A British Medical Association Lecture

ON THE

RELATION OF EARLY MISTAKES IN INFANT FEEDING TO THE DYSPEPSIAS OF LATER INFANCY AND CHILDHOOD.*

BY

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At a recent meeting of the Section for the Study of the Diseases of Children at the Royal Society of Medicine, Dr. Jules Taillens,¹ professor of paediatrics at the University of Lausanne, gave a very interesting address on the same subject as that which I have chosen for this lecture—that is to say, “The dyspepsias of childhood.”

Professor Taillens claimed that these dyspepsias often took a misleading form and produced symptoms of general weakness, wasting, pyrexia, and even coughing, which were frequently mistaken for those of tuberculosis. He further stated that gastric hyperchloracidity, or, as we usually call it, hyperchlorhydria, was the most frequent cause of these dyspepsias. Owing to the limitation of time, Professor Taillens did not explain exactly how hyperchlorhydria developed into the above group of symptoms, and although I agree with him that hyperchlorhydria may ultimately produce such results, I am not sure that I should agree with him as regards the explanation. His address, however, created so much interest that I thought that the same subject might well form the basis of this lecture.

If hyperchlorhydria, or the excessive secretion of hydrochloric acid in the stomach, is accompanied by a corresponding supply of pepsin, there must be a great capacity for digesting protein, and, as a matter of fact, there is usually in cases of hyperchlorhydria a considerable appetite or craving for nitrogenous foods. Individuals who are the subjects of such gastric hyperacidity do not, as far as my experience goes, suffer at first from any of the recognized symptoms of dyspepsia. They are usually, at least during the early years of life, individuals with a healthy appetite. They enjoy their food, are impatient for their meals, and appear to be well nourished. They sometimes feel a so-called “sinking of the stomach” if their cravings for food are not immediately satisfied, but these discomforts pass off rapidly after a meal. Such individuals deserve to be regarded as “eupeptics” rather than dyspeptics. None the less, if these individuals, as they grow older, continue with full meals, they begin to have epigastric pain about two hours after food—a premonition of duodenal ulcer. Similarly, infants and quite young children with various degrees of hyperchlorhydria do not suffer from ordinary dyspeptic symptoms, but they do suffer from periodic attacks which, according to my own interpretation, depend on a disturbance of liver function. Among the several functions which the liver performs, one of the chief is the de-aminization of amino-acids, and their conversion into urea or ammonia. There is a limit to the tolerance of all organs with respect to each of their functions, and when these limits of tolerance are overstepped they will show signs of fatigue and cause the development of various symptoms which are the direct result of such breakdowns; the liver is no exception to this rule—with overwork its power to de-aminize amino-acids begins to wane.

Now with hyperchlorhydria there are strong inducements to consume excessive quantities of protein foods, which, if indulged, will impose on the liver the duty of de-aminizing very large quantities of amino-acids. The result is easy to predict. When hepatic function fails and the de-aminizing process is feebly conducted, it follows that the amino-acids in an unchanged condition will gain access to the systemic circulation.

In a very interesting paper² contributed by Dr. H. W. Barber and Dr. G. H. Oriel to the Section of Dermatology at the Annual Meeting of the British Medical Association held at Cardiff, the results of such flooding of the blood

with amino-acids were described as an asthma-prurigo symptom-complex. The group of symptoms described by these authors is made up of:

1. Cutaneous symptoms of the urticarial type.
2. Subcutaneous symptoms of the nature of angioneurotic oedema.
3. Respiratory symptoms of the asthmatic type.
4. Gastro-intestinal symptoms representing certain forms of vomiting and diarrhoea.
5. Nervous symptoms, including migraine and epilepsy.
6. Articular symptoms, including paroxysmal hydrarthrosis.

All these symptoms are, of course, never found combined in any one person suffering from excess of amino-acids in the blood. In infants and young children the symptoms are of rather a specialized form, but the point which I wish to emphasize is, that there are a large number of symptoms in children which follow as a result of the condition of hyperchlorhydria which cannot properly be classified as dyspeptic symptoms, but rather as those of liver inefficiency, and associated with an increase of amino-acids in the blood. I venture, therefore, once more to suggest that many or most of the results of hyperchlorhydria are due to too active a digestion rather than to a bad one.

