AUSTRALIAN

ORTHOPTIC JOURNAL

1972

Volume 12

TRANSACTIONS OF

29TH ANNUAL SCIENTIFIC MEETING

including a Cumulative Index of Volumes 1 to 12 (1959 – 1972)

Wholly set up and printed by ROTOGRAPHIC PRODUCTIONS, 41 Turner Street, Redfern ● 698 3624

EDITORIAL

Volume 12 of the Australian Orthoptic Journal has been revised since its Editor, Neryla Jolly went overseas and now is largely a Journal of papers read over the past few years on the subject of Dyslexia and associated reading problems.

These papers were presented in Sydney, April, 1971, and Adelaide, April, 1972.

Thanks are due to Dr. R. Hertzberg who presided as patron at the Sydney conference, and to Dr. C. Moore who was our patron in Adelaide.

We are indebted for the work Mrs. Jolly did in editing, to Mrs. Dennison, Mrs. Stanley and Miss McCormack for compiling and proof reading; to Mrs. Hitch and Miss J. Russell for their task of compiling the cumulative index, and to Dr. D.B. Dunlop for the introduction.

Helen Hawkeswood

INDEX

CUMULATIVE INDEX

Volumes 1...12

1959 to 1972

Emmie Russell Prize *

INDEX OF AUTHORS WITH FULL TITLES OF ORIGINAL CONTRIBUTIONS

INDEX OF AUTHORS

INDEX OF AUTHORS

SUBJECT INDEX

SUBJECT INDEX

INTRODUCTION

Orthoptists have not previously been expected to play a significant role in the diagnosis or treatment of dyslexia. Many may have rather hazy notions of what the word is supposed to convey.

The Research Group on Developmental Dyslexia of the World Federation of Neurology, which comprises an international body of experts - neurological, paediatric, psychological, pedagogic - met in 1968 and drew up two definitions:-

"Specific Developmental Dyslexia"

"A disorder manifested by difficulty in learning to read despite conventional instruction, adequate intelligence, and socio-cultural opportunity. It is dependent upon fundamental cognitive disabilities which are frequently of constitutional origin."

"Dyslexia"

"A disorder in children who, despite conventional classroom experience, fail to attain the language skills of reading, writing and spelling commensurate with their intellectual abilities."

The latter definition covers a wider, more general range of defects and the term "learning disability" is often preferred in order to avoid confusion with the more precisely defined "specific developmental" condition which is characterised by:- the peculiar and specific nature of the errors in spelling and reading; its tendency to run in families; to affect males much more than females; to persist to a greater or less degree into adult life; the presence of normal if not superior intelligence; and the complete absence of any evidence of brain or neurological disease or damage.

It will be noted that the diagnosis of dyslexia involves an assessment of reading ability in addition to an assessment of intelligence. It follows that the diagnosis can only be valid if it is made by experts in each field.

Thus an inter-disciplinary group is necessary comprising at least a trained psychologist and a special teacher. Now, as you will see from the following papers, an expert orthoptist will be a very valuable member of such a group.

AN INTERDISCIPLINARY APPROACH TO DYSLEXIA

PAPER 1

Mr. B. Fenelon, M.A.
Senior Lecturer in Psychology, University of Newcastle.
(Presented in Sydney, October, 1972)

I want to thank the Orthoptic Association for giving me this opportunity to talk to such a wide audience.

Let me say something about how the problems of dyslexia appear to me, which may not be the way they appear to you. Most dyslexic people I see are in a child health care setting, and I find that most of them are part diagnosed. It is always part, because, no matter how far the diagnostic procedure has gone, there is usually still more to do. I would prefer to say "described and understood" to "diagnosed", because the diagnosis gives an impression of a procedure which is formal and relatively specific, and this does not necessarily apply in these cases.

Almost all the parents are concerned parents. Some may be anxious, some tense, some very aggressive, and many of them depressed to some degree. These are levels of concern that the parents exhibit because their children have been revealed to them as being intractable kinds of learning problems. In some cases you can register these levels of concern and take them into account in your prognosis of what you can hope to achieve. I think it is reasonable and natural for all parents to be exhibiting some level of concern, and frustration and to hope that something better can be achieved in the individual case.

There is the tremendous impact of the initial diagnosis which may have been offered at school, or in an educational guidance clinic or may even have been suggested by a friend. You often have to handle the falsely informed parent, to whose child a label has been applied, whose child has "dyslexia", a term which is bandied about a great deal in the community. You may be confronted with a child who shows signs of all round retardation, in which case you have the problem of assisting the parent to face up to the sum of the implications of that more generalised condition. But I do not think that relieves you in any way of the responsibility of dealing with problems that are very similar, in many ways, to the ones that are associated with children who actually have dyslexia type learning problems. One of the jobs, of course, is to handle the frustrations of the parent concerning the system. As a general statement, I should say that there is a problem of helping the child to achieve some self respect in the knowledge, as he often has, that he is different from other children in the school and in the community. It is a matter of helping him over a prolonged period of time through rather difficult periods, to an understanding that he is a person who is worthwhile in himself, that there are many things that he is capable of doing and that he is capable of making both others and himself happy, given the right types of opportunity. With the parent, the task is, in my opinion, a somewhat easier one, though it may appear, in the early stages, to be the more difficult.

The contract that is entered into involves these concerns and not infrequently some particular group, or some particular specialist, is the pivot of the whole treatment programme. This pivot group can be from any specialist area; the psychologist, for example, may co-ordinate all of the various diagnostic and remedial measures which are undertaken, but any specialist in any of the disciplines belonging to a team for the treatment of these disorders may undertake the co-ordination.

I think that we have an obligation to carry out some **optimally exhaustive diagnostic** procedure in any case that is presented.

I will outline the procedure for an assessment of a child with a handicap. A full coverage of sensory and motor functions, testing of visual and perceptual functions and testing of laterality and of any right/left confusions which may exist are required. Close observation is terribly important with these children. I want to say that whatever tests are used they have to be interpreted and sometimes it is not so much the formal aspects of tests and the objective scores which are derived; as the observations on behaviour that are made whilst the tests are going on that are important. The psychologist, for one, has a very good opportunity to record a lot of verbal and nonverbal behaviour in the course of any testing session, no matter what tests he happens to be using. Of course there have to be reading, spelling and writing tests, and these call for the sharpest observation of all. It is in this area that one can make observations and set them down for the future remedial teachers who are going to be involved in the case, and these are the things that teachers and remedial teachers want to know about because ultimately they are going to be charged with the responsibility for applying some system and getting the best learning possible out of the child. They want to know specifically what it is that the child is able and is not able to do and an abstract report from any discipline will not necessarily give them very much guidance in that direction.

What is the team to deal with this problem? I don't know, but frequent members listed are ophthalmologists, audiologists, paediatricians, neurologists and psychologists, but I would like to add orthoptists, optometrists, special teachers, speech therapists, and perhaps there are many others who could figure because I am not particularly discipline-oriented in this; I believe that there are people who develop specialist inclinations and directions in their work within any discipline and, providing they are prepared to be flexible and open-minded, there is a lot that can be achieved by people in probably the most unlikely associated disciplines. Those mentioned are some that I would include in a team approach, but I am using the word "team" somewhat loosely; I would think of this in most communities as being a loose, inter-referral structure and if the members of that team are aware of their own limitations then they will take advantage of the structure that exists.

Now shortcut diagnosis is something that worries me. I often hear people saying that it is possible to determine the diagnosis of dyslexia with a few quick flicks of the wrist and a couple of diagrams, and a short one-minute performance from the child. Psychology has been very good on that kind of trick for a long time. We have been producing major tests and then shortening them, and I would warn that the correlations of the shortened forms of the tests with the major form of the test are often only moderate. These are shorter forms to which a great deal of effort has been applied and a good deal of psychometric acumen was needed to produce them. Now when one produces an ad hoc procedure and bungs it on as a short way to diagnosis, I believe that the risks are very great indeed that false diagnoses will be made. I cannot see the necessity of engaging in this kind of activity when, with a bit of open-mindedness and flexibility, it is possible for people to make referrals and, since they are interested in saving themselves time, to actually save themselves time and get the specialist that they know is capable of doing a job in a certain area to carry it out. And I say that as much in criticism of psychologists as anybody else. The all round diagnostician is a possibility, but a rarity I believe. Nevertheless he may exist and if he does, then he is a force unto himself, but he will spend most of his time in diagnosis - I am sure of that; and the all round diagnostician with such composite abilities could probably not afford to use that time up when other people could be relied upon to do the job.

Each specialist and each speciality has some important individual contribution to make. For example, the cerebral palsy specialists probably know a great deal about the critical developmental behaviours which may lead to learning problems in later life. They also know something about the application of remedial methods in relation to motor performance. I am thinking of techniques such as the Bobath technique where sequential reflex organisations may be manipulated. I would say that we know nothing of that in the specific learning disability field, and this is an area of specialising to which we could turn with great profit. It is also an area from which people could turn to us and give us the benefit of their knowledge.

Most of us, I think, are committed to pragmatism when we handle these children: we do what works and sometimes we have little idea of what it is we are doing, except that it works. We have not much theory to go on and some of us don't place much trust in the theory that does exist. So many of us become pragmatists up to a point. But, I think, we need to know ultimately just what is going on underneath and we should not discourage or ridicule basic research or intermediate level research in relation to these problems.

This open versus closed mind that I am talking about affects whole orientations. I have spoken about the rather restrictive, narrow and destructive attitudes on EEG as something that affects me personally. You hear, "It is an educational problem", "It is a medical problem." It is both of those things. It is many other kinds of problems too, but if any particular speciality can arrogate to itself the authority to advise on the whole diagnosis and treatment of this condition, then it is time that the guide lines were laid down in the literature, because we would all like to know more about it. We have to face our own limitations in respect to diagnosis and treatment all the time, In my own case, they are prodigious indeed.

The speaker then went on to outline research projects which had been conducted in Newcastle, by himself and colleagues:

(a) psychological and educational response of severe reading disability children to drug therapy over a period of four months;

(b) motor-skill profiles of children (M. Lawson);

(c) perceptual thresholds when auxiliary sensory stimulation is applied (Fenelon and Wortley, to appear in Perceptual and Motor Skills);

(d) change in spatial distribution of the EEG of learning problem children under treatment by Nitrazepam (Fenelon, Holland, and Johnson, (1972) Cortex, 8:4:444–464

(e) left-right visual half-field identifications of verbal and non-verbal stimuli, presented by computer (Fenelon, Hall and Kelly, to appear in Cortex);

(f) a study with Mrs. Dunlop and Dr. Dunlop, where orthoptic examination indicators were combined successfully to categorise subjects as learning disability cases or normals (Dunlop, Dunlop, and Fenelon, to appear in Cortex).

Editor's note:- As Mr. Fenelon left for an overseas lecture tour shortly after giving us this paper, he did not have any opportunity of revision. The minor changes made by the editor to the original draft, despite the care taken to preserve the author's meaning, may not have captured all of the author's views nor the subtleties of the author's desired emphasis.

AN INTERDISCIPLINARY APPROACH TO DYSLEXIA

PAPER 2: THE BINOCULAR BASIS OF DYSLEXIC CONFUSION

Dr. D.B. Dunlop

(Presented in Sydney, October, 1972)

Basic Concept

The basic concept underlying this study and the new method of binocular testing is that the two eyes do not make an equal contribution to the function of the central binocular region and that this difference in central visual function is necessary and should be related in a specific manner to the lateralisation of other special functions within the nervous system such as speech and the normally right handed motor system.

It has been known for a long time that children suffering from a certain type of specific reading and writing difficulty were more likely to be left handed or ambidextrous or to belong to families so affected (Berlin 1887). Such children appear to lack the ability to develop normal lateralisation of special functions to the appropriate cerebral hemisphere so that effective specialisation of development can proceed.(Rosennerger 1970)

Many investigators have sought to correlate this crossed laterality of motor and other functions with some similar crossed laterality of visual functions, but without success. (Walls, 1951, White, 1969)

We believe their failure was due to the fact that the great majority of their test procedures involved essentially monocular activities like aiming a gun, looking through keyholes or the super-imposition of monocular images without true fusion. Berner and Berner (1953) first suggested the necessity of maintaining binocularity but even their stereoscopic tests rely on suppression which is the breakdown of binocular function to the monocular state.

The new test described in Mrs. Dunlop's paper, is specifically designed to maintain the patient in the fully binocular state, in his central field of vision, despite relative movements of lateral control features, as in Ogle's experiments with disparate images in stereopsis (1953)and Burian's similar studies (Adler, 1965).

From a practical point of view the new test has shown a statistically high degree of correlation with the patient's clinical state (Dunlop, Dunlop & Fenelon, 1973). This means that we may now, for the first time by ocular tests, differentiate one group of children with visual learning problems from normals.

The concept of a master eye and a minor eye has been declared to be "neurologically unsound" (Flax 1966), even by such eminent experienced neurologists as John Money (1968); we must therefore re-examine the neuro-physiology underlying the concept.

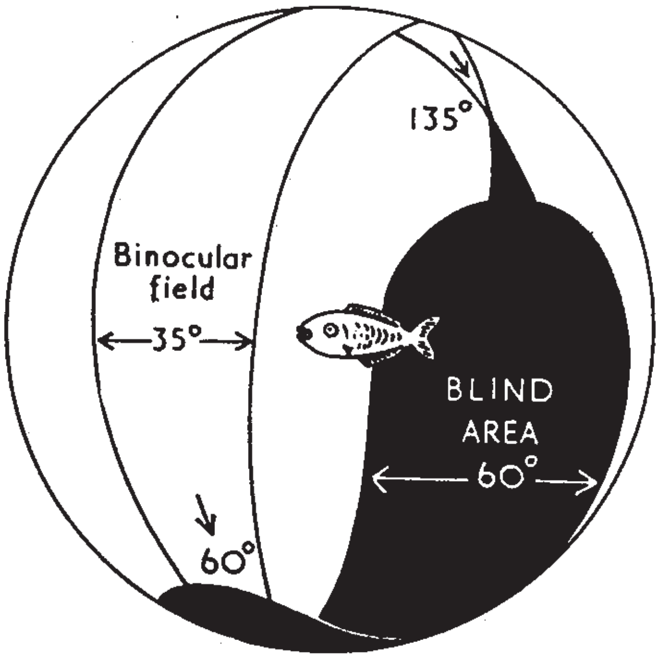
The critics state that since the visual fields are divided down the midline and there is an equal and symmetrical distribution of visual nerve fibres from each eye to each hemisphere, neither eye can dominate the visual process.

