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Australian Orthoptic Journal

AUSTRALIAN ORTHOPTIC JOURNAL – 2010 VOLUME 42, NUMBER 1

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2010 Volume 42 (1)

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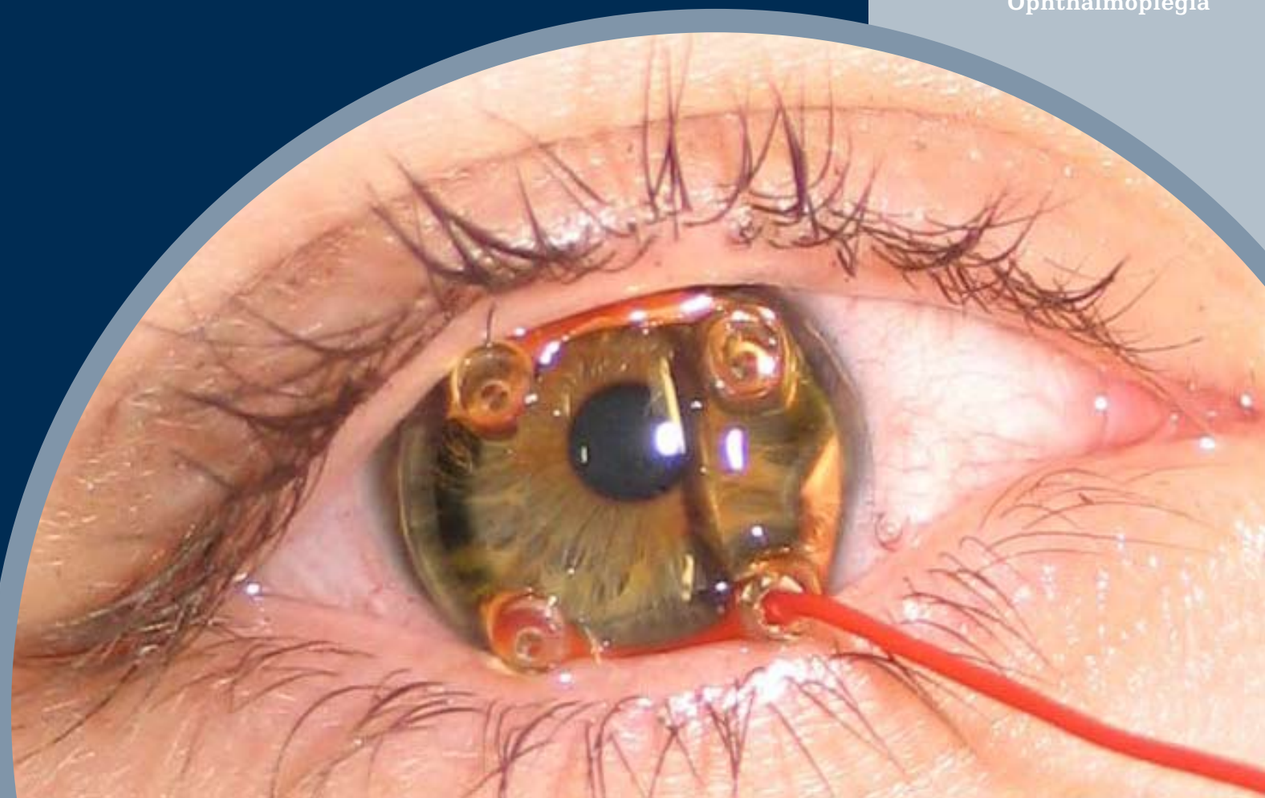

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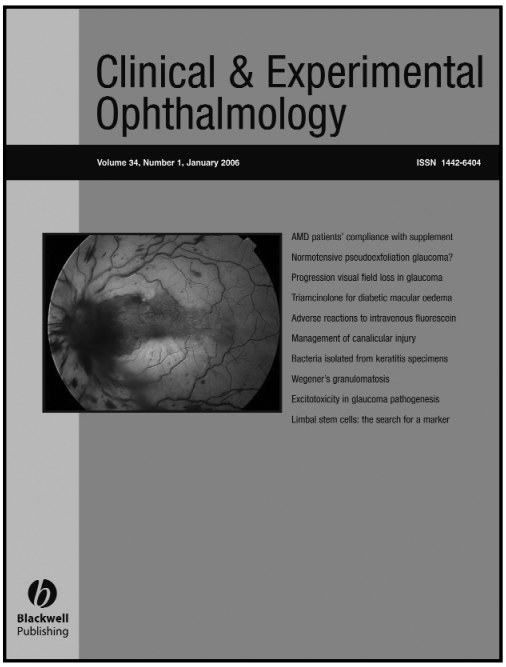

Edited by: Charles N.J. McGhee
Print ISSN: 1442-6404
Online ISSN: 1442-9071
Frequency: 9 issues per year
ISI Journal Citation Reports® Ranking: 2005: 24/44 (Ophthalmology)
Impact Factor: 1.193

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Australian
Orthoptic Journal
2010 Volume 42 (1)

The official journal of Orthoptics Australia
ISSN 0814-0936

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Published by Orthoptics Australia (Publication date: July 2010).

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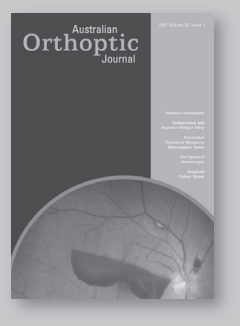

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The types of manuscripts accepted are as follows:

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MANUSCRIPT SUBMISSION

Submitted manuscripts must include a cover letter, title page, abstract (including keywords), the paper itself, any acknowledgements, references and tables and/or figures. Each of these sections should begin on a separate page. Pages should be sequentially numbered. The manuscript submission should be electronic, via email to: AOJ@orthoptics.org.au

Cover Letter: The cover letter must include information regarding ethical considerations, informed consent and potential conflicts of interest , in addition to the statement regarding the originality of the manuscript.

Ethical Considerations: Authors must state that the protocol for any research project has been approved by an appropriate Ethics Committee that conforms to the provisions of the Declaration of Helsinki in 1995 (as revised in Edinburgh 2000). Investigators who do not have a formal ethics review committee must indicate they have adhered to the aforementioned provisions.

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Abstract and Keywords: The abstract should not exceed 250 words. It should be a clear and succinct summary of the paper presented and need not be structured into subsections. However, where appropriate, it should relate to the format of the paper, including aim, methods, results and conclusion. Beneath the abstract, include up to five keywords or terms suitable for use in an index or search engine.

Text: Manuscripts should not exceed 3,000 words. Where appropriate the structure of the text should be as follows: Introduction, Method, Results, Discussion and Conclusion. For scientific research the methods section of the manuscript should also address ethical considerations and informed consent. Authors should also use subheadings for Case Studies, generally as follows: Introduction, Case Report and Discussion (Conclusion is optional). Case Studies should not exceed 1,500 words.

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Book: Kline LB, Bajandas FJ. Neuro-ophthalmology: Review Manual. 5th Ed. Thorofare: Slack Inc; 2004.

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Acknowledgements: Identify all sources of financial support including grants or sponsorship from agencies or companies. Include any acknowledgements to individuals who do not qualify for authorship.

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Australian
Orthoptic
Journal

2010 Volume 42 (1)

orthoptics australia

CONTENTS

Editorial

Orthoptic Practice Variations and Effective Care: The Need for Clinical Practice Guidelines to Improve Care

In 1981 50% of the award for the Nobel Prize for Physiology or Medicine was given to David Hubel and Torsten Wiesel for the discovery of the pathophysiology of amblyopia[1] and thus marked a turning point in the management of children with this condition. Recognition that early visual experience is essential for the development of the visual brain has fundamentally changed the way we manage disorders that interfere with image formation in the eye during early life. However, since that time amblyopia treatment history has been littered with abandoned methods such as the Cam vision stimulator, red filter treatment and pleoptics and has seen a variety of regimes that include occlusion of a few minutes a day to all waking hours of the sound eye, Bangerter foils of different densities, use of atropine ranging from daily to exclusive weekend only instillation, contact lenses and spectacles combined with occlusion or as a period of exclusive treatment and refractive surgery.[2] There is also evidence that there is a lack of adherence to standardised amblyopia treatment regimes and practice differences between centres and countries exist.[3-7] The results of these studies highlight the lack of standardisation in the treatment of the various types of amblyopia in apparently similar eye care communities. Patients with amblyopia receive different treatment depending on their clinician, hospital or location. While variations in amblyopia treatment practice are well documented, there has been less progress in explaining these variations.

