restoration of blood loss is more rapid in cases of haematemesis treated by the Meulengracht method. The effect of the more rapid restoration of the vitamin C nutrition in the present series of cases cannot as yet be estimated. Whether peptic ulcers heal more soundly, whether haemorrhages recur less frequently, and whether the blood regeneration is still more rapid in patients whose vitamin C nutrition as well as their general nutrition is maintained at what is regarded as physiological level are questions that have still to be answered.

It appears from the results of this investigation that the purée diet given to these cases of haematemesis during the winter months did not contain enough vitamin-C-rich foods to restore rapidly their vitamin C deficiency. The addition of preparations or foods rich in vitamin C is thus necessary. Harris (1928) recommended such additions to the diet of patients with peptic ulcer, and Witts (1937) has recently added adequate amounts of orange juice, marmite, and cod-liver oil to the purée diet given in cases of haematemesis and melaena. As regards the amount of ascorbic acid usually required by patients with haematemesis to replenish the vitamin C stores of their tissues, 200 mg. daily by mouth will as a rule accomplish this in ten to fifteen days. After this a maintenance dose equivalent to 30 to 60 mg. is advisable. This could be achieved very satisfactorily by giving 60 to 100 c.cm. of orange juice, or smaller amounts of tomato juice, together with additions of fresh milk, vegetables, and potatoes.

Summary

1. The state of vitamin C nutrition of fifteen cases of haematemesis and melaena was investigated by the estimation of the excretion of ascorbic acid in the urine.
2. Thirteen of the fifteen cases were found to be in a condition of vitamin C subnutrition or "subclinical scurvy." This was of a severe degree in seven of the cases.
3. Three cases of peptic ulcer uncomplicated by haemorrhage were similarly found to be in a state of vitamin C subnutrition.
4. Estimations carried out on three healthy control subjects did not reveal any vitamin C subnutrition.
5. The addition of 200 mg. of synthetic ascorbic acid to the purée diet given to the cases of haematemesis led to the correction of the nutritional defect in ten to fifteen days, and produced no untoward symptoms even when started four days after the actual bleeding.
6. Ascorbic acid, 200 mg. daily by mouth for ten to fifteen days, is recommended to be followed by the liberal addition of orange juice (60 to 100 c.cm.) to the diet of these patients.
7. The general indications for correcting this nutritional defect are discussed.

It is a pleasure to record my indebtedness and gratitude to Professor E. Meulengracht for hospitality and direction. My thanks are also due to Dr. Knud Secher for permission to investigate cases under his care, and to Roche Products Ltd. for a generous supply of redoxon and ascorbic acid.

REFERENCES

- Abbasy, M. A., Harris, L. J., Ray, S. N., and Marrack, J. R. (1935). *Lancet*, 2, 1399.
- Archer, H. E., and Graham, G. (1936). *Ibid.*, 2, 364.
- Davidson, P. P. (1928). *J. Amer. med. Ass.*, 90, 1014.
- De la Viesca, P. (1935). *Arch. Verdaugsk.*, 58, 22.
- Göthlin, G. F. (1931). *Scand. Arch. Physiol.*, 61, 225.
- (1934). *Nature*, 134, 569.
- Harde, E., Rothstein, I. A., and Ratish, H. D. (1935). *Proc. Soc. exp. Biol., N.Y.*, 32, 1088.
- Harris, L. J., Ray, S. N., and Ward, A. (1933). *Biochem. J.*, 27, 2011.
- Abbasy, M. A., Yudkin, J., and Kelly, S. (1936). *Lancet*, 1, 1488.
- Harris, S. (1928). *J. Amer. med. Ass.*, 91, 1452.
- Hawley, E. E., Frazer, J. P., Button, L. L., and Stephens, D. J. (1936). *J. Nutrit.*, 12, 215.
- Hurst, A. F., and Ryle, J. A. (1937). *Lancet*, 1, 1.
- Johnson, S. W., and Zilva, S. S. (1934). *Biochem. J.*, 28, 1393.
- Kelly, S. (See Harris, L. J., Abbasy, M. A., Yudkin, J., and Kelly, S.)
- Lund, H., and Lieck, H. (1936). *Scand. Arch. Physiol.*, 74, 255.
- McCarrison, R. (1921). *Studies in Deficiency Diseases*, pp. 95 and 225, London.
- Meulengracht, E. (1935). *Lancet*, 2, 1220.
- (1936). *Wien. klin. Wschr.*, 49, 1481.
- Platt, R. (1936). *Lancet*, 2, 366.
- Schiödt, E. (1936). *Amer. J. med. Sci.*, 192, 163.
- (1937). *Ibid.*, 193, 313.
- Schroeder, H. (1935). *Klin. Wschr.*, 14, 484.
- Schultzer, P. (1934). *Acta med. scand.*, 81, 113.
- Smith, D. T., and McConkey, M. (1933). *Arch. intern. Med.*, 51, 413.
- Witts, L. J. (1937). *British Medical Journal*, 1, 847.
- Wood, P. (1935). *Lancet*, 2, 1405.