Before I leave this subject I would like to say a few words with respect to the etiology of the very common condition of hyperchlorhydria in young children. It arises as a natural result of the modern practice of feeding infants with cow's milk with an excessive content of caseinogen, and it matters very little whether the milk is ordinary dairy milk or dried milk, for in both cases it is almost invariably given in a way which calls for a high degree of gastric acidity, owing to its considerable protein concentration.

Breast milk, or rather its protein content, can be digested by pepsin when comparatively little acid is secreted in the stomach, owing to its low buffering capacity, whereas cow's milk requires the addition of a large amount of acid, owing to the high buffer effect of its proteins and phosphates. Hence it follows that if infants can be fed on cow's milk without the development of vomiting and other gastric symptoms the amount of acid secreted must be very great, much greater than would have been the case if breast milk alone had composed their diet. Now, owing to various resources, such as the addition of sodium citrate to such milk dilutions (which prevents the coagulation of casein in a heavy clot with consequent vomiting), gastric tolerance can usually be acquired by infants, even though the milk is only slightly diluted or not diluted at all. This tolerance, with its natural concomitants hyperchlorhydria and a large appetite for protein foods, leads to overfeeding, overfeeding leads to overproduction of amino-acid bodies, and these in turn to hepatic failure and to a flooding of the system with unaltered amino-acids—hence the appearance of symptoms belonging to the “asthma-prurigo” syndrome.

I do not wish to complicate the subject with unnecessary detail, but the accompanying graph, which has been constructed for me from figures supplied by Mr. Leslie J. Harris, D.Sc., working in the Nutritional Laboratory at Cambridge, and confirmed by Dr. Montague Maizels, working in our own laboratory at the Infants Hospital, shows the amount of acid which it is necessary to add to breast milk and cow's milk respectively to bring them up to the degree of acidity at which pepsin will effect the digestion of the protein.

From this graph it will be seen that while about 24 c.cm. of decinormal hydrochloric acid must be added to 100 c.cm. of breast milk to activate the pepsin and make it effective, between 40 c.cm. and 60 c.cm. must be added to cow's milk to obtain the same result. This difference is due to the high buffering effect of the cow's milk. From this it is clear that if an infant is fed on cow's milk instead of breast milk the acid-secreting cells of the gastric mucous membrane will tend to be more highly developed than in the case of breast-feeding, and a state of hyperchlorhydria will result, which will give an appetite for protein foods and cause the condition to “grow worse by what it feeds on.”

Mr. Leslie Harris tells me that the buffer value of different foodstuffs may generally be represented with fair accuracy in the ratio of their protein content—that is to

* Delivered before the Cardiff Division, December 20th, 1928.

say, it makes very little difference what be the source of the protein, the actual amount of acid which must be secreted to digest it will be proportional to the actual quantity of protein present in the food. Hence cow's milk, with its high protein content and consequently high buffer value, will necessitate the secretion of large quantities of

In this graph the vertical line is graduated in accordance with its pH or degree of acidity: pH 7 is neutral point; 6, 5, 4, 3, and 2 represent respectively increasing degrees of acidity. The degree of acidity at which pepsin digestion is at its maximum is about 3 pH, although it begins at a lower point—that is, about 4 pH. The horizontal line marked 0, 20, 40, 60, 80, 100 indicates the volumes of decinormal (N/10) HCl added to every 100 volumes of food.

acid in the stomach, and will lead ultimately to a condition of hyperchlorhydria.

I hope it will not be assumed that I attach little importance to the dyspepsias which result from a deficient secretion of hydrochloric acid, owing to the fact that I have given special prominence to the condition of hyperchlorhydria.

One of the most important lessons I have learned from certain investigations¹ which we have made at the Infants Hospital is that the pH of gastric juice in infants shows an immediate fall with the onset of pyrexial and other constitutional symptoms. With a falling off in efficiency of gastric digestion, followed in all probability by similar falls in the efficiency in pancreatic and intestinal digestion, there results a considerable danger of undigested food entering the large intestine, which gives opportunity there for bacterial decompositions. Under normal conditions neither decompositions nor fermentations occur to any appreciable extent in the small intestine, for although the chyme in this part of the bowel is not absolutely sterile, it is so relatively, owing to the antiseptic influence of the acid gastric juice. Under conditions of hypochlorhydria the stomach does not exercise the same influence, and the chyme when it enters the small intestine is in a less sterile condition, and consequently more liable to fermentative and decomposition changes. Hence a condition of hypo-acidity in the stomach has a tendency to lead later to intestinal toxæmia.