But let us look at the situation in greater detail and consider the actual visual functions involved in each part of the fields of vision. The work of Julesz, Hubel and Weisel, Blakemore, Bishop & Pettigrew should indicate that you must abandon many of your old concepts of binocular vision. We will first consider the lateral areas of the fields of vision, then the overlapping areas of binocular vision and finally the central mid-line areas of special binocular sensitivity and high acuity.

Dissection of the Visual Fields into Three Distinct Areas with Three Distinct Functions

The light stimuli from each lateral field activates the nasal half of the ipsilateral retina; all the nerve fibres from the nasal retina cross in the optic chiasm to the contralateral geniculate body and eventually the contralateral lateral visual cortex. No one will deny that each eye is totally dominant in its own far lateral field of monocular vision. The relevant contralateral cortical cells receive impulses from one eye only. The function of this lateral dominance is to enable the animal to react quickly and unequivocally to important stimuli in its peripheral field of vision on either side. The reaction in all species is to turn the visual apparatus so that the object of interest is now pictured on the central region of the visual field where high visual acuity and high stereo-acuity are available.

FIGURE 1(FROM DUKE ELDER)



THE BINOCULAR VISUAL FIELD OF A TORPEDO-SHAPED FISH WITH LATERALLY DIRECTED EYES

The field of vision is not a flat area but is curved in 3 dimensions with horoptors suitable for the activity of the animal. Thus the fish has a very extensive total field of vision but a narrow binocular field which extends from below and in front to above and behind (Fig. 1, Duke Elder 1958). The rabbit similarly has a binocular field in front, above and behind. This enables the pursued animal to use his binocular functions to assess the nearness of the safety of his burrow in front and simultaneously the closeness of his death-dealing enemy behind or above, e.g. the fox or the eagle. (Fig. 2, Duke Elder 1958).

FIGURE 2 FROM DUKE ELDER

FIGS. 805 AND 806.—BINOCULAR FIELDS.

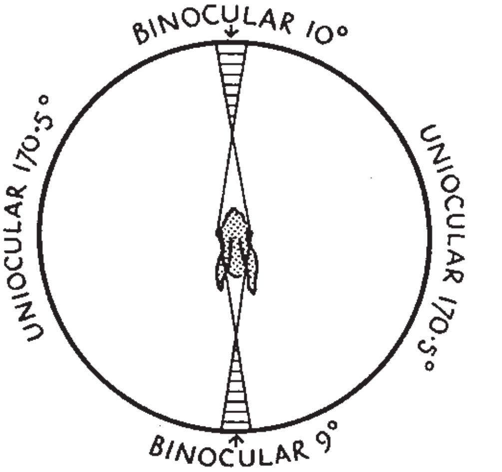


FIG. 805.—The panoramic field of a hunted animal (the rabbit) with a small binocular segment in front (10°) and behind (9°), and a large uniocular area (170·5° on each side).

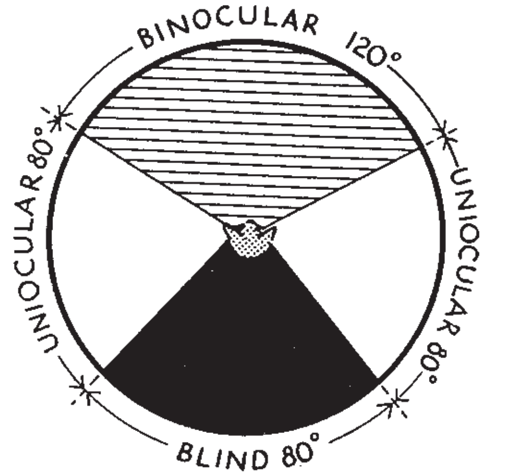


FIG. 806.—The binocular field of a predator (the cat) showing a large anterior binocular area (120°) a large posterior blind area (80°) with relatively small uniocular area (80°).

Survival Depends on Accurate Distance Assessment

The essential point is that his central field is used for the assessment of distance. Although his binocular field is narrow it is his quickest and most accurate means of judging his margin of safety (Rubin & Walls). It is his best way of measuring his chance of survival. It does not need to be wide in the rabbit. He just has to assess his enemy's distance behind and the distance of safety in front. It needs to be wider in the hunting animal, such as a cat, which must be able to assess the distance of the most opportune member of a group of prey which may be spread out camouflaged in front of him. He has no binocular field behind because he does not need it; but he has a fairly wide binocular field in front so that he can measure relative distances of several prey and their escape routes at the same time.

How does this distance measuring mechanism work? Hubel and Weisel (1962) have demonstrated that there are special binocular cells in each para-striate cortex which respond best when each eye is stimulated simultaneously and in the same manner in very specific small areas of each field of vision. These "receptive areas" are accurately located in a permanently fixed relation to each other and to the other parts of the field of vision. The receptive field of one eye rarely corresponds exactly to the receptive field of the opposite eye and this lack of correspondence is called "disparity."

The receptive areas can be mapped out very accurately by putting a needle in the appropriate visual cortex of a conscious, but immobilised cat so that the activity of a singular binocular cell is recorded while discrete areas of the field of each of the cat's eyes are suitably illuminated, separately and together, at different degrees of disparity (Pettigrew, 1972). Some of these cortical cells respond poorly or not at all when only one eye field is illuminated but all respond much more actively when the corresponding receptive field of each eye is stimulated simultaneously at the correct degree of disparity. The response is quickly inhibited if the disparity becomes too large or too small, indicating that these cells respond to stimuli from specific horoptors at a definite distance in front of the animal (Joshua and Bishop, 1970). In those binocular receptive areas recorded away from the midline it is found that the corresponding areas in each eye are not equal. There are differences in area, and in intensity of response contribution, indicating that the receptive area of one eye is used as a reference against which the stimulus on the receptive area of the other eye is compared. This comparison is made by single cortical binocular cells which receive one nerve fibre from each eye, one ipsilateral and one crossed (or contralateral). (Barlow, Blakemore and Pettigrew, 1967)

The majority of these reference or controlling areas are found in the contralateral eye. In considering the distribution of nerve fibres this would be appropriate because the crossed fibres are the oldest phylogenetically and the more recently developed and expanding function of binocular comparison is mediated by the phylogenetically younger ipsilateral fibres (Holmes., 1945).

Contralateral Dominance of the Lateral Alerting Areas

To summarise, we find that both the far peripheral field and the non-central areas of the binocular field of vision are dominated by the contra-lateral eye. To quote Bishop (1972) "There is a clear cut dominance of one eye over the other at the cellular level."

From a functional point of view the lateral field is seen as an alerting mechanism while the non central binocular areas are "relative distance" measuring areas, assessing distances of non central points of interest in relation to each other and to the central fixation area of maximum interest.

The central midline binocular region is quite different to the lateral areas in structure, neuronal connections and function.

The Essentially Different Midline Binocular Region

The distance of any one point of interest from the individual can be assessed by a single cortical binocular cell which is comparing a receptive field from one eye with a receptive field from the other eye, provided that the point of interest is not on the midline, or very close to the midline. Fig. 3.

The relative distances of two points of interest, neither of which is on the midline, can be assessed by the relation of the activity of two binocular cells within the same hemisphere, provided that the two points of interest are on the same side of the midline and not on or very near the midline. Fig. 3 (Mitchell & Blakemore, 1970)

The distance of any point of interest which is actually on the midline must be assessed by a totally different mechanism.

FIGURE 3 FROM DAVSON

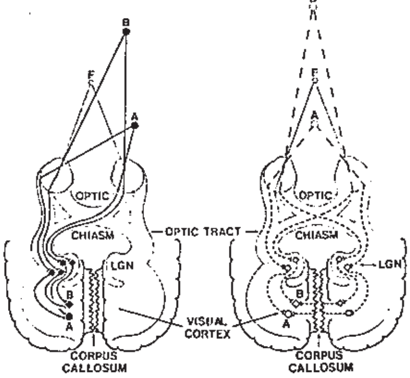


FIG. 333. The possible neural system for binocular depth perception in the split-brain human. On the left is the arrangement of neurones which enabled the subject to recognize the depth of the peripheral objects A and B, relative to the fixation point, F. For both objects the image in the right eye falls on nasal retina and that in the left upon temporal, and all the information projects to the left optic tract. The messages from the two eyes remain segregated at the lateral geniculate nucleus (LGN). Solid circles represent neurones within whose receptive field the image of A falls. Solid diamonds are cells to which object B projects. The two binocular cells, A and B, shown as large symbols in the cortex, encode the disparities of the objects. In this case the sectioned corpus callosum is of no hindrance. The binocular information is processed entirely in the left hemisphere and the judgements can therefore be vocalized.

In the right diagram objects A and B lie directly in the midline and, therefore, their images fall on temporal or nasal retina, respectively, in *both* eyes. The interrupted lines and open symbols in the visual pathway show the neurones normally responsible for the recognition of the disparity of these objects. The binocular cells A and B in the cortex receive one of their inputs from a fibre from the opposite visual cortex, crossing in the corpus callosum. Section of the commissure has severed this link and binocular integration is impossible. (Mitchell & Blakemore, *Vision Res.*)

If the point is just beyond fixation its image will fall on the nasal half of the retina in each eye and the resulting nerve impulses will each travel by crossed fibres through the optic chiasm to a cell in the opposite visual cortex on each side. Comparison of the receptive fields must now be made, not within a single cell in the contra lateral cortex but by the comparative integration of the activities of two cells, one in each hemisphere. This comparison can only be made by transfer of information across the corpus callosum.

Similarly, if the point of interest is just within fixation (closer) its image will fall on the temporal half of the retina in each eye and the resulting nerve impulses will each travel by uncrossed fibres to a cell in the ipsilateral visual cortex on each side. Again comparison can only be made via the corpus callosum.

Experimentally, callosal units have been recorded which could serve this purpose (Berluchi, Gazzaniga and Rizzolatti, 1967).

Choudhury, Whitteridge & Wilson (1965) have confirmed the function of these units by recording specific cortical cells before and after callosal section and before and after cooling of the corresponding point on the opposite visual cortex.

Hubel and Wiesal have recorded simultaneously the activity of separate cortical cells at the junction of areas 17 & 18 in each visual cortex and have found that some had receptive fields which overlapped the midline. Blakemore (1970) has shown that the centres of some receptive fields can be on the wrong side of the midline by as much as 1½ °. The size of each one of a pair of these overlapping receptive fields is much more equal than that of relative receptive fields in more lateral regions (Bishop, 1972).

Clinically, it has been proved that patients who have suffered a division of their corpus callosum lose stereopsis for objects in front of or behind fixation on the midline, but retain stereopsis for objects to the right side or the left side of the midline (Mitchell & Blakemore, 1970).

Stone (1965) has shown histologically that there is a midline overlap of the temporal and nasal retinal elements and this overlap has been shown to persist in the lateral geniculate body (Sanderson & Sherman. 1971) and the cortex (Leicester, 1968).

So the anatomical and neurophysiological differentiation of the central midline region of the visual function has been effectively established and it is important to realise that this differentiation results in greater stereo-acuity (ability to judge small differences in the distances of objects from the observer). (Rubin & Walls, 1965)

Human vision is not concerned with stationary objects nearly as much as with relative movement of parts of its field.

Much of the analysis of visual function is in terms of a static two dimensional situation. Each should be regarded as applicable only for an instant in the constantly changing three and four dimensional real model.

Many different analyses of lateral transverse movements are available but few of the all important (to the subject) "far to near" movements. Blakemore's discovery of constant depth columns of cortical cells in contrast to constant direction columns should emphasise the necessity for more clinical investigation of the central "near-far" distance assessment function.

The central region also has another extremely interesting attribute. It is subject to reversals which are not apparent to the observer. In other words the subject cannot differentiate "right versus left" or "left versus right" lateral reversals presented on the midline unless he has been specially trained to do so.

Many investigators have found that normal animals cannot distinguish mirror images: N.S. Sutherland with the octopus, Karl Lesley with rats, David Thomas, Nancy Wells, Michael Corballis and Ivan Beale with pigeons, John Noble with monkeys and Sir Frederick Bartlett and Norman Haber with humans (Corballis & Beale, 1971).

Noble's experiments with monkeys who had had the optic chiasma divided by midline sagittal section are particularly revealing. (Fig. 4) (Noble, 1966).

Each occipital lobe of an animal so treated can receive direct visual impulses only from its ipsilateral eye. With the chiasm divided, the only other communication of the occipital lobe with the contralateral eye is via the corpus callosum.

Noble trained monkeys with divided chiasms to react to an asymmetric figure (e.g. 7) with one eye occluded. When they were tested with the opposite eye covered and the originally occluded eye open, they reacted to the mirror image of the test figure (e.g. ꓶ).

Berluchi (1968) has shown that it is possible to train one half of the brain of an animal to react to one stimulus and the other side to react to the opposite provided that both optic chiasm and the corpus callosum are divided and the animal is trained with alternate eyes appropriately occluded.

