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* Awarded the Emmie Russell Prize

The Emmie Russell Prize for 1973 was awarded to Shane Brown for her paper on
"Uses of Fresnel Prisms", published in Volume 13.

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EDITORIAL

This is a time of growth and change for orthoptics in Australia. The transfer of management of orthoptic schools from the Orthoptic Board of Australia to government-financed Colleges of Advanced Education has given teaching orthoptists a fascinating but difficult task in planning how best to use the wider resources available. There is a corresponding stir among non-academic orthoptists towards defining their role and the qualifications necessary to fulfil it, and with this, towards reviewing the ideas, traditions and procedures which must be expounded and demonstrated to students who come for clinical training. This is in part stimulated by the increasing opportunities for involvement in community health. Some of these are here described in papers read at the Sydney conference which had as its theme "The Orthoptist's Role in the Community".

To all new developments, members of the College of Ophthalmologists contributed - instigating, advising and assisting.

One member of the College, liked and respected by interstate orthoptists for his work on the Orthoptic Board, is no longer with us. Shane Brown, president of the New South Wales Branch of the Orthoptic Association, writes:

"Dr. Bryan Cooper M.B., B.S. (Sydney), D.O. (London), F.R.C.S., F.R.A.C.S., M.A.C.O., died on 8th November after a lengthy illness. His untimely death at the age of 43 is a tragic loss to the world of ophthalmology. He had become one of the leaders of neuro-ophthalmology in Australia. It was said that he always prized his position as neuro-ophthalmologist to Sydney Eye Hospital; it was well known that the Hospital felt honoured to have him on the staff. The New South Wales Branch of the Orthoptic Association is indebted to Dr. Cooper for his contribution to orthoptics in this State. He was honorary treasurer of the Orthoptic Council of New South Wales, later its chairman. He was vitally interested in the education of orthoptists; he began lecturing in 1965 and continued until the first signs of his illness last year. Many of us will long remember those lectures, always informative and witty, certainly never dull. He was a great friend to orthoptics, and will be sadly missed by orthoptists."

We also record with sorrow the death of Margaret Theile (Hamp) a Victorian member of the Orthoptic Association since 1959, of frail physique but of gallant spirit.

Diana Craig

COMMUNITY INVOLVEMENT IN THE WESTERN METROPOLITAN HEALTH REGION*Ann Macfarlane**Presented in Sydney, April 1975.*

As our theme is "Community Involvement" my paper is about the work we are doing for the Health Commission of N.S.W. in the Western Metropolitan Health Region.

The Western Metropolitan Health Region was established in August, 1973, with Dr. Gary Andrews as Regional Director. It contains fifteen local government areas in Sydney's Western suburbs with a population of 1.2 million.

The Western Region is staffed by "Community Health Nurses". Each nurse is based in a school and works in a small geographically defined area with a population of 3,000 to 5,000. Her many functions include a health screening programme for school children. This replaces the School Medical Service in this area.

All school children in New South Wales are health screened in kindergarten and again in 5th class.

Each community health nurse does a three months course. Dr. S. Sarks (consultant ophthalmologist for the Western Metropolitan Health Region) and I were asked to organise their training in ocular screening. We have been allocated a 3 hour lecture and a 1½ hour demonstration. The lecture basically covers medical eye conditions, refractive errors and strabismus. Also methods of testing are discussed.

The next day, in groups of six, the girls are given a practical demonstration. This is held at a school under the conditions in which they will be working. It is a 1½ hour demonstration with five main parts -

- (1) vision testing,
- (2) general anatomical features and positions of light reflections,
- (3) cover test for near and distance
- (4) muscle movements,
- (5) convergence

Each child examined is given this series of tests. The nurses receive a comprehensive set of notes also. The lecture has been given by me, and the demonstrations by myself at Fairfield and Mrs. Wong at Mt. Druitt.

When the nurses have had time to settle into their schools, a follow-up visit is made to each girl by an orthoptist to see that she is screening correctly and so that she may discuss difficulties she is finding. All obvious ocular cases have already been referred by her for treatment. She presents the children of whose diagnosis she is unsure. The orthoptist writes a brief report on each positive case, and referral for treatment is arranged.

This follow-up visit has been the main role of the orthoptist. We are aiming to have an orthoptist visit each nurse once a term for half a day. We also suggest that all children with reading problems have an ocular examination.

In every school the equipment is standardised. There is a wooden test cabinet having a Snellen's test panel with internal lighting, set at twenty feet or with a mirror at ten feet, a Sheridan-Gardiner magnetic board with removable letters, a fixation torch,

an occluder, a fixation stick and squeaky toys. Also a set of Matsubara colour vision test plates for infants. Teachers are more and more often asking about colour blindness because of the use of cuisenaire rods for mathematics and reading by colour.

In New South Wales, many schools, particularly in the Western suburbs are having pre-school kindergartens built by the Government within the public schools. Children enter these one year earlier, that is at three years and nine months. It is good to be able to detect defects even one year earlier but, because of the earlier age, the easiest method of vision testing had to be adopted. It has been unanimously decided that Sheridan-Gardiner is the easiest because it does not involve eye-hand co-ordination to a fine degree or right versus left decisions. From the equipment point of view, it has meant that only the one Snellen's panel is needed. We have developed the Sheridan Gardiner Test further.

If you are on your own pointing to the chart, it is not possible to see which letter the child is pointing to on the Sheridan Gardiner cardboard sheet. Plastic letters which the child could lift from a frame were not practical as they were too easily dropped.

Mr. Harris (of Marsden Industries Sheltered Workshop, N.S.W.) and I then came up with the idea of a magnetic board with large removable letters. The children love this as it is a "matching" game and as the letters are printed on the back of the block, they can be easily seen by the tester at the end of the room. Even severely retarded children at Evandale School at Croydon with I.Q.'s between 30 and 60 can do Sheridan Gardiner by this method and certainly the three to four year child can cope easily.

Conclusion

The most important aspect of our programme to me is that it means that referrals will be made at a much earlier age. It has happened to all of us that a child of nine (i.e. the fifth class examination instead of the earlier one) has been referred with severe amblyopia. Beside the fact of the dramatically reduced chance of responding to treatment, the trauma of occlusion is great for this child personally, and the effect bad on the school progress. As these nurses will see the whole family in the scope of their work, we hope that they will detect defects from the baby stage on.

Mrs. Wong has been following-up in the Mt. Druitt area, and from the end of July, 1974 to the end of March, 1975 has confirmed 192 cases, doing only a few hours per week of follow-up screening. In the Fairfield area, I have in five weeks confirmed 29 cases. In one morning, I was asked to look at 11 children; 4 of them had no defect, (other than a large epicanthus in two), 1 had a large esophoria (and a behaviour problem); 1 had a "V" sign and intermittent divergent strabismus; 3 had convergence deficiency, and 2 had amblyopia only. I feel this programme is effective because screening is more thorough, and would like to see it implemented in other Regional Health Areas of New South Wales. Previous screening omitted cover test for distance, and convergence, and muscle movements were very crudely checked.

Acknowledgements

I would like to thank Dr. Andrews and Marie O'Connor of the Western Region of the Health Commission for their help with this paper and also Dr. Carter (Medical Superintendent of Lidcombe Hospital) and Mr. Loman - the clinical photographer. Lastly but certainly not least I would especially like to thank Dr. Shirley Sarks, Consulting Ophthalmologist, without whose help and enthusiasm this programme would never have reached this stage.

SURVEYS AND ALL THAT!

by Patricia Wister

1970 saw the beginning of the involvement in the community of Victorian orthoptics. In November of that year the first amblyopia survey sponsored by Lions International was held at Healesville, Victoria, orthoptics being involved in assessment of visual acuity, cover test et cetera, and orthoptists by doing the work. This survey showed that of approximately 300 children screened (age group 3½-6 years), 10% had a visual defect.

These results inspired other Lions Clubs in Victoria to sponsor surveys; thus orthoptics and orthoptists became more involved with the community of Victoria.

Since the end of 1972 I have been directly involved, asking, begging, pleading, bribing orthoptists to spend their Saturday at amblyopia surveys.

It is on these surveys that I wish to comment.

From January 1973 to April 1975 we have carried out 81 surveys, 46 being in country areas and 35 in metropolitan areas.

During this time approximately 10,000 miles have been travelled in country areas alone. From Portland in the West, Bairnsdale and Orbost in the East, to the New South Wales border, Phillip Island and Tasmania in the South. All manner of transport, car, plane, hydra-foil, and on one occasion, tow truck, have enabled us to reach our destinations, more or less on time, depending on the map reading capabilities of one's navigator. In these areas one is totally involved with the community, especially in more remote areas. The hospitality and friendship shown us are extraordinary. Some families travel fifty miles or more and combine attendance at the survey with a picnic in the local gardens. A very noticeable aspect of these surveys is the way the children are dressed, all scrubbed and in their Sunday best. The people are so grateful for our coming, School Medical Service visits being few and far between, that the opportunity is taken to mention among other things, speech, hearing, reading and writing difficulties. All are listened to sympathetically, and tactfully within our capabilities, advice regarding possible means of treatment is offered.

Orthoptists also benefit from this involvement inasmuch as we get to know and get along with all manner and type of person; to know our State highways intimately; to become acquainted with fascinating bits of history and local folk-lore. One never realised what a wealth of interest there is in our own home State.

Metropolitan surveys are a test of our fractured Italian and Greek, especially in the Western suburbs, where there is a high density migrant population. Usually an interpreter is on hand; if not, one has to seek a bi-lingual parent.

Sometimes one meets with resentment from parents who don't like to think there is anything wrong with their child. A great deal of tactful persuasion, on the orthoptist's part, has to be employed. Notice of such a situation is brought to the attention of the sponsoring Lions Club, who follow up all referrals but these cases in particular, to see that the child receives appropriate medical attention and treatment.

Children up to the age of six years in these areas speak little if any English, which makes one ponder on their difficulties when they start school, and the burden of language barriers placed upon the teachers in Prep and Grade 1. Mostly the eastern suburbs are on a higher socio-economic level. The warmth, spontaneity and intense involvement, i.e. "we're all in this to help the children", seem to be lacking.

81 surveys have been conducted - 50 in 1973, 15-20 orthoptists being available. In 1974 - travel fever, home and family commitments depleting our numbers - only 24 surveys were possible; to this date 7 have been run in 1975.

No two surveys are alike. They range from the sublime - adequate venue, superb organisation - to the ridiculous - icy east wind blowing into a concrete floored fire brigade station and no organisation - total disaster.

On these latter occasions the true mettle of the Victorian orthoptist comes to the fore. Smiling faces greet every child, charming tactful voices advise parents, all the while legs and hands are freezing, feet hurting and I'm sure wishing me (the organiser) to the other end of the earth. As yet I haven't been lynched!!

The average age of the children seen is $3\frac{1}{2}$ -6 years. Marked differences are noted in the intelligence, hand-eye co-ordination and attention span of this age group, especially $3\frac{1}{2}$ - $4\frac{1}{2}$ years, not to mention the many and varied ways of demonstrating the "E" game, from gymnastic enthusiasm to the merest indication with one finger held tight against the chest or leg.

In screening, we use the cover test, and also test ocular movements, convergence and visual acuity. Psychology and patience play a big part in our professional lives, but never so great as when one is confronted with one's 75th tearful, shy, obstinate or rude child for the day. An interesting note is the number of children who cannot converge voluntarily.

The average number screened during each survey is about 300. One momentous day at Bendigo in 1972, 1074 children were seen. The approximate total, over 81 surveys, is 24,300, and the average referral rate is 10%, i.e. 2,430. This means that 2,500 children in this age group have a visual defect, a very sobering thought when one considers there are 141,051 (1973) school children in Victoria in Preparatory and Grade 1.

The greatest percentage of these referrals are refractive errors, followed by latent intermittent and manifest deviations, then amblyopia in its strict definition.

Apart from visual defects, i.e. children with low or unequal vision, we find those with equal vision, but with possibly every known syndrome and muscle imbalance, e.g. Duane's, S.O. Tendon Sheath, Jaw Winkers, C.H.P. head posture with and without muscle imbalance, A & V patterns, I.O. overaction for no apparent reason, and of course sore eyes, itchy eyes, sticky eyes and runny eyes; these are all referred for further assessment.

The sponsoring Lions Club follows up all referrals by visiting the family concerned. If no action is taken, two or three repeat visits are made. In fact these clubs do all in their power to see the child received appropriate medical advice and treatment. Under-privileged families have been assisted with transport and offers of financial help, e.g. for glasses.

Occasionally one comes across parents seeking a second opinion; here one can only tactfully suggest they go back to their own doctor. In nine cases out of ten, one finds they have an appointment anyway!

In these 81 surveys we have found a very meaningful role for orthoptists in Victoria, and an almost frightening rate of visual defects in the $3\frac{1}{2}$ -6 year old age group. We believe that orthoptic assessment could with advantage be incorporated into the School Medical Service.

I would like to thank Glenys Van Den Breck of the Photography Department at Royal Victorian Eye and Ear Hospital, who prepared my slides at such short notice, the Lions of District 201M for their financial generosity to our Department at R.V.E.E.H., Anne-Marie Mahoney and Anne McIndoe for slides taken at our last survey, and most importantly, the Victorian orthoptists for their friendship, stamina and continued loyalty to me.

VISION SCREENING – PILOT SURVEY

S. Brown, S. Gillis, MD.,CL.B.,M.H.A., S. Stanley

Submitted December, 1975

Following discussions with the Department of Education and the Health Commission of N.S.W., a pilot survey was undertaken of 400 children between the ages of 7.0 – 8.11. The children were 2nd and 3rd Grades from the Public Schools of Rose Bay, Redfern and Paddington.

Personnel

Examinations were conducted by three orthoptists from the Orthoptic Department of the Sydney Eye Hospital.

Tests Performed

1. Vision with an 'E' chart at 6 metres.
2. Cover test for near and distance
3. Convergence near point
4. Ocular movements
5. Stereopsis and three dimensional acuity
6. Reference eye test
7. Reading age tested with the Holborn Reading Scale

Recording

Copies of code and coding sheets are attached

Breakdown of Results

The four categories of defects found were:

1. Defective Visual Acuity
2. Squint
3. Convergence Insufficiency
4. Stereopsis

1. Defective Visual Acuity

Visual acuity was tested with an 'E' chart at a distance of 6 metres. 48 (12%) children were found to have defective vision in one eye only; 28% children had defective vision in both eyes.

