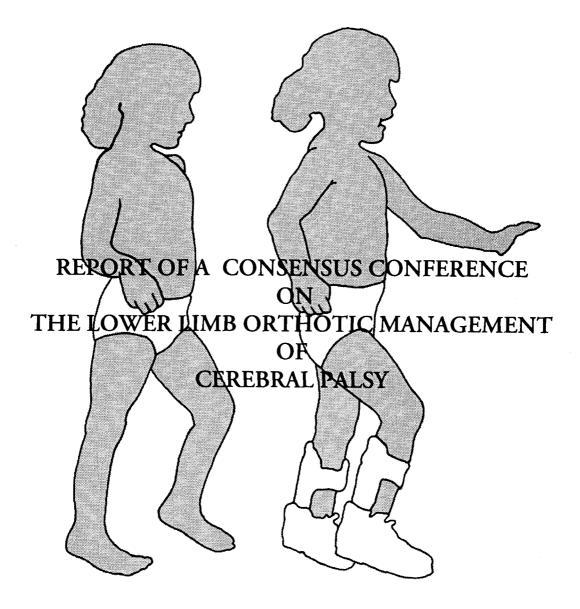


INTERNATIONAL SOCIETY FOR PROSTHETICS AND ORTHOTICS



Held at Duke University, Durham, N. Carolina 10 - 12 November, 1994

> EDITED BY DAVID N. CONDIE AND C. BARRY MEADOWS

Published in 1995

by

The International Society for Prosthetics and Orthotics Borgervaenget 7, 2100 Copenhagen -0-, Denmark

ISBN 87-89809-02-5



INTERNATIONAL SOCIETY FOR PROSTHETICS AND ORTHOTICS

REPORT OF A CONSENSUS CONFERENCE ON THE LOWER LIMB ORTHOTIC MANAGEMENT OF CEREBRAL PALSY

Held at Duke University, Durham, N. Carolina 10 - 12 November, 1994

> EDITED BY DAVID N. CONDIE AND C. BARRY MEADOWS

.

CONTENTS

PART 1

THE CONFERENCE ORGANISATION AND OUTCOME

Background	1
Participants	3
Programme	6
Bibliography	8
Conclusions and Recommendations	15

<u>PART 2</u>

THE PRESENTATIONS

	Aspects of the Cerebral Palsies elevant to the Orthotic Management of r Limbs		B Neville D Scrutton and K Richardson	21
The Aims	of Lower Extremity Orthotic Treatment in Cere	bral	W Stuberg	27
Palsy: A	Critical Review of the Literature			
The Scien	tific Basis of Treatment			
IJ	To Prevent or Control Deformity		I Ziv and L Cardomone	35
	Fo Provide a Base of Support and to Facilitate Learning		J Kluzik	47
III 1	To Improve the Dynamic Efficiency of Gait		C B Meadows	57
How do T	Therapists Achieve these Aims?		R Boyd	66
How do Surgeons Achieve these Aims?				
		I П	J R Fisk G A Carter	83
		11	G A Carter	88
	iew of Components and Concepts Involved c Prescription for Children with Cerebral Palsy		B Cusick	94

Current Orthotic Practise

Ι	The Orthotic Management of the Foot in Cerebral Palsy	G Small	123
п	The Orthotic Management of Deformity in Cerebral Palsy	C J Drake	127
III	Orthotic Management of the CP Child	R S Lin	137
IV	The Use of Orthotics to Improve the Dynamic Efficiency of Gait	T Supan	140
V	Gait Related Orthotic Prescription Criteria for Children with Cerebral Palsy	D Weber	145
Biom	echanical Considerations		
I	Current Orthotic Practise in Relation to the Improvement of Gait Efficiency	R E Major	175
Ш	Biomechanics of Orthotic Management of Gait in Spastic Diplegia	D L Vaughan W E Carlson D L Damiano and M F Abel	181
	ntegration of Orthotic Treatment in an Il Management Programme	J Becher	192

PART 1

ORGANIZATION AND OUTCOME

BACKGROUND

The past few decades have seen significant advances in the treatment of both children and adults with cerebral palsy. During this period several "schools" of thought and clinical practise have emerged, espousing a wide range of views of varying degrees of compatibility or conflict. Sometimes these views have been very deeply held, even although based largely on clinical experience rather than scientific evidence.

Throughout this period opinion on the use of orthoses with this group of patients has varied, with those in favour tending to come from the more "mechanical" or orthopaedic approach and those opposed coming from the primarily neurological approach.