FIGURE 4 FROM DAVSON

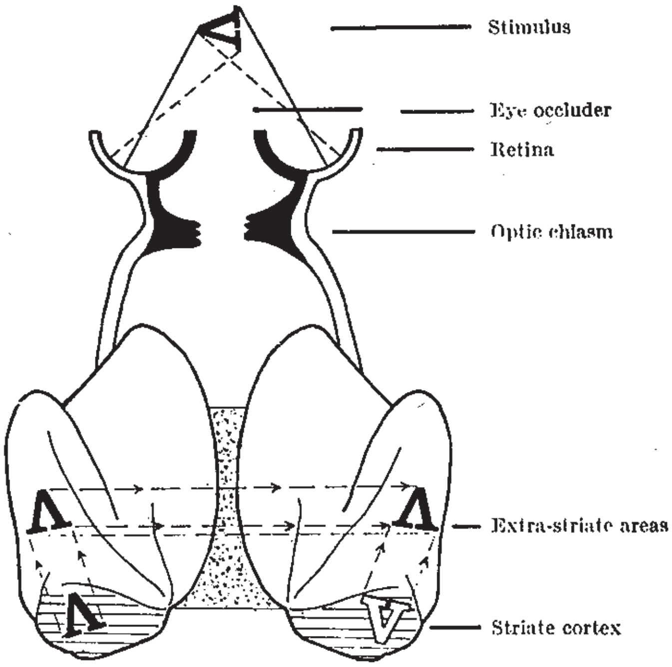


Diagram to explain mirror-image reversal. With right eye occluded, the input to left striate cortex is propagated to extrastriate areas (Λ) and is laterally reversed in crossing the midline via the forebrain commissures (stippled). When the left eye is occluded the input to the right striate area (V) does not match the stimulus (Λ) transposed from the left hemisphere; it does, however, match its mirror-image. (Noble, *Nature.*)

Beale, Corballis (et al 1972) etc. have shown with pigeons "that normal birds showed a tendency to respond to mirror-image sloping lines as equivalent, whereas split brain birds do not. This is consistent with the view that the inter-hemispheric commissures are critical to mirror image confusion." They further remark that "the finding is of possible significance in understanding mirror-image confusions of reading and writing commonly observed in children and in cases of dyslexia."

It is about the midline that reversals occur. They occur when the individual has no means to distinguish left from right.

But why should he differentiate R/L or L/R profiles? Nothing in Nature requires such differentiation and it should be no surprise that birds, fish and animals cannot distinguish reversed figures. The fish that swims up his pool looking at the left bank with his left eye need recognise no difference in the lateral reversal of this scene as he swims back again with the same bank on his right side surveyed by his right eye.

And to the rabbit either profile of the fox is equally dangerous.

Nowhere in Nature is the animal required to differentiate lateralisation. Even trained humans have difficulty in assessing laterality of remembered picture profiles. (Bartlett & Haber).

Only in man's world of symbolic communication and in the use of complex tools is the special technique of the L v R code necessary.

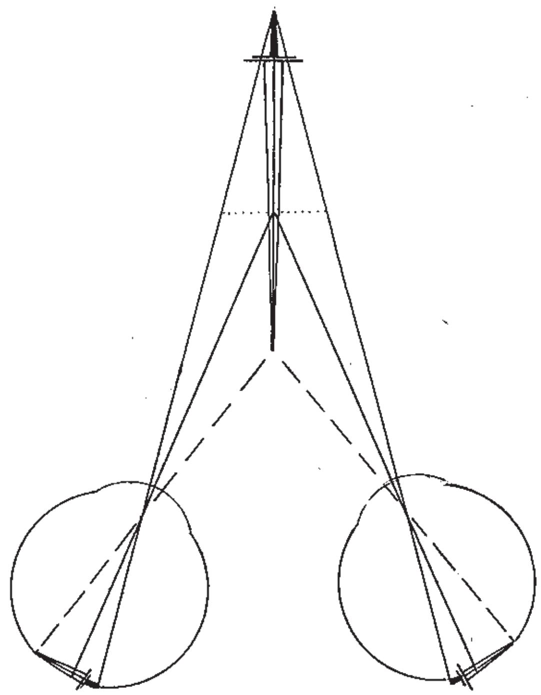
And having adopted this special technique of codifying asymmetric symbols and objects, what mechanism does man use to decode the information thus hidden from animals and the uninitiated? He can hardly escape the necessity to use the mechanism which yields the highest visual acuity. The central 2°- 5° of his visual field which has the highest cone density, (Davson, 1972). The same narrow central region which yields his highest stereo-acuity (Ogle, 1962)- The region where receptive fields are found on the "wrong side" of the midline (Blakemore, 1970) and where naso-temporal overlap occurs (Stone, 1965).

He uses his age old "relative distance" survival mechanism.

Over countless milleniums the developing animal has evolved more and more special nerve pathways (up to 50% uncrossed nerve fibres in the human) to effect a more and more accurate and rapid assessment of relative distance. Now suddenly man has chosen to convert this "too near, too far" assessment mechanism to a "left of centre versus right of centre" mechanism.

From the diagram (Fig. 5) one can see how this transformation works.

FIGURE 5



If a sword is pointing between the eyes of a frightened observer he cares little if he sees the right side of the sword with his right eye or the left side with his left eye or the fused stereo combination of both. He watches the point which will first hurt him and the hand behind that will guide the weapon. He is vitally interested in both, and, if he fixes his focus half way in between, so that both point and hand are in relatively good focus, the image of the sword will lie from right to left across the midline of his right retina and from left to right across the midline of his left retina.

Here we see the primary reason for the animal's acceptance of L & R images as equal and the basis of the decoding dilemma of the individual who wants to use his far near distance assessment mechanism for "right versus left" lateralisation assessment.

Using lateralised symbols for communication , modern man must accept one image as the standard correct orientation and reject the other as incorrect. Thus it is essential for one eye to become the master or reference eye in the central binocular region when it is used for interpreting symbols. (Note that it is not one half of his central field which becomes dominant, but one whole central field.)

It is astounding that this transformation of function should fortuitously work so consistently when one considers that no such transformation was necessary to the first nomadic hunters nor to the later more civilised tillers of the soil.

Up till this century the majority of humans were illiterate and had no use for such a lateralised decoding mechanism. Now, almost every human is expected to make a successful attempt to learn the code. It is not surprising that a few fail or find great difficulty.

But why do they fail?

It can be seen from the previous explanation and diagrams that there is normally little necessity or opportunity for dominance of one image on the narrow central binocular field over the other. And if the animal is itself perfectly symmetrical it would have no internal mechanism with which to attach a significance to a lateralised element of its environment. It would have no decoding mechanism.

Here, at last, we can see some significant meaning in the frequent finding that patients with severe dyslexia have faultlessly equal vision in each eye.

The individual must develop some means of attaching significance to right or left. If he is unable to do so he will remain in a state of permanent dilemma. The more perfectly symmetrical his functions, the more difficult is his dilemma.

It thus becomes clear that some "lateralisation" of the central visual function is essential. While the lateral receptive fields of both the binocular and monocular areas will each dominate the individual cells of the contra lateral visual cortex, due to their essentially fixed primordial neuro-anatomy and physiology, the receptive fields of the central binocular region with their double crossed and double uncrossed representation in the two occipital lobes carry no such obligatory dominance.

But some relative difference must be developed or imprinted before the central binocular area can be used for the modern symbolic decoding and coding involved in reading and writing.

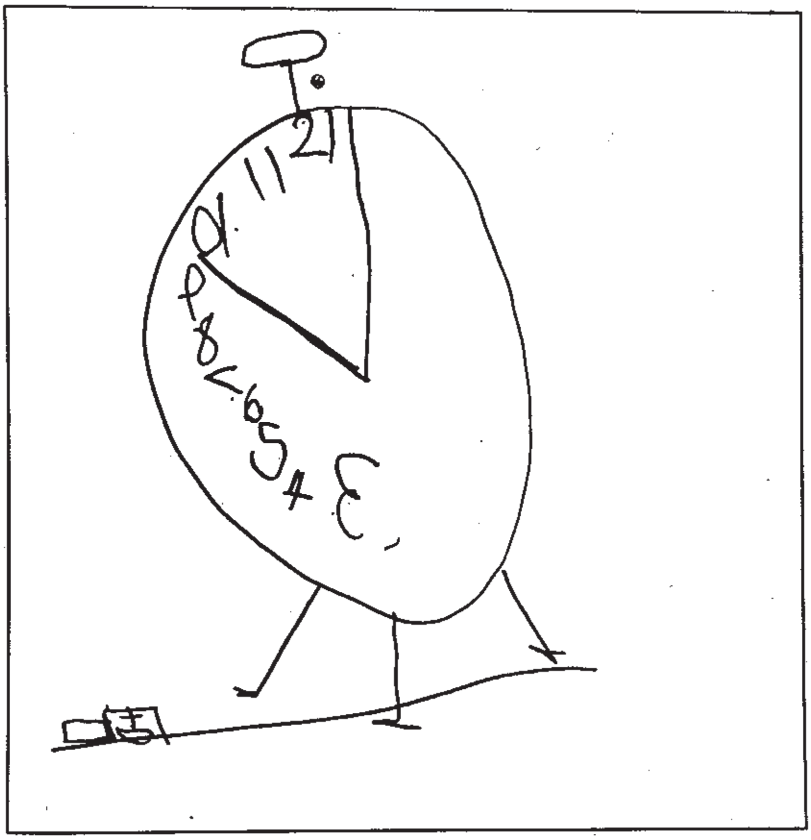
If the individual fails to achieve a consistent and reliable lateralisation of his central visual functions he will be no more able to tell whether any one symbol or any group of symbols are correctly orientated, than a totally colour blind person can assess whether an object is coloured or not. He can only keep guessing and wondering how other people find the task so easy. Like the partially colour blind, he will develop various tricks which seem to help him to guess a little better. He will twist his book and turn his head and may achieve some degree of lateralisation by keeping the image in a more peripheral part of his visual field so that a natural lateralisation is achieved.

But as soon as he reverts to the central high acuity region of his visual field he will be just as confused as ever. Eventually he may become quite distressed whenever he is asked to use his central field for symbolised detail. In such a situation a normally happy child will suddenly become fidgety and upset, almost hyperactive, as he squirms to avoid the insoluble confusion of his central visual field. He seems to try to avoid fixing his eyes with "movements oculaires inutiles." Lesèvre (1964). Just as the occasional cases of strabismus will exhibit "horror fusionis" this child will exhibit "horror confusionis centralis."

The spacial arrangements of his work become wildly erratic as he desperately tries to write in his lateral field of vision or anywhere off centre; letters and groups of letters (up to a few degrees wide) become reversed and sequentially muddled as he guesses which image is acceptable; and if he twists his book and his head far enough in opposite directions ($>45^{\circ}$ is enough) he may achieve inversions of letters (Corballis & Beale).

The figure of the 6 year old boy's drawing of a clock from McDonald Critchley's book "The Dyslexic Child, (1970)" shows all these features except inversions (Fig. 6).

FIGURE 6



Drawing of a clock, showing severe spatial difficulties, neglect of the right half of the dial, and various rotations and reversals.

The child was an intelligent boy of 6 years 8 months with a family history of dyslexia. Seen again at the age of 9 years and 1 month, his reading and spelling ages were at a 7-year level. His spontaneous drawing of a clock was then well executed.

This boy was able to draw a clock successfully a few years later but he still was unable to overcome all his reading (decoding) problems.

This case illustrates the fact that, as with strabismus, it is necessary to initiate the correct pattern of visual function early in life for best facilitation of the definitive neural pathways. (Schiffman, Lawson, 1970).

We have been discussing the most difficult of the dyslexic type of visual problems. A different type arises where the individual eventually achieves visual lateralisation but allows the eye which does not correspond to the cerebral lateralisation of his speech centre and motor functions to become the master or reference eye.

Here again, reversals of the central part of the binocular image and the resulting confusion of visual sequences occur owing to the natural tendency of callosal transfer to give preference to the mirror image, while the more direct neuronal pathways give the opposite lateralisation.

Both these types of case will tend to occur much more readily in families whose hereditary features lead to a low emphasis on lateralisation.

Thus we can understand how the change demanded of man's central binocular visual function, which has been violently sudden in relation to the time scale of man's development as an animal, has caused great difficulty for those who cannot adapt themselves to the new sophistication of the visual environment.

We can help such individuals very substantially and perhaps almost eliminate this specific problem if we can develop tests to isolate those who lack the ability to lateralise easily and if we can show them early in life how to achieve lateralisation of central binocular vision corresponding to that of other functions lateralised for more efficient specialisation.

The orthoptic test Mrs Dunlop will describe is only a beginning. Once the neurophysiology of central binocular vision and the essential necessity of developing harmonious lateralisation of various functions is recognised, many other and possibly better tests will be evolved (e.g. using Julesz stereograms) and suitable regimes of treatment should be developed.

Other implications of these concepts within the area of symptomatic phorias and strabismus are already becoming evident and will be the subject of later communications.

REFERENCES:

Adler, F.H. (1965) *Physiology of the Eye,* 4th edition. St. Louis C.B. Mosby. p.869.

Barlow, H.B., Blakemore, C., Pettigrew, J.D. (1967) *The Neural Mechanism of Binocular Depth Perception.* J. Physiol. 193, 327-342

Beale, I.L., Williams, R.J., Webster, D.M., Corballis, M.C. (1972) *Confusion of Mirror Images by Pigeons and Interhemispheric Commissures.* Nature. 238, 348-349.

Berlin, R., (1887) *Eine besondere Art de Wortblindheit. Wiesbaden:*

Berlucci, G., Gazzaniga, S., Rizzolatti, G. (1967) *Microelectrode Analysis of Transfer of Visual Information by the Corpus Callosum.* Arch. ital. Biol. 105, 583-596.

Berlucci, G. Rizzolatti, G. (1968) *Binocularly Driven Neuroses in Visual Cortex of Split-Chiasm Cats.* Science 159. 308-310.

Berner, G.E. and Berner, Dorothy E. (1953) *Relation of Ocular Dominance, Handedness and the Controlling Eye in Binocular Vision,* Arch. Ophthal. 50. 603-608.

Bishop, P.O. (1972) *Eye, Brain and Mind.* 44th ANZAAS Congress, Presidential Address, Sydney.