6/9	29 Children	7.25%
6/12	8 Children	2.00%
6/18	8 Children	2.00%
6/24	1 Child	0.25%
6/36 & 6/60	None	
<6/60	2 Children	0.50%

2. Squint

The presence of a squint was tested by the cover test, the stereoscopic function on the Synoptophore and the Titmus Tests.

24 Children had squints	6.00%
Intermittent Convergent Squints – 3 Children	0.75%
Convergent Squints (Constant) – 12 Children	3.00%
Intermittent Divergent Squints – 9 Children	2.25%
Divergent Squint (Constant)	–
Of the 24 children – 9 had Amblyopia	2.25%

3. Convergence Insufficiency

131 (32.7%) children were found to be suffering from this condition – some complained of symptoms, but perhaps more noteworthy is that 62(47.3%) of the 131 had defective reading ability.

4. Binocular Vision

Defects in stereoscopic depth perception may cause problems in school subjects involving visual judgment and of course in many sports.

93 children in total were found to have defects in binocular stereopsis –	23%
37 had associated convergence insufficiency –	9%
28% of children with convergence insufficiency had defective stereopsis.	
20 had associated phorias –	5%

36 did not have associated phorias or overt squint or convergence insufficiency. Some of these may have had associated refractive errors, examination for which was not included in the survey, but for which electronic refraction, as a screening procedure, is now available. – 9%

Recommendations

1. The figures presented in this report are interesting, but 400 is a relatively small group, and it is felt that a larger survey of 5,000 would give more significance to the figures.
2. The method of examination should be trimmed to a simple screening procedure, which would still adequately detect defective visual acuity, squints, convergence insufficiency and binocular function.
3. The age group should be lowered to 5.0 – 6.11 years.
4. A simple screening procedure would enable more children to be seen in a shorter period. It is estimated that 5,000 could be screened by five orthoptists in twelve weeks – orthoptists could be drawn from a number of hospitals.
5. The recording system used in this survey was simple and most efficient.

VISUAL SCREENING OF CHILDREN IN WESTERN AUSTRALIA

Beverley Balfour

Visual screening of children by orthoptists has been sponsored in Western Australia as in other States, by the Lion's Save Sight Foundation.

The response has not always been good, and is possibly better in the country areas. We conducted screenings in various metropolitan areas up to November 1974 since then the Lions seem to be engaged in other Save Sight projects. Some of us have had trips to country areas, mainly by plane, covering the South as far as Esperance, the East to Meekatharra, and the northern mining areas and on to the far North, Wyndham and Kunurra.

Those of us who were fortunate enough to go on the trips have gained much greater insight into Community Welfare problems, and a healthy admiration for the welfare workers and others who try to help the people in their areas, often under the most trying conditions, day in, day out. The Health Department allows children requiring medical treatment to travel free to Perth if they live north of the 26th parallel, and others can get assistance according to circumstances.

It seems to me a pity that this is stereotyped, as there could be obvious advantages in seeing them in their own environment, and sending those who need it south for further examination and treatment. Various groups of people do go, or are sent, north but I feel that a co-ordinated approach involving, say, both an orthoptist and an ophthalmologist would be of more benefit. Children of white, particularly of British parentage, appear to suffer from divergent squints.

The aborigines appear to be the most disadvantaged. They are not accustomed to our signs and symbols, and I feel that they need a special chart. The tribal ones are the most delightful children, well disciplined, and their elders stand by. They make no fuss if they don't understand, merely becoming silently shy, with lovely liquid brown eyes, charming smiles, and clouds of flies around their eyes and runny noses. All the full bloods I tested had excellent visual acuity. I tested some in camps in the open, hanging my E chart on the nearest gum tree.

The semi-civilized ones suffered from malnutrition, and their eyesight seemed to be less of a problem than their general health.

Tests for trachoma are done by the Welfare officers as a routine now. Even though the percentage screened was not always high, often due to public apathy, I feel that the number we have "saved" from amblyopia and refractive errors was worth it; news of grateful parents filters through to me.

My main country practice involves approximately 700 miles and four country towns in four days once every three months. The contacts there, particularly if patients or parents keep in touch by phone or mail between times, can speed the treatment of amblyopia in particular, and encourages them to keep at it. Results in the end are often just as good as those of town patients.

ORTHOPTICS AND CEREBRAL PALSY

V.C. Elliot

Presented in Sydney, April, 1974

To quote a widely accepted definition: "Cerebral palsy is a persistent but not unchanging disorder of movement and posture appearing in the early years of life and due to a non-progressive disorder of the brain, the result of interference during its development."

There are four main types of cerebral palsy, all of which refer to the types of muscle abnormality found in the child. More than one type may be present in a particular case with the degree of involvement and distribution varying from child to child. The types are:-

1. SPASTIC

This is the most well known of the groups, having been the first to be described and made known to the public. These cases show increased muscle tone which may involve one or all of the limbs, trunk muscles, and/or the muscles of respiration, of the head or of the neck. In mild spasticity there may be only a disturbance of co-ordination and balance.

2. ATHETOID

In this type, the muscle tone fluctuates between hypertonus and hypotonus resulting in involuntary movements due to fluctuations in tone in the opposing muscles.

3. ATAXIC

This involves a loss of balance. Equilibrium reactions are well developed but are abnormal and unco-ordinated.

4. HYPOTONIC

This is a low muscle tone. These children usually become either athetoid or spastic and some develop normal muscle tone.

The associated defects of cerebral palsy include respiratory problems, epilepsy, deafness, speech defects, behaviour disorders and learning disabilities. Mental retardation (i.e. an IQ less than 90) is found in many cerebral palsy cases. The Spastic Centre of NSW has found that 25% have an IQ below 50; 25% between 50 and 70; 25% between 70 and 90; and 25% over 90.

The Spastic Centre School at Mosman caters for cerebral palsy children from nursery to 6th Form High School, with the classes grouped mainly in academic potential and degree of handicap. Included in these groups are: one Junior O/D (opportunity deaf); one Senior O/D O/L (opportunity deaf and language); three O/F classes (IQ 35 to 55 i.e. moderately retarded); and two O/A classes (IQ 55 to 70 i.e. mildly retarded).

The children are in school for the normal school hours but one half of the day may be interrupted by treatment in Physiotherapy, Occupational Therapy, Speech Therapy or Orthoptics.

Over a period of twelve months working as an orthoptist at the Spastic Centre of NSW, it became obvious that the ocular defects found there were much greater than those found amongst normal children. The 154 cerebral palsied children at the Mosman Spastic Centre School were screened and compared with 100 children from a suburban public school with the following results:-

	CEREBRAL PALSY 154 CASES	NORMAL 100 CASES
	%	%
Squint	58	7
Nystagmus	21	1
Vision below normal	43	11
Convergence Insufficiency	29	7

Surveys under ophthalmologists have revealed significant refractive errors in between 42% (Graham 1968) and 54% (Schachat 1957) of cerebral palsied children.

Overall 81% of the cerebral palsied children were found to have an ocular defect as opposed to 18% in the normal cross section.

Valium and Serepax are used in selected cases for muscle relaxation and a proportion of the children take anti-convulsants regularly. These tend to accentuate convergence insufficiencies and to reduce the control of heterophorias and squints.

Orthoptic treatments are those carried out in a normal clinic. There is occlusion for amblyopia, elimination of suppression and teaching of diplopia, convergence exercises, treatment for accommodative squints and so on, though treatment may be modified and unfortunately often has to be minimised.

A test is often done with the help of the teacher, speech therapist or psychologist to obtain the maximum response and this is especially valuable in visual acuity assessment. Comments on the visual acuity, the effect of a particular defect and, when necessary, the position or direction of greatest ocular comfort and best vision are made in reports available to the doctors, the school and the various therapies.

Over the past eighteen months, I have become increasingly aware of the part orthoptics can play in the assessment of potential and in the management of the cerebral palsied child and to say that the work there is rewarding would be quite an understatement.

Acknowledgments

I am grateful to Dr. G.C. Hipwell, consultant ophthalmic surgeon, and to Dr. C. Reye, Medical Superintendent of the Spastic Centre of NSW, for their help and encouragement.

REFERENCES:

- H.V. Graham *The Spastic Child*, Vol. 62 American Journal of Ophthalmology Vol.62
Reye, C., *Changing Patterns in Treatment of Cerebral Palsy* Med. J. Aust. (1971): 1187-1188
Bobath, K., *The Motor Deficit in Patients with Cerebral Palsy*
Bobath, K., *The Motor Deficit in Cerebral Palsy* (1966) Spastic Society Medical Education and Information Unit, in association with Wm. Heineman Medical Books Ltd. London.

THE ORTHOPTIST'S CONTRIBUTION TO THE MULTI-DISCIPLINARY GROUP CONCERNED WITH LEARNING DIFFICULTIES

Patricia Dunlop

Presented in Sydney, April 1975

The term "learning difficulty" includes a wide range of conditions varying from obvious brain damage to classical dyslexia where intelligence is normal or even super-normal.

Until recently, the world of ophthalmology was curiously unable to offer any substantial help to those who sought to elucidate dyslexia, literally "difficult reading" which must, so obviously have an important visual element. There seemed to be no relation between eye functions and the tendency to reverse letters or small groups of letters, with omissions and multiple errors of sequencing.

However the recent discovery of a central pool of neurones in the visual cortex which respond only weakly or not at all to monocular stimuli, but respond vigorously to binocular input (Joshua and Bishop 1970), emphasised the need for detailed binocular investigations in place of the former emphasis on exclusively monocular visual acuity tests, simple muscle balance tests and monocular tests of ocular dominance.

This should be the home field of the orthoptist. It proved a fruitful field when we realised that it was important to look most critically at the very central binocular region of overlap, a few degrees wide, where both eyes are represented in each half of the visual binocular cortex. (Bishop & Henry, 1971; Stone, Leicester & Sherman, 1973)

The orthoptist can now demonstrate that in this central field of binocular overlap, dyslexic patients often show no preference, a variable preference or a preference which conflicts with the established dominance of speech and manual motor functions.

Here we have a visual factor which can be correlated with the other disturbed dominance factors, long accepted as an important characteristic of dyslexia. So far, it is only the orthoptist who can demonstrate this factor. She has a critical role in the documentation of all binocular and some non-binocular cases of learning difficulty.

Orthoptists have received limited acceptance at a few centres for investigation of learning difficulties around the world, particularly in the United States. Ward (1970), Hurtt (1971), Kambara (1971).

The special clinic at Broken Hill where Miss Peoples works, is a pioneer in Australia.

It is important to realise that the orthoptist's role is limited to ocular factors - she can have no part in the difficult parallel task of assessing relative intellectual potential or in the detailed analysis of writing and reading abnormalities. The psychologists and special remedial teachers have had many years of training to become competent in these fields. Hence the need for a multi-disciplinary team of which the orthoptist should become a natural member, just as in the past she has worked with ophthalmologists, neurologists and paediatricians. She must work also with psychiatrists, audiologists, speech pathologists, social workers, remedial and regular teachers. She should not forget her important role in communicating with parents who may be just as disturbed as the child. Of course, she may not meet with all members of such a team at once. But even if she only communicates by telephone or letter she should understand what is required and be prepared to undertake purposeful investigation and treatment.

When the patients come to the orthoptist, certain information will be available through earlier investigations. The child will have been assessed medically and educationally. There should be some estimate of his intelligence and performance and some notes as to whether the problem appears to be essentially visual, auditory, emotional or due to some lack of motor co-ordination, or combinations of these factors. The medical history will give the orthoptist some idea whether she is dealing with a normal child or not. The ophthalmologist will have assessed the state of the child's eyes and prescribed glasses where necessary. Speech and auditory tests may have been done.

Very often the child will be a normal, active (perhaps hyperactive) intelligent child, with no refractive error and no apparent ocular muscle imbalance. Parental and teacher's comments on the child's reading, writing, spelling and maths are helpful and the incidence of reversals, inversions, place holding whilst reading, misreading or skipping words, blurring, diplopia, head tilting and headaches should also be noted. Inquiry into family history of learning difficulty, left handedness and ambi-dexterity should also be made, and any stress situation, particularly during the early years of schooling will also help in the understanding of the child's problem. Attention should be given to the child's handedness and history of how it has developed, taking into account any injuries to fingers, hands and arms. Head injuries and birth complications will be noted from the medical history. All these factors are relevant in relation to the child's present age and performance.

Orthoptic Examination should include cover test; ocular movements; visual acuity; convergence and accommodation; muscle balance for near and distance - either Maddox rod and wing or prism cover test; full detailed assessment of binocular vision including the angle of deviation and fixation of either eye; subjective tests of fusion and fusional reserve; stereopsis and stereo-acuity; suppression should be noted and reference eye

in the central binocular field recorded. Sighting eye (monocular test) may be noted for historical interest - this does not necessarily correspond with the reference eye.

The Reference Eye Test has been described (Dunlop, 1974) but discussion of the responses to the test may be helpful. The first essential is to determine whether useful binocular single vision exists in the central binocular field of 2° ... 3° around the point of bifoveal fixation. Where binocular single vision is present responses may be - a) R reference, b) L reference, c) alternating reference, d) lack of development of reference.

Where suppression of binocular vision is marked in some squints, anisometropia or amblyopia, there is no real binocular vision in the central overlapped field and thus no visual confusion of laterality can occur.

For example, one sometimes sees a child with anisometropia and amblyopia of a line or two in the eye corresponding to the preferred hand, coping well with reading and school work, because in such a case the reference eye mechanism can not exist and no such confusion of visual perception is possible. (Of course confusion of laterality of speech, auditory and motor functions could still be present.)

In the classic case of visual dyslexia, suppression is very rarely a problem and the child can appreciate physiological diplopia very easily. This is an interesting finding because these children often have trouble with figure ground work, where they have to pick out certain specific shapes from a complex drawing of many shapes. They have difficulty in suppressing the irrelevant background.

The reference eye assessment should be correlated with the existing handedness of the child so that the relation of the visual function to the manual laterality of the child may be classified as—

- a) normal correspondence,
- b) crossed correspondence
- c) alternating correspondence
- d) undeveloped correspondence

Following full orthoptic investigation and diagnosis the orthoptist is in a position to offer treatment in an effort to promote normal binocular functioning. This will include treatment for defects of —

- a) visual acuity such as amblyopia and suppression,
- b) ocular muscle balance and convergence and
- c) treatment to promote the desired corresponding reference eye in the central binocular field. a) and b) are normal parts of orthoptic therapy but c) is a new area for orthoptists and treatment is experimental at present.

