.

He drank heavily till three years ago, and has been occupied with exceedingly heavy lifting work as porter at a goods' station. Denies syphilis.

On admission (October 1, 1894). Largely built man of 38. Face of a uniform pinkish-yellow colour. No anæmia. No cyanosis. No jaundice. No œdema. Exceedingly well nourished. Looks as if he had gained weight.

Pulse 90, regular, somewhat high tension. Arterial wall a little hard for a man of 38. Pulses equal.

No capillary pulsation.

No venous pulsation.

Heart's apex beat very feeble, and cannot be definitely localised. No pulsations over the upper part of the chest. No thrill.

Cardiac dulness begins at third rib, extends $\frac{3}{4}$ in. (2 cm.) to the right of the middle line of the sternum and $5\frac{1}{2}$ in. (14 cm.) to the left of the mid-sternal line.

Over the upper part of the sternum, and in the second right interspace, a sharp and ringing second sound is heard. The first sound is inaudible; and a distinct systolic murmur is heard over the upper part of the sternum to the aortic area. It is not conducted over the apex; the first sound is very short and wanting in tone, and closely resembles the second sound, so that the sounds at the apex have a "tick tack" character closely resembling that of a

fœtal heart. No murmur heard at the apex, or at the angle of the left scapula.

No cough or expectoration. He is short of breath on exertion, and cannot sleep flat upon the back.

No abnormal physical signs in the lungs.

Tongue moist and a little grey. Appetite poor. No vomiting. Bowels confined. Does not usually suffer from flatus.

Liver and spleen not free.

The pupils are equal to react to light and accommodation. The left does not dilate quite so well to shade as the right.

Urine normal.

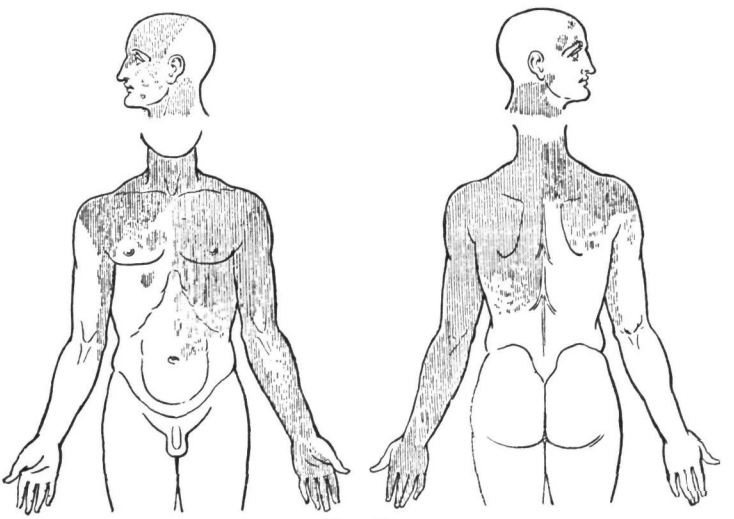

FIG. 7.

To show the widespread tenderness present after a major anginal attack in Case 56.

Nature of the Attacks and of the Pain.—On October 7 he was completely free from pain. During the night, at 1 a.m., on October 8, a patient in the ward became violent. William D. was sitting up to watch the struggle when he was noticed to lie back with his head thrown back over the pile of pillows, and his neck extended. His face was sweating, and he said he had a cold, dead feeling over the forehead, nose, and lips, which spread all over his face. The jaws felt stiff. He felt exactly as if he were dying. As he lay back he had a feeling over the region of the apex beat, and in the epigastrium, as if something were getting tighter and tighter. Then a shooting, tearing pain came on over the second and third interspaces, rapidly followed by a piercing pain over the anterior and posterior aspects of the left shoulder joint. Then the pain ran

over the left sterno-mastoid to the occipital region, and down the left arm to the top of the little finger. At the same time he suffered from marked headache over the left half of the forehead. During the later part of the attack, his heart was beating rapidly, but was not irregular. The attack lasted two minutes. It was followed by considerable mental depression, and he said he felt utterly exhausted. He complained of widespread soreness, and said he could scarcely bear his shirt, owing to the tenderness of his chest and back. (Fig. 7.) About a quarter of an hour after the attack he passed a considerable quantity of pale urine.

This represents a complete, fully-developed attack, characterised by the following symptoms:—(1) feeling of coldness; (2)

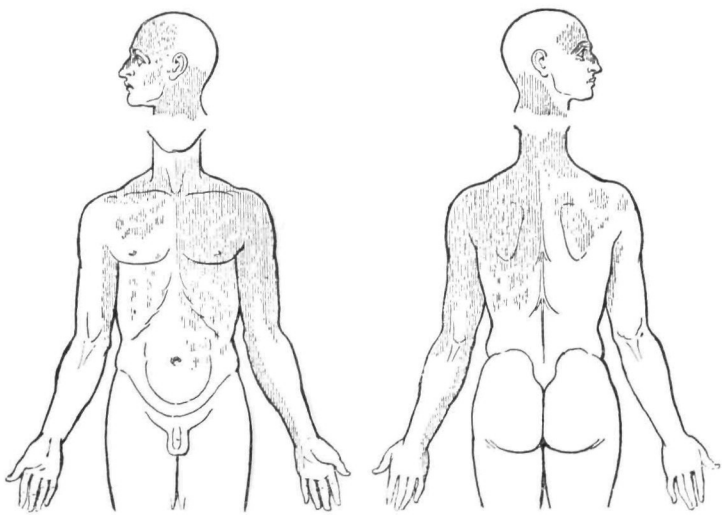

FIG. 8.

To show the tenderness present after an attack of paroxysmal pain [minor attack of the first type].

feeling of death and suffocation; (3) local bursting feeling at the heart; (4) violent shooting referred pains in chest and head, followed by intense widespread superficial tenderness. But during a long period of observation it became plain that he also suffered from minor attacks of two separate types.

First type (October 21, 1894). He had not slept well on account of pain in the upper part of left side of chest and back. In the morning his chest ached and felt sore. At 10 a.m. the pain began to shoot, and to spread as in a major attack, but it was of much less intensity. This shooting gradually ceased, and left behind it a sore aching feeling wider spread and more intense than before. Superficial tenderness was present on the left side of the

trunk, from the level of the jaw to the horizontal level of the tenth dorsal spine. (Fig. 8.) It extended down the ulnar border of the arm to the little finger. Some tenderness, but to a much less degree, was present on the right side of the chest. The whole fronto-temporal, mid-orbital, and frontal and nasal areas of the scalp and face were tender. There was no feeling of death or suffocation, and no sensation in the throat. This type of attack occurred when he was recovering under rest in bed and treatment with nitro-glycerine.

Second type (March 12, 1896). He woke feeling quite well. Whilst washing himself he raised both hands to the top of his head, and immediately sank back on to his pillows with a feeling of tightness across the chest. He felt suffocated, as if he would never get his breath again, with a cramped feeling in the throat as if something was being tightened. He felt a rolling, fluttering, irregular movement at the heart. It passed off, but returned again in a few minutes, and for an hour and a half he continued to have repeated small attacks of this nature. Before they began he was completely free from pain, but throughout the hour and a half during which they lasted a feeling of soreness over the upper part of the left chest and in the left arm gradually developed. There was no shooting or stabbing pain, as in the major attack, but a slow increase, with each little attack, of the sore aching pain. Finally he showed exceedingly marked superficial tenderness of the left side of the chest and back, from the level of the jaw to the horizontal level of about the tenth spine, extending down the left arm. There was very slight tenderness on the right side of the chest. Marked tenderness of the anterior part of the scalp. This type of attack seemed to occur when he returned to work after the major attacks had been abolished for a period of many months by treatment.

March, 1896.—The physical signs are still exactly as on admission in October, 1894, except that the systolic murmur is somewhat less marked.

Now this man not only had many major attacks, demonstrating all the symptoms of angina pectoris, but he also had minor attacks of two distinct types of much importance in attempting to analyse the phenomena of a major attack.

The one type was always preceded by some referred pain in the arm and shoulder. The attack consisted in the rapid spread of this pain to other parts. The heart did not become irregular. The sense of impending death was absent.

The second type came on after a definite exertion, and consisted of small exacerbations in a condition which was

present for an hour or more. He felt a tightness across the chest, a cramped feeling in the throat, and a rolling, irregular feeling at the heart. The heart seems to beat irregularly. These sensations lasted almost two minutes, leaving him with an uncomfortable feeling as if he would drop, and then recurred again and again at short intervals. On one occasion the whole state lasted one hour and a half. When these attacks begin there is no pain, but referred pain slowly appears, and becomes more marked after each little attack of suffocation. Finally, at the end of a considerable period, the pain and superficial tenderness become more marked. Thus the main feature of this type of attack is the suffocative feeling, and referred pain is not developed until these attacks are repeated many times.

Now just as this case showed minor attacks of two separate types, so we find that cases of primary and secondary angina pectoris range themselves at a point somewhere between two extreme types. On the one extreme lies the type in which the feeling of suffocation or strangulation and the sense of impending dissolution predominate. Referred pain may actually be absent, or if it is present, it simply follows the cardiac disturbance exactly as we should expect. On the other extreme lies the type that usually suffers from more or less pain and superficial tenderness apart from the attack. Here the spread of the referred pain and its increase in intensity are the main features of the attack. The heart's action is at first undisturbed, but as the pain continues to spread the heart begins to beat, not only with great rapidity but with increased force. The sense of suffocation and impending death may be absent.

Case 57 more nearly approached the first extreme type. He was an old man with profoundly degenerate arteries. The sounds of the heart were exceedingly feeble, and a musical systolic murmur was audible, apparently originating at the aortic valves. The attacks occurred in groups, and consisted firstly of palpitation and a sense that he was dying, and then secondarily of pain. The superficial tenderness before the first recognised attack was slight, but after the attack was over it had spread widely.

Case 57.—*To illustrate the type of anginal attacks, in which the suffocation and sense of impending death are the main features, and referred pain and tenderness a secondary phenomenon.*—John K. (V.P.H., Dr. Eustace Smith), aged 64; a canvasser.

In 1888 he began to suffer from palpitation of the heart. In 1889 he was in the hospital; but at that time suffered from no paroxysmal attacks. In 1890 he began to have slight attacks in which he felt faint. They began with gasping; he then felt as if he was going to die. Pain was not a marked feature of these attacks. In November, 1893, pain became a marked feature of the attacks, which then assumed the form described below.

He drank heavily as a young man, but has been a teetotaler for ten years. Has had many accidents, but no diseases, excepting one attack of gout in the left toe.

On admission. Medium-sized man of 64; looks considerably older. Marked arcus senilis. No cyanosis. No jaundice. No œdema. No wasting.

He has never fainted. On exertion he suffers from palpitation, which he says is quite different from the palpitation of the attacks. Not very short of breath, excepting during the attacks. Sleeps with two pillows, but does not need to be otherwise propped up.

Pulse 72, regular; of increased tension. The arterial wall is excessively tortuous and rough to the feel. Every artery of any size is tortuous and visible. The pulses are equal.

No capillary pulsation.

No venous pulsation.

Heart's apex beat seen and felt in fifth space ½ inch (1·5 cm.) outside nipple line. Not heaving. No thrill. No other pulsation visible in chest.

Cardiac dulness much diminished by emphysema. All over the front of the chest, within the areas enclosed by the right and the left nipple line is heard a high pitched, exceedingly musical systolic murmur. It is not conducted to the angle of the left scapula. Over the aortic area both first and second sounds are inaudible, whilst over the apex beat the first sound is not heard, but the second is occasionally very feebly audible. The weakness of the sounds of the heart was a very noticeable feature of this case.

Respiration 20: with the expiratory period prolonged. Marked elevation of the chest. Alæ nasi quiet. Single, non-paroxysmal cough; not very troublesome now. Very small quantity of grey mucous expectoration.

Physical signs in chest those of emphysema, but none of bronchitis.

Tongue clean, moist. No vomiting.

Liver and spleen not enlarged.

Urine 1010, acid. No albumen or sugar.

Nature of the pain and of the paroxysmal attacks.—The following account of an attack seen on February 24 is typical

of all his present attacks. In the morning he complained that he had slept badly, and felt a good deal of soreness in his chest. (Fig. 9). There were several spots of superficial tenderness confined to the left side of chest and inner aspect of left elbow. He had no headache, but there was some superficial tenderness above and around the left eye. At 10.30 p.m. he suddenly sat straight up in bed and said he was going to have an attack. His pulse was beating at the rate of 150 in the minute. He was sweating over the face, but there was no marked alteration of colour. Thirty seconds after the beginning of the attack he began to wheeze violently. In sixty seconds the pulse dropped in rate, and became very irregular. He complained that his heart felt as if

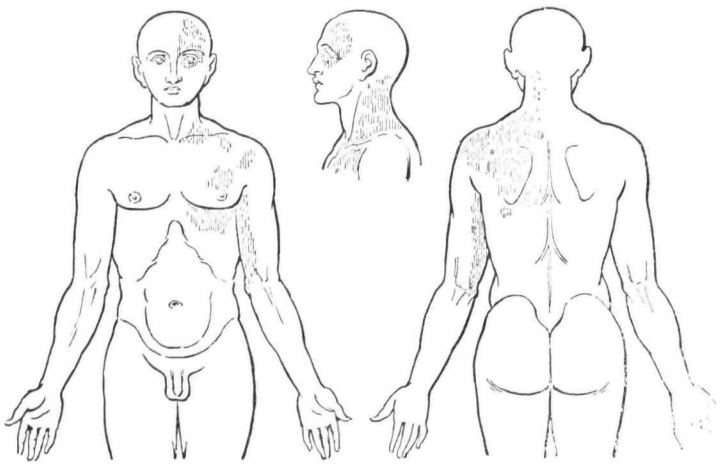

FIG. 9.

To show the tenderness which was present before the attack of angina pectoris in Case No. 57.

The dotted portions are those over which the tenderness was ill-defined and of no marked intensity.

it were swelled. The attack was over in two and a half minutes. He said that, when he called me, the palpitation had begun at the heart. Then the pain began to gather force. He had had pain in the chest and left arm before, and these pains rapidly increased, and were joined by a pain in the region of the hyoid. He complained that the skin of his forehead felt bulged, and that his nose felt swollen. Profound and widespread superficial tenderness of scalp, chest, and arms after the attack.

At 11 p.m., whilst I had my hand upon his pulse, he had a second attack. In the first ten seconds the heart beat 27 (at the rate of 162 in the minute). At the beginning of the second half minute it beat 24 in ten seconds (144 in the minute). At the

beginning of the third half minute it beat 14 in ten seconds (84 in the minute), but was very irregular both in rhythm and force. The pulse at the wrist was now very feeble and irregular. At the beginning of the fourth half minute it still beat 14 in ten seconds. At the end of the first half minute he began to wheeze violently and to clutch at his throat, complaining that he suffered pain there. The pain in the chest became much worse, and he felt as if he were dying. His body and head were sweating, and his hair became quite moist. He then gave a great yawn, and the attack was over.

He always insisted that the increase in the shoulder pain did not begin till after his attack was well on him; and that the attacks began with palpitation and were followed by pain.

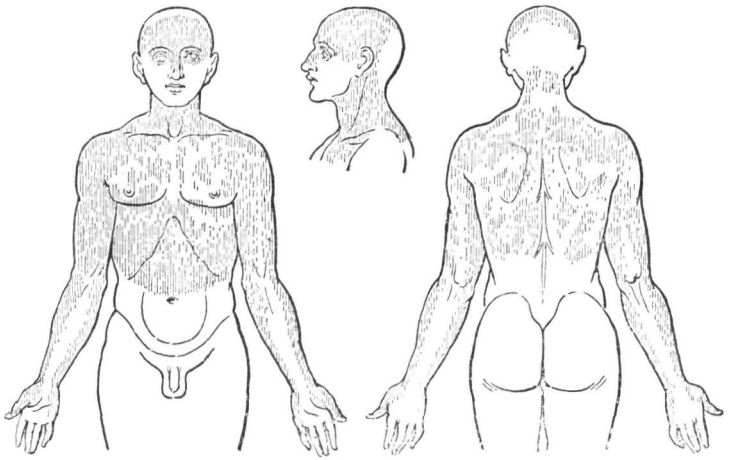

FIG. 10.

To show the extent of the superficial tenderness after the attack of angina pectoris in Case 57.

After the attack he had much widespread feeling of soreness of trunk and arms, and a feeling as if his head had swelled and tightened his scalp. Much widespread superficial tenderness of body and scalp. (Fig. 10.) The left arm felt numb, as if he had been lying on it. He could make no fine movements of his fingers, but there was no absolute paralysis. The loss of power seemed to him to be below the elbow. He seemed to be utterly exhausted, and was very depressed after the attack was over. Shortly afterwards he passed a considerable quantity of pale urine.

During the attack the systolic murmur was still heard, but neither first or second sound were audible anywhere. There were no moist sounds in the lung during the stage of wheezing.

These attacks enormously improved under treatment, and particularly with strict rest in bed. During the time he was in bed he had a few slight attacks, and it was very evident that the referred pain and superficial tenderness followed the attacks and were directly produced by them. For he would have an attack when all referred pain and superficial tenderness were absent, but after the attack marked referred pain and superficial tenderness would make their appearance mainly over the left side.

On the other hand, Case 58 more nearly approached the second extreme type. This case suffered from aortic regurgitation and stenosis as the sequel of acute rheumatism, and similar phenomena are found in some cases where the aortic regurgitation is of non-rheumatic origin. Here the attacks consisted mainly of a violent radiation of the referred pain present before the attack. An appreciable time elapsed between the beginning of the radiation of pain and the increase in the rapidity and force of the heart's beat.

It is interesting to notice that when the left ventricle failed after a series of attacks and a mitral regurgitant murmur became audible, abolishing the apical first sound, all attacks ceased. When the heart recovered the attacks returned. It is also interesting to note that this case died, not in an attack, but from cardiac failure.

