7. These preliminary observations showed that throat carriers of haemolytic streptococci are extremely common, and that the organism in that locality is very susceptible to treatment and other hazards of its existence. This last is proved by a later observation, which showed that fifteen nurses reported positive in November were negative in January without any local treatment being used, while six nurses reported negative in October were positive in January.

The known prevalence of the organism, and the rarity and trivial nature of the temperatures which occurred among the clean cases following the established precautions regarding contagion, made it clear that a conclusive test must be made which would leave no vestige of doubt regarding the reputed danger of throat carriers to parturient women.

There were two weak links in the chain of evidence to date: (1) positive carriers had been submitted to an antiseptic throat routine; and (2) only those who delivered the patients had been examined—not the dressers.

To make the test conclusive it was necessary: (1) That all the nursing staff should be examined. (2) That all antiseptic treatment of the throat should be discontinued. (3) That only clean cases, Groups A and B, should be investigated. (4) That no masks should be worn.

The test lasted from November 1st to December 31st. The entire staff—doctors, students, and nurses—were examined. Of the 50 persons from whom swabs were taken, 34 (68 per cent.) were positive.

A record was kept of the work done by each person reported to be positive. This was easy as regards deliveries, but it was a little difficult to keep a complete list of all dressings done by the nurses. The officially recorded number of patients dressed by "carriers" is certainly an underestimate.

Clean cases were delivered or dressed by "carriers" as follows:

Deliveries.—Hospital: total deliveries 183; by carriers 102. District: total deliveries 99; by carriers 47. The district sister-midwife (positive) assisted at nineteen of these deliveries, while the district medical officer (positive) delivered with forceps, or repaired lacerations, on twenty-six occasions.

Dressings.—Hospital and district 478. This number represents individual patients dressed by carriers, but as only 282 patients are under review, it will be clear that a number of patients were dressed by more than one carrier.

Results—Indoor.

Chart 4 shows that the incidence of sepsis in hospital during November and December was the same as during the previous four months. The detailed statistics are as follows:

Group A cases:				Sepsis.
September–October, 1929	...	...	...	1.1 %
November–December "	...	...	...	2.1 %
Group B cases:				
September–October, 1929	...	...	...	2.8 %
November–December "	...	...	...	nil
Combined Groups A and B:				
September–October, 1929	...	...	...	1.4 %
November–December "	...	...	...	1.6 %

The three cases of sepsis among the 183 clean cases delivered in hospital were analysed. All three were trifling, and the temperatures were notifiable on 1, 1, and 2 days respectively.

Case 1.—One day's pyrexia. Delivered by a positive and dressed by negative.

Case 2.—One day's pyrexia. Delivered by a negative and dressed by two positives.

Case 3.—Two days' pyrexia. Delivered by a negative; dressed by two negatives.

Throat and vaginal cultures were taken from the patients in Cases 1 and 3. It is significant that all were negative except the vaginal culture in Case 3, which was positive.

The total district deliveries for September–October, 1929, numbered 114; sepsis 0.8 per cent. The total for November–December was 99; sepsis nil.

It will be seen that the excellent record resulting from the precautions regarding contagion was not in any way affected by this exposure to presumed gross potential infection.

The suspect department afforded a certain amount of material for this investigation. During November and December 19 suspect cases were delivered by carriers and showed no ill effects.

Conclusions.

It is possible that an individual with an acutely septic throat may cause puerperal sepsis by coughing on his or her hands, dressings, instruments, or patients. Any attempt to obtain positive proof of this would be on a par with observations regarding the results of vaginal examinations made with fingers suffering from whitlows, or of surgeons with septic throats coughing into abdomens at laparotomies.

The sum total of the combined investigations indicate:

1. That outbreaks of fever or definite sepsis in maternity hospital practice are due to contagion, and are preventable.

2. That throat carriers of the *Streptococcus haemolyticus* are very prevalent, and that apart from a negation of surgical and aesthetic principles—for example, coughing on hands, instruments, dressings, or patients—they are not a danger to parturient women.

In carrying out these observations special thanks are due to the matron, Miss Ferlie, and her nursing staff, and to Drs. Ind, Cover, and Blacklock for their valuable and most willing assistance.