Some centres in New South Wales are experimenting with total occlusion of the opposite eye to the desired reference eye during all close work and sometimes for TV also. This is supported by convergence training in the form of home exercises. It is necessary to keep convergence active because of the considerable periods of monocular activity for close work; convergence training is also necessary because these children characteristically show poorly maintained convergence. No case of amblyopia has yet occurred in spite of occlusion extending up to 18 months, during experimental work over three years.

This type of treatment has been successful in many cases. The children begin to respond after several months on the occlusion routine. Generally, writing begins to improve first, reversals tend to disappear and later reading begins to improve. Spelling appears to take longest to improve.

However, not all cases respond to this type of therapy. Even 12 - 18 months occlusion may not alter the side of the reference eye. In such cases parents and teachers have said that the child has improved but is still functioning below a desirable level. These cases seem to be quite resistant to our efforts just as do some cases of squint who fail to develop binocularity. It is probable that confusion of laterality in other modalities perpetuates their sequencing difficulties.

Orthoptic treatment is still experimental, and will take some years to fully assess in controlled studies.

The following analysis of 345 cases currently undergoing orthoptic treatment is not a pre-planned study, but a review of a fortuitous group of my patients with a common problem.

TABLE I
ANALYSIS OF 345 CASES CURRENTLY UNDERGOING ORTHOPTIC
TREATMENT

DIAGNOSIS	MALES	FEMALES	TOTAL
Crossed and unstable correspondence	201	49	250
Lack of and undeveloped correspondence	40	11	51
Convergence insufficiency with normal correspondence	31	3	34
Intermittent deviation			
Convergent	4	1	5
Divergent	2	1	3
S.O.T.S.S.	2		2
	280	65	345

Table I shows the distribution of various groups. **Crossed correspondence** (reference eye opposite to preferred hand) and unstable correspondence are grouped together and form the largest group, as they appear to have similar problems with reversals, reading and spelling. **Lack of reference and undeveloped reference** form another group where the patient gives no indication of lateralisation of visual function. When this persists (beyond the age of about six years) he often has poor spatial orientation and difficulty with lateralisation in reading and writing. **Convergence insufficiency** with normal correspondence forms quite a small group. These children rarely have any trouble with reversals, but misread, lose the place and lack concentration; they respond well to conventional orthoptic training in a relatively short time. **Intermittent deviation** forms another small group with a fairly even distribution of convergent to divergent deviations. It is interesting to note two cases of Brown's superior oblique tendon sheath syndrome in this group. There were no cases of constant strabismus. Half of the group with intermittent strabismus required surgery and subsequently responded well to treatment.

TABLE II
HANDEDNESS

	MALES	FEMALES
Right handed	235	51
Left handed	41	14
Ambidextrous	4	0
Total	280	65

$$\chi^2 = 1.944, df = 1. \quad \text{Not significant}$$

Table II shows the distribution of handedness by sex. Despite an apparent tendency toward sex differences in incidence of left-handedness in this group, the difference in incidence between males and females is statistically not significant.

**TABLE III
TREATMENT**

	MALES (280)	FEMALES (65)	TOTAL (345)
Glasses Prescribed	24	0	24
Convergence Training	280	64	344
Occlusion	252	60	312
Operation			
(con.)	2	1	5
(div.)	1	1	
Change of Hand	3	0	3

**TABLE IV
REPORTED RESULTS**

	MALES (280)	FEMALES (65)	TOTAL (345)
	%	%	
Fewer Reversals	265 (94.6)	59 (90.8)	324
Neater Writing	241 (86.1)	60 (92.3)	301
Better Reading	224 (80)	52 (80)	276
Improved Spelling	147 (52.5)	36 (55.4)	183

Table III shows how few children required refractive help although it is a real advantage to have even a small cylinder prescribed if necessary for the desired reference eye. Convergence training played a very important role in conjunction with occlusion in the treatment regime of most of the children. Convergence training by the usual daily home exercises has proved satisfactory. Occlusion is only used for close work at school and at home. (The eye opposite to the desired reference eye is occluded.) Total occlusion seems to work better than opaque transparencies, either on elastic around the head or on spectacle frames. Atropine occlusion was not used on any of these cases, but is being used as a trial in another group. Atropine has the disadvantage of lasting some ten days after instillation before it is possible to carry out a reasonably accurate test for reference eye even for a check up procedure; while the effects are present it masks the test.

Where surgery for an intermittent deviation was performed the response to subsequent orthoptic treatment was good.

Only three children made a change of hand for writing with favourable results in each case. This change was only undertaken after careful consideration by all involved members of the multi-disciplinary team.

Two boys in the crossed correspondence group had suffered broken arms during the pre-school period and had changed handedness of necessity. Neither of these children responded to occlusion therapy and the reference eye did not change over in 18 months. Learning was still a problem for them.

A number of young children had not established a dominant hand and in most, the reference eye was undeveloped also. Where the reference eye was established the hand was encouraged on that side. Otherwise it seems best to wait until more definite lateralisation is apparent, at least in the younger child. In the older child occlusion can be used to promote the desired dominance, again in consultation with other members of the team in deciding which hand is to be encouraged. New procedures to establish the side of the speech centre may be of great value in this type of case. Low (1973), Fenelon.

Table IV - Results of treatment for this group have been assessed from comments by teachers, parents, school counsellors and psychologists. Ideally all the children should have had educational assessments before and after treatment but this was not possible for various reasons in this unplanned group.

Fewer reversals were reported in a high percentage of the cases and this seemed to relate to co-operation and diligence in wearing the patch. Neater writing also occurred in high percentage and seemed to coincide with better co-ordination generally; the children seemed to be less clumsy. Reading was said to have improved, and many mothers reported a better attitude toward books and school work. Spelling was the slowest to improve and many children still had trouble even after eighteen months training.

The most pleasing outcome of treatment was the happier outlook of the children both at school and at home. The children seemed to be more relaxed and co-operative than before.

TABLE V
LENGTH OF TREATMENT

	NO. OF CASES
6 months	158
12 months	99
18 months	88
	345

Length of Treatment - 158 of the cases here reported have been under treatment for at least 6 months., 99 for 12 months, and 88 for 18 months. Treatment seems to take about a year to complete; if the reference eye has not changed in that time, it is dubious whether it ever will with further occlusion. In some cases it may be necessary to allow a child with an apparent crossed correspondence to maintain a small suppression area with persistent poor convergence, i.e. monocularly in the central overlapped field.

The orthoptist has a responsibility to fully assess all cases of learning difficulty referred to her and to communicate adequately with other members of the team. In this way a careful selection of cases suitable for orthoptic training may be given treatment, while useless and unwarranted interference may be avoided.

Acknowledgements:

My thanks are due to all the parents and children who have given me the opportunity to investigate this fascinating subject; to the school teachers, counsellors and psychologists who have referred the children and carried out much of the initial assessments; to the other members of the multi-disciplinary team for their expertise; to the ophthalmologists who have allowed me to pursue investigations when their own tests so often proved normal; to Mr. B. Fenelon for helpful advice with the statistics and to my typist, Christine Stringer, for her dedication to the production of this manuscript.

REFERENCES:

- Bishop, P.O., Henry, G.H., (1971) *Spatial Vision*, Ann. Rev. Psych. 22:152
- Brown, H.W., (1957) *Isolated Inferior Oblique Paralysis: An Analysis of 97 Cases*. Trans. Amer. Ophthal. Soc. 55: 415-154
- Dunlop, D.B., Dunlop, P. (1974) *Orthoptic Assessment of Children with Learning Difficulties*. Aust. J. Ophthal. 2: 113-116.
- Feneion, B., Personal communication.
- Hurt, J. (1971) *Role of the Orthoptist in Evaluation of Reading Disorders* Current Concepts in Dyslexia. Hartstein J. (Ed.), C.V. Mosby Co., St. Louis. 148-160.
- Joshua, D.E., Bishop, P.O., (1970) *Binocular Vision and Depth Discrimination: Receptive Field Disparities for Central and Peripheral Vision etc.* Exp Brain Res. 10: 389-416.
- Kambara, G.K. (1971) *Mirror Writing: Its Role in Reading Problems and Practical Suggestions in the Management of these Patients*. Amer. Orth. J. 21: 110-119.
- Low, M.D., Wada, J.A. and Fox, M., (1973) *Electro-encephalographic Localisation of Conative Aspects of Language Production in the Human Brain*. Paper read at the 3rd International Congress on Event Related Slow Potentials of the Brain. Bristol Aug., 1973
- Stone, J., Leicester, J. and Sherman, S.M. (1973) *The Naso-Temporal Division of the Monkey's Retina*. J. of Comp. Neurology 150: 3, 333.
- Ward, J., (1970) *Clinical Testing of Children for Reading Disability*. Amer. Orth. J. 20: 81-86.

DEVELOPMENT OF VISUAL ACUITY

Presented in Sydney, April 1975

Valerie Spooner, D.B.O.(T)

Has anything new been discovered about the actual visual acuity of the infant? It was decided to find out what has been written recently on this subject.

The optokinetic drum has been used to produce nystagmus to evaluate vision. Optokinetic nystagmus is a visuo-motor reflex which can be elicited in normal circumstances at an even earlier age than the light fixation and following reflexes. However, until very recently an accurately graded drum could not be bought. To quantify the response it is necessary to have the black and white stripes so graded as to be equivalent to the Snellen's test chart. It is also important that the drum should rotate at a given speed.

Now two ophthalmologists have improved this instrument. Their version has six circular targets, of values equivalent to Snellen type. A motor rotates the drum. It is held 60cms from the baby and is a quick and easy test to assess visual acuity. Using their improved drum, Catford and Oliver (1973) did a survey on adults whose vision was known. The drum proved very accurate. Encouraged by this they did a survey of children under the age of one year. They found that babies fixate from two weeks of age, but optokinetic nystagmus could not be induced until six weeks. Acuity increases to 6/18 at five months, to 6/12 at 18 months, to 6/9 at two years and to 6/6 at three years.

Thus they have proved that the infant sees better than other authorities believed. Many of our text books state that vision is much lower in babies under one year. Their estimates were based on deduction. The work of Catford and Oliver has proved the better vision objectively.

It is interesting to realise that a three year old child already has normal adult vision for distance and near. The ciliary muscle is fully developed at this age and so the child is capable of good, prolonged accommodation.

Perhaps therefore we should not insist that toys and picture books should be large. The only reason to give a learner large print to read is that it enables him to consider a few letters at a time, a question of comprehension rather than vision.

Other authors including Ruskell (1967) agree that there is a high level of visual acuity at birth. Yet in the general literature it is stated that the fovea and macula are

not developed in the neonate. Mann (1928) showed that the macula continues to differentiate for more than four months after birth. Modern research has shown that the fine structure of the foveal cone is present in the neonate, which supports the belief that the infant is capable of good vision.

It has also been stated in many text books that the neonate is hypermetropic. However some surveys have been done recently.

In U.S.A. Cook and Glasscock (1951) found 25% were myopic.

In Japan Miyake and others (1962) found 5% were myopic.

In India Mehre and others (1965) found 20% were myopic.

In U.K. Graham and Gray (1963) found 5% were myopic.

These figures appear to disagree, but they do indicate that many children (rather than isolated cases) have congenital myopia. It would be interesting to follow up these cases. Does the myopia diminish or increase with age?

Until the motorized optokinetic drum is available in Australia, orthoptists should know the following typical stages in the development of visual acuity.

- | | |
|------------------|---|
| At 2/52 | The child is capable of fixing a light,
He will turn his face towards a light source.
Some convergence is elicited
Doll's head phenomenon is present. |
| At 5/52 - 6/52 | He stares at large objects. Versions with fixation and refixation are possible, i.e. eyes follow movement. |
| At 2/12 | Eyes follow person or large toy. The near-spherical lens of the infant has a fixed focus of about 12 inches, which gradually changes as accommodation develops.
Convergence is good and well maintained
The child may be able to overcome a 20 ^Δ base out prism. |
| At 3/12 | Eyes follow small toy; the head is moved as well. |
| At 4/12 | The child holds head up. Inspects his own hand, reaches towards object, can overcome 20 ^Δ base out. |
| At 6/12 | The body follows head and eyes to the extent of sitting up. |
| At 12/12 - 18/12 | Child is able to pick up small sweets such as jelly beans or smarties. |
| At 1½ - 2½ yrs. | Child is able to pick up small cake decorations such as "hundreds and thousands". |

The orthoptist is also aided in the assessment of visual acuity by the comparison of behaviour when one eye is covered. If one eye, but not the other, can be covered without fuss, then vision is not equal. Again, if small sweets or cake decorations can be picked up with one eye covered, but not with the other eye covered, this too indicates inequality.

The assessment of visual acuity is always difficult in the pre-school child, but it is even more difficult in those under the age of 3½ years. The orthoptist needs patience, experience, and keen observation.

REFERENCES

- Brown, I.A.R., *Brit. Orthopt. J.* (1974) 31, 83
Catford, G.V., & Oliver, A., *Arch. Dis. Child.* (1973) 48, 47
Cook, R.C. & Glasscock, R.E., *Amer. J. Ophthal.* (1951) 34, 1407
Ruskell, G.L., *Brit. Orthopt. J.* (1967) 24, 25
Mann, I.C., "*The Development of the Human Eye*" (1928), pp. 114, 236 Cambridge University Press, London
Mehra, K.S., Khare, B.B. & Vaithilingam, E., *Brit. J. Ophthal.* (1965) 49, 276
Miyake, S., Katayama, S. & Honma, C., *Pract. Ophthal.* (1962) 56, 774
Wybar, K. *Brit. J. Ophthal.* (1974) 58, 483.

THE EDUCATION OF ORTHOPTISTS IN NEW SOUTH WALES

Patricia M. Lance (Sydney)

Presented in Sydney, 1975.

Recent events, which have brought about startling changes in the education of orthoptists in the State of New South Wales, make it desirable to review the education of orthoptists in the past and to consider how far such education should be extended in the future.

In the early 1930's when orthoptics commenced in this country, the training of orthoptists was conducted on a very personal, rather amateurish but highly ethical basis. The first orthoptists were trained by a small number of very enthusiastic young ophthalmologists who had worked with orthoptists in the United Kingdom. These men were helped and encouraged by some of their older colleagues whose interest in the treatment of Strabismus had been aroused by their experience in practice. Orthoptic clinics were established and many of the early difficulties were overcome by very close co-operation between orthoptists and ophthalmologists. As these pioneer orthoptists gained in confidence and experience they were encouraged to pass on this knowledge to others and so the numbers of practising orthoptists gradually increased.