The diagnosis, management, and treatment of amblyopia in clinical practice is ideally guided by evidence accrued from high-quality clinical trials, cohort studies, epidemiological studies, observational data, and a consensus of clinical experience. Recommendations are proffered in many guidelines from the continents of the world. Patients benefit from adherence to clinical practice guidelines[8] and appropriate treatment. The expectation would be that the practice of amblyopia treatment would be similar, or almost so, in all parts of the world. Any differences, which exist in amblyopia treatment, would be accounted for by unique clinical features of this disorder in different parts of the world. If that were so, and it is not,[9] then outcomes measured as mortality, morbidity, treatment procedures and regimes would be universally similar, and measurement of those outcomes would provide an indicator of performance, which would have validity within regions of a particular country, between countries, and between continents. What nirvana that would be for providers of health care. But the reality is otherwise.

Too often orthoptic practice has had only limited success in improving the scientific basis of everyday clinical practice. Patterns of practice among eye care teams are often idiosyncratic and unscientific, and local medical opinion and parental opinion are more important than science in determining how care is delivered. Few practices have written guidance for occlusion treatment.[7] While occlusion therapy is widely accepted as the first choice treatment of amblyopia[6,10] there are clinician, regional, country and continent differences in the age at which treatment is started, how quickly treatment was discontinued, whether full or part-time occlusion is selected, the intensity of occlusion therapy, whether refractive correction is used alone as a treatment for anisometropic amblyopia before using occlusion therapy, and whether amblyopia patients received surgery, and if so, whether treatment is continued postsurgically.[3-6]

Clinicians, and health care policy makers see clinical practice guidelines (CPG) as a tool for making care more consistent and efficient, and for closing the gap between what clinicians do and what scientific evidence supports. The Institute of Medicine defines CPG as "systematically developed statements to assist practitioners' and patient decisions about appropriate health care for specific clinical circumstances".[11] It has been shown in rigorous evaluations that clinical practice guidelines can improve the quality of care.[8] Guidelines promote interventions of proved benefit and discourage ineffective ones while making it more likely that patients will be cared for in the same manner regardless of where or by whom they are treated.

CPG can improve the quality of clinical decisions. CPG based on critical appraisal of the literature offer explicit recommendations for clinicians who are uncertain about how to proceed, overturn the beliefs of outdated practices, improve the consistency of care, and provide authoritative recommendations that reassure practitioners about the appropriateness of their treatment policies. They alert clinicians to interventions unsupported by good science, reinforce the importance and methods of critical appraisal. The methods of guideline development that emphasise systematic reviews focus attention on key research questions that must be answered to establish the effectiveness of an

intervention which benefit researchers by drawing attention to gaps in evidence.

CPG can support quality improvement activities. The first step in designing quality assessment tools (standing orders, critical care pathways, algorithms, audits, etc.) is to reach agreement on how patients should be treated.

For orthoptists, there is a need to determine whether actual amblyopia treatment approaches the established standard of care, if it exists at all. Establishing CPG and validating uniform standards across the world, so that clinical outcomes in amblyopia treatment can meaningfully be compared, may take many years. The challenge is daunting but necessary; the need is timely.

Karen McMain

Dalhousie University, Halifax, Nova Scotia, Canada

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Electroretinography in a Paediatric Setting: A Useful Diagnostic Tool

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ABSTRACT

Assessing visual behaviour in young children is a challenging task. When children present with poor vision, nystagmus, photophobia or nyctalopia, it can be difficult to determine the cause. The electroretinogram (ERG) plays an important role in the diagnosis and management of paediatric retinal eye conditions and can be a useful diagnostic tool for the paediatric ophthalmologist. The ERG records electrical activity of the retina in response to ocular stimulation with either a light or pattern source.

Patients are referred to the visual electrophysiology clinic when a diagnosis is uncertain or when the ERG result will help confirm a diagnosis. When a diagnosis is confirmed the ERG can be used to monitor progression of the disease. These results, along with genetic counselling, allow patients and their families to be informed on prognosis and progression of retinal disease and its impact on vision.

A retrospective review of patients attending The Children's Hospital at Westmead for ERG assessment over a two-year period from 2007 to 2009 was carried out. This paper discusses methods of paediatric ERG assessment, indications for testing and common paediatric retinal dystrophies.

Keywords: electroretinogram, paediatrics, retinal dystrophy

INTRODUCTION

The electroretinogram (ERG) is utilised as part of a group of tests which assess visual and retinal function. These tests include visual acuity, colour vision, contrast sensitivity, visual fields, fundoscopy and other electrodiagnostic testing. The ERG records electrical activity from the retina in response to ocular stimulation via either a light or pattern source. It is used to investigate rod and cone photoreceptor retinal function as well as inner and outer retinal function.

Paediatric ophthalmological investigation is hindered in young infants and children by the limited number of objective diagnostic tests available, and the patient's inability to communicate symptoms and subjective visual responses. Fundoscopy examination in young infants is not always conclusive and may reveal a normal-looking retina initially, even in cases of severe retinal dystrophy.[1] The ERG is a useful tool in the paediatric population as it is objective, and although it does require some co-operation from the patient to enable adequate positioning of both the patient and the electrodes during the test, it requires minimal participation and interaction throughout the test.

The role of the ERG in paediatric ophthalmology is crucial in the diagnosis and management of paediatric retinal eye conditions. The benefit of the ERG in providing a diagnosis should not be underestimated as it can impact the patient's visual rehabilitation with low vision training and support from low vision services, schooling choices and future employment possibilities.

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TYPES OF ERG ASSESSMENT

There are three types of ERG assessment. These are the full field, pattern and multifocal ERG. The full field ERG (ffERG) is used to assess the retina with light stimulation. It investigates rod and cone photoreceptor function and inner and outer retinal function. It requires minimal patient interaction and can be assessed while the patient is asleep or under sedation. It is recorded in a minimum of five stages in scotopic and photopic conditions to isolate rod, mixed rod and cone, and cone stimulation in the retina. It is useful in diagnosis of retinal dystrophies such as retinitis pigmentosa, Leber's congenital amaurosis, congenital stationary night blindness and cone dystrophies.

The ffERG waveform is comprised of a series of peaks and troughs known as the a, b, c and d waves. It is analysed by the amplitude and timing of the first initial negative trough – the a wave, and the subsequent positive peak – the b wave (Figure 1). The a wave originates from the photoreceptor layer of the retina, the rods and cones, while the b wave originates from the Muller and bipolar cells. Differences in the electrical potential caused by hyperpolarisation of the apical membrane of the retinal pigment epithelium (RPE) and hyperpolarisation of the distal end processes of the Muller cells result in the c wave. This is a slow positive wave that follows the b wave but is not always identifiable. The d wave is a positive response that occurs after the b wave when the retinal illumination is turned off in the light adapted eye. It is produced by the interactions of the on (depolarising) and off (hyperpolarising) bipolar cells.

