FIFTH EDITION

OCULAR SYNDROMES & SYSTEMIC DISEASES



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JAYPEE

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1. A-ESOTROPIA SYNDROME

General

Esotropia greater looking up by 15 prism diopters than looking down; an overaction of superior oblique muscles or underaction of inferior rectus muscles; fusion may be obtained by chin elevation; mongoloid (upward) slant of lid fissures; may be accommodative, nonaccommodative or paralytic esotropia components.

Laboratory

Diagnosis is made by clinical findings.

Treatment

Recess the medial rectus bilaterally with a half tendon upshift. If unilateral surgery recess medial rectus with upshift and resect the lateral rectus (LR) with a half tendon downshift. With significant oblique overaction—recess the medial rectus bilaterally or recess the medial rectus and resect the LR unilaterally for esotropia in primary position weaken the superior oblique bilaterally if 25 diopters from up to down.

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1. Beyer-Machule C, von Noorden GK. Atlas of Ophthalmic Surgery. New York: Thieme; 1984.
2. Hwang J, Wright KW. Strabismus syndromes. In: Wright KW (Ed). Pediatric Ophthalmology and Strabismus. St. Louis: Mosby; 1995. p. 223.
3. Roy FH. Practical Management of Eye Problems: Glaucoma, Strabismus, Visual Fields. Philadelphia: Lea & Febiger; 1975. pp. 32-134.

2. A-EXOTROPIA SYNDROME

General

Exotropia greater looking down by 15 prism diopters than looking up; mongoloid (upward) slant of lid fissures; alternating sursumduction and associated vertical divergence; overaction of superior oblique muscles or underaction of inferior oblique or inferior rectus muscles; fusion obtained by chin depression.

Laboratory

Diagnosis is made by clinical findings.

Treatment

Recess the lateral rectus with downshift. If unilateral surgery recess the lateral rectus with downshift and resect the medial rectus with upshift. With oblique dysfunction—recess the rectus bilateral and weaken the superior oblique bilaterally.

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1. Hardesty HH. Superior oblique tenotomy. Arch Ophthalmol. 1972;88:181-4.
2. Hwang J, Wright KW. Strabismus syndromes. In: Wright KW (Ed). Pediatric Ophthalmology and Strabismus. St. Louis: Mosby; 1995. p. 223.
3. Roy FH. Practical Management of Eye Problems: Glaucoma, Strabismus, Visual Fields. Philadelphia: Lea & Febiger; 1975. pp. 144-5.

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3. A-PATTERN STRABISMUS

General

Visual axis greater in upgaze than downgaze by at least 15 prism diopters.

Clinical

None

Ocular

A-esotropia; A-exotropia; amblyopia.

Laboratory

Diagnosis is made by clinical findings.

Treatment

A-pattern esotropia without oblique dysfunction—recess medial rectus bilateral with upshift or recess the medial rectus with upshift and resect lateral rectus with downshift in one eye. A-pattern esotropia with superior oblique overaction—recess the medial rectus bilaterally or recess medial rectus and resect lateral rectus unilaterally and weaken superior oblique by tenectomy. A-pattern exotropia without oblique dysfunction—recess the lateral rectus bilaterally with downshift or unilaterally recess the lateral rectus with a downshift and resect the medial rectus with a upshift. A-pattern exotropia with superior oblique overaction—recess the lateral rectus bilaterally or unilaterally recess the lateral rectus and resect the medial rectus and weaken the superior oblique bilaterally by tenectomy.

Additional Resource

1. Plotnik JL (2011). A-Pattern Esotropia and Exotropia. [online] Available from http://www.emedicine.com/oph/TOPIC560.HTM [Accessed April, 2012].

4. AARSKOG-SCOTT SYNDROME (FACIOGENITAL DYSPLASIA)

General

Sex-linked; characterized by ocular hypertelorism, anteverted nostrils, broad upper lip and saddle-bag scrotum.

Ocular

Ptosis; hypertelorism.

Clinical

Hyperextensibility of fingers; genu recurvatum; flat feet; hypermobility in cervical spine with neurologic deficit; cleft lip and palate; anteverted nostrils; broad upper lip; abnormal penoscrotal relations; "saddle-bag scrotum".

Laboratory

For full and detailed physical examination, a radiologic evaluation is essential. Standard posteroanterior and lateral views are used for the initial evaluation.

Treatment

Primary goal is to prevent the development of a severe deformity rather than treating the current deformity.

