The Schorstein Lecture

ON

CONGENITAL PYLORIC STENOSIS.

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BY

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THE title of this lecture is "Congenital Pyloric Stenosis," but it must be admitted that the term "congenital" is really a "question-begging epithet," for it assumes as proved what is one of the most disputed points in the natural history of the disease. Nevertheless I have resolved to adhere to it, not only because it was the title by which the condition was christened when it was first seriously introduced to professional notice, but also because the alternatives which have been suggested ("congenital gastric spasm," "congenital spastic hypertrophy of the pylorus," etc.) are all open to the same objection in an equal degree. At all events, and whatever one chooses to call it, this form of pyloric stenosis exhibits clinical and pathological problems of the most interesting and baffling character; indeed it would not be going too far to speak of it as a morbid mystery. It presents, too, a conspicuous example of the difficulties of clinical observation and of the humiliating possibility of the whole profession overlooking for years something which must all the time have been lying, so to speak, under its nose. In this respect the history of the disease has a lesson for us all. That history is soon told. Although the *flair* for medical antiquities which characterizes my distinguished predecessor in this lectureship—Professor Osler—has enabled him to unearth¹ a probable example of the disease published so long ago as 1788 by one who bore the prophetic name of Hezekiah Beardsley, and although more unequivocal examples were published by Williamson² and by Dawosky³ in the middle of last century, it was not until the year 1888 that Hirschsprung⁴ first really attracted attention to the condition. Even after that some years elapsed, so slowly did it attain recognition, before its existence was at all widely acknowledged, the result being largely brought about—in this country, at least—by the writings of my old teacher, Dr. John Thomson. It has now at last attained a place in the hierarchy of disease, and almost every day adds, with almost embarrassing profusion, to the published examples. I have said that this record is painful reading for the clinical physician, but lest any pathologist present should be inclined to scoff I hasten to add that it reflects no great credit on *them*, for undoubtedly babies must have been dying—dozens of them—from congenital pyloric stenosis every year, and yet its existence was as completely, and less excusably, overlooked on the *post-mortem* table as at the bedside. You say that it may be a new disease, or, as is sometimes said of appendicitis and duodenal ulcer, that it has greatly increased in frequency. I am afraid I do not believe it, for my personal experience gives me only too good reason to believe that it was simply overlooked. It was only some six or seven years ago that I was disposed to regard congenital pyloric stenosis as a myth, or, at all events, as a clinical occurrence of the greatest rarity. Then suddenly I learnt how to recognize it, and since that time I have seen a large number of cases, including twenty in private alone. Now, the only possible interpretation of this experience is, not that cases suddenly began to occur in my practice six years ago, but simply that up to that time I had been blind to their existence, and this, I believe, has also been the experience of the profession at large. Even yet it does not seem to be recognized everywhere. Cases have been published in abundance in Germany, America, and England, but in the Latin countries scarcely one. Is it conceivable that congenital pyloric stenosis does not occur in France and Italy? Is it not much more likely that, like some other diseases of Teutonic nativity, it has found difficulty in establishing its identity outside the Fatherland except in England and America, which are notoriously favourable to the admission of aliens?

CLINICAL FEATURES.

In the study of the clinical features of this disease one is met at the outset by two curious facts. One is its greater frequency in boys, the available statistics giving about 80 per cent. of all cases as occurring in the male sex, whilst the other is the relatively large proportion of first-born children who are thus affected. Thus, out of 19 cases recorded by Davidson,⁵ no fewer than 10 were in the first child of the family. I had almost added to these peculiarities the frequency with which the children of doctors are affected. At all events, in my own experience, no less than 6 out of 20 cases seen in private were the children of medical men.

That the greater frequency of the condition in boys and in the firstborn is without significance one cannot believe, but no plausible explanation of these facts has yet been offered, nor am I prepared to suggest one.

The symptoms are few and suggestive, the most prominent being persistent vomiting. It should be specially noted that the vomiting rarely dates from birth, but usually makes its appearance between the second and fourth weeks. This, as we shall see later, has an important bearing upon the pathology of the disease. The vomiting is often what is called "projectile" in character, and more material is apt to be brought up than was swallowed at the last meal. It usually comes on soon after a feed, the vomited material consisting of curdled milk, mixed sooner or later with mucus, and occasionally with streaks of blood. There is one curious fact about the vomiting which I do not remember to have seen described, but which I have often verified—namely, its tendency to cease for some hours with any change of food and to recur again as violently as ever. This is another of the many anomalous features exhibited by the disease.