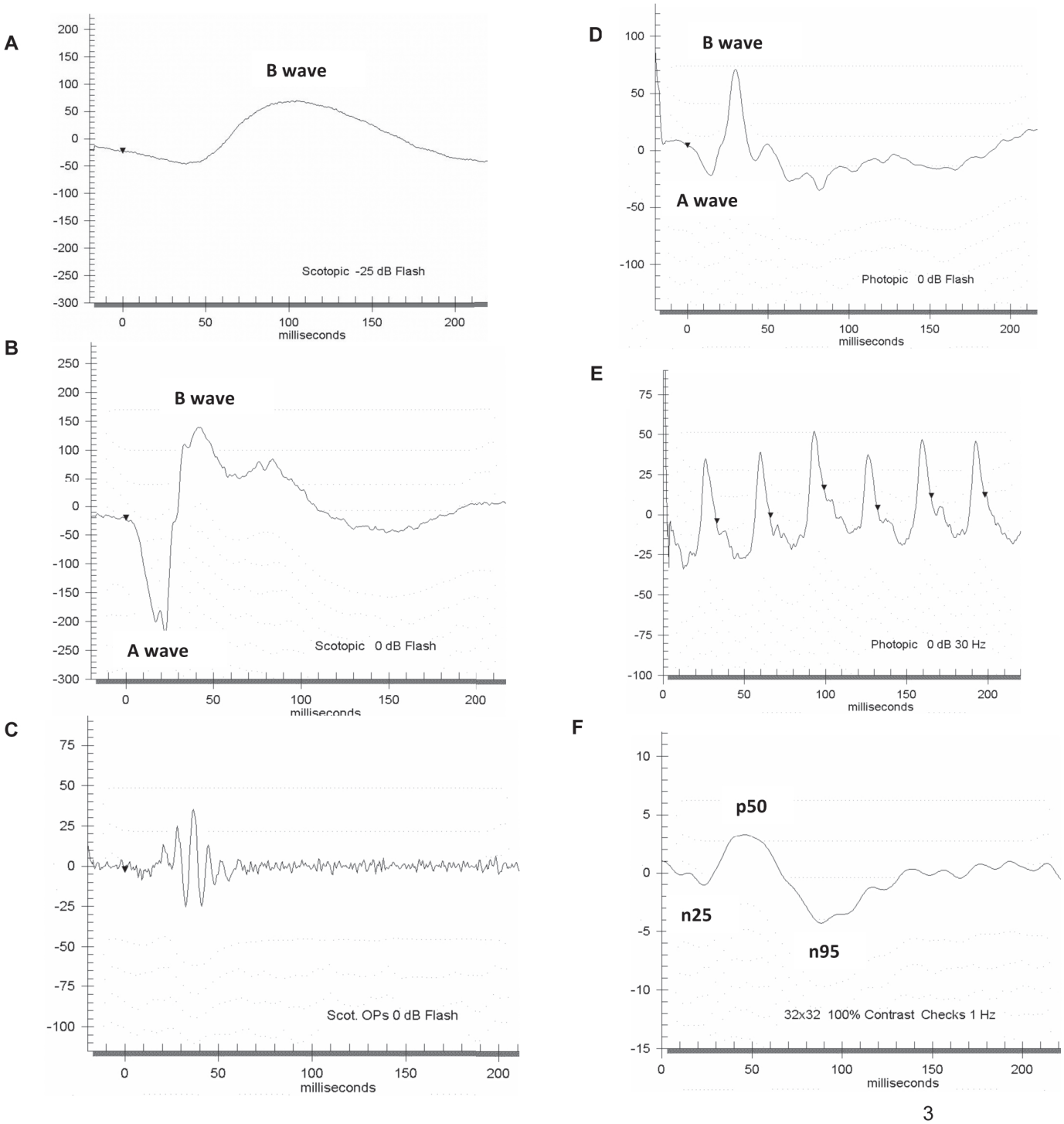

Figure 1. A normal full field ERG recording and pattern ERG recording. (A) scotopic rod response, (B) scotopic combined rod cone response, (C) scotopic oscillary potentials, (D) photopic cone response, (E) photopic cone flicker response, (F) pattern ERG.

The pattern ERG (pERG) assesses ganglion cell function and is used to investigate macular function and maculopathies. It is recorded to pattern stimulation and is often performed in conjunction with a visual evoked potential (VEP) to differentiate between optic neuropathies and macular pathway dysfunction. It requires co-operation and steady fixation from the patient and therefore the patient can not be sedated during the test.

The pERG waveform is comprised of a negative trough at approximately 25 milliseconds, a positive peak at approximately 50 milliseconds and another trough at approximately 95 milliseconds (Figure 1).

The multifocal ERG (mfERG) is used to assess localised retinal lesions within the central 20 to 30 degrees of retina and is recorded with pattern stimulation. It is a cone initiated response and requires co-operation and central steady fixation from the patient and like the pERG the patient can not be sedated for the test.

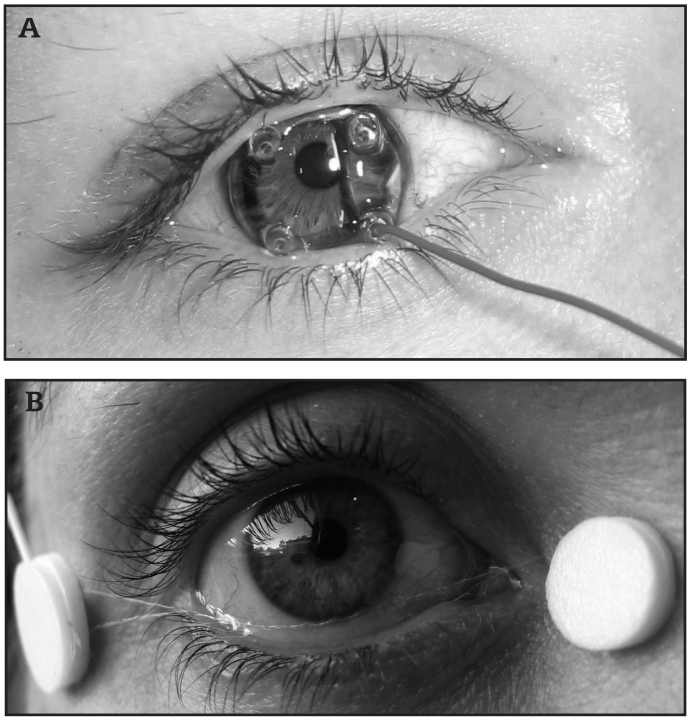

Figure 2. Two types of ocular contact electrodes (A) ERG Jet contact lens electrode, (B) DTL.

ELECTRODES

There are different types of electrodes that can be used to record the ERG. Ocular contact electrodes record from the cornea or the conjunctiva. Corneal recording electrodes come in the form of a contact lens with or without a lid speculum – Burian Allen and ERG Jet respectively. Gold foil and DTL thread electrodes record from the conjunctiva (Figure 2). Skin electrodes are attached to the skin surrounding the eye (Figure 3).

The ERG result will differ in scale amplitude depending on the type of electrode used and the proximity of the electrode to the cornea. Skin electrodes record the lowest amplitude and Burian Allen electrodes record the highest. In comparison with a Burian Allen electrode the amplitude will be reduced to 89% when recorded with an ERG Jet electrode, 56% when recorded with a gold foil electrode, 47% when recorded with a DTL electrode and 12% when recorded with a skin electrode.[2] The waveform morphology when recorded with skin electrodes is similar to corneal contact electrodes, and after scaling responses, amplitudes are similar also.[3] Skin electrodes have been proven to be an effective and reliable, non-invasive technique of recording the ERG in the paediatric population.[4,5]

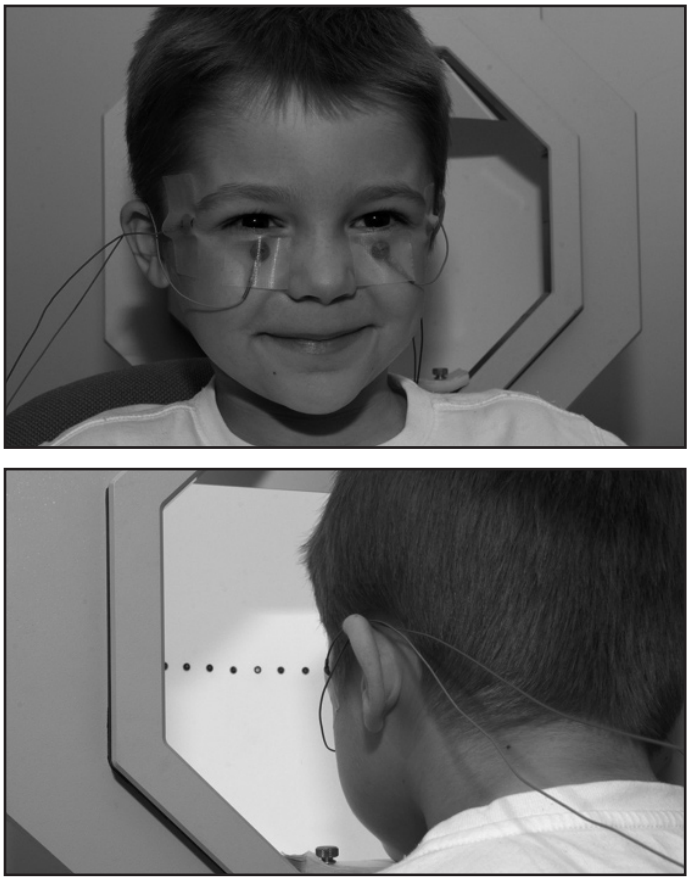

Figure 3. Child with skin electrodes at the Ganzfeld bowl.

TESTING PROTOCOLS

The International Society for Clinical Electrophysiology of Vision (ISCEV) is an international body that establishes standard protocols for all visual electrophysiology testing. This includes the ERG as well as the VEP and electrooculogram (EOG). These international guidelines enable comparison of data amongst different recording centres and different recording equipment. The current ISCEV protocol for recording of the full field ERG includes

the following responses, named according to conditions of adaptation and the stimulus (flash strength in $cd \cdot s \cdot m^{-2}$)[6]

1. Dark-adapted 0.01 ERG (Rod ERG)
2. Dark-adapted 3.0 ERG (Standard combined ERG)
3. Dark-adapted 3.0 (Oscillatory Potential ERG)
4. Light-adapted 3.0 ERG (Cone ERG)
5. Light-adapted 3.0 Flicker ERG (30Hz Flicker ERG).

ISCEV recommends a minimum of 20 minutes dark adaptation with maximal mydriasis prior to scotopic rod testing and a minimum of 10 minutes light adaptation prior to photopic cone testing.

PAEDIATRIC ERG ASSESSMENT

Recording of the ERG in a paediatric population can prove to be more difficult than in an adult population. This is due to limited co-operation and compliance from paediatric patients. Testing protocols are often adapted to combat these restrictions.

