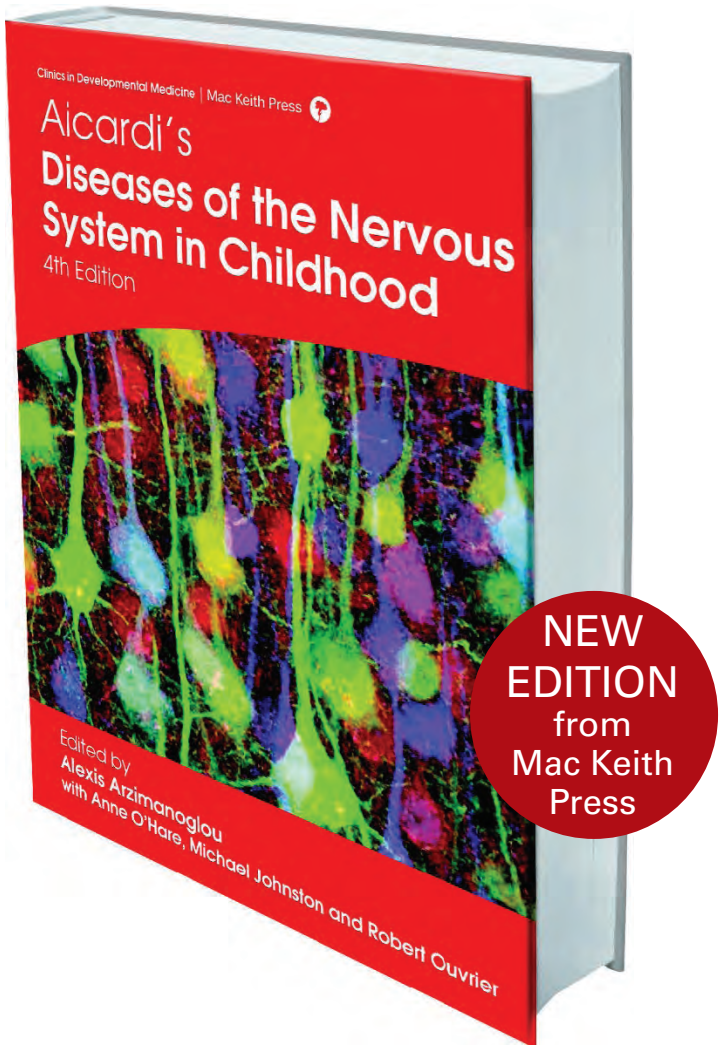
Aicardi's
Diseases of the Nervous System in Childhood
4th Edition

Edited by **Alexis Arzimanoglou with Anne O'Hare, Michael Johnston and Robert Ouvrier**



Includes sample material

For more information visit www.mackeith.co.uk

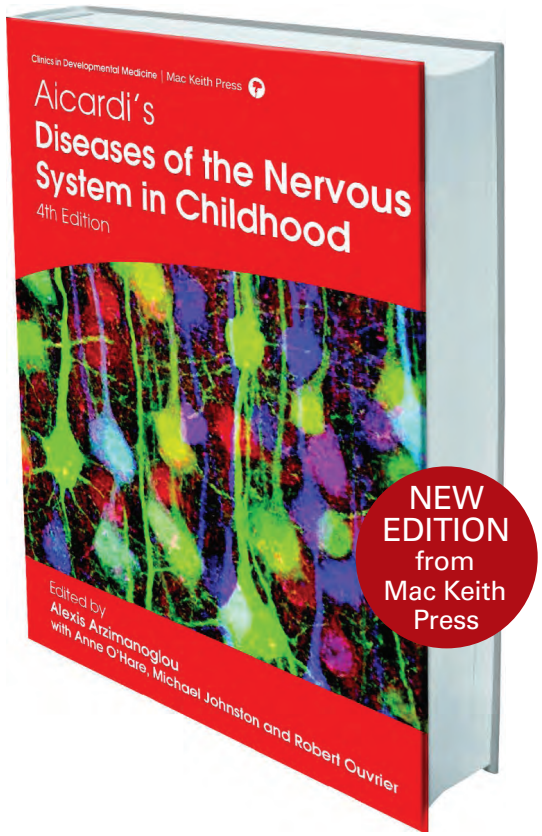
Aicardi's
Diseases of the Nervous System in Childhood
4th Edition

Edited by **Alexis Arzimanoglou**
with Anne O'Hare, Michael Johnston and Robert Ouvrier