Fortunately it would appear that in recent years there has been some degree of convergence of views, presumably as a result of the generally improved level of knowledge of practitioners of all disciplines in this area of clinical practise. For example today's orthotic practitioners are much more aware of the broader neuromuscular issues and the potential roles of orthoses in the overall management programme. In addition the development of a range of new and improved designs of orthoses, mostly fabricated in plastic, has greatly enhanced the quality of control which it is possible to provide orthotically.

In spite of this welcome trend there is still considerable divergence of opinion as to precisely how and when these orthoses should be used. It was for this reason that the Executive Board of ISPO decided to convene a Consensus Conference on this topic.

The Board entrusted me with the task of organizing the Conference and to assist me I recruited a small team of UK experts in this field, comprising Barry Meadows, a bioengineer from Glasgow and Chris Drake, an orthotist and David Scrutton, a physiotherapist, both from London.

After considerable discussion, this team formulated a list of what we considered were the "Aims or Objectives of Orthotic Treatment" for this category of patient. These were:

- To prevent and/or correct deformity
- To provide a base of support
- To facilitate training in skills
- To improve the efficiency of gait

The Conference programme was then designed to examine systematically both the published evidence and the clinical experience of the use of orthoses to achieve each of these aims.

Invited experts from all the involved disciplines, physicians, surgeons, therapists, orthotists and bioengineers, eighteen in total, were asked to prepare and present "review papers" dealing with a specified aspect of the subject.

1

To assist them with this task a selected bibliography of recent journal articles and books dealing specifically with the lower limb <u>orthotic</u> management of cerebral palsy was compiled by Ms Heather Smart, the Librarian at the National Centre for Training and Education in Prosthetics and Orthotics at the University of Strathclyde, and a full set of copies of the journal articles was dispatched to each reviewer.

The meeting was held in the Washington Duke Inn and Country Club within the campus of Duke University, Durham, N Carolina. The on-site arrangements for the meeting and the technical and secretarial support were the responsibility of the staff of the Department of Prosthetics and Orthotics of The Duke University Medical Centre under their Head of Department, Michael Schuch.

An "audience" of a further 24 invited experts joined with the reviewers to debate each of the review papers with the reviewer immediately after its presentation.

A small group of the participants, comprising Diane Taillon, Terry Supan, George Carter and myself was responsible for identifying the key issues arising from these debates which were then further examined in Syndicate Groups before being debated once again in a subsequent plenary session.

The entire event lasted three very full days and was very demanding, both physically and intellectually, but also absolutely fascinating.

The conclusions and recommendations of the Conference were drafted by me, based on the transcripts of the Syndicate Reports and the further discussions, and were subsequently submitted to every participant for their comment and approval. They can, therefore, truly be described as representing the agreed views of a multidisciplinary group of practitioners who are extremely knowledgeable and very experienced in the management of cerebral palsy.

The Executive Board of ISPO has decided that the review papers prepared for the meeting represent such a valuable, not to say unique, body of knowledge that they should be published in their entirety along with this report of the organisation and outcome of the meeting.

The publication of this report represents the culmination of a two year period of intensive activity encompassing the planning and organisation of the meeting, the event itself and the follow-up from it. The conclusions and recommendations do, however, include proposals for further initiatives by ISPO, intended to advance the quality of clinical care for this group of patients generally and to improve our understanding of the functions and roles of orthoses specifically.

It is my sincere hope that both ISPO and the practitioners who so diligently applied themselves to the tasks they were set during the Conference will rise to the further challenges offered by these proposals.

David N Condie August, 1995

LIST OF ATTENDEES

DR K SKOW ANDERSEN DEPARTMENT OF ORTHOPAEDIC SURGERY HERLEV UNIVERSITY HOSPITAL HERLEV RINGVEJ 75 2730 HERLEV DENMARK

PROF. DR J U BAUMANN LEITENDEK ARZT NEURO-ORTHOPAEDIE ANGENSTEINERSTRASSE 10 CH-4052 BASEL SWITZERLAND

DR J BECHER DEPARTMENT OF REHABILITATION MEDICINE FREE UNIVERSITY HOSPITAL PO BOX 7057 1007 MB AMSTERDAM NETHERLANDS

MS ROS BOYD NEWCOMEN CENTRE GUY'S HOSPITAL ST THOMAS STREET LONDON SE1 9RT ENGLAND

DR GEORGE CARTER 24 MAY STREET FREMANTLE 6158 WESTERN AUSTRALIA AUSTRALIA MR MICHAEL DeLACEY LOT 7 34-37 BLUFF ROAD CEDAR VALE QUEENSLAND 4285 AUSTRALIA

