seems not to have been secondary to any excessive haemolysis, but to have been primary in the bone marrow, and I would regard it as a myelogenic form of splenic anaemia of adults, doubtless allied to what French authors have termed "anémie splénique myéloïde." Therefore the case cannot be adduced as an example of pernicious anaemia being followed by or becoming transformed into leukaemia, though it is probable that similar cases have been erroneously supposed to illustrate such a course of events. It is also highly probable that similar cases, owing to enlargement of spleen and liver, have been clinically regarded (in spite of the poikilocytosis) as examples of Banti's disease.

In the German cases, I believe, enlargement of haemolymph glands was not noted.

It is very tempting to suggest that in the present case both the spleen (haemal gland) and the prevertebral haemolymph glands were actively engaged in supplementing the erythrocyte-forming functions of the diseased bone marrow, but I have no conclusive evidence to support such a supposition. From what has been lately written on the subject⁶ it seems that no really satisfactory evidence has been brought forward to show that in adults erythrocytes are produced in the spleen and haemolymph glands either in health or in disease.

NOTES AND REFERENCES.

¹ W. von. Leube, *Deutsche Klinik*, Berlin, 1902, No. 42; and J. Arneith's haematological account of Leube's case in *Deut. Arch. für klin. Med.*, Leipzig, 1901, vol. lxxix, p. 331; also H. Luce, "Ueber Leukanämie," *Deut. Arch. für klin. Med.*, Leipzig, 1903, vol. lxxvii, p. 215. "Leukanæmia" is obviously a term coined out of "leukaemia" and "anaemia," and will, I believe, be found well suited as a clinical term for the present class of cases. ² This comparison of the appearance of very long prevertebral haemolymph glands to leeches has been borrowed from Dr. H. D. Rolleston. ³ I do not use the term "large mononuclear cell" in the limited special sense often attached to it. ⁴ No Prussian blue reaction was obtained by the "long" method employed by Abbott for pieces of tissue which had been lying in formalin (*Vide Trans. Path. Soc. London*, 1900, vol. li, p. 76). ⁵ *Vide* Vaquez and Aubertin, "Nature de l'Anémie Splénique Myéloïde," *Société de Biologie*, Paris, May 14th, 1904. ⁶ See especially "The Pathology of Pernicious Anaemia with special reference to the changes occurring in the Haemolymph Nodes," by Dr. A. S. Warthin, *American Journ. Med. Sci.*, 1902, vol. cxxiv, p. 674.

NOTE ON THE ANTI-HAEMOLYTIC (HAEMOSOZIC) PROPERTIES OF NORMAL URINE.

BY

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NORMAL aseptic human urine does not, as a rule, dissolve human red blood corpuscles, even when the mixture of urine and red blood corpuscles is kept for many days at the temperature of the room (20° C. to 30° C.). The red blood corpuscles of many pathological urines also sink to the bottom of the vessel and often remain apparently intact, even when no care is taken to secure the asepticity of the urine. In a previous paper we showed the serum of rabbits which had received injection of human urine dissolved human red blood corpuscles in vitro.

We were thus led to inquire whether normal urine contained an antihaemolytic (haemosozic) substance neutralizing the haemolysins present in the sera of animals previously injected with urine.

Experiments showed that a serum markedly haemolytic for sheep's red blood corpuscles is obtained by injecting sheep's urine into rabbits, and that the injection of the urine of the ox produces serum haemolytic for the red corpuscles of the ox. Miss B. Sheldon Amos, working in this laboratory, has found that the injection of small doses of urine produces marked changes in the composition of the blood, and that, occasionally, the injection of so small a quantity as 2 c.cm. of normal urine into a rabbit causes the serum of that animal to become haemolytic.

In connexion with these experiments, it must be remembered that rabbit's serum always dissolves to some extent sheep's red blood corpuscles, and less frequently it haemolyses slightly those of man.

In order to demonstrate the haemosozic property of normal urine, we mixed a given quantity of human red blood corpuscles, previously washed twice in normal salt solution and centrifugalized, with a given quantity of haemolytic serum. In another tube, we added to a similar mixture a certain quantity of freshly passed normal urine. In order to avoid errors

due to greater or lesser dilution, physiological salt solution or normal rabbit's serum was added whenever necessary, so that each tube contained exactly the same quantity of fluid. The following experiment may serve as an example.