Case 58.—*To illustrate an anginal attack in which the spread of referred pain was the primary phenomena.* Mary S. (V. P. H., Dr. Sainsbury), aged 18. In December, 1888, she had her first attack of rheumatic fever, which left her with an affection of the aortic valves. She remained fairly well, excepting that she suffered from pain in the chest and shortness of breath, until June, 1889, when she had a second attack of acute rheumatism. When she recovered from this second attack she had pain in the chest more or less continuously. Shortly afterwards, at 9 p.m. one night, she had her first attack.

From July, 1893, to March, 1894, she was under my observation day by day, and the following account is a *resumé* of the notes taken during this period by myself.

She is a finely-built rather stout girl of 18; looks a good deal older. Face pale, with occasional irregular flushes on cheeks and chin. No œdema; no jaundice; no cyanosis; no fainting; short of breath on exertion, and she occasionally suffered from periods during which she could only breathe when propped up. Palpitation on exertion and occasionally when lying in bed.

This palpitation always leaves her with a pain in the left breast and left temple, and if pain was present before, it is markedly increased by the palpitation.

Pulse, as a rule, about 80, regular, but easily runs up on exertion; typically low tension, with a strong stroke and marked regurgitant characteristics. Arterial pulsation visible in neck as high as the ears.

Extreme capillary pulsation; no venous pulsation. Cardiac pulsation visible over whole left half of chest. What appears to be the apex beat can be felt in the fifth space in the line of the anterior axillary fold. Cardiac dulness begins at second space above, extends to the right edge of the sternum and to a point to the left on the fifth rib in the line of the anterior axillary fold.

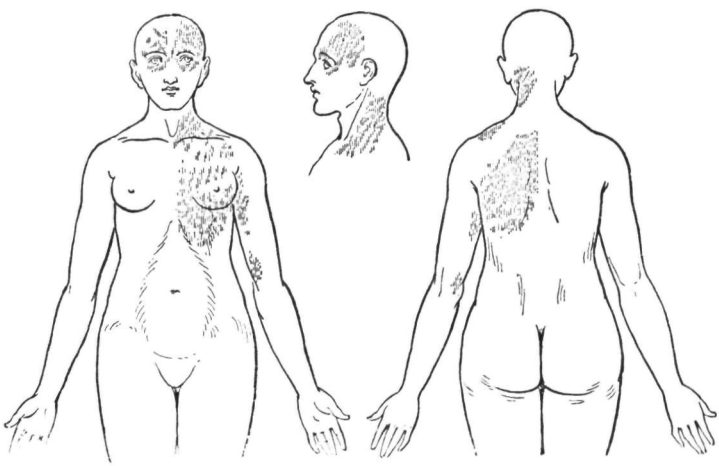

FIG. 11.

To show the extent of the superficial tenderness in Case No. 58, apart from an attack of angina pectoris.

Over the aortic area neither first nor second sound are heard but all over the sternum is a loud double aortic murmur (systolic and diastolic). Over the apex beat both first and second sounds are audible. The first sound is also heard at the angle of the left scapula. There is no mitral murmur either at the apex or angle of left scapula.

No abnormal physical signs in the lungs. Tongue clean; appetite bad. No pain after food; no vomiting; bowels somewhat confined. Liver and spleen not enlarged. Urine 1030 acid, no albumen, no sugar.

Nature and distribution of the pain and tenderness.—She usually complained of pain over the left breast and in the left interscapula region of the back. At times the pain was situated

over the inner side of the left arm. More or less superficial tenderness was usually present, usually on the left side, but sometimes, if the pain was severe, on both sides of the chest. The most common distribution of this tenderness was between the second and fifth rib in front, and from the level of the third dorsal to the seventh dorsal spine behind, and the tenderness involved either the upper third or the whole of the inner aspect of the arm to the elbow. (Fig. 11.) It was always most marked over the left half of the chest and back, and remained longer on the left side than the right.

The headache extended from the fronto-temporal region forwards over the eyes to a greater or less extent. This headache was accompanied by superficial tenderness of the forehead, and

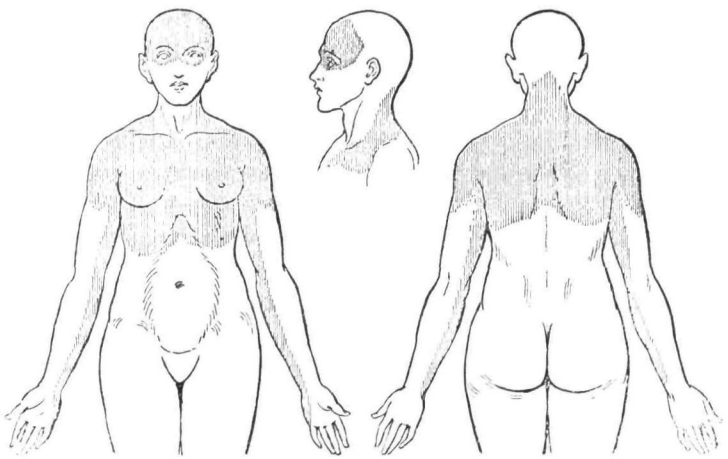

FIG. 12.

To show the extent of the superficial tenderness in case No. 58 during the time the pain was spreading, and 3 minutes before the quickening of the heart, in the attack of angina pectoris described in the text.

anterior portion of the temporal regions; it was usually bi-lateral, but occasionally unilateral confined to the left side. (Fig. 11.)

The attacks, which occurred at one time as often as once in twenty-four hours, were as follows: she suffered from pain for hours or even days before an attack. As a paroxysm came on, she would call out that she was going to have an attack. She said she felt the pain spreading. (Fig. 12.) She said it was going down the left arm and into the shoulder. The forehead was moist, the lips of a grey white, the area around the mouth pale, but the face still retained its pink colour. The pulse was 80. Then she called out that the pain was all over her chest. She threw herself backwards, arched her back, and clung to the rail at the head of the

bed. The forehead and cheeks flushed, and intense capillary pulsation was visible all over the face; the arteries of her neck pulsated intensely. The pulse ran up to 160, and became of extremely low tension. The heart beat seemed to shake the whole front of the chest. The arching of the back then ceased, the arms fell flat on the bed and she began to cough. The pulse rate rapidly fell. The attack occasionally ended in crying, and after it was over she generally passed a considerable quantity of pale urine of low specific gravity. It was followed by the most intense and wide-spread superficial tenderness of the thorax, back, arms, and scalp. The extent of the tenderness over the scalp and trunk closely resembled that shown on Fig. 10. The whole attack lasted about three minutes, but occasionally the first attack had scarcely passed off before a second came on.

As the pain spread it was noticed that the superficial tenderness became of much wider distribution. Thus I was, on more than one occasion, able to watch it spread down the inner side of the arm to the elbow, and then to the ulnar border of the hand. I also watched it spring into the neck and to the opposite side of the body. All this occurred before the pulse altered in rate. (Fig. 12.)

This I took to be an indication for treatment, and on several occasions, after the pain and tenderness had begun to spread, an injection of morphia or gr. xxx. of phenacetin prevented the great rise in pulse rate. The attack then completely altered in character. For the pain began to spread as usual, reached a certain point, accompanied by wide-spread superficial tenderness, and then slowly died away without the occurrence of the flushing, rapid pulse beat, or arching of the back.

During the attack she felt very faint, but the faintness was not sufficient to alarm her. She had no fear of impending death, although on more than one occasion the pulse became imperceptible at the wrist, and she seemed on the point of death.

The permanent pain, and the frequency of the attacks, were much lessened by treatment, and she left the hospital. I saw her three months later in a condition, shortly, as follows: the double aortic murmur was present as before, but the first sound at the apex was replaced by a systolic murmur, which was audible at the left angle of the scapula; the liver was distinctly enlarged; the mitral valve had evidently failed. She had had no attacks for three weeks, and she remained free from attacks for a further two weeks until the mitral murmur disappeared. With the marked improvement in the general condition of the left ventricle the anginal attacks returned.

She died in December, 1894, of cardiac failure.

To understand how severe attacks, like those of Case No. 58, may be developed in cases of aortic regurgitation, I give Case No. 59. Here the pain was, as a rule, not

paroxysmal, but closely resembled that usually seen in aortic disease, when unaccompanied by mitral regurgitation. However, under the influence of slight exertion, distinct paroxysms of slight intensity, but of the same nature as those of Case No. 58, appeared at infrequent intervals. Such cases are not uncommon; and are very instructive for the study of the origin of paroxysmal attacks arising in patients with organic disease of the heart.

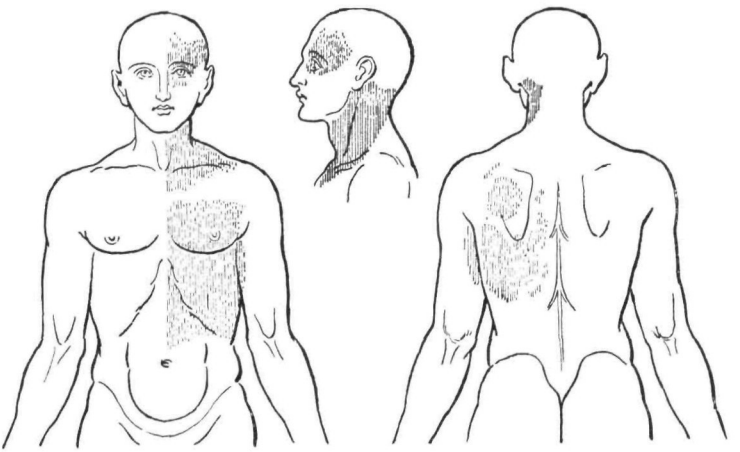

FIG. 13.

To show the extent of the superficial tenderness in Case No. 59, after the attack of "faintness" described in the text. Before the attack the tenderness was much less extensive.

Case 59.—*To illustrate the origin of paroxysmal attacks of pain in cases of organic disease of the heart.* Samuel F. (V.P.H. Dr. Eustace Smith), aged 40; labourer.

About Christmas, 1891, he was running to catch a train when he felt a pain "at the heart." This pain was exceedingly sharp, and forced him to stop. It never quite left him; and in April, 1893, it began to affect his left arm, but he continued with his work in spite of it until November, 1893. He was then seized with great shortness of breath, and suffered from much pain. A bad cough came on, and he spat up blood-stained phlegm. He was admitted to the hospital on December 11, 1893.

No history of acute rheumatism could be obtained, and he denies all venereal disease. His work is exceedingly heavy; and consists in unloading chests of oranges from the hold of a vessel,

and carrying them on his back up a flight of stairs into the warehouse.

On admission.—He is a well-built man of 40, and looks quite his age. No anæmia. No jaundice. No œdema. No clubbing of the fingers.

He fainted several times before admission, and within the first twenty-four hours after admission, felt faint more than once. He says his heart beats irregularly when he exerts himself. He is very short of breath when he walks about, but not when he lies in bed. He lies on the right side in bed on account of the pain in the left side.

He complained of pain over the inner side of the left arm as far as the elbows. Occasionally the pain runs down to the fingers of his left hand, and he has a stinging, numbing feeling in the middle, ring, and little fingers of the left hand. He also complained of pain at the epigastrium, and over points above and internal to the nipple, and under the outer part of the clavicle. All the pain was on the left side.

He also complained of headache in the left temple, extending over the left half of the forehead.

There was marked superficial tenderness over the chest, neck, temple, and forehead on the left side only.

Apex beat is feeble, but is felt in fifth space as far out as 2 in. (5 cm.) outside the left nipple line. No pulsation felt elsewhere in the chest.

The cardiac dulness begins above, at the third space, extends to the right sternal border, and to rather over 1 in. (2·5 cm.) outside the left nipple line.

Over the whole of the sternum, and over the apex, a loud aortic diastolic murmur is heard. No systolic murmur is audible. At the aortic cartilage the first and second sounds are not heard, whilst both sounds are distinct at the apex.

Pulse 100, regular, somewhat low tension. Pulses are equal on the two sides.

No cough or expectoration now.

No abnormal signs beyond a few *râles* at the right base.

Tongue a little furred. No vomiting now. No pain after food.

Liver and spleen not enlarged.

December 31.—He was free from pain until he got up to tea. At teatime he complained of a pain which started under the left breast and in the epigastrium. This pain then spread to the upper spaces, and then travelled slowly until it involved the left half of the neck and the inner side of the left arm. He then had a feeling as if he was going to drop. When I reached him he had crawled into bed. He was crying, and said he felt faint. The aching pain was still severe, but had ceased to spread. He was sweating profusely.

He showed exceedingly marked tenderness of the superficial structures of the chest, back, and neck, as in fig. 13. Headache

and tenderness were also present over the left temple and forehead.

There was no marked alteration in the physical signs, but the pulse was somewhat more rapid. The temperature had risen to 100° F. (37·8° C.)

He remained in bed for three weeks, and was treated with a pill containing extr. belladonna gr. ¼, pulv. opii gr. i. Although he had some occasional pain he had no further attacks.

Thus we must conclude that paroxysmal cardiac pain can be developed in one of two ways :—

(1) The pain is the expression of the cardiac disturbance and consists of a local epigastric pain, followed by more or less widespread referred pain. The attack is accompanied by a feeling of impending death and suffocation.

Attacks, more or less approaching this type, are best seen in men well on in life with calcareous arteries and feeble heart sounds.

(2) The pain is the main feature. The attack consists in the rapid radiation and increase in intensity of the referred pain that has been present before the paroxysm began. The feeling of suffocation or impending death may be absent.

Such attacks require a visceral lesion to produce the antecedent referred pain. They are, therefore, best seen in cases of aortic regurgitation, whether of rheumatic origin or not.

CHAPTER II.

THEORETICAL.

§ 1. *The Conditions within the Heart which lead to Referred Pain.*

Up to this point I have only attempted to show an association between certain physical signs and the presence or absence of referred pain. I shall now consider the significance of these signs, and attempt to show why they are associated with referred pain.

In an ordinary case there can be but little doubt about the presence of aortic stenosis and regurgitation. However, it

is not always so easy to be certain of the amount of the stenosis or the regurgitation. For it is only necessary to remind the reader that a systolic murmur over the aortic area, with the distribution of a murmur of aortic stenosis, may appear as a consequence of a roughening of the valves only, without any true stenosis. In the same way, a diastolic murmur, resembling the murmur of aortic regurgitation, may be present without abolishing the aortic second sound. In both these cases the pulse will help to show that but little stenosis is present in the one case, and little regurgitation in the other.

True aortic stenosis throws more work on the wall of the left ventricle during systole. The ventricle, therefore, hypertrophies, compensation takes place, and the patient suffers from no symptoms. Not so aortic regurgitation. For here the ventricle empties itself during systole, but as soon as the wall relaxes, at the beginning of the diastole, the blood pours back from the aorta and distends its cavity. In pure aortic stenosis the ventricle has to perform increased work during the systole, but is at rest during diastole. In pure regurgitation, on the other hand, there is not necessarily any increase in the work required during systole; but just at the moment when the wall is not toned to meet an effort, the blood comes pouring back into its cavity from the aorta. Now it is this distension, at the moment when the walls of the cavity are at rest, that is most pernicious, and it is this condition, above all others, that is most liable to lead to referred pain.

When both regurgitation and stenosis are present the condition is still worse. For not only has the systole to overcome a greater intra-ventricular resistance, but the diastole is broken by a back rush of blood from the aorta. The left ventricle hypertrophies to meet this increased resistance in front, but the increase in the force of the ventricular contraction will also increase the force with which the blood rushes back from the aorta at the beginning of the diastole. Thus referred pain is more likely to be present with aortic regurgitation than with stenosis, but most certainly when both lesions are combined.

But pressure within the cavity of the ventricle during the diastolic period gives rise to a second evil. The walls of the cavity are particularly liable to dilate, and the contents of the cavity to permanently increase. The ventricular wall now acts at a disadvantage on its contents, and they may possibly not be completely expelled during the systole. Thus the ventricle is never actually empty, but is always somewhat distended, an ideal condition for the production of referred pain. In most cases this is probably but a temporary condition, arising from some additional exertion in a heart normally working at the top of its power. So long as the patient remained more or less at rest, the ventricle is able to empty itself during systole; but it is working at the extreme limit of its power, and a little extra difficulty will render the complete emptying of the cavity at each systole impossible. In young people some excessive effort is necessary to produce this condition, but in older people, and especially in those cases where the lesion of the aortic valve is not of rheumatic origin, it may arise after any small exertion, such as getting up after a rest of a day or two in bed.

This permanent distension of the left ventricle cannot exist long, or to any very great extent, without the mitral valve becoming incompetent. Directly this happens the pressure within the ventricle becomes relieved, and the cavity is again emptied of blood at each systole. An enormous load is taken off the walls of the ventricle, for the blood can now leave its cavity by two roads. If the resistance in front is great, a considerable quantity of blood is now pumped, at each ventricular systole, into the pulmonary circulation, through the incompetent mitral valve, for the highest pressure in the lungs is low compared with that in the aorta. The aorta then receives less blood, the pressure within it falls, and the tension in front is relieved. Thus, as soon as the mitral valve yields, the permanent pressure within the left ventricle is relieved by a self-regulating mechanism, and referred pain disappears.

In the same way the essential conditions for the production of referred pain are absent in those cases where the

incompetence of the mitral valve arises coincidently with disease of the aortic valves during acute rheumatism.

Thus, whether the yielding of the mitral valve be a primary or a secondary occurrence, free regurgitation through this orifice acts as a safety vent to the ventricle, and prevents that distension of its cavity during diastole, necessary for the production of referred pain.

So far, the physical signs, upon which we have depended for a knowledge of the condition of the circulation in valvular disease, are fairly easy of interpretation. But a similar attempt to translate the physical signs produced by disease of the mitral valves, is beset with many difficulties. I will, therefore, begin with those cases where the interpretation of the physical signs presents least difficulty.