MR TOM DI BELLO 6910 FANNIN, SUITE 105 HOUSTON TEXAS 77030 USA

MR CHRIS DRAKE REHABILITATION CENTRE QUEEN MARY'S UNIVERSITY HOSPITAL LONDON SW15 5PR ENGLAND

DR JOHN FISK SOUTHERN ILLINOIS UNIVERSITY SCHOOL OF MEDICINE PO BOX 19230, SPRINGFIELD ILLINOIS 62794-9230 USA

MR ROBERT GREIG NATIONAL CENTRE FOR TRAINING & EDUCATION IN PROS/ORTH UNIVERSITY OF STRATHCLYDE CURRAN BUILDING 131 ST JAMES ROAD GLASGOW G4 0LS SCOTLAND

MRS M. ELIZABETH CONDIE NATIONAL CENTRE FOR TRAINING & EDUCATION IN PROSTHETICS & ORTHOTICS UNIVERSITY OF STRATHCLYDE CURRAN BUILDING 131 ST JAMES ROAD GLASGOW G4 0LS SCOTLAND

MS BEVERLY CUSICK PO BOX 62 PLACERVILLE COLORADO 81430 USA MS GRETCHEN HECHT SAWTOOTH ORTHOTICS INC 509 W. HAYS BOISE, IDAHO 83702 USA

MS JOANN KLUZIK DEPT OF PHYSICAL THERAPY BOSTON UNIVERSITY 635 COMMONWEALTH AVENUE BOSTON, MASSACHUSETTS 02115 USA MRS DULSEY LIMA OAKBROOK ORTHOPAEDIC SERVICES FOX VALLEY COMMONS II 4255 WESTBROOK DRIVE # 215 ILLINOIS 60504 USA

MR BOB LIN NEWINGTON CHILDREN'S HOSPITAL 181 E CEDAR STREET NEWINGTON CONNECTICUT 06111 USA

MR RICHARD MAJOR ORTHOTIC RESEARCH AND LOCOMOTOR ASSESSMENT UNIT ROBERT JONES AND AGNES HUNT ORTHOPAEDIC HOSPITAL OSWESTRY SHROPSHIRE SY10 7AG ENGLAND

DR PETER MASSO SHRINERS HOSPITAL 516 CREW STREET SPRINGFIELD MA 01104 USA

DR C BARRY MEADOWS HEALTHCARE INTERNATIONAL (SCOTLAND) LTD BEARDMORE STREET GLASGOW G81 4DY SCOTLAND

MS MARY MILLER C/O CLINICAL ORTHOTICS CONSULTANTS 2601 MATHESON BLVD. E UNIT #10 MISSISSAUGA ONTARIO L4W 5A8 CANADA

MR CHRIS MORRIS NUFFIELD ORTHOTICS AND RESEARCH CENTRE NUFFIELD ORTHOPAEDIC CENTRE NHS TRUST WINDMILL ROAD HEADINGTON OXFORD OX3 7LD ENGLAND DR ADRIANO MURRI STOLZALPE 39 A-8852 STOLZALPE AUSTRIA

PROF. BRIAN NEVILLE INSTITUTE OF CHILD HEALTH UNIVERSITY OF LONDON NEUROSCIENCES UNIT THE WOLFSON CENTRE MECKLENBURGH SQUARE LONDON WC1N 2AP ENGLAND

MR C NOPPE NOPPE ORTHOPEDIE BV VD WEYDENLAAN 2 2211 JK NOORDWIJKERHOUT NETHERLANDS

MR JOHN PATRICK ORLAU ROBERT JONES AND AGNES HUNT ORTHOPAEDIC HOSPITAL OSWESTRY SHROPSHIRE SY10 7AG ENGLAND

DR DAVID PRATT ORTHOTICS & DISABILITY DEPT RHEUMATOLOGY & REHAB DERBYSHIRE ROYAL INFIRMARY LONDON ROAD DERBY DE1 2QY ENGLAND