At the request of the Hospitals Commission of New South Wales, the Ophthalmological Society of New South Wales in 1938 appointed a sub-committee, known as the Orthoptic Council of New South Wales, to regulate the training and registration of orthoptists in this State. This committee drew up a syllabus based on that of the British Orthoptic Board, and in 1939 two students were accepted for the first regular course in orthoptics. In this and the next two courses conducted in 1941 and 1943 each student was assigned to one orthoptist for her whole training, so there was typical master-apprentice relationship. A few lectures in ocular anatomy, physiology and optics were given by ophthalmologists. Students were required to study these subjects from post-graduate texts.

In 1947 on the advice of all practising orthoptists in Sydney, the Orthoptic Council appointed a part-time orthoptic tutor and course co-ordinator (Patricia Lance) to assist the students. At this time orthoptists worked only a few sessions per week at any hospital, so most students had to travel with the clinical orthoptist to different hospitals during the week. With no one hospital for the training centre, the tutor orthoptist had to move her lecture room from place to place, and had no permanent office for many years. As the need for more intensive training of orthoptists became apparent the course increased from twelve months to eighteen months, and then to two years in 1956. Annual intake of students commenced in 1953 and an average of six new students per year were accepted by 1958 and the Sydney Eye Hospital became the headquarters for the training.

In 1938 the newly-formed Ophthalmological Society of Australia appointed a sub-committee, the Orthoptic Board of Australia, to co-ordinate the training and registration of all orthoptists in Australia, and in 1947 reciprocity was granted by the British Orthoptic Board. Revision of the syllabus was made from time to time and joint examinations between the Schools in Melbourne and Sydney were held from 1962. Meanwhile, the Orthop-

tic Association of Australia, formed in 1943, facilitated many useful discussions between orthoptists concerned with the training of students, especially in 1964 when a teaching seminar was held.

In 1970 it became obvious that orthoptists would have to be even more closely concerned with the education of their profession, when the Werner report indicated that orthoptics should join other paramedical professions in a special college of paramedical studies. On July 1st, 1973 the New South Wales College of Paramedical, (now renamed as the Cumberland College of Health Sciences) took over the orthoptic course at that time conducted by the Orthoptic Board of Australia (New South Wales Branch), and an orthoptist, (Patricia Lance) was appointed Head of the School.

Thus, for the first time an orthoptist had full responsibility of the education of orthoptic students. It has proved to be a heavy one. We have found to our cost that it is far harder to describe our teaching plans to a government body than to a professional one.

It became necessary to prepare a document for the Advanced Education Board outlining the course, describing its nature, the need for such a course, its relation to other courses in Australia and overseas, its proposed content, organisation and implementation. It supported a submission for a three year UG2 Diploma of Applied Science. This considerable task was achieved on time, thanks to assistance from the Principal of the College, (Dr. J.O. Miller), the Assistant Principal, (Dr. R. Rawlinson) and some of the educationalists who helped with the layout and writing of the submission, while Miss Valerie Spooner D.B.O. (T), the visiting lecturer from the United Kingdom, took over the orthoptic lecturing. From outside the College the profession rallied in its usual spirit of co-operation, to help with the clinical training of current students, to act on the School's external advisory committee, and even on the Advanced Education Board's external advisory committee. Similar co-operation was received from members of the Orthoptic Board of Australia (New South Wales Branch) and other ophthalmologists.

It was a great disappointment for all concerned when in December 1974 word came back that the three year (six semester) proposal for a UG2 Diploma Course in Orthoptics had been disallowed, but that the Board had agreed on a two and a half year (five semester) UG3 Associate Diploma Course. This meant more work. A complete restructuring has now been completed. In February this year (1975) the first group of students commenced the new five-semester course from which they should graduate in mid 1977. Consideration will be given to re-submission of a three-year diploma course proposal in the future.

In developing the orthoptic course, our broad aims were considered, of developing the student's professional competence to meet community needs while furthering her growth in knowledge, skill and wisdom. The submission listed the specific abilities desirable in the graduate, and the basic directional and educational principles underlying the course. The orthoptic process was seen as the unifying concept. This was expressed in the submission as follows:

"The concept of an orthoptic process is derived from a functional analysis of the role of an orthoptist. The process steps are defined in broad terms as:

- Investigation of the patient's visual and ocular motor system.
- Classification of the patient's ocular deviation based on the results of investigation.
- The development of a plan of patient management based on an accurate assessment and prognosis.
- Orthoptic management including orthoptic procedures for therapeutic processes, continuing supervision and assessment.
- Evaluation of patient progress including recommendation regarding further treatment or cessation of treatment (based on recognised standards for cessation of treatment)."

In the proposed six-semester course, the first phase was orientated towards gaining an overview of the orthoptic process. Considerable emphasis was placed on the underpinning studies in behavioural and biological sciences and on an introduction to orthoptic management techniques. In the second phase of the course the orthoptic process provided a recurring framework for the study of each type of deviation and in the third phase the student was introduced to specialised areas of orthoptics, including the visual problems of the handicapped, and special areas of ophthalmology. Provision was made in the form of electives, for students to pursue particular areas of clinical interest.

The module structure of this course has been retained in the five semester course, the main differences being in the hours spent on each unit and the dropping of the twelve-week elective. It is hoped that our aims will still be achieved. For this it is necessary to improve both the teaching methods and the student's learning techniques. Teaching now is not just a matter of giving lectures and handing out a few notes - it involves tutorials and seminars, written work and viva voce examinations, projects and assignments, case study and discussion. Audio-visual equipment is available to improve the standard of lectures and demonstrations, and the lecturers are becoming familiar with the use of overhead projectors, slides, films and video-tapes.

Learning is most effective when the student works things out for herself. The "orthoptic process" concept provides for this. In following through the orthoptic process, applying her theoretical knowledge to the interpretation of clinical data of increasing complexity, the student is helped to integrate orthoptic theory with clinical practice; she gains competence and confidence in decision making, and is stimulated towards an analytical and evaluating approach.

The most obvious difficulty in augmenting the curriculum is the relation and balance between formal academic work and clinical experience. As has been seen, orthoptic education in its early stages was largely a matter of clinical training. The academic side has been gradually increased and grafted into the existing clinical experiences. Now with absorption of the academic side into a college of advanced education, there is a danger that a gulf could grow between these two aspects of the student's education.

The School must not grow too far away from the clinical field; by the same token, clinical orthoptists must strive to keep up to date with theoretical knowledge. Students tend to take more notice of what they see than of what they read or are told. Their clinical experience must be of the highest standard. It is essential that academic and clinical staff retain the closest possible contact.

The final problem to consider is assessment of the student's performance, one of the most difficult aspects of teaching. What criteria are to be used? How is it possible to know who are safe to be let loose on an unsuspecting public? Most authorities seem to agree that continual assessment is fairer than for all to depend on one final examination. But is there any objective way of assessing clinical expertise? Can it be equated with academic expertise? Which is more important in the education of the orthoptist? There are no clear cut rules to follow. Lecturers, examiners, and clinical supervisors can only do their best to be fair both to the student and to the community at large.

All these points are matters of much concern for those involved with students at the Cumberland College. Workshops and seminars are being held on teaching methods and there is an excellent forum for multi-disciplinary discussion, especially between the "health professionals" and those teaching behavioural and biological science. The educationalists in the College are available to help those who need it in the planning stage or in the implementation of programmes.

The field of education is very challenging but always exciting. The orthoptic members of the College hope that from time to time they will be able to report interesting developments to these conferences, and that the relationship between the College and the Orthoptic Association of Australia will always be close and mutually stimulating.

THE USE OF "HALF PATCH" OCCLUSION

Melinda Hansor

Presented in Sydney, April 1975

Most orthoptists are familiar with the use of occlusion of part of a lens in cases of paralytic squints, i.e. in occluding the area in which diplopia is experienced, thus allowing binocular vision in other directions of conjugate gaze. Also, in cases where hyperopia is manifest in depression only, suppression of the hypertropic eye is prevented by occluding the lower half of the other lens.

In the treatment of intermittent convergent squints with a refractive error, where the cover test shows unstable esophoria for distance and a convergent squint for near or on depression, I have found the use of half patch occlusion to be helpful in the elimination of suppression and in stabilising binocular function.

The lower section of the lens of the fixing eye is covered, the upper edge of the cover being so placed that binocular single vision can be maintained in the primary position and in all positions of gaze except depression. Thus for activities such as reading or writing during which manifest deviation is likely to occur, the fixing eye is occluded. The material chosen depends on the personal preference of the patient or the orthoptist.

The use of half patch occlusion has particular advantages in the following areas -

- (1) to prevent suppression, once it has been overcome by the usual methods, until other treatment is begun, whether it be surgery, orthoptics, or temporary measures such as miotics, bifocals, or Fresnel prisms.
- (2) during the course of orthoptic treatment
- (3) where there has been no response to the usual methods of eliminating suppression
- (4) for the patients who do not respond well to transparencies, and others, particularly older people, who find it difficult to fuse one clear and one blurred image, or are disturbed by continued diplopia for near.
- (5) in cases where the maintenance of binocular single vision for distance is considered important because binocular vision is weak.

As with most orthoptic treatment, when contemplating half patch occlusion one must consider the age and co-operation of the patient, and if the patient is a child, the parents' co-operation as well. A simple explanation is usually sufficient. After some problems in adapting to this method in the first couple of weeks, most patients have no complaints. With the younger ones, there is always the problem of "peeping", which is easy for the tempted child.

The following are summaries of three case histories where half patch occlusion was used in conjunction with other treatment.

Louise aged 12 years wore a low hypermetropic correction. Her intermittent right convergent squint had apparently responded well to orthoptic treatment some five years previously. Now, with increased close work, she noticed her right eye wandering. Progress with part-time left occlusion was slow, so half-lens clear occlusion was suggested. Louise found she could adapt easily. In the initial stages, whenever the right eye felt strained after close work, she could look over the occlusion. She reached a stage at which she could use the right eye comfortably for all near work, with no sign of suppression. After surgery and further orthoptics Louise was able to go without her glasses, and remained symptom-free.

Helen, aged 5, wore a low hypermetropic correction and had a history of left convergent squint and amblyopia. After constant right occlusion to equal visual acuity, part time occlusion was continued until she was able to control the deviation for distance at times. But owing to marked left convergent squint for near, suppression recurred whenever occlusion was stopped. As the deviation was extremely variable and the binocular function unstable, surgery although probably inevitable had been delayed. Clear half-lens occlusion was commenced with explanation to parents and child, and continued until miotics were started. With these treatments, suppression has been prevented and the binocular function stabilised, Helen maintaining control over a moderate esophoria.

Michael, an intelligent and helpful child aged 4, was esophoric for distance when wearing glasses, but after left occlusion which brought the right vision up from 6/12 to 6/6 there was still a right convergent squint with troublesome diplopia for near. With half patch brown paper occlusion, suppression was overcome, the manifest deviation for near became an esophoria, and there was no more double vision. This standard was maintained after all occlusion was abandoned.

From my experience, having used half patch occlusion in most cases where part-time occlusion might be considered, the former really only has value in the treatment of intermittent convergent squints, as here described. More than anyone else, I can appreciate the limitations of such a method, but am convinced it definitely has merits for certain patients. Perhaps the greatest advantage is as a continuous form of management.

REFERENCE:

Duke Elder, S., and Wybar, K. (1973) *System of Ophthalmology VI*, Kimpton, London.

OBJECTIVE ORTHOPTIC TREATMENT

Diana Craig

Presented in Sydney, April 1975

Introduction

This paper is about a search for more effective methods of orthoptic treatment than are offered in text books. Where guidance from neurology leaves gaps, these have been tentatively filled in the light of orthoptic experience. "Those that go down to the sea in ships ... these see the works of the Lord and his wonders in the deep." The orthoptist deeply involved in the troubles of her patient across the synoptophore, perhaps gains glimpses of the workings of visuo-motor mechanisms that are hidden from others.

The dual control of ocular movements

In 1938 Gordon Holmes described the functions of the two cerebral oculo-motor centres in man, as deduced from study of the eye disabilities occurring when the projection tracts from either centre to the brain stem were injured or diseased. When the frontal projection fibres were affected, the patient's eye moved freely, but not by his intention. They would follow people moving in the room, or an object slowly moved towards or away from him, but by no effort of will could he turn them from one thing to another. When the occipital cortico-tecal pathways were bilaterally interrupted, voluntary movement was free, but ability to watch moving objects or to maintain clear single vision of near or distant objects was lost.

From the sum of his observations, Holmes concluded that "the frontal oculomotor centre is concerned in those movements and reactions of the eyes which we may call voluntary. Through it we can by an effort of will look or turn our eyes in any direction

and converge them on a near object." .."In normal conditions there is an accurately adjusted co-operation between all parts of the cortex concerned in ocular movements. Through the occipital centres some visual reflexes excite movements which turn our eyes towards objects in the field of vision; other reflexes determine by fusion and accommodation the accurate perception and the unification of binocular images; others keep the eyes fixed on any point which claims attention or which excites interest. The frontal centres make possible the turning of gaze in any desired direction and the exploration of space, but they also keep under control, or inhibit, reflexes that are not appropriate to our conduct or our reactions to the world around us."

Dual control applied to convergence deficiency

Mann (1940) called attention to the significance of Holmes' paper in explaining the successful management of convergence deficiency (insufficiency). She suggested that the signs and symptoms were due to lack of frontal oculo-motor involvement, and that by teaching voluntary convergence one activated frontal convergence to facilitate the reflex occipital function.

Inhibition and facilitation

For the meaning of facilitation one must turn to Sherrington (1906). From study of the interacting spinal reflexes of dogs, Sherrington recognised that when different reflex arcs share the same muscle or muscles as effector organ, they also share a final common path in the motor nerve supplying that organ; the motor nerve nucleus, the point at which the various reflex arcs and output paths from higher centres impinge on each other, has a special role. A spinal nerve nucleus is a co-ordinating mechanism arranged to fire in response to incoming signals in such a way that a useful response is produced. An algebraic summing of incoming impulses is not to be expected. If the separate trains of impulses are antagonistic, some tending to inhibit and some to excite the action of the effector muscle(s), the resultant action is not a compromise; at any time, one set of impulses dominates the final common path.