Now although the poisonous products of decompositions and fermentations in the large and small bowel may lead to local disturbances, such as catarrhal enteritis, diarrhoea, colic, or flatulence, the more serious effects are, as in the case of hyperchlorhydria, remote and constitutional. The toxic products of such decompositions are absorbed and carried along the portal channels to the liver, where under normal conditions they are detoxicated and rendered harmless, in a manner comparable to the de-aminization which occurs in the liver in the case of the normal products of protein digestion. In the same way that the liver can fail in its de-aminizing functions from overwork, so also can it fail in its detoxicating functions with respect to the poisonous products which result from bacterial activity in the bowel.

Now when the liver fails in its detoxicating functions the unaltered toxins enter the systemic circulation and exercise their deleterious influences on the body as a whole, and on the cells of the central nervous system in particular. The symptoms of this condition are, therefore, largely nervous, and were it not that the resulting crises are accompanied by anorexia and vomiting, which preclude the further taking of food, the symptoms would be continued indefinitely until death terminated the scene. Thus we see that, both in hyperchlorhydria and hypochlorhydria, the symptoms are remote and not directly concerned with the function of the digestive system proper.

The primary symptoms associated with dyspepsia are vomiting, gastric pains, colic, wind, diarrhoea, and constipation—symptoms which represent disturbances of the motor and not the digestive functions of the alimentary tract. Almost without exception, however, motor symptoms are combined with the deferred constitutional symptoms. In infants it is the motor symptoms which mainly attract attention, whereas in older children and grown-up individuals the main symptoms are of the deferred constitutional type.

TYPES OF DYSPEPSIA IN INFANCY AND EARLY CHILDHOOD.

In the time at my disposal it would obviously be impossible to give any detailed account of all the common varieties of dyspepsia, nor, indeed, do I feel competent to classify them on any rational or comprehensive basis, but I think if I take certain representative types which in my experience are the most frequently met with in infancy and early childhood, I shall be able to give certain indications for the recognition of the many combinations of them with which we are often perplexed. The three types that I propose to take are as follows:

1. Vomiting, which occurs in infancy, and is due to overaction of the pylorus.
2. The coeliac type of diarrhoea, generally found in children between 1 and 6 years of age.
3. The chronic dyspepsias of early childhood (3 to 7 years), accompanied by periodic attacks of vomiting with acidosis (cyclic vomiting).

Type 1.

A considerable number of the infants under 3 months of age who are brought to the Infants Hospital for so-called indigestion have a history of vomiting suggestive of pyloric stenosis, and in quite a large proportion of such cases a diagnosis to this effect has already been made by the practitioner or welfare centre sending up the case. Although the surgical treatment by Rammstedt's operation is a most satisfactory and rapid means of alleviating the pyloric obstruction, I never refer such cases to the surgeon unless the symptoms refuse to yield to ordinary medical measures. The presence of a palpable tumour with visible peristalsis, even when these symptoms are combined with a history of intractable vomiting, is not, in my opinion, sufficient evidence of pyloric stenosis to justify an operation without further corroboration. I have had many cases with these symptoms in which the x-ray examination after a bismuth meal has revealed rapid emptying of the stomach through the pyloric opening. In such cases, however, obstinate constipation is not one of the symptoms, as it is in true pyloric stenosis, and there is usually faecal matter as well as mucus in the motions. A bismuth meal is of the greatest help in distinguishing pyloric stenosis from simple overaction of the motor functions of the stomach, but I have known a bismuth meal pass quite normally through the pylorus and yet ordinary food be retained in the stomach until it is finally evacuated by vomiting. I regard lavage of the stomach some three hours after a feed as giving better evidence of the degree of motor efficiency of the stomach, or at least of the capacity of the stomach to empty itself, than a bismuth meal, although both, taken in combination, give still more reliable evidence. A wash-out of the stomach three hours after a meal will throw light on several points—the character and quantity of the residue, its degree of acidity or alkalinity, and the actual volumetric capacity of the stomach itself.

All these items of evidence pieced together are of great assistance in diagnosing the cause of the vomiting, and

in formulating an opinion as to whether medical or surgical means are indicated in the treatment.