REFERENCES Contd.:

Blakemore, C. (1969)
Binocular Depth Discrimination and the Nasotemporal Division. J. Physiol. 205. 471...497.

Blakemore, C. (1970) *Binocular Depth Perception and the Optic Chiasm.* Vision Research. 10: 43-47.

Blakemore, C. & Pettigrew, J.D. (1970)
Eye Dominance in the Visual Cortex. Nature 225:426-429.

Choudhury, B.P., Whitteridge, D., Wilson, M.E. (1965)
The Function of the Callosal Connections of the Visual Cortex. Quart. J. Exp. Physiol. 50, 214-219.

Corballis, M.C., & Beale, I.L. (1971)
On Telling Left from Right. Scientific American 224. (3) 96...104.

Critchley, M. *The Dyslexic Child.* Heinemann. London

Davson, H., (1972) *The Physiology of the Eye.* London, Churchill Livingstone. P. 126.

Duke-Elder, W.S. (1958) *System of Ophthalmology.* Vol. 1. 673 and 678.

Dunlop, D.B., Dunlop, P., Fenelon, B. (1973) Cortex Vol.9 227-236

Flax, N. (1966) *The Clinical Significance of Dominance.* Amer. J. Optom. 43. (9) 566-581.

Holmes, G. (1945) *Organisation of the Visual Cortex in Man.* Proc. Roy. Soc. B. 132. 348-361.

Hubel, D.H., Wiesel, T.N. (1962)
Receptive Fields, Binocular Interaction and Functional Architecture in the Cat's Visual Cortex. J. Physiol. 160, 106-154.

Joshua, D.E., Bishop, P.O. (1970)
Binocular Single Vision and Depth Discrimination: Receptive Field Disparities for Central and Peripheral Vision etc. Exp. Brain Res. 10:389-416.

Julesz, B. (1964) *Binocular Depth Perception without Familiarity Cues.* Science. 145. 356-362.

Leicester, J. (1968) *J. of Neurophysiology* 31, 371-382.

Lesèvre, N. (1964) *Les Mouvements Oculaires d'Exploration. Thèse de Paris.*

Mitchell, D.E., Blakemore, C.(1970)
Binocular Depth Perception and the Corpus Callosum. Vision Res. 10, 49-54.

Money, J. (1968) *Handbook of Clinical Neurology.* Vol. 4. Vinken, P.H. Bruyn, G.W.Ed. Amsterdam.

Noble, J. (1966)
Mirror Images and the Forebrain Commisures of the Monkey. Nature, 211, 1263-1266.

Ogle, K.N. (1953)
Precision and Validity of Stereoscopic Depth Perception from Double Images. J. Opt. Soc. Am. 43, 906-913.

Ogle, K.N. (1962) *The Eye* Davson Ed. Vol. 4, p. 290. Academic Press, New York and London.

Pettigrew, J.D. (1972) *The Neurophysiology of Binocular Vision.* Scientific American 227 (2) 85-95.

Rosennerger, P.B. (1970)
Visual Matching and Clinical Findings among Good and Poor Readers.
Amer. J. Dis. Child. 119, 103-110.

Rubin, M.L. Walls, G.L. (1965) *Studies in Physiological Optics,* Springfield. Charles C. Thomas.

Sanderson, K.J. & Sherman, S.M. (1971) *J. of Neurophysiology* 34, 453-466.

Schiffman, quoted by Lawson, C.J. (1970)
Reading and Learning Problems, Ophthalmological Management. Illinois Med. Journal.137,623-6.

Stone, J. (1965) *J. of Comp. Neurology* 124, 337-352.

Walls, G.L. (1951) *A Theory of Ocular Dominance.* Arch. Ophthal. 45, 387-412.

White, M.J. (1969)
Laterality Differences in Perception. A Review. Psychol. Bull. 72, 387-405.

AN INTERDISCIPLINARY APPROACH TO DYSLEXIA

PAPER 3: DYSLEXIA – THE ORTHOPTIC APPROACH

Patricia Dunlop, D.B.O. (London)

(Presented in Sydney, October, 1972)

In this paper are described a pattern of ocular conditions found in a group of children with specific developmental dyslexia, and a new orthoptic test which will demonstrate a distinct difference in the visual functions of the children with this type of dyslexia.

Up to the present, orthoptists have seen only those children with learning difficulties who have distinct muscle imbalance. While this does occur in some cases of learning difficulties it is not characteristic of "specific developmental dyslexia." These children usually have good vision and only a small degree of muscle imbalance. It is the analysis of binocular vision in this specific developmental group which I would like to discuss.

In the early stages of the work I tried the usual methods of determining the controlling eye and found them to be unsatisfactory, largely because, at the point of decision, the patient was actually monocular in the central field. So this new test was specifically developed to determine the reference or controlling eye in the central binocular field in the binocular state as opposed to the monocular state.

It would be better to call this a test for the "reference" eye because it is essentially different to the test described by the Berners' and others for the so-called "controlling" eye (Berner & Berner, 1953, Bettman et al. 1967, Helveston et al. 1970).

Ogle (1962) notes that the phenomena of directional difference of fused disparate images within Panum's area is a possible basis of tests for ocular dominance.

Assessment of the reference or controlling eye is carried out using a pair of fusion slides on the synoptophore and performing disjunctive movements so that some fusion disparity within Panum's area is apparent. Thus it is possible to discern the eye used as a reference while central fusion is still maintained.

FIGURE 1

Fusion slides with indicators (large and small trees) used to determine reference eye.

Figure 1 shows the pair of slides used in the synoptophore to determine the reference or controlling eye in central binocular vision. The fused image of the two slides subtends an angle of 5° on the retina. The child fixes on the door which is about 1° and both trees, the indicators, are seen 1° to either side. Disjunctive movements are carried out until fusion breaks giving the measurement of the amplitude of fusion. The child is further questioned on the movement of one or other tree which occurs before fusion breaks. This movement of the tree is more easily seen in divergence than in convergence because in convergence the accommodative element can be confusing. Divergence must be done very slowly and fixation maintained steadily on the central object, only answers taken before fusion break occurs are valid as alternation quickly takes place after fusion breaks.

Mr. Fenelon selected children for an experimental and a control group. The group consisting of 15 experimental and 15 control children was sent in random manner so that, at the time of testing, I was unaware of the category to which each child belonged.

All these children had passed the routine school medical service examination as being without significant ocular defect. These experimental children were severely retarded readers and were diagnosed as follows:-

1. reports of marked reading retardation from school and home
2. average or near average intelligence
3. low achievement in standardised reading tests
4. presence of specific dyslexic signs, e.g. inversions of letters; reversals of letters and words
5. no history of brain damage
6. normal childhood health
7. absence of uncorrected sensory defect
8. normal school attendance
9. no deprivation of educational opportunity

The normal or control group were of average or above average intelligence with no evidence of learning problems.

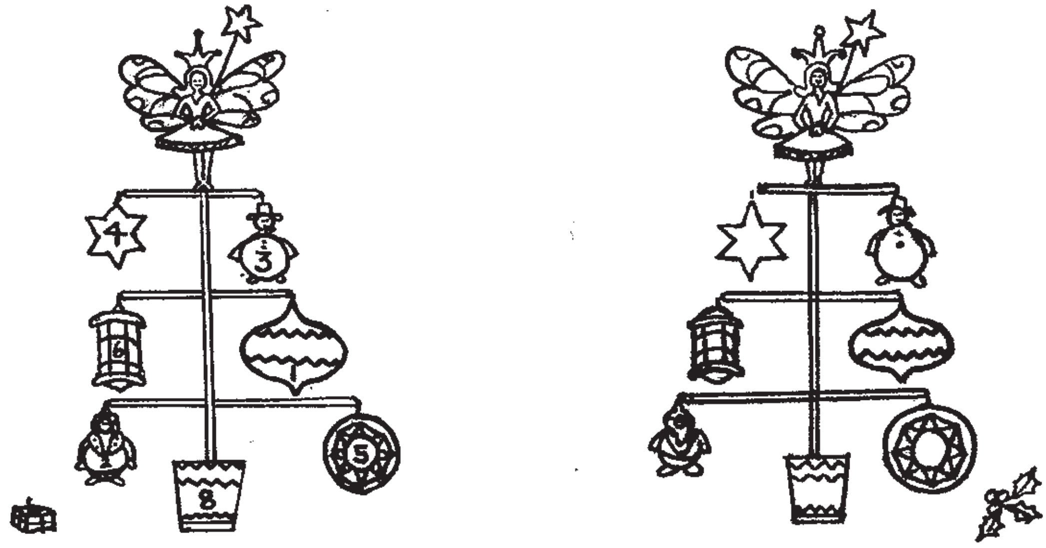
Table 1 shows the defects found and their statistical significance. (See Page 19)

Visual acuity was generally equal in both groups and was about the level of 6/6. Unequal visual acuity was in the order of ½ line difference. The esophoria found in these subjects was the group 4 type of esophoria, Mayou (1968). The esophoria measured on the Maddox Rod is small 2-3$^{\Delta}$ and the Maddox Wing measures exophoria between 2..6$^{\Delta}$. While this imbalance is small, it is consistent in the children with specific dyslexia.

Convergence is generally up to 6-8 cms (but can be only 12-15 cms). This is not well maintained and voluntary convergence is poor. Sometimes there is head retraction on attempting full convergence. Convergence deficiency has previously been noted in children with learning difficulties, Guthrie and Bermingham (1972).

Stereopsis is present but is defective, in that its appreciation is slow and often needs stimulation. On using the Titmus-Wirt card they manage up to 80 secs of arc disparity with ease (stereo-acuity). One sometimes sees the child trying to view the spots from one side in order to appreciate the depth. However, a more complex pattern of eight stimulus objects (see Fig. 2) used on the synoptophore and placed in the central binocular field gives rather poorer results and stimulation is often necessary (stereo-perception).

FIGURE 2



Stereoscopic slides used to test stereopsis

It must be realised that, as Ogle (1962) states, "that the magnitude of the stereoscopic depth perception must be carefully distinguished from the stereoscopic acuity or the precision of that depth."

The lack of significance of the crossed hand and sighting eye (a monocular test as shown in Table 1) is a finding supported by, and all too evident in, the massive accumulation of papers which have ineffectively attempted to correlate the dominant (or sighting eye) with the dominant hemisphere. Walls (1951) Lederer (1961), Gronwall & Sampson (1971) Norn (1969).

Certain factors show a significant difference when one group is compared with the other, but prediction from single indicators is hazardous and it is advisable to seek effective combinations of variables. Esophoria is not a good single predictor, but it shows possibilities in combination with defective stereopsis and crossed correspondence (handedness and reference eye). This triple combination mis-classified only one member of each group. The chi-squared value was 19.2 ($p < 0.001$), which is highly significant.

Attempts by other authors to use the Berners' concept (1953) of the controlling eye to correlate ocular and cerebral lateralisation, e.g. Bettman (1967) and Helveston (1970), have produced disappointing results because the authors have failed to realise the difference between the truly binocular state and the monocular state which immediately follows the breakdown of binocular vision. No definitive results are likely from investigations which fail to distinguish between observations made in a truly binocular state and those observations based on suppression, alternation or retinal rivalry. In all these conditions binocular vision has already broken down. A significant feature in the two most recent of such investigations using the Berners' test is the author's inability to demonstrate any control" in half their cases, even the normals. Bettman, (1970) Helveston, (1970).

TABLE 1

ORTHOPTIC EXAMINATION DATA SUMMARY

NUMBER OF SUBJECTS DEMONSTRATING OCULAR CONDITIONS

Groups	Unequal Visual Acuity	Convergence Deficiency	Defective Stereopsis	Crossed Corres-pondence*	Crossed Dominance**	Esophoria	Exophoria
Normal (n= 15)	3	4	0	1	3	7	6
RD (n =15)	5	13	9	10	8	11	4

(* *Reference eye in binocular vision opposite to handedness)*

(** *Sighting eye in monocular viewing opposite to handedness)*

Differences exist in the incidence of ocular conditions in the two groups. These differences are statistically significant for:-

Convergence Deficiency — $^*chi^2 = 8.7$, ($p<0.005$)

Defective Stereopsis — $^*chi^2 = 5.2$, ($p<0.025$)

Crossed Correspondence (handness and reference eye) — $^*chi^2 = 9.1$, ($p<0.001$)

but **not** significant

Crossed handedness– Sighting Eye — $^*chi^2 = 1.4$

Combination of Esophoria, Defective Stereopsis and Crossed Correspondence

$^*chi^2 = 19.2$, ($p<0.001$) is highly significant.

(All chi-square tests involved a single degree of freedom and Yates correction was applied).

The main value of these new procedures must be the fact that it gives us a new ability to predict the child at risk before his disability is magnified by habitual confusion and subsequent failure and frustrations. It may also prove to be useful in the assessment of treatment. As in any work on binocular function, the earlier treatment is initiated the more likely it is to be successful.

Treatment of the dyslexic child must involve multiple workers in multiple disciplines. The main treatment will always be Educational. The Orthoptist can help in an attempt to establish normal laterality and to alter the reference eye to the side which corresponds with the child's cerebral dominance. It is known that the sighting eye becomes fixed early in life, but the reference eye can be altered until a much later age.

The concept of the reference eye in relation to laterality has application in other fields, such as in symptom-producing heterophoria and the effects of acquired defects of visual acuity, including the effects of prolonged occlusion in some cases.

Orthoptic treatment of children with learning difficulties is only experimental at present. Further well controlled research programmes will have to be instituted including workers in all the disciplines involved, to truly assess the merit of orthoptic procedures. The value of orthoptic treatment will have to be assessed by remedial teachers and psychologists with adequate and detailed assessment of each child before and after treatment. The orthoptist on her own is not in a position to assess the true value of her treatment.

REFERENCES:

Berner, G.E., & Berner, Dorothy E. (1953)
Relation of Ocular Dominance, Handedness & the Controlling Eye in Binocular Vision. Arch. Ophthal. 50, 603-608.