If a systolic murmur, audible at the apex and well conducted to the back, abolishes the first sound at the apex, in the axilla, and at the angle of the left scapula, and especially if the pulse is at the same time one of low tension, the lesion of the mitral valve is probably pure incompetence.

In such cases the blood is expelled by each contraction of the ventricle, not only into the aorta, but also into the auricle. A free path is open into the pulmonary circulation, and the pressure in the ventricle, therefore, always remains low. With each diastole the ventricle dilates, and the blood flows in again from the auricle at the pulmonary pressure. Thus the heart has to act more frequently to circulate the same amount of blood, and the pulse is increased in rate, but its tension is low. As extra work has to be done by the left ventricle, its wall hypertrophies, and complete compensation may occur. It is true the auricle receives blood from the left ventricle during the auricular diastole, but the blood does not enter at high pressure. Moreover, the left auricle can empty itself freely at each stroke through the free opening of the mitral valve, and thus the mean pressure within its cavity does not rise. Hence such cases of pure mitral regurgitation are the least serious of all cardiac lesions, and are unattended by referred pain.

Let us now consider the condition of the circulation in a pure case of mitral stenosis, characterised by a pre-systolic

murmur of the typical quality, terminated by a sharp first sound. It is almost universally acknowledged that this murmur is produced by a jet of blood forced through the narrowed mitral orifice by the sharp contraction of a well-acting and probably hypertrophied auricle. For it is the most capricious of all cardiac murmurs; it disappears as soon as the heart shows signs of failing, but reappears as the patient recovers. It is always absent some time before death. If absent when the patient is at rest, it reappears when, by any exertion, such as lifting the arms, extra work is thrown on the heart. The blood enters the auricle during the diastole. Then follows the auricular systole, which drives the blood forcibly through the narrowed orifice, producing the pre-systolic murmur. The ventricular systole follows immediately, and terminates abruptly the flow from the auricles. Then follows the auricular diastole, during which the cavity of the auricle can remain at rest, except for the somewhat high tension in the pulmonary circulation, which always follows a narrowing of the mitral valve.

Under such conditions the pressure in the auricle is high, but the contractile force of the auricle is increased to meet it; the diastole of the auricle is uninterrupted, and there is nothing to cause referred pain. It is, of course, possible that some exertion, which, though actually not great, is relatively considerable, owing to the diseased condition of the valves, may upset this circulation. Blood would then accumulate in the auricle, and it would remain permanently distended, and referred pain would result. This probably accounts for the occasional presence of referred pain in these pure cases of mitral stenosis. Pain is, however, the exception, and only occurs after some definite indiscretion, and not during the even tenour of a quiet life.

But imagine that just when the powerful auricular systole has expelled the blood through the narrowed mitral orifice, a strong jet of blood is forced back into its cavity, just as its walls are relaxing, we should then have exactly the condition which we found so fruitful a source of referred pain in aortic regurgitation, except that the auricle here

bears the brunt of the diastolic pressure instead of the ventricle.

This is the condition that I believe exists in those cases where an apical systolic murmur is present, accompanied by a short, sharp, accentuated first sound.

The actual mechanism of this short, sharp, first sound in mitral stenosis is still uncertain. But whether its origin be valvular or muscular, it seems to be associated with the contraction of a well-acting left ventricle on a light load. If the ventricle loses power, and dilates, this sharp first sound disappears. At the same time, the systolic murmur may become audible over the back and in the axilla. Thus, whatever the mechanism of this accentuated, sharp, first sound may be, it seems to be associated with a well-acting left ventricle and a stenosed mitral orifice.

It might be objected that in pure mitral regurgitation blood is also forced into the auricle at the moment of its diastole by the contraction of the left ventricle, and yet that this condition gives rise to no referred pain. But we must remember that in such cases the mitral orifice offers no obstruction. The contracting auricle can empty itself with ease into the ventricle, and the blood which regurgitates from the left ventricle passes easily into the pulmonary circulation. In mitral stenosis, on the other hand, before the left ventricle dilates and the heart fails, the tension in the pulmonary veins and left auricle are high, owing to the obstruction to the onward flow. This increase of tension is evident from the frequency of hæmophysis in cases of mitral stenosis. Out of twenty-five consecutive cases, eighteen suffered from hæmoptysis at one time or another. Thus even a slight increase in the auricular contents during its diastole, especially if it take place with rapidity and considerable force, must act injuriously.

The following considerations also show that the regurgitation in these cases of considerable mitral stenosis occurs at a considerable pressure. The pulse of mitral stenosis, at any rate in the earlier stages, is of well-maintained tension, though the stroke is poor. Now, before blood can leave the left ventricle, and pass into the aorta, the tension in the

aorta must be overcome. Thus the contents of the left ventricle must be under fair pressure. If regurgitation takes place from the left ventricle, this regurgitation must, then, also take place under fair pressure. On the other hand, the pulse of pure mitral regurgitation is distinctly one of low tension, and easily compressible. This points to the regurgitation through the mitral orifice also occurring at low pressure.

Thus, in conclusion, it would seem that the most potent conditions for the production of referred pain are: firstly, the maintenance of considerable tension within a cavity of the heart, accompanied by, secondly, a sudden accession of tension (owing to regurgitation) at the moment when the walls of that cavity are dilating after systole.

Throughout this paper I have omitted all consideration of complex combinations of valvular lesions. For, although such cases form interesting problems for solution, when the principles which govern the presence or absence of referred pain are once determined, the conditions of the circulation are too complicated to aid in the determination of such principles. Moreover, the various groups into which such cases fall, do not occur with sufficient frequency. I have also neglected an interesting group of cases where referred pain appears as the accompaniment of acute endocarditis, arising during the course of an attack of acute rheumatism.

§ 2. *The Sensory Supply of the Various Portions of the Heart.*

I have attempted to show, in the previous section, that the referred pain, which occurs in certain cases of mitral stenosis, is due to conditions of pressure within the left auricle. Now, on examining the records of such cases (p. 186 to 189), we find that the referred pain and superficial tenderness lie within the areas from the fifth to the ninth dorsal, but mainly within the sixth and seventh dorsal segments. Thus we may assume that if the explanation of the causes which give rise to referred pain in such cases is correct, the sensory impulses from the auricle pass into the

central nervous system at the level of the fifth, sixth, seventh, eighth, and possibly the ninth dorsal segments, but mainly at the level of the sixth and seventh dorsal.

In the same way the referred pain, in cases of aortic regurgitation, or of combined aortic regurgitation and stenosis, were attributed to abnormal pressure in the left ventricle. Now the pain and superficial tenderness in such cases lies mainly over the second, third, fourth and fifth dorsal, with occasional implication of the fourth cervical and sixth dorsal. Thus the impulses from the left ventricle probably pass into the cord at these levels.

A study of aneurisms of the aorta (p. 168) taught us that the referred pain and superficial tenderness they produce may be divided into three groups. Firstly, with aneurisms of the ascending arch, the pain and superficial tenderness lie over one or more of the following areas —first, second, third dorsal, third and fourth cervical. Secondly, if the transverse arch is implicated, the pain and tenderness may lie over the anterior surface of the neck, especially within the inferior laryngeal area. Thirdly, when we have reason to think that the aneurism has involved that part of the aorta which lies beyond the entrance of the ductus arteriosus, the pain and superficial tenderness may lie within the fifth, sixth and seventh dorsal segmental areas.

These results may be tabulated as follows :—

Sensory Supply of the Heart.

Transverse arch of aorta ...	Inferior laryngeal segment
Ascending arch of aorta ...	3rd and 4th cervical 1st, 2nd and 3rd dorsal [? 4th dorsal.]
Ventricle	2nd, 3rd, 4th and 5th dorsal [? 6th dorsal.]
Auricle	5th, 6th, 7th and 8th dorsal [? 9th dorsal.]

§ 3. *The Physiology of Cardiac Pain.*

Pain does not exist without a cause. It is simply the expression of the stimulation of sensory nerves within the heart, and this stimulation has a definite aim.

The first effect of the presence of pain will be to warn the patient against exertion. Under the influence of this warning the body is placed more at rest, and the heart will have a chance of recovering, in consequence of the diminished call made upon it. Pain, wherever it occurs in organic heart disease, is a warning and an intimation that rest is required.

But, probably, the stimulation of the sensory nerves, of which pain is the expression, performs a second function. We know that the depressor nerve conducts sensory impulses from the heart to the medulla, which cause dilatation of the visceral capillaries, and so lower the pressure in the distal portion of the vascular system. We also know that augmentor fibres pass from the dorsal cord to the heart, increasing not only the rate but the strength of its contractions. Now, these fibres have been found in the second, third, fourth, and fifth anterior roots, and it is quite possible that the sensory portion of the reflex arc is of much wider extent.

Thus it is possible that these pain impulses may be the expression of the afferent arc of one, or both, of these reflex mechanisms; at any rate, in those cases where the pain lies over the upper dorsal or third and fourth cervical areas.

It is, possible, therefore, that the implicated segments of the central nervous system are both acted on, and react upon the heart. This must be borne in mind during the study of paroxysmal attacks of referred pain. A very widespread disturbance of the heart, especially if it occur suddenly, will cause extremely widespread referred pain, owing to the intensity of the stimulus poured into the central nervous system. Before the disturbance in the heart's activity, no referred pain or tenderness will be present; but during and after the manifestation, both will be intense and widespread. This is the one extreme type of anginal attack. But, supposing pain impulses of considerable in-

tensity have been pouring into the segments of the central nervous system for a considerable time, the resistance within the nervous system may ultimately break down, and the impulses will spread up and down the cord. When the cord is widely implicated, especially if the impulses are of considerable intensity, the motor mechanism will become involved, and not only will the tension fall rapidly [depressor action], but the heart will beat with greater rapidity and force [augmentor action]. This is the second extreme type of anginal attack, in which the antecedent referred pain is the main feature, and the cardiac disturbance appears to be secondary to the spread of the pain.

But in order that the stimulation of this reflex arc may occur, the heart must not be worn out by disease of long existence. For, as I pointed out above, much referred pain may appear when the liver enlarges for the first time; but if it remains enlarged, or if the enlargement has occurred many times, the pain ceases to make its appearance, owing to the changes undergone by the liver substance. In the same way, so long as the condition of the heart remains fair, referred pain appears when the necessary conditions for its appearance are fulfilled, and the heart then reacts to the stimulus so produced. But, later on, towards the end of the case, the heart becomes worn out, and structural changes occur. Such hearts react neither to changes of internal pressure nor to drugs; and the patient passes away painlessly, and in spite of all our efforts.

§ 4. *The Distribution of Referred Pain and Tenderness in Diseases of the Heart considered from the Developmental Aspect.*

In section 2 of this chapter I attempted to map out the sensory supply of the heart, and the result must appear, at first sight, somewhat startling. For it would appear that the ascending arch of the aorta receives its innervation from the highest segments, whilst the impulses from the auricle enter the lowest segments that stand in relation to the heart. Thus the sensory visceral innervation of the heart is

upside down in relation to the present position of the adult human heart.

Now it is worth considering, shortly, what we know of the development of the heart in attempting to explain this remarkable fact.[1]

Let us take up the development of the heart at the stage where the two tubes have fused to form a single tubular heart. The hinder end is continuous with the two vitelline veins, whilst the anterior end bifurcates into two primitive aortæ. At this time, the heart lies in the middle line under the upper part of the alimentary canal, and clearly shows the division into three portions. The hindmost of these becomes the auricle, the middle portion the ventricles, and the most headward portion the bulbus aortæ, and ultimately the ascending arch of the aorta. This tube then becomes bent on itself, so that the hindmost, or auricular portion, comes to lie above the middle or ventricular portion.

Now the plan upon which the sensory supply of the sympathetic system to the viscera is laid down, seems to be very antique, and to represent an early stage of vertebrate phylogeny. The sensory supply of the heart bears out this statement, in that the impulses enter the segments of the central nervous system just as if the auricles were still the hindmost portion of the heart, and as if the heart were a median organ.

That portion of the aorta, from the valves to the innominate artery (ascending arch) is innervated like the ventricle, from the second, third, (and, perhaps, the fourth) dorsal segments, and also refers into the first dorsal, and third and fourth cervical areas. Thus the next segment, headward of the first dorsal, is the fourth cervical. The fifth, sixth, seventh, and eighth cervical are skipped, and a gap exists, as I pointed out in my two previous papers.

[1] The following considerations will be rendered clearer, to those of my readers who are not familiar with the development of the mammalian heart, by a reference to Figs. 176, 177, 178, 179, and 181 in Quain's "Anatomy," 10th edition, vol. i., part 1. The originals of these beautiful figures are found in His. "Anat. d. Mensch. Embryonen," a work that is in the hands of but few persons. I have, therefore, referred the reader to Prof. Schäfer's excellent modifications of the original figures.

Now the third and fourth cervical areas lie over a part of the neck, which is definitely somatic in origin, and has no connection with the structures formed from gill arches.[1] This area of skin grows forwards from the dorsal surface of the body, encroaching on the gill arches and their appendages. Now, as I pointed out before,[2] the sensory portion of the first cervical segment is contained in the trigeminal (fifth nerve), and the second cervical (whose sensory fibres enter in the cervical region at the proper level) receives its afferent impulses from the posterior part of the scalp. Thus the third cervical segment comes to be the highest segment which receives impulses from a true somatic skin surface, and is the uppermost segment which supplies true somatic viscera, *e.g.*, the ascending arch of the aorta, and the apex of the lung.

In front of the anterior border of each sterno-mastoid, lies a triangular area, which the dissector knows as the anterior triangle of the neck. Within this area referred pain and superficial tenderness appear, with lesions of such essentially branchial structures as the larynx and its appendages. The ventricles and the lung never primarily refer into this area. Thus, I pointed out that the areas within this region, like those of the jaws and face (excluding the nose, eyes, and forehead) belonged to a branchial system of segmentation, apart altogether from the somatic segmentation, and that of the areas on the forehead and scalp.

Let us turn for a moment to what we know of the development of the aorta in man and the higher vertebrates. The bulbus arteriosus is the most anterior or headward portion of the heart. It ends in front in a series of lateral vessels, which run from the ventral surface towards the back. The front four of these vessels run in the four branchial arches; of these the first three form the carotids and their branches, and do not concern us here. The fourth of these vessels, which runs in the fourth branchial bar, behind the

[1] *Cf.* Part II., BRAIN, 1894, p. 477; also His. "Anatomie Menschlicher Embryonen," Leipzig, 1885.

[2] BRAIN, 1894, p. 477.

third branchial cleft, forms the transverse arch of the aorta on the left side, and the innominate artery on the right side. The fifth vessel, which runs behind the fourth cleft, forms the pulmonary artery. On each side, dorsally, these five vessels are connected with one another. Thus, the fourth vessel, running in the fourth arch on the left side (the future aorta), unites, on the dorsal surface of the body, with the fifth vessel (the future pulmonary artery). The point at which this union takes place is marked on the adult aorta by the entry of the obliterated ductus arteriosus, which originally united the stem of the pulmonary artery with the aorta, and is the remains of the dorsal portion of the fifth branchial vessel. Thus we have now divided the aorta as we know it, in the adult with three portions: (1), from the semi-lunar valves to the level of the origin of the innominate. This is the representative in the adult of the bulbus arteriosus, the most headward division of the tubular heart. (2), from the innominate to the point of entry of the ductus arteriosus. This part represents, in the adult, the fourth branchial vessel. (3), all the aorta beyond this point—a true somatic dorsal vessel.

It is, therefore, peculiarly interesting to find that aneurisms of the ascending, or first part of the aorta, cause tenderness over the third and fourth cervical, and first, second, third dorsal segmental areas; all of which are purely somatic, and either identical with, or a continuation headwards of the areas to which the ventricle refers. On the other hand, aneurisms of the transverse arch, the remains of a true branchial vessel, cause pain and tenderness within the inferior laryngeal areas of the neck, in common with so truly a branchial organ as the larynx. This is the more instructive, from the fact that the larynx proper is developed from the structures of the fourth branchial bar, of which the transverse arch of the aorta represents the branchial vessel.

Much of what I have said in this section may be fanciful, and may be negatived by future investigation. But, at any rate, for the present, such embryological considerations seem to me to offer the only hypothesis to explain the distribution of referred pain and superficial tenderness in diseases of the heart and aorta.

CHAPTER III.

Pain in Diseases of the Respiratory Organs.

§ 1. *Character of the Pain.*

In my first paper I scarcely touched on the pain produced by diseases of the lungs, and neglected the pain which may occur in the course of phthisis; for whatever pain is present in this disease throws little light on the nature and causes of referred pain, and on the limits and anatomical distribution of areas of superficial tenderness. But although of but slight scientific interest for the determination of localisation, in the central nervous system, the distribution of pain and tenderness is of considerable practical importance, and occupies a considerable field in the clinical picture of many diseases of the lungs. The study of pain in its different forms in various diseases of the lung and pleura, also forms a useful demonstration of the methods by which widespread pain and large territories of superficial tenderness are to be interpreted.

Two quite distinct varieties of pain, accompanied by two even more distinct types of tenderness, may make their appearance in the course of diseases of the lung and pleura. Pain of the first type is local, and occupies no predetermined spots or areas. It is situated over the focus of disease, and usually in those intercostal spaces which cover a patch of pleural friction. It is accompanied by no true superficial tenderness, but marked deep tenderness is evoked by pressure or percussion. Pain of the second type is referred—that is to say, it is not necessarily situated over the focus of disease. It runs through or around the body, and has a focus behind and one in front. If severe, or of any duration, it is accompanied by more or less marked tenderness of the superficial structures of the chest, and this tenderness lies over predetermined spots or bands. Thus, referred pain and tenderness do not point directly, but only indirectly to the focus of disease.