SQUINT AND HETEROPHORIA

WITH SPECIAL REFERENCE TO ORTHOPTIC TREATMENT*

BY

W. H. McMULLEN, F.R.C.S.

Consulting Ophthalmic Surgeon, Charing Cross Hospital

The rational treatment of any disorder must be based upon the study of its aetiology, of its pathology, and of its clinical features and course. The aetiology of concomitant strabismus is a problem as yet unsolved. We have hypotheses in plenty, but none that gives a completely satisfactory explanation of the development of squint. Pathological changes are absent, except perhaps occasional hypertrophy and shortening of one muscle with thinning and lengthening of its opponent. It is only in the study of the clinical features and course of the disorder that we obtain indications for treatment based on exact observation. The treatment of squint has, however, been largely influenced by the theories held as to its aetiology, so it seems well to begin with a short critical review of these theories.

In ancient times very fantastic ideas prevailed as to the cause of squint, and it is a curious fact that one of the first writers to propound anything like modern views on the subject was the notorious charlatan, the Chevalier Taylor, who flourished in the eighteenth century (1702-72). He said that squint was nothing but a misdirection of the eyes on looking at objects; that if such misdirection were produced artificially by pressing on the eye diplopia resulted, because the image fell on an abnormal area of the retina; that diplopia did not occur in squint because many squinters saw better with one eye than with the other, or if they saw equally well with both they used one eye more than the other, and therefore directed its axis on the object. He explained that an image was indeed formed in the squinting eye, but it was so weak that it did not hinder clear vision with the other, and that with practice one learned to disregard a second image. Elsewhere he made more questionable statements, as that a child squinted owing to association with squinting persons, or from too many objects being brought to his notice at the same time, which caused him to look simultaneously in different directions.

The theories most generally held in recent times are: (1) The muscular theory; (2) the accommodation theory; and (3) the theory of defective fusion faculty. In addition to these one has to consider the influence of anisometropia, of heredity, of nervous factors, and of social conditions.

* Read in opening a discussion in the Section of Ophthalmology at the Annual Meeting of the British Medical Association, Belfast, 1937.

The Muscular Theory

Until the publication of Donders's theory in 1866 a muscular defect was generally held to be the essential cause of squint. Many have continued to support this theory, but varying views have been held as to the exact nature of the defect—for example, congenital or acquired paresis, a developmental anomaly in the muscles, innate imbalance, or imperfect nerve control. In favour of the theory it is noted that slight anatomical abnormalities are sometimes found at operation, especially undue thickness of the internal rectus and thinness of the external; but some consider that such abnormalities are always secondary, due to long-standing deviation. The hypothesis receives some support from the observation that cases which at first appear to be obvious examples of congenital palsies may develop in time into concomitant squints. Further support has been received from the recognition by modern methods of examination that there is a slight degree of incomitance in a considerable proportion of apparently concomitant squints. Gayer Morgan found this in 20 per cent. of a series of cases. The frequent onset of squint after acute illness, such as measles or whooping-cough, has been held to support the muscular hypothesis. It is suggested that slight degrees of toxic paralysis may be caused by these diseases, comparable to post-diphtheritic paralysis. I am not aware, however, that paralyzes are known to occur in other parts of the body after the common acute fevers, other than diphtheria.