Bettman, J.W., Stern, E.L., Whitsell, L.J., & Gofman, G.F. (1967)
Cerebral Dominance in Developmental Dyslexia. Arch. Ophthal. 78, 722-729.

Gronwall, D.M.A. and Sampson, H. (1971)
Ocular Dominance. A Test of Two Hypotheses. Brit. Journal Psychol. 62, 175-185.

Guthrie, D.I. and Bermingham, I.H. (1972)
A Clinic for Children with Reading Problems. Med. Journal Aust. 1. 149-158.

Helveston, E.M., Phillips, W.C. & Weber, J.C. (1970)
Controlling Eye — Dominant Hemisphere Relationships as a Factor in Reading Ability. Amer. Journal Ophthalm. 70:96-100

Lederer, J. (1961) *Ocular Dominance.* Aust. Journal Optom. 44, 531-574.

Mayou, S. (1968) *A New Approach to Esophoria.* Brit. Orth. Journal 25, 50-65.

Norn, M.S., Rindziunski, Eve, Skydsgaard, H. (1969)
Ophthalmic and Orthoptic Examinations of Dyslectics. Acta. Ophthal. 47, 147-160.

Ogle, K.N. (1962) *The Eye* Davson H. Ed Vol 4 page 280. Academic Press Inc., New York.

Ogle, K.N. (1962) *The Eye* Davson H. Ed Vol 4 page 297. Academic Press Inc., New York.

Walls, G.L. (1951) *A Theory of Ocular Dominance.* Arch. Ophthal. 45, 387-412.

READING DIFFICULTIES

Neryla Jolly

Presented in Adelaide, 1972 (Emmie Russell Prize)

Reading difficulties are becoming increasingly important. During the past eighteen months, methods such as flash cards, sentence building, Frostig, Initial Teaching Alphabet and programmes initiated by Science Research Associates, are coming under discussion when orthoptically examining and teaching children.

The aim of the education system is to train children to read at as early an age as possible. However, this is not without some effects on the physical components of children's reading mechanisms.

Reading is a long time development progressing over many years. Physical handicaps present during the basic and formative stages will generally cause low grades and slow progress, because of lack of initial understanding. A physical handicap occurring later in the learning process may hinder further progress and lead to frustration which can be aggravated by pressure to succeed from parent and school. As orthoptists we see patients after the frustration has become manifest and caused worried parents to seek medical assistance.

In this paper, four questions relating to reading difficulties are investigated.

A. Is the age at which a child can read decreasing?

B. Are we finding the need to treat children with latent eye defects which become decompensated and produce symptoms, at a younger age?

C. What is the frequency of occurrence of various symptoms?

D. What is the probability that any child who is attending the clinic is attending for treatment of a latent deviation.

The data used in this investigation was analysed statistically, using the chi-squared method for comparative tests. This method tests the probability that for every one hundred trials examined similar results will occur. If we can expect similar results in 95 of these trials then we are 95% confident that our results are true. To be significant the results must have a confidence level of 90% or over.

A. With regard to the first question, there is for every orthoptist a mental equating of age, class and reading ability. This provides some guide as to when a child can transfer from doing the vision test with the illiterate "E" chart, to the more accurate Snellen letter chart. Thus the age at which the transfer occurs has been used in this study as a criterion of reading ability.

In 1949 the 12th International Conference of Public Education put out a publication which stated that "formal reading from simple books begins at just over 6 years in N.S.W. (Class 1)."

In an orthoptic clinic approximately eight years ago, it was observed that children in second class were the youngest who could cope with the Snellen letter chart. In 1971, children in kindergarten were able to do this test. However, to investigate this development, a further survey of children referred to the orthoptic clinic in each of the three years, 1967, 69 and 71 was carried out.

All the above mentioned children were included in the survey regardless of the nature of their eye defect. Their ages varied between 4 and 8 years. The age of 4 years was chosen so that those children who were reading prior to formal education could be included. All patients had been referred by an ophthalmologist and so had been examined and given suitable glasses where needed.

Although the results only cover a span of four years, they show that a significant decrease in the age at which 50% or more of the children are reading, has occurred.

The results are shown in Table 1 and graphed in Figure 1.

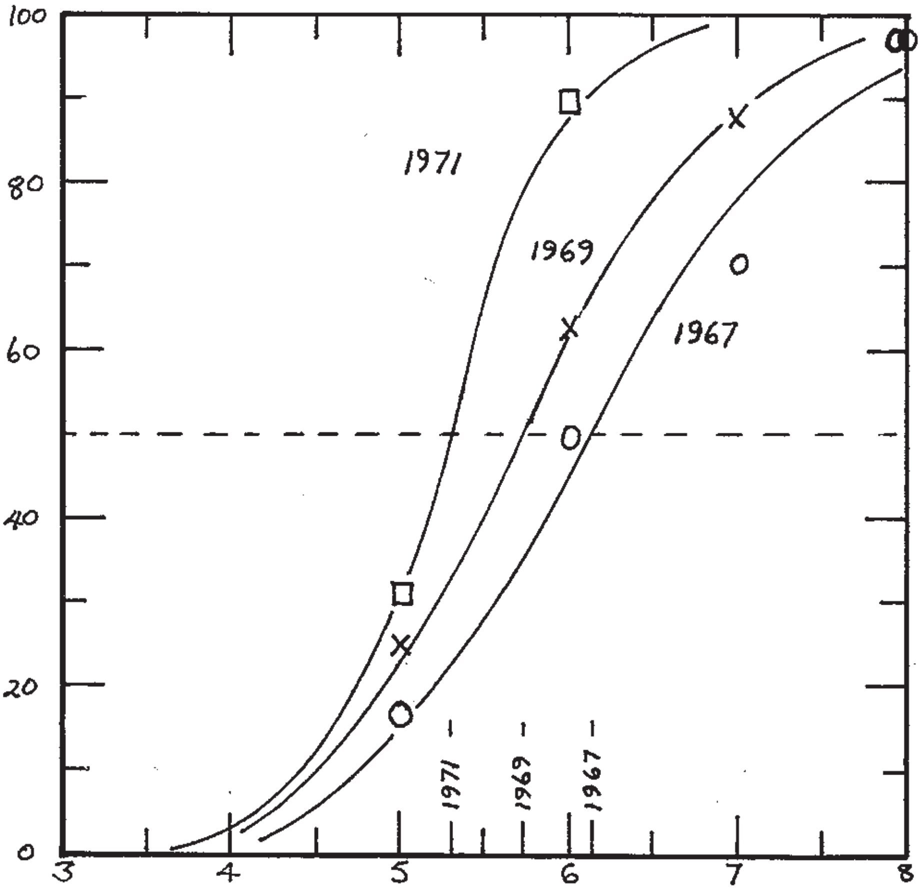
PROBABILITY OF READING

TABLE 1

AGE	1967		1969		1971	
	NUMBER	PROBABILITY	NUMBER	PROBABILITY	NUMBER	PROBABILITY
3	1	0	4	0	5	0
4	14	0	20	0	21	0
5	18	17%	30	23%	30	33%
6	33	52%	24	62%	20	90%
7	12	67%	16	88%	16	100%
8	13	100%	21	100%		

FIGURE I

% READING AT GIVEN AGE



Referring to the table it can be seen that between 1967 and 69 there was an increase in the probability that a child of a given age would be reading. However, these differences when tested statistically are not significant. During the interval 1969 to 71 there was a further increase in the probability that a child of a given age would be reading. This increase from 1967 to 1971 for 6 year old children was highly significant (99% level of confidence) a finding which supports our hypothesis that the average age at which a child learns to read at Sydney suburban schools is decreasing. The increase over the same period for 5 year old children is significant (at the 95% level).

It is interesting to note that in the three years under study the average age per class has remained constant.

B. With regard to the second question, age and symptomatic heterophorias, an additional survey was carried out.

The age range in this instance was 4 to 12 years. Twelve years was chosen as the cut off point since above this age children have, in general, completed primary education and are undergoing a more strenuous close work programme.

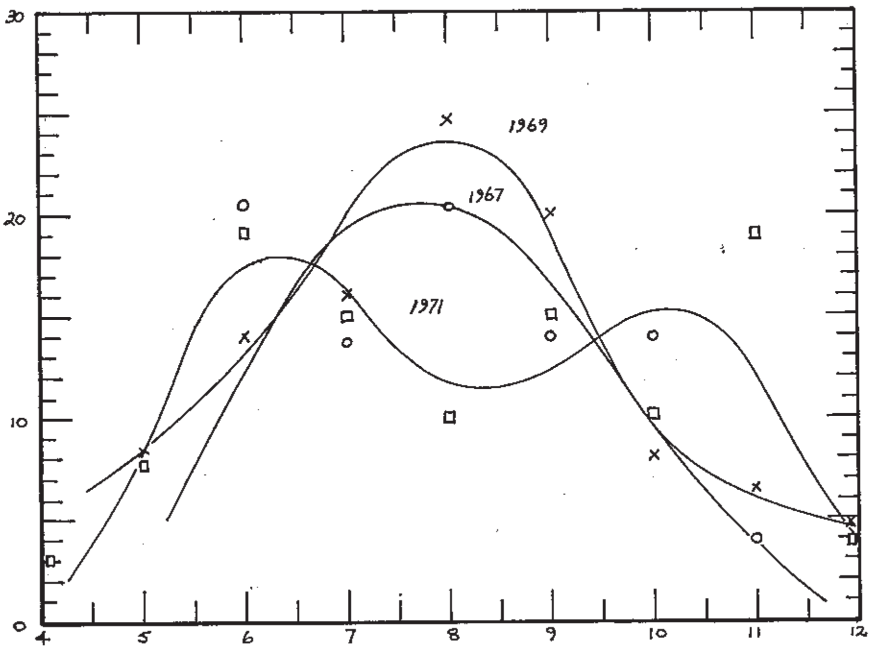
The distribution by age of the patients with latent deviation was investigated. The results can be seen in Table II and are plotted in Figure 2..

AGES AT WHICH LATENT DEVIATIONS ARE DISTRIBUTED

TABLE II

YEAR / AGE	1967		1969		1971	
	NUMBER	PROBABILITY	NUMBER	PROBABILITY	NUMBER	PROBABILITY
4	–	–	–	–	2	3%
5	–	–	4	8%	5	7%
6	6	21%	7	14%	13	19%
7	4	14%	8	16%	10	15%
8	6	21%	13	25%	7	10%
9	4	14%	10	20%	10	15%
10	4	14%	4	8%	7	10%
11	4	14%	3	6%	13	19%
12	1	3%	2	4%	2	3%
TOTAL	29		51		69	

FIGURE 2



Points of interest are:-

1. the proportion of 8 year olds in the 4-12 years age group of heterophoric patients has decreased, from 1969 to 1971. This decrease is significant at 98% confidence level. (Highly significant)

2. the proportion of 11 year olds in the 4-12 years age group of heterophoric

patients has increased, from 1969 to 1971. This increase is significant at 98%. (Highly significant)

3. the proportion of 6 year olds has increased from 1969 to 1971. The increase in both groups is significant at only an 80% level of confidence.

4. in the curve for 1971 there are two maxima. The first is at 6 years of age, rhe second at 11 years of age.

When considering the two maxima in 1971 it was thought possible that if the group of patients under study were extended to include patients older than 12 years, then both the 1967 and 1969 curves would exhibit a second maxima similar to that of the 1971 curve. The presence of two maxima would alter the curve shape.

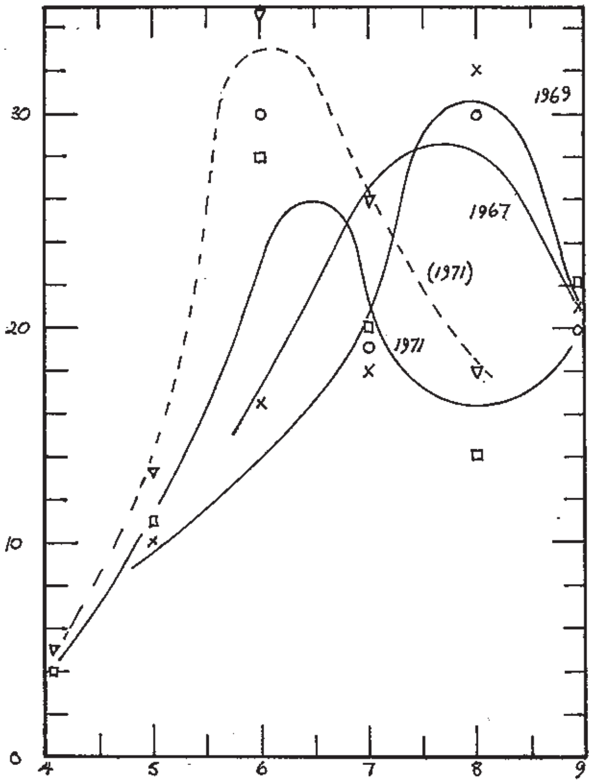
It was then decided to move the cut-off age to 9 years, that is to restrict the study to include those patients aged 4 to 9 years. This removes the levelling effect on the 1971 curve of the second maxima, and results in a more realistic comparison, since the original 1967 and 1969 curves did not show this second peak. The curves of the reduced data (Figure 3) all show a single maximum. The curve for 1971 data, recalculated with an even lower cut-off age (8 years) is also plotted in Figure 3 (broken line). The change in peak positions over the period 1967 to 1969 is not significant, however, the change from 1969 to 1971 is highly significant, and appears to support the theory that, in 1971, children with symptomatic heterophorias are appearing for treatment at a younger age than in previous years.

AGE DISTRIBUTION OF LATENT DEVIATION

TABLE III (Restricted Study: patients 4 yrs. to 9 yrs.)