In the vast majority of cases of vomiting in young infants investigated on these lines it is found unnecessary to have resort to surgical intervention. The cause is nearly always spasm and not true stenosis of the pylorus. This spasm almost invariably yields to ordinary medical treatment and appropriate feeding. Now I am sure I am not exaggerating when I say that in 99 per cent. of cases of persistent vomiting in young infants I have found a history of gross errors in feeding during the first few days or weeks of life, which has adequately and satisfactorily accounted for the setting up of pyloric spasm. There is almost invariably a history of overfeeding in cases of breast-fed infants, and of unsuitable feeding in the case of those artificially fed.

Spasm is very easily set up in the sensitive pyloric sphincter, the true function of which is to protect the delicate intestinal tract from unsuitable varieties or quantities of food. Spasm once set up in this sphincter during the early days of life, when it is learning its functions, is very apt to persist and continue even though unsuitable food is no longer to blame. A well-organized pyloric reflex—a reflex which protects the intestines from excessive quantities of semi-digested products, or from the irritation of totally undigested food—is one of the essential conditions of a good intestinal digestion. This pyloric reflex can be so disorganized by early mistakes in feeding that re-education may become difficult or impossible, and symptoms of dyspepsia persist during the whole of life.

Excessive acidity, or hyperchlorhydria, sometimes delays the emptying of the stomach, and when this occurs long-continued contractions of the pyloric muscles may lead to their hypertrophy. On somewhat comparable grounds subacidity of the gastric juice also leads to delayed emptying and long-continued contraction of the pylorus and hypertrophy. Inasmuch as the pH of the stomach is mainly dependent on the nature of the food, as previously explained, we see, too, how to a great extent the establishment of a normal pyloric reflex, and all that this portends, is dependent on a correct adaptation of the food to the physiological requirements.

Now let me state quite briefly how all the troubles which depend on a perverted pyloric reflex can be avoided. In breast-feeding the first secret is to regulate the quantity of milk which enters the stomach during the early days of life by the invariable employment of the "test" feed, a control which is particularly necessary when lactation is first established and when there are great dangers of flooding the stomach with an excess of milk.

In artificial feeding the desideratum is never to allow any variety of food to gain access to the stomach which can provoke any exaggerated or excessive gastric or pyloric reaction. The worst offender of all is cow's milk, no matter what be the degree of dilution. Personally I always use as the basis for the artificial feeding of newborn babies very thoroughly peptonized milk modifications. If this precaution is taken and the proper quantities are given at regular intervals, I guarantee that a case of so-called hypertrophic pyloric stenosis will never be met with; indeed, I will go so far as to assert that there will be no case of persistent vomiting of any kind.

I am often asked what is the ultimate fate of these so-called pyloric cases. As far as my experience goes, their fate depends on the degree of aggravation which has in the first instance brought about the spasm of the sphincter. Cases in which only slight defects in the feeding have produced spasm of the pylorus necessitating operation usually do badly, because a violent reaction to a slight degree of stimulation implies a very sensitive or spasmophilic state of the nervous system. I have noticed in such cases a general tendency in later life to increased sensibility to all forms of injurious agents—a sort of general anaphylaxis. Cases in which very gross liberties have been taken with the feeding during the early days of life, without producing very violent reaction—that is to say, reactions which are easily controlled by simple medical measures without resort to operation—usually do extremely well as soon as the vomiting ceases.

Type 2.

Now with respect to the "coeliac" type of dyspepsia, I will preface my remarks by saying that I do not regard the syndrome to which this name has been applied as a clinical entity or definite disease. I regard it merely as a combination of symptoms resulting from a variety of causes, mainly concerned, however, with an atrophic condition of the intestinal mucosa and atony of the intestinal walls, but sometimes due to an atrophic or undeveloped condition of the pancreas. The liver is certainly small in many of these cases, but this is better explained on the ground that the liver has little work to do owing to restricted absorption, than that it is small owing to innate or congenital causes. Moreover, the fact that this smallness of the liver itself disappears as recovery takes place also militates against the view that there is any congenital defect in the liver itself, or that coeliac disease itself is of congenital origin.