The Premier Textbook of Clinical Child Neurology

This classic text on paediatric neurology is now in its fourth edition. Leading academics from around the world present evidence from their various areas of expertise, placing it firmly in the clinical context. Each chapter covers the basic science of the disorder and relevant genetic advances. New to this edition are chapters on fetal neurology, movement disorders, basal ganglia disease and psychogenic disorders. References at the end of each chapter are thorough, guiding the clinician to relevant further reading. The international team of editors has maintained the clinical approach of the late Jean Aicardi, which distinguishes this book from others in the field. Succinctly given, patient-focused information makes *Aicardi's Diseases of Nervous System in Childhood* a comprehensive yet readable resource for busy clinicians in paediatric neurology, general paediatrics, neurodisability and all the related medical disciplines.

- Covers large areas of paediatric neurology
- Patient-focussed with succinct clinical information
- Pertinent chapter references for further reading



New for the 4th Edition

NEW Completely revised and updated
NEW International team of expert editors
NEW Updates on genetic advances
NEW Sections on Fetal Neurology, Basal Ganglia and Movement Disorders
NEW Easy to use presentation
NEW Full colour illustrations throughout

Clinics in Developmental Medicine Series

June 2017 / Hardback / 928 pp/ 280 x205mm, 4-colour / ISBN 978-1-909962-80-4 / £199.95

Meet the Editors behind this updated and revised new edition

Professor Alexis Arzimanoglou is the Director of the Department of Paediatric Clinical Epileptology, Sleep Disorders and Functional Neurology at the University Hospitals of Lyon, France and Visiting Professor at the Universitat de Barcelona, Spain, coordinating the Epilepsy Research Program at the Hospital San Juan de Dios. He graduated from the Salonica University, Greece, trained in Neurology at Great Ormond Street, London, UK and at the Hôpital de la Salpetriere and in Child Neurology as a fellow of Jean Aicardi at the Hôpital des Enfants Malades in Paris, France. He then with worked with Jean Aicardi for over 25 years. He served as: Chair of the Scientific Committee of the European Paediatric Neurology Society; Editor-in-Chief of the International League Against Epilepsy (IALE) educational journal *Epileptic Disorders*; Elected member of the European Commission of the ILAE. Together with Jean Aicardi and Renzo Guerrini he authored *Aicardi's Epilepsy in Children*. He is the editor of seven books and an author or co-author of over one hundred scientific articles in the fields of cognition, medical and surgical treatment of childhood epilepsies.

Professor Anne O'Hare is Professor of Community Paediatrics and Director of the Salvesen Mindroom Centre for Learning Difficulties, at the University of Edinburgh. She is a developmental paediatrician with extensive clinical experience in neurodisability, neuroscience and child protection. Her research interests include how neurodevelopmental conditions impact on the development of speech, language, communication, motor skills and learning and the development of effective interventions.

Professor Michael V Johnston is a Professor of Neurology, Pediatrics and Physical Medicine and Rehabilitation at the Johns Hopkins University School of Medicine and the Chief Medical Officer and the Blum Moser Endowed Professor of Pediatric Neurology at the Kennedy Krieger Institute in Baltimore, Maryland, USA. He trained in pediatrics, neurology and neuroscience at Johns Hopkins, and his clinical and research interests include fetal and neonatal neurology, as well as care for older children with cerebral palsy and neurogenetic disorders including Rett syndrome. He has been active in development of strategies to protect the developing brain from hypoxic-ischemic injury. He is one of the founding faculty members of the Neurosciences Intensive Care Nursery (NICN) research and clinical care group at Johns Hopkins Hospital, and he has also been a leader in of the Phelps Cerebral Palsy Center at Kennedy Krieger.

Professor Robert Ouvrier is the Emeritus Professor of child neurology in the University of Sydney. After training in general paediatrics in Sydney, Perth and Papua-New Guinea, he undertook specialist training in child neurology at the Royal Children's Hospital, Melbourne, the University of Kentucky (1969-70) and the Johns Hopkins Hospital, Baltimore USA (1971-72). He was then Head of the Department of Neurology at the Children's Hospital at Westmead, Sydney for 25 years. In 1999, he became the Foundation Head of the Institute for Neuroscience and Muscle Research at The Children's Hospital, Westmead. He was President of the International Child Neurology Association from 2006-2010. He is the author of two books, thirty book chapters and an author or co-author of over 150 scientific articles on paediatric neurology.

