He can find an account of this treatment in the brochure *Chemotherapeutic Researches on Cancer*, published by Messrs. Arrowsmith, price half a crown. As it has not been reviewed so far by the English medical journals of wide circulation, Mr. Hume perhaps has some excuse for his unscientific statement.—I am, etc.,

Department of Pathology, Bristol University, Jan. 13th. A. T. TODD.

THE "CARDIAC SIGN" OF CARCINOMA OF THE STOMACH.

SIR,—In his valuable paper on carcinoma of the stomach in the *Journal* of December 29th (p. 1163) Dr. William Gordon states that diminution or absence of the cardiac dullness is a very valuable confirmatory sign when carcinoma is suspected, and he mentions only two other conditions in which this sign occurs—namely, starvation and severe diarrhoea.

If this statement goes unchallenged it may lead to many errors of diagnosis, for this absence of the cardiac dullness is very frequently found in simple cardiac weakness such as that which occurs after influenza or diphtheria; and complete absence of the cardiac dullness may sometimes occur when the heart weakness is not enough to prevent the patient from walking about and living a life of normal activity.

With regard to the explanation of this physical sign, several factors may be concerned. One factor—though not the commonest—is, I believe, a lessening of the elastic tension of the heart walls due to poor muscular tone. This is, I believe, the explanation of the cases occasionally met with where a heart is so greatly dilated that the apex may be in the seventh interspace in (or even external to) the anterior axillary line, but nevertheless the whole of the cardiac area may be resonant and no area of cardiac dullness can be defined. If a solid organ overlies one containing air, it will only give a dull note when sufficiently elastic to absorb the vibrations due to the percussion stroke, and thus prevent them from causing vibration of the air in the subjacent "resonant" organ. This change of quality owing to lowered elastic tension of an organ is well illustrated by the disappearance of the dullness of the liver in cardiac weakness, even when only slight.

These questions are discussed in a paper of mine in the *British Medical Journal* for June 27th, 1925, on "The estimation of the cardiac output"; and also in my book *The Early Diagnosis of Heart Failure* (London, John Murray; pp. 184–208).—I am, etc.,

Birmingham, Jan. 9th. T. STACEY WILSON.

SERUM TREATMENT OF SCARLET FEVER.

SIR,—With reference to Dr. W. Brown's article in the *British Medical Journal* of January 5th (p. 13) on the subject of the Dick test in scarlet fever, I notice that he calls attention to the possibility of patients treated with anti-scarlatinal serum giving a positive Dick reaction, indicating that they have probably not developed their own antitoxic immunity. The defensive forces of the body have not had to fight as they would have done in the absence of serum.

Having treated over 800 patients without a single death directly due to scarlet fever during the past three years in Darlington I have become convinced of the marvellous value of anti-scarlatinal serum in the treatment of all early severe toxic scarlet fever patients. I have not, however, tested the Dick reaction in cases treated with serum, but my attention has been drawn to the increased tendency for late complications in convalescence, particularly in the form of minor articular rheumatism with occasional cardiac involvement, beginning about the third or fourth month after attack, in patients who left the hospital perfectly fit in their fourth or fifth week. One of my serum-treated patients developed a typical attack of scarlet fever five months after the first attack, but, as Dr. Brown states, it is too early yet to look for confirmation of this aspect of the effect of serum administration, since the occurrence of second attacks of definite scarlet fever was not so rare as to escape recognition before the introduction of serum therapy.—I am, etc.,

January 7th. G. A. DAWSON, Medical Officer of Health, Darlington.

CARDIOSPASM OR ACHALASIA OF THE CARDIA.

SIR,—It is evident that Dr. Hurst believes that those who sow the wind should reap the whirlwind; his tempestuous comments on my contribution to this intricate subject (December 15th, 1928, p. 1110) are tinctured by a dogmatism that tends to cloud rather than clarify the obscurity. At the outset he and Mr. Rake challenge my statement that "the existence of any hypertrophy of the muscular coat remains a subject of contention." That statement may appear ambiguous, but reference to the context would have saved both of them much superfluous criticism, referring, as it does, to the cardiac sphincter and not to hypertrophy of the oesophageal wall. Hypertrophy of the oesophageal wall has been found by all observers so constantly that it has become accepted as an invariable concomitant of the disease. In the matter of hypertrophy of the cardiac sphincter it should suffice that Moore has recorded two cases and Brown Kelly one, where definite hypertrophy of the sphincter was found, to justify the assertion that there remains difference of opinion as to fact.