A paediatric ERG assessment will often be of longer duration, and will require interaction and skill from the technician recording the test. The testing environment may need to be adapted to allow it to appear less threatening and enable better co-operation from the child. Due to these challenges paediatric ERG assessments are a highly specialised area of electrophysiology with many centres not performing paediatric assessments on a regular basis.

In a recent survey of seventy-one visual electrophysiology centres worldwide, it was found that only 13 (21%) of the centres performed a high volume (more than ten patients per month) of paediatric ERG assessments in infants and young children less than 6 years of age, and only seven (11%) centres performed a high volume of ERG assessments on patients less than 12 months of age. Eighty-seven percent of respondents indicated that they rarely or never used sedation or anaesthesia. Twenty-nine percent of respondents used skin electrodes and 88% used ocular contact electrodes.[7]

MATERIALS

The Eye Clinic at The Children's Hospital at Westmead (CHW) provides a visual electrophysiology service where ERG, along with other visual electrophysiology tests such as VEP and EOG are performed. These tests are recorded either in the clinic or in operating theatres under sedation with a general anaesthetic. Patients are referred to the visual electrophysiology clinic when a diagnosis is being investigated, subnormal visual responses can not be explained, or if a patient with a known retinal dystrophy is being monitored for progression of the disease. All patients are referred from an ophthalmologist or paediatric consultant.

The clinic services paediatric patients aged from birth to 18 years. Rarely an adult assessment will be undertaken. Testing of an adult will only occur during a genetic investigation in conjunction with the genetic eye clinic at CHW, or if an adult patient has a developmental delay and would benefit from being tested in a paediatric environment with specialised staff.

The visual electrophysiology clinic at CHW is led by orthoptists and a consultant ophthalmologist. It benefits from the help and support of the play therapy department within the hospital. Their expertise has been vital in establishing an environment that is non-threatening to the patient. This enables better compliance and co-operation during testing and has lead to the ERG test being a more enjoyable experience for the majority of patients.

The ERG is recorded by an orthoptist and in most cases two orthoptists will be present during the test, one to operate the recording equipment and one to monitor the patient and encourage co-operation from the patient. This is achieved with toys, games and music.

Previously sedation and ocular contact electrodes were used routinely for ERG assessments at CHW. This proved difficult in many ways, being confronting for parents to observe and requiring additional nursing staff for patient observation. With new advances in technology and revised paediatric protocols including the use of skin electrodes and play therapy advice, sedation is now rarely undertaken and is never undertaken within the clinic. All attempts are made to have the ERG performed in the clinic. If this proves too distressing for the patient, or there are other complicating factors such as systemic disease or developmental delay the ERG may be performed under general anaesthetic administered by a paediatric anaesthetist in the operating theatres at CHW. Often the consultant ophthalmologist will be present to perform an examination under anaesthetic after the ERG is completed.

METHOD

The medical records of patients who underwent ERG testing either in the Eye Clinic or in operating theatres under a general anaesthetic, between January 2007 and January 2009 were retrospectively reviewed.

ISCEV standards were followed where possible. If a patient was unco-operative a shorter period of dark adaptation was used. All patients underwent a full orthoptic assessment prior to the ERG, including visual acuity, cover test, ocular motility, and if achievable colour vision, contrast sensitivity, visual fields and fundus photos.

The parents or guardians of the patient were present for the duration of the test. The Ganzfeld bowl light source was

always attempted initially. Younger children sat on their parent's lap and older children sat by themselves. Infants were swaddled and held into the bowl, lying in their parents arms. If recording was unnoticeable with the Ganzfeld, a hand held Kurbisfeld light source was used. The duration of the consultation lasted on average 60 minutes.

The type of electrode used for the ERG was determined by the age and co-operation of the child. Young children or older children who were unco-operative, were tested with skin electrodes. Older children and children who had been sedated were tested with an ocular contact electrode.

RESULTS

In total there were 131 patients reviewed and a total of 139 tests performed. Ages of the patients ranged from 10 weeks to 22 years with a mean age being 6.3 years (SD ± 5.7). The two most common groups were patients older than 10 years at 27% (n=35), and those aged between 6 and 12 months 19% (n=25) (Figure 4).

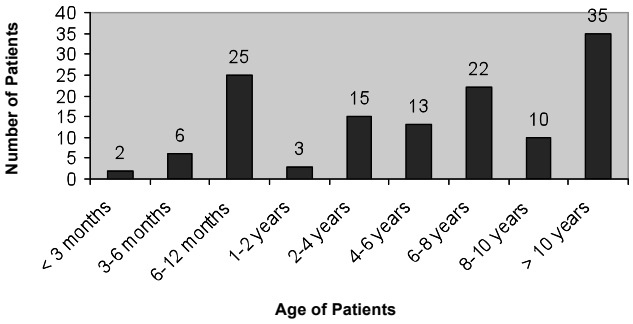

Figure 4. Distribution of ages of patients seen for ERG testing.

The most common type of ERG assessment was the ffERG. Eighty-six percent (n=119) of patients were assessed with this method. Nine percent (n=12) of these patients were assessed with a ffERG in operating theatres under sedation with a general anaesthetic. A much smaller proportion of patients were assessed with a pERG or mfERG, 5% (n=7) and 1% (n=1) respectively (Figure 5).

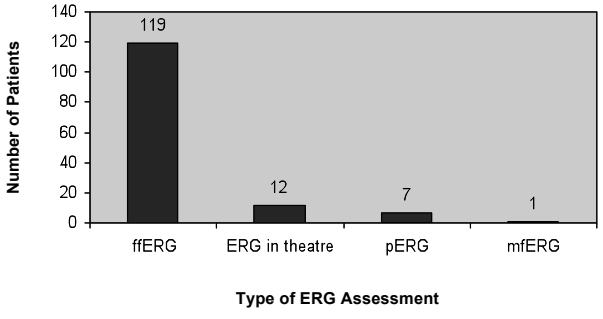

Figure 5. Distribution of types of ERG assessment performed.

The most frequent electrode used was the skin electrode. This was used in 81% (n=113) of patients, by far the majority. ERG Jet electrodes were used in 14% (n=19) of patients, and all patients who underwent ffERG assessment under general anaesthetic in operating theatres were assessed with an ERG Jet electrode. Therefore seven patients were assessed with an ERG Jet electrode in the Eye Clinic without sedation. Gold foil and DTL electrodes were used in 3% (n=4) and 2% (n=3) of patients respectively. A corneal electrode was used for all pERG and mfERG recordings (Figure 6).

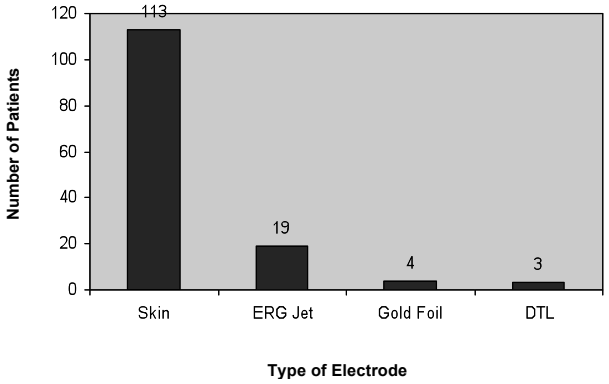

Figure 6. Distribution of types of electrodes used for ERG assessments.

All patients were referred for an ERG by an ophthalmologist. Subnormal visual acuity was the most common reason for referral 38% (n=53). This was followed by visual inattentiveness 22% (n=31), high refractive error 11% (n=15), and nystagmus 9% (n=13) (Table 1). All of these clinical features can occur with or without retinal dysfunction. If retinal dysfunction is detected alongside

Table 1. Reasons for referral

Reasons for referral	Number	Percentage
Subnormal visual acuity	53	38
Visual inattentiveness	31	22
High refractive error	15	11
Nystagmus	13	9
Nyctalopia	7	5
Maculopathy	6	4
Functional vision loss	5	4
Optic nerve disease	3	2
Retinal toxicity to medications	3	2
Photophobia	3	2

these other clinical findings, it can help to either explain the clinical features, or diagnose the patients with a disease or syndrome.

Of the 139 ERG assessments performed on 131 patients, 34% (n=45) were normal, 10% (n=13) were diagnosed with Leber's congenital amaurosis and 17% (n=22) had a dysfunction of their photoreceptors (rod, rod-cone and cone dystrophies) (Figure 7). Eight percent (n=11) of patients were found to have a functional or non-organic visual problem (Table 2). The diagnosis of a patient with a functional vision problem is a diagnosis of exclusion. As the ERG is an objective test it is an accurate method of ensuring there is no underlying retinal pathology.