Table of Contents

AUTHORS' APPOINTMENTS

Alexis Arzimanoglou	Director of the Department of Paediatric Clinical Epileptology, Sleep Disorders and Functional Neurology at the University Hospitals of Lyon, France; Visiting Professor at the Universitat de Barcelona, Spain
Nadia Bahi-Buisson	Consultant, Neurologie Unité, Necker-Enfants Malades, Paris, France
Alasdair Bamford	Paediatric Infectious Diseases Consultant, Great Ormond Street Hospital, London, UK
Karen Barlow	Cumming School of Medicine, University of Calgary
Peter Baxter	Sheffield Children's Hospital, Sheffield, UK
Nathalie Boddaert	Head of department, Pediatric Radiology, Necker-Enfants Malades, Paris, France
Aabir Chakraborty	Lead for Paediatric Neurosurgery, Southampton General Hospital, Southampton, UK
Russell Dale	Petre Associate Professor Paediatric Neurology Research, Paediatrics & Child Health, Children's Hospital, University of Sydney, Westmead, Australia
Bernard Dan	Professor of Neuroscience, Université libre de Bruxelles (ULB), Brussels; Director of Rehabilitation, Rehabilitation Hospital Inkendaal, Vlezenbeek, Belgium
Linda De Meirleir	Head of Pediatric Neurology and Metabolic Diseases and Medical Coordinator for Rare Disorders, Universitair Ziekenhuis Brussel, Brussels, Belgium
Linda S de Vries	Professor in Neonatal Neurology, Department of Neonatology, UMCU, Utrecht, the Netherlands
Gabrielle deVeber	Professor, Alberta Children's Hospital Research Institute, University of Calgary, Canada
Michael S. Duchowny	Emeritus Director, Comprehensive Epilepsy Program, Nicklaus Children's Hospital, Miami, Florida, USA
Adré J Du Plessis	Chief, Division of Fetal and Transitional Medicine; Director, Fetal Medicine Institute, Children's National Medical Center, George Washington University School of Medicine, Washington DC, USA
Michael Eyre	Specialty Registrar in Paediatric Neurology, Great Ormond Street Hospital, London, UK
Emilio Fernandez-Alvarez	Hospital Sant Joan de Deu Servico de Neuropediatria, Barcelona, Spain
Robert Forsyth	NHS Consultant Paediatric Neurologist, Great North Children's Hospital; Senior Lecturer, Newcastle University, Newcastle, UK
Patricia Franco	Pediatric Sleep Unit & INSERM U1028, Mother-Children Hospital, University of Lyon 1, France
Paddy Grattan-Smith	Clinical Associate Professor, Paediatrics & Child Health, Children's Hospital, Westmead, Sydney, Australia
Richard Hayward	Professor, Paediatric Neurosurgery, Great Ormond Street Hospital for Children, NHS Foundation Trust, London, UK
Cheryl Hemingway	Paediatric Neurology Consultant, Great Ormond Street Hospital, London, UK
Michael V Johnston	Chief Medical Officer and Blum Moser Endowed Chair for Pediatric Neurology, Kennedy Krieger Institute; Professor of Neurology, Pediatrics and Physical Medicine and Rehabilitation, Johns Hopkins University School of Medicine, Baltimore, Maryland, USA
Colin Kennedy	Professor in Neurology and Paediatrics, University of Southampton, UK
Adam Kirton	Associate Professor, Pediatrics and Clinical Neurosciences Cumming School of Medicine, University of Calgary, Alberta Children's Hospital Research Institute (ACHRI); Director, Calgary Pediatric Stroke Program Pediatric Neurologist, Alberta Children's Hospital, Calgary, Alberta, Canada
Ingeborg Krageloh-Mann	Professor of Paediatrics; Director Paediatric Neurology and Developmental Medicine, University Children's Hospital, Tübingen, Germany
Kenneth J Mack	Mayo Clinic, Child and Adolescent Neurology, Rochester, Minnesota, USA

SAMPLE MATERIAL FROM CHAPTER 6

Craniosynostosis/ Paediatric Craniovertebral Anomalies

Richard Hayward FRCS

INTRODUCTION

Craniosynostosis is defined here as the premature closure of one or more of the skull vault sutures. While advances in molecular genetics have revolutionised our understanding of the various syndromes that may include craniosynostosis, and improved imaging techniques have provided new information about not only the calvarial sutures but also changes affecting the skull base and facial skeletons, the initial diagnosis (or more accurately the initial suspicion) of craniosynostosis still depends primarily on the patient's – usually a child's – appearance.