EXPERIMENT.—Rabbit (No. 23) received on the first, fifth, and tenth days 3 c.cm. of normal human urine (total 9 c.cm.). The serum was found haemolytic for human corpuscles. The serum of this animal we call Serum 23.

	Serum 23.	Serum normal (rabbit).	Human urine.	Red corpuscles (human).	Result.
Tube I.....	20 drops	o	20 drops	2 drops	+ in 18 hours
" II. ...	20 "	20 drops	o	2 "	++++ "
" III....	10 "	o	30 drops	2 "	∞
" IV....	10 "	30 drops	o	2 "	+++ in 18 hours

(N.B. +++ means complete haemolysis. + means slight haemolysis: that is, red corpuscles remain undissolved at the bottom of the tube, the supernatant serum being slightly tinged with haemoglobin. ++ and +++ are immediate stages. ∞ indicates that no haemolysis has taken place.)

Haemolysis, therefore, was only very slight or totally absent in the tubes to which urine had been added.

Similar experiments made with various samples of haemolytic sera (produced by the injections of urine) always gave the same results.

The haemosozic property of the urine of the same individual varies, for whereas 20 drops of urine passed by one individual at 9 a.m. may prevent the haemolysis of a given quantity of haemolytic serum, the same quantity of urine of the same man passed at 11 a.m. may no longer prevent the haemolysis of the same serum, though all the other conditions of the experiment remain the same.

The red corpuscles remaining in contact for some hours with urine, when removed from the medium, washed and suspended in haemolytic serum are haemolysed just like those which have never been in contact with urine, and the urine in which such corpuscles have been suspended for two or more hours loses none of its haemosozic properties. The red corpuscles, therefore, do not absorb in that time any haemosozic substance present in urine. The haemosozic substance or substances have not been isolated from the urine, but the following properties have been ascertained.

First, the haemosozin is practically destroyed by heating to 120° C. or to 100° C for 30 minutes.

Secondly, the haemosozin is precipitated almost completely by absolute alcohol, but a slight quantity of haemosozic substance is always found in the filtrate. One may argue, either that the haemosozin is slightly soluble in alcohol, or that there are two haemosozins one of which is soluble in alcohol, and the other is not.

Thirdly, the haemosozin is precipitated almost entirely by saturating the urine with ammonium sulphate. The precipitate dialysed until it is entirely free from salts has marked haemosozic properties.

THE DETERIORATION OF VISION DURING SCHOOL LIFE.*

By ETTIE SAYER, M.B., B.S.,

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THERE are nearly a million children attending the Board schools in London and such a large number of these had obvious defects of vision that two years ago the Board decided thoroughly to investigate the condition of the children's eyesight. The children in our schools are divided into three departments:

1. Boys between 8 and 14 years of age.
2. Girls " 8 " 14 "
3. Infants " 4 " 8 of both sexes.

The work was carried out on purely scientific principles. A child was taken to have normal vision who distinguished objects subtending an angle of one minute, this being determined by the ability to recognize letters 9 millimetres square from a distance of six metres. Only children in the boys' and girls' departments were to be included in the test, and it may

* Paper read at the International Congress of School Hygiene at Nuremberg, 1904.

be stated briefly that 10 per cent. of boys and 11 per cent. of girls on an average throughout school-life were found to have such serious defects of vision that their visual acuity was $\frac{1}{8}$ or worse.

In addition to this I last summer very carefully tested the visual acuity of 1,864 infants between 6 and 8 years old, 100 of each sex for every three months increase of age. With children younger than this it was impossible to get trustworthy results.

As a large number of these infants did not yet know their letters, the letter E was made in various sizes and positions to correspond with Snellen's test types $\frac{1}{2}$, $\frac{1}{3}$, etc. The infant was placed six metres away, given a large E to hold, and told to turn it in the same direction as the one indicated—upwards, downwards, backwards, or forwards—and rewarded with a sweet every time it got it the right way upon the first trial.

The results were very surprising, for it was found that at exactly six years of age 81 per cent. could read $\frac{1}{2}$ with each eye separately, and only 3.5 per cent. had such serious defects that they could only see $\frac{1}{8}$. The defects, however, steadily increased with every quarter of a year of age, until at 8 years old only 77 per cent. could see $\frac{1}{2}$ with each eye, whilst 8 per cent. had serious defects.