(*A*) *Local Pain in Pulmonary Diseases.*

This type of pain occasionally appears in the neighbourhood of phthisical centres, or large areas of pneumonic consolidation; but in the large majority of cases it stands in definite relation to a pleuritic rub. It is said to be "stabbing" or "catching" in character. It is absent when the side is at rest, is intensified by each inspiration, and becomes agonising during the long and deep inspiration which follows a cough, a laugh, or a sneeze. It is relieved by any treatment that puts the side at complete rest, such as successful strapping. The patient describes the pain as like the sudden stab of a knife into the side. But this stab does not go through the chest from back to front, as is the case with severe referred pain, but is localised to one spot.

This pain is frequently associated with marked tenderness on pressure or percussion in the intercostal spaces.[1] Thus, by fairly heavy percussion, an area can generally be marked out within which deep tenderness exists. Now within this area pleuritic friction will usually be audible. I must warn the reader from supposing that referred pain and superficial tenderness never co-exist with an acute pleurisy. But where superficial tenderness, of the usual type that accompanies referred pain, exists, it is due to the disease of the lung, of which the pleurisy is a secondary phenomenon, and is not due directly to the pleurisy. For in a considerable number of cases of acute pleurisy all superficial tenderness is absent, and the presence of the patch of deep tenderness, corresponding more or less closely to the area of friction, can be easily demonstrated. Thus, before proceeding to test for deep tenderness, it is well to determine that all superficial tenderness is absent, or the results of the examination will be fallacious.

This deep tenderness, in most cases, seems to be definitely due to pressure upon the inflamed pleura. For it corresponds very fairly with the area of audible friction, and is less easily obtained over the ribs than over the inter-

[1] Walshe ("Diseases of Lungs," third edition, p. 269) recognised this tenderness on pressure.

costal spaces. It disappears with time, even though the friction may remain, and thus it is sometimes a useful guide to the duration of a friction sound, present when the patient first comes under observation. Local pain and deep tenderness usually disappear when serous fluid is effused; but I have more than once seen it persist over the upper part of the purulent effusion in empyema.

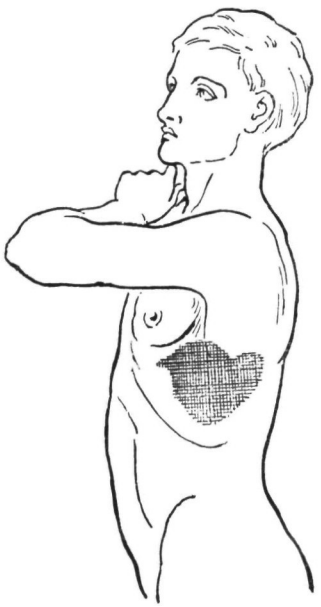

FIG. 14.

To show the area of deep tenderness evoked by pressure or percussion in a case of acute pleurisy [Case No. 60].

Case 60.—*To illustrate the local pain and tenderness on deep pressure which accompanies a pleurisy.* John K. (V.P.H., Dr. Heron); aged 44; coachbuilder.

In 1888 he is said to have suffered from pleurisy, but no detailed account of the attack can be obtained. Some cough each winter since then, but he has been able to do his work. Is a teetotaller. No family history of phthisis.

On admission (December 11, 1893).—Medium-sized well-built man of 44. Looks older. No marked anæmia. No wasting. No cyanosis. No night sweating.

Respiration, 24. No marked dyspnœa. Hard cough in paroxysms, which seem to choke him. Expectorates mainly aerated mucus. No tubercle bacilli found in sputum.

He complains of pain in seventh, eighth, and ninth spaces in the left axilla. The pain is increased by taking a deep breath, and becomes exceedingly bad in a paroxysm of coughing.

No superficial tenderness, but marked tenderness on pressure or percussion over a patch the size of the palm of the hand in the seventh, eighth, and ninth spaces (fig. 14). No headache. No scalp tenderness.

Both sides of the chest move equally. Percussion note somewhat hyper-resonant. Breath sounds everywhere weak, but expiration is not prolonged. Vocal resonance everywhere feeble. A few scattered râles are heard over the lower part of the left lung.

In the left axilla marked rough friction is heard, extending over the seventh, eighth, and ninth spaces. The area within which friction is heard almost exactly corresponds to the area of deep tenderness (fig. 14).

No other signs or symptoms of importance. Temperature not raised.

December 26.—Friction is less marked. All pain has practically gone, except when he takes a very deep breath. The area of deep tenderness is contracting, and is less marked.

January 3.—All pain and deep tenderness gone.

Sometimes an infarct in the lung will cause a small patch of pleurisy if situated near the surface. Such cases form exquisite examples of the localising value of deep percussion tenderness. I have several times had the opportunity of verifying the position of the infarct which caused this local tenderness. In Case 61 both infarcts were diagnosed during life, and verified after death.

Case 61.—*To illustrate the deep tenderness sometimes present when infarcts of the lung cause small patches of pleurisy.* Emma D. (V.P.H., Dr. Harris); aged 47; nurse.

At the age of 14 years she had chorea, "due to a fright." It lasted fifteen months. At no time in her life has she had anything resembling acute rheumatism.

She had been working very hard as a sick nurse, and on December 10, 1893, began to feel very tired, hot, and feverish. December 11 her temperature was 104°. She was in bed for a fortnight, with a varying temperature, and then got up. January 10, 1896, the temperature rose again, and she felt very ill.

On admission (January 18, 1896).—She is a small, used-up looking woman of 47. Arcus senilis beginning to appear in both eyes. Face of a grey, dusky pallor.

Respiration rapid. Very short of breath. Some orthopnœa.

Directly after admission she began to complain of pain in the *right* axilla in the sixth, seventh, and eighth spaces. There was no superficial tenderness, but exceedingly marked deep tenderness in the sixth, seventh, and eighth spaces in the mid-axilla.

January 19.—The percussion note is diminished over the lower axilla on the right side, extending back to the line of the angle of the scapula. Over this area the breathing is tubular, and the vocal resonance increased. Over the area of deep tenderness in sixth, seventh, and eighth spaces, coarse friction is audible.

Elsewhere lungs are normal.

Pulse 88, and irregular in rate and force. The pulse is small, but hard.

Apex beat in fifth space and nipple line. Epigastric pulsation. No thrill. The first sound at the apex is sharp, and the second re-duplicated. Pulmonary second sound accentuated. No murmur heard at first.

Liver and spleen not felt.

Urine showed a faint cloud of albumen.

January 20.—She began to spit up red or purple-coloured sputum.

January 22.—All deep tenderness has gone; but friction was occasionally heard over the same area.

January 23.—She began to complain of pain in the *left* axilla, situated in the seventh and eighth spaces in the mid-axilla.

There is exceedingly well marked deep tenderness in the seventh and eighth spaces in the mid-axilla, but no superficial tenderness anywhere.

On the night of January 23, friction appeared over the area in the left axilla.

The diagnosis was made of an infarct in the right lung, followed by a second infarct in the left lung; both reaching the surface, and causing slight pleurisy.

February 20.—She suddenly became hemiplegic, without loss of consciousness.

She then gradually got weaker, and died on March 20.

Post-mortem.—Before the examination was begun, the areas of deep tenderness in the right and left axilla were marked out upon the skin. Dr. Fyffe made a careful dissection, and found two infarcts, as follows. In the right lower lobe was an infarct, a little more than an inch across the base, which had caused thickening of the pleura in its immediate neighbourhood, and this area of thickening lay under the area marked out on the surface. A similar recent infarct, causing a patch of pleurisy, was found in the base of the left lung, and this infarct also lay under the area marked out in the left axilla.

Mitral orifice much stenosed, admitting only one finger. About the edge of the valves were some recent vegetations, and a small erosion. Other valves normal. Hypertrophy and dilatation of left ventricle. No micro-organisms could be found in the valves.

Liver and spleen enlarged.

In the brain an infarct was found, causing softening of part of the internal capsule.

(*B*) *Referred Pain in Pulmonary Disease.*

The second type of pain met with in diseases of the lungs is that which is usually known as referred pain. It is of no direct localising value, but is situated at certain spots on the chest, or may appear to run round the body at various levels. In these spots we recognise the maximum points of certain areas connected with the central arrangements of the nervous system. Pains of this type are usually described as "intercostal neuralgia," "myalgia," "intercostal rheumatism"—names which are either meaningless, or else imply a false pathology. For these pains do not follow the course of the intercostal nerves, but occupy part of the territory of one nerve, and part of that of another. They are said to be "stabbing," "catching," "aching," or "tight;" and in some cases the patient complains of a true girdle sensation, with a feeling as if his chest were tied in at a certain level. The pain is not usually increased, to any marked extent, by each act of inspiration, but is markedly increased by anything that throws increased work upon the diseased lung. Thus the dyspnœa produced by going up a flight of stairs, will frequently bring on, or increase, referred pain, although each respiratory act is not peculiarly associated with an increase of pain.

Such referred pains are associated with tenderness of the superficial structures of the chest. Now it has been long known that phthisical patients are subject to soreness and tenderness of the chest walls, but this tenderness has been considered of little importance. Walshe[1] noticed this superficial tenderness, and points out that "firm pressure over a broad surface gives relief." Valleix[2] pointed out that what he called intercostal neuralgia, was accompanied by three tender points: one behind, one in the axilla, and one on the front of the chest. This observation, Walshe confirms; and

[1] Third Edition, p. 192. [2] "Traité des Névralgies."

points out that no one intercostal nerve is affected, "the pain usually involves the nerves from the sixth to the ninth —in rare instances a single trunk only."

But, in spite of the common neglect of superficial tenderness by the physician, this feature of the disease is well recognised by many patients. They complain that the neck, chest, back, or upper abdomen feels sore and tender, as if it had been beaten or was bruised. They tell you that they have been obliged to loosen their clothes, to wear soft things next the skin; or, in the case of women, that they have been obliged to loosen or remove their corsets. On picking up the superficial structures between the finger and thumb, areas are found, within which the patient complains that the manipulation, however gentle, causes pain. By using the blunt head of a pin these areas can be defined; for the patient either complains that certain parts are sore when touched with the head of the pin, or else he experiences a sensation as if he were being pricked. I must, however, here warn the observer of the fallacies which are attached to the use of the head of the pin upon the anterior and lateral aspects of the chest. He must be very careful, in these regions, that the head of the pin does not impinge on the edge of a rib; for this will cause pain even in a normal individual. Thus I always prefer to lift the skin between the finger and thumb when testing areas on the chest, excepting over such parts, at the breasts,[1] where the pin is obligatory. Here, however, the ribs are so well covered that fallacious results are less likely to be obtained with the head of a pin.

Another fact which shows how purely such pain and tenderness depend upon impulses passing into the central nervous system, is the association of such referred pain on the chest, with areas of referred pain and tenderness on the scalp. This association takes place according to definite rules, which I have laid down in Part II.[2] of this investigation;

[1] In multiparæ the superficial structures of the breast are comparatively insensitive; and superficial tenderness that is well marked over the back and rest of the chest may frequently be almost absent over the breasts, although the breast lies within the tender areas.

[2] BRAIN, 1894, p. 436.

and the occurrence of such scalp tenderness in diseases of the lungs will be repeatedly alluded to in subsequent sections of this chapter.

§ 2. *Conditions necessary to the production of referred pain in diseases of the lungs.*

Referred pain is produced by distention of a sensitive organ from within, or by the exertion upon it of some tearing or rending strain from without. Thus, if the intestine is distended, referred pain may result. If, on the other hand, it is cut with a sharp knife, as in the second stage of inguinal colotomy, no pain is produced; but if, in the process, it is torn or pulled upon, the patient complains of pain. Moreover, it is essential that, at any rate, some of the end organs within the diseased area should be intact, in order that referred pain may result. Thus no true referred pain and superficial tenderness result from a tooth in which the pulp is dead, although much local pain may be caused by inflammation around the root. Thus any disease which prevents all movement in the diseased organ, or any disease that rapidly destroys its tissues, including the nerve endings, will be unaccompanied by referred pain.

Now, in acute lobar pneumonia, the essential process of the disease is a consolidation of one or more lobes of the lung, with complete obliteration of the alveoli by exudation. The air no longer enters the alveoli of the whole consolidated lobe. But, in spite of its profoundly diseased condition, such a consolidated lobe gives rise to no referred pain, for the whole of the end organs are removed from the influence of pressure from within, or traction from without, by this consolidation. Thus the large majority of cases of acute lobar pneumonia exhibit no referred pain or superficial tenderness, and associated with this absence of referred pain there is no referred headache, or tenderness of the scalp. But in a considerable number of cases of acute lobar pneumonia, the consolidation of the lung is associated with pleurisy of greater or less extent. To this is due the local pain, frequently accompanied by tenderness on percussion or pressure, which is not

an uncommon feature of this disease. I give an instance in which both lower lobes were consolidated; but the only pain that was present was localised over the left base, where marked friction was audible.

Case 62.—*To illustrate the absence of referred pain and superficial tenderness in Acute Lobar Pneumonia, and the dependence of the pain frequently present in this disease on coincident pleurisy.* Luke L. (V.P.H., Dr. Thorowgood); aged 38; Wood carver.

On the evening of March 29, 1894, he was seized with cold shivers. The next day pain came on in the left side.

On admission (April 3).—Well-built man. Face of a pinkish-yellow colour, with a patch of herpes about the right, lower lip, extending outwards to the cheek. No marked cyanosis.

Respiration 24. Sterno-mastoids acting slightly. Some subjective dyspnœa. He can lie on both sides, but prefers to lie on the left side. Short paroxysms of cough. At infrequent intervals he spits up a small quantity of greenish-brown, frothy expectoration.

He complains of pain over the middle of the left axilla in the seventh, eighth, and ninth interspaces. No superficial tenderness, but extremely well-marked tenderness on percussion, or deep pressure was evoked in the sixth, seventh, eighth, and ninth spaces from about an inch from the costal margin below to the line of the latissimus dorsi above.

No headache. No scalp tenderness.

The percussion note in the left axilla, and below the level of the sixth spine on the left side, is absolutely dull and resistant. Over this area the breath sounds and vocal resonance are almost completely abolished, and in the sixth, seventh, eighth and ninth spaces in the axilla, marked friction is audible.

The percussion note over the right base and in the right axilla is tubular. Over this area the heart sounds are weak, and the vocal resonance fair. Numerous fine crepitations are heard, but no friction is audible.

The temperature was 102·8° F. (39·3° C.) The pulse was good. The urine contained no albumen.

All pain and deep tenderness rapidly disappeared, and on May 6 the temperature was absolutely normal; and the lungs presented no abnormal signs, except a very slight diminution of note, with weak breathing and vocal resonance below the angle of the left scapula.

Thus the form assumed by the disease is all important in determining the presence or absence of pain in the course of phthisis and other diseases of the lungs.

Three stages—invasion, consolidation, and excavation

—are usually described for tubercular phthisis; but experience shows that they occupy a very variable amount of the field in the clinical picture of any one case. Thus, in the one extreme type, the patient scarcely complains of anything beyond a cough in the mornings, and a feeling that he is not up to the mark. And yet, when he seeks advice, marked signs of consolidation, and, perhaps, of excavation, are present over the upper part of one lung. The temperature may be very irregular—greatly raised one night and scarcely raised at all the next. The patient may gain weight under treatment, and take his food well; but in spite of this, the cavitation may steadily advance. For whilst, at first, the signs of a cavity may extend to the level of the third dorsal spine, in two or three months similar signs may be present as low as the sixth spine. Yet during this time, all referred pain, referred headache, tenderness of the scalp, and superficial structures of the chest may be entirely absent.

From this point onwards the course followed by the case may be very variable. A sharp rub may make its appearance about the angle of the scapula, or in the axilla; and the whole base of the lung may become adherent; or the upper lobe, or the apex of the lower lobe of the opposite lung, may become rapidly excavated. But neither of these processes is associated with referred pain or superficial tenderness. Thus, in an extreme case of this type, the patient may advance far with the disease without suffering from any referred pain.

Moreover, when excavation, or pleurisy, or an association of both processes, form the main feature in the course of a case of phthisis, the parts which are not visibly affected by tubercle tend to become markedly fibrotic. Thus, in such a case, the adherent, but otherwise unaffected, bases are found, *post-mortem*, to contain excess of connective tissue. They are thus in an unfavourable condition to give rise to referred pain when actually invaded by tubercle.

Thus, what may be called the excavation and pleuritic type of phthisis may run on to the end without showing any referred pain or superficial tenderness.

Case 63.—*To illustrate the absence of referred pain and superficial tenderness in those cases of phthisis where excavation is a marked and early feature of the disease.*—Herbert K. (V.P.H., Dr. Heron); aged 26; groom.

He was in splendid health until October, 1893. He was then seized with some acute illness. On the second and third days he lay in bed. On the fourth day he got up, and on the fifth day a cough came on, and has not left him since.

He felt languid and ill, but was able to continue work till November. At the end of November he began to sweat at night, and about the beginning of December he began to spit up with his cough. He has lost 7½ lbs. in four months. He has been free from pain throughout.

On admission (January 22, 1894.)—Well-built man of 26. Not anæmic. No marked cyanosis. Sweats at night.

Respiration 28. Short of breath on considerable exertion, but not when walking quietly. Cough, single; not very troublesome. Small quantity of expectoration, containing tubercle bacilli.

No pain. No headache. No superficial tenderness anywhere.

The right apex moves less than the left, both in front and behind. The percussion note is diminished on the right side, over the first two spaces in front, and as low as the level of the fourth spine behind. Over this area, both in front and behind, marked cavernous breathing and whispered pectoriloquy are heard, with many metallic râles. A few scattered râles are heard behind over the upper part of the right lung; but the breath sounds at the bases and at the left apex are normal. Larynx, normal. No abnormal signs in heart. Tongue, moist. Appetite, poor. No nausea or vomiting. Bowels, normal. Urine contains no albumen, and is acid.