The aim of this chapter is to provide for the paediatric neurologist a broad overview of this complex subject. For convenience craniosynostosis affecting a single vault suture (sometimes referred to as 'simple' synostosis) will be dealt with separately from the various complex/syndromic forms in which premature closure of several – sometimes all – of the skull sutures is usual although overlap between the two groups is not uncommon. Johnson and Wilkie have provided a useful overview from a craniofacial surgeon and geneticist's perspective (Johnson and Wilkie 2011).

AETIOLOGY OF CRANIOSYNOSTOSIS

The various causes of craniosynostosis (or conditions with which it may be associated) can be broadly classified as shown in Table 6.1.

Single Suture Synostosis

The direct consequences of premature fusion of a single skull vault suture are for the most part cosmetic – the result of a usually characteristic alteration in head shape. Effects on the facial skeleton, if any, are modest and severe functional consequences affecting vision, breathing and feeding, for example, are rare. This is in marked contrast to the complex/syndromic forms of craniosynostosis in which such issues are common.

Table 6.1

Primary	Idiopathic	No genetic cause either known or suspected[a]
	Genetic cause known or suspected	Gene mutation[b]
		Chromosomal abnormality[c]
Secondary	Metabolic:	Vitamin D deficient rickets
		Hyperthyroidism
	Disorders of bone metabolism	Cranio-meta/diaphyseal dysplasia
		Osteopetrosis
	Storage disorders	Hurler syndrome
		Morquio syndrome
	Drug induced:	Metopic synostosis, as part of the fetal valproate syndrome
	Physical distortion:	Post-CSF shunting positional scaphocephaly

[a]The majority of single suture synostosis fall into this category – in particular those affecting the sagittal and metopic sutures.
[b]This includes not only the syndromes once described eponymously (Crouzon, Apert etc.) but also many previously labelled as non-syndromic that have now had their underlying gene mutation mapped.
[c]The metopic suture appears to be the most vulnerable when craniosynostosis forms part of a chromosomal abnormality – a deletion of part of the short arm of 7, for example.
CSF, cerebrospinal fluid

Aetiology

While the majority of cases of single suture synostosis arise as isolated events the possible genetic implications of the diagnosis should not be overlooked particularly for unicoronal synostosis (Moloney et al. 1997) (see Unicoronal [and Fronto-Sphenoidal] Synostosis). Whereas no common candidate genes are presently known for isolated sagittal and metopic synostosis, it is essential that all children with unicoronal synostosis are referred for evaluation by a geneticist (Johnson and Wilkie 2011).

infection, for example) never absent from what is never a minor cranial operation.

The various surgical interventions (and their timing) currently employed in the management of each single suture synostosis are here briefly described. For a more detailed summary, see the review by Garza and Khosla (2012).

Sagittal Synostosis

Premature closure of the sagittal suture is the most frequent form of craniosynostosis and leads to a characteristic scaphocephalic (boat-shaped) deformity of the skull. A prevalence of approximately 1 in 5 000 children has been estimated and the condition is more frequently seen in boys. Six per cent of cases are familial with transmission following an autosomal dominant pattern with a penetrance of 38% (Lajeunie et al. 1996).

The affected skull has an increased antero-posterior diameter, its bi-parietal diameter is reduced (Fig. 6.1) and a bony ridge can often be both seen and felt along the line of the fused suture. Victims of teasing may be called 'peanut head'. The synostotic process does not always involve the entire suture and even when it does the severity with which the child's head shape is affected is very variable, with a mild prominence of the forehead at one end of the spectrum to gross elongation (frontal and occipital bossing) plus narrowing (particularly in the pterional regions) at the other.

The variety of surgical treatments presently employed for the correction of sagittal synostosis (for those children whose parents have opted for intervention) suggests either that all are equally effective – or equally non-effective! The operations vary in scale from removal of the fused suture (suturectomy) combined with internal springs (de Jong et al. 2013) or external (helmet/orthosis) manoeuvres (Proctor 2012) designed to induce a more round shape (all of which need to be performed before six months of age to be most effective) to increasingly major forms of skull reconstruction for which there are no age limits.

Unicoronal (and Fronto-Sphenoidal) Synostosis

Craniosynostosis of a single coronal suture produces a characteristic asymmetry of the forehead: frontal plagiocephaly. The supra-orbital ridge on the affected side is recessed as is the forehead above while the temporal region is unusually prominent. On the contralateral side the frontal region is often bossed, accentuating the asymmetry and the nose is set an angle, its root 'pointing' towards the side of the affected suture. The net result is to give the face a characteristic 'scoliosis' or curve convex to the affected side. The anterior skull base is also curved – but concave to the affected side (Fig. 6.2).