Allied impulses from different sources, whether all excitatory, reinforce each other. The main effect of reinforcement is that the action is more certain to occur and better maintained. It may or may not make the action more vigorous or more extensive; there is wide variation in this respect, related to the importance of various actions for the individual's well-being.

Facilitation in action

Ida Mann's application of this concept was light in darkness to orthoptists of that era. The development of strong "fusion" by synoptophore training was accepted as the orthoptist's essential task. It was often a disappointing one. The value of voluntary convergence training in certain cases was recognised. The news that a different cerebral centre was involved in this gave a good physiological reason for its greater success, and justified its more whole-hearted use.

There can be no doubt that in treating convergence defects, results are quicker and surer if one aims first at obtaining convergence awareness and control away from the synoptophore. The most potent stimulus to convergence as well as accommodation is to arouse curiosity about detail in an approaching object. Once control is established, the synoptophore is useful in allowing the patient to feel the interaction of the two mechanisms in maintaining or rejoining fusion over the required range. One can, so to speak, watch the process of facilitation at work.

The improved results in convergence training naturally led to a trial of similar methods in the treatment of esophoria and accommodative squint. Of course it was found again that results are better if attention is primarily concentrated on exercises which de-

velop conscious control over convergence, rather than on fusion training. (The question of suppression will be discussed later). But if a new approach is to be accepted wholeheartedly, particularly if it is to be presented to students, a satisfactory explanation is desirable. It was doubtful whether the reinforcement explanation was applicable here. Another journal article led to the answer.

Ittelson & Ames : 3-card Trick and Card-shrinking

A very interesting series of experiments were carried out by Ittelson and Ames (1950) to whom Holmes' findings were apparently unknown. In what one might call their Three Card Trick, subjects were shown in turn each of three playing cards, visible monocularly at a distance of 32 inches in an otherwise completely darkened room. The cards were of normal, double and half sizes. Having looked at a card, the subjects turned to direct the experimenter in adjusting the distance of a post seen binocularly in normal lighting until it appeared to be the same distance from him as the card.

Four men observers, aged 30 to 45 years showed good judgment in choosing distances which would account for the size differences if all the cards had actually been of normal size. A 15-year-old boy was influenced by the card sizes, but to a lesser extent.

Each card was in fact seen through a half-silvered mirror, and a similar mirror with a shield beyond it confronted the other eye. Each mirror reflected a star point to the corresponding eye. Therefore the observer saw two star points, superimposed on the playing card in front of him. Suitable adjustment of lenses and of the mirror angle, until a single and clearly defined star appeared on the card, provided measurements of the accommodation and convergence exerted by the viewer. (Incidentally this method might also allow time for the subjects first response to the size of the target to subside somewhat.)

Measurements of the boy's accommodation varied only slightly, and in the wrong direction; his convergence hardly varied at all. In the adult subjects, convergence and accommodation were always greatest for the large card, and least for the small one. Although the changes were less than would be fully appropriate for the apparent changes in distance, one might say there was evidence, not only of a near response, but of a far response also.

In a further experiment which might be dubbed the Card-Shrinking Trick, the image of a playing card was projected on a screen at 16 inches. While each observer watched it, the size was changed continuously from that of a normal card, to one-third of that size. This gave the effect of movement to 4 feet away.

This time the measured changes in convergence and accommodation were greater. Most subjects, moreover, reported that as the card grew smaller, it blurred. One of them accused the experimenter of mismanaging his projector. But when the image was held at the small size, most of the observers reported that it "slowly, and completely beyond their control, cleared up."

Finally, three observers watched the image binocularly, while the size was rapidly and continuously changed. Accommodation decreased. There was no change in convergence. The observers all reported that the card moved further away, and blurred as it reached the far point. In addition, all reported a feeling of strain about the eye with discomfort which lasted for about an hour afterwards. Ittelson and Ames comment that this may account for complaints of eye strain after prolonged close television viewing.

The Near/far response and voluntary convergence

The card-shrinking trick gives evidence of three separate but interacting involuntary functions, namely

- 1) one providing accommodation and convergence changes in response to apparent changes in distance of a familiar object, which one might call the near/far response,
- 2) the accommodation reflex, action of which is seen in the unexpected clearing of the target at the distal point,
- 3) the fusional vergence reflex which in the binocular part of the experiment, kept the eyes accurately converging on the target in spite of the size change.

Significantly, symptoms occurred when fusion(3) acted in opposition to (1).

The fact that responses to the size changes were made quite unconsciously and without intention forces one to reconsider the meaning of "effort of will" and "voluntary convergence" as used by Holmes and Mann. In fact, just such responses are likely to be typical of frontal oculo-motor activity as it affects disjugate movement. Holmes wrote "Through it we can by an effort of will...converge (our eyes) on a near object." But the truth is that none of us under ordinary circumstance do converge our eyes intentionally on a near object. Even when by training we become aware of convergence as a positive action and of our power to control it, we do not make any sort of decision to converge or diverge as we look from far to near, or consider vergence as an act of choice as we sometimes do of conjugate changes of gaze.

Vergence movements in fact fit Sherrington's (1955) description of habits: "they are reactions to a mental situation rather than to any simple stimulus...These trains of reaction have become automatic, though at first attended by acute and critical awareness," Changes in fixation must be among the earliest, intentional acts to be mastered, and perhaps for that reason we are often unaware of making them. When we are aware, we do not subjectively distinguish vergence components from the conjugate components of the change.

One can feel sure, for instance, that Holmes' patients could be aware of, and could complain of, their inability to bring their eyes quickly to focus on a near object, without ever having been aware of this necessary ability while they still had it.

Thus we believe there is justification for accepting the near/far responses demonstrated by Ittelson and Ames as being typical of normal frontal vergence activity. It is probably acquired in the first year of life, conditioned by experience to reinforce the vergence and accommodation reflexes during slow changes of fixation distance, and to initiate immediate greater changes when required.

The etiology of convergence insufficiency

Mann suggested that voluntary convergence, the power to converge the eyes without a near fixation object, is usually acquired early in life, and that those who fail or are slow to acquire are liable to difficulties over near work. For reasons to be recounted, some of us now believe that there is actual unconscious inhibition of convergence in most cases of convergence insufficiency.

We have found that many patients under treatment make no progress until the subject of physiological diplopia is raised. Some bring it up themselves; others welcome the orthoptists' mention of it, others deny any concern. But most of them recall

childhood incidents when they were scolded for attempting the dangerous game of going cross-eyed. Once the matter is explained, and reassurance given, progress is normal.

Is it too much to accept that we can unconsciously inhibit actions that are unconsciously performed? The writer of this paper, well experienced in converging and in training those who could not, used to take pride in flower arrangement, but became concerned about the common after-effect, a blurring of vision which persisted 30 minutes or more. She wondered if some allergic reaction might be involved. Then a psychology professor consulted her about his problem, a blurring of vision which occurred repeatedly when, in his work as journal editor, he selected papers for publication. He did not, he emphasised, have to read them in detail - a general impression of topic and setting-out was sufficient. The orthoptist already knew her psychology, primed by his own lectures. She pointed out that the psychologist must be, in effect, instructing himself not to look closely - i.e. not to accommodate. He had only, on such occasions, briefly to examine his own finger tip, and he would recover. At this moment, she realised she had the answer to her own problem. She had deliberately ignored detail, looking for broad effect, in her flower arrangement. Now and again similar cases crop up.

In short, we came to the conclusion that convergence insufficiency as we know it is probably due to habitual inhibition of convergence and accommodation, by a mental rejection of the near response and due in some way to the thoughts, intentions and experiences of the sufferers. This explains the familiar signs and symptoms. We (skilled in orthoptic tricks) can if we wish interrupt fusion so that a book in our hands appears blurred and double; so we know that if inhibitory innervation is relatively strong, there must be manifest deviation for near. If it is less intense, there must be exophoria and discomfort without deviation, just as in Ittelson & Ames diminishing card trick.

Clearly there is here a departure from Sherrington's one-or-the-other rule for competing innervations. But he was dealing with spinal co-ordinating centres, we with mid-brain ones. (Discomfort as a result of unresolved cerebral conflicts is all too common!)

The dual involuntary vergence systems: a hypothesis

The considerations outlined in the last two sections led us to believe that two involuntary systems regulate convergence and accommodation, working in parallel. On one hand are the occipital, essentially unconditioned, accommodation and fusion reflexes, on the other the habits or conditioned responses mediated by frontal oculo-motor centres, and typically seen as the near response and its antithesis, the far response, in normal binocular vision.

What is called proximal convergence is one aspect of the near response. For those interested in accommodation - convergence relationships this is worth noting. Burian, following Morgan, denied the existence of proximal accommodation because experimentally, the total accommodative change is the same, whether measured on a distant fixation target with and without added -3.00 dioptre spheres, or with normal correction only at 6 metres and at 1/3 metre. Their reasoning assumes that innervation from 2 sources necessarily stimulates a greater reaction than from one acting alone; this is an unjustifiable assumption according to Sherrington. Proximal accommodation was demonstrated by Ittelson and Ames, and is known to us when we experience blur as well as diplopia during voluntary convergence.

Next to the inhibition of convergence which gives rise to convergence insufficiency the most common abnormal habit is excessive use of the near response for the sake of clear vision in hypermetropia, evident as accommodative squint. Similar habits of more obscure origin produce what we call convergence spasm. In fact variability in concomitant deviations, may usually be attributed to acquired responses of one sort or another.

Clearly much of this is supposition. Those who accept the concept of convergence habit, do so because it is a useful guide, which makes treatment shorter and which patients accept and work at because they can see its purpose.

Treatment

The methods of treatment are fairly traditional but the emphasis is different, as described earlier. They are surprisingly effective in cases of convergence spasm and esophoria.

Once attention was concentrated on motor responses it became clear that fusional vergence is a complex process. If one eye has been favoured over a period of time a conditioned inhibition of fixation develops, affecting the deviating eye. Even when by voluntary effort it is brought into position for binocular fixation, this eye fails to fixate into position. We judge the need for occlusion now, on fixation behaviour rather than on subjective evidence of suppression...finding that this ensures sufficient treatment and avoids what is unnecessary.

Acknowledgement

The writer is indebted to colleagues in England, the U.S.A. and all Australian States for the fruits of their experience and particularly to the late J. Ringland Anderson as teacher, Emmie Russell for an objective approach to Suppression and Beverley Balfour for unflinching encouragement.

Summary

Gordon Holmes' distinction between the rôle of the occipital oculo-motor centre, concerned with fixational, fusional and accommodation reflexes, and that of frontal centres which are concerned with voluntary eye movements, was quoted by Mann to explain the cure of convergence deficiency through instruction in voluntary convergence. Convergence and accommodation are not ordinarily conscious actions, but the frontal oculo-motor centre is seen by the writer as responsible for changes in both functions in response to perceived changes in fixation distance, as demonstrated by Ittelson and Ames, and also for certain mal-adaptive convergence habits. Such habits, it is claimed, underly any binocular disorder which is susceptible to improvement by orthoptic treatment. They may be cured by making the patient aware of vergence movements and of his power to control them. Fusional vergence is regarded as innate reflex, but a complex one, involving active fixation of each eye. Its function in the control of ocular deviations can be improved not by direct training, but by frontal facilitation and by suitable occlusion of the preferred eye.

REFERENCES:

- Burian, H.M. (1960) *Brit. Orthopt. J.* 17, 12
 Holmes, Gordon (1938) *Brit. Med. J.* 2, 107.
 Ittelson, W.H. & Ames, A. (1950) *J. Psychol.*, 30, 43
 Mann, I. (1940) *Brit. J. Ophthal.*, 24, 373.
 Morgan, M.W. (1952) *A.M.A. Arch. Ophthal.*, 47, 745.
 Pratt-Johnson, (1969) *Brit. Orthop. J.* 26, 15.
 Sherrington, C.S. (1906) *The Integrative Action of the Nervous System*, Constable, London.
 Sherrington, C.S. (1955) *Man and his Nature*. Penguin Books, Mitcham.

SUPERIOR OBLIQUE SURGERY

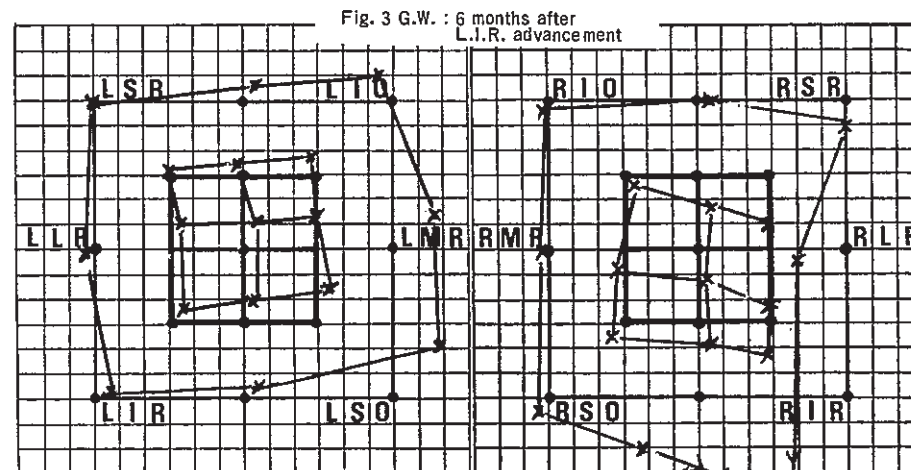
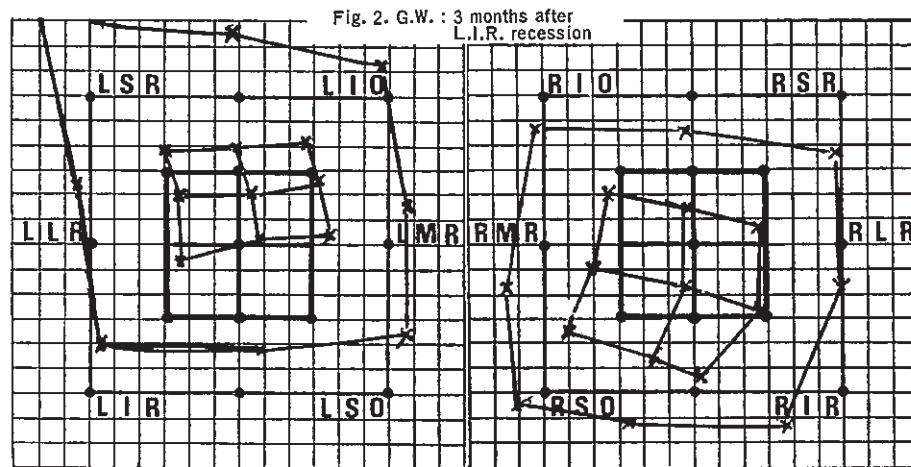
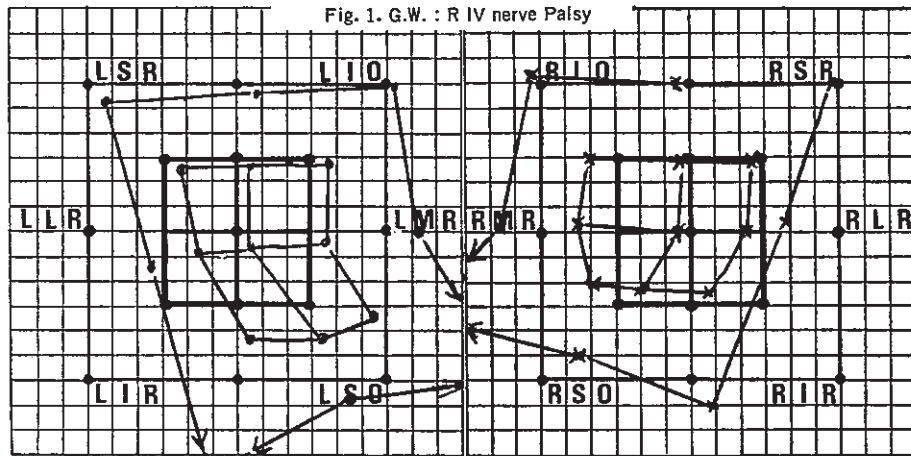
Jeanette Yap

Presented in Sydney, April 1975

The logical treatment of a paralytic deviation is to strengthen the affected muscle and/or to weaken its direct antagonist. Other methods of attack are unsound, in our experience. We have frequently seen disastrous results from the surgical treatment often recommended for superior oblique palsy, namely weakening of the contralateral inferior rectus or of the ipsilateral inferior oblique. The following case history illustrates this point.