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Bibliography

1. Bowle E, Tyrkus M, Lipman S, et al. Aarskog syndrome: full male and female expression associated with an X-autosome translocation. Am J Med Genet. 1984;17:595-602.
2. McKusick VA. Mendelian Inheritance in Man: A Catalog of Human Genes and Genetic Disorders, 12th edition. Baltimore: The Johns Hopkins University Press; 1998.
3. Scott CI. Unusual facies, joint hypermobility, genital anomaly and short stature: a new dysmorphic syndrome. Birth Defects Orig Artic Ser. 1971;7:240-6.

5. AARSKOG SYNDROME (FACIAL-DIGITAL-GENITAL SYNDROME)

General

X-linked recessive; males fully affected; females exhibit partial features; normal birth weight and length.

Ocular

Telecanthus; hypertelorism; unilateral or bilateral blepharoptosis; strabismus; hyperopic astigmatism; large cornea.

Clinical

Short stature; triangular facies; deformity of hands and feet; anomalies of external genitalia; inguinal hernia; protruding umbilicus; abnormal cervical vertebrae; cryptorchidism.

Laboratory

For full and detailed physical examination, a radiologic evaluation is essential. Standard posteroanterior and lateral views are used for the initial evaluation.

Treatment

Primary goal is to prevent the development of a severe deformity rather than treating the current deformity.

Bibliography

1. Kirkham TH, Milot J, Berman P. Ophthalmic manifestations of Aarskog (facial-digital-genital) syndrome. Am J Ophthalmol. 1975;79:441-5.

6. ABDOMINAL TYPHUS (ENTERIC FEVER; TYPHOID FEVER)

General

Causative agent is *Salmonella typhi*.

Ocular

Conjunctivitis; chemosis; corneal ulcer; tenonitis; paralysis of extraocular muscles; endophthalmitis; panophthalmitis; optic neuritis; retinal detachment; central scotoma; central retinal artery emboli; iritis with or without hypopyon; choroiditis; retinal hemorrhages; bilateral optic neuritis; abnormal ocular motility (likely secondary to thrombotic infarcts affecting the ocular motor nerve nuclei, fascicles, brainstem or cerebral hemispheres).

Clinical

Fever; headache; bradycardia; splenomegaly; maculopapular rash; leukopenia; encephalitis. Salmonella may produce an illness characterized

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by fever and bacteremia without any other manifestations of enterocolitis or enteric fever, which is particularly common in patients with acquired immunodeficiency syndrome (AIDS).

Laboratory

Gram-negative bacillis isolation from blood culture (50–70% of cases). Stool culture positive less frequent.

Treatment

Early detection, antibiotic therapy, adequate fluids, electrolytes, and nutrition reduce the rate of complications and reduce the case-fatality rate.

Additional Resource

1. Brusch JL (2011). Typhoid Fever. [online] Available from http://www.emedicine.com/med/TOPIC2331.HTM [Accessed April, 2012].

7. ABDUCENS PARALYSIS (SIXTH NERVE)

General

Unilateral or bilateral, multiple etiologies because of long course of sixth nerve.

Clinical

Childhood neoplasm, raised intracranial pressure, hypertension, diabetes.

Ocular

Inability to turn the eye laterally.

Laboratory

Diagnosis is made by clinical findings.

Treatment

Tape or base-out prism on one eyeglass may be useful, botulinum toxin type A into the antagonist medial recturs muscle, if no improvement after 6–12 months—recess/resect of medial and lateral rectus.

Additional Resource

1. Ehrenhaus MP (2012). Abducens Nerve Palsy. [online] Available from http://www.emedicine.com/oph/TOPIC158.HTM [Accessed April, 2012].

8. ABERFELD SYNDROME (CONGENITAL BLEPHAROPHIMOSIS ASSOCIATED WITH GENERALIZED MYOPATHY SYNDROME; OCULAR AND FACIAL ABNORMALITIES SYNDROME; SCHWARTZ-JAMPEL SYNDROME)

General

Etiology not known; autosomal recessive inheritance, although there are reports of dominant inheritance; progressive disorder.

Ocular

Blepharophimosis; exotropia; myopia; congenital cataracts; microcornea.

Clinical

Myopathy; bone deformities; arachnodactyly; dwarfism; hypoplastic facial bones; hypertrichosis; kyphoscoliosis.

Laboratory

Physicians might consider referring suspected cases to genetic clinics that have affiliations with

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groups actively research so that genetic studies can be performed. Muscle biopsy findings are consistent with a myopathy.