The elevation of the lateral wing of the sphenoid bone on the affected side is responsible for the characteristic 'harlequin eye' appearance on an antero-posterior skull X-ray. The deformation of the orbit results in a subtle malposition of the extra-occular muscle attachments that may in turn cause a complex abnormality of eye movement and a secondary compensatory head tilt (Gosain et al. 1996). All children with unicoronal synostosis should therefore be referred to a paediatric ophthalmologist.

Although the cause of unicoronal synostosis is in many cases unknown it can result from a variety of genetically mediated disorders. Most prominent amongst these is the Muenke (or FGFR3-associated synostosis) (Muenke et al. 1997). Saethre–Chotzen syndrome (Reardon and Winter 1994) and craniofrontonasal dysplasia (Cohen, Jr 1979) may also involve premature closure of a single coronal suture but their other features are usually sufficiently characteristic to suggest the diagnosis.

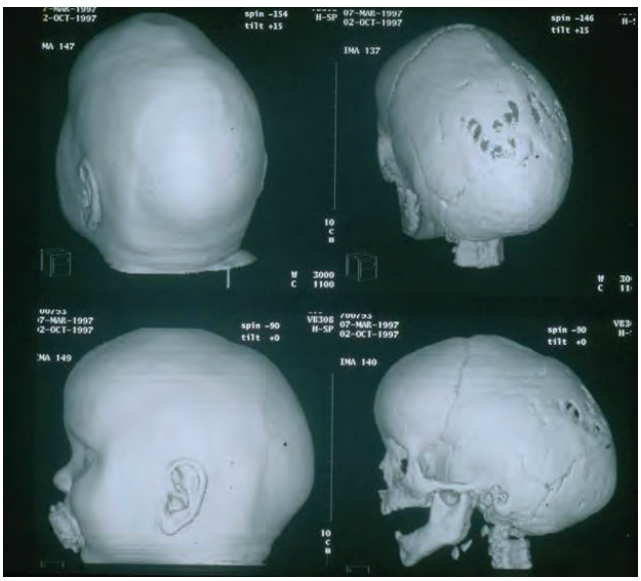


Figure 6.1 Three-dimensional CT scans showing the typical scaphocephalic head shape caused by sagittal synostosis.

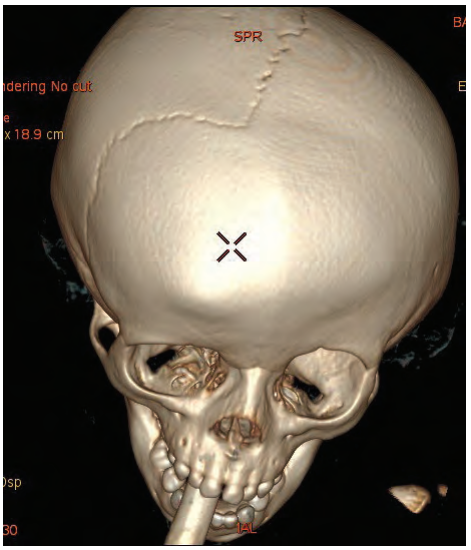


Figure 6.2 Three-dimensional CT scan showing the typical deformity caused by unicoronal synostosis.

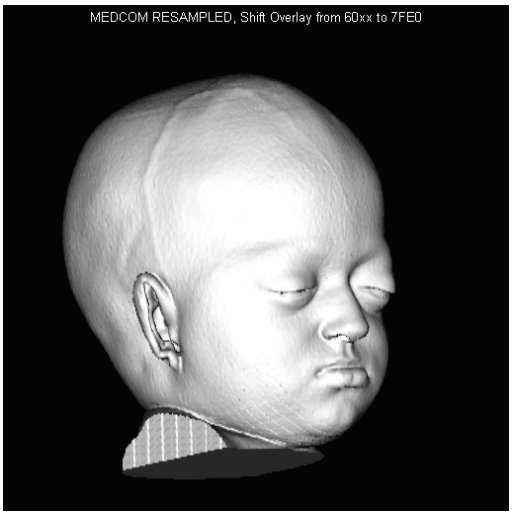


Figure 6.5 Three-dimensional CT of a child with Crouzon syndrome showing the typical facial appearance. Note also the enlarged scalp veins suggesting raised intracranial pressure secondary to intracranial venous hypertension.

developmental and learning difficulties. Marked intellectual compromise was present in 3% of Kreiborg's series (Kreiborg 1981).