These results were so contrary to statistics published hitherto that Dr. Kerr, the Principal Medical Officer to the Board, suggested submitting a large number to re-examination by the ordinary methods, and he and I together went over them. It was now found that when tested with the ordinary Snellen's test types a much larger number apparently had bad vision, but with this method of E the vision of infants was as is stated above.

The reason for this difference I shall endeavour to prove is simply that the higher brain centres are as yet undeveloped, and that the methods hitherto employed for testing the eyesight of very young children have been at fault. Defective visual acuity, in short, has been confused with want of perception by the brain and the serious defects among children aged 6 years are only 3 per cent., a percentage which steadily increases until at 11 years old it reaches 11 per cent., and then gets somewhat better again.

This great deterioration is extremely serious. The eye being the chief inlet for knowledge it means that at least 25,000 children are being seriously handicapped in all their pursuits. The shortsighted child cannot see the blackboard, a ball in play comes suddenly out of nowhere, a human face is an expressionless patch, and, never seeing its expression, he grows up without any power of estimating character from observation. As he does not care to look at what he cannot see his chief delight is to pore over a book. This, as I shall show, only increases his defect of vision, and, moreover, since he sits all doubled up with his head bent low over the book, the movements of respiration are hampered, which predisposes him in after-life to organic diseases of the heart and lungs.

Therefore, it is of the utmost importance to discover what is the cause of the deterioration of vision, and to prevent it or cure it as the case may be.

Normal Changes in the Eye from Birth to Old Age.

A normal eye is one in which the focal length of the lens at rest is equal to the axial length of the eyeball posterior to the lens, and in which the retina is of normal structure and sensitiveness to receive the disturbances and vibrations of ether known as light. Given these conditions a perfect image is thrown on to the retina from every object which is placed in front of it at a sufficient distance for rays of light from it to arrive on the front of the eye practically parallel. In order that correct images of near objects should be accurately focussed on to the retina the normal eye possesses three faculties: (1) Adaptation, (2) accommodation, (3) convergence. The intelligent appreciation and interpretation of things visible, however, is a function of the very highest order, necessitating not only that the eye should be perfect as an optical instrument, but in addition that the higher visual centres situated in the occipital cerebral cortex and elsewhere in the brain should be fully developed and in good physiological working order.

The changes which occur in the eye itself during life are chiefly concerned with accommodation which depends partly on muscular contraction and partly on the elasticity of the lens. If, therefore, that elasticity is diminished (as it is in old age) beyond a certain point, accommodation will fail. The eyeball in infancy is short or flat and hypermetropic, but the lens is relatively larger, more hemi-

spherical, and much more elastic than it is in after-life; therefore its amplitude of accommodation is greatest at birth—so much so that the shortness of the eyeball is then more than compensated. By about 11 years of age the eyeball has grown and elongated so as to be emmetropic—that is to say, the focal length of the lens equals the length of the posterior chamber. Throughout life the elastic tissue in the whole of the body gradually lessens, and the elastic tissue in the coats of the eyeball shrinks, causing it to become somewhat flatter again. The lens itself loses its elasticity most markedly and steadily, and with this loss the amplitude of accommodation gradually diminishes until by the age of 50 the normal eye is incapable of accommodating for objects nearer than 10 in.

Changes during Life in the Visual Centres of the Brain.

The dissociation between the reception of visual impressions by the eye and the perception of them by the brain is a matter of everyday experience. Who is there amongst us that has not at some time or other pulled out a watch, looked at it, and after replacing it in the pocket realized that the time had not been noticed? There is a distinct remembrance of the facepiece having been impressed upon the retina, but owing to the ultimate visual cerebral centres or the passages to these centres having been obstructed by thoughts or cerebration on other subjects the details of the facepiece of the watch passed unnoticed, and failed to arouse any consciousness of their significance.

Somnambulism is another familiar example of the cutting off of these higher from the lower centres. Here all actions are performed automatically, visual impressions being received and acted upon without the concurrence of conscious thought. The young infant, whose higher centres are only partially developed, will be unable to explain which letter is D and which is G and to give a name to them, but yet the eye may be capable of receiving a perfectly accurate visual impression, and by exercising the faculty of mimicry (which is developed extremely early and is present in several of the lower animals), it may copy the position of an E held up to it every time it receives the impression; especially will it do this if its sympathies are engaged by its being rewarded every time it gets the position right on the first trial.