The temperature rose to 102° F. (39° C.) every night, but fell to normal in the morning.

The signs of cavitation at the right upper part of the right lung spread downwards rapidly, and by March 3 cavernous breathing and whispered pectoriloquy could be heard as low as the level of the sixth spine.

Then sharp, dry friction appeared at the angle of the right scapula, and by April 12 the movement of the right base was much impaired, the note was diminished, and the breath sounds very weak all over this base.

The temperature still remained very high, and he had lost 6½ lbs.

No referred pain, headache, or superficial tenderness appeared throughout the whole time he was under observation.

This case is an extreme instance of phthisis, running a course unattended by referred pain. But the disease may advance in a manner that is so different to the instance I

have just given as to form an almost exactly opposite type. When the patient first seeks advice, there is, perhaps, a little deficiency of movement at one apex. The percussion note is not definitely altered in front or behind, but a few sharp crepitant râles are heard over the upper part of the lung on the side where the movement is deficient. There is a good deal of cough, and some expectoration, in which tubercle bacilli can be found, generally in considerable numbers. The patient may complain of some aching pain about the clavicle, but there may be little or no superficial tenderness. Then the signs rapidly increase, and fine crackling râles may become audible over the upper part of the lung as low as the level of the fourth or fifth dorsal spine, and a few râles and rhonchi may be heard creeping round the vertebral border of the scapula. The breath sounds over the upper part of the affected lung are feeble, and the vocal resonance perhaps a little increased. The patient now complains of referred pain in the affected side of the chest, and more or less superficial tenderness is present both on the chest and on the scalp.

Let us suppose that the left side is first affected. This side may now remain quiet for a time, and, although the lower part of the interscapular fossa may be free from râles, they persist over the upper lobe and apex of the lower lobe of the left lung. All referred pain and superficial tenderness may die away. Then, after a varying interval, a sudden outburst of râles and rhonchi may appear over the right side, extending to the level of the angle of the scapula. Referred pain will now reappear mainly over the right side of the chest and back, accompanied by widespread superficial tenderness, more marked on the right side than the left, but present on both sides of the chest.

The acute signs on the right side may now die away, leaving the upper lobe and apex of the lower lobe damaged, as shown by the weak breath sounds and a few irregular râles. The referred pain and superficial tenderness may either disappear, or be very much diminished in intensity and extent.

The next acute outburst may occur on the same or on

the opposite side, and be accompanied by a further development of referred pain and superficial tenderness of the chest and scalp.

Each such acute outburst subsides, but leaves the lung more damaged than before, and, sooner or later, signs of excavation make their appearance, generally at the upper part of the lung first affected. But for some time after the first signs of excavation have appeared, the progress of the case is characterised by periodic outbursts of râles and rhonchi over the lower portions of the lungs.

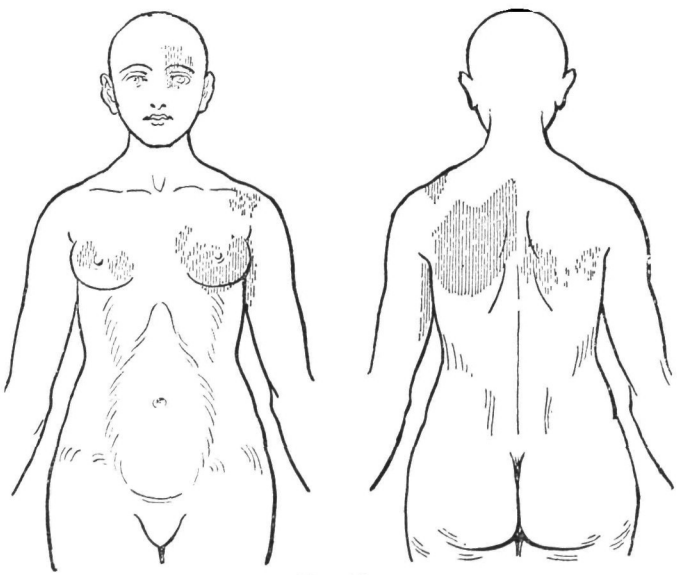

FIG. 15.

To show the superficial tenderness present in a case of phthisis [No. 64] during the implication of the left lung [Jan. 25].

It is this type of phthisis (of which I have given an extreme example in Case No. 64) that is accompanied by referred pain and superficial tenderness in its more exquisite form. The reason for this, I conceive, to be that, during the bronchitic type of invasion, the alveoli are rather encroached upon than primarily affected. The end organs are irritated but not destroyed, and are capable of conveying impressions even from the diseased portions of the lungs.

Case 64.—*To illustrate the type of Phthisis in which the advance takes place by repeated so-called bronchitic attacks.* Catherine C. (V.P.H., Dr. Harris), aged 24; housewife.

About August 1893, she began to have a cough and to spit phlegm, occasionally streaked with blood. She then began to lose flesh. November, 1893, she began to have pain in her left side.

On Admission (December, 15, 1893).—She is a largely-built woman, distinctly wasted. Face pale. No cyanosis. Sweats profusely at night. The temperature was 102° F. (38·9° C.) on the first night. This fell to 98° F. (36·7° C.), in the morning; rising to 100·4° F. (38° C.) the second evening.

Respiration 28. The alæ nasi dilate. Some subjective dyspnœa. Cough not very troublesome. Expectoration, in considerable quantity, consists of small masses of muco-purulent material, streaked with blood, and contains tubercle bacilli.

No pain. No headache. No superficial tenderness.

The movement at the apices is somewhat poor, and the right base moves rather more freely than the left. There is no very marked difference in the percussion note over the apices, but the note over the left apex in front is perhaps a little less resonant than over the right. On the left side, in the first and second interspaces in front, and as low as the second spine behind, the breath sounds are weak, vocal resonance increased, and numerous crackling râles are audible. Otherwise there are no markedly abnormal signs in the lungs.

Pulse 94. Small stroke and regular. Heart sounds normal. Tongue moist, and a little grey; papillæ prominent, not actually furred. Appetite good. Bowels irregular. No nausea. No vomiting. No pain after food. Liver and spleen not felt. Urine acid; no albumen, nor sugar.

January 14.—The temperature ranged to 101° F. (38·3° C.) every night during the first month. She has had very slight referred pains, but now no definite superficial tenderness could be found.

The signs at the left apex have increased. There is no distinct diminution of note over the upper two spaces on the left side in front. Over this area the breath sounds are feeble, expiration distinctly prolonged, and vocal resonance is markedly increased. Numerous fine crackling râles are heard as low as the third rib.

Behind, on the left side, the note is diminished as low as the level of the fourth spine. The breath sounds are exceedingly feeble, and the vocal resonance is a little increased. Fine crackling râles are heard to the level of the fourth spine behind.

No abnormal signs on the right side.

January 25.—She complains of shooting pains in the left side of the chest and left half of forehead, accompanied by superficial tenderness, as in fig. 15.

The cough is not more troublesome, nor the expectoration more profuse. The crackling râles at the upper part of the left

lung are more numerous, and extend down to the level of the sixth spine behind. When the left hand is placed on the right shoulder, these crackling râles are heard along the vertebral border of the scapula, but when the arm is dropped to the side they are not heard at the angle of the scapula.

January 30.—The signs on the left side of the chest have extended a little lower, but have not materially altered. A sudden outburst has, however, taken place on the *right* side, and crackling

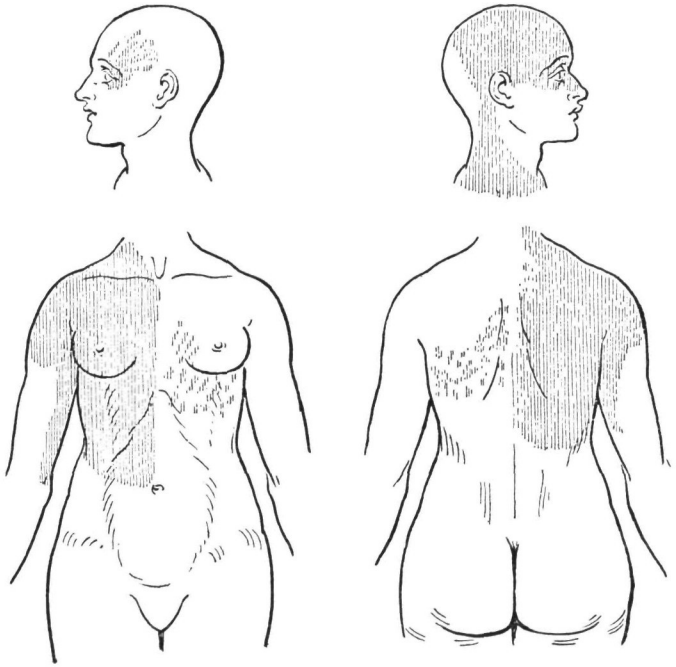

FIG. 16.

To show the superficial tenderness present in the same case of phthisis [No. 64], in a later stage, during the implication of the right lung [Jan. 30].

râles are heard over the whole upper part of the inter-scapular fossa, and about the angle of the right scapula.

The pain has shifted. She complains now of much pain all down the right side, and over the right half of the head. Extremely widespread tenderness is present over the right side of the body and scalp, such as not infrequently accompanies an acute outburst of this kind. (Fig. 16).

February 15.—She is now complaining of pain over the epigastrium, lower ribs, and under the shoulder blades behind. The

headache is mainly on the top of her head. Marked superficial tenderness as in fig. 17.

The appetite is poor. The tongue is clean. Bowels opened daily. No diarrhœa or constipation now. She is beginning to feel a little sick, but has not vomited.

On the left side in front, the dulness extends to the fifth rib. Cavernous breathing and whispered pectoriloquy are heard in the upper two spaces. Behind the second space the heart sounds are very weak, and the vocal resonance increased. Numerous râles audible.

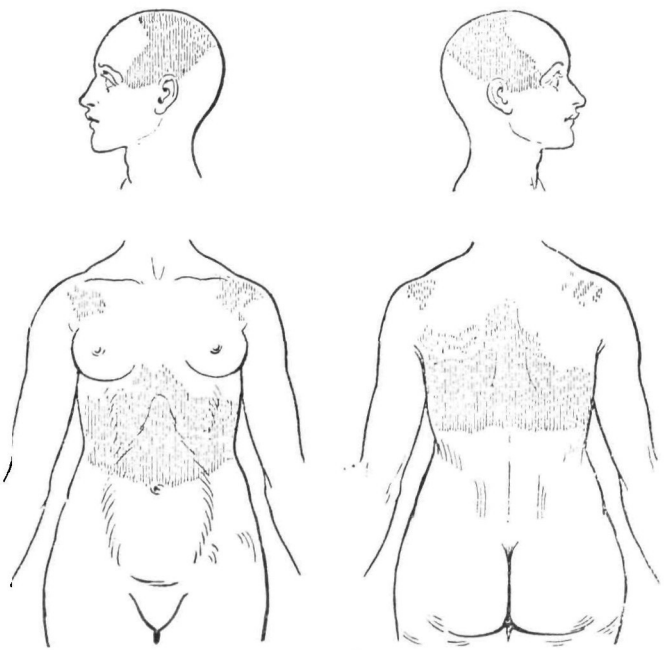

FIG. 17.

To show the superficial tenderness present during a later stage of the same case [No. 64].

Over the upper part of the left lung behind, the note is dull as low as the sixth space. The breath sounds over the apex are very weak, but expiration is hollow, and bronchophony is heard. Behind this to the base the breath sounds are very feeble, expiration is prolonged, and vocal resonance is increased. Râles are heard behind from the apex to the level of the eighth spine.

On the right side in front, râles are heard to the level of the fifth spine. Behind the note is somewhat diminished, but less so than on the left side. The breath sounds are feeble, and

expiration is prolonged as low as the angle of the scapula, and over the whole of this area, vocal resonance is increased, and numerous râles are heard.

Thus both bases are now being steadily affected.

She remained in this stage till March 11, when another outburst took place over the whole right lower lobe, especially about the angle of the right scapula, and in the right axilla.

This produced another widespread outburst of superficial tenderness of both sides of the body and scalp.

The temperature now rose to 103° F. (39·5° C.) every night, and she became very weak.

Thus, in the space of three months, the march of the disease was as follows :—Firstly, affection of the left apex, not associated with referred pain. Secondly, spread along the upper border of the left lower lobe (fig. 15). Thirdly, affection of the apex of the right lower lobe, together with an acute outburst within this lobe (fig. 16). Fourthly, steady advance down both sides within the lower lobe (fig. 17). Fifthly, an acute outburst over the whole of the right lower lobe, from which it never even partially recovered.

Between these two extreme types of invasion and progress innumerable variations occur, but the more nearly the course of a case of phthisis approaches the bronchitic type, the more certainly will referred pain and superficial tenderness tend to appear, and form a feature in the clinical picture. The more nearly it approaches the excavation and pleuritic type, the less likely are referred pain and superficial tenderness to make their appearance.

Thus, when considering the origin of referred pain in any particular case of phthisis, many different factors have to be taken into account. No referred pain accompanies rapid and absolute consolidation of one lobe, or its subsequent excavation. On the other hand, partial implication of the lung by multiple foci of disease [not acute miliary tubercles] scattered amongst relatively healthy tissue, or the gradual opening out of a collapsed or airless patch, are particularly liable to be associated with referred pain. Thus, the physical signs which most frequently stand in connection with the development of referred pain in the course of a case of phthisis, are small sharp, dry râles and sibilant rhonchi. But these small râles may be present for months over some particular area of the chest without there being any referred pain. Thus, it is not the quality nor the nature of the

adventitious sounds which is important, but the sudden appearance of these small râles and sibilant rhonchi over parts where they have not been audible before. The larger and moister râles are not usually associated with referred pain.

§ 3. *An attempt to connect the presence of certain areas of superficial tenderness with disease of certain portions of the lung.*

Now that we have considered the conditions and physical manifestations, which are associated with referred pain in the course of a case of phthisis, it remains to be seen how far such referred pain represents the position of the disease.

At the outset, we are met by the difficulty that every referred pain, when sufficiently intense, or of sufficiently long duration, passes over and affects the same areas on the opposite side of the body. Thus, if the patient complains of pain under the shoulder blades and across the lower part of the chest, we have to enquire on which side the pain began, and on which side it is worse. Both, however, depend upon the patient's memory and observation, and the answers are, therefore, of but little use in practice. Far more valuable is an examination of the condition of the superficial tenderness on the two sides of the body. If the tenderness on the one side is very marked, and extends completely round the body from the middle line behind to the middle line in front; whilst on the opposite side the intensity is less, and the distribution spotty, and only the maximum points well marked, the referred pain probably began over the former and spread to the latter side. Sometimes the headache and scalp tenderness afford a guide to the side upon which the disturbance began, for the headache and scalp tenderness are usually more marked upon that side upon which the pain on the body is most marked.

By intensity of tenderness, I do not mean an expression of the patient's opinion as to which side is the most tender. I judge of the intensity of the tenderness (1) by the sensation produced by the blunt head of a pin. Stimulation with the

blunt head of a pin may only cause a feeling of soreness over the tender area, if the tenderness is but slight, or, if the tenderness is more acute, the patient may complain that he is being pricked or stabbed. Thus, in the minor degree of tenderness, the patient recognises a change in the sensibility of his skin, whilst in the major degree he complains of a change in the nature of the stimulus. (2) By the completeness with which the bands of tenderness are represented on the surface of the chest. (3) By the exaggeration of the superficial reflexes.

But another disturbing factor in the attempt to determine the localising value of referred pain in phthisis lies in the fact that a fresh outbreak in one part of the lung is usually associated with a rise of temperature. Now, wherever referred pain and tenderness are present, and a sudden rise of temperature occurs, the pain, and particularly the tenderness, tend to spread widely. Thus a sudden acute outburst over the upper part of one lower lobe of the lung will not unfrequently lead to exceedingly widespread tenderness of the neck, trunk, and scalp, mainly on the side of the fresh lesion, but also on the opposite side. But though a rise of temperature tends to cause spreading in those cases where referred pain and tenderness are already present, it does not tend to generate tenderness in those cases where it would not otherwise appear. Thus where the lesion consists of a large cavity, the temperature is frequently raised, and even a rigor may occur; but no referred pain or superficial tenderness of the body or scalp appear.

A third difficulty that meets us is the tendency to spreading, and the consequent diminution in the localising value of the referred pain, produced by the cachexia which accompanies the disease. Any referred pain and tenderness which occurs in phthisis tends to spread, owing to the deficient resistance within the nervous system, produced by the phthisical cachexia. Thus the referred pain and superficial tenderness produced by the inflammation of the pulp of a tooth will tend to spread beyond the limits usual when that particular tooth is affected. Hypermetropia headaches

also tend to become much more troublesome, and the tenderness to spread beyond the forehead, in consequence of the patient's cachectic condition,

Case 65.—*To illustrate the tendency to spread shown by all referred pain, of whatever origin, owing to the cachexia of phthisis.* Sarah Ann T. (V.P.H., Dr. Sainsbury).

Within the last six months she has had cold after cold, and repeatadly lost her voice. She has grown much thinner, and sweated at nights throughout this period. Slight hæmoptysis a month before admission.

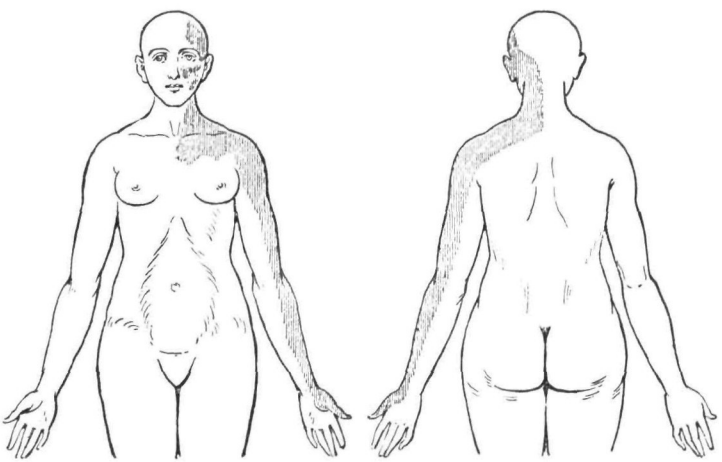

FIG. 18.