Treatment

The goal is to reduce the abnormal muscle activity that causes stiffness. Botulinum toxin therapy may be considered for the treatment of blepharospasm. However, if ptosis is present, this is contraindicated. Some medications that have been found useful in myotonic disorders such as the anticonvulsants and the antiarrhythmics.

Additional Resource

1. Ault J (2012). Schwartz-Jampel Syndrome. [online] Available from http://www.emedicine.com/neuro/TOPIC337.HTM [Accessed April, 2012].

9. ABSENCE OF FOVEAL REFLEX

The absence of foveal reflex is caused by drugs, including amodiaquine, chloroquine, diiodohydroxyquin, hydroxychloroquine, iodochlorhydroxyquin or quinine.

Bibliography

1. Fraunfelder FT. Drug-Induced Ocular Side Effects and Drug Interactions. Philadelphia: Lea & Febiger; 1982.

10. ACANTHAMOEBA

General

Caused by *Acanthamoeba polyphaga* and *Acanthamoeba cartel* (see Herpes Simplex Masquerade Syndrome); all types of contact lenses have been associated with acanthamoeba keratitis, particularly daily-wear soft contact lenses.

Ocular

Hypopyon; uveitis; conjunctivitis and chemosis; keratitis; pannus; corneal ring abscess; papillitis; vitreitis; retinal perivasculitis; secondary glaucoma; postkeratoplasty acanthamoeba keratitis may present as an infectious crystalline keratopathy in the periphery of the graft.

Clinical

Meningoencephalitis; meningitis; hemorrhagic encephalitis.

Laboratory

Polygonal double-walled cysts, under bright-field or phase-contrast microscopy or stained with hematoxylin and eosin, Gram, Giemsa or celluflor white.

Treatment

Medical therapy for Acanthamoeba infection is not well established. Topical antimicrobial agents that achieve high concentrations at the site of the infection can be considered. Treatment of keratitis consists of early diagnosis and aggressive surgical and medical therapies.

Additional Resources

1. Crum-Cianflone NF (2011). Acanthamoeba. [online] Available from http://www.emedicine.com/med/TOPIC10.HTM [Accessed April, 2012].
2. Wang JC (2010). Ophthalmologic Manifestations of Herpes Simplex Keratitis. [online] Available from http://www.emedicine.com/oph/TOPIC100.HTM [Accessed April, 2012].

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11. ACANTHOSIS NIGRICANS

General

Rare skin disease of unknown etiology; occurs at any age; equal frequency in males and females; thickening and hyperpigmentation of the skin of the entire body, especially in flexural areas; autosomal dominant inheritance.

Ocular

Conjunctivitis; pigmentation of palpebral conjunctiva; tumors on lids and lid margins; madarosis; trichiasis.

Clinical

Hyperkeratotic lesions on face, neck, oral mucosa, axillae, groin, antecubital fossae and umbilicus.

Laboratory

Basic workup for underlying malignancy and screen for insulin resistance.

Treatment

The goal of therapy is to correct the underlying disease process. Treatment of the lesions of acanthosis nigricans (AN) is for cosmetic reasons only.

Additional Resource

1. Miller JH (2010). Acanthosis Nigricans. [online] Available from http://www.emedicine.com/derm/TOPIC1.HTM [Accessed April, 2012].

12. ACCOMMODATIVE ESOTROPIA

General

Hereditary, onset between the ages of 6 months and 5 years.

Clinical

None

Ocular

Uncorrected hyperopia with insufficient fusional divergence. The hyperopia averages 5 diopters. The esotropia is equal distance and near.

Laboratory

Diagnosis is made by clinical findings.

Treatment

Amblyopia therapy: Spectacle correction—full cycloplegic refraction if esotropia is at same distance and near, greater near than distant esotropia—bifocals up to +3.50 diopters set high at mid-pupil, gradually reduce the power to maintain fusion, contact lens if patient is capable, echothiopate iodine use to reduce the use of spectacles and if residual esotropia after full correction consider strabismus surgery.

Additional Resource

1. Noyes C (2012). Accommodative Esotropia. [online] Available from http://www.emedicine.com/oph/TOPIC554.HTM [Accessed April, 2012].

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13. ACCOMMODATIVE INSUFFICIENCY (ACCOMMODATIVE EFFORT SYNDROME)

General

Male or female, 10–35 years of age.