THE MECHANISM OF REFERRED PAIN IN DYSPEPSIA:

ITS REFLECTION ON THE RESPIRATORY AND
CIRCULATORY SYSTEMS.*

BY

A. T. TODD, M.B. Ed., M.R.C.P. Lond.,

HONORARY ASSISTANT PHYSICIAN, BRISTOL ROYAL INFIRMARY.

THE term "dyspepsia" is here employed literally to mean a group of symptoms associated with abnormal digestion produced by any cause. Pain is a symptom in many cases, by no means in all. Though the cause of the dyspepsia may vary within rather wide limits the pains tend to conform to a few types or mixtures of types. It is well known that the pains of ulcer, inflammation, or ulcerated cancer may strongly resemble each other; as a result the nature of the pain and its site have little or no value in differential diagnosis. The timing of the pain, too, is of limited significance, early and late pain being regarded as due to a higher and lower lesion respectively. This similarity of pain brought about by so very different lesions raises the thought that the nature of gastric pain is not understood.

There has been controversy on the mechanism of gastric and other visceral pain for many years; several theories have been advanced, but no single one has met with general acceptance. This article describes a modification of the theory propounded by the late Sir James Mackenzie.¹ The observations on which it is based have extended over some years, and their accuracy is proved by the finding that the signs can be predicted from the symptoms.

The current theories to explain pain in gastric disease fall under two main heads:

1. *That the pain is felt in the affected viscus.* This is the popular theory; physician and patient speak of a pain in the stomach. The pain is regarded as being due to: (a) the action of hydrochloric or other acid on a sensitive mucosa or exposed submucosa; (b) abnormal contraction of the smooth muscle of the wall of the viscus; (c) stretching of the sensory nerves in the wall of the viscus; (d) stretching of the nerves or muscles of the supporting structures. There is abundant clinical and experimental evidence for each of these being a factor.

2. *That the pain is not felt in the viscus but is referred to some other structure.* It is of interest to us that one of the earlier propounders of this theory was a professor of physiology at the Bristol Medical School,² but his views were never widely known. The main upholders of this theory were Mackenzie and Head. Mackenzie considered that stimuli passed from the stomach via the lower dorsal sympathetic fibres to the corresponding spinal segments;

*The substance of a lecture delivered to the January meeting of the Bristol Medico-Chirurgical Society.

these segments became irritable after a time and then picked up normally subliminal stimuli, which were referred to the periphery as ordinary painful sensations. Most of us have experienced a demonstration of this theory—the throbbing of a septic finger or tooth socket. The subliminal sensation of the neighbouring pulsating arteries becomes perceived as a painful throb so long as the corresponding spinal segment is kept on the *qui vive* by irritation of the sensory nerves in and around the septic focus.

Mackenzie described areas of cutaneous hyperaesthesia related segmentally to the various viscera. In the case of the stomach he states that the segments affected are the lower dorsal. He refers also to muscular hyperaesthesia, but is vague as to its site, and is content to call it epigastric. It was found that the systematic investigation of cutaneous hyperaesthesia was not of great help in the diagnosis of gastric conditions. On concentrating on the hyperaesthetic muscles it was found that the cases fitted into patterns—certain groups of muscles were repeatedly found to be hyperaesthetic. It is considered that these muscles are frequently spasmodically contracted, because the tension of the muscle is greater than that found in a relaxed normal, and greater than that of the other side of the body when pain is unilateral. At times, especially in the intercostal muscles, which lend themselves to this observation, it can be seen that the muscle is shorter than that of the unaffected side. It is granted that proof of spasm is not always complete.

It was soon seen that the muscles which became hyperaesthetic were those classed under the muscles of respiration; in dyspeptics complaining of pain a section of the respiratory musculature has consistently been found to be hyperaesthetic and often spasmodically contracted. The most frequently affected muscles are the abdominal recti and obliques, the anterior scaleni, the interchondrals, the lower intercostals, the triangularis sterni, and the diaphragm. In cases of dyspepsia showing present or recent pain some, or all, of these muscles have been found to be hyperaesthetic, and pressure upon them has produced more or less severe pain; it is often found that pressure on one or more of them will reproduce the pain for which the patient seeks advice. This finding appears to be significant, for if pressure upon a fifth left interchondral or triangularis sterni reproduces the pain felt after food, it is unnecessary to throw the blame directly upon abnormal acidity or peristalsis.