The progress of the development of these higher centres is best watched by noting the development of the fusion faculty—that psychical blending of the two sets of visual impressions when the two eyes act together as a pair which produces binocular vision. A newborn child fixes a light from a mirror flashed into its eye only momentarily, therefore the fixation must be purely reflex. In two or three weeks it fixes it for two or three seconds, but does not converge accurately. It is only by the fifth or sixth month that experiments with prisms show a distinct desire for binocular vision.

During the first few months want of control by the higher centres resulting in muscular inco-ordination causes the movements of the eyes to be uncertain, the slightest disturbance, even a little flatulence, causing one or other eye to deviate. But by the end of the first year the desire for binocular vision is so strong that unless there is some insuperable obstacle the vision of one eye will be suppressed in preference to enduring diplopia. Experiments with Worth's amblyoscope prove that this fusion faculty is not fully developed normally until the sixth or seventh year. Therefore the higher centres cannot be developed until then.

The Cause of the Rapid Deterioration of Vision during School Life.

At 6 years of age 3 per cent. have seriously bad vision, and 88 per cent. can see $\frac{1}{2}$ with each eye; at 11 years of age 11 per cent. have seriously bad vision, and only 58 per cent. see $\frac{1}{2}$ with each eye. The rest have slight defects.

In farming districts, where vocations demand no accurate vision, no necessity for care of the eyes arises; but if children from these communities are sent to school the problem of how to prevent injury to the eyes soon obtrudes itself by the appearance of headache, pain in the eyes, impaired vision, undue sensitiveness to light, and increased lachrymation. Examination reveals tonic cramp of accommodation and hyperaemia of the optic nerve, choroid, and retina. These symptoms subside under rest, and recur when work is resumed.

If accommodation and convergence are not sufficient to overcome the hypermetropia of infancy, the child brings its eyes very low down on to its work, as the larger retinal image and increased illumination more than compensate for the less

accurate definition and the muscles of accommodation are strained to their utmost. A sustained and strenuous effort is demanded of all the ocular muscles. The continuous contraction of the internal recti renders them liable to overpower their antagonists and become a factor in the causation of squint. More important is the pull which the muscles of accommodation exert upon their points of insertion; they distort the eyeball and tend to lengthen it antero-posteriorly. This elongation produces myopia, which when caused in this way is more likely to attain a high degree and become ultimately perilous than when it existed as an original proportion. All recorded cases which have passed from hypermetropia to myopia have done so by astigmatism, no one having been emmetropic at any stage.

The small or hypermetropic eye is always found in animals, in infants, in uncivilized races, and in those who pass their lives in employments of the grosser sort, whilst myopia is almost unknown under these conditions of life. It is with the greatest rarity that the adult Indian outgrows his hypermetropia. Unfortunately I did not take statistics, but, when doing a large practice amongst Kaffirs, I was never once consulted about short sight, or even asthenopia.

Myopia results only under the stress of those employments which require the protracted use of the eyes for near work. Half a century ago Professor Donders laid down the law that "the myopic eye is a diseased eye." Myopia increases steadily with the progress of pupils in school; it is the characteristic of the student or of the artisan who began accurate near work early in life. Among the compositors of Breslau, Cohn found that no less than 51 per cent. had myopia. Risley found the same great percentage among the compositors and brass-founders of Philadelphia. Among the students in various German universities Cohn found 22 per cent. to 23 per cent. were myopes. Erisman showed that among 1,245 myopic children in St. Petersburg, only 5 per cent. were free from pathological conditions of the choroid. Horner kept 1,875 myopes under observation, and no less than 34 per cent. developed the greatest complications later on, such as haemorrhages into, or detachment of, the retina, optic atrophy, etc.

Indications for Treatment.

A child compelled to struggle with a sign is negligent of the thing signified. Its nervous energies cannot be directed into two channels at the same time. Therefore if its senses are defective it cannot take the full advantage of its education.

The public duty of providing an education which is to be the preparation for the child's after-life should include the duty of ascertaining whether that education is likely to inflict serious and permanent physical injury upon it.

It is only reasonable to expect some degree of physical deterioration will result from depriving the child of its freedom and confining it to lessons, and since the eye is the organ upon which the greatest strain falls, it will be the eye which is most likely to suffer.

All eyes should be tested immediately on the child beginning its education: (1) As to visual acuity; (2) as to rapidity of perception; (3) as to colour blindness.

All children whose visual acuity is subnormal should be atropinized at once, and if there is any excessive degree of hypermetropia simple *plus* lenses should be worn until it is outgrown. Spectacles for them should be regarded not as an evil, but as a temporary protection against the evil of defective vision.