There are weighty arguments against the theory that a congenital muscular or neuro-muscular defect is the essential cause. In the great majority of cases no defect of mobility of the squinting eye can be detected. It is true, as already mentioned, that a considerable minority of cases at first sight appearing concomitant are found to be slightly incomitant, and that precise measurements of the eye movements in young children are impossible; but if a squint is concomitant over a full range of movements, as tested by a modern orthoptic instrument, it seems most improbable that paresis is the cause of the squint. It is difficult, on the hypothesis of congenital muscular defect, to account for the common age of onset. It is said that most infants do not acquire full co-ordination of eye movements for several months after birth, but it is generally agreed that good co-ordination is normally developed by the time a child is a year old, so one would expect the effects of congenital anomalies to become manifest by that time. Yet the common age of onset is later.

Another objection to the purely muscular theory is that an obvious congenital or very early acquired paralysis may be present without leading to constant strabismus. For instance, in some cases of congenital paresis of abduction the eyes are ordinarily straight, though the head may be held turned slightly to one side. Still more striking are the typical cases of ocular torticollis.

It is probable, therefore, that while the presence of a slight muscular weakness or imbalance may often be a factor in the aetiology of squint it is not alone sufficient to cause it.

The Accommodation Theory

The theory of Donders is so familiar that it is unnecessary to describe it. It was the first to offer a rational explanation of the occurrence of strabismus in the absence of recognizable muscular anomaly; but while no one now doubts that errors of refraction may play an important part in strabismus, few, if any, consider that this theory affords a full explanation of its development.

The most convincing evidence in its favour is that some cases are completely cured by correction of errors of refraction without any other treatment. In many others the angle of squint is reduced. Strong support was given to the hypothesis in a paper published by Chavasse in 1930. In a comparative study of 200 normal children and 200 squinters he found a very striking correlation between the degree of hypermetropia and the liability to squint. The age of onset is explained on the accommodation theory by the assumption that in infancy and early childhood little effort is made to focus near objects clearly, and that squint develops at the time when a child begins to take an interest in pictures, etc., close at hand, and so makes greater demands upon his accommodation.

The objections to the theory are many. Most hypermetropes, even of high degree, do not squint. In myopes convergent squint is commoner than divergent: E. Thomson found among squinting myopes thirty-eight convergent to twenty-seven divergent, a ratio of 1.4 to 1.0. In hypermetropic squinters the convergence is usually greater than that normally associated with the accommodation required to overcome the error of refraction.

The difficulty of accounting for the excess of convergence in relation to accommodation has been met by the suggestion that at the onset of squint a child gets diplopia, and that with the comparatively slight convergence attributable to excessive accommodation the images are but slightly separated, and suppression of one image is difficult. Further convergence occurs to separate the images more widely, and so facilitate suppression and overcome diplopia. There is no doubt that disproportion between accommodation and convergence is easier in the direction of excess of convergence than in that of deficiency; it is easier to converge without accommodating than to accommodate without converging; so it is reasonable to suppose that a child may overcome diplopia more easily by excessive convergence than by relative divergence, although the latter would enable him to maintain binocular vision. It is probable that in some individuals there is innate disproportion between convergence and accommodation. Certainly some children, even when their errors are fully corrected, squint much more when looking at near objects.

The Fusion Theory

One of the most important events in the history of our subject was the publication in 1903 of Worth's book on squint, in which he expounded the theory that the essential cause of squint was a defect in the fusion faculty. This hypothesis has been widely accepted, and has played an important part in stimulating the study of orthoptic methods of treatment. Nevertheless it remains unproved. It is certain that the faculty of binocular vision is defective in most squinters; but this defect may well be a result and not the cause of the squint. It has been found in orthoptic clinics that when good binocular vision has been developed by training it may be rapidly lost, in cases with much persisting heterophoria, if exercises are stopped too soon. It seems reasonable to suppose that in very early life binocular vision may be even more easily lost. Worth himself admitted that hypermetropia might sometimes cause a squint in early infancy, before the fusion faculty had developed, and that if the squint were allowed to persist the development of the faculty might be prevented. On the other hand, one occasionally sees cases of squint in which the fusion faculty is quite good. Worth's original assumption of the existence of a fusion centre in the brain, for which there was no anatomical or clinical evidence, is no longer regarded as essential to the theory.