At the outset, at least, the vomiting is accompanied by constipation, which helps to distinguish it from dyspeptic vomiting in which diarrhoea is usually present. The constipation is sometimes spoken of as a pseudo-constipation, inasmuch as it is not due to any disorder of the intestine, but simply to a failure of the food to find its way into the bowel at all.

Meanwhile the child rapidly loses weight and soon becomes extremely emaciated. The wasting, however, is not like that met with in cachexia. The child is not anaemic, but simply looks starved, and often remains surprisingly bright in spite of it. In this respect it differs from the wasting of most forms of organic disease, and to the experienced eye the difference often suggests a correct diagnosis.

The physical signs are few but sufficiently characteristic. In the first place, the stomach is dilated and may cause a swelling in the upper part of the abdomen, which is all the more striking in contrast with the attenuation of the rest of the trunk. On light percussion it can often be found that the greater curvature reaches down to or even much below the level of the umbilicus. Most characteristic of all is the appearance of waves of visible peristalsis. These are best seen shortly after a feed has been given, and if they are lazy in making their appearance they can often be called forth by a little gentle massage of the upper abdomen. The waves of contraction begin at the left side, where a rounded swelling about the size of a golf ball appears below the left costal margin and travels somewhat leisurely across the abdomen in an oblique direction and downwards and to the right. Before one wave has disappeared another makes its appearance, so that an hourglass-shaped swelling can be seen reaching across the abdomen. Sometimes three waves are present simultaneously. These peristaltic waves are often so marked as to attract the attention of the mother or nurse, and I have even been assured in one case that the swelling they caused was so great that it could be seen through the clothes.

This vigorous peristalsis is pathognomonic in the child, as in the adult, of some obstruction at the outlet of the stomach, and is therefore of great diagnostic value.

Curiously enough it does not seem to cause much, if any, pain, and the child will often lie quite placidly whilst the waves course in succession across the organ.

The third sign is the presence of a pyloric tumour. It should be felt for to the right of the middle line, and one should try to get underneath the stomach and compress the pylorus against the right side of the vertebral

column. As the late Dr. George Carpenter pointed out, it is often best felt by trying to pinch up the deeper structures. Sometimes the pylorus is so tucked up under the liver that no tumour can be felt, but if one is successful it feels like a hard lump almost the size of a hazel nut, but somewhat elongated.

Ibrahim⁶ has pointed out that there is often a pronounced diastasis of the recti muscles in these cases, but this is no doubt due simply to the distension of the upper abdomen and is of no special value in diagnosis.

The combination of symptoms and signs, then, on which one relies for establishing the existence of congenital pyloric stenosis is: (1) Vomiting of the character already mentioned coming on within the first few weeks of life and accompanied by "pseudo-constipation" and wasting; (2) the presence of visible stomach peristalsis; (3) the existence of a pyloric tumour, if it can be felt.

PATHOLOGY AND PATHOGENY.

Morbid Anatomy.—The stomach in early life exhibits very clearly the division of the pyloric portion into the two segments known as the pyloric vestibule and the pyloric canal. The latter is particularly well marked, forming a tube about 3 in. long, which begins at the sulcus intermedius and ends at the pyloro-duodenal junction. If one examines the stomach from a case of congenital pyloric stenosis after death, one is at once struck by the enormous development of the whole pyloric canal, which forms a dense, almost cartilaginous, tube, the extremity of which projects into the duodenum in much the same way, it has been remarked, as the cervix uteri projects into the vagina. In addition, the wall of the pyloric vestibule is also thickened, and, indeed, that of the whole stomach to some extent, including even in some cases the lower end of the oesophagus.

It will be observed, too, that the mucous membrane of the pyloric canal is thrown into longitudinal folds, the ends of which often project into the vestibule.

On microscopic examination, it is found that the thickening of the wall of the pyloric canal is due almost entirely to an enormous development of the circular muscular fibres, although the longitudinal layer is also thickened, and the mucous and submucous coats to some extent. More careful examination shows that the muscle fibres are not only increased in number, but that each individual fibre is also broader than normal (Davidson).

So far all observers are agreed, but it is when one passes on to inquire how these changes are brought about that differences of opinion begin. Two distinct views are held:

1. That the thickening of the pyloric canal is to be regarded as a congenital malformation.
2. That it is due to overwork, the consequence of a long-continued spastic contraction.

We shall consider immediately which of these views best fits in with the clinical history and symptoms of the disease, but meanwhile we may examine them in greater detail.