Table 2. Diagnosis of patients

Diagnosis	Number	Percentage
No retinal dystrophy	45	34
Leber's congenital amaurosis	13	10
Functional	11	8
Cortical vision impairment	10	8
Cone dystrophy	10	8
Rod-cone dystrophy	10	8
Delayed visual maturation	8	6
Congenital motor nystagmus	6	5
Congenital stationary night blindness	5	4
Optic neuropathy	4	3
Maculopathy	3	2
Inconclusive	3	2
Rod monochromatism	2	2

DISCUSSION

GENETIC COUNSELLING

Retinal dystrophy is investigated by a comprehensive ophthalmological exam, electrodiagnostic testing and a thorough genetic pedigree. Electroretinography is not a tool used in isolation to provide a diagnosis for retinal dystrophies, nor does it determine the genetics of a retinal dystrophy. When a diagnosis is confirmed the ERG can be used to monitor progression of the disease. These results, along with genetic counselling, allow patients and their families to be informed on prognosis and progression of the disease and its impact on vision. This is useful for families as patients diagnosed with a retinal dystrophy benefit from low vision support services.

Prenatal diagnosis and medical genetics are 'traditional' genetic counselling roles. More comprehensive knowledge of genetic disorders has led to speciality areas developing in genetic counselling, such as cancer and ophthalmology.[8]

Patients with inherited eye disorders and their families have complex needs, which include clinical services for diagnosis and management, social and genetic counselling to help them cope with the disease. Specialist genetic eye clinics are set up to help meet these needs.[9]

CHW runs a Genetic Eye Clinic (GEC) which is held once a month. It is led by a clinical geneticist together with a consultant ophthalmologist who specialises in genetic eye disease. The team also comprises of a clinical geneticist fellow, a genetic counsellor, ophthalmology registrars and fellows, and orthoptists.

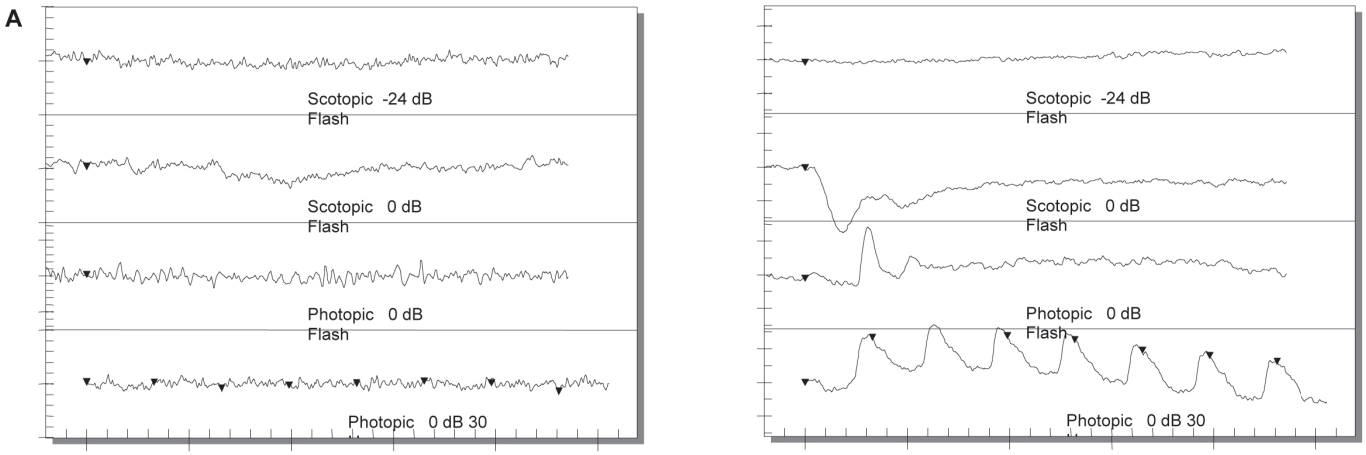

Figure 7. ERG results for (A) advanced rod-cone dystrophy and (B) congenital stationary night blindness. Note (A) shows extinguished responses for both photopic and scotopic stimuli, (B) shows an extinguished rod response on the dim -24dB flash and a negative b wave on the brighter 0dB flash with present photopic responses.

Each patient who attends the GEC along with their family may have tests performed such as visual acuity, orthoptic examination, colour vision, visual fields, fundus photography and electrophysiology. They will also have an ophthalmological examination, including cycloplegic refraction and fundoscopy performed when necessary. Genetic counselling and testing will also be carried out along with any necessary referral to other services, either internal or external to the hospital such as low vision services.

The GEC is invaluable to patients and their families in providing a comprehensive consultation regarding their genetic eye disease and will discuss in layman's terms their clinical diagnosis, family pedigree, patterns of inheritance, risk for future pregnancies as well as prognosis of vision. This enables patients and families to gain a better understanding of the implications of their inherited eye condition.

LOW VISION

Early intervention from a low vision service will better prepare patients and families with skills needed in the future. Awareness of a child's level of vision plays a vital role in the overall development of the child. For example if a child can not see, they will be less likely to learn how to reach for toys, roll to an object or understand their environment. Low vision specialists play an important role in teaching parents the necessary skills required to ensure their child continues to develop in all areas. A diagnosis aids the patient in registering for low vision services, which in turn, ensures they receive vital early intervention as soon as possible.

CONCLUSION

ERG assessment is an essential tool in diagnosing retinal dystrophies in paediatrics. Testing procedures may need to be adapted to suit the clinical environment where the test is being performed. Skin electrodes are an effective way of assessing the ERG without causing discomfort to the patient. It is possible to accurately record the ERG in the majority of paediatric patients without the use of sedation, however this is reliant on the examiner's expertise and ability. Early electrophysiology has become a vital component to the paediatric ophthalmology clinic at CHW and is utilised well by both internal and external paediatric ophthalmologists.

The ERG assists in the early diagnosis of retinal dystrophies. This is vital in the patient receiving early intervention low vision services and enables development of the child in all areas in the presence of a vision impairment.

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A Case of Diplopia Following Monovision with Contact Lenses

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ABSTRACT

A 46-year old woman presented with a 12-month history of diplopia after being prescribed monovision contact lenses. The iatrogenic anisometropia caused decompensation of an esophoria, resulting in diplopia. A normal binocular state was reinstated with glasses, but it was necessary to incorporate prisms to achieve single vision.

Keywords: diplopia, monovision, contact lens

INTRODUCTION

In the era of 'throw away your glasses', the early stages of presbyopia present a challenge for the patient and the eye care professional. The successful contact lens wearer may now be needing glasses for reading. The mid-forties patient considering refractive surgery should be advised that throwing away their distance glasses will mean wearing reading glasses. Monovision, where one eye is corrected for distance vision and the other corrected for reading vision, is becoming an increasingly popular method to overcome these problems.

However, not everyone can tolerate monovision, with limitations including the lack of an intermediate focal distance, visual discomfort caused by anisometropic blur and binocular disruption. Success rates have been reported between 59% and 67% using contact lenses in patients who have already adapted to contact lenses wear.[1,2] A Sydney-based study offered monovision with contact lenses to 1,133 presbyopes who were not already contact lens wearers. Only 28% were interested in trying monovision, and only 6.4% were actually fitted with contact lenses. Only one-third of these were interested in continuing with monovision after a one-month trial period, meaning only 2.8% (n=32) of the original participants continued with contact lens wear.[3] The success rates of surgically-induced monovision are reportedly higher, ranging from 73%[4] to 96%.[5] This could be due to the difficulty handling contact lenses, residual astigmatism or the constant optical correction of a permanent surgical procedure facilitating binocular adaptation.[5]

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The literature on success rates highlights the key issue of patient selection.[6] Certainly some occupations are not suitable for monovision. The airline pilot or professional driver should be steered away from this option due to the decrease in binocular vision and blur factor.

CASE REPORT

A 46-year old woman, Ms Y, presented to the Ocular Motility Department at the Royal Victorian Eye and Ear Hospital with diplopia for driving and television for the past 12 months. She was distressed by these symptoms and had undergone several consultations previously elsewhere.

Ms Y had no past history of strabismus or occlusion, had moderate myopia and anisometropia with a glasses prescription of -3.50DS and -5.00DS for the right and left eye respectively, and was a contact lens wearer. Monovision contact lenses had been prescribed, with the right eye used for distance and the left eye for near. Diplopia was noticed three months later.

Subjective refraction whilst wearing contact lenses showed 1.50DS of uncorrected anisometropia. Spectacle prescription was correct according to subjective refraction. No cycloplegic refraction was done. Ocular examination showed a constant left esotropia measuring 20PD for both near and distance with no diplopia in the clinical setting. Ocular movements were full indicating no paretic or restrictive element and, with her correct spectacle prescription, vision was 6/5 in each eye. At this point the differential diagnosis was between a childhood esotropia which had increased in size and moved out of a suppression scotoma, a decompensated esophoria, and an acquired esotropia which had occurred during the period of monovision wear.