Further support emerges from the examination of the dyspeptics who do not complain of pain. The common type labelled "hepatic" shows flatulence; feeling of weight and vague distress after food; inability to eat some or all fats, milk included; often inability to eat potatoes, however they are cooked; often showing a small or absent liver dullness and telangiectases about the diaphragmatic origin. This type seldom experiences pain of any severity, and muscular hyperaesthesia is little marked or absent. Thus, when pain is present hyperaesthesia, with or without spasm, of some of the respiratory muscles is found. Pressure on these muscles will frequently elicit the pain when absent and intensify it when present. When pain is not a symptom of any importance no hyperaesthesia is found. The deduction follows that gastric pain is referred to some of the respiratory muscles, and that the visceral sensation of the stomach is perceived as weight or vague discomfort.

The findings are derived mainly from inspection and palpation. Instrumental demonstration is not necessary. The suspected muscles are gently palpated, and, if found to be hyperaesthetic, the tone is compared with a similar muscle free from pain. The diaphragm cannot be palpated. Evidence of diaphragmatic involvement is obtained from the amount of diminution of air entry at the bases of the lungs, measurement of the expansion of the lower thorax, degree of phrenic trunk tenderness (nerve palpated by pressing directly backwards between the two heads of the sterno-mastoid muscles), and direct observation with the fluorescent screen. The anterior scaleni are palpated against the transverse processes of the cervical vertebrae by dipping behind the sterno-mastoids. The triangulares are reached by hooking the finger upwards and outwards in the angles between the xiphisternum and chondral margins.

The value of the hyperaesthesia of the individual muscles or groups of muscles in the diagnosis of the site of the lesion is not great. In oesophageal and gastric disease the left side is more affected; in duodenal and gall-bladder disease the right side. Usually both sets are involved, but one side predominates, and this would be expected, for strict localization of lesions to one viscus is rare in the patient though common in the textbooks.

The exact cause of this reflection of gastric pain upon the respiratory musculature cannot be given with any certainty. There is no direct path over which a reflex like that of a tendon reflex could pass. An indirect path is known via the sympathetic nerves to the dorsal segments. We may accept Mackenzie's hypothesis of segmental irritability due to bombardment by impulses carried by the rami communicantes. Assistance from animal experimentation is not likely to be of very great help in demonstrating such a reflex path, for the animals do not use their thoracic respiratory muscles so much as man does—thus, one can straddle the thorax of a winded horse without much discomfort.

Another explanation becomes possible when the actions of the individual muscles are reviewed. It is generally accepted that disease of the abdominal viscera is usually accompanied by a protective immobilization, or spasm, of the overlying muscles; this is regarded as an attempt to rest or shield the part. Such a defence would involve the rectus and oblique muscles in disease of the upper abdominal viscera. Immobilization of these would diminish abdominal respiration, and, more important, would occasion a drawing down and fixation of the thorax. The thoracic respiratory muscles would have to labour against this load, the more so because of the diminution of abdominal respiration. It may readily be understood that these muscles might become fatigued and show the pain and spasm of fatigued muscles. A closer review of the muscles most affected shows that these muscles are regarded as having double function—they are inspiratory and expiratory. This appears to be significant; granted the overwork, then the muscles which work double time become the most fatigued. It is too early, and anyhow foolish, to be dogmatic in pressing this theory of muscular ill-balance; it is my impression that the severity of thoracic muscular hyperaesthesia is proportional to the degree of defence in the upper abdomen.

The significance of this involvement of the respiratory musculature was not grasped for some years, but when one adds to the clinical findings the results of certain experiments, it appears astonishing that disorders of respiration are not even more frequent than is claimed. Thus, distension of the stomach, or a painful stimulus such as cold, occasions prolonged elevation and diminished expansion of the diaphragm³—perhaps the most important respiratory muscle. A stimulus to the stomach produces an expansion of the left side of the chest with eversion of the lower ribs; this impedes the action of the diaphragm,⁴ and is perhaps a partial explanation of the left-sided preponderance of hyperaesthesia in gastric disease. Increased intra-abdominal pressure, from gas or fluid, puts the diaphragm under stretch and diminishes thoracic expansion.⁵ It would appear that any interference with the stomach will result in widespread upset of the respiratory muscles.