Ocular

Asthenopia with near vision appearing within a few minutes after reading, sewing or observing a near object; increased amplitude of accommodative adduction; abnormal relaxation of accommodation induced by relative divergence at close distances; latent convergence insufficiency; may result secondary to ciliary dysfunction associated with Adie's pupil.

Clinical

It may be secondary to a systemic disorder such as Parkinson disease or oral lithium.

Laboratory

Accommodation testing are the basic tests that used to help diagnose this condition.

Treatment

Convergence exercises including orthoptics, vision therapy and/or base-in prisms are the mainstays of treatment of convergence insufficiency.

Additional Resource

1. Bartiss MJ (2011). Convergence Insufficiency. [online] Available from http://www.emedicine.com/oph/TOPIC553.HTM [Accessed April, 2012].

14. ACCOMMODATIVE SPASM (SPASM OF THE NEAR REFLEX)

General

Episodic excessive contraction of the ciliary muscle.

Clinical

Posterior fossa tumor, central nervous system (CNS) infection, head trauma, cerebrovascular injuries.

Ocular

Diplopia, esotropia, accommodation, convergence, miosis.

Laboratory

Computed tomography (CT) and magnetic resonance imaging (MRI).

Treatment

Cycloplegics to break the accommodative component, refractive correction for distance with reading addition.

Bibliography

1. Roy FH, Fraunfelder FW, Fraunfelder FT. Roy and Fraunfelder's Current Ocular Therapy, 6th edition. Philadelphia: WB Saunders; 2008.

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15. ACERULOPLASMINEMIA

General

Autosomal recessive, adult-onset.

Ocular

Maculopathy which resembles aging macular degeneration.

Clinical

Associated with increased levels of copper in the retina.

Laboratory

Increased urinary copper level, greater than 100 mcg/24 hours (normal, 10–80 mcg/24 h).

Treatment

A low copper diet is recommended. Foods that are high in copper content include shellfish, liver, mushrooms, broccoli, chocolate and nuts.

Bibliography

1. Miyajima H. Aceruloplasminemia, an iron metabolic disorder. Neuropathology. 2003;23:345-50.

16. ACHARD SYNDROME

General

All features of Marfan syndrome, with the addition of dysostosis mandibulofacialis; arachnodactyly; receding lower jaw; joint laxity limited to the hands and feet; differs from Marfan syndrome in that the skull is broad and brachycephalic with small mandible.

Ocular

Myopia; lens dislocation; spherophakia.

Clinical

Mandibulofacial dysostosis; skeletal anomalies; arachnodactyly; high-arched palate; heart disease.

Laboratory

Genetic testing, molecular studies.

Treatment

Dislocated lens can be treated with careful phacoemulsification, topical steroids to control ocular inflammation.

Bibliography

1. Achard D. Arachnodactylie. Bull Mem Soc Med Hop Paris. 1902;19:834.
2. Jackson LG. Genetic principles for the ophthalmologist. Trans Pa Acad Ophthalmol Otolaryngol. 1974;27:20-8.
3. Magalini SI, Scrascia E. Dictionary of Medical Syndromes, 2nd edition. Philadelphia: JB Lippincott; 1981.
4. McKusick VA. Mendelian Inheritance in Man: A Catalog of Human Genes and Genetic Disorders, 12th edition. Baltimore: The Johns Hopkins University Press; 1998.
5. McKusick-Nathans Institute for Genetic Medicine, Johns Hopkins University and National Center for Biotechnology Information, National Library of Medicine. (2007). Online Mendelian Inheritance in Man (OMIM). [online] Available from http://www.ncbi.nlm.nih.gov/omim [Accessed April, 2012].

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17. ACHONDROPLASIA

General

Dwarfism; etiology unknown; occurs in both sexes; inheritance is autosomal dominant with almost complete penetrance; characterized by rhizomelic dwarfism (reduction most marked in the proximal limbs); mid-face hypoplasia; exaggerated lumbar lordosis; limitation of hip and elbow expansion; location of achondroplastic gene reported to be in the short arm of chromosome 1.

Ocular

Strabismus; optic atrophy; hypermetropia.

Clinical

Osseous impingement upon cranial nerves; rhizomelic short stature; facial features include frontal bossing, depressed nasal bridge, relative mandibular prognathism; connective tissue dysplasia; hypotonic in infancy; paraplegia may develop in the second or third decade.

Laboratory

Plasma can be analyzed for the fibroblast growth factor receptor 3 (FGFR3) mutation in the mother when a short-limb skeletal dysplasia is diagnosed prenatally on ultrasound. This can be confirmatory for achondroplasia.