MR MIKE SCHUCH DUKE UNIVERSITY MEDICAL CTR DURHAM NORTH CAROLINA 22710 USA

MR GREG SMALL 104 EAST BROWN STREET 40 DENTON TEXAS 76208 USA MR BARRY SMITH ORTHOTIC PROSTHETIC SERVICES PTY LTD UNIT 9 LEEDERVILLE VILLAGE 663 NEWCASTLE STREET LEEDERVILLE WESTERN AUSTRALIA 6007 AUSTRALIA

DR WAYNE STUBERG MEYER REHABILITATION INSTITUTE UNIVERSITY OF NEBRASKA MEDICAL CENTRE 600 SOUTH 42ND STREET BOX 985450 OMAHA NEBRASKA 68198-5450 USA

MR TERRY SUPAN O + P SERVICES SOUTHERN ILLINOIS UNIVERSITY SCHOOL OF MEDICINE PO BOX 19230 SPRINGFIELD ILLINOIS 62794 - 9230 USA

MS DIANE TAILLON THE BRACE CENTERS LTD 720 S VAN BUREN, SUITE 104 GREEN BAY WISCONSIN 54301 USA

MR JACQUES VAN ROLLEGHEM INTERBOR 67 RUE DE LAEKEN 1853-STROMBEEK BELGIUM

MR KIT VAUGHAN DEPARTMENT OF BIOMEDICAL ENGINEERING UNIVERSITY OF CAPE TOWN OBSERVATORY CAPE 7925 SOUTH AFRICA

MR DON WEBER CHEDOKE-McMASTER HOSPITALS BOX 2000 STATION A HAMILTON ONTARIO L8N 3Z5 CANADA MS JOAN WEINTROB THE ORTH/ PROS CENTER INC 8330 PROFESSIONAL HILL DRIVE FAIRFAX VIRGINIA 22031 USA

DR ISRAEL ZIV DEPT OF ORTHOPAEDIC SURGERY UNIVERSITY OF BUFFALO STATE UNIVERSITY NEW YORK 162 FARBER HALL 3435 MAIN STREET BUFFALO NEW YORK 14214-2692 USA

PROGRAMME

0900	<u>SDAY 10th NOVEMBER</u> Cerebral Palsy - Aetiology and Presentation		Prof. B Neville
0900	Cerebrai Faisy - Actiology and Fresentation		(London)
0930	Discussion		(Dondon)
0945	The Aims of Orthotic Treatment		Dr W Stuberg
			(Omaha)
1030	Discussion		
1100	COFFEE		
	The Scientific Basis of Treatment		
1130	- To Prevent and/or Correct Deformity		Dr I Ziv
			(Buffalo)
	Discussion		
	LUNCH) (- TT/1
1330	- To Provide a Base of Support and to Facili	itate Training	Ms J Kluzik
1415	Discussion		(Boston)
1415 1430	Discussion To Improve the Dynamic Efficiency of Gri	+	Dr C B Meadows
1430	- To Improve the Dynamic Efficiency of Gai	l	(Glasgow)
1515	Discussion		(Chasgow)
1515	Syndicate Session (A)		
1615	COFFEE		
1645	Syndicate (A) Reports		
1730	Discussion		
1800	End of Session		
1000			
FRIDA	AY 11th NOVEMBER		
0830			
0930	Syndicate (B) Reports		
1000	How do Therapists Achieve these Aims		Ms R Boyd
			(London)
1045	Discussion		
1100	COFFEE		
1120	How do Surgeons Achieve these Aims	(a)	Dr J Fisk
			(Springfield IL)
1205	Discussion		
1215		(b)	Dr G Carter
			(Perth, Aus)
1245	Discussion		
1300	General Discussion on Therapy and Surgery	,	
1330	LUNCH		
	Current Orthotic Practise		M. D. Consist-
1430	- An Overview		Ms B Cusick
		.	(Colorado)
		and	Mr G Small
			(Texas)

6

1515 Discussion

	Current Orthotic Practise in Relation to		
1530	- The Management of Deformity		Mr C Drake (London)
1600	Discussion		()
1615	COFFEE		
1645	- The Provision of a Base of Support and th	ne	
	Facilitation of Training		Mr R Lin (Newington)
1715	Discussion		
1730	End of Session		
<u>SATU</u>	RDAY 12th NOVEMBER		
	Current Orthotic Practise in Relation to the Improvement of Gait Efficiency		
0845	- Biomechanical Considerations	(a)	Dr C Vaughan (Charlottesville)
0915	Discussion		(enanottosvino)
0930		(b)	Mr R Major (Oswestry)
1000	Discussion		
1015	COFFEE		
1045	- Orthotic Designs	(a)	Mr T Supan (Springfield IL)
1115	Discussion		
1130		(b)	Mr D Weber (Hamilton)
1200	Discussion		
1215	Syndicate Session (C)		
1330	LUNCH		
1430	Syndicate (C) Reports		
1515	Discussion		
1545	COFFEE		
1615	Integration of Treatment	(a)	Dr J Becher
1700		(b)	(Amsterdam) Dr P Masso
1700		(0)	(Springfield MA)
1730	Closing Discussion		(opinigneta MA)