GW suffered a traumatic right IV nerve palsy. A typical Hess Chart (Fig.1) was obtained 12 months after the accident. Recession of the left inferior rectus resulted in left hypertropia, as shown in the chart (Fig.2) taken 3 months later. A second operation, left inferior rectus advancement was then performed. Six months later the Hess Chart (Fig. 3) shows further incomitancy, and the patient is no better than before surgery.

In such cases a more desirable procedure is one which increases the efficiency of the paralytic muscle. In the past, attempts to do this in cases of superior oblique palsy have been avoided as too difficult.



During the past few years, surgeons at the Prince of Wales Hospital have been using a simple technique called Sagittalisation of the Superior Oblique.

Operative procedure: the usual fornix incision is made through the conjunctiva and Tenon's capsule. The superior rectus muscle is pulled up with a squint hook, and then half the superior oblique is split back to the trochlea (Fig.4). The anterior half of the muscle is cut off its original insertion, and sutured to the sclera above the upper border of the lateral rectus muscle. (Fig.5)

Sagittalisation of Superior Oblique

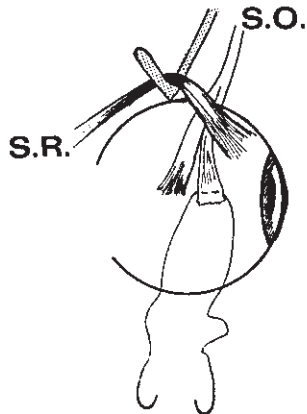


Fig. 4
Superior Oblique split back to trochlea

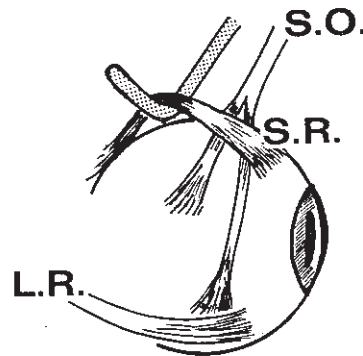


Fig. 5
Advancement of anterior half of Superior Oblique

Advancement of the rectus muscle has little effect because it is such a short distance from the limbus. The oblique, however, is ideally suited for the advancement procedure because of its sagittal approach to the globe. Up to 15mm of advancement can be performed on the superior oblique muscle, thereby increasing its effectivity in a direction close to that of its normal action. In no other way can the torsion, hypertropia, and esotropia caused by superior oblique weakness be so reduced.

The following are case histories of two patients on which this operation has been used.

CASE I

JS, aged 32, was involved in a motor-bike accident 10 months before seeing us in the orthoptic clinic; he was complaining of diplopia and difficulty walking down steps. He suffered severe head injury and had a traumatic brain stem syndrome.

The Hess Chart (Fig. 6) and synoptophore chart confirmed the presence of a left superior oblique palsy. The Bielchowsky test with head tilt to the left was positive.

Synoptophore angles were

in primary position, fixing right eye 0, L/R 4 Δ , excyclo 1 $^{\circ}$

fixing left eye 0, L/R 5 Δ , excyclo 5 $^{\circ}$,

in 15 $^{\circ}$ depression fixing right eye + 2 $^{\circ}$, L/R 9 Δ , excyclo 5 $^{\circ}$

fixing left eye + 3 $^{\circ}$, L/R 10 Δ , excyclo 8 $^{\circ}$

14 months later, there being no significant change, sagittalisation of the left superior oblique muscle was performed.

The post-operative Hess Chart (Fig. 7) showed an overcorrection; and there was 5 Δ left hypertropia. As the oedema and swelling reduced, the hypotropia decreased. JS was instructed to try to join the diplopia, and to extend his field of binocular vision.

A further chart (Fig. 8) was plotted 6 weeks after surgery; there was now an exophoria of 4 Δ with binocular single vision in all directions of gaze.

Fig. 6 JS Left IV nerve palsy
duration 10 months

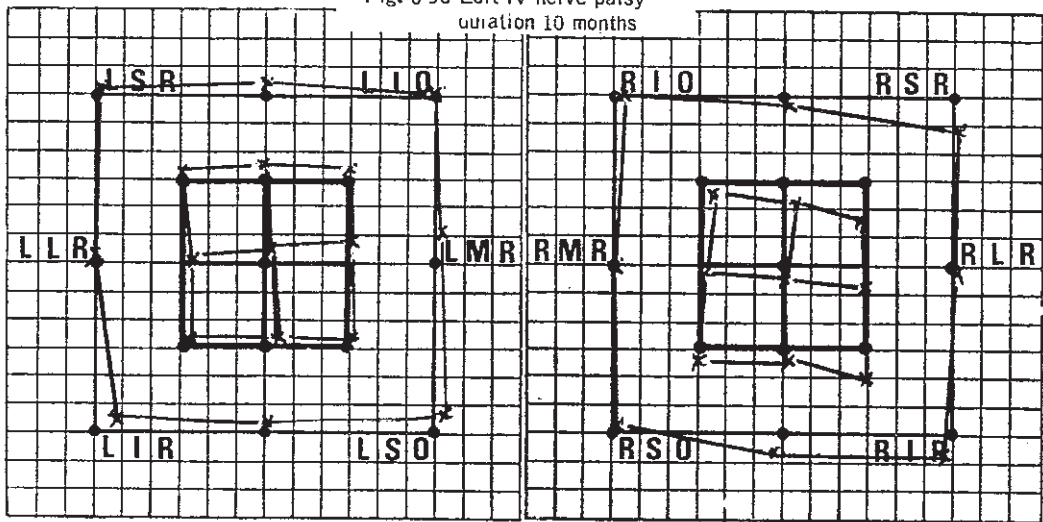


Fig. 7 JS After sagittalisation
of L.S.O.

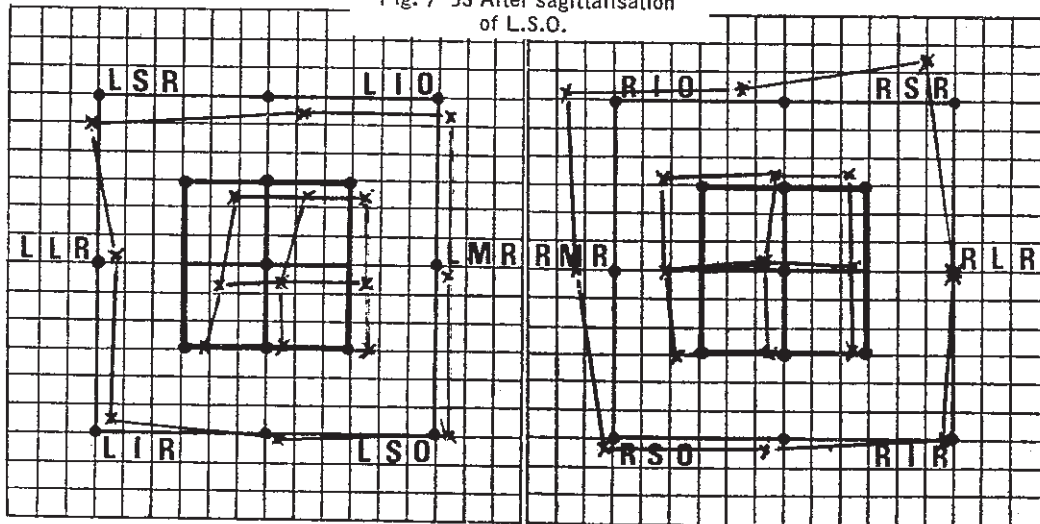
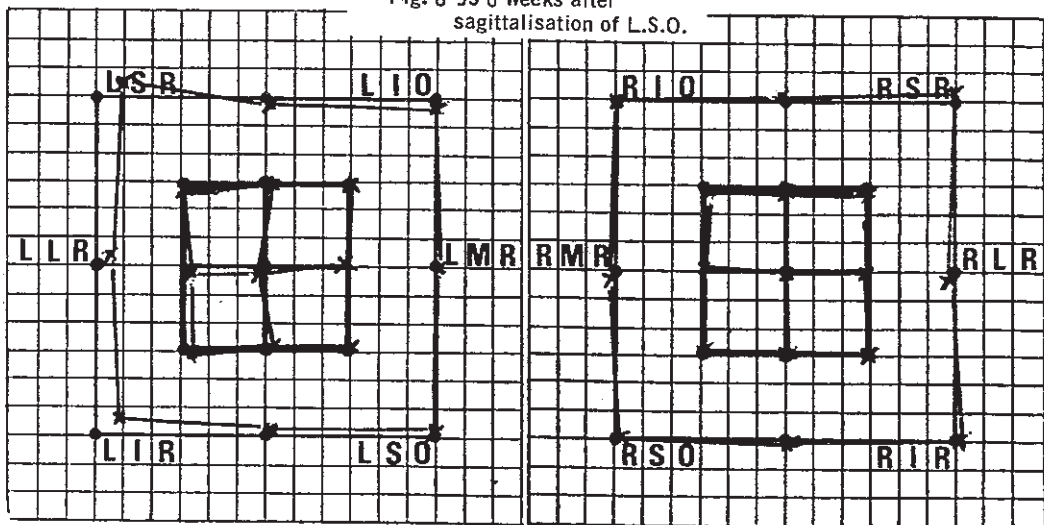


Fig. 8 JS 6 weeks after
sagittalisation of L.S.O.



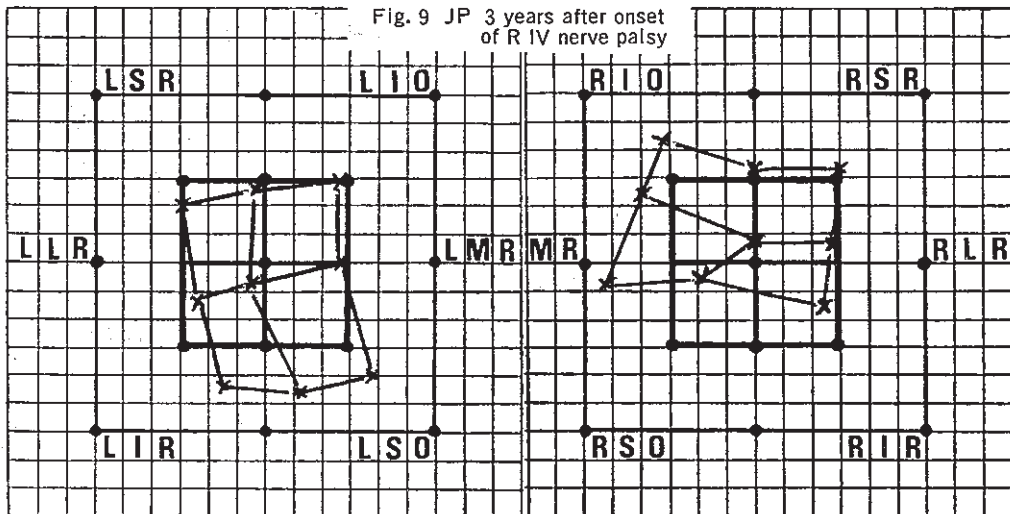
CASE II

JP, also was involved in a motor vehicle accident and was seen 3 years later. He had suffered severe brain stem injury. His Hess Chart (Fig. 9) showed a classical pattern of right IV nerve palsy.