YEAR / AGE	1967		1969		1971	
	NUMBER	PERCENTAGE	NUMBER	PERCENTAGE	NUMBER	PERCENTAGE
4	–	–	–	–	2	4.3%
5	–	–	4	9.5%	5	10.7%
6	6	30%	7	16.7%	13	27.7%
7	4	20%	8	19%	10	21.2%
8	6	30%	13	31%	7	14.99%
9	4	20%	10	23.8%	10	21.2%
TOTAL	20		42		47	

FIGURE 3



C. Of the patients with latent deviation, some complained of a single symptom, others of multiple symptoms. The frequency of various symptoms is summarised in Table IV.

PERCENTAGE FREQUENCY OF OCCURRENCE OF SYMPTOMS

TABLE IV

SYMPTOMS \ YEAR	1967	1969	1971
Poor reading	17%	8%	26%
Diplopia	59%	57%	39%
Headaches	24%	39%	43%
Sore Eyes	41%	45%	40%
Observed Problems	21%	27%	32%
Total Patients	29	51	69

The following points are to be noted from the table:

1. Headaches - there has been a steady significant increase in the occurrence of headaches reported over the period of study. The increase 1967 to 1969 is significant at a 90% level of confidence, whilst the increase over the past four years is significant at a 96% level.

2. Poor readers - those referred because reading was noted to be below standard. Problems included losing place and apparent wave-like motion of print.

Over the past two years the increase in the number of patients with this difficulty is highly significant (99%). However, the level in 1969 had shown a decrease from 1967, so that the overall increase from 1967 to 1971 is significant only at an 80% confidence level.

3. Observed problems - that is difficulties reported by the parents and not noticed by the patient.

   The frequency of this sign has increased steadily over the past years. The difference between 1967 and 1971 figures is significant at the 85% level of confidence.

D. The study of the probability that a child is attending for treatment of a latent deviation.

Points of particular interest are that

1. there is an overall increase in the percentage frequency of referral of latent deviations between 1967 and 1969. This increase is significant at a 99% confidence level. (highly significant)

2. there is an overall increase in the percentage frequency of referral of latent deviations between 1969 and 1971. This increase is significant at a 99% confidence level. (highly significant)

3. for 5 year old patients there is an increase from 1967 to '69 in the probability that a child will be undergoing treatment for a latent deviation. This increase is significant at level greater than 99%. (very highly significant)

4. for 6 year old patients there is an increase from 1969 to '71 in the probability that the child will be undergoing treatment for latent deviation. This increase is significant at level greater than 99%. (very highly significant)

5. for 7 year old patients there is an increase from 1967 to '69 in the probability that a child will be undergoing treatment for latent deviation. The increase is significant at only 80 to 85%.

6. for 7 year old patients, between 1969 and '71 there was an increase significant only at a 77% level of confidence.

7. for 7 year old patients between 1967 and '71 there is an increase significant at 98%. (highly significant)

8. for 8 year old patients, between 1967 and '69 there is an increase in the probability that the child will be undergoing treatment for latent deviation. This increase is significant at 90%.

9. in 1967 the age of the youngest child attending for treatment was 6 years in 1969 it was 5 years, and 1971, 4 years. Statistically these figures are only significant at the 80% level of confidence. Hence little importance can be attached to this observed decrease in age but it does indicate a trend for children to require treatment at an earlier age.

PROPORTION OF PATIENTS REFERRED AT A GIVEN AGE WHO SHOW A LATENT DEVIATION

TABLE V

YEAR / AGE	1967			1969			1971		
	Total Number	Patients with latent defect	Proportion	Total Number	Patients with latent defect	Proportion	Total Number	Patients with latent defect	Proportion
3	1	–	–	4	–	–	5	–	–
4	14	–	–	20	–	–	21	2	10%
5	18	–	–	30	4	13%	30	5	16%
6	33	6	18%	28	7	25%	20	13	65%
7	12	4	33%	16	8	50%	16	10	63%
8	13	6	46%	21	13	62%	14	7	50%
9	6	4	67%	13	10	77%	12	10	83%
10	4	4	100%	5	4	80%	8	7	88%
11	5	4	80%	3	3	100%	13	13	100%
12	1	1	100%	2	2	100%	2	2	100%
Total	107	29	19%	142	51	35%	141	69	49%

CONCLUSION

The conclusions that can be drawn from this investigation seem to answer the four questions initially raised.

A There is significant evidence that children are reading at a younger age.

B Thereseems to be a trend for children to be attending for treatment of latent deviations at a younger age.

C Amongst the children referred for clinical treatment of latent deviation, symptoms of headache and poor reading have increased in frequency.

D Of all the children referred to the clinic, the percentage who are suffering from latent deviations has increased.

Although it has not been proved, these factors may well be inter-related. It is interesting to consider why there is a trend for a decrease in age in occurrence of a latent deviation causing symptoms. Points to consider are:

i. increased awareness from the authorities, such as the Education Department through the School Medical Service and literature distributed by the optometrists.

ii. the change in pattern of the referral of patients.

iii. increased pressure from the school at an earlier age causing physical inabilities to become manifest. Knowledge of the aims and methods of the various reading techniques could be of great help in connection with this point.

A PROBLEM READERS' CLINIC

Mary Peoples

Presented in Adelaide, 1972.

Dyslexia, which Mrs. Drummond ably explained to our Melbourne conference, has had a lot of publicity lately. We prefer to call our patients "Problem Readers."

My reason for writing this paper is to show that the orthoptist can play a part in the diagnosis and assessment of children with reading difficulties.

The growing awareness of children with reading difficulties prompted the formation of a "Problem Readers' Clinic" by the director, Dr. Guthrie at the Rehabilitation Centre of the Broken Hill and District Hospital in September, 1969.

Originally, the clinic was to be experimental, with as little publicity as possible. A survey showed that 15% of the district's school population had reading problems that needed attention.

The idea was to form a diagnostic team working with the Director of Rehabilitation. The team consisted of the District School Counsellor, speech therapist, social worker and orthoptist with remedial teachers, physiotherapists, school medical officer and physician as consultants. The approach was to ensure a comprehensive assessment of the child.

As the clinic could operate only one afternoon a week, it was decided to limit referrals to children with good intellectual ability whose school progress was being retarded by reading difficulties.

After medical assessment, the child and parent or parents were interviewed by the diagnostic team and director who had already given the neurological tests. It is interesting to note that the children were not confused when confronted by this group of people, very often seeming to gain confidence when they realised they were talking to people who understood their problem.

Vision, hearing and speech were checked later and the parents interviewed by the social worker to determine emotional or social problems.

The District School Counsellor gave the standardised reading tests, I.Q., visual and auditory perception tests and other tests which help to isolate the area of weakness.

A case conference was held after the assessment with the remedial teachers often being present. The parents were again interviewed and advised on possible ways to help and understand the child's difficulties and reports were sent to the Principals, Doctors, District School Counsellor and teachers. Teachers were invited to a monthly meeting to discuss their pupil's problems in this field.

A review in 1970 proved the demand for our services. Unfortunately, a waiting list was inevitable and the need for remedial teachers great.

A grant from the Hospital made purchase of books and equipment possible. As a result, selected children were able to have three half hourly sessions weekly at no expense to the parent. They were taught by professional remedial teachers who volunteered their services.

So far this has said little of the part played by the orthoptist. I will quote from the "Australian Medical Journal", 22nd January, 1972, where anyone interested can read the whole story of our clinic.

"VISION. This examination was done by the orthoptist. The tests included visual acuity for near and distance, ocular movements, binocular vision and red and green recognition.

Results showed that there were no causes of uncorrected loss of visual acuity, but in 32% of 50 subjects there were some clinically observed difficulty, such as squints, poor convergence, slow stereopsis or muscular fatigue, 14% had eye exercises prescribed and 12% had been referred to the ophthalmologist.

It is felt, that the orthoptist, made a significant contribution to the assessment in that as a matter of procedure both visual acuity and eye movements were checked. Although none of the children referred for specialist attention had serious impairment, the number of children who admitted to blurring and to seeing double after some period of reading, suggested that in some cases fatigue and discomfort may be related to lack of motivation for reading.

This simple check, we feel, may be often overlooked in assessments of reading difficulties."

Being part of a team, has been a rewarding experience. Our clinic has continued to grow, we are members of 'SPELD' and have created a lot of public interest. Six remedial teachers give time to teaching these children, in small groups after school, and we find splendid co-operation between principals and teachers. We have had encouraging reports from them on children reviewed at the end of last year.

Undoubtedly, remedial reading is an educational responsibility. It seems that by using the combined knowledge of medicine, para-medical groups and educationists, difficulties, which are revealed in an educational context, are the responsibility of a number of disciplines. The multi-disciplinary approach assessment has helped in a problem which has caused many children difficulties. It is now being recognised that there is a group of children with good mental ability who are plagued by words without meaning.

I would like to thank Dr. D.I. Guthrie for permission to quote from his article in "Medical Journal of Australia", 22nd January, 1972.

REFERENCE:

Guthrie, D.I., and Bermingham, I.H. *Med. Journ. Aust.* 1 : 149-158 (1972).

A NEW ROLE FOR ORTHOPTICS

Jan Alexander
Presented in Sydney, 1971

Frostig reading methods are now being introduced into schools in N.S.W. This method, of American origin, is called the Development Programme in Visual Perception and is described by the promoters, as a "break-through in literacy." It consists of three series of pictures and patterns: Beginner, Intermediate and Advanced.

The books which describe the method, define visual perception and visual motor co-ordination in a manner which seems to refer to normal neurological development of the whole body. Dr. Leckie and I can find no reference to binocular vision or abnormalities of binocular function in these books.

I became involved with the Frostig method when approached by a remedial reading teacher, in private practice in Bathurst, who was worried by the way a child she was teaching was responding to the Frostig exercises. His eyes became red, he rubbed them excessively and was worried by small print.

The boy, N.W. aged 8 years, had been refracted by Dr. Leckie 5 months previously and was found to have no refractive error. Dr. Leckie agreed to his having an orthoptic examination.

This child, according to the representative from the Guidance Department, had an abnormal growth pattern and no visual motor co-ordination.

Cover Test distance: steady

Cover Test near: exophoria to L.D.S.

Convergence near point: 12 inches; with effort and the left eye failing.

Visual acuity: R.E. 6/6–2 L.E. 6/9

Worth's lights: 4

Maddox wing: exophoria 2° and blurred.

Synoptophore angles were –IR/L 1^{Δ}, fusion was held to $+5^{\circ}$ and to -3° and there was deep left suppression, stereopsis was appreciated, correspondence was normal. Part-time occlusion of the right eye after school was advised. One week later the vision was equal, 6/6–2, the left eye being slightly slower. Thereafter he attended for orthoptic treatment, involving elimination of his left suppression and improvement of convergence. After 6 treatments he was satisfactory.

His remedial teacher reported that there had been a marked improvement in his reading and stated that he is not a "brilliant" child but has progressed from being a non-reader to one who can read lengthy stories.

Four other children who had similar reading problems and showed ocular defects for near have been treated and these also have shown improvement in general school work as well as in reading.

I do not pretend to understand reading procedure - it is not my field - but as an orthoptist, I am amazed that such a method can be introduced and embarked upon without first eliminating the possibility of obstacles such as brain damage, congenital word blindness, refractive errors and abnormalities of binocular function, including convergence insufficiency.

I would like to thank Dr. T.D. Leckie for letting me present this case.

PRELIMINARY REVIEW OF 325 CONSECUTIVE CASES OF LEARNING DIFFICULTY

Patricia Dunlop, D.B.O. (London)

Presented at Canberra, April 1973 Annual Orthoptic Conference.

Children with learning difficulties are sent for eye investigation when authorities suspect a visual impairment of some kind - motor, sensory or perceptual, or when a child who is not performing for some reason, such as mental retardation, brain damage or aphasia may need to be examined to see whether there is also a visual problem, which, if treated, might make the general outlook brighter.

This is a preliminary review of 325 such cases seen during the past 18 months.

The main problem was failure to **learn** at a rate comparable to other children of the same age, or failure to **achieve** to the level of their intelligence.

This was manifested by a problem of inco-ordination, generally; poor reading or lack of fluent reading, e.g. skipping words, losing the place, difficulty in progressing from one line to the next; untidy writing; poor spelling; reversals e.g. b and d; p and q; was and saw; etc., and more rarely inversions e.g. b and p; n and u; ' and sequencing, making spelling difficult. Most of the children were failing in reading and associated skills although many were good at maths. Some were behind in all phases of learning (51 cases).

The group of 325 included 255 males and 70 females - a ratio of 3.5:1, the mean age was 10.02 years - varying between 4½ years and 21 years of age. This is a biased sample of children with learning difficulties, as they were all suspected of having a component of visual defect in their problem at the time of referral. All were seen as private patients, except two groups, (1) 15 Primary School children, (2) 19 High School children, who had participated in two research projects during that time. No. 1 group was specially selected as having specific developmental dyslexia, age variation between 8 years and 12 years. This study has been accepted for publication in Cortex. No. 2 group (aged between 12 years and 15 years) was specially selected by their remedial teacher as probably having a visual difficulty but not necessarily having specific dyslexia or signs of reversal and confusion. Reversal is not generally a problem at high school age but sequencing and adequate form perception have still to be coped with. This study was undertaken for Mr. E. Gray, Director of Education for the Newcastle area.

REVIEW OF 325 CASES OF LEARNING DIFFICULTY

TABLE I

AGE	4½ years .... 21 years.	Mean 10.02 years.		
SEX	Males... 255	Females... 70	Total... 325	Ratio M:F... 3.5:1
SOCIO-ECONOMIC BACKGROUND				
	Low.................... 22	Average 259	High 44	
INTELLIGENCE				
	Below Average..... 13	Average............... 232	Above Average... 80	
BEHAVIOUR				
	Aggressive......... 22	Normal................. 267	Hyperactive...... 36	
VISUAL ACUITY				
	Equal V.A......... 272	Unequal V.A......53	6/6 or 6/5...261	6/9 or <6/9..64
REFRACTION				
	Emmetropic....... 275	Myopic and/or Astigmatic..... 11	Hypermetropic and/or Astigmatic............ 39	
	Refractive Error.. 50			

As most of these patients were seen privately, the socio-economic background is average to high; similarly the intelligence quotient broadly speaking is also average to high, because people with this economic background would generally have higher motivation to seek orthoptic analysis. Only 22 children came from a below average socio-economic background. 44 came from a high background and the remaining 259 were from an average background.