1800 End of Conference

SELECTED BIBLIOGRAPHY (JUNE 1994)

Journal Articles

- 1 ANDERSON J P, SNOW B, DOREY F J ... (et al.) Efficacy of soft splints in reducing severe knee-flexion contractures. Dev Med Child Neurol 1988 <u>30</u>, 502-508.
- 2 BAMBERG H. Orthopadie-technische versorgung für patienten mit zerebralparese (orthopedic technical management of cerebral palsy patients). Orthop Tech 1988 <u>39</u>, 731-735.
- 3 BANZIGER E, HEWITT C, FORD RL. Dynamic dorsiflexion assist polypropylene ankle foot orthosis. J Assoc Child Prosthet Orthot Clin 1991 <u>26</u>, 65-68.
- 4 BERTOTI D B. Effect of short leg casting on ambulation in children with cerebral palsy. Phys Ther 1986 <u>66</u>, 1522-1529.
- 5 BINDER H, ENG G D. Rehabilitation management of children with spastic diplegic cerebral palsy. Arch Phys Med Rehabil 1989 <u>70</u>, 482-489.
- 6 BOYD R, DRAKE C. Effectiveness of the hip abduction and spinal orthosis for postural management in a group of non ambulant bilateral cerebral palsy children (abstract). Newsletter ISPO 1993 <u>Summer</u>, 26-27.
- 7 BROWN K. Positional deformity in children with cerebral palsy. Physiotherapy Practice 1985 <u>1</u>, 37-41.
- 8 BRYCE J. The management of spasticity in children. Physiotherapy 1976 <u>62</u>, 353-357.
- 9 BUTLER P B, THOMPSON N, MAJOR R E. Improvement in walking performance of children with cerebral palsy: preliminary results. Dev Med Child Neurol 1992 <u>34</u>, 567-576.
- 10 CAMPBELL J, BALL J. Energetics of walking in cerebral palsy. Orthop Clin North Am 1978 <u>9</u>, 374-377.
- 11 CARLSON J M. Biomechanik und orthetische versorgung der unteren extremitaten bei kindern mit zerebraler lahmung (lower extremity biomechanics and orthotics for the child with cerebral palsy). Orthop Tech 1987 <u>38</u>, 497-507.
- 12 CHALMERS D D, HAMER G P. Three point dynamic orthosis: technical note. Prosthet Orthot Int 1985 <u>9</u>, 115-116.
- 13 CUSICK B, SUSSMAN M D. Short leg casts: their role in the management of cerebral palsy. Phys Occup Ther Pediatr 1982 <u>2</u>, 93-110.