In nearly all cases of "coeliac" disease which have come under my notice there has been a history of good or normal progress up to a certain point, and that then there has been a crisis of some kind with diarrhoea and all the usual symptoms. Moreover, in the majority of these cases there has been a history of bad feeding—generally excess in the total quantity combined with deficiency of vitamins, which will explain all the subsequent events. But in whatever way these cases commence, there can be no doubt that if an atrophic and unhealthy condition of the intestinal mucous membrane exists, it can be perpetuated and aggravated by the treatment to which nearly all "coeliac" cases are subjected. Owing to the peculiar character of the stools, fats are almost universally eliminated from the diet, and consequently there is almost complete starvation as regards fat-soluble vitamins as well as fat itself; secondly, the consumption of fruit is vetoed because of the diarrhoea; and thirdly, vegetables and other cellulose-containing foods are forbidden because of their stimulating effect on peristaltic action. So-called "coeliac" disease, or at least a very large proportion of the cases which pass under this name, are associated with, if they are not definitely due to, dilated and atrophied intestines, and these two latter conditions are almost certainly caused by bad feeding, and very probably chiefly by want of vitamins. If this is so, no course of treatment could be worse than the one outlined above. It illustrates the therapeutic principle of "treating a bite with a hair of the dog that bit"—that is to say, a bad dietary is treated by a worse one. I find these cases always improve and generally become cured by acting on the principle that since little food can be absorbed little food must be given; and since the cause of the disease is mainly a want of correct balance in the dietary, the diet must be properly balanced, and contain a good representation of vitamins and mineral elements.

The presence of fat in the stools is no contraindication for the giving of fats. Fats in the form of insoluble soaps furnish, in the case of infants, their only source of roughage, and they serve the same purpose at any time of life. But in order that insoluble soaps may be formed out of the neutral fats consumed as food two conditions must be fulfilled. First, the fats must be "split," and secondly, there must be an adequate supply of calcium or magnesium with which the fatty acids can combine to form the insoluble soaps. Hence the stools of "coeliac" patients should be examined to see whether soaps are present or not. If either fatty acids or soaps are present it proves that the fats consumed are split, or digested, and that lipases are provided by the pancreas or other source. If only, or mainly, neutral fats are present in the stools, pancreatic preparations must be given independently to ensure the splitting of the fat, and in all cases a liberal supply of calcium should be provided to neutralize the fatty acids and so form insoluble soaps. For this purpose either prepared chalk or aromatic chalk powder must be given in full amount. One of the chief essentials in the treatment of these cases is unlimited patience and rigid adherence to the main principles in spite of obvious temptations. The dangerous time is when improvement begins to take place and appetite returns, for then there are strong inducements to relax rules and increase quantities. Since in

these cases little food can be absorbed, such sources of energy as are available must be conserved by keeping the patient thoroughly warm—in bed for preference—and by limiting exercise as far as possible.

The ultimate fate of the majority of "coeliac" cases is recovery, but even after what may be regarded as complete recovery there is always a tendency to recidivism, and some dilatation of the intestines usually persists.

Type 3.

As I have already indicated, the symptoms of certain forms of dyspepsia are so remote and general that they are liable to be attributed to tuberculosis or other constitutional trouble, rather than to actual defects in the digestive processes. The kind of case which I am about to describe is essentially of this character in its most typical form. It occurs in children between 2 and 16 years of age. It is chronic and remittent in character.

Children suffering from this form of dyspepsia are never really well, neither do they look well. They usually do not complain of feeling ill, but they have little energy, are easily tired, and have capricious appetites. There is often a continued temperature, usually under 100° F., sometimes lasting for many months. The complexion is sallow, with dark rims to the eyes, the tongue is dirty, and the breath tainted. There is irritability of temper, restlessness at night, or night terrors. The children often complain of abdominal pain or stitch in the side on running. The urine is usually thick, cloudy, or ammoniacal, and the stools are offensive. One of the most diagnostic and characteristic of the symptoms is the regular manner in which periodic attacks of biliousness supervene. These symptoms make up a clinical picture which must be perfectly familiar to everyone; but since all of them in combination never occur in one case the presence of even a few of them should put the practitioner on his guard.

All these symptoms can be rationally and satisfactorily explained on the assumption that the food consumed is not completely digested, owing either to its quality or total quantity, or to the relatively deficient powers of the digestive functions to cope with the task imposed upon them. Whatever may be the cause bringing about this failure of digestion the result is the same. Food which should have been digested normally by the normal enzymes of the alimentary tract remains undigested, or only partially digested, and becomes the pabulum of bacteria in the large bowel, and, as a consequence, the colon assumes an unhealthy condition, with offensive stools, flatulence, abdominal pains, and stitch in the side. This stitch in the side, so very characteristic of intestinal toxæmia, is, I believe, caused by the dragging of a heavy and sagging colon on its two points of support—its hepatic and splenic flexures—a dragging which is intensified by exercise in the upright position.