With regard to commencing myopia, it is very questionable whether the child should be allowed to continue its studies at all. Certainly no more near work should be done than is absolutely necessary. A special curriculum should be devised for these children which will fit them for a congenial career in after-life and not involve any strain on the eyes.

Children with errors of refraction always get as near to their work as possible. The hypermetrope, to get a larger image; the myope, to get any image at all—spectacles may do away with the necessity, but the objectionable habit may be formed. The thing to do is to use a face rest which will keep the face at least 10 in. off the book. Kallman's of Breslau is the best.

Squint occurs when there is an error of refraction combined with a defect in the "fusion faculty." I have found it present in 4.5 per cent. of children under 8 years of age. It is of the utmost importance to cure squint with the greatest speed as early as possible. Before the child is 6 years old it is easy to do so by correcting the refraction and training the fusion faculty with Worth's amblyoscope. After this it is a

matter of considerable difficulty. Only 30 per cent. of squints can be cured by glasses alone, the rest requiring operative treatment, etc. Squinting eyes become amblyopic from continued suppression of vision to prevent diplopia.

Colour sense is as often untrained as absent, therefore attempts should be made to develop it.

The Type of Children's Books.—It has already been most ably pointed out by Dr. Kerr that pens, pencils, paper, etc., are utterly out of place in an infants' school. They are much too minute. Infants should only be allowed chalk on card-board. Reading should be learnt from the blackboard. Every child in a 6-year-old class should be able to print its name in 6 in. letters on the blackboard with a free arm. Fine writing should be absolutely forbidden. In order to be distinguished, letters must subtend an angle of at least five minutes, and the lines of which they are composed at least one minute. This requires the utmost endeavour of an emmetrope, being the limit of normal acuity of vision. Therefore it is obvious that a hypermetropic infant should not be set to small print. At no time during school life should type smaller than *pica* be used.

Parents among the lower orders are so astoundingly ignorant with regard to the care of their children's eyesight, that it is impossible to rely on them to get defects attended to. They invariably imagine the child will in time "grow out of" any defect. Glasses they regard as a disfigurement worse than sore eyes, or else as an adornment for Sundays. Many children also run grave risks by their parents taking them to sight-testing establishments, certified opticians, etc., who are rightly prevented by law from using atropine, and who therefore prescribe wrong glasses.

CONCLUSION.

No child's eyes were intended by Nature to undergo the strain of accommodating over lessons for six or seven hours every day of their lives between 4 to 14 years of age.

If, however, compulsory education enforces it, it becomes the duty of the Board of Education annually to separate those 20,000 children or more whose vision is so defective that they are unfit physically, to devise for them a specially modified curriculum, and to provide them with glasses if their parents are too poor or too ignorant to do so. This is the type of case, common enough, I am referring to.

Boy, aged 13; rest of his class I examined yesterday, but he was absent, as he stopped at home to help his mother (who is a widow with five younger children) do the washing. He suffers from headaches, squints, sees double, and has blepharitis. His class is at present doing algebra, a subject in which he makes no progress, as sometimes the figures are all a blur and at others they jump about.

Such children are as unfit to become students as cripples are to become soldiers.

There is plenty of skilled manual work to be done by the class which is so poor that the public has to pay for their education, and they should be taught from their earliest infancy to regard this as their special lot in life.

For if the child with defective vision has its weak eyes overstrained, not only does he as an individual suffer, but also future generations, to whom the welfare of the nation is to be entrusted, it being a well-proved fact that there is no defect more likely to be transmitted from parent to child than that of defective eyesight.

OBSERVATIONS ON AN EPIDEMIC OF SCARLATINA.

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THE following notes on a recent epidemic of scarlatina will be of interest on account of the mildness of the symptoms in a large proportion of the cases, only 31 children out of a total of 96 showing any signs of a rash.

The not infrequent occurrence of very mild cases of scarlatina, with little or no perceptible rash or indisposition, is not dwelt upon with sufficient emphasis in the usual textbooks.

The epidemic occurred in a school of 300 children (boarders), to which I am medical officer, and was a source of much anxiety on account of the absence of all symptoms of illness in a large proportion of cases and the consequent difficulty of selecting cases for isolation. At the outset I may note that the milk supply was above suspicion, and that there was no scarlatina in the neighbourhood.

Early in October, 1902, my attention was called to a child