- 14 DEMOPOULOS J T, ESCHEN J E. Variations in plastic orthoses for the developing child. ICIB 1976 <u>15(1/2)</u>, 1-6.
- 15 DIAMOND M. Rehabilitation strategies for the child with cerebral palsy. Pediatric Annals 1986 <u>15</u>, 230-236.
- 16 DONOVAN E M, ARONSON D D. Serial casting for equinus contracture in children with spastic cerebral palsy (abstract). Orthop Trans 1992 <u>16</u>, 8.
- 17 DRAKE C, BOYD R. The design and manufacture of a thermoplastic hip abduction/spinal orthosis for bilateral non ambulant cerebral palsy children (abstract). Newsletter ISPO (UK) 1993 <u>Summer</u>, 25-26.
- 18 DRAKE C J. Results from a comparison study of the effectiveness of the fixed ankle foot orthoses versus the hinged ankle foot orthosis. Newsletter ISPO (UK) 1993 <u>Summer</u>, 23-24.
- 19 DUNCAN W R. Foot reflexes and the use of the "inhibitive cast". Foot Ankle 1983 <u>4</u>, 145-148.
- 20 EMBREY D G, YATES L, MOTT D H. Effects of neuro-developmental treatment and orthoses on knee flexion during gait: a single-subject design. Phys Ther 1990 <u>70</u>, 626-637.
- 21 FULDNER R V, ROSENBERGER J. The Newington brace for cerebral palsy. Clin Orthop 1958 <u>12</u>, 151-158.
- 22 GANS B M, ERICKSON G, SIMONS D. Below-knee orthosis: a wrap-around design for ankle-foot control. Arch Phys Med Rehabil 1979 <u>60</u>, 78-80.
- 23 GARRETT A L, LISTER M, BRESNAN J. New concepts in bracing for cerebral palsy. Phys Ther 1966 <u>46</u>, 728-733.
- 24 GRITZKA ... (et al.) Serial short leg casts for the treatment of equinus deformity (abstract). J Pediatr Orthop 1987 <u>7</u>, 490.
- 25 HANSON C J, JONES L J. Gait abnormalities and inhibitive casts in cerebral palsy. J Am Podiatr Med Assoc 1989 <u>79</u>, 53-59.
- 26 HARRINGTON E D, LIN R S, GAGE J R. Use of the anterior floor reaction orthosis in patients with cerebral palsy. Orthot Prosthet 1983 <u>37(4)</u>, 34-42.
- 27 HARRIS S R, RIFFLE K. Effects of inhibitive ankle-foot orthoses on standing balance in a child with cerebral palsy: a single-subject design. Phys Ther 1986 <u>66</u>, 663-667.
- 28 HARRYMAN S E. Lower-extremity surgery for children with cerebral palsy: physical therapy management. Phys Ther 1992 <u>72</u>, 16-24.

- 29 HINDERER K A, HARRIS S R, PURDY A H ... (et al.) Effects of "tonereducing" vs. standard plaster-casts on gait improvement of children with cerebral palsy. Dev Med Child Neurol 1988 <u>30</u>, 370-377.
- 30 HYLTON N M. Postural and functional impact of dynamic AFOs and FOs in a pediatric population. J Prosthet Orthot 1989 <u>2</u>(1), 40-53.
- 31 KASSER J R, MACEWEN G D. Examination of the cerebral palsy patient with foot and ankle problems. Foot Ankle 1983 <u>4</u>, 135-144.
- 32 KENDALL P H, ROBSON P. Lower limb bracing in cerebral palsy. Clin Orthop 1966 <u>47</u>, 73-76.
- 33 KHODADADEH S, PATRICK J H. Force plate studies of cerebral palsy hemiplegic patients. J Hum Move Stud 1988 <u>15</u>, 273-278.
- 34 KNUTSON L M, CLARK D E. Orthotic devices for ambulation in children with cerebral palsy and myelomeningocele. Phys Ther 1991 <u>71</u>, 947-960.
- 35 KOOP S E, LONSTEIN J E, WINTER R B ... (et al.) The natural history of spine deformity in cerebral palsy (abstract). Orthop Trans 1993 <u>17</u>, 213.
- 36 LANGE L R. Management of a severely involved cerebral palsy patient: case study. Orthot Prosthet 1987 <u>40</u>(4), 24-27.
- 37 LOUGH L K, SODERBERG G L. The effects of fixed and hinged ankle foot orthoses on gait myoelectric activity and standing joint alignment in children with cerebral palsy (abstract). Phys Ther 1991 <u>71</u>(Suppl), S57-S58.
- 38 MANOLIKAKIS G. Individuelle versorgung mit sitzpreiz- und spreizleige-schalen bei adduktionskontraktur und drohender paralytischer huftluxation bei infantiler zerebralparese (individual care of adduction contractures and threatening paralytic hip dislocations in cerebral palsy using sitting and lying expanding casts). Orthop Tech 1992 <u>43</u>, 810-815.
- 39 MCDONALD K C, VALMASSY R L. Cerebral palsy: a literature review. J Am Podiatr Med Assoc 1987 <u>77</u>, 471-483.
- 40 MESSINGER A L, HAVILAND C W. A new hip-action adjustable abduction orthosis. ICIB 1975 <u>14(1)</u>, 1-4 17.
- 41 MIDDLETON E A, HURLEY G R B, MCILWAIN J S. The role of rigid and hinged polypropylene ankle-foot-orthoses in the management of cerebral palsy: a case study. Prosthet Orthot Int 1988 <u>12</u>, 129-135.
- 42 MOLLY J P, BERGER J, BRIGUE J P. Les coques moulees avec recouvrement du scaphoide chez l'enfant infirme moteur cerebral (moulded shells covering the scaphoid bone for children with cerebral palsy). J Readapt Med 1987 <u>7</u>, 84-86.