1. A study of the "architecture" of the thickened muscle layer of the pyloric canal shows that it is certainly not to be regarded as a tumour formation (Wernstedt⁷), but rather as a hyperplasia. As Cautley⁸ has put it: "Nature in her extreme anxiety to provide an efficient pyloric sphincter has over-exerted herself, and produced too great a quantity of muscular tissue." Granted that this were so, it is difficult to see why obstruction should result, for the mere thickness of the muscular coat should not prevent its relaxing to allow the escape of the stomach contents when required. It has also been pointed out as an *a priori* objection to this theory that infants who suffer from congenital pyloric stenosis have no special tendency to exhibit malformations elsewhere, although it is well known that, as a rule, a malformation in one part of the body is apt to be accompanied by congenital abnormalities in others.

2. The supporters of the spasm theory have got to show: (a) How spasm may be set up; (b) that it is capable of leading to consecutive hypertrophy.

(a) As regards the cause of the spasm, it has been suggested that it is due to an inco-ordination in the pyloric mechanism—a sort of "stammer" of the pylorus—beginning, perhaps, in intrauterine life (Thomson⁹), whereby, when the waves of peristalsis reach it, instead of opening, as it should do, it remains closed. It has also been

attributed (Shattock¹⁰) to a hyperaesthesia of the mucous membrane in the neighbourhood of the pylorus, which induces reflex contraction—a "hysterical" pylorus, as it has been called. A third group of writers would attribute the spasm to a disorder of the chemical mechanism of the pylorus. They have tried to show that there is hyperacidity of the gastric contents in these cases, and that this hyperacidity induces the spasm (Engel,¹¹ Pfaunder¹²). This theory, however, will certainly not hold water, for (i) it has been shown that there is no real hyperacidity of the gastric juice in these cases at all (Miller and Wilcox¹³). (ii) Where hyperacidity exists it is almost certainly the result of the pyloric obstruction, and not the cause of it (Ibrahim,¹⁴ Feer¹⁵), being comparable to the hyperacidity found in cases of benign stenosis of the pylorus in adults, which is now admitted to be due to the continuous stimulation of the gastric mucous membrane by the retained contents. (iii) In any case experimental evidence (Cameron¹⁶) goes to show that acid on the gastric side of the pylorus induces relaxation of the pyloric sphincter, and not contraction of it.

(b) The assumption that prolonged spasm of the pyloric canal can lead to hypertrophy of its muscular coat also involves certain difficulties. It would be contrary to pathological law that this should happen, unless the muscle is not merely contracted, but contracted *upon* something, so that its fibres are kept in a state of "extension" at the same time. If it be admitted, however, that the pyloric canal in infancy is a regulating mechanism which controls the passage of the gastric contents from the stomach to the duodenum, this difficulty disappears, for in that case it would be comparable to the arterioles which regulate the flow of blood from the arteries to the capillaries; and it is admitted that prolonged contraction of the arterioles can lead to hypertrophy of their muscular coat.

A greater difficulty is to explain the rapid development of the hypertrophy by this theory. Operation within as short a time as three weeks from the first appearance of symptoms has shown the hypertrophy to be already present in a marked degree, and it is difficult to conceive how "overwork" of the muscle could have led to so great an overgrowth of it so quickly.

It will be observed that there are great difficulties in the way of accepting either the congenital hyperplasia or the spasm theory, whilst the compromise suggested by some—that there is both hyperplasia and spasm—has the difficulties of both combined.

Putting aside these *prima facie* objections, let us now inquire which theory best fits the clinical facts.

We have already seen that there is some difficulty in understanding how a mere hyperplasia of the sphincter can produce obstruction at all; but, waiving this objection, any acceptable interpretation of the pathological appearances must explain these two important facts in the clinical history of the disease:

1. That the symptoms are not truly congenital, but often only come on some weeks after birth.
2. That the condition is capable of being recovered from without operation.

Now it is obvious that the spasm theory fits in best with both of these facts. Granted that spasm can be brought about somehow it is really easier to understand its supervention some time after birth than its occurrence congenitally or *in utero*, whilst there is no reason why, when it passes off, a return to a normal state should not be possible. On the other hand, no plausible reason has been advanced for the delay in the appearance of the symptoms on the assumption that the obstruction is due to a congenital abnormality of structure, nor can we understand how, if that were the true pathology, cures without operation, such as I shall have to describe immediately, would be possible.