Treatment

Growth hormone is currently being used to augment the height of patients with achondroplasia. Orthopedic physician will need to be consulted concerning problems related to the spine.

Additional Resource

1. Parikh S (2012). Achondroplasia. [online] Available from http://www.emedicine.com [Accessed April, 2012].

18. ACHOO SYNDROME (AUTOSOMAL DOMINANT COMPELLING HELIO-OPHTHALMIC OUTBURST SYNDROME; PEROUTKA SNEEZE; PHOTIC SNEEZE REFLEX; SNEEZING FROM LIGHT EXPOSURE)

General

Autosomal dominant; sneezing in response to bright light, especially sunlight; association between photic sneeze and nephropathic cystinosis has been reported.

Ocular

Photic sneeze reflex by sudden exposure of dark-adapted subject to bright light.

Clinical

Successive sneezing, as many as 43 in a row.

Laboratory

Diagnosis is made by clinical findings.

Treatment

None

Bibliography

1. Collie WR, Pagon RA, Hall JG, et al. ACHOO syndrome. Birth Defects Orig Artic Ser. 1978;14:361-3.
2. Katz B, Melles RB, Swenson MR, et al. Photic sneeze reflex in nephropathic cystinosis. Br J Ophthalmol. 1990;74:706-8.
3. McKusick VA. Mendelian Inheritance in Man: A Catalog of Human Genes and Genetic Disorders, 12th edition. Baltimore: The Johns Hopkins University Press; 1998.
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19. ACID BURNS OF THE EYE

General

Acid injuries of the eyes are characterized by protein coagulation and precipitation with the anion. Direct tissue damage produced by the hydrogen ion.

Clinical

None

Ocular

Chemosis, corneal epithelial defects, limbal blanching, corneal clouding, photophobia, corneal neovascularization, symblepharon formation.

Laboratory

Diagnosis is made from clinical history and findings.

Treatment

Immediate irrigation. Topical antibiotics for bacterial infection if needed. Contact lenses aid in re-epithelialization of the cornea.

Bibliography

1. Roy FH, Fraunfelder FW, Fraunfelder FT. Roy and Fraunfelder's Current Ocular Therapy, 6th edition. Philadelphia: WB Saunders; 2008.

20. ACINETOBACTER (ACINETOBACTER IWOFFI; MIMA POLYMORPHA)

General

Gram-negative pleomorphic bacillus *Mima*; generally occurs in patient with lowered resistance.

Ocular

Conjunctivitis and chemosis; corneal ulcer; blepharitis; iris prolapse; endophthalmitis.

Clinical

Meningitis; pneumonitis; endocarditis; urethritis; vaginitis; arthritis; dermatitis; intracranial abscess; subdural empyema.

Laboratory

Culture of the appropriate body fluid that is properly transported, plated and incubated grows *A. baumannii*.

Treatment

An infectious disease specialist should be consulted to differentiate colonization from infection and for antibiotic recommendations. Fluoroquinolone is the treatment of choice.

Additional Resource

1. Cunha BA (2011). Acinetobacter. [online] Available from http://www.emedicine.com/med/TOPIC3456.HTM [Accessed April, 2012].

21. ACKERMAN SYNDROME

General

Autosomal recessive; characterized by pyramidal molar roots.

Ocular

Juvenile glaucoma.

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Clinical

Unusual upper lip and dental roots; pyramidal molar roots.

Laboratory

Diagnosis is made by clinical findings.

Treatment

Congenital glaucoma: Primary congenital glaucoma almost always is managed surgically, both goniotomy and trabeculectomy may be useful. When multiple goniotomies and/or trabeculectomies fail, the surgeon usually resorts to a filtering procedure.

Bibliography

1. Antine BE, Brown FM, Arisco MJ. Fibroma of the cornea. Arch Ophthalmol. 1974;91:278-80.
2. Kenyon KR. Mesenchymal dysgenesis in Peter's anomaly, sclerocornea and congenital endothelial dystrophy. Exp Eye Res. 1975;21:125-42.

22. ACL SYNDROME (ACROMEGALOID; CORNEAL LEUKOMA SYNDROME; CUTIS VERTICIS GYRATA)

General

Autosomal dominant; rare; three features include: (1) cutis verticis, (2) associated with acromegaly and (3) corneal leukoma; onset by age 1 year.

Ocular

Bilateral corneal leukoma; keratitis.