To show the wide-spread tenderness produced by disease of the pulp of the second lower molar [Case No. 65]. The great extent of this tenderness is probably due to the cachexia produced by phthisis.

I will not enter into great detail, for this case is quoted to show how any visceral pain tends to spread in phthisis whatever may be its origin.

Dulness was present over the right side behind to the level of the sixth spine. Bronchial breathing and pectoriloquy were heard over the upper part of this area, and a considerable number of râles were audible. There was a small ulcer on the left vocal cord.

She remained free from pain till five weeks after admission, when she developed pain in the second lower molar on the left side. The pulp cavity was exposed. Neuralgia came on at the same time in the face, with pain in the left ear. Then all the

teeth in the left half of the lower jaw up to the canine began to ache. The whole left side of the face, neck, and a part of the left arm began to ache. She feels as if all the power of lifting her left arm were gone, but she can use her fingers perfectly.

There is extensively widespread superficial tenderness over the left side of her face, neck, and radial side of left arm. (Fig. 18).

The temperature was 100·2° F. (37·9° C.).

The whole disappeared within four days of removing the second lower molar tooth.

Now such a widespread disturbance is not usual,[1] except where the patient is anæmic and cachectic. This case shows how the cachexia of phthisis leads to a condition in which referred pain spreads with great rapidity.

Bearing in mind these sources of fallacy, the referred pain and superficial tenderness appear on the same side of the body and scalp as the lesion in the lung.

An attempt to further differentiate the referred pain and superficial tenderness which occur in phthisis is met by a difficulty in nomenclature. To say that certain referred pain is due to an affection of the upper lobe, and pain in another position is associated with affection of the lower lobes would be simple enough. But the upper lobe of one lung is rarely affected apart from the apex of the lower lobe, and thus a statement in this form would be both unpractical and indefinite. Again, from the point of view of the advance of the disease, the lower lobe is made up of several portions. For, as Fowler[2] showed in his admirable paper, the apex of the lower lobe is easily affected in the course of tuberculous phthisis, and the disease then progresses along the upper and outer portion of the lower lobe beneath the interlobular septum. Spreading later to the other portions of the lower lobe of the lung.

Again, the affection of the lower lobe is more often the cause of referred pain than the upper lobe. For the ordinary process of destruction in the upper lobe is one of consolidation followed by excavation. But the mode of extension towards the base is not usually by an advancing line of

[1] *Vide* part ii. of this investigation, BRAIN, 1894, p. 414, and p. 361, fig. 10.

[2] *The Localisation of the Lesions of Phthisis.* Kingston Fowler. London, 1888.

consolidation, but by scattered nodules of infiltration often arranged in a racemose manner. Now this is just the condition which is likely to lead to referred pain and superficial tenderness, and thus the implication of the lower lobes is far more commonly the cause of referred pain in phthisis than that of the upper lobes.

Another difficulty that meets us at the outset is familiar to everyone who has performed many *post-mortem* examinations. The amount of the lesion discovered after death is frequently considerably in excess of the signs during life. Thus all the following attempts to show that certain portions of the lung are associated with certain segments of the central nervous system must be accepted with a liberal margin for error. Such an attempt at differentiation would be quite impossible if chronic phthisis advanced steadily down the lung. It is only rendered practicable by the fact that it is usually the small acute outbursts in the course of the chronic disease that cause referred pain and superficial tenderness, and that the signs audible during such an outburst, more nearly represent the extent of the disease than the chronic signs present between any two such outbursts of fresh activity.

Purely for the purpose of attempting to associate the extent of the pulmonary lesion in phthisis, with the areas of superficial tenderness on the chest, I have been led to divide the lung into the following parts. I do not suppose these divisions have any permanent value, they are purely rough empirical divisions, by which the commoner lines of spread may be classified for association with areas of referred pain and tenderness in the one disease, *i.e.*, phthisis. These divisions are marked out as follows :—

(1) Place the hand upon the opposite shoulder,[1] so that the angle of the scapula becomes tilted upwards and outwards. Draw a line from the fifth dorsal spine (sixth dorsal vertebra) outwards and slightly downwards to the angle of the tilted scapula. Above this line lies the upper part of the lower lobe.

(2) Let the hand fall to the side. Then draw a line from

[1] *Cf.* Fowler, *op. cit.*

the seventh dorsal spine outwards and slightly downwards following the line of the eighth rib, and passing below the angle of the scapula. Above this rough line lies the middle division of the lower lobe.

(3) From this line to the base of the lung lies the third division of the lower lobe.

The upper lobe of the lung is but rarely affected without some implication of the apex of the lower lobe. But in those few cases where the disease was very early, and the râles were mainly present in front, and in the supra-clavicular fossa, the pain and superficial tenderness lay mainly within the third and fourth cervical and third dorsal areas. Now, early phthisis is usually said to cause "pain over the lung," or "tenderness over the upper intercostal spaces." But where this pain and tenderness are present, a careful examination shows that the pain is frequently referred and not local, and that the tenderness is superficial. Thus, the tenderness frequently runs up the back of the neck, and is present over parts of the neck under which no lung tissue is situated. In the same way, the little tag of tenderness on the arm belonging to the third dorsal area can frequently be demonstrated at a distance from the lung.

As soon as acute signs become audible in the neighbourhood of the fourth and fifth dorsal spines, and the râles are found creeping round the vertebral border of the scapula when the hand is placed on the opposite shoulder, the third, fourth, and fifth dorsal areas tend to make their appearance.

When the disease attacks the middle division of the lower lobe, and the râles are found clustering about the angle of the scapula (with the arm by the side) and extend into the lower part of the axilla, the fifth, sixth and seventh, but particularly the sixth dorsal areas, make their appearance.

When the lowest division of the base of the lung becomes affected, the tenderness lies mainly over the seventh, eighth, and ninth dorsal areas, but most commonly over the seventh and eighth in phthisis.

I have entirely neglected the middle lobe of the right lung, because it is almost impossible to localise the disease

affecting it, even with the roughness that is alone possible elsewhere. Moreover, it is frequently found, *post mortem*, to have escaped, although the greater part of the right lung is affected.

The lung is, therefore, associated with the third and fourth cervical, and the third to the ninth dorsal areas inclusive. The second dorsal escapes, as a rule, except in such cases where we have reason to suspect that the tenderness has spread. Sometimes, however, it seems to make its appearance even where such spread seems improbable. The possibility of variation must always be borne in mind.

In my first paper, I did not place the supply of the lung lower than the fifth dorsal segment; for from this point downwards we reach the gastric areas, and at that stage of the research, I did not feel justified in associating any pain and tenderness accompanied by gastric disturbances with lesion in the lung. My present opinion on this matter is embodied in §4 of this chapter.

As lesions of the upper part of the lung are associated with pain and tenderness within the third and fourth cervical and upper dorsal areas, they are accompanied by superficial tenderness over the forehead and around the eyes. On the other hand, as soon as the lower lobes are considerably affected, the scalp tenderness will travel back to the temporal, vertical, or even to the parietal regions. The lower the lesion in the lung, the nearer the tenderness over the body approaches the ninth dorsal area, and the further back is the tenderness on the scalp.

To summarise the conclusions to which we have arrived in this section :—

(1) All referred pain and superficial tenderness which makes its appearance, from whatever cause, in the course of subacute phthisis tends to spread widely. This is probably due to (A) the cachexia, and (B) the temperature which accompanies the disease.

(2) Referred pain produced by the disease of the lung was most marked, and of greatest intensity on the same side of the trunk as the fresh lesion. Both pain and tenderness may, however, secondarily make their appearance on the

opposite side of the lung, but appear later, disappear earlier, and are not of the same intensity as on the side of the lesion.

(3) The innervation of the lung is connected with the third and fourth cervical segmental areas, and with all the dorsal segmental areas from the third to the ninth. The lower lobe of the lung is particularly connected with the dorsal areas, especially from the fifth dorsal downwards to the ninth.

§ 4. *Gastric Disturbances in the course of Phthisis.*

We have just seen that referred pain and superficial tenderness appear over the middle dorsal areas when the bases of the lungs become affected with disease of a certain type. But these areas, from the sixth to the ninth dorsal, stand in close relation with the stomach, especially those on the left side. Thus two organs, the bases of the lungs and the stomach, send sensory impulses into the same segments of the central nervous system. Now, when any two organs send impulses into the same segments of the central nervous system, an affection of the one which causes referred pain and superficial tenderness, tends to cause a reflex disturbance in the other organ that refers into the same areas. For instance, an inflammation of the pulp of the wisdom tooth of the lower jaw causes a soreness of the fauces and a sense of pain and swelling on the same side as the diseased tooth. Glaucoma will set those teeth aching which refer into the same areas that have become tender owing to the rise of tension in the globe. Thus, it is not surprising that the sudden appearance of referred pain and tenderness over the sixth to the ninth dorsal areas, in consequence of a fresh outburst in the lung, should be accompanied by symptoms referred to the gastro-intestinal tract.

But tubercular phthisis is in itself associated with gastric symptoms, and it thus becomes very difficult to say whether the pain, tenderness, nausea, and vomiting are, in reality due to the fresh affection of the lung, or to some intercurrent gastric attack. In some cases, however, the gastric dis-

turbance seems to be clearly secondary to the fresh outburst in the lung.

The dyspepsia which may occur in the course of tubercular phthisis can be divided clinically into three groups:—

(1) The *prodromal* dyspepsia which occurs during a period of the disease when the signs in the lungs are either absent, or exceedingly slight.

(2) The dyspepsia of *invasion*, which occurs during the gradual invasion of the lung by the tubercular disease.

(3) The dyspepsia of *dissolution*, occurring in the last stages of the disease.

Now, the dyspepsia of dissolution is very rarely accompanied by referred pain and superficial tenderness. It is mainly characterised by flatulence, eructations, and signs of dilatation of the stomach, and feeble motor and sensory activity. Thus, this form of gastric disturbance does not come within the scope of this enquiry, owing to the fact that it does not cause referred pain and superficial tenderness.

The prodromal dyspepsia, on the other hand, is accompanied by marked and widespread referred pain and superficial tenderness. But at the time it occurs the signs in the lungs are either absent, or exceedingly slight; and thus it is certainly not due to what may be called sympathetic reference. It seems to stand in relation with the cachexia which signalises the onset of tubercle of the lungs in the same way that gastralgia so frequently accompanies another general state — anæmia. I do not mean to say that this state is an anæmia, for it differs in certain striking particulars from a true anæmia, and I, therefore, prefer to speak of it as a cachexia. Now there are certain general states, which in themselves seem to be peculiarly associated with, more or less, widespread referred pain and superficial tenderness. To these general states belong the prephthisical cachexia, and certain anæmias, and I shall, therefore, postpone the full consideration of this prodromal dyspepsia, the outcome of the prephthisical cachexia, until I come to treat of these general states. I give the following case (No. 66) as an instance of this form of dyspepsia.

Case 66.—*To illustrate the referred pain and superficial tenderness sometimes associated with the prodromal dyspepsia of phthisis.*—Janet N. (V.P.H., Dr. Sainsbury).

January, 1887.—At the age of 15, she was admitted to V.P.H., complaining of loss of appetite and weakness. She had not begun to menstruate. She was then pale and thin. She vomited her food as soon as she took it, and had much pain at the epigastrium, increased by taking food. The abdominal walls were

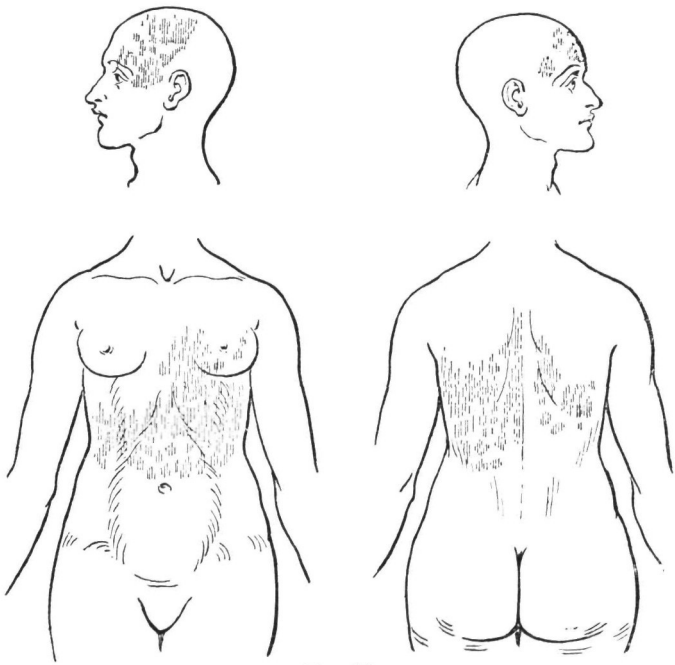

FIG. 19.

To show the superficial tenderness present during the course of the prodromal dyspepsia in Case No. 66.

hyper-æsthetic. Tongue, red and raw looking. Teeth, good. A systolic (hæmic) murmur was heard over the upper part of the sternum; the pulmonary second sound was increased. The temperature was normal throughout.

She left the hospital much improved, but throughout the next four years had several similar attacks.

January, 1891.—At the age of 19 she was again admitted. She had now developed a slight hacking cough, which was only followed by expectoration in the early morning. The sputum contained scarcely any purulent material, and Dr. Hadley was

unable to find any tubercle bacilli. She had been losing flesh for three months, and sweated slightly at night. No definite abnormal signs could be found in the lungs. A systolic hæmic murmur was heard over the second left interspace, and a hum was audible in the veins of the neck. The teeth were still good. Tongue, red. Appetite, bad. Suffered a good deal from flatulence.

February 10.—The tonsils swelled, and the lymphatic glands in the neck became enlarged. The gland in the neck remained enlarged for a considerable time.

She much improved, but in July, 1893, began to feel tired and faint, and to suffer from nausea, whether she took food or not. Vomiting now returned, and she again began to suffer from a slight cough.

October 24, 1893.—She was again admitted to V.P.H. She was now 21, but looked older. The face was pale, of a somewhat opaque white, with a slight flush on each cheek. The ankles swell at the end of the day, but there is no œdema when she is kept in bed. She sweats slightly at night. She was fairly well nourished, and showed no signs of wasting.

She complained of palpitation and shortness of breath. No objective dyspnœa when in bed. Slight single cough, especially in the morning. Expectoration scanty, consisting of a few muco-purulent lumps, in which no tubercle bacilli could be found. She suffers from pain in the epigastrium and back, accompanied by considerable superficial tenderness as in fig. 19. She complains mainly of bitemporal headache, and this headache is accompanied by considerable superficial tenderness of the scalp.

The only abnormal physical signs in the chest were slight diminution of movement over the left apex, with very slight diminution of the percussion note. A few crackling sounds were heard in the upper two spaces on the left side in front. Pulse, 72; low tension. Hæmaglobin, 50 per cent. Red corpuscles, 60 per cent. (3,000,000 in a cub. mm.). No hæmic murmur could now be heard in the chest.

The teeth have decayed very rapidly. The upper central and lateral incisors have gone, and are replaced by a well-made plate. The left second bicuspid, first molar, and right second molar, in upper jaw, exist only as stumps. In addition, many teeth in both jaws show signs of caries.

Tongue, cracked transversely, dry in the centre, and red, but not furred. Appetite, bad. Feels sick at varying times after food. She cannot take fat in any form. Vomiting occasionally. Some flatulence. Bowels opened daily; not constipated.

Liver and spleen cannot be felt.

Menstruation began at 17. Irregular, at intervals of one to two months. She menstruated during her stay in hospital, and suffered from considerable pain and widespread superficial tenderness, which gradually disappeared when the menstruation ceased.

The pain generalised two days before the flow came on. The flow lasted two and a-half days, and the amount was scanty.

The temperature was very irregular. It was invariably raised in the evening, but whereas it sometimes only reached 99° F. (37·2° C.), it occasionally ran up to 100·6° F. (38·1° C.), falling again in the morning to 98° F. (36·7° C.).

During a stay of eight weeks in the hospital she gained 7 lbs. in weight, and much improved.

January, 1894.—The cough returned, and she again became weak and languid, but without vomiting. She had been under continuous treatment with iron, and the hæmaglobin is now 100 per cent., and the red corpuscles 97 per cent. (4,860,000). The movement of the left apex is still somewhat deficient, and a few crackling râles have reappeared both in back and in front on the left side.

August, 1894.—She married. When she became pregnant the vomiting and pain came on again, and she was in bed four months.

August, 1895.—The child was born, and from that time the vomiting and gastric symptoms have much improved, but she began to suffer from extreme weakness. She began to sweat at night again.

February, 1896.—She is obviously thin, and has lost a stone in weight. Sweats profusely at night.

Tongue red and moist. Appetite was very good, but she cannot bear fat of any kind. No pain after food. No headache. No nausea. No vomiting. She now takes large quantities of food, and after meals bitter stuff rises into the mouth, but does not cause her any discomfort. A good deal of flatus at all times.

Coughs, especially in the morning. Expectoration, thick and yellow.

Deficiency of movement at left apex, back and front. Note at left apex not good, but note at right apex, back and front, is considerably diminished. Over upper two spaces, on right side in front, breath sounds are weak, and vocal resonance is increased. Over upper part of right lung behind, breathing is bronchial, vocal resonance is increased, and fine crackling râles can be heard as low as the level of the fifth spine.

Thus the signs of phthisis have now become evident, and the whole character of the clinical picture has altered.