The *a priori* difficulties, therefore, are all in the way of spasm; the clinical difficulties in that of hyperplasia.

Meanwhile I have no alternative theory to offer, and the part of wisdom is to suspend judgement and wait for more information. In particular we want observations with the *x* rays which might throw light on the mechanism of the pylorus in such cases, and we want also more evidence as to the state of the pylorus in those which have recovered without operation and have subsequently died some months or years afterwards from some other disease.

TREATMENT, PROGNOSIS, AND COURSE.

I propose to consider the treatment of the disease and its prognosis together, because I believe that the mortality depends very much upon the line of treatment adopted.

When I first began to recognize cases of congenital pyloric stenosis I was deeply imbued with the idea—derived from one's preconception of the pathology of the affection and the writings of others—that surgical treatment alone offered any chance of success. In the first case which I recognized in private, therefore, I strongly advised that an operation should be performed. To this the parents objected, and the case passed from my care. I subsequently learned that the child had made a complete recovery under medical treatment carried out by someone else. I need not say that this experience made a deep impression upon my mind. I was, in fact, converted to a belief in the efficacy of medical measures in this disease, a belief which subsequent experience has only deepened and strengthened, for since that time I have seen 20 cases in private practice, 17 of which were treated at home, and all these recovered without operation. Of the 3 sent into hospital for treatment 2 died.

I have perhaps been unusually fortunate, but the experience of others has not been dissimilar. Thus Heubner¹⁷ records 19 recoveries out of 21 cases treated medically, and Starck¹⁸ 11 out of 12, whilst of 6 cases reported by Bloch¹⁹ and not operated upon all did well, and Bendix²⁰ has had no fewer than 30 recoveries out of 32 cases treated by medical measures alone.

The more enthusiastic advocates of operation have not hesitated to explain away such results as these by the assumption that the cases which recover under medical treatment are not true examples of congenital hypertrophic stenosis, but merely instances of pyloric spasm. Assuredly, however, this explanation will not do. Of my own cases, at least, I can say that they all exhibited the classical features of pyloric stenosis in a pronounced degree; and in most of them the severity of the condition was shown by the extreme illness of the child, which in many cases hung literally between life and death for weeks together. I do not propose to trouble you with particulars of these cases, but three²¹ of them have been published in detail by the practitioners under whose immediate care they were, and I would ask any unprejudiced person who will take the trouble to study their descriptions whether there can be any doubt that we were dealing with extreme examples of the affection.

I have given you the results of my own experience of cases treated in private, but I admit that hospital statistics present much more unfavourable reading. In the Children's Hospital, for instance, where medical treatment alone is adopted, our mortality has been 78 per cent. in a series of 64 cases. The great majority, too, of those we have had here have died. The greater mortality in hospital cases is partly to be explained, I think, by the fact that they tend to come later under treatment when the child's strength has already been greatly reduced by the constant vomiting. I have also an impression that these babies stand the conditions of hospital life badly. They seem to become "hospitalized," and even if one succeeds in stopping the vomiting, they hang on in a miserable marasmic condition and readily fall victims to intercurrent disease. This fact, however, does not invalidate the claims of medical treatment to be successful when begun early and carried out under favourable conditions.

One may contrast with the statistics I have put before you the results yielded by operative treatment. Of these the most favourable with which I am acquainted are those of Mr. Burghard²² who had 11 recoveries out of 16 cases—a mortality of 31 per cent. Cautley²³ reports 6 recoveries out of 7 cases operated upon in private, but a mortality of 4 out of 5 hospital cases. Of 23 cases under the care of Dr. John Thomson²⁴ which were treated by operation (most of them by Mr. Stiles) 15 died—a mortality of 65 per cent. We will probably not be far from the truth if we accept Ibrahim's estimate of 50 per cent. as the probable mortality amongst all cases submitted to operation.

I fully admit that such statistics do not do justice to the surgeon, for no doubt in many of the cases operation was only performed as a last resort and when the chances of recovery in any event were but small, but they are the only material available for forming a judgement.

I shall return again later on to the question of operation, but meanwhile I propose to describe briefly the medical treatment usually adopted. The essentials are careful feeding and daily lavage of the stomach. As regards the kind of food, breast milk is probably the most suitable, but owing to the frequent intervals at which food must be given suckling is impracticable. The breast milk may be drawn off and given by a spoon, but usually this practice results in a speedy cessation of the appearance of milk and has to be abandoned. In that case, or if, as often happens, the child has already been weaned before the diagnosis has been made, thoroughly peptonized and diluted cow's milk is the best substitute, but I have also used dried milk, such as Glaxo, and mixtures of whey, egg-albumen and lactose, supplemented occasionally by somatose.