The toxic products of intestinal putrefaction are absorbed and carried to the liver, where for a time they may be effectually dealt with and rendered innocuous. Failure, however, is evidenced by offensive breath, dirty tongue, irritability, and other nervous symptoms. After varying periods of endurance the liver breaks down in function, and then the systemic circulation is inundated with a flood of unaltered toxic products, with intensification of the nervous symptoms, which take the form of vomiting, high temperature, migraine, or sick headache.

Under these conditions, and especially owing to the vomiting, the further consumption of food becomes impossible, and the liver obtains a much-needed rest. General recovery then takes place, the appetite returns, and the whole cycle of events is repeated with systematic regularity. The treatment of these bilious attacks, or cyclic crises, is usually by calomel or grey powder. This treatment, which as a rule gives immediately good results, is not really rational or far-seeing, for its efficacy is mainly due to stimulation of the liver, and not to removal of the cause. It acts, in fact, on the principle of "spurring on the tired horse," instead of taking the load off its back. The toxic bodies which directly bring about these breakdowns of liver function are due to the decomposition of nitrogenous foods, such as butcher's meat, fish, eggs, poultry, and milk. Hence it is obvious that the treatment should be concerned with a limitation of the intake of these particular

foods. On the other hand, the fermentations which take place in carbohydrate and cereal foods lead to the formation of considerable quantities of acid bodies, such as acetic acid, butyric acid, lactic acid, propionic acid, and so on; these do not seriously tax hepatic function after absorption, but they do seriously drain the bicarbonate reserves of the blood and lead to those true conditions of acidosis which so often complicate the actual hepatic crisis. It should be clearly recognized that conditions of acidosis proper, in which there is a reduction of the alkaline reserves of the blood, should be distinguished from conditions of ketosis in which there is usually acetone in the blood, the breath, and the urine. A true ketosis seems to be one of the results of defective fat metabolism and hepatic insufficiency, and therefore it is scarcely to be wondered at that in these cases of periodic liver attacks due to intestinal toxæmia the condition should be complicated by a ketosis. In fact, I think it may be assumed with some confidence that in cases of intestinal toxæmia due to defective digestive processes there is generally a combination of all these conditions—hepatic crisis, ketosis, and true acidosis. The rational treatment of these cases involves three main principles of therapeutics: First, and mainly, the limitation of nitrogenous foods. Secondly, the limitation of all foods within the bounds of physiological requirements. Thirdly, the prevention as far as possible of intestinal decomposition by sterilization of the intestinal tract. Finally, by the prevention of conditions of true acidosis by the free administration of alkalines.

I hope that the three types which I have chosen to illustrate my thesis will help to show that if the digestive functions are imperfectly performed certain immediate, and, if I may use the term, proximal, symptoms, are liable to be produced which are mainly concerned with disturbances of the neuromuscular mechanisms that control the transit of food through the alimentary tract. These symptoms carry a warning note which, if attended to, should obviate the development of other, more serious and more distal, symptoms connected with the breakdown of liver function and with the poisoning of the system with various toxic substances.

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THE SIGNIFICANCE OF INTRADERMAL TUBERCULIN TESTS.

A PRELIMINARY NOTE ON AN INVESTIGATION IN PROGRESS BY
THE MEDICAL STAFF OF THE WELSH NATIONAL MEMORIAL
ASSOCIATION.

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THE intradermal method of tuberculin testing was introduced by Mendel¹ and Mantoux² in 1908. For quantitative tests it has the advantage over the von Pirquet reaction in that the whole of the tuberculin used does actually enter the tissues. Happ and Casparis³ consider that 0.01 mg. of tuberculin given intradermally corresponds to the usual von Pirquet test. While the von Pirquet test carried out according to the technique described by me in the *British Medical Journal* of February 2nd, 1924, appears to give results equivalent at least to 0.1 mg. of tuberculin intradermally, there can be no question but that the intradermal method is a much more sensitive test than the cutaneous one, and gives a greater amplitude in the elicitation of latent infection.

As compared with the subcutaneous tuberculin test, the intradermal method aims at a local, not a general, reaction, and it is an interesting fact that doses of tuberculin capable of giving rise to severe focal and constitutional reactions in infected persons can be given intradermally without exciting any appreciable response other than the local one at the site of inoculation. An excellent account of this and other forms of the tuberculin test was given by Burhans⁴ in 1927, and should be consulted by all interested in the subject.