Apert Syndrome

The child with Apert syndrome has a head that is tall and shortened from front to back (turri-brachycephaly), midfacial (maxillary) retrusion, proptosis, a downward cant to the palpebral fissures and hypertelorism (Lajeunie et al. 1999) (Fig. 6.6a). The essential clinical feature however is a complex fusion (syndactyly) of the fingers and toes (Anderson et al. 1997d, Anderson et al. 1999, Cohen and Kreiborg 1995b) that may require multiple surgical procedures before functional effectiveness is achieved (Guero et al. 2004) (Fig. 6.6b). Visceral (Cohen and Kreiborg 1993) and cutaneous (Cohen and Kreiborg 1995a) abnormalities can also occur. Palatal abnormalities (Kreiborg and Cohen 1992) ranging in severity from frank clefts to a bifid uvula are common and occur with a frequency of up to 75% (Peterson and Pruzansky 1974, Slaney et al. 1996). Cervical vertebral fusions that may be progressive occur in over half of affected children although it is unusual for them to become clinically significant (Thompson et al. 1996).

Developmental and learning difficulties are the norm in Apert syndrome although a combination of developmental assessment tools designed for non-Apert children and low societal expectations may overestimate their severity (Shipster et al. 2002). While a small percentage of children may complete secondary education (and usually only with assistance in the classroom), many drop out of mainstream education during their primary school years while a small percentage are too affected for the mainstream education system at anything above kindergarten level (Patton et al. 1988, Renier et al. 1996).

Pfeiffer Syndrome

Although described separately for historical reasons, the genetic overlap between Pfeiffer and Crouzon syndromes is such that they are now often considered together as 'Crouzon–Pfeiffer syndrome'.

The 'traditional' Pfeiffer syndrome is an autosomal dominant condition characterised by suture fusions that range from bicoronal synostosis alone to pan-synostosis (with or without the cloverleaf skull deformity – see next section) (Winter 1994). Affected patients also have digital abnormalities (Panthaki and Armstrong 2003) that include curved and shortened thumbs and great toes (Anderson et al. 1998b) and, less commonly, digital fusions (although to a lesser degree than in Apert syndrome [Panthaki and Armstrong 2003]).

Cohen (1993) divided children with Pfeiffer syndrome into three types based on their clinical severity. Type 1, those least affected, may display little more than bicoronal synostosis and midface retrusion (in addition to their digital

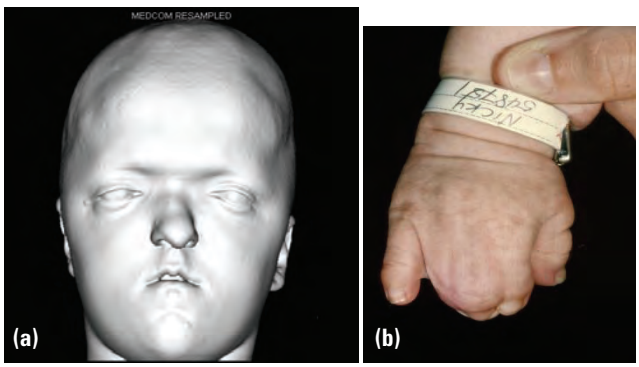


Figure 6.6 (a) Apert syndrome. Syndactyly. (b) Three-dimensional CT showing the typical facial features.

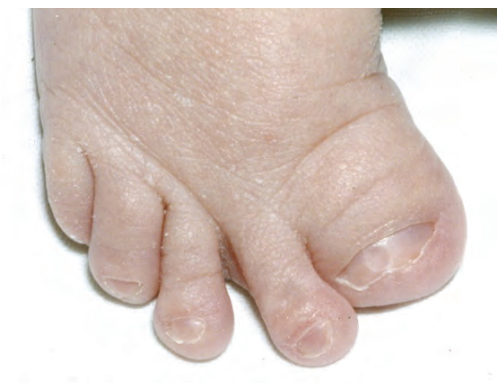


Figure 6.7 The typical hallux deformity of Pfeiffer syndrome.

ed chromosomal abnormalities. It may also occur as a post-operative complication, a consequence of the frontal lobe retraction required during fronto-facial monobloc and bipartition procedures in children with severe frontal bone recession (Cobb et al. 2013).