The marked fat retention, which has been found to take place where there is a block at the pylorus,²⁵ has induced some to exclude all fat as much as possible from the food. It has not been found, however, that this is of any great benefit in practice, and if the stomach is being regularly washed out I believe it to be unnecessary.

One must feel one's way as to the amount to be given at a time, beginning, perhaps, with an ounce every hour, and gradually increasing the quantity and intervals as the vomiting subsides. I think this plan is much better than feeding at long intervals, as some advise. After the feed the child should be kept lying flat and not moved about, in order to minimize the chance of vomiting.

I am convinced of the great value of regularly washing out the stomach once or twice a day, according to the frequency of the vomiting. That it is possible to treat cases successfully without this the results obtained by Heubner, who does not employ it, clearly show, but it certainly adds to the comfort of the patient, and, by lessening the frequency of the vomiting, diminishes exhaustion and facilitates sleep. By removing retention products and mucus it also lessens the catarrh and swelling of the mucous membrane, and may in this way favour the escape of the contents through the narrowed pylorus.

The technique of gastric lavage in infancy is now so well known that I need not describe it.

As subsidiary measures, I believe in the value of the German plan of applying continuous poultices to the epigastrium, and great care should be taken, as in all underfed babies, to maintain the body heat.

If vomiting be severe and the amount of food which passes out of the stomach very small, fluid should be supplied per rectum in the form of normal saline enemata, 2 to 3 oz. being given at a time twice or thrice daily. These are of great value in supplying a sufficiency of fluid to the tissues and in promoting elimination of waste products by the kidneys.

Drugs are of little help. I have tried most of them—alkalis, on the assumption that there is hyperacidity of the gastric contents; atropine and bromides as antispasmodics, carminatives and antiseptics—but the only one which has ever seemed to be of any use is opium. It may be given in minute doses (say, $\frac{1}{16}$ minim before each feed), and seems sometimes to help in allaying the vomiting, besides diminishing restlessness and promoting sleep, but I believe one can get on quite well without it. It is more highly esteemed in Germany than it is here, perhaps because lavage is less systematically employed in that country.

Under such a plan of treatment as the above the vomiting usually ceases quite soon; but it will be found that in spite of this the infant does not gain weight; indeed, it may even go on losing, and there may be little or no progress for weeks, sometimes not until the fifth month. It is at this period that one's faith in the efficacy of the treatment is most sorely tried. With perseverance, however, improvement finally sets in, and often quite suddenly the child starts to gain weight. This sudden change for the better is often very striking, and has also been remarked by Heubner.

Sometimes, on the other hand, and but all too often in hospital cases, the issue is not so happy, and, in spite of the cessation of vomiting and the consumption of a fair amount of food, the weight refuses to rise, and the child eventually succumbs apparently as the result of an "alimentary toxæmia" which seems to be the consequence of an atrophy of the bowel with impairment of the

power of absorption, probably brought about by the long period of virtual starvation through which the infant has passed. In other cases, again, one is disappointed by a sudden and inexplicable collapse just when all seemed to be going well.

What, it may be asked, is the ultimate result in those cases which recover under medical treatment? Are they entirely cured, or is there only a state of compensation established with some persistent narrowing of the pylorus and dilatation of the stomach? In some cases, at least, I believe that the cure is both complete and permanent. At all events I have examined some of my cases—and those of the worst type—five years after treatment, and found them to be perfectly healthy children, with no sign of gastric disorder or dilatation of the stomach. I know that it seems incredible, when one examines the stomach from a typical case after death, that the almost cartilaginous thickening of the pylorus which is present should entirely disappear, and a complete return to a normal condition be brought about. But we have no right to set a limit to the powers of the *vis medicatrix Naturae*, and I can only repeat that, as far as clinical observation can determine it, such a restitution to the normal does, in fact, take place. But not, probably, in all; I have certainly seen one case in which three years after apparent recovery the stomach was still somewhat dilated, and I was told that the child was subject to "stomach upsets," although well nourished and otherwise in good health. Some of the cases described by Russell²⁶ and Maylard,²⁷ in adults, and to which Dent²⁸ also has referred, may, perhaps, have originated in this way, although others probably belong to the Landerer-Maier group, and it is quite certain that compensation can be established with a gain in weight and disappearance of all symptoms even although some degree of pyloric obstruction, as shown by the persistence of visible peristalsis, persists. Most cases, indeed, pass through this phase, although in the great majority, probably in all, the peristalsis finally disappears. What we really need to determine the question is more *post-mortem* evidence as to the state of the stomach some years after recovery under medical treatment. Such evidence is not yet forthcoming, but we shall no doubt get it in time, and meanwhile the fact of clinical cures is undoubted whether they be real anatomical cures or no, and that, after all, is all we want.