The assessment of intelligence is not a job for the orthoptist and it is often difficult to get an accurate estimate of this in cases of learning difficulty sent for orthoptic analysis. Sometimes the parents will know (having been informed by a psychologist or hinted at by a teacher), occasionally the ophthalmologist or referring Dr. will know, or the psychologist will give an estimate on referral. Children with specific developmental dyslexia can be thought to have rather lower intelligence using some methods of testing than would be the case using another method where reading does not play such a prominent role.

I will consider the groups "average" and "above average" as a single group in this review. I single out the "below average" intelligent children as there is less chance of error in this small group (13) who appear to be well below average and many of them have evidence of brain damage (8). 3 had manifest squint with no binocular vision. Considered as a whole, the background of the total group was normal. Very few had history of neurological disorder (31), hearing defect (13), uncorrected refractive error or deprivation of educational opportunity. Some had emotional problems, a few were aggressive (22) others were hyperactive (36).

Refractive Errors. Most children were emmetropic (275) 11 had myopia and small astigmatism and 39 had hypermetropia with some astigmatism. Visual acuity was good, 6/6 or 6/5 in both eyes in 261 cases. 64 had 6/9 or less in one or both eyes. Unequal vision was present in 53 cases leaving 272 with equal or very nearly equal vision.

From these figures it is apparent that these children seem to be fairly normal young people from many angles except that they have a strange disability in learning to read and interpret symbols correctly.

TABLE II

ANALYSIS OF BINOCULAR VISION

Evidence of functional Single Binocular Vision.	Eso	Eso Group iv	Exo	Inter D.S.	Inter C.S.
322	30	236	52	3	1
No Binocular Vision.				ADS 1	LCS 1
3				LDS 1	

Convergence rating. 0...4
322 Cases. Mean 1.8

0 — 6 cms, well maintained
1 — 6 cms, with effort to maintain
2 — 8 cms, with head retraction
3 —10 cms .. 15 cms
4 — >15 cms

Stereoscopic Vision Rating. 0...4
322 Cases. Mean 1.8

0 — full
1 — good, but not full
2 — good, with stimulation
3 — fairly good
4 — poor – nil

S.V. Using Wirt – Titmus Test 9...0
282 Cases Mean 8

The Orthoptic Analysis took the usual form of orthoptic examination:- history; visual acuity; muscle movements; cover test; Maddox rod and wing: Titmus stereo-acuity test spots; visuscope check on fixation; accommodation and convergence ability; sighting eye (using a hollow cylinder to view a distant spot; preferred hand for writing; assessment of binocular vision - simultaneous perception, fusion fusional amplitude and stereopsis; and reference eye in central binocular vision (a new orthoptic test).

In this study I use the term "crossed correspondence" for a condition where the preferred hand is opposite to the reference eye in central binocular vision.

The test for reference eye in central binocular vision was developed in a previous study on Primary school children specially selected as having specific developmental dyslexia. In these cases the evidence of crossed correspondence was found to be a definite statistical significance. The triad esophoria, defective stereopsis and crossed correspondence was found to be highly significant for specific developmental dyslexia.

Previous tests to ascertain the dominant eye in binocular vision have been unsatisfactory. Recent workers in this field have been unable to make any discrimination of dominance in half their cases - even in the controls. (Bettman 1967 and Helveston 1970). These tests are based on suppression or alternation (Berner & Berner 1953). In either case true binocular vision has already failed at the point of decision.

Ogle (1962) notes that the phenomenon of directional difference of fused disparate images within Panum's area is a possible basis of tests for ocular dominance.

The new reference eye test is based on fixation disparity within Panum's area. A decision is made while true binocular vision holds in the central field, using a pair of fusion slides with slightly dissimilar indicators, e.g. house with 2 trees; house with a man and a woman.

Table II shows that most children had functional single binocular vision, only 3 squints had no binocular vision.

The evidence of esophoria (group iv) Mayou (1968) is interesting. This is a small esophoria for distance with an exophoria for near.

Convergence and stereopsis were rated (Table II) for convenience in applying statistical methods. The mean value of convergence ability is 1.8 which is between "effort to maintain" and "head retraction" at 6 cms.

Stereopsis as tested on slides with 8 stimulus objects (Xmas tree) gives a mean of 1.8 putting it near "stimulation needed to achieve almost correct answer." The Wirt-Titmus gives a mean of 8, i.e. 50 secs of arc. One should realise that these two tests for stereopsis can give strangely different results. Ogle (1962) states "that the magnitude of the stereoscopic depth perception must be carefully distinguished from the stereoscopic acuity or the precision of that depth.

TABLE III
ANALYSIS OF LATERALITY

		R. Handed	L. Handed	Ambidextrous
Total No. of Cases	325	273	47	5
Triple combination: Eso, defective S.V. Crossed Correspondence	207	182	21	4

TABLE IIIa
Handedness & Sighting Eye

Rh:Rs 138	Lh:Ls 18	A:Rs 3
Rh:Ls 130	Lh:Rs 27	A:Ls 2
Rh:Es 5	Lh:Es 2	A:Es 0

TABLE IIIb
Handedness & Reference Eye

Rh:Rr 46	Lh:Lr 13	A:Rr 1
Rh:Lr 205	Lh:Rr 27	A:Lr 0
Rh:Er 19	Lh:Er 6	A:Er 4
Rh:nil r 3	Lh:nil r 1	A:nil r

R — Right
L — Left
A — Ambidextrous
E — Either
h — handed
s — sighting eye (Monocular test)
r — reference eye (Binocular test)

Table III shows the correlation of handedness with sighting eye and handedness with reference eye. This shows clearly the high incidence of crossed correspondence 232 (reference eye test) compared with the crossed dominance 157 in 325 cases.

Taking the combination of esophoria, defective stereopsis and crossed correspondence, which was proved highly significant in the earlier study of specific developmental dyslexia there are 207 instances among the 325 cases. This indicates that there is a high incidence of the visual components of specific dyslexia, contributing to the difficulties of the children in this group.

203 cases had a family history of left handedness and 85 had history of others with learning difficulties. There were 8 adopted children in the series and no family history was available about them.

33 children had full stereopsis and analysis of this group showed an interesting factor of fluctuating reference eye in 10 of them. Taking the entire group there were 28 who had fluctuating reference eye or "lack of dominance."

There were only 49 cases of amblyopia, which was only one or two lines on the Snellen chart.

In this review I have tried to analyse the ocular side of learning difficulty, in particular, that part concerned with binocular vision.

It is clear that in cases of learning difficulty, unless binocular vision is investigated in detail the ocular findings will be within the limits of normal eye function.

With information based on binocular vision including reference eye, it is much easier to understand why these children have trouble.

To date, I have been experimenting with treatment in an effort to set ocular functions in the path of the normal as far as possible.

Intermittent squint, refractive error (particularly in the desired reference eye) convergence deficiency can all be treated readily. But the treatment for crossed correspondence is still experimental and as yet I have no controlled data on which to base facts.

However, certain aspects of the treatment are becoming clear and general orthoptic principles still apply. If you wish to alter a reflex you must get in as early as possible before normal development has stabilised. I believe the upper age limit is around 12 years of age with some variation depending on motivation etc. Using the routine of total occlusion during lessons and homework with convergence exercises to support, I have not had trouble with patch amblyopia as has been suggested by overseas authorities (Helveston 1970). I have seen children's behaviour improve, reading become more accurate and fluent, writing become legible and flowing, overall coordination of the limbs occur and willingness to approach reading material which had been the source of many a family argument previously.

The basic need to catch up in learning will of course be still necessary and special remedial teaching will have to be undertaken.

The real value of the orthoptic test for reference eye in central binocular vision lies in its unique potential to differentiate the child at risk at an early stage so that the necessity for treatment of the fully developed syndrome with all its complicated overlays may never be necessary.

The point of the orthoptic treatment is to put the child in or near the position of a normally learning child in so far as visual function is concerned, so that he will be able to pursue his studies with more reward and in time should be able to work to his full potential.

REFERENCES:

Berner, G.E. & Berner, Dorothy E. (1953)
Relation of Ocular Dominance, handedness and the controlling eye in Binocular Vision
Arch. Ophthal. 50: 603-608

Bettman, J.W., Stern, E.L., Whitsell, L.J. & Gofman, H.F. (1967)
Cerebral Dominance in Developmental Dyslexia. Arch. Ophthal. 78, 722-729.

Helveston, E.M., Phillips, W.C. & Weber, J.C. (1970)
Controlling Eye - Dominant Hemisphere relationships as a factor in Reading Ability.
Amer. Journal Ophthal. 70: 96-100

Mayou, S. (1968) Brit. Orth. Journal 25: 50-65

Ogle, K.N. (1962) *The Eye* Davson H. Ed. Vol. 4 Page 280. Academic Press Inc. New York.

Ogle, K.N. (1962) *The Eye* Davson H. Ed. Vol. 4 Page 297. Academic Press Inc. New York.

TRAINING SCHOOLS

Sydney School of Orthoptics:	Miss P.M. Lance, Orthoptic School, NSW College of Paramedical Studies, *209 Salisbury Rd., Camperdown.*
Postal Address:	*P.O. Box K348, Haymarket, 2000*
Melbourne School of Orthoptics:	Mrs. E. Cornell, Royal Victorian Eye & Ear Hospital, *126 Victoria Pde., East Melbourne, 3002.*

LIST OF CLINICS, HOSPITALS & PRACTICES

QUEENSLAND

BRISBANE

Private Practice

	Miss J.E. Kirby, *1 Wickham Terrace, 4000*		31-1597

Sponsored Practices

	Dr. M. Harrison, *137 Wickham Terrace, 4000*	Miss J. Hart	21-4972
	Dr. E.F. McGuiness, "Inchcolm", *Wickham Terrace, 4000*	Mrs. A. Dickinson	21-0437

Hospitals

	Mater Children's Hospital, *Annerley Rd., South Brisbane, 4101*	Mrs. A. Dickinson	44-0141
	Princess Alexandra Hospital, *Ipswich Rd., Woolloongabba, 4102*	Mrs. S. Daly	91-0111
	Royal Brisbane Children's Hospital, *Herston Rd., Herston, 4029*	Miss J. Kirby, & Mrs. A. Dickinson	52-0111

SOUTHPORT

Private Practice

	Miss M. Hansor, *Sunstate Building, Nerang & Scarborough Sts., 4215*		32-2455

SOUTH AUSTRALIA

ADELAIDE

Private Practice

Miss J. Taylor, *229 Melbourne St.,* *North Adelaide, 5006*		67-3304

Sponsored Practices

Dr. H. Handley, *198 North Tce., 5000*	Miss J. Gers	
Dr. J. Landers, *104 Brougham Place,* *North Adelaide, 5006*	Mrs. P. Hall	
Drs. M. Moore, J. Murchland & D. Bennett, *163 North Tce., 5000*	Miss J. Gers, Miss J. Stewart	
Drs. J. Slade, R. Renton & I.Hobbs, *79 Pennington Tce.,* *North Adelaide, 5006*	Mrs. B. Walch	
Dr. P. Stobie, *182 Ward St.,* *North Adelaide, 5006*	Mrs. P. Hall	

Hospitals

Adelaide Children's Hospital, *King William Rd.,* *North Adelaide, 5006*	Miss J. Taylor	
Royal Adelaide Hospital, *North Tce., 5000*	Miss J. Gers	

TASMANIA

HOBART

Private Practice

Miss J. Clerk & Miss B. Ross *201 Macquarie St., 7000*		23-5306

Hospitals

Royal Hobart Hospital, *48 Liverpool St., 7000*	Miss J. Clerk, Miss B. Ross	23-0521
Bruce Hamilton School, *Albuera St., 7000*	Miss J. Clerk, Miss B. Ross	23-7510

TASMANIA

LAUNCESTON

Private Practice

	Miss B. Ross *52 Elphin Rd., 7250*		2-2832
Hospital			
	Launceston General Hospital *Charles St., 7250*	Miss B. Ross	2-2221

AUSTRALIAN CAPITAL TERRITORY

CANBERRA

Sponsored Practices

	Drs. J. Lodge & D. Coop, City Chambers, *East Row, Canberra City, 2601*	Mrs. J. Garfit-Mottram	48-6128
	Dr. Y. Pittar, Prudential Building, *Darwin Place, 2601*	Mrs. S. Baird	48-9555
	Dr, L. Shanahan, *P.O. Box 718, 2601*	Miss A. Terrell	49-6000
	Dr. L. Shanahan, *Theodore St., Curtin, 2605*	Miss P. Boath	49-6000
	Dr. T. Walker, *13 Barry Drive, Turner, 2601*	Miss L. Dalmazzo	47-9606

WESTERN AUSTRALIA

ALBANY	Miss B. Balfour		
BUNBURY	Miss B. Balfour		
FREMANTLE			
Private Practice	Miss B. Balfour, *26 Queen St., 6160*		35-6048
Hospital	Fremantle Hospital, *Alma St., 6160*	Miss P. Bulford	35-2141
PERTH			
Private Practice	Miss B. Balfour, *48 Hilda St., Shenton Park, 6008*		81-2980
	Miss P. Bulford, *35 Richardson St.,* *West Perth, 6005*		21-4650
	Miss M. Lewis, *254 St. Georges Terrace,* *Perth, 6000*		22-3454

WESTERN AUSTRALIA

Hospitals			
	Princess Margaret Hospital, *Thomas St., Subiaco, 6008*	Miss Balfour Miss Bulford Miss Lewis	81-3222
	Royal Perth Hospital, *Wellington St., 6001*	Miss Lewis	33-0111