- 43 MOSSBERG K A, LINTON K A, FRISKE K. Ankle-foot orthoses: effect on energy expenditure of gait in spastic diplegic children. Arch Phys Med Rehabil 1990 <u>71</u>, 490-494.
- 44 MUELLER K, CORNWALL M, MCPOIL T ... (et al.) Effect of a tone-inhibiting dynamic ankle-foot orthosis on the foot loading pattern of a hemiplegic adult: a preliminary study. J Prosthet Orthot 1992 <u>4</u>, 86-92.
- 45 MURRAY W T, GREENFIELD J E. The cosmetic below-knee brace. Orthot Prosthet 1970 24(4), 27-30.
- 46 MURRI A. Grundprinzipien der orthopadisch-technischen versorgung von IZPpatienten aus der sicht des neuroorthopaden (neuroopedic principles of fitting CPpatients with orthotic devices). Med Orthop Tech 1990 <u>110</u>, 112-114.
- 47 NUZZO R M. Dynamic bracing: elastics for patients with cerebral palsy, muscular dystrophy and myelodysplasia. Clin Orthop 1980 <u>148</u>, 263-273.
- 48 OUNPUU S, BELL K J, DAVIS R B ... (et al.) An evaluation of the posterior leaf spring orthosis using gait analysis (abstract). Dev Med Child Neurol 1993 <u>35</u>(Suppl 69), 8.
- 49 PHELPS W M. The cerebral palsies. Clin Orthop 1966 44, 83-88.
- 50 POLLOCK G A. Appliances used in the treatment of cerebral palsy. Prosthet Int 1965 2(3), 10-16.
- 51 PORTELA A L M. Lower extremity casting in the treatment of cerebral palsy. Phys Occup Ther Pediatr. Phys Occup Ther Pediatr 1990 <u>10</u>, 121-132.
- 52 POWELL M M, SILVA P D, GRINDELAND T. Effects of two types of anklefoot orthoses on the gait of children with spastic diplegia (abstract). Dev Med Child Neurol 1989 <u>31</u>(Suppl No.59), 8-9.
- 53 RINE R M, WARD J, LINDEBLAD S. Use of angle-angle diagrams to analyze effects of lower extremity bracing in children with cerebral palsy (abstract). Phys Ther 1992 <u>72</u>(Suppl), S57-S58.
- 54 ROBSON P. Lower limb deformity and prevention of scoliosis in cerebral palsy. Arch Dis Child 1987 <u>62</u>, 547-548.
- 55 RODGERS M M, ALBERT M C, SCHRAG D R ... (et al.) Assessment of orthotics in cerebral palsy: kinematic and kinetic responses (abstract). Dev Med Child Neurol 1992 <u>34</u>(Suppl 66), 15-16.
- 56 ROGERS J P, VANDERBILT S H. Coordinated treatment in cerebral palsy where are we today? J Prosthet Orthot 1989 2(1), 68-81.
- 57 ROSE S A, OUNPUU S, DELUCA P A. Strategies for the assessment of pediatric gait in the clinical setting. Phys Ther 1991 <u>71</u>, 961-980.