To return, then, to the question of surgical treatment. Is operation never to be recommended in these cases? In practice I think not. I fully believe that there may be some cases which would have been saved by operation that die without it; but I know of no method by which such cases can be recognized, for one can never say of any given case that it is so bad that operation alone can give a chance of life. Of that experience has thoroughly convinced me. As a practical policy, therefore, one is obliged either to operate upon all cases in which the symptoms and signs are well marked or upon none of them, and that the former plan would result in a much heavier mortality than the latter I have not a shadow of doubt.

The only alternative to this dilemma is to treat all cases medically at first, and then to advise operation upon those that do not show any improvement after a certain lapse of time—three or six weeks have been given as the limit beyond which the surgeon should be called in. The objection to this plan, however, is that it means operating at a time when the child's strength is already much reduced and when success is not likely to be achieved, whereas, on the other hand, my own cases show that no case of the disease can be pronounced to be incurable by medical means until the patient is dead.

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An Address

ON

PRIZES AND PERFORMANCES.

DELIVERED AT THE OPENING OF THE MEDICAL SESSION
AT ST. GEORGE'S HOSPITAL, ON OCTOBER 1ST.

BY

S. SQUIRE SPRIGGE, M.D. CANTAB.,

EDITOR OF THE "LANCET."

GENTLEMEN,—I will not waste your time by expressing at any length my sense of the high honour that has been done me in allowing me to present the prizes at my old medical school, and to address you at the beginning of the new session. It would be ungracious of me, however, not to acknowledge my feeling in the matter, as the position in which I find myself is one so totally unexpected—I am, then, surprised at myself for being in this position, and I know that a good many of you, having regard to the scientific eminence of my distinguished predecessors in this function, and to their services to St. George's Hospital, must share my sentiment.

The ceremony which we have just witnessed—the award to the successful students at the medical school of the prizes which their industry, their ingenuity, and perhaps a happy blend of both qualities, which I will call their wisdom, have enabled them to win—I will take as a suggestion that to look a little into the significance of prize-giving might be interesting. All the year round, and in every class of academic, institutional, and professional training, men are winning prizes, losing prizes, competing for prizes or scratching for those competitions; and a system that is so universally in employment among us may easily become accepted by us in a mechanical way, may cease to have a reason which appeals to us, and be merely regarded as one of those things that happen in the course of education. It is these frequent and general features in the scheme of life that pass unnoticed by us because of their very familiarity. If familiarity with such a ceremony as we have just seen were all that were needed to grasp its full significance we should all know all about prizes—their uses, their abuses, and their relation to the spread of wisdom generally, and to the efficiency of students and scholars in particular. Our opinion upon the matter would be unanimous, and there would be no need for any discussion. But although we cannot conceive any system of professional training without them and their attendant troubles—namely, examinations—such clear comprehension is not ours with regard to prizes and the principle of rewarding early merit in examination by gifts, for among my hearers it is perfectly certain that there are those who have expressed opinions of diametrically opposite natures on the value of prizes, upon the systems under which they are awarded, and upon the utility of examinations generally. I am certain that many of us have said that prizemen are too often found not to fulfil their early promise. This is when we hear of the insignificant future which some brilliant student has made for himself—perhaps when we find the first mathematica scholar in our batch at school as an under-master in a second-rate seminary, or notice the best classic of his year at our college dozing away a valueless existence in front of a club fire, or using his recollection of the classical dictionary in an acrostic competition. It is equally certain that many of us, in congratulating a prize winner, or perhaps in the case of those of us who are middle-aged, in congratulating the father of a prize-winner, have said that nowadays, in a world of stress and competition, there is nothing like making a good start. And some of us have certainly said both these things, suiting our words to the circumstances. And the same diversity of view is expressed by us with regard to the value of examinations as a test of present merit or as a guide to prognostications of future success. Inevitably those who profess to hold cheaply the winning of prizes believe that the easy and rapid passing of examinations must not be credited as furnishing any sure ground for future honours.

There is, then, a considerable difference of estimate with regard to the value of prizes and the significance of passing