VICTORIA

BALLARAT			
Sponsored Practice	Dr. M.H. Brennan, *16 Errol St., North Ballarat, 3350*	Mrs. J. Brownfield	
Hospital	Ballarat & District Hospital, *Sturt St., 3350*	Miss S. Blake-Lane	
BENTLEIGH			
Sponsored Practice	Dr. J. Foster, *8 Bent St., 3204*	Mrs. B. Williams	97-5867
BOX HILL			
Sponsored Practices	Dr. P. Hardy-Smith, *50 Nelson Rd., 3128*	Mrs. K. Edwards	897-9166
	Dr. J. Murphy, *601A Station St., 3128*	Miss B. Richardson	89-8526
BRIGHTON			
Sponsored Practice	Dr. R. Lindsay Jones *175 Bay St., 3186*	Mrs. E. Cornell	96-5556
CAMBERWELL			
Sponsored Practice	Drs. E.N. & P.A. Rosen, *687 Burke Rd., 3124*	Mrs. V. Gordon	85-2525
DANDENONG			
Sponsored Practice	Drs. J. Stewart & D. O'Connor, *229 Thomas St., 3175*	Miss B. Richardson	792-2125

VICTORIA

DONCASTER

Sponsored Practice	Dr. M. Burkett, *619 Doncaster Rd., 3108*	Mrs. T. Peberdy	848-4747

FITZROY

Sponsored Practice	Dr. G. Serpell, *181 Victoria Pde., 3065*	Miss L. Maclarn	419-3393
Hospital	St. Vincents Hospital, *Victoria Pde., 3065*	Miss M. Carter Mrs. P. Wister	41-0221

FOOTSCRAY

Sponsored Practice	Dr. E. Finkelstein & B.L. Wilberham, *34 Paisley St., 3011*	Miss M. Carter	68-1959
Hospital	Footscray West General Hospital, *Eleanor St., 3011*	Miss M. Carter	317-0211

FRANKSTON

Sponsored Practice	Drs. Goldman, Shuter & Donovan, *42 Beach St., 3199*	Miss W. Brown Mrs. B. Williams	783-5245

GEELONG

Sponsored Practices	Dr. J. Bishop, *10 Wallace Ave., 3220*	Miss S. Royston	
	Dr. M. Morton, *68 Bellerine St., 3220*	Mrs. P. Towell	9-9399
	Dr. J. Robinson *Ryrie St., 3220*	Miss S. Royston	
Hospital	Geelong & District Hospital, *Ryrie St., 3220*	Miss S. Royston	

GLEN WAVERLEY

Sponsored Practice	Dr. P.L. Wong, *272 Springvale Rd., 3150*	Mrs. V. Gordon	560-6818

VICTORIA

HAWTHORN

Sponsored Practice

	Drs. J. McBride-White & G. Morlet, *Barkers Rd., 3122*	Miss J. O'Brien	81-8700

HEIDELBERG

Hospital

	Austin Hospital, *Studley Rd., 3084*	Mrs. P. Towell	45-0411

HORSHAM

Sponsored Practice

	Dr. Mangan, *Lister House, 3400*	Miss B. Richardson	

MELBOURNE

Private Practices

	Mrs. J. Craig, *14 Collins St., 3000*		63-1744
	Mrs. V. Gordon, *127 Collins St., 3000*		63-2639

Sponsored Practices

	Dr. P. Cowen, *2 Collins St., 3000*	Mrs. B. Williams	63-5184
	Professor G.W. Crock, *148 Victoria Pde., East Melbourne, 3002*	Mrs. E. Cornell	662-1644
	Dr. W.E. Gillies, *110 Collins St., 3000*	Miss J. O'Brien	63-5869
	Dr. G. Harley, *15 Collins St., 3000*	Miss J. O'Brien	63-4829
	Dr. E. Ryan, *4 Collins St., 3000*	Miss J. O'Brien	63-5394
	Dr. H. Ryan, *100 Collins St., 3000*	Miss S. Blake-Lane	63-5706

VICTORIA

MELBOURNE

Hospitals

	Prince Henry's Hospital, *St. Kilda Rd., 3000*	Miss L. Maclarn, Miss J. O'Brien, Mrs. P. Wister	62-0621
	Queen Victoria Hospital, *172 Lonsdale St., 3000*	Mrs. V. Gordon	66-6046
	Royal Victorian Eye & Ear Hospital, *126 Victoria Pde., East Melbourne, 3002*	Mrs. Cornell, Craig, McIndoe, McNamara, Strentz, Theile, Towell, Williams & Wister, Misses Ball, Blake-Lane, Byrt, Carter, Maclarn, Mahoney, Maloney, Nevill, O'Brien, Richardson & Royston	662-2000

MORWELL

Sponsored Practice

	Drs. Bonner & Morgan, *6 Church St., 3840*	Miss B. Richardson	

PARKVILLE

Sponsored Practice

	Dr. F. Billson, *159 Flemington Rd., 3052*	Mrs. J, Kristic	30-1285

Hospital

	Royal Children's Hospital, *Flemington Rd., 3052*	Misses M. Gyaw, Heinz, Kristic	34-0288

PRAHRAN

Hospital

	Alfred Hospital, *Commercial Rd., 3181*	Mrs. L. Collins	51-0281

PRESTON

Hospital

	Preston & Northcote Community Hospital, *265 Bell St., 3072*	Mrs. B. Williams	44-0241

RINGWOOD

Sponsored Practices

	Dr. J. Mitchell, *21 Ringwood St., 3134*	Mrs. G. Burgess	870-5995
	Dr. G.N. Vaughan, *43 Ringwood St., 3134*	Mrs. B. Williams	870-8621

VICTORIA

SHEPPARTON

Sponsored Practice

	Dr. M. Roche	Miss J. Moloney	

WANGARATTA

Sponsored Practice

	Dr. G. Barty, *Ely St., 3677*	Mrs. M. Theile	

WARNAMBOOL

Sponsored Practice

	Dr. G. Jones, *Liebig St., 3280*	Mrs. P. Towell	

NEW SOUTH WALES

ALBURY

Sponsored Practice

	Drs. Goddard & Howsam, *541 Kiewa St., 2640*		

ARMIDALE

Private Practice	Mrs. L. Brent *123 Rusden St., 2350*		3655
Hospital	Armidale & New England Hospital	Mrs. L. Brent	

BALMAIN

Hospital	Balmain & District Hospital, *Booth St., 2041*	Mrs. E. Duncan	82-0501

BANKSTOWN

Private Practice	Mrs. A. Macfarlane, *17 Kitchener Pde, 2200*		709-1910
Hospital	Bankstown & District Hospital, *17 Kitchener Pde., 2200*	Mrs. A. Macfarlane	70-0444

BATHURST

Sponsored Practice	Dr. T.D. Leckie, *101 William St., 2795*	Mrs. J. Alexander	

NEW SOUTH WALES

BONDI JUNCTION			
Private Practice	Miss G. McCormack, *253 Oxford St., 2022*		389-0488
BOWRAL			
Private Practice	Mrs. A. Mikus, *Bowral County Council Chambers,* *Bong Bong St., 2576*		
BROKEN HILL			
Hospital	Broken Hill & District Hospital, 2880	Miss M. Peoples	
BURWOOD			
Private Practice	Mrs. A. Macfarlane, *13 Belmore St., 2134*		747-2518
CAMPERDOWN			
Hospitals	Royal Alexandra Hospital for Children, *Bridge Rd., 2050*	Miss S. Innes Miss A. Knoblanche	51-0466 Ext.460
	Royal Prince Alfred Hospital, *Missenden Rd., 2050*	Miss M. Eyres	51-0444
CAMPBELLTOWN			
Private Practice	Miss S. Innes, *258 Queen St., 2560*		2-2422
CARINGBAH			
Private Practice	Miss J. Cumines, *Room 11, Caringbah Chambers,* *Cnr. Port Hacking Rd. & Mansfield Ave., 2229*		525-5190
CHATSWOOD			
Private Practice	Mrs. T. Duggan, *Railway Chambers,* *Help St., 2067*		412-2130
CROWS NEST			
Private Practice	Mrs. J. Kunst, *20 Burlington St., 2065*		43-0230 771-3333

NEW SOUTH WALES

CROWS NEST			
Hospital	Mater Misericordiae Hospital, *Pacific Highway, 2065*	Mrs. B. Macpherson	929-7022
CROYDON			
Hospital	Western Suburbs Hospital, *Liverpool Rd., 2132*	Mrs. A. Macfarlane	747-5311
DARLINGHURST			
Hospital	St. Vincents Hospital, *Victoria St., 2010*	Mrs. B. McDougall Miss K. McKern	31-0411
DEE WHY			
Private Practice	Misses P.M. Lance & H.R. Hawkeswood, *14/1 Redman Rd., 2099*		98-9503
DOUBLE BAY			
Private Practice	Mrs. A. Rona, *83 Manning Rd., 2028*		36-7086
DUBBO			
Sponsored Practice	Dr. M. Hargrave, *Macquarie Chambers, Macquarie St., 2830*	Miss F. Horton	
EPPING			
Private Practice	Mrs. S. Stanley, *41 Rawson St., 2121*		86-5855
FAIRFIELD			
Sponsored Practice	Dr. T. Keldoulis, *87 Ware St., 2165*	Mrs. D. Dixon	72-4811
GOSFORD			
Private Practice	Miss A. Philpotts, *William Court, William St., 2250*		

NEW SOUTH WALES

HORNSBY			
Private Practice	Mrs. N. Jolly, *32 Florence St., 2077*	Locum: Miss D. McCorquodale	47-2494
Hospital	Hornsby & District Hospital, *Palmerston Rd., 2077*	Miss D. McCorquodale	476-3622
HURSTVILLE			
Private Practice	Miss C. Muir, *9/346 Forest Rd., 2220*		57-1203 451-5977
LEWISHAM			
Hospital	Lewisham Hospital, *West St., 2049*	Miss M. Eyres	560-8666
LISMORE			
Sponsored Practice	Drs. Kerkenezov & Tester, *96 Molesworth St., 2480*	Mrs. R. Whitney	21-2215
LIVERPOOL			
Sponsored Practice	Drs. T. Keldoulis & E. Gregory, *17 Moore St., 2170*	Mrs. P. Erikson	602-8066
Hospital	Liverpool Hospital, *Elizabeth St., 2170*		602-6988
MANLY			
Sponsored Practice	Dr. Y. Barnard, *2 Wentworth St., 2095*	Miss B. Paul Miss B. Marshall	97-5728
Hospital	Royal Far West Children's Health Scheme, *18 Wentworth St., 2095*	Mrs. A. Mikus	977-4377
MARRICKVILLE			
Sponsored Practice	Dr. M. Sterling-Levis, *324 Marrickville Rd., 2204*	Miss B. Paul	560-8589

NEW SOUTH WALES

NEWCASTLE

Private Practice	Mrs. P. Dunlop, *17 Bolton St., 2300*		2-3535
Sponsored Practices	Drs. G. Stuckey & C. Joneshart, *CML Building, Hunter St., 2300*	Miss P. Monteath	
	Drs. C. Walter & W. Porter, *16 Union St., 2300*	Miss P. Monteath	
Hospital	Royal Newcastle Hospital, *2300*	Miss P. Monteath	

PARRAMATTA

Private Practice	Miss S. Innes, *57 Macquarie St., 2150*		635-3460
Sponsored Practices	Drs. Moxham & Cumming, *20 Macquarie St., 2150*	Mrs. J. Williams	635-8509
	Drs. Hogg, Darvall, Morrissey, Henry, Hopkins, du Temple, & Thomson *152 Marsden St., 2150*	Mrs. Dixon	635-7795 635-7077
Hospital	Parramatta District Hospital, *Marsden St., 2150*		635-0333

PENRITH

Sponsored Practice	Drs. Cooper & Chatfield, *314 High St., 2750*	Mrs. J. Wilson	21-3110

RANDWICK

Private Practice	Miss A.M. Dwyer, *Medical Centre, 66 High St., 2031*		39-3921
Hospital	Prince of Wales Hospital, *High St., 2031*	Miss J. Cumines Miss B. Marshall Miss B. Paul	399-0111

NEW SOUTH WALES

REDFERN

Hospital

	Rachel Forster Hospital, *Pitt St., 2016*	Miss C. Muir	69-2591

RYDE

Private Practice

	Miss E. Sullivan, *6 Smith St., 2112*		807-1532 807-3939

ST. LEONARDS

Private Practice

	Miss M. Hall, *North Shore Medical Centre, 66 Pacific Highway, 2065*	Locums: Mrs. P. Erikson, & Mrs. B. McDougall	43-4614

Hospital

	The Royal North Shore Hospital, *Pacific Highway, 2065*	Miss J.Russell Miss A. Herlihy	43-0411 Ext. 2589

SYDNEY

Private Practices

	Miss P. Lance & H.R. Hawkeswood, *106/67 Castlereagh St., 2000*		28-6775
	Mrs. A. Rona, *195 Macquarie St., 2000*		211-1714

Sponsored Practice

	Drs. Price, Cumming & Moxham, *235 Macquarie St., 2000*	Miss C. Muir	28-1390

Hospitals

	Medical Eye Service, *27 Commonwealth St., 2000*	Miss A. Philpotts Miss K. McKern	61-6534
	Sydney Eye Hospital, *Sir John Young Cres., Potts Point, 2011*	Mrs. Kunst, Doyle, Merrick, Rivers, Stanley, Misses Brown & J. Russell	2-0311 Ext.2217

TAMWORTH

Sponsored Practice

	Drs. Baker, Barnett & Wilson, *Bank of NSW Chambers, 2340*	Miss J. Henning	

NEW SOUTH WALES

WAGGA WAGGA

Sponsored Practice

	Drs. D. Thornton, G. Nixon, & M. Halley *1 Trail St., 2650*	Miss A.M. Mahoney	

WOLLONGONG

Sponsored Practices

	Drs. Brown, O'Connor & Higgins, *82 West Market St., 2500*	Mrs. J. Armstrong	2-1086
	Dr. Scott, *68 Market St., 2500*	Mrs. P. Toogood	2-1708