Synoptophore readings were

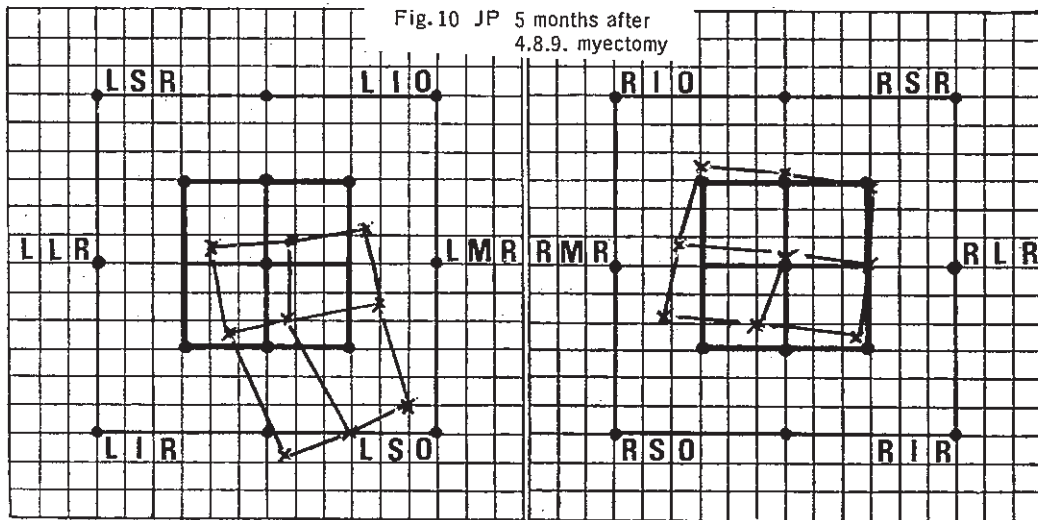
in primary position, fixing right eye $+2^{\circ}$ R/L 18A, excylo 10°
 fixing left eye $+1^{\circ}$, R/L 7A, excylo 15°
 in 15° depression, fixing right eye $+3^{\circ}$, R/L 20A, excylo 15°

The first operation, in accordance with accepted theory, was a myectomy of the overacting direct antagonist, in this case the right inferior oblique. The immediate post-operative result was most favourable, but as the weeks progressed the deviation grew more inconstant, and JP was more distressed by diplopia.



Five months later, the Hess chart (Fig. 10) confirmed this inconstancy. The synoptophore angles had increased, and in primary position were:

fixing right eye $+3^{\circ}$, R/L 11A, excylo 11°
 fixing left eye $+3^{\circ}$, R/L 6A, excylo 4°



Sagittalisarion of the right superior oblique combined with a left inferior rectus recession of 4mm was carried out.

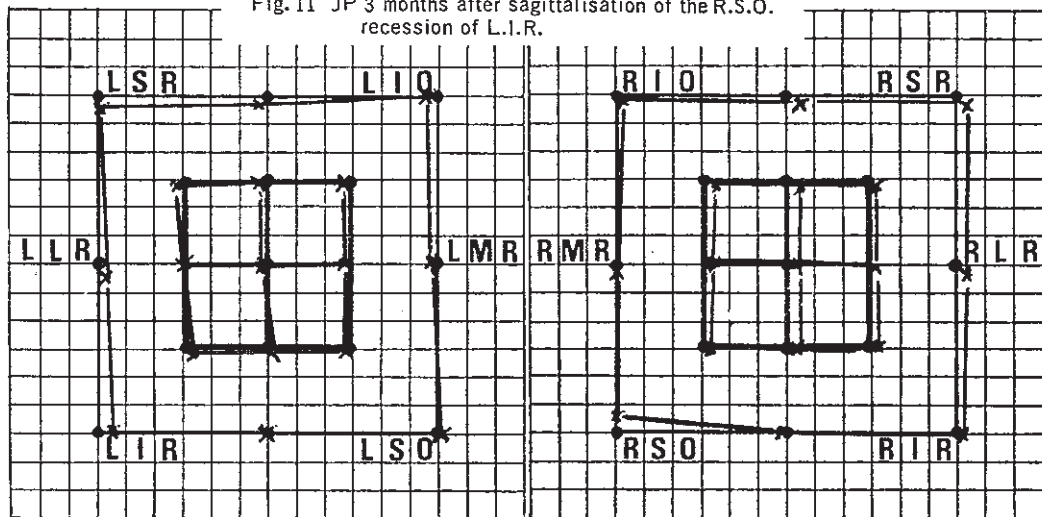
The immediate post-operative chart showed a constant small right hypotropia of 10^{Δ} . Constant diplopia was present.

As the oedema and swelling decreased, so did the hypotropia.

2 weeks after operation, the hypotropia measured 5A, and single vision was obtained in the primary position. JP was instructed to try to control the diplopia and continued to practice exercises at home.

3 months after surgery the Hess Chart (Fig.11) was almost perfect, there was orthophoria for distance and a small exophoria for near, with binocular single vision in all directions of gaze.

Fig.11 JP 3 months after sagittalisation of the R.S.O.
recession of L.I.R.



The simple procedure of advancing the paresed muscle appears to be the treatment of choice in partial IV nerve palsy. It may well be the treatment of choice for all oblique muscle weakness.

Acknowledgment

I would like to take the opportunity of thanking Professor Hollows, Dr. Paul Beaumont and the orthoptists at the Prince of Wales Hospital for their help and encouragement in preparing this paper.

ALTERNATING SURSUMDUCTION: THREE CASE HISTORIES

Diana Craig

Presented in Adelaide, April 1974.

Alternating sursumduction presents the most fascinating and challenging problems that orthoptists encounter. When it is the only disorder, it is no obstacle to binocular vision; it is evident on dissociation only, disappearing when both eyes are uncovered. But it commonly occurs together with other motor anomalies, the most usual being latent nystagmus and the A phenomenon (Lang 1968). Signs of alternating sursumduction (=ASD) and the other anomalies present may be marked or very slight, often affecting one eye more than the other. The picture may be further complicated by refractive error and temperament.

Our problem is to distinguish the signs of ASD and nystagmus, which we cannot treat, from secondary effects which we may be able to reduce by orthoptics and accompanying disorders which may be corrected by glasses or surgery. The following cases illustrate some of the phenomena encountered, and methods of examination which are sometimes helpful.

CASE 1

Ms.SD. aged 3½, wore +1.50 sph. each eye, (Retinoscopy +3.25 +4.25 each eye). Intermittent left or alternating squint had been noticed from birth. Glasses and atropine (R eye) had helped but the squint was becoming more frequent.

22.8.73 Cover test: left convergent squint for near and distance

Chin down

V eso, marked overaction of both inferior obliques

Vision: 6/12 (single E) each eye

Angle with glasses : +20°, occasionally less. Fusion +10° to +40°.
without glasses: +25°, variable

At end of visit eyes momentarily appeared straight.

Part time occlusion advised

19.9.73 Small LCS/ACS with alternating sursumduction on accommodation

Vision 6/12 each eye. Turns head to reduce nystagmus when either eye occluded

28.11.73 Intermittent convergent squint with ASD and nystagmus

Angle with glasses +25°

without glasses +35°

Fusion: two pictures seen, but eyes converge steadily, following pictures from +25° to +50°

Father convinced that eyes are often straight, undertakes to give exercises

19.2.74 Intermittent ACS with ASD. SD can usually straighten eyes with encouragement.

Vision : right eye 6/6, left eye 6/6 partly, (single E)

Angle with glasses: -5°, found subjectively, correct objectively;
without glasses: +5° to +20°

Vergence with SMP slides: -5° to +50°;

normal correspondence (lion in cage) -5° to +20° approx.,

lack of NRC (lion out, on contralateral side of cage) +20° to +50°

Fusion: steady with both controls -5° to +20° approx.

continued steady vergence, pictures separate, to +50°

Latent nystagmus was not noted on the first visit, probably because of the excessive convergence, nor on the last visit when the child had grown accustomed to us. Latent nystagmus in congenital squint is usually more noticeable in distant fixation, and when the subject is excited or apprehensive. It tends to be less in convergence, and children so afflicted are particularly likely to converge violently (possibly to reduce the nystagmus) under stress, in the presence of strangers. A single visit might, and sometimes does lead the examiner to a mistaken diagnosis of constant squint requiring surgery. In this case, fortunately, eyes were occasionally straight on the first visit.

The vertical deviation was attributed on the first visit to overacting inferior oblique muscles, and to alternating sursumduction subsequently.

ASD in association with marked convergent squint in an unco-operative child often leads to mistaken diagnosis of superior oblique overaction, because of the upturn of the convergent eye. This has applied in some cases in which underaction of inferior obliques or inferior rectus muscle, with A phenomenon was later demonstrable.

The measurements given show no vertical deviations, although if this paper had at that time been planned, such measurements could have been provided. In any case of alternating sursumduction, different measurements are obtainable according to the procedure adopted. The typical updrift occurs

- 1) when the eye is not fixating
- 2) when its illumination is reduced

Covering one eye tends to "send it up" on both these counts. Other factors including accommodation, attention, and degree of amblyopia, appear to affect it also, but unpredictably.

In measurement of the angle of deviation, a choice must usually be made. Does one need to learn the best, or the worst? To estimate the potential for binocular single vision, or to record the presence and extent of the sursumduction? A four-year-old's attention soon flags.

Once SD was keeping her eyes relatively straight, ASD was readily seen by the upward deviations under cover. Our aim in measurement was to discover the resting angle when the eyes were unembarrassed. On her fourth visit when asked to put the lion in the cage, she chose angles at which corneal reflections appeared symmetrical. If, with targets still in this position, one switched off either light, the other eye made no movement other than a slight nystagmoid one, so one could accept her choice objectively as correct. As both lights are on at the moment of choice, our notation for such a measurement is "angle, lights on". Had we used the common "light off" procedure of objective measurement, beginning by switching off the light of the non-fixing eye, alternate hyperphoric measurements would have been obtained.

The readiness to fixate binocularly was extraordinary. Even with glasses at the synoptophore, SD would occasionally converge one eye noticeably. If one moved the target to confront the converging eye, and then returned it to the zero position, the deviating eye readily followed while the other continued to fixate its own target. Both converging and diverging movements could be obtained in this way, with slow or quite quick movements of the target. One had the impression that each eye was maintaining fixation independently.

Subjectively, SD reported that the lion remained in the cage, or in the case of fusion slides, that there was one picture only with correct stereopsis, for angles up to about $+20^\circ$. During convergence to greater angles the pictures appeared to separate, and remained apart heteronymously. I have noted the same surprising phenomenon in at least two other cases.

The response to training, or perhaps one should say to gentle encouragement at home, was impressive. Single binocular vision to cover test, improved visual acuity, normal retinal correspondence, and a small divergent angle on the synoptophore were gained in the interval between the last two visits. That is, latent normal function had become manifest as the child gained confidence and learnt what was required in the test situation.

CASE II

MC, aged 6, wore no glasses. Intermittent alternating convergent squint and ASD were noticed soon after birth, Atropine occlusion had been given at 10 months for a constant left convergent squint. There had been operations at $2\frac{1}{2}$ years (RMR recession, RLR resection) and at 4 years (LMR recession, RIO myectomy)

Further surgery for a conspicuous left hypertropia was under consideration when she was referred to us for investigation.

We found a left esotropia of 16^Δ for distance and 2^Δ for near, with 15^Δ hypertropia. Left fixation was well held, with similar degrees of R esotropia, and 2^Δ R hypotropia. These measurements refer to the deviations normally manifest; the appropriate prism was placed before the uncovered deviating eye, then checked by covering the fixing eye. Under cover, the left eye drifted further up, and the right eye became hyperphoric.

Vision right eye 6/9, left eye 6/12

Synoptophore angles: right fixing $+15^\circ$, L/R 10^Δ } objective measurements under
left fixing $+17^\circ$, L/R 2^Δ } "lights on" conditions

Subjectively the state of correspondence was uncertain, because of variability in horizontal and vertical angles. No fusion was demonstrable.

First inspection of ocular movements gave an impression of marked left inferior oblique overaction (see Figures 1a and 1c) with upshoot of the left eye in dextroversion and eyes in approximate alignment in laevoversion. But if one watched the left eye carefully while a target was moved slowly from right to left, a wave-like or rolling motion was seen, the left hypertropia increasing steadily (Fig. 1b) until well beyond the midline, then subsiding rather suddenly. A cover test with eyes in laevoversion showed that in fact the left eye had now taken up fixation, with the right eye deviation slightly inwards and down. If one held a screen before the left eye to ensure right fixation in laevoversion, one could see, looking behind the screen, that the left hypertropia reached a maximum in full abduction.

Figure 1. Diagram of appearance as MC follows target right to left.



(a) Fixing right eye in dextroversion

(b) Fixing right eye in primary position.

(c) Fixing left eye in dextroversion

Showing changing deviations when following a target from right to left.

The investigation of muscle palsy

To investigate the signs of muscle palsy, if any, in a case of ASD, the effects on vertical deviations of fixation changes must be temporarily eliminated. This may be done by screening one eye from the target, and watching the screened eye while the fixing eye watches a moving target. It may also, in many cases, be done using the Hess screen.

One's first attempts to obtain a Hess chart from an ASD patient are apt to be discouraging. The patient complains that he cannot see both together; they keep moving away. The secret is to insist that he watches the fixed light or target while moving the other towards it. An alert child of six or even five years can do it, providing one reiterates the fixation instructions at every step. He soon appreciates the way steady monocular fixation prevents disconcerting changes in the apparent relative position of targets.