Anisometropia and Squint

Supporters of all the main theories as to the essential cause of squint agree that inequality of vision in the two eyes favours its development. It is not easy to assess the importance of this factor. Anisometropia is common in squinters, but most persons with anisometropia do not squint, even when the visual acuity in the worse eye is much below normal. Many years ago, when investigating a series of cases of anisometropia, I was surprised to find what good binocular vision was possessed by some individuals with very unequal eyes. I found in one case excellent stereoscopic vision with a difference of nearly 6.5 D between the two eyes. Another had good stereoscopic vision with a difference of 4.25 D, although in the worse eye vision without glasses was less than 6/60 and with correction was only 6/18: he had never worn glasses, vision in the better eye being 6/6.

Heredity in Squint

The influence of heredity has been variously estimated. Mackenzie noted its importance. Worth obtained a history of squint in parent, grandparent, brother, or sister in 51 per cent. of 1,373 cases. The question of heredity is closely linked with that of nervous or psychological factors. Cosse, including certain unstable mental characteristics, traced inheritance in as many as 74 per cent. of his cases.

Nervous Factors in Squint

The onset of squint is very often attributed to a fright, a fall, or some other nervous shock. Inman, especially, has emphasized the importance of psychological factors. It is significant that one is often told that the squint is worse when the child is with strangers and feeling shy, or when overexcited or tired. Lyle and Jackson, in their recently published book on practical orthoptics, quote cases illustrating the importance of psychological factors. In this connexion it is interesting to note that Cantonnet and Filliozat lay great stress upon the conscious mental effort required of the patient for successful training, on the patient's will to be cured, and upon the encouragement given by relatives and friends. Personally I think we should not ignore the possibility that in some cases at least squint may be a functional nervous disorder comparable to a hysterical pose, and that the advice of a competent psychiatrist may be helpful in treatment.

Influence of Environment

I think that most ophthalmic surgeons find that squint is commoner among children attending school clinics and hospitals than among private patients. The most striking demonstration of the relation between social conditions and prevalence of squint was given by Ernest Thomson in a study of the incidence among 32,000 children attending schools classified according to the social status of the parents. The proportion of squinters was found to increase from 1.9 per cent. in the best class to 3.5 per cent. in the poorest.

Amblyopia and Squint

The amblyopia associated with squint presents a difficult problem. It is common ground that congenital amblyopia must favour the development of squint by hindering good binocular vision. But opinions differ

greatly as to the frequency of congenital amblyopia. In the past many believed that the amblyopia of a squinting eye was always due to some developmental defect or to some lesion in very early life, such as macular haemorrhage at birth, which disappeared leaving no trace, and that there was no such thing as amblyopia ex anopsia. The very common presence of retinal haemorrhages in the newly born was shown by the investigations of F. Juler published in 1926. Observations confirming and extending those of Juler were communicated to the Ophthalmological Section of the Royal Society of Medicine last November by Dr. Frances Richman of Brooklyn, N.Y. She hopes to be able to follow up many of her cases to determine whether there is any relation between such haemorrhages and squint or amblyopia. Juler has been able to follow up only five of his cases. In none of these did he find either squint or amblyopia.