Dr. Hurst three times refers contemptuously to the futility of founding deductions on my "one case." Yet in the *Proceedings of the Royal Society of Medicine* of January, 1915, we find (following his injunction to "consult the extensive literature") that Dr. Hurst reported "a case of achalasia of the cardia," and on the strength of that "one case" deduced his belief that the term "spasm" was incorrect and that "similarly many cases of so-called pylorospasm are due to pyloric achalasia, and ileo-caecal achalasia is the most frequent cause of ileal stasis"—sweeping conclusions to base on so slender a foundation.

Despite the implications of his declaration that "the word was first applied by me in 1914 to the cardiac sphincter," etc., the plain truth is that the term "achalasia" (*α*, not; *χαλάω*, I relax) was coined by Sir Cooper Perry and adopted by Dr. Hurst. Many years previously, in the *Transactions of the Pathological Society*, 1896, xlvii, 37, Sir Humphry Rolleston first suggested the pathology so strenuously advocated later by Dr. Hurst. It was Sir Humphry Rolleston's conception of a pathology that suggested to Sir Cooper Perry an alternative nomenclature. Since those days an enlarged experience and a more critical examination of the known facts have led many to believe that this pathology is inadequate, so that the term "achalasia" represents a premature attempt to crystallize in a word our conception of the disease. From 1913 onwards, until Walton's significant article in 1925, most of the published cases, treated by operation, were single cases.

Lest Dr. Hurst should feel any remorse for his temerity in publishing, with comments, his one case of achalasia, I offer this consolation, that more than fifty years ago (1877) James Paget reported one case of osteitis deformans so accurately and with such a wealth of acute observation that little of essential importance has since been added to our knowledge of that disease. Nor did Newton need to see more than one apple fall to enable him to make some very pertinent deductions as to the laws of gravity.

Treatment by bougies proving sometimes dangerous, often ineffective, and always tedious, a dilatable bag was devised by Plummer, who (with Vinson) reported in 1921 no fewer than 301 cases—some of these, however, required operation. The mercury tube is claimed to be more effective and to pass painlessly, but on these points there is divergence of opinion. In February, 1927, Mr. Harry Morley recorded in the *Lancet*, under the title "Cardiospasm," four cases. There emerge from his experience, from that of Walton, and from the experience of others, these facts—that the mercury tube does not always pass painlessly, that the passage is often difficult, that there is usually marked gripping of the tube, and that treatment by this means is tedious and often ineffective. Economic considerations (loss of time, loss of work, and the factor of expense) cannot be disregarded where alternative procedures are available. Mutch instances a case where the mercury tube required passing thirty-five times in one week, with little or no ultimate benefit; one of Morley's

patients, at the end of two years' treatment by the mercury tube, was "given a mercury tube to use daily." On the other hand, in Walton's fourteen cases, as in mine, operation conferred on the patient immediate, complete, and permanent cure, healing and complete convalescence being ensured within a fortnight.

Dr. Hurst might cease to refer to "Walton's operation" since the procedure of dilating digitally the cardiac orifice, after a preliminary gastrostomy, was first used and described by Mikulicz in 1881. Incidentally, Walton refers to it as "Mikulicz's operation."

Dr. Hurst seems to display a carelessness of statement and a disregard for logic when he says, "Hirschsprung's disease is probably acquired in early childhood, and is not congenital." Here he passes from the tentative "is probably" straightway to the definite "is not congenital." Again, "The relief following Walton's operation . . . the effect of which is to produce a more favourable postural tone of the sphincter" can scarcely be regarded as a pattern of lucidity; the term "postural tone" may hold some occult meaning for Dr. Hurst, but to less agile minds it requires further elucidation. In what way, precisely, is the tone of the cardiac sphincter modified by posture? Again, he tells us, "There is no question of *curing* this condition by any form of treatment . . . as the changes in Auerbach's plexus are of a permanent character, the hypertrophy and dilatation . . . remaining unaltered." It might induce Dr. Hurst to modify the confidence displayed in this *non sequitur* to learn that Plummer, out of the experience of 301 cases, declares that both hypertrophy and dilatation do disappear after the operation. The term "cure" has a definite meaning, being with reason applied to patients when all symptoms have completely disappeared, leaving no physical defect or depreciation in health. In this sense the cases treated by operation are assuredly cured, whatever term Dr. Hurst cares to apply to those treated by the mercury tube, who seem not infrequently to require daily, weekly, or monthly repetition of the treatment over an indefinite period.

There is no evidence of any change in the nervous mechanism of cases that recover. From such a mechanical obstruction death can only ensue as a direct result by starvation, or indirectly by a complication, as, for example, a mediastinitis. It must be a rare occurrence that, for lack of a gastrostomy, a case is allowed to terminate fatally from starvation alone, so that the changes in Auerbach's plexus found *post mortem* are not demonstrably due to the primary disease, but to the complication that caused death. Mere end-products seen in other terminal conditions are interesting, of course, but there are pitfalls in the way of correctly correlating them with the essential primary cause of the disease.