It is therefore not surprising that dyspeptics suffer from respiratory disorders, but it is rather surprising that complaint of such disorder is relatively uncommon. Shortness of breath is not often volunteered, possibly because the dyspeptic is frequently underfed, and does not engage in any work entailing exertion; the clerk will work for months with dyspepsia, but the labourer will give up in a week or so. Some do complain of shortness of breath, and occasionally it is the most noticeable symptom. A majority, when questioned, state that shortness of breath is present, and a large proportion of the cases state that exertion, as well as food, intensifies the pain. One can readily convince oneself of the frequency of shortness of breath in dyspepsia by timing the duration of voluntary apnoea; after a few deep breaths the patient is instructed to take as large an inspiration as possible and retain it as long as he can. Voluntary apnoea lasts about sixty seconds in the average healthy adult, but longer times are

acquired by training. In painful dyspepsia periods of five to twenty seconds are usually found. In a small proportion of the cases the respiratory symptoms so overshadow the other symptoms that cardiac disease is suspected by patient and physician.

It is well known that the dyspeptic often thinks himself to be suffering from disease of the heart. How often do we find ourselves saying: "That is not your heart, but your stomach"? It is sometimes noted that the patient is still sceptical, especially if he is a physician or has a smattering of medicine derived from the lay manuals. He feels a pain where he knows his heart to be (due to left triangularis or fifth left interchondral), he is short of breath on exertion, he often experiences disordered action of the heart or palpitation, and sometimes has distinct syncopal attacks. Is he to blame for suspecting his heart to be wrong?

On careful consideration the patient is in the right. The cardiologists have thrown the blame of heart disease on every part of the heart and vessels, but have, so far, failed to remember that the heart and vessels—the circulatory system of the present day—are but a half of the vascular pump. The heart and vessels can pump blood onwards efficiently if healthy and if blood is supplied to them, but the heart, by itself, cannot get blood to pump. The auxiliary pump is the respiratory musculature, which derives, or sucks, blood from the great venous cisterns to the right auricle. When the respiratory pump is thrown out of action the heart will stop, unless the body is in the horizontal position, for then respiratory suction is not necessary. The climber who falls on the rope soon succumbs to syncope, as the rope throws the respiratory pump out of action; the laboratory rabbit suspended in a vertical position dies of syncope in a few minutes, for the weight of its abdominal viscera and the poor tone of its abdominal respiratory muscles prevent respiration. It was noted above that the lower animals have little or no thoracic respiration.

We have seen that the dyspeptic frequently shows respiratory disability; he does not use his abdominal muscles because they increase his distress; he cannot use his thoracic muscles because they are painful. He has two causes for shortness of breath—his respiratory movements are inadequate to aerate his blood, and his respiratory pump fails to supply the heart properly with blood.

Another reflection of this referred pain in dyspepsia is seen in the cases which show anginoid pain, or pain which must be regarded as identical with angina pectoris. Such cases are not uncommon. Numerous observers have described the dyspeptic symptoms of angina, but few the anginal symptoms of dyspepsia.

The explanation of the syndrome angina has produced as big a literature as dyspepsia, for though not so common it is much more spectacular. Time forbids a long excursion into the various theories of its causation; in brief they are: disease of the proximal part of the aorta with pain radiating into the upper dorsal segments; disease of the coronaries with anoxaemic cramp of the myocardium; Briscoe's conception of fatigue of the respiratory muscles owing to a change over from youthful thoracic to elderly abdominal respiration; vaguer nervous mechanisms. Evidence for and against all these theories is abundant. It is not my object to deny that each or all of these may not be causes of anginal pain, but to point out the possibility that these lesions may also result from the cause which produced anginal pain.