Magnetic resonance imaging was normal, finding no suggestion of a recent onset deviation. Further orthoptic investigation showed normal binocular functions on the Synoptophore, with normal retinal correspondence and a negative fusional amplitude of two degrees and a positive fusional amplitude of seven degrees, giving a fusion range of nine degrees. Sbisa bar gave diplopia from filter 2, showing a shallow suppression scotoma and, when the deviation was fully corrected with prisms, Worth Lights gave a binocular response. A diagnosis of decompensated esophoria was made. Presumably the iatrogenic anisometropia created by the monovision contact lenses had disrupted fusion and precipitated an esotropia.

Initial treatment was to stop monovision contact lenses and Ms Y resumed wearing her multifocal glasses. Her symptoms improved initially but diplopia was still very bothersome. A program of prism therapy was instigated. With a 20PD base-out Fresnel prism on the left lens a binocular response was achieved with Worth Lights. Any less prism showed left suppression. This prism was fitted and one month later the patient was symptom-free with glasses, but still diplopic without the prism. Over the next 10 months attempts were made to wean off the prism. As Ms Y was still suppressing in the clinical setting, binocularity could only be assessed using Worth Lights. Prisms were gradually reduced and a regime of physiological diplopia and stereogram exercises began. Prisms were reduced through 15PD, 12PD to 10PD, beyond which a binocular response on Worth Lights was not achieved. The patient continued to suppress in the clinical setting, the angle of deviation always remained the same and diplopia persisted without the prism in daily life.

Despite improving negative relative fusion to some degree, there remained a small, symptomatic manifest esotropia. Surgery was an option that Ms Y declined. She was very happy to be diplopia-free and finally the 10PD prism (5PD base-out each eye), was incorporated into her glasses. However, this outcome means contact lenses are no longer an option and Ms Y will need to permanently wear glasses with a prism.

DISCUSSION

It is unusual for contact lens monovision to precipitate an esotropia and diplopia. An extensive literature review on monovision by Evans[6] found no cases of diplopia following monovision with contact lenses in patients without pre-existing strabismus. Only one paper presented three cases of fixation switch diplopia precipitated by monovision contact lenses. All these cases were adults with a pre-existing history of strabismus. In this instance, diplopia is elicited by forcing the strabismic eye to fixate. The suppression scotoma that is present in the strabismic eye may not be present in the dominant eye when the non-dominant eye is fixing and so diplopia results.[7]

No cases of monovision contact lens wear causing an esotropia with diplopia could be found in the literature. However, this is not the case with monovision produced by refractive surgery. Schuler et al[8] described a decompensated IVth nerve palsy with vertical diplopia after bilateral refractive surgery resulting in monovision. In this case the interrupted fusion caused decompensation of a previously controlled vertical deviation, with the patient finally needing glasses and a prism. Kushner and Kowal[9] found five mechanisms to account for diplopia following refractive surgery; technical problems, prior need of prisms, aniseikonia, iatrogenic monovision and improper control of accommodation in patients with strabismus. Monovision was accountable for seven of the 28 patients with diplopia following refractive surgery, with three of these due to decompensated intermittent deviations, three due to fixation switch diplopia and one a decompensated IVth nerve paresis previously well controlled. The anisometropia produced in this group was between 1.50DS and 2.50DS. As with Schuler, this disruption to the binocular state decompensated a previously well controlled strabismus.

It has been shown that long-standing monovision in adults results in the absence of foveal fusion and reduced stereoacuity.[10] Fawcett et al[10] compared 32 adults with longstanding monovision (greater than six months) through refractive surgery with a control group. Even when the binocular state was restored with optical devices, patients in the monovision group showed reduced stereopsis on random dot stereo tests and suppression on Worth Lights, lending evidence to the view that the adult binocular visual system is susceptible to change throughout life. Indeed the success of monovision seems to depend on the adult patients' ability to learn to suppress the blurred image.

How can we identify which patients will be at risk from monovision? The American Academy of Ophthalmology guidelines for the management of refractive surgery suggest a pre-operative evaluation of ocular motility and alignment.[11] Kushner and Kowal[9] go further, suggesting a trial of monovision contact lenses if there is more than a minimum of heterophoria, although this amount was not defined. However, it should be remembered that in our case it was three months before the monovision contact lenses produced symptoms of diplopia. It is unknown what, if any, ocular motility assessment was performed prior to giving monovision. It is also of interest that Ms Y was myopic with a convergent deviation.

Refractive surgery is a state not easily reversed. On the other hand, contact lenses can easily be removed and the binocular state restored. However, this case demonstrated that even the restitution of a normal binocular state may not be enough to restore a fusional amplitude sufficient for binocular single vision once it is disrupted. This finding is in agreement with Fawcett at al's conclusions that fusion

in adults can be lost if the visual system is disrupted.[10] Ms Y had symptoms of diplopia for 12 months before coming to our clinic. In this time she had developed a shallow suppression scotoma which remained despite prism and orthoptic treatment. This suppression scotoma may well have impeded the full recovery of binocularity.

CONCLUSION

Diplopia caused by monovision use of contact lenses is an unusual occurrence. However, it is advisable to know the binocular state of each patient before prescribing monovision. A simple cover test is enough to elicit any significant heterophoria. In the case of significant heterophoria, the patient may be informed of the risks of monovision and advised not to proceed. Close supervision should follow if the patient chooses monovision despite advice.

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A Case of Orbital Cellulitis with Accompanying Bilateral Ptosis

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ABSTRACT

A case study of a young male with right orbital cellulitis secondary to sinusitis is presented. Ocular signs are described, including decreased visual acuity, ptosis, proptosis, pain, and restriction of ocular movements. The patient had a number of clinical signs, including a decompensating intermittent exotropia and the continued presence of bilateral ptosis following resolution of the orbital cellulitis. It was concluded that the patient likely had previously unknown pre-existing conditions, which meant that he will continue to require ophthalmic and orthoptic management beyond the resolution of the orbital cellulitis.

Key Words: orbital cellulitis, sinusitis, orbital abscess, ptosis

INTRODUCTION

Orbital cellulitis is an infection of the soft tissues of the orbit posterior to the orbital septum.[1-3] It is a serious condition with many dangers, including optic nerve involvement that can result in decreased vision, cavernous sinus thrombosis, inflammation of meninges and brain abscess.[1,2,4-8] This condition needs to be treated as a medical emergency, with hospital admission often necessary, requiring medical and surgical intervention. It often develops suddenly and is generally accompanied by unilateral chemosis, ocular movement restrictions, severe pain, proptosis and lid swelling, and may also have decreased visual acuity and an afferent pupillary defect.[1,3,5,7,8]

CASE REPORT

In early September 2009, 14-year old Master C attended the clinic for review of his resolving orbital cellulitis. He had previously attended in August, following his original treatment in hospital.

At initial presentation in hospital during July, Master C complained of severe pain and swelling, resulting in trouble opening his eye. Right visual acuity (VA) was count fingers 2 m (right side of visual field) and count fingers ½ m (left side of visual field); left VA was 6/6. He had no afferent pupil defect. Ocular movements were affected, with underactions of -3 in all gazes except elevation, laevoelevation and laevodepression, which were -4. He was diagnosed with right orbital cellulitis secondary to sinusitis, and admitted to hospital where he remained for 14 days. Swab results found two streptococci species as the cause of the infection. Management included initial superior orbital abscess drainage, followed by drainage of ethmoid, maxillary and frontal sinuses; with intravenous benzylpenicillin for two weeks, vancomycin for five days, and metronidazole (Flagyl) for two weeks. Post surgery he complained of an increase in eye pain and further decreased VA, and proptosis was noted.

At discharge, VA had improved to 6/9 part and ocular movements were only mildly restricted in upward gaze. The right upper lid had mild swelling and ptosis, however a left ptosis was also noted. The right palpebral fissure width was 5 mm and the left 7 mm, and right levator function was 5 mm and left 13 mm. A significant exophoria was present that decompensated to an intermittent exotropia with consequent diplopia, however it was concomitant in both right and left gaze, with no medial rectus underaction. Due to the diplopia being intermittent, no treatment was given at this stage to allow for the resolving orbital cellulitis. Master C was reviewed monthly and since discharge he was treated with chloramphenicol (Chloromycetin) ointment and dexamethasone (Maxidex) drops twice daily (bd) in the right eye and ciprofloxacin (Ciloxan) drops bd to the right nostril.