Professor Langmead is reported to have said at a meeting of the Brighton and Sussex Medico-Chirurgical Society that it was difficult to understand how a paralytic lesion could lead to hypertrophy down to the zone of structure, or how so much hypertrophy could result from obstruction imposed by a zone of muscle in normal tone.[1] Accepting the evidence adduced by Mr. Rake and others derived by *post-mortem* dissection of advanced cases as to degeneration of the nerve fibres, it seems strange that an isolated area of degeneration can occur as a primary lesion in an otherwise healthy individual—it is assumed to be unnecessary even to hint at any first cause such as a toxaemia from syphilis or other known antecedent of a neuritis, etc. Again, as regards "spasm," Professor Langmead is said to have referred to the positive evidence of gripping by the cardia which was vouched for by some surgeons. Somewhere in the region of the diaphragm there must be a physiological sphincter, one capable of holding back for an indefinite period a column of food, 8 inches or more, against the powerful peristaltic contraction of an oesophagus with grossly hypertrophied walls. For this tonic contraction no better term than "spasm" has yet been found.

In cured cases, as frequently figured in the literature, and in my case, there remains for a time a flaccid dilated oesophagus down which the barium meal is seen on the screen to fall like a tenuous waterfall. As surely as hypertrophy of muscle only occurs in response to an intermittent obstruction, just as surely does atrophy occur when the obstruction has been removed and the muscle ceases to have work to do. The hypertrophy of the muscle of the oesophageal wall may be expected on first principles to disappear, and even if it can be proved that the dilated tube never regains its original calibre, so long as it fulfils its function of conducting food without let or hindrance to the stomach and no symptoms reappear, the mere existence of dilatation does not invalidate the claim to "cure."

In saying that cure does not occur because "the changes in Auerbach's plexus are of a permanent character," Dr. Hurst is again guilty of a *non sequitur*, since it is by no means proved that the changes found by Mr. Rake and others have anything to do with the true cause of the disease. To describe the essential cause as being due to an inco-ordination of the neuro-muscular control of deglutition only leads us to ask what causes the inco-ordination, and to that question there has hitherto been no adequate answer.

There is a paucity of information as to the habits of life, etc., in the recorded cases; perhaps a more searching inquiry into the previous history as to imperfect mastication and the bolting of masses of solid food, suggested by my case, may establish this as one at least of the primary causes. It is true that cases occurring at so early an age as 2 years are on record, but even at that tender age the bolting of food is not unknown. There remain those said to occur within a few days or weeks of birth; these are few in number and demand very careful analysis before being accepted as true cases of cardiospasm; Professor Langmead instanced two of these juvenile cases, one of which was completely cured by suggestion.

The operation is admittedly severe, but in careful hands it is reasonably safe (none of Mr. Walton's operated cases died); recovery and cure followed with impressive rapidity and with a completeness that inevitably challenges the tedious, protracted, and expensive course of those treated by the mercury tube; the scope of the latter method would appear to be in mild cases, or in cases complicated by some other serious disease. Certainly the results achieved by the mercury tube do not prove it to be the counsel of perfection in all cases, nor do they confer on their protagonist any prescriptive right to generalize and to refer disparagingly to the deductions of those who report single cases.—I am, etc.,

Swindon, Jan. 9th. H. H. GREENWOOD.

[1] *British Medical Journal*, December 29th, 1928, p. 1180.

TONSIL AND ADENOID OPERATIONS.

SIR,—"Whenever a laryngologist brings forth some fresh instrument for the prevention of haemorrhage or to simplify the operation for the removal of tonsils and adenoids, I can tell exactly how far he has got in laryngology" is the saying of one of my former chiefs.

I can suggest no golden rule to meet every difficulty that may be encountered in the course of this perplexing operation; but having performed many thousands of operations by the guillotine method, and many hundreds by the method of dissection, perhaps I may be allowed to express an opinion on this somewhat worn subject. I have had only seven cases of haemorrhage in over 5,000 cases treated by the guillotine. Three of these required ligaturing; a gauze plug and Watson-Williams pressure forceps sufficed for the remainder. Haemorrhage after the guillotine operation usually means that a piece of tonsil has been left behind.

While admitting that dissection is the ideal surgical method, I suggest that it is one that cannot, as a general rule, be practicably applied in such large clinics as I have had to deal with. In dissection, haemorrhage will result unless care is exercised not to go beyond the capsule or to injure the faucial pillars. Anything that Mr. Tilley says must be taken as advice from a teacher; I am heartily in agreement with him.—I am, etc.,

Maidstone, Jan. 7th. J. ALDINGTON GIBB.