The possibility of amblyopia ex anopsia is now generally accepted, although opinions differ still as to its frequency relatively to the congenital form. The great supporter in Great Britain of the theory of amblyopia ex anopsia was, of course, Claude Worth. He believed that very few cases of amblyopia in squint were congenital, and accepted as such only those in which there was scarcely a possibility of acquired amblyopia. I find it difficult to accept without question Worth's criteria for the discrimination of congenital and acquired amblyopia, but his careful analyses of a large number of cases prove very conclusively some facts of great practical importance. He showed that the intensity of the amblyopia and the difficulty of curing it increased in direct proportion to the fraction of the child's life during which it had squinted before treatment was commenced, also that the earlier the age at which constant unilateral squint developed the more rapidly and the more seriously did the vision of the squinting eye deteriorate. He found that amblyopia ex anopsia seldom occurred to any great degree when squint commenced after 6 years of age. A valuable study by F. Juler of the vision after operation in cases of unilateral traumatic cataract supports Worth's views. Juler found that none of the eyes which became cataractous before 5 years of age retained a final central acuity of 6/60, while of those developing cataract after 6 years of age the great majority had 6/12 partly or better. The development of amblyopia ex anopsia is one of the few things connected with squint that have been proved by experiment, albeit accidentally. Worth observed development of amblyopia in cases in which treatment was neglected, and others have found that unduly prolonged occlusion may cause amblyopia in an originally fixing eye.

I think there is now fairly general agreement that in most cases amblyopia can be prevented or cured if treatment is undertaken early enough and efficiently carried out. Probably we should see little amblyopia in squint if every child were brought under competent treatment within a few weeks of the first appearance of squint.

In this very brief and inadequate review of the problem of the aetiology of squint I have considered many factors that may be to a greater or less extent responsible. My conclusion is that we cannot say that any one of them is the essential cause of the disorder. It may be that there is some one underlying cause in all cases, but I think it more probable that squint is a clinical condition due to many causes, differing in character and importance in different cases. Probably at the present time progress in treatment will be furthered rather by the more exact study of the clinical features and course of the disorder than by the elaboration of theories as to its aetiology.

Development of Orthoptic Treatment

The most notable development in the treatment of squint during the past decade has been the elaboration of methods of orthoptic treatment. The idea of remedying squint by exercises is no new thing. Roux, in 1814, described the cure of his own external squint by exercises, but it is said that the cure was not complete. Mackenzie in 1841 referred to the use of Wheatstone's stereoscope. But Javal is usually, and I think rightly, regarded as the true pioneer of orthoptic training. Javal, who used a stereoscope chiefly, was followed in France by Rémy, inventor of the diploscope, and many others. In Great Britain the names of Maddox and Worth stand pre-eminent in this connexion.

The development of orthoptic treatment in France and Great Britain has been largely influenced by the general adoption in the two countries of two different instruments—in France the diploscope of Rémy, in Great Britain the amblyoscope of Worth. Exercises with the diploscope call for a relatively high degree of intelligence and power of concentration on the part of the patient. Hence we find that French writers generally advise commencement of training at a later age than British. Cantonnet and Filliozat consider that it is seldom of any use to begin training before the age of 7. They say that better results are obtained in a girl of 17 than in a child of 6 or 7, because her will is stronger and her intelligence more developed, and that there is fair hope of success from training up to the age of 30 to 35 years. In the system of training described by these writers mental concentration and effort of imagination play a very important part. Worth, on the other hand, claimed that it was quite easy to use the amblyoscope at 3 to 3½ years of age, that fusion training of children under 5 or 6 years of age was in suitable cases easily accomplished, and that after the age of 7 the results were seldom worth the time and trouble that they cost. Experience in the orthoptic clinic at the Royal Westminster Ophthalmic Hospital has led Lyle and Jackson to the conclusion that probably 6 years is the ideal age at which to commence treatment by exercises, but that good results are obtainable in some cases up to early adult life.

There is no doubt that much depends upon the character and intelligence of the patient and upon the skill and personality of the trainer. Many, including myself, who attempted treatment on the lines advocated by Worth failed to obtain satisfactory results and became sceptical as to its value. A few, however, maintained faith in it. In the year 1914, on the suggestion of William Ettles, a much improved form of amblyoscope was designed by Mr. J. R. Howard, now a member of the firm of Messrs. Curry and Paxton. This instrument was named the synoptoscope. It was the prototype of all the improved amblyoscopes now used for training under the names of synoptoscope (the original instrument with improvements), synoptophore, and orthoptoscope. Only one synoptoscope was constructed until after the war, when Mr. Howard brought out an improved model, which he showed to me soon after its introduction. At that time I had little belief in the value of orthoptic treatment and, I regret to say, took little interest in the instrument. Maddox was one of those who had continued to use the amblyoscope, and was one of the first to employ the synoptoscope. Maddox was very largely responsible for the recent development of orthoptic treatment. He taught his daughter the methods he employed, especially the use of his cheiroscope and of the synoptoscope. Early in 1929 Miss Maddox came to London and started a