- 58 ROSENTHAL R K. The use of orthotics in foot and ankle problems in cerebral palsy. Foot Ankle 1984 <u>4</u>, 195-200.
- 59 ROSENTHAL R K, DEUTSCH S D, MILLER W ... (et al.) A fixed-ankle belowthe-knee orthosis for the management of genu recurvatum in spastic cerebral palsy. J Bone Joint Surg 1975 <u>57A</u>, 545-547.
- 60 SCHAARS A H, HENDRIKSEN H, TOORNEND J L ... (et al.) Orthotic management of the lower extremity in cerebral palsy: lower extremity orthoses in cerebral palsy. J Rehabil Sci 1990 <u>3</u>, 113-117.
- 61 SCHRAG D R, ROGERS M M, ALBERT M C. Compensatory responses to fixed ankle foot orthoses in the uninvolved limb of hemiplegic cerebral palsy children (abstract). Gait Posture 1994 <u>2</u>, 59.
- 62 SHAMP J K. Neurophysiologic orthotic designs in the treatment of central nervous system disorders. J Prosthet Orthot 1989 2(1), 14-32.
- 63 SHARRARD W J W. Paralytic deformity in the lower limb. J Bone Joint Surg 1967 <u>49B</u>, 731-737.
- 64 SIMON S R, SUTHERLAND D, GAGE J ... (et al.) A multi-institution prospective study of ambulatory patients with spastic diplegia: part 1. Dev Med Child Neurol 1989 <u>31</u>(suppl No.59), 9.
- 65 STADELMANN A. Orthoses and gait of patients with hemiplegia or club foot (abstract). Eur J Phys Med Rehabil 1992 2(4) Suppl 1, 35.
- 66 STAMP W G. Bracing in cerebral palsy. J Bone Joint Surg 1962 <u>44A</u>, 1457-1476.
- 67 SUNDANCE P, SUTHERLAND D L, BRNCICK M. Motion analysis evaluation of orthotic intervention on gait in children with cerebral palsy (abstract). Arch Phys Med Rehabil 1989 <u>70</u>(11), A99.
- 68 SUSSMAN M D. Casting as an adjunct to neurodevelopmental therapy for cerebral palsy. Dev Med Child Neurol 1983 <u>25</u>, 804-805.
- 69 SUSSMAN M D, CUSICK B. Preliminary report: the role of short-leg tonereducing casts as an adjunct to physical therapy of patients with cerebral palsy. John Hopkins Med J 1979 <u>145</u>, 112-114.
- SUSSMAN M D, SMITH M. Instant orthosis the Aquaplast AFO (abstract).
 Dev Med Child Neurol 1987 <u>29</u>(Suppl No.55), 34.
- 71 TAYLOR A G, SAXON L K. Prone board/standing frame. Physiotherapy 1988 74, 410.

- 72 TAYLOR C L, HARRIS S R. Effects of ankle-foot orthoses on functional motor performance in a child with spastic diplegia. Am J Occup Ther 1986 <u>40</u>, 492-494.
- 73 THOMAS S S, MAZUR J M, WRIGHT N ... (et al.) Quantitative assessment of AFOs for children with cerebral palsy (abstract). Dev Med Child Neurol 1989 <u>31</u>(Suppl No.59), 7.
- 74 THOMAS S S, SARWARK J F, DIAS L ... (et al.) Preliminary report on the effect of gait analysis on the clinical decision making process for children with cerebral palsy (abstract). Orthop Trans 1992 <u>16</u>, 10.
- 75 THOMPSON S B. An anti-scissoring device for patients with cerebral palsy. J Bone Joint Surg 1957 <u>39A</u>, 218-219.
- 76 VOLKERT R. Die orthopadie-technische versorgung bei kindern mit infantiler cerebral parese (ICP) (orthopediac technical management of infantile cerebral palsy in children). Orthop Tech 1988 <u>39</u>, 71-73.
- 77 VOLKERT R. Individuelle versorgungen bei zerebralen bewegungsstorungen (individual management of cerebral movement disturbances). Orthop Tech 1988 <u>39</u>, 736-739.
- 78 WATT J ... (et al.) A prospective study of inhibitive casting as an adjunct to physiotherapy for cerebral-palsied children. Dev Med Child Neurol 1986 <u>28</u>, 480-488.
- 79 WEAVER K M, HAYNES R J, HANSEN T ... (et al.) The effect of a shoe lift on gait in spastic hemiplegia (abstract). Orthop Trans 1992 <u>16</u>, 378.
- 80 WEBER D. Use of the hinged AFO for children with spastic cerebral palsy and midfoot instability. J Assoc Child Prosthet Orthot Clin 1990/91 <u>25</u>, 61-65.
- 81 WILSON B D R, ALLEN D. Splints in the treatment of cerebral palsy. Physiotherapy 1962 <u>48</u>, 41-44.
- 82 ZACHAZEWSKI J E, EBERLE E D, JEFFERIES M. Effect of tone-inhibiting casts and orthoses on gait: a case report. Phys Ther 1982 <u>62</u>, 453-455.

Other Reading

- 83 ANDERSON D M, MEADOWS C B. Some influences on the design and production of polypropylene ankle-foot orthoses for the young cerebral palsied child. In: Disability; proceedings of a seminar on rehabilitation of the disabled./ edited by RM Kenedi, JP Paul and J Hughes.- London: Macmillan, 1979. p462-470.
- 84 BUNCH W H, DVONCH V M. Cerebral palsy. In: Atlas of orthotics: biomechanical principles and application./ 2nd edition./ American Academy of Orthopaedic Surgeons.- St Louis: CV Mosby, 1985. p259-269.
- 85 CONDIE D N, MEADOWS C B