Clinical

Unusually tall; large hands, feet and chin; skin of hands very soft; skin of scalp lies in folds; frontal bosses; ear calcification; pituitary tumors; abnormal dermal ridge patterns; enlargement of supraorbital arch of frontal bone.

Laboratory

Anticardiolipin (aCL) antibodies react primarily to membrane phospholipids, such as cardiolipin and phosphatidylserine. Of the three known isotypes of aCL [i.e. immunoglobulin G (IgG), IgM, IgA], IgG correlates most strongly with thrombotic events.

Treatment

Ocular—patients with bilateral and visually disabling corneal opacity, penetrating keratoplasty (PK) is recommended.

Bibliography

1. Antine BE, Brown FM, Arisco MJ. Fibroma of the cornea. Report of a case associated with congenital generalized fibromatosis. Arch Ophthalmol. 1974; 91:278-80.

23. ACNE ROSACEA (ACNE ERYTHEMATOSA; OCULAR ROSACEA)

General

Etiology unknown; usually occurs in women 30–50 years of age; pathogenetic mechanism remains unclear.

Ocular

Conjunctivitis; corneal neovascularization (wedge-shaped); keratitis; meibomianitis; blepharitis; recurrent chalazion; conjunctival hyperemia; superficial punctate keratopathy; corneal vascularization, thinning, perforation and scarring; episcleritis; scleritis; iritis; nodular conjunctivitis.

Clinical

Symmetrical erythema; papules; pustules; telangiectasia; sebaceous gland hypertrophy of the forehead, malar eminences and nose.

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Laboratory

Diagnosis is made by clinical findings.

Treatment

Systemic antibiotics are useful in most cases.

Additional Resource

1. Banasikowska AK (2011). Rosacea. [online] Available from http://www.emedicine.com/derm/TOPIC377.HTM [Accessed April, 2012].

24. ACOSTA SYNDROME (MOUNTAIN CLIMBER SYNDROME; MOUNTAIN SICKNESS; MONGE SYNDROME; SOROCHE SYNDROME)

General

Cause is cerebral hypoxia at high altitudes; Monge syndrome is the chronic form of mountain sickness.

Ocular

Acute blurred vision; difficulties in color discrimination; impaired light adaptation; retinal hemorrhage; chronic lid edema; bluish scleral injection; decreased visual acuity.

Clinical

Restlessness and irritability; headaches; impaired judgment at approximately 15,000 feet; confusion, cyanosis, muscular incoordination and possible loss of consciousness at approximately 18,000–20,000 feet; exertional dyspnea; epistaxis; gum bleeding; hemoptysis; anorexia; nausea; vomiting; tinnitus; cough; loss of libido; paresthesian extremities; coma; clubbing of fingers; hepatosplenomegaly.

Laboratory

Computed tomography (CT) is useful in patients with focal neurologic findings or in atypical cases of suspected high altitude disease.

Treatment

The symptoms improve dramatically with descent, and by the time a patient reaches the emergency department, further treatment is rarely indicated. Oxygen 4 L/min or to keep SaO_2 above 90% should be used in patients who continue to be acutely ill. Dexamethasone should be continued in symptomatic patients.

Additional Resource

1. Kale R (2012). Altitude-Related Disorders. [online] Available from http://www.emedicine.com/med/TOPIC3225.HTM [Accessed April, 2012].

25. ACQUIRED EXOTROPIA

General

Exodeviation characterized by visual axes that form a divergent angle. Intermittent exotropia, sensory exotropia and exotropia with neurologic causes are all types of acquired exotropia. More common in Middle East, Africa and Asia.

Clinical

Neurological issues.

Ocular

Exophoria, intermittent exotropia, diplopia, bitemporal suppression, asthenopia, and closing of one eye in bright light, amblyopia.

Laboratory

Diagnosis is made by clinical findings.

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- The latest edition of this book includes more than 1,600 entries related to different ocular syndromes and systemic diseases.



Frederick Hampton Roy is in private practice of ophthalmology, specializing in cataract surgery. He is highly regarded for his research and writing in the field of ophthalmology, having published nearly 136 articles, 10 book chapters and 41 books, as well as pamphlets, filmstrips, and other educational materials. In addition, he played a leading role in the formation of the World Eye Foundation, a nonprofit organization established to foster eye care in the Third World. He has traveled, taught, and lectured on the eye care practices, throughout the world including India, China, Egypt, Bolivia, Argentina, and many other countries.

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