When we consider the limitation of effort; the pain when effort is not limited; the immobility of the whole of the respiratory musculature, perceived as a sensation of a vice-like grip; the pallor and breathlessness; the absence of evidence of circulatory failure in the early stages; the dyspeptic symptoms—we have enough evidence to constitute a case for the dyspeptic etiology of angina, at least in some cases. In dyspepsia one can select a perfect sequence of cases to show all steps upward from simple pain after food, through the effort syndrome, to true anginal pain. The group showing this pain have recovered, up to the present, when the condition has been treated as a failure of the respiratory pump due to dyspepsia. It is admitted that the number showing severe pain of this kind is few, but a large number have shown milder degrees.

The importance of the vascular lesions is not denied, and it may well be found that after a time the vascular lesion may become the chief one; it is contended that the vascular lesion may be produced by the angina. Proof for this contention is not complete, but there is some evidence: Cunliffe Shaw, Manouélian, Stopford, and H. Todd⁶ have shown that prolonged irritation of the nerves of a part is followed by vascular degeneration in the territory supplied by these nerves. Is it not possible that the prolonged irritation of the sympathetic fibres proceeding from the abdominal viscera, and the sensory nerves supplying the hyperalgesic muscles, may cause vascular degeneration in the areas supplied by these nerves? It appears suggestive that the segments postulated to be concerned in gastric pain by Mackenzie are the same as those which are blamed by the upholders of some of the neurogenic theories of angina.⁶

It should be noted that hyperalgesia of the muscles of respiration is not limited to patients who suffer from painful dyspepsia. Similar hyperalgesia is found in subjects suffering from fatigue of respiration from purely respiratory and circulatory causes.

Summary.

1. Pain of some of the upper abdominal viscera is shown to be referred to some of the muscles of respiration.
2. This respiratory muscular hyperalgesia may interfere with the respiratory pump and the supply of blood to the right auricle.
3. It is considered possible that some cases of pain resembling angina pectoris are due to this factor, and that properly directed treatment may result in cure in the early stages.

REFERENCES.

- ¹ Mackenzie, Sir J.: *Symptoms and their Interpretation*, third edition, Shaw and Sons.
- ² Martyn, S.: *British Medical Journal*, 1864, ii, p. 296.
- ³ Briscoe, Sir C.: *Lancet*, 1927, i, p. 751.
- ⁴ *Ibid.*, p. 752.
- ⁵ *Ibid.*, p. 643.
- ⁶ See Cunliffe Shaw, R.: *British Medical Journal*, 1927, i, p. 1046.

CHRONIC HYPOGLYCAEMIA.

BY

P. J. CAMMIDGE, M.D.,

LONDON.

CHRONIC HYPOGLYCAEMIA is regarded by most clinicians as a rare phenomenon of scientific rather than of practical importance, but, as I have met with a fasting blood sugar of 70 mg. per cent., or less, in more than 200 patients during the past five years, it cannot be so uncommon as is generally assumed. Unfortunately there is no characteristic sign to draw attention to hypoglycaemia, as glycosuria does to hyperglycaemia, but in most cases the history and symptoms are sufficient to suggest a diagnosis if the possibility of its presence is borne in mind. It will, I hope, help others to recognize chronic hypoglycaemia more frequently if I classify and discuss my cases, most of which had been treated for a variety of symptoms without success until the abnormal condition of their blood was discovered.

TABLE I.

Age.	No.	Sex.		Heredit.	Chronic Infection.	Faeces.				Urine.				Blood.	
		Male.	Female.			Enterocolitis.	Enteritis.	Colitis.	Normal.	Sugar.	Acetone.	Albumin.	Calcium Excess.	Calcium Low.	Calcium Normal.
0-10	44	27	17	31	27	31	4	4	5	20	28	3	3	3	3
11-20	19	10	9	12	8	9	4	5	1	12	7	2	5	5	4
21-30	32	18	14	12	7	11	11	5	5	20	10	5	8	10	11
31-40	32	15	17	7	6	13	7	9	3	17	6	7	4	11	9
41-50	35	27	9	5	15	16	13	1	6	18	4	5	12	13	7
51-60	19	11	8	1	2	6	6	5	2	7	2	3	4	4	7
61-	18	14	4	1	6	9	4	4	1	10	1	3	7	6	6
	200	122	78	69	71	95	49	33	23	107	58	28	43	52	57