Figure 2

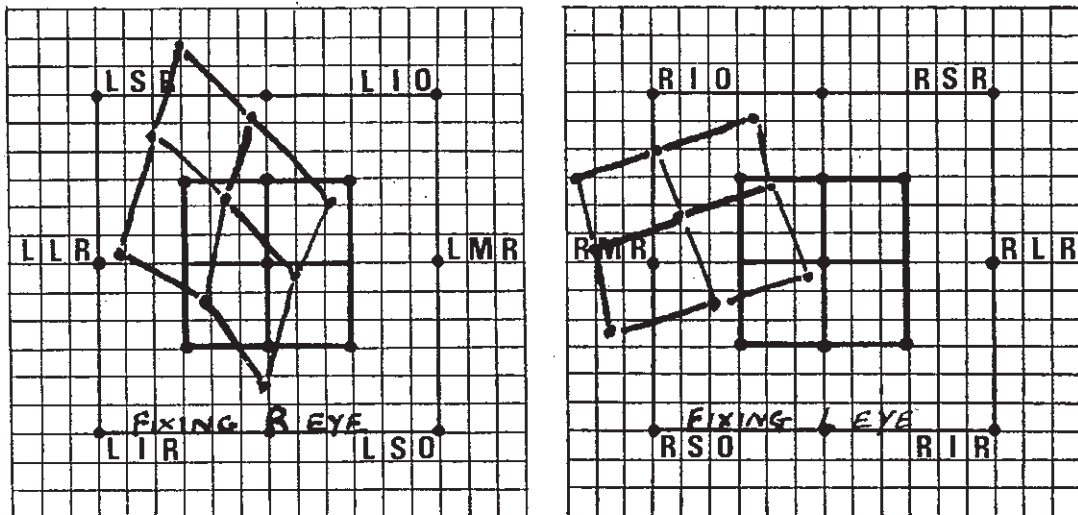
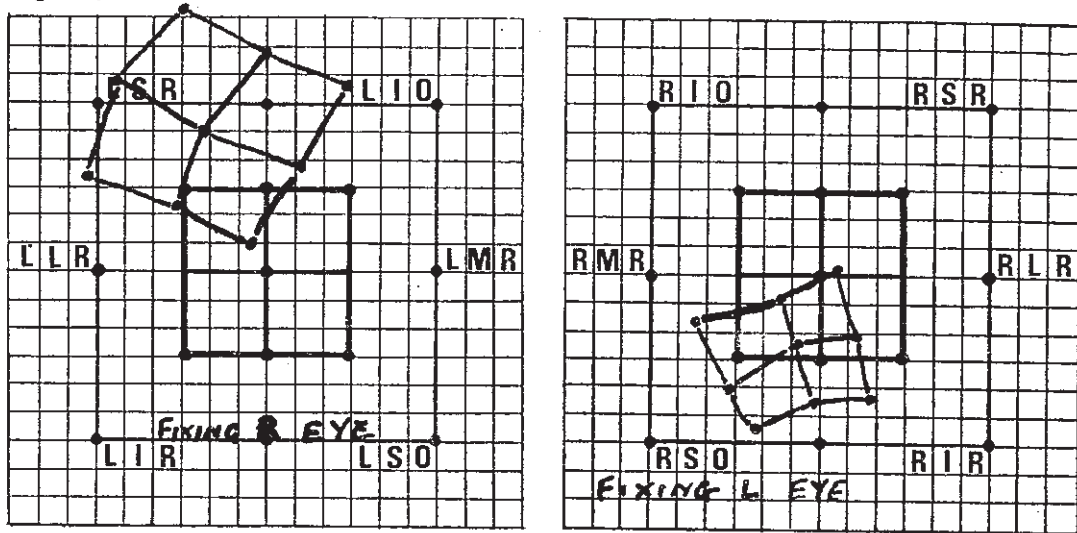


Figure 2 shows the first record obtained in this way from MC. Because of the up-drift of the non-fixing eye, the right and left eye charts are not complementary in the normal way. The record suggests that each eye is hyperphoric, so that one cannot determine whether elevators or depressors are at fault. But it does reveal a marked A pattern, with hyperophorias which increase in abduction, indicating relative underactions of inferior obliques or inferior recti. The chart also suggests an esophoria with left eye fixing, and exophoria with the right eye fixing. I cannot explain this.

The second chart (Fig.3) was obtained some weeks later, when the right eye had been atropinised to reduce left suppression. It is included as another of the unexpected findings in ASD cases. Possibly the dilated pupil, admitting more light to the right retina, caused the greater left hyper/right hypophorias. But why has the right chart shrunk? There must be information here which as yet we cannot unravel.

Figure 3



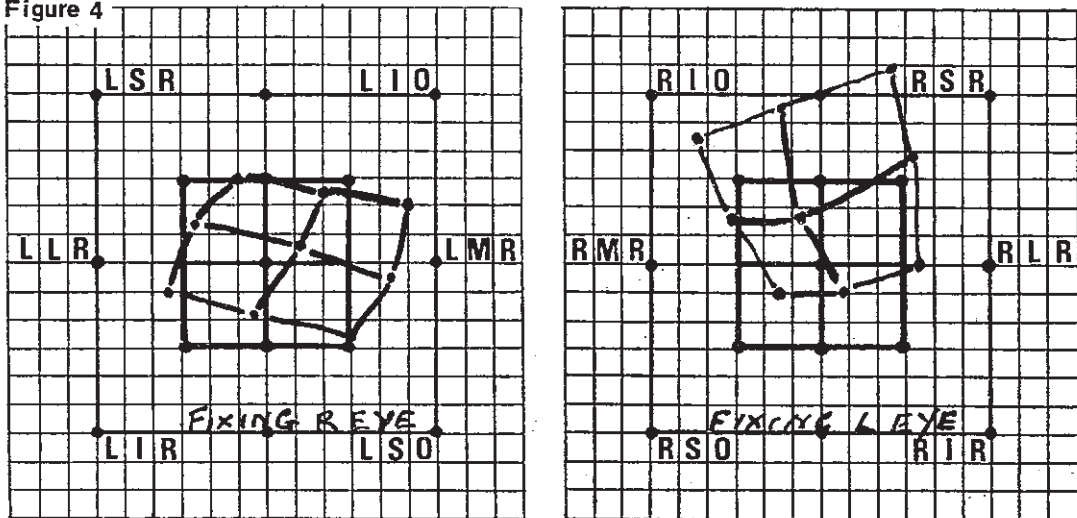
Retention of binocular single vision

In spite of her muscle anomalies, MC used binocular single vision for reading. When we gave her a book, she held it a little to the right, tilted her head a little to the left, kept her chin up, and was able to read N8, if not smaller letters, with both eyes fixing.

CASE III

Ms. JF. had shown a right or alternating convergent squint from birth
 RMR recession, RLR resection aged 2
 LMR recession aged 8
 Orthoptic exercises from 7½ to 8½ years.
 She came to Melbourne aged 9 years, when we found on cover test
 at 6m.: right hypertropia/small LDS : each eye up under cover
 at 1/3m: right convergent squint,

Figure 4



There was a marked A pattern of movement, with esotropia in elevation, exotropia in depression, hypertropia of each eye in abduction. Synoptophore angles, obtained subjectively and checked objectively, were

with right eye in primary position: $+5^{\circ}$

with left eye in primary position: $+5^{\circ}$ R/L 3

with much alternating suppression. Fusion was doubtful, not surprisingly in view of the great variation in vertical and horizontal angles for different directions of gaze.

Nevertheless, when JF was asked to demonstrate the exercises which Miss Kirby had taught her, she turned her head a little to the right, tilted it left a fraction, and followed a pencil with steady binocular fixation from 33 cm to about 10 cm, maintaining physiological diplopia all the while.

The Hess Chart (Fig.4) agrees with the observed pattern of ocular movements and is similar to Case 2, but without the gross asymmetry.

Comment

These cases serve to show that alternating sursumduction is no bar to binocular single vision. The potential for good or useful binocular single vision is far better retained in ASD cases than in any others of birth onset and complex muscle anomalies.

Unless one is forewarned, it would be easy to misdiagnose such cases. As regards objective signs, a marked intermittent convergent squint, apparently purposive to reduce nystagmus or vertical deviation, often suggests a constant convergent squint on early visits; and the alternating vertical movement might wrongly be interpreted as bilateral or unilateral inferior oblique overaction (This does not imply that constant convergent squint and/or inferior oblique overaction are never associated with ASD) Subjectively, difficulty in superimposing targets, due to alternating vertical deviation, may be misinterpreted as loss of correspondence.

These problems may be solved by caution in long-term management, and by particular techniques of investigation using careful direction of the patient in fixation with one eye or the other or both.

In addition, bizarre behaviour occurs from time to time, such as vergence movements in pursuit of SMP slides, or changes in vertical deviations on Hess charts after monocular instillation of atropine. These (who knows) may be clues by which, when carefully collected and collated, the aetiology of ASD may ultimately be unravelled.

Acknowledgment

I am grateful to Drs. G. Morlet, K. Lidgett and F. Billson for allowing me to use the case histories recorded here.

REFERENCE:

Lang, J., *First International Congress of Ophthalmology*, 231

CASE HISTORY : HEADACHES

J. Fitzsimmons

Presented in Sydney, April 1975.

Michael A, aged 12 years, first attended the Outpatients Department of the Sydney Eye Hospital complaining of blurred vision in his left eye, and of headaches after school. On referral to the Orthoptic Clinic he was found to have a convergence insufficiency; one month later, on his third visit, he was discharged as symptom free.

Michael returned twelve months later, again complaining of headaches. His convergence was fair, his accommodation somewhat below normal. A refraction showed that no glasses were necessary. Treatment was again attempted. Michael's convergence and accommodation improved steadily. His responses to all orthoptic tests were soon satisfactory, but his headaches persisted without improvement, and were now always in the morning.

Michael was referred back to the Outpatients Department, where the ophthalmologist found

visual acuity 6/5, 6/9 (no improvement with pinhole)

pallor of the left disc

headaches always present in the morning, growing worse during the day,

youthful appearance for a 14-year-old,

left temporal hemianopia.

A pituitary tumor was suspected. This was confirmed by skull X-rays, and Michael was referred to the neurologist.

This case was a reminder that if a patient complains of headaches, one should note when and where they occur, and bear in mind that although there may be evidence of convergence insufficiency, it is not necessarily the only cause. Particularly, vision should be tested to 6/5; any deterioration, no matter how slight, may indicate a neurological or ophthalmological problem.

CASE HISTORY : INDUCED HYPO ACCOMMODATIVE SQUINT

Maree Sullivan

Presented in Sydney, April 1975.

Miss H.P. aged 25 years, was referred for an orthoptic examination, complaining of blurred vision for near work during the past 6 weeks. She had found that her mother's glasses (+3.00 D. sphere R and L) enabled her to see print clearly and to read quite comfortably. Her history revealed that she was schizophrenic, and had been prescribed two drugs, Cogentin (a depressant with a cycloplegic agent) and Melleril (a tranquiliser).

Clinical examination revealed a small left convergent squint for near, and approximate orthophoria for distance. Both binocular and monocular accommodation were grossly defective and at her convergence near point (6 cms.) there was no constriction of the pupils. The Maddox rod reading was esophoria 4^A, while the Maddox wing showed esophoria 15^A. Miss H.P. could bar read N48 with an effort.

A week later, having suspended the Cogentin on her doctor's advice, this patient presented with a small esophoria for near and distance. Her accommodation was no longer defective, and there was normal constriction of the pupils on convergence. She could bar read N5 easily without the use of her mother's glasses, and was symptom free.

It was interesting to find that a squint could be induced in this way. If there had not been an underlying esophoria, would the result have been the same?

I would like to thank Dr. David Benjamin for allowing me to present this case history.

HYPOPLASIA OF LATERAL RECTUS AND ABNORMAL INFERIOR OBLIQUE INSERTION

Jill C. Stewart

Presented in Sydney, April 1975.

G.M., a girl aged two years, was first seen by the ophthalmologist six months ago. Her mother said she had had a convergent squint since birth. There was no family history of squint, and the birth was normal. She had a left convergent squint of 45° with left hypertropia of 10° , judged by corneal reflections. Ocular movements showed a right inferior oblique overaction.

She was given a +3.50 sphere each eye for constant wear, with total full-time occlusion of the right eye. This was changed to alternate eye occlusion once she could take up left fixation.

When I first saw G.M., she had a left or alternating convergent squint with hypertropia of the convergent eye. Both right and left abduction were limited to a few degrees at most past the midline, and both inferior obliques were overacting, the right more noticeably than the left.

In January this year both medial recti were recessed by 5mm and the right lateral rectus was resected 6mm. There appeared to be a complete absence of the left lateral rectus, while the left inferior oblique had an anterior insertion; or possibly the left lateral rectus insertion was misplaced and the left inferior oblique inserted elsewhere. The ophthalmologist found no muscle or fibrous band in place of the left lateral rectus, and what he believes to be the left inferior oblique is inserted 7mm from the limbus.

After the operation G.M. still had a left convergent squint of about 15° with 5° left hypertropia. Right and left abduction were much better, although left abduction was still limited. Possibly the eye is more free to move and the misplaced muscle, either left lateral rectus or left inferior oblique, is able to abduct the eye. Cosmetically the result is still not good, so a Hummelshiem's operation may be done a little later.

Although squints of peripheral origin are not as common as neurogenic squints, they are by no means rare; but from the evidence of reported cases, the absence of a muscle is rare. Absence of the lateral rectus was reported by Heuck in 1879, Bahr 1896, Rabstan and Goar 1921, Bielchowsky 1932, and Stockwell 1952. In 1967, two cases were reported in America by Pritikin; after surgery both were cosmetically satisfactory, and the affected eye could be abducted past the midline.

Abnormal insertions are the most common congenital defects in ocular motility. The insertion of the inferior oblique varies chiefly in the degree of obliquity and convexity of the curve, and in gross irregularities such as angular serrations or gaps.

Acknowledgment

I would like to thank Mr. P. Munchenberg for allowing me to present this case history, and Dr. M.C. Moore for his assistance with the literature on the subject.

REFERENCES:

- Duke-Elder, S. *System of Ophthalmology* (1973), Volume VI, Henry Kimpton, London.
Pritikin, R.I., *American Journal of Ophthalmology* (1967) 63.
-

CASE HISTORY : CONVERGENT SQUINT OF SUDDEN ONSET

Frances Merrick

Presented in Sydney, April 1975

S.B. was born with a double or bifid nose (3 nostrils). At the age of 4 years she underwent extensive cranio-facial surgery to improve her appearance. This involved multiple bandaging of skull and face, and when the bandages were removed, the right eye had turned in. Her mother had never noticed a squint previously.

About 5 months later, at the age of $4\frac{1}{2}$, S.B. was presented in the orthoptic clinic of Sydney Eye Hospital with a 30Δ right convergent squint and slight right hypertropia; and visual acuity right eye 6/18, left eye 6/9. The synoptophore angle was 13° , R/L 2Δ , and fusion with simple SV was held to $+32^\circ$ R/L 2Δ and to $+10^\circ$ R/L 2Δ .

S. gave an appearance of bird-like fragility; she wanted desperately to have straight eyes. She accepted constant and total left patch occlusion stoically but when it was removed she had the sensation that the delicate bridge of her nose was "coming right off". Large cotton wool pads were accepted instead. The vision became equal but fine S.V. was still confused: we changed to part-time pads. A date was set for the operation: on went the pads full time again and anti-suppression treatment at the synoptophore was begun. When the small bird retained 2 black legs without stimulation, S. and I sat back and cheered. She wore her cotton wool pad right up until surgery.

Three weeks later there was only a small exophoria with good recovery,

6/5, N5 each eye,

zero angle and 20° of fusion,

SV accurate on Titmus fly, animals, & 6/9 circles.

At this stage a newspaper article recorded the withdrawal of the drug Duogynon for pregnancy testing, as research had shown it to be the cause of some cases of cleft palate. When S's mother read this, her "heart stopped" (she said). Doctors and plastic surgeon had questioned her exhaustively about her medication during pregnancy; she knew without a shadow of a doubt that she had taken no drugs once she knew a child was on the way. But Duogynon had been used to determine her pregnancy, and the palate is part of the nose.

Acknowledgement

I would like to thank Dr. Geoffrey Hipwell for referring this child to me, and for allowing me to present the case history.

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