Cosmesis

The cosmetic disabilities that most trouble patients with syndromic synostosis and their families include a misshapen forehead, eyes that protrude, eyes set too far apart (hypertelorism) and an upper jaw set back while the lower jaw protrudes.

When correcting for cosmesis alone it is important to remember that surgery carried out on a part of the craniofacial skeleton that is still growing may need to be repeated either wholly or in part in order to achieve a result that will prove stable over time. Our own policy, based more on clinical observation than measurement, is to assume that a forehead and supra-orbital region in a satisfactory configuration at around 10 years of age is unlikely to need further correction and essentially cosmetic reconstructions after that age can focus more on the maxilla and mandible where growth will continue until secondary dentition is complete – the mid to late teens.

CONCLUSION

Primary craniosynostosis whether it affects one or multiple sutures and is associated or not with a particular syndrome is rare and its management should only be undertaken by a unit with sufficient experience to ensure affected children achieve their developmental potential.

Early assessment by such a unit will enable the correct diagnosis (both genetic and clinical) to be made, the risk of complications assessed and a management plan made that is tailored to each individual child's needs.

While in non-syndromic unisutural synostosis treatment may require no more than a single reconstructive operation, more complex cases require input from a wide range of specialists including the paediatric neurologist often until the completion of skeletal maturity.

REFERENCES

Abou-Sleiman PM, Apessos A, Harper JC, Serhal P, Delhanty JD (2002) Pregnancy following preimplantation genetic diagnosis for Crouzon syndrome. *Mol Hum Reprod* **8**: 304–9.

Agochukwu NB, Solomon BD, Gropman AL, Muenke M (2012) Epilepsy in Muenke syndrome: FGFR3-related craniosynostosis. *Pediatr Neurol* **47**: 355–61.

Ahmed J, Marucci D, Cochrane L, Heywood RL, Wyatt ME, Leighton SE (2008) The role of the nasopharyngeal airway for obstructive sleep apnoea in syndromic craniosynostosis. *J Craniofac Surg* **19**: 659–63.

Alden TD, Lin KY, Jane JA (1999) Mechanisms of premature closure of cranial sutures. *Childs Nerv Syst* **15**: 670–5.

Allam KA, Wan DC, Khwanngern K, et al. (2011) Treatment of apert syndrome: A long-term follow-up study. *Plast Reconstr Surg* **127**: 1601–11.

Anderson PJ (1997) Crouzon syndrome: Anomalies not limited to the skull and the face. *J Craniomaxillofac Surg* **25**: 356–7.

Anderson PJ, Hall C, Evans RD, Harkness WJ, Hayward RD, Jones BM (1997a) The cervical spine in Crouzon syndrome. *Spine* **22**: 402–5.

Anderson PJ, Hall CM, Evans RD, Hayward RD, Harkness WJ, Jones BM (1997b) The cervical spine in Saethre-Chotzen syndrome. *Cleft Palate Craniofac J* **34**: 79–82.

Anderson PJ, Hall CM, Evans RD, Hayward RD, Jones BM (1996a) The hands in Saethre-Chotzen syndrome. *J Craniofac Genet Dev Biol* **16**: 228–33.

Anderson PJ, Hall CM, Evans RD, Hayward RD, Jones BM (1998a) The elbow in syndromic craniosynostosis. *J Craniofac Surg* **9**: 201–6.

Anderson PJ, Hall CM, Evans RD, Hayward RD, Jones BM (1999) The feet in Apert's syndrome. *J Pediatr Orthop* **19**: 504–7.

Anderson PJ, Hall CM, Evans RD, Jones BM, Harkness W, Hayward RD (1996b) Cervical spine in Pfeiffer's syndrome. *J Craniofac Surg* **7**: 275–9.

Anderson PJ, Hall CM, Evans RD, Jones BM, Hayward RD (1998b) The feet in Pfeiffer's syndrome. *J Craniofac Surg* **9**: 83–7.

Anderson PJ, Harkness WJ, Taylor W, Jones BM, Hayward RD (1997c) Anomalous venous drainage in a case of non-syndromic craniosynostosis. *Childs Nerv Syst* **13**: 97–100.

Anderson PJ, Smith PJ, Jones BM (1997d) New classification for the hand anomalies in Apert's syndrome. *J Hand Surg Br* **22**: 140–1.