Two months following discharge, uncorrected RVA was 6/18 and LVA 6/6-1. Subjective refraction RE was -0.75/-

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0.50 x 105° = 6/6, and LE -0.50/-0.50 x 105° = 6/5-1. He demonstrated a 20 to 25 dioptre exophoria with moderate recovery at both near and distance. Convergence function was of no concern, his near point being closer than 10 cm. Distance prism cover test in nine directions was performed with changes in head position whilst he fixed on a light source (Table 1). This was performed to clarify any residual vertical limitation.

Table 1. Distance prism cover test in nine directions (left eye fixing)

	30BI 6L/R	35BI 3L/R	25BI
Right Gaze	18BI	22BI	10BI Left Gaze
	10BI	12BI	8BI

Ocular movements had continued to improve with only a minor underaction in dextroelevation and all medications were finished. The orbital cellulitis had resolved, diplopia was no longer present and bilateral ptosis was now Master C's greatest concern. At the next review, consideration was to be given to the prescription of glasses and discussion of ptosis surgery.

DISCUSSION

Cellulitis has been reported as occurring in 3% of cases of sinusitis.[3] The incidence of sinusitis as a cause of cellulitis has been reported as between 66% and 91%.[1,3,5,7] Multiple sinus involvement, as in this case, has been reported in 20% to 35% of cases.[1,5,8] Orbital cellulitis occurs following sinus infection, most commonly the ethmoid and maxillary sinuses, either by direct spread to the orbit through the thin porous walls, or through normal venous drainage channels.[3,7-9] Surgical treatment involves drainage of any orbital or subperiosteal abscess, which allows the condition to resolve.[6,8,9] Drainage of the abscess aims to prevent potential visual function loss[7] and any damage to extraocular muscles.[6] Orbital cellulitis is commonly a result of the bacteria staphylococcus aureus or streptococcus pyogenes,[1,2,5,7-9] which was the case with Master C, where two streptococci species were found. As with Master C who is aged 14 years, it is usually children or young adults who present with orbital cellulitis,[5,7] the condition occurring most commonly in children aged 0 to 16 years, as sinusitis becomes more prominent as they reach preteen years.[1,2,7,8] It has been stated that there is a gender preference with twice as many males affected than females,[1,2,8] and that seasonal changes, in particular colder weather may play a part in the development of the condition.[1,5] The presence of an orbital abscess would have resulted in Master C's condition falling into the category of severe,[7] with the average length of stay for these patients being reported as 10 to 11 days due to surgical intervention being required.[7-9] Master C's stay of 14 days was therefore longer than the average, but this was as a result of the necessary surgeries that occurred and the decrease in VA and prolonged pain that resulted. In the case of Master C, upon initial presentation his condition reflected all the classic characteristics with results consistent with those found in reported studies.

Of interest in this case is the suspicion that there were pre-existing undiagnosed conditions. He presented with problems with his right eyelid, however examination at discharge noted a bilateral ptosis. A note in the patient's history stated 'noticed droopy eyelids' previously, but he had never had an ocular examination. Lid swelling due to the accumulation of fluids, rather than ptosis, is a feature of orbital cellulitis,[1-3,5,7-9] and it is generally considered that a bilateral ptosis indicates a congenital origin.[10] Normal palpebral fissure width is 10 mm, indicating that both the left and right eyes can be considered abnormal, with a bilateral asymmetrical ptosis is present.[11,12] As normal levator muscle function is defined as 12 to 17 mm, the left may be considered normal, however the right eye would be graded as 'fair' as it is between 5 and 8 mm.[13,14] It may also be of note that strabismus has been reported as occurring in 20% of cases of cases of congenital ptosis, compared to 1% to 5% of the general population, with horizontal strabismus accounting for two-thirds of these.[13,14]

The second issue is the presence of a moderately large exophoria of mixed type for near and distance, with a V-pattern. During the acute phase of the cellulitis this decompensated to an intermittent exotropia. In the Sydney Myopia Study it was reported that exophoria was present for near fixation in 52.2%, and for distance in 7.8%, of 12-year old children, though the incidence of any heterophoria of 10 dioptres or larger was only 3.2%.[15] It was also reported that 12.3% of 12-year olds were 0.50 dioptres or more myopic, and that those who were myopic were 2.1 times more likely to have an exophoria for near and 3.1 times more likely for distance.[15] This would support the hypothesis that increasing myopia and its effect on the accommodative convergence control mechanism may also have contributed to the decompensation.

With the one-line difference in final vision, the question was raised of the possibility of a residual defect from the cellulitis or a pre-existing amblyopia. Amblyopia has been defined as a visual acuity difference of two lines or more,[16] and one line of a LogMAR chart is considered a normal interocular difference.[17] So, the one-line difference in best corrected visual acuity would be considered normal.

In summary, after full recovery from an acute episode of orbital cellulitis, it is suspected that Master C had a combination of pre-existing ocular conditions that manifested or became obvious during or after his recovery. These included a bilateral asymmetric ptosis, a moderately

large exophoria of mixed type, and increasing myopia. During the acute phase the exophoria decompensated, most likely due to a combination of the decreased visual acuity and the vertical limitations. It remains to be seen whether he will maintain the good control of his exophoria, or whether myopia will become an increasingly dissociative factor.

CONCLUSION

Orbital cellulitis is potentially dangerous to the eye and may be a life-threatening condition, however with suitable treatment it can be resolved. Many patients like Master C are of a young age, and obtain the infection from sinusitis. If the patient fails to respond to antibiotics, VA is decreasing, or an abscess is present, surgery is indicated. Sinusitis is the most common cause of orbital cellulitis, and with appropriate intravenous antibiotics and surgical care, recovery is optimistic with the major complications of the condition all but eliminated.[1,8] It is important to acknowledge that in this case the clinical dilemma was whether the ocular signs remaining after the resolution of the condition were pre-existing or a residual effect of the orbital cellulitis. Ocular conditions such as orbital cellulitis may not present as a textbook case, and awareness of this enables the clinician to manage the patient effectively.

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A Case Study: Bilateral Internuclear Ophthalmoplegia

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ABSTRACT

A 30-year old female presented with a five-day history of vertical diplopia. Clinical examination revealed bilateral restriction of adduction and nystagmus of the abducting eye, diagnosed as a bilateral internuclear ophthalmoplegia. A three-day course of intravenous methylprednisolone was prescribed and her signs and symptoms soon resolved. Later, magnetic resonance imaging revealed no signs of demyelination.

Keywords: internuclear ophthalmoplegia, multiple sclerosis, medial longitudinal fasciculus, nystagmus, methylprednisolone

INTRODUCTION

Internuclear ophthalmoplegia (INO) is a disorder of conjugate horizontal gaze. Typically it is elicited as an adduction paresis of one eye and nystagmus of the abducting eye on lateral gaze.[1] It can be either unilateral or bilateral, and is caused by a lesion of the medial longitudinal fasciculus (MLF) between the third and sixth cranial nerve nuclei in the brainstem, with or without involvement of the vergence midbrain control mechanisms.[1,2]

As the MLF is a highly myelinated tract within the brainstem, the most common cause of INO in young people is demyelinating disease secondary to multiple sclerosis (MS) (41%-54%). Other aetiologies can include cerebral/brainstem vascular accidents (23-27%), infection (5-14%), head trauma (6%), brainstem tumour (4-5%), systemic lupus erythematosus (<5%), nutritional and metabolic disorders, or degenerative disorders.[3-6]

Patients are unlikely to experience diplopia in primary position with most being orthophoric. In fact bilateral INO may be asymptomatic.[1] Horizontal diplopia on lateral gaze is the most common complaint, with or without the presence of oscillopsia due to the lateral gaze nystagmus.[1]

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CASE STUDY

Ms Z, a 30-year old female legal assistant, presented with a five-day history of vertical diplopia in left gaze with no loss of vision. Ms Z also reported that on the second day of her symptoms she noticed a transient decrease in her "mental acuity". There had been no history of head trauma. Aside from slight asthma and being clinically overweight, her general health was good and she took no medications. Ms Z did, however, report that she had recently been under a lot of stress at work.

Ms Z's past ocular history was uneventful and revealed only a slight myopic refractive error. Her mother has a history of diabetic eye related problems.

On examination, visual acuity was 6/5 and N5 both eyes. Cover testing revealed orthophoria at near and a small exophoria with rapid recovery at distance fixation. Ocular motility assessment revealed slight bilateral limitation of adduction on horizontal gaze. Nystagmus was noted on both right and left abduction, left worse than right, with no oscillopsia. Small amplitude downbeat nystagmus was also noted on down gaze. The patient reported vertical diplopia on left gaze, although no vertical muscle anomaly was noted. There was no pain on eye movements. Colour vision testing with Ishihara showed no defect. Brightness saturation was estimated at 90-95% right, and 100% left. Red saturation was estimated at 80-85% right and 100% left. Pupils showed no sign of relative afferent pupil defect. Upon ophthalmic examination, anterior chamber, lens, peripheral retina and macula were all found to be healthy in either eye, with the optic nerve showing no signs of papilloedema. Routine testing of blood pressure recorded 150/80.