There now remains for consideration, a group of cases of phthisis in which referred pain and tenderness make their appearance over the sixth to the ninth dorsal areas, during the course of the invasion of the lung by the disease. The difficulty in such cases lies in tracking the referred pain and tenderness to its fundamental cause, and, in many

cases, it is practically impossible to say whether the lung or the stomach is the organ that gives birth to the referred pain. However, I think, some help in this difficult task can be gained by dividing this *clinical* group into three *physiological* subdivisions. These three subdivisions of the dyspepsia of invasion I will now consider seriatim.

(*A*) *Pseudo-Gastric Disturbance.*

The patient's general condition may be good, the tongue clean, appetite fair, bowels regular, and referred pain may be absent, superficial tenderness may be absent or only slightly marked, or may, perhaps, be situated entirely over the upper areas of the chest and the front of the scalp. All tenderness over the sixth to the ninth dorsal areas, on the body, and from the fronto-temporal area backwards on the scalp are absent.

Then a slight recrudescence of activity takes place in the lung disease. Fine dry râles, perhaps accompanied by sibilant rhonchus, appear behind over that part of the lung which lies at the level of the seventh dorsal spine. Referred pain accompanied by more or less marked superficial tenderness, appears in one or more of the areas from the sixth to the ninth dorsal, accompanied by scalp tenderness over the temporal, vertical, or parietal areas. The outburst of fresh activity in the lung will probably be accompanied by some rise of temperature, and thus the pain and superficial tenderness, though more marked on the side of the affected lung, will tend to double at the same level of the central nervous system; this is peculiarly liable to happen on the scalp.

But the disturbance of any organ, whether of the head or trunk, which causes well-marked bilateral referred pain and superficial tenderness of the temporal region is associated with nausea, loss of appetite, and possibly, vomiting. Thus, this nausea, and even vomiting, can be excited by affections of the eye, although there is no tenderness over the gastric areas of the trunk.

Thus, when a slight recrudescence of activity occurs in that part of the lung which lies below the angle of the

scapula, the patient may complain, not only of referred pain and tenderness over the gastric areas of the trunk and temporal region of the scalp, but also of loss of appetite and nausea; later, actual vomiting may occur. The tongue is at first quite unaltered, the bowels remain regular, and the evacuations are unchanged in character, the pain is not increased by food, and vomiting does not necessarily relieve the pain and headache.

On the other hand, anything which relieves the pain and headache, will remove the nausea and vomiting. Thus, mustard leaves applied to the maximum spots of the affected areas of the chest or back, or a dose of phenacetin (which acts more quickly on the headache, but also on the referred pain of the trunk) will remove the nausea and vomiting in this mild and purely reflex type of gastric disturbance.

Such a condition rarely lasts long, for, if it does not pass off within forty-eight hours, it tends to merge into the second form next to be considered.

I might have selected a case of ordinary chronic phthisis as my example of this form of gastric disturbance, I have, however, chosen case No. 67, on account of the shortness of the attack of pain and headache, and the complete return of appetite, with the cessation of the nausea and vomiting.

Case 67.—*To illustrate the first, or reflex type, of gastric disturbance which occurs in the course of phthisis.* Ada W. (V.P.H., Dr. Harris), aged 17. Admitted, August 17, 1894.

When a child she had a fever, about which no details can be obtained. Since then she has always been under a doctor for her chest. Her cough has become worse during the last five years.

She is a small girl of 17, but looks much older. She is ill-developed, and the fingers are markedly clubbed. There is no wasting, the body is well covered, and the breasts well formed. She does not sweat at night. There is a marked lateral curvature with the concavity towards the left in the mid-dorsal region.

Respiration 24. No objective or subjective dyspnœa, except when she exerts herself. The dyspnœa then becomes marked. Sometimes she does not cough throughout the day. Then, on first waking, she has a paroxysm of coughing, in which she retches, bringing up a large quantity of greenish-yellow masses of stale smelling muco-pus. Many most careful examinations failed to show any tubercle bacilli.

She is usually free from pain and superficial tenderness.

The whole left side is somewhat retracted, and does not move with respiration. Back and front this side is dull. Over the upper part of the left lung the breath sounds are weak, expiration prolonged, and vocal resonance diminished, but below the level of the sixth spine the breath sounds are almost tubular, and bronchophony is audible. These signs extend to the extreme base. From top to bottom on the left side large moist consonating râles are heard. Thus the disease on the left side seems to be old, and the signs did not alter appreciably in three months.

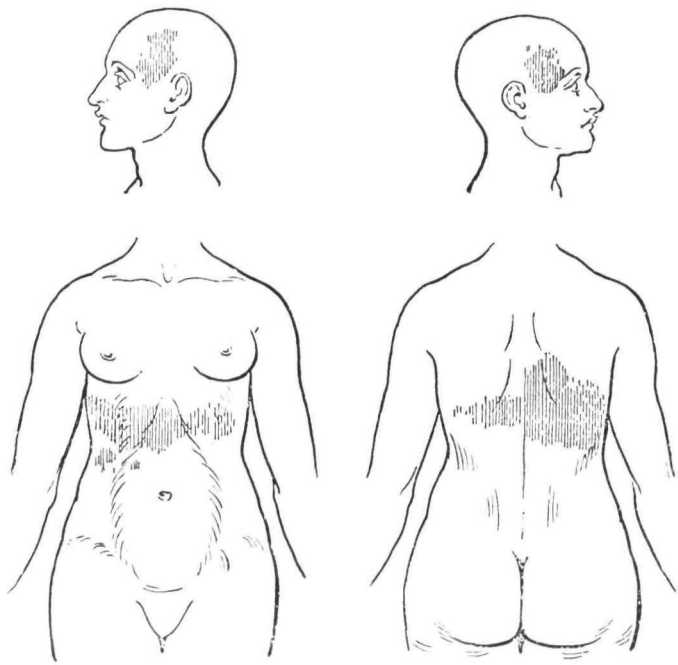

FIG. 20.

To show the areas of tenderness present during an attack of reflex gastric disturbance in Case No. 67.

The right side of the chest is free from abnormal signs, except for a few very large moist râles at the right base.

The pulse was 100, very small, regular, and somewhat hard.

The apex beat seems to be drawn up into the third space on the left side. Heart sounds are normal.

Tongue normal and moist. Appetite good. No pain, nausea, or vomiting (except with the paroxysms of coughing). Bowels normal.

Urine 1015, acid, and containing a considerable quantity of albumen.

She continued in this condition until September 18, when she began to complain of pain under the right shoulder, and in the epigastrium. Some pain on left side, but to a less degree. Bi-temporal headache. This pain was associated with marked superficial tenderness, as in fig. 20, both of body and scalp.

She felt very sick, and vomited twice. The appetite was not good, but she could take her food, and the food caused no pain. The pain was always present whether she took food or not. The tongue was clean and moist. The motions were unaltered.

The cough was not increased, and the sputum still had the stale smell, but was not truly offensive.

At the right base the breath sounds are bronchial, the vocal resonance increased, and a large number of râles have made their appearance below the angle of the right scapula.

The temperature which usually only touched 99° F. (37·4 C.), ran up to 101° F. (38·3° C.)

September 19.—By the evening the pain, tenderness, nausea, and vomiting had disappeared. She took her tea with a good appetite. The temperature did not rise above 99·4° F. (37·5° C.)

She had several similar slight attacks, in the course of the three months she was under observation, apparently due to an infection of the comparatively healthy side by the pus from the dilated bronchi of the left lung, which was in a fibroid condition.

(B)—Sympathetic Hyper-æsthesia of the Stomach.

If the referred pain and tenderness over the gastric areas, produced by the condition of the lung, continues for some length of time, or is of marked intensity from the beginning, the stomach seems to become hyper-æsthetic by sympathy. For not only do nausea and occasional vomiting make their appearance but the pain is distinctly increased by the ingestion of food, and is partly relieved by vomiting. Such patients complain that pain is always more or less present, but that it is markedly increased after taking food and, if vomiting occurs, the pain is relieved, and sinks back to the amount that was present before the food was taken. In this connection it is interesting to notice that if the sixth and seventh dorsal areas are mainly affected, the pain will be increased almost immediately the food is taken into the stomach, and the patient may vomit practically unaltered food within twenty minutes of its ingestion. If, however,

the ninth dorsal area be mainly affected and the upper gastric areas are either absent, or but slightly developed, the pain will not be increased until a considerable time after the food has been taken, and vomiting may be completely absent.

The tongue remains unaltered, as a rule, in this form of gastric disturbance. If it were clean and red, or a little furred, soft and moist it does not alter, at any rate, at first, although the gastric symptoms are very marked. In the same way the action of the bowels and the character of the evacuations are not markedly altered, excepting in consequence of the rejection of food by vomiting, or some alteration in diet prescribed by the physician. Flatulence and the eructation of fluid into the mouth, are not usually symptoms of this condition.

Thus the whole of the symptoms are those of irritability of the stomach without obvious signs of gastric mischief. As soon as the outburst of fresh disease in the base of the lung clears up, or ceases to advance, the referred pain and superficial tenderness over the gastric areas ceases, or diminishes, and with this cessation or diminution of the pain and tenderness the gastric symptoms disappear. The explanation would seem to be that the outbreak in the lung pours pain impulses into those segments of the nervous system which stand in nervous connection with the affected part of the lung. But the stomach also stands in connection with the same segments, and thus becomes hyperæsthetic, in exactly the same way as the testicle may become hyper-æsthetic owing to a renal calculus.[1]

The first case I have chosen as an illustration shows the outburst of referred pain and tenderness, with the implication of the left base. The base of the lung was permanently damaged, but the referred pain and tenderness disappeared when the acute increase in the disease passed away.

The second case shows the disappearance of the gastric symptoms, with the clearing up of an acute outburst at the left base, and their reappearance when the base became again affected.

[1] *Cf.* BRAIN, 1893, p. 78.

Case 68.—*To illustrate the second type of gastric disturbance associated with the invasion stage of phthisis.* (*Sympathetic gastric hyper-æsthesia*). Edward O. (V.P.H., Dr. Thorowgood); aged 34, wire layer for electric light.

In the winter of 1890 had a bad cough and spat much phlegm. Got better, but continued to cough all through the summer. In the winter of 1891 got very bad again, and could not work. He attended Brompton Hospital, and much improved, but in October, 1892, became worse again. Wasted greatly, sweated at night; cough very troublesome. He again improved somewhat, but in October, 1893, got worse than ever.

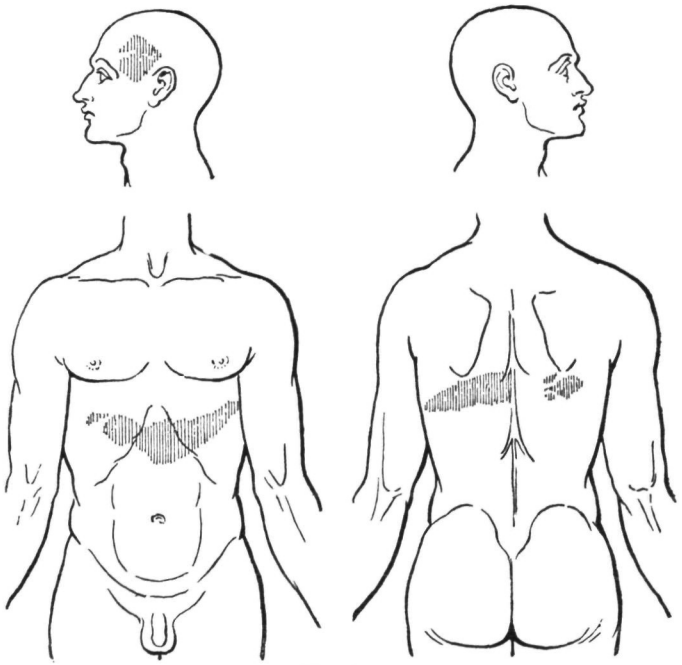

FIG. 21.

To show the areas of tenderness present in Case No. 68 during an attack of sympathetic gastric hyper-æsthesia.

On admission (April 9, 1894).—Small man of 34. Face, cachectic, of a somewhat greasy pallor. Slight pink congestive flush on the cheeks. No cyanosis. Considerably wasted.

Respiration 22. Accessory muscles of respiration acting slightly. Very short of breath, on exertion. Cough troublesome in the morning, but not troublesome in the daytime. Large amount of greenish-yellow, round masses of muco-purulent expectoration containing tubercle bacilli.

He complains of pain in the epigastrium, over the left costal margin, and below the angle of the left scapula. This pain is worse on the left side, than the right. Beautifully marked superficial tenderness. (Fig. 21). Headache over left temple accompanied by well-marked, superficial tenderness.

In Front: movement poor over both apices. Percussion note diminished over the upper three spaces on the left side. Over the left apex the breathing is bronchial, and bronchophony is heard. Over right apex breath sounds are weak, expiration prolonged, and vocal resonance diminished. Many crackling râles all over the front of the chest.

Behind: on the right side the note is dull, to the level of the angle of the scapula; over this area expiration is prolonged, vocal resonance increased and many rhonchi and râles are heard. From the angle of the right scapula to the base the breath sounds exceedingly weak, vocal resonance is much diminished and irregular, crackling sounds are heard. On the left side the percussion note is diminished to the level of the fifth spine. Over this area expiration is prolonged, vocal resonance increased, and many crackling râles and much inspiratory rhonchus is audible. From the level of the fifth spine downwards the breath sounds are harsh, expiration a little prolonged, but vocal resonance is unaltered. Many sharp râles and inspiratory rhonchi are heard over the lower part of the left lung.

Thus the signs in the lungs pointed to an old affection of the right base, and a recent affection of the left base.

Pulse 88, regular. Heart sounds normal.

Appetite very bad. He complains that the pain is increased by food. Feels sick, and retches within half-an-hour of taking food. Has vomited several times with this retching, apart from coughing. The vomit consists of almost unaltered food. Tongue small, moist, a little tooth indented. Bowels opened daily without drugs. Motions normal.

Liver and spleen not enlarged. Urine normal. Temperature rose every night to 100° F. (37·8° C.) falling in the morning to normal.

By April 20 all pain, headache and superficial tenderness had disappeared. He no longer felt sick after food. Food caused no pain. Appetite good.

The signs in the lungs on the right side remain the same. Over the upper part of the left lung behind, to the level of the fifth spine, the dulness is unaltered, and breath sounds and adventitious signs remain as before. Below this point all over the left base the breath sounds are good, vocal resonance normal, and absolutely no râles or rhonchi can be heard, or have been audible for several days.

On May 9. An exactly similar attack of nausea, vomiting, pain after food began, associated with another outburst at the left base. He left the hospital with persistent signs below the angle of the left scapula.

Case 69.—George R. (V.P.H., Dr. Sainsbury); aged 39, cellarman.

He was quite well till 1891, when he had an attack which was called "Influenza." He remained in bed for two weeks, and for the next three weeks was troubled with cough and spitting. July, 1892, began to suffer with pain in the left side and cough returned. January, 1893, had another attack of "Influenza" with pain in the left shoulder. He coughed, sweated at night, and lost much flesh. The cough has continued ever since. April, 1893, began to spit blood and the expectoration continued to be blood-stained for two weeks.

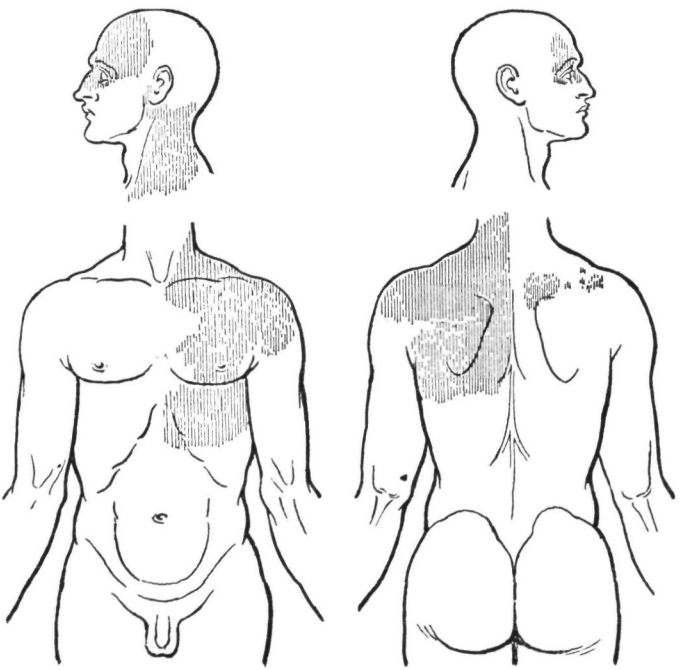

FIG. 22.

To show the areas of superficial tenderness in Case No. 69, produced by the invasion of the base of the left lung. This tenderness was associated with marked gastric disturbance.

Owing to the nature of his employment he takes alcohol somewhat in excess; five or six pints of beer daily.

On admission (October 16, 1893). Large well-built man, evidently somewhat wasted. Says he has lost 14 lbs. in six months. Face of a yellowish pallor. No flush in cheeks. No cyanosis. No profound anæmia.

Respiration 20. Accessory muscles quiet. Dyspnœa on exertion. Hard hacking cough. Spits up a considerable quantity of small muco-purulent yellowish masses tinged with bright or dark red blood.

Some pain in left supra-clavicular fossa, and over the posterior aspect of the left shoulder joint. Distinct superficial tenderness over the spots to which the pain is referred.

In front: right apex moves somewhat better than the left. Percussion note diminished in first two spaces on left side. Expiration is prolonged and vocal resonance increased as low as third space on left side. Fine dry râles heard over the same area. Over the right apex breath sounds are fair, vocal resonance a little increased, and a few scattered râles are heard.

Behind: on the left side the dulness extends to the level of the sixth spine. Expiration is prolonged and vocal resonance is increased over the area of diminished note. Crepitations are heard from the left apex to the level of the sixth spine, and when the left arm is placed on the right shoulder these crepitations are found to extend along the vertebral border of the left scapula. At the extreme right apex the percussion note is diminished, expiration prolonged, and a few crepitations are heard.