private clinic. In 1930, on the suggestion of the medical staff, the board of management of the Royal Westminster Ophthalmic Hospital invited Miss Maddox to start an orthoptic clinic at the hospital. She accepted the invitation, and under her guidance, with the close co-operation of the medical staff, the clinic soon gained wide recognition as a centre for orthoptic treatment and as a school of instruction in methods of training. Other clinics and training schools were soon started, and it became obvious that something should be done to ensure that persons undertaking orthoptic training were properly qualified for the task. An Orthoptic Board was therefore established, consisting of representatives from hospitals with orthoptic clinics. This board has prescribed certain educational standards for entrants to orthoptic training schools and prepared a syllabus for the course of instruction extending over a minimum period of a year. At the end of that period students are examined and successful candidates are granted certificates, which may be accepted as evidence that the holders have been adequately trained and have satisfied competent examiners that they are fitted to undertake training. Candidates are required to give an undertaking to work only in collaboration with qualified medical practitioners.

BIBLIOGRAPHY

- Cantonnet, A., Filliozat, J., and Fombeure, G. (1934). *Strabismus*, London.
 Chavasse, B. (1930). *Trans. Ophthalm. Soc. U.K.*, **50**, 168.
 Inman, W. (1921). *Ibid.*, **41**, 181.
 Juler, F. (1921). *Ibid.*, **41**, 129.
 — (1926). *Ibid.*, **46**, 42.
 — (1937). *Proc. roy. Soc. Med.*, **30**, 279.
 Lyle, K., and Jackson, S. (1937). *Practical Orthoptics in the Treatment of Squint*, London.
 Mackenzie, W. (1840). *Diseases of the Eye*, third edition, p. 283, London.
 — (1841). *Ibid.* (Appendix), p. 27.
 McMullen, W. H. (1912). *Ophthalm. Rev.*, **31**, 353.
 Morgan, O. Gayer. Personal communication.
 Richman, Frances (1937). *Proc. roy. Soc. Med.*, **30**, 277.
 Taylor, the Chevalier, cited by Coats, G. (1915). *Roy. Lond. Ophthalm. Hosp. Rep.*, **20**, 1.
 Thomson, Ernest (1924). *Trans. Ophthalm. Soc. U.K.*, **44**, 240.
 — (1924a). *Ibid.*, **44**, 250.
 Worth, C. (1903). *Squint*, London.

W. Heim (*Dtsch. med. Wschr.*, August 27, 1937, p. 1321) gives an account of the 283 cases operated on for perforated gastric or duodenal ulcer at a hospital in Berlin in the period 1920 to 1936. About five-sixths of all the patients had suffered from gastric symptoms for more than six months before perforation. The stomach was the seat of the ulceration in 213 cases, and the distribution of the cases over the years under review was fairly even. But during this period there was a notable rise in the number of cases of duodenal ulcer. Only twenty-one of the 283 patients were women, and the immediate mortality was 36.7 per cent. (107 deaths). The mortality showed no sex inequality, but it rose after 45 and was as high as 71 per cent. of the cases operated on more than sixteen hours after perforation. Eighty-one patients were re-examined after discharge, and forty-five were found to be symptom-free. Seven had required another operation, and thirty could follow their occupations only when they dieted strictly. The patients rendered symptom-free were for the most part elderly, whereas post-operative disturbances were comparatively common among the patients under 30. The shorter the duration of symptoms before perforation, the higher was the proportion of complete cures after operation. One of the lessons emerging from this follow-up study was that the success of any operation for perforated gastric or duodenal ulcer largely depended on the conscientiousness with which after-treatment was carried out, whether dietetic or medicinal.