The patient was subsequently diagnosed with bilateral INO. Possible aetiology was suspected to be MS due to her young age. She was therefore immediately admitted to the Royal Victorian Eye & Ear Hospital (RVEEH) for treatment. Upon arrival, the patient was re-assessed, confirming mild bilateral limitation of adduction on contralateral gaze, clinically observed slow saccades and abducting nystagmus of either eye. Convergence was intact and no proptosis was observed. Diagnosis of bilateral INO was verified and the patient was admitted as an inpatient for intravenous (IV) pulse methylprednisolone for three days. Urgent magnetic resonance imaging (MRI) was also ordered.

The following day her fasting blood glucose levels, HbA1c, measured 9.0% (normal ≤7%). The patient was unwell and complaining of a headache. She was also tachycardic, although this was attributed to her anxiety. Intermittent horizontal diplopia on extreme dextroversion and laevoversion was present, now with no vertical component. A Humphrey Visual Field was performed and showed no defects. A second dose of methylprednisolone was commenced.

On the third day, the last dose of IV steroids was commenced and blood sugar levels via the finger prick test were measured at 9.8mmol/L (normal 4-8mmol/L). The patient's general health had improved, the headache resolved and a reduction of the nystagmus amplitude was observed. The patient was discharged later that day and was due for follow-up with a neuro-ophthalmologist.

Two months later, the MRI scan revealed two non-specific supratentorial T2 hyperintense white matter foci, which of themselves were not diagnostic of demyelination. Cerebral, brainstem and cerebellar parenchymal signals were normal, in particular there were no callosal septal interface, corpus callosum, midbrain, middle cerebellar peduncle or temporal lobe lesions. There was no evidence of atrophy. The optic nerves had a symmetrical size and signal.

DISCUSSION

Optic neuritis and internuclear ophthalmoplegia are the most common ocular presenting signs in MS,[2,4,7 8] with optic neuritis present in 50% to 90% of MS patients[8,9] and INO in 17% to 53%.[3,9-11] MS is said to be the most common cause of bilateral INO in young adults.[1-5,8]

Bilateral INO affects both sides of the MLF, producing bilateral adduction deficits, abducting nystagmus, as well as horizontal diplopia on lateral gazes. Horizontal gaze is mediated by the abducens nucleus, from which abducens motor neurons innervate the ipsilateral lateral rectus via the sixth nerve. Abducens interneurons cross to the contralateral MLF to the oculomotor nucleus, with motor neurons innervating the contralateral medial rectus. A bilateral MLF lesion results in disruption of adduction on horizontal gaze, with the abduction nystagmus thought to be a compensatory mechanism.[1,7,12] Convergence is generally intact in INO,[7] with only 10% of MS patients with eye movement problems having vergence affected.[10] This indicates a more caudal lesion, sparing the vergence control centres in the rostral midbrain.[1,7]

Vertical gaze-evoked nystagmus commonly occurs with bilateral INO,[1,8,9,11-13] with one study reporting 55% of cases with bilateral INO having vertical nystagmus.[3] This is due to a disruption of the vestibulo-ocular and cerebello-ocular pathways through the MLF to the vertical gaze integrator, the interstitial nucleus of Cajal.[1,7,8,13] Skew deviation, a supranuclear vertical misalignment with hypertropia and incyclotorsion on the ipsilateral side to the INO may also occur in unilateral cases,[1,2,4,7,12] with 20% demonstrating skew deviation.[3] This is due to an interruption of the otolithic pathways ascending the MLF.[1,8] It could be hypothesised that Ms Z originally had a unilateral INO with a skew deviation that progressed to a bilateral INO, hence explaining her change in diplopic symptoms from vertical to horizontal, as in a similar reported case.[4] With hindsight, a more detailed examination of ocular motility with a prism cover test or Maddox Rod test would have elicited more information of the minor vertical muscle imbalance, and may have explained her initial complaint of vertical diplopia.

Although this patient had nystagmus on both lateral gazes, lateral recti defects were eliminated by the detection of full abduction and the presence of an adduction defect. A differential diagnosis of ocular myasthenia gravis was eliminated due to the patient's reduced saccadic velocity on adduction. Patients with myasthenia gravis have normal saccades, despite their pseudo-INO appearance at times.[2,14] The uncommon occurrence of INO is always reported in the context of previously diagnosed SLE, rather than as a presenting disorder, is rarely bilateral, and almost always resolves with corticosteroid treatment,[15,16] which would eliminate SLE as a cause in this case.

Optic neuritis presents as a sudden unilateral loss of visual acuity, caeco-central scotoma on visual field testing, pain on eye movement, afferent pupil defect, colour vision impairment (predominantly red) and photopsia.[17] Even though Ms Z reported subtle brightness and red desaturation on the right, she had no pupillary, visual acuity or visual field defects, and in particular, no signs of optic neuritis. A more appropriate colour vision test would have been the City University Colour Vision Test or the Farnsworth Munsell 100 Hue Test as these are more sensitive in detecting acquired defects. It has recently been demonstrated that subjective measurements of brightness intensity and red saturation are clinically significant tests able to detect optic neuropathy to a high degree of sensitivity and specificity.[18] Optic neuritis is the most common ocular manifestation, and the initial presenting sign, in up to 20% of MS patients.[9] The 10-year probability of developing MS after an acute episode of optic neuritis, for a female with no brain lesion found on MRI, is

25%.[19] This risk may be applied to any isolated demyelinating episode, including optic nerve, brainstem or spinal cord.[19-21]

Despite an urgent MRI scan request, this was not available for two months, which caused the patient further distress and anxiety. This waiting time is of some concern, as in this case the opportunity to detect an early and transient aetiology has been missed. However, it is not unusual for MRI to be normal in the presence of an INO, with 31% reported by Bolanos.[3] According to the 2005 McDonald Diagnostic Criteria for Multiple Sclerosis, the diagnosis of MS cannot be confirmed until deterioration over time is established by the collective data of repeat MRI, abnormal visual evoked potential test, lumbar puncture positive for oligoclonal bands or increased immunoglobulin G, or another neurological episode occurs.[22]

Methylprednisolone is commonly used to treat inflammatory, haematological, neural and ophthalmic disorders. Its prescription is usually the first line of treatment for acute episodes in patients with MS, hence why it was prescribed to Ms Z. IV methylprednisolone reduces the duration and severity of attacks, and was found to reduce the 2-year risk of MS, 8% versus 18% placebo, however there was no difference in the longer term development of MS.[23,24] There can however be collateral diabetic signs and symptoms as well as a manifestation of latent diabetes mellitus whilst on methylprednisolone, explaining the increase in blood glucose levels in this patient's case.[24,25] Despite this, with a family history of diabetes, Ms Z's elevated blood glucose levels cannot solely be attributed to the treatment of methylprednisolone without further investigation.

CONCLUSION

There is no specific treatment for the eye signs of INO, as the diplopia in extreme lateral gaze precludes the use of prism therapy, orthoptic training or ocular surgery. The patient should be treated according to the underlying cause. In this patient's case, given the diagnosis of bilateral INO and a possible right optic neuritis, MS was the tentative diagnosis, and high-dose IV pulse corticosteroids were prescribed. Given that only 25% of female patients will develop MS after 10 years, this natural history must be considered when deciding on prophylactic treatment at the time of the first acute demyelinating episode.[26] For Ms Z, the tentative diagnosis of MS relies on the only sign being the bilateral INO. As this does not fit the McDonald Diagnostic Criteria as yet, it would be wise to repeat MRI testing in approximately six months with ongoing ophthalmic and neurologic review.

ACKNOWLEDEGMENTS

The authors wish to acknowledge Dr Renuka Bathija for her support.

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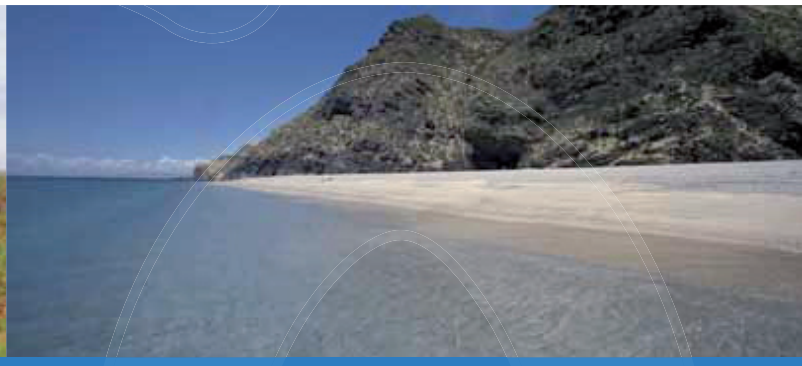

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