Pulse 80 regular. Heart sounds good. No murmur.

Tongue moist, a little flabby. Appetite excellent. Bowels opened daily. No vomiting or pain after food.

The temperature did not rise above 99° F. (37·2° C.).

November 17.—He complained of marked increase in intensity and extent of the pain on the left side of the chest. Wide-spread superficial tenderness. Headache and scalp tenderness.

The cough is more troublesome, expectoration no longer contains blood.

The right side of the chest remains as before, but there is a marked outburst of râles below the angle of the left scapula and in the left axilla.

Has lost his appetite. Feels sick after food. No diarrhœa.

November 26. Much pain on left side of the chest with superficial tenderness over the areas in the figure. Headache more marked on left side than right, and accompanied by marked scalp tenderness. (Fig. 22).

His appetite has completely gone. He feels sick and has vomited after food. The tongue remains clean. Bowels opened daily. Motions show no marked alteration.

The crepitations over the left base and in the left axilla have considerably increased since November 17.

Temperature reached 99·8° F. (37·7° C.) every night.

By December 20 all referred pain, superficial tenderness and headache had disappeared. He was taking his food well, and the nausea and vomiting were absent.

The left side of the chest behind was dull from the apex to the base. Over the upper part of the left lung behind expiration was prolonged and vocal resonance much increased, but below the

angle of the left scapula the breath sounds were very feeble, and vocal resonance was diminished. A few scattered râles were heard over the left base No active signs in right lung. Heart's apex beat was normal.

Thus, the dyspeptic symptoms in this case came on with the implication of the left base, and disappeared when the base of the left lung became permanently affected.

(*C*) *True Gastric Dyspepsia.*

In the third type of gastric disturbance met with during the course of the invasion of the lung by phthisis, the referred pain and superficial tenderness bear no relation to the condition of the physical signs in the lungs. This is a true dyspepsia of gastric origin. The pain and tenderness extend over the sixth, seventh, eighth and ninth dorsal areas to a greater or less extent, but there is a marked tendency to the simultaneous development of referred pain and tenderness within the fourth cervical area. Now, this association of tenderness over the gastric areas of the trunk, with the areas about the clavicle and shoulder joint, is a common feature of true gastric disorders.

Pain after food with occasional vomiting, especially in the morning (apart from coughing), flatus, and the regurgitation of fluid into the mouth, are symptoms of this form of dyspepsia. The tongue is usually more or less furred, frequently with a red tip, sometimes it is soft and tooth indented, the bowels are always disturbed, constipation alternating with attacks of diarrhœa, the evacuations are frequently made up of small hard lumps of dark fæces with some leathery curds and darkish brown yellow fluid, and are distinctly offensive.

This form of dyspepsia is more directly amenable to treatment directed towards the gastro-intestinal tract, and a considerable number of the cases are improved by the ordinary rhubarb and soda mixture accompanied by the occasional use of cascara sagrada.

Thus, the distribution of the referred pain and tenderness, the condition of the tongue and evacuations, the flatus and regurgitation of fluid into the mouth, and the results of

treatment, all point to some disturbance of the stomach itself as the cause of this type of dyspepsia in phthisis.

Case 70.—*Dyspepsia of gastric origin, accompanied by referred pain and superficial tenderness, arising in the course of a case of phthisis.* Bernard McD. (V.P.H., Dr. Harris); aged 21, cook.

In May, 1892, he was serving in the Marine Artillery, when he broke his ankle. For this he was invalided, and he spent the

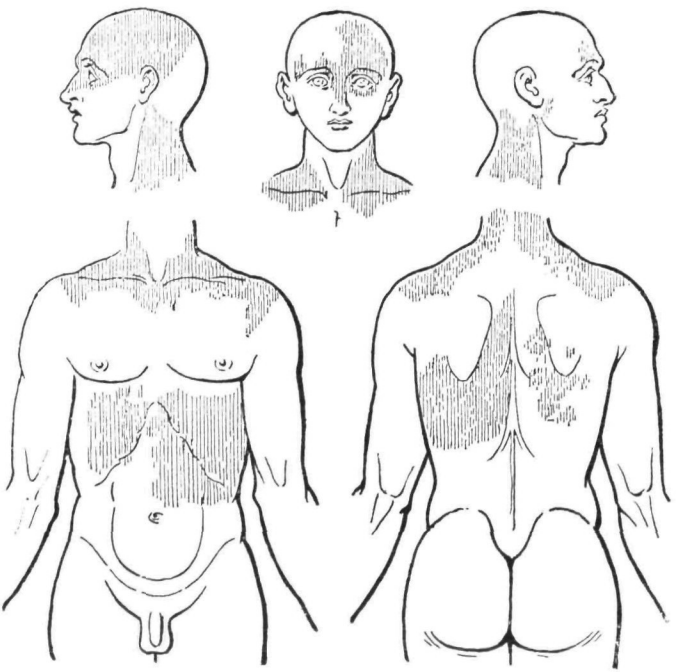

FIG. 23.

To show the areas of superficial tenderness accompanying an attack of dyspepsia, of true gastric origin, arising in the course of a case of phthisis [No. 70]

next five months nursing a friend, who died of "consumption of the lungs and throat." He then began to feel languid and faint, and "caught cold after cold."

August, 1893.—Cough came on badly; he began to sweat at night, and lost 7 lbs (3·4 kgm.) in weight.

February, 1894.—Hæmoptysis. Continued to bring up blood in small quantities for weeks.

Family History.—He has been entirely separated from his family for four years. One sister (who is in a convent) has had repeated hæmorrhage "from the lungs," and is dying of "consumption."

Admitted to V.P.H., July, 1894.

He suffered much from pain, vomiting, and alternating constipation and diarrhœa; coming on in attacks at varying intervals. The condition on September 7 was typical of several attacks which were watched during his stay in hospital.

September 7, 1894.—He is a well-built man of 5 feet 10 inches. Face thin; and there is much wasting about the neck. He has lost 3 stone (over 20 kgm.) in twenty months. No marked sweating at night. Face pale, and of a uniform earthy white. No flush. Lips pale, but distinctly blue. Temperature ranges to 101° F. (38·3° C.) at night, but sinks to 98° F. (36·7° C.) in the morning.

He has been somewhat freer of gastric disturbances until today. Tongue somewhat blue-red, moist; over the posterior part of the dorsum there is a triangular area of brown fur. Just before dinner he has a feeling of hunger and emptiness. Then he feels sick, and occasionally vomits. An hour after food, this same sinking feeling comes on again. He is passing green, very offensive, motions, three or four times in the twenty-four hours.

He complains of much pain, particularly in the left side, both back and front, from about the level of the sixth rib to the level of the umbilicus. It is most marked in the epigastrium, and behind the angle of the scapula. He also has pain in the posterior aspect of both shoulder joints, running up the back of the neck, and causing a feeling as if his neck was stiff. The pain is sharp, and leaves behind it a kind of "benumbed, painful feeling." Then, when he moves, the sharp pain comes on again within the "benumbed areas."

Headache over the forehead and temples. Hair is sore when brushed.

Marked superficial tenderness of body and scalp, as in fig. 23.

This pain is always more or less present now. It is worse before food; is somewhat relieved by food; and is worse again about an hour after a meal. Some flatulence; and the pain is easier when he brings up the wind.

Respiration 20. Sterno-mastoids acting. Very short of breath on exertion. Cough in occasional paroxysms; especially in the morning, when the cough causes vomiting. Expectoration in irregular, greenish muco-purulent masses, but not very profuse. (It has been tinged with blood since admission).

The left side of the chest is now dull from top to bottom, both back and front. Expiration is prolonged, and vocal resonance increased all down the left side, and in left axilla; and many crackling râles are heard. (The left base became affected on August 18, and has remained in the same condition ever since).

Over the right side of the chest the note is impaired as low as

the fourth rib in front, and the fifth spine behind. Over this dull area the breath sounds are bronchial. Vocal resonance is increased, and numerous fine crackling râles are heard. (The right side only was affected on admission in July.) The right base around and behind the angle of the scapula, and the lower part of the right axilla, are clear.

Palpitation and feeling of faintness on exertion. Pulse 88; small, regular. No abnormal sounds in the heart.

Urine, specific gravity 1022, acid, and with some albumen.

He died at the beginning of November, 1894.

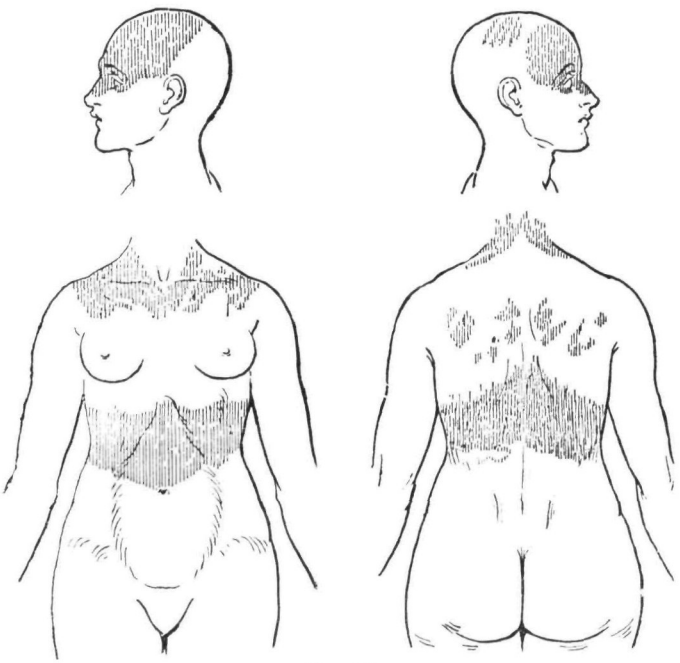

FIG. 24.

To show the areas of superficial tenderness accompanying an attack of dyspepsia of true gastric origin, arising in the course of a case of phthisis [No. 71].

Case 71.—*Dyspepsia of gastric origin, accompanied by referred pain and superficial tenderness, arising in the course of a case of phthisis.*—Minnie E. (V.P.H., Dr. Harris); aged 23, waistcoat maker.

In the winter of 1888-89 she began to have a dry, harsh cough, which passed off with the coming of the spring.

October, 1889.—She began to suffer from pain after food, and much vomiting. With this attack a hard cough came on again.

She was admitted to a general hospital, and there told she had a gastric ulcer. (Her abdomen still shows the scars of the frequent blisters that were then applied to the epigastrium.) She was well in ten weeks.

June, 1890.—The cough came on again, with nausea and pains in the limbs. Said to be influenza. In five weeks she was better, but very weak. August, 1890, a second similar attack, from which she did not recover for a long time.

Every winter the cough reappeared, but cleared off with the summer, until 1893. The cough then became constant.

September, 1893.—She began to waste rapidly, and to sweat profusely at night. The cough became much looser.

February, 1894. *On admission.*—Very marked purple flush upon the cheeks. Marked wasting. Sweats at night.

Respiration 35 to 40. Sterno-mastoids acting vigorously. Much subjective dyspnœa. Single troublesome cough. Brings up a large quantity of stringy aerated mucus.

Much pain at epigastrium and under shoulders, especially on the left side. Marked superficial tenderness over the sixth and seventh dorsal areas, with spotty tenderness over the fourth cervical area, on the left side. Widespread fronto-temporal headache, with superficial tenderness over the frontal mid-orbital, fronto-temporal, and temporal areas of the scalp.

Right apex moves better than the left. Percussion note diminished over left front. Over the upper two spaces on left side, in front, breath sounds are cavernous; bronchophony is audible, and many crackling râles are heard as low as the fifth intercostal space. Over the first two spaces on the right side the breath sounds are bronchial, and bronchophony is present, and numerous crackling râles are heard.

Behind: on left side, diminished note as low as level of fourth spine; breath sounds cavernous, with whispered bronchophony. Crackling râles from apex to around angle of scapula, and in the axilla. Over the right side: note diminished, as low as sixth spine. Over upper lobe: bronchial breathing and bronchophony; but opposite the level of third and fourth spines, an area of profound cavernous breathing and whispered pectoriloquy.

Pulse 100; small, regular. Apex beat, fifth space, internal to nipple line. Cardiac dulness slightly diminished. No abnormal sounds.

Tongue somewhat dark red, and not well moistened. Appetite poor. Bowels opened two to three times daily.

Liver and spleen not felt. Nothing abnormal felt in the abdomen.

Temperature sometimes touched 103° F. (39·4° C.) at night, and did not fall below 100° in the morning.

The signs on the left side became quieter, but the right side progressed rapidly. The pain and superficial tenderness became more marked on the right side.

At the end of March the gastric symptoms became a much

more prominent feature, and the superficial tenderness was very marked.

April 30.—She is vomiting green watery fluid in the morning, apart from cough, and before breakfast. The tongue is dry and red, with some irregular whitish fur, but not heavily coated. Appetite is very bad. She has been constipated, but the motions are now three or four in the twenty-four hours. The evacuations consist of some irregular brownish lumps, with much brown offensive liquid material.

She is complaining of much pain in the epigastrium and under the shoulder blades. She also has pain over the neck on both sides and over the shoulder joints. Very marked superficial tenderness, as in fig. 24. Headache over the temples and forehead, with superficial tenderness, as in fig. 24.

Liver and spleen not enlarged. Nothing abnormal felt in the abdomen. No marked signs of dilatation of the stomach.

The signs in the lungs have advanced rapidly. *In front*, there is dulness, with cavernous breathing and whispered pectoriloquy, as low as the fourth rib on the left side. On the right side, hollow breath sounds and whispered bronchophony, as low as third rib. *Behind:* on left side there is dulness to level of sixth spine, with cavernous breathing and whispered pectoriloquy to level of third spine. On the right side, dulness, with cavernous breathing and whispered pectoriloquy, as low as angle of left scapula.

The temperature does not vary more than about a degree, and the mean temperature is 101° F. (38·3° C.).

This case illustrates the true gastric disturbance of phthisis, and shows the association of the cervical areas of tenderness with those of the epigastrium.

It is frequently very difficult, in practice, to decide in which of these three groups to class some particular gastric disturbance that occurs during the course of a case of chronic phthisis. From a clinical point of view, such a division into three forms of dyspepsia would be scarcely necessary. But, from the physiological aspect, such a division is absolutely essential; for the three types are the expression of, at any rate, two fundamentally different processes.

Moreover, if we remember that two of these forms of dyspepsia are produced reflexly, whilst one is of gastro-intestinal origin, we can understand the following facts, upon which most observers are agreed. It would seem that in spite of the notorious proneness of phthisical patients in this stage of the disease to suffer from gastric disturbance,

in the large majority of cases no abnormal activity, either of the mucous membrane or of the muscular walls of the stomach, can be discovered. On the other hand, certain cases show distinct secretory and motor abnormalities.

Again, it helps us to understand why, in a large proportion of the cases, the dyspepsia tends to disappear, or to alter in type, as the destruction of the lung progresses. For the two purely nervous forms, due to the destruction of the bases of the lungs, must cease when the changes in the lung assume a form incapable of generating referred pain. Thus the "reflex dyspepsia" is a very prominent feature of the invasion stage of the disease, when organic changes in the walls and coats of the stomach are uncommon; whilst it may be entirely absent in the third stage, when alterations in the walls of the stomach are of much more frequent occurrence.

This assumption of a reflex origin for many of the dyspepsias, which appear during the invasion stage of phthisis, helps us to understand why gastric symptoms are not a marked feature of pneumonia, pleurisy, or of those cases of phthisis where the destruction of the lung is mainly by cavitation. For complete consolidation prevents the appearance of referred pain, by throwing the nerve endings of the diseased portion of the lung out of play. Cavitation destroys the nerve endings in the lung, and is, therefore, unaccompanied by referred pain. Pleurisy causes a local pain, in which the splanchnic system of afferent nerves is not implicated. Thus we should no more expect reflex dyspeptic symptoms to accompany a pure pleurisy than we should expect them to accompany an inflammation of the skin of the chest wall of similar extent.

To sum up the results to which we have arrived in this section:—

The gastric disturbances, which occur in the course of phthisis, may be divided, *clinically*, into three groups:—(1) The prodromal dyspepsia, appearing before there are any marked signs in the lungs. This dyspepsia is frequently accompanied by marked referred pain and superficial tenderness. It seems to be associated with the cachexia which not infrequently heralds the onset of the disease.

(2) The dyspepsia of invasion, also accompanied by referred pain and superficial tenderness.

(3) The dyspepsia of dissolution, accompanied by marked signs of organic change in the walls and mucous membrane of the stomach, but unaccompanied by referred pain and superficial tenderness.

Physiologically, the second of these clinical divisions can be again divided up into three types, according to the causes which give rise to it :—

(*a*) A pseudo-gastric disturbance, due to the nausea and vomiting produced by the referred temporal headache, which stands in definite relation to the referred pain produced by the implication of the base of the lung.

(*b*) Gastric hyper-æsthesia, which owes its origin to the fact that the bases of the lungs and the stomach are supplied from the same segments of the central nervous system. Thus an affection of the base of the lung causes a disturbance within the sixth to the ninth dorsal segments, and the stomach becomes hyper-æsthetic by sympathy, just as the testicle becomes tender in some cases of stone in the kidney.

(*c*) True dyspepsia of gastro-intestinal origin, having no relation to the nature of the phthisical process, or the distribution of the disease within the lung.

In conclusion, I have to offer my sincere thanks to the staff of the Victoria Park Hospital for Diseases of the Chest, for the extremely liberal manner in which they have permitted me to make use of their cases, not only during the time I acted as house physician, but also during the last three years.

My thanks are also due to Mr. Lunn for permitting me to make use of some of the cases under his care at the St. Marylebone Infirmary.