Antley R, Bixler D (1975) Trapezoidocephaly, midfacial hypoplasia and cartilage abnormalities with multiple synostoses and skeletal fractures. *Birth Defects Orig Artic Ser* **11**: 397–401.

Bellus GA, Gaudenz K, Zackai EH, et al. (1996) Identical mutations in three different fibroblast growth factor receptor genes in autosomal dominant craniosynostosis syndromes. *Nat Genet* **14**: 174–6.

Bottero L, Lajeunie E, Arnaud E, Marchac D, Renier D (1998) Functional outcome after surgery for trigonocephaly. *Plast Reconstr Surg* **102**: 952–8.

Bradley JP, Kawamoto HK, Taub P, Wexler A, Cahan L (2003) Antley-Bixler syndrome: Correction of facial deformities and long-term survival. *Plast Reconstr Surg* **111**: 1454–60.

Britto JA, Chan JC, Evans RD, Hayward RD, Jones BM (2001a) Differential expression of fibroblast growth factor receptors in human digital development suggests common pathogenesis in complex acrosyndactyly and craniosynostosis. *Plast Reconstr Surg* **107**: 1331–8.

Britto JA, Evans RD, Hayward RD, Jones BM (1998) Maxillary distraction osteogenesis in Pfeiffer's syndrome: urgent ocular protection by gradual midfacial skeletal advancement. *Br J Plast Surg* **51**: 343–49.

Britto JA, Evans RD, Hayward RD, Jones BM (2001b) From genotype to phenotype: the differential expression of FGF, FGFR, and TGFbeta genes characterizes human cranioskeletal development and reflects clinical presentation in FGFR syndromes. *Plast Reconstr Surg* **108**: 2026–39.

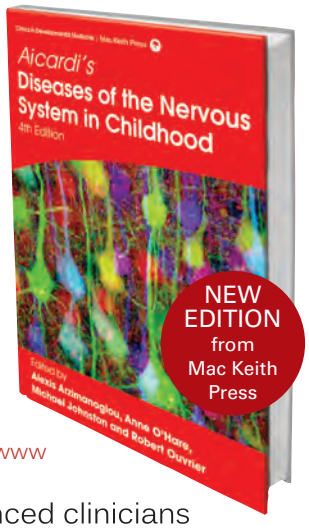
Britto JA, Moore RL, Evans RD, Hayward RD, Jones BM (2001c) Negative autoregulation of fibroblast growth factor receptor 2 expression characterizing cranial development in cases of Apert (P253R mutation) and Pfeiffer (C278F mutation) syndromes and suggesting a basis for differences in their cranial phenotypes. *J Neurosurg* **95**: 660–73.

Mac Keith Press

The leading source of publications on child neurodisability and developmental medicine

www.mackeith.co.uk

Aicardi's Diseases of the Nervous System in Childhood



4th Edition

Edited by Alexis Arzimanoglou
with Anne O'Hare, Michael Johnston and Robert Ouvrier

New edition completely updated and revised. Now in full colour.

Contact us at admin@mackeith.co.uk to receive further details.

June 2017 / Hardback / 928 pp / 280 x 205mm, 4-colour / ISBN 978-1-909962-80-4 / £199.95www

- Patient-focussed with succinct clinical information from experienced clinicians
- Compiled by a new international team of expert editors
- Includes updates on genetic advances
- New sections on Fetal Neurology, Basal Ganglia and Movement Disorders

Purchases can be made at our website or by using the order form below

www.mackeith.co.uk

I WOULD LIKE TO PURCHASE _____ COPIES OF Aicardi's Diseases of the Nervous System in Childhood, 4th edn

NAME	CARD NUMBER (VISA/ MASTERCARD) _____
BILLING ADDRESS	EXPIRY DATE _____ SECURITY CODE _____
SHIPPING ADDRESS (IF DIFFERENT)	SIGNATURE
CONTACT DETAILS IN CASE OF QUERY: TELEPHONE _____ EMAIL _____	PRICE PER BOOK £ 199.95 SHIPPING FREE TOTAL £ _____ Payment will be taken in £ sterling and will appear on your card statement at the exchange rate applied by your card company.

PLEASE TICK HERE IF YOU WOULD LIKE TO JOIN OUR MAILING LIST TO RECEIVE MARKETING INFORMATION FROM MAC KEITH PRESS ☐

Please return completed forms to:

Mac Keith Press, 6 Market Road, London N7 9PW, UK T +44 (0) 20 7619 7320 F +44 (0) 20 7619 7207 E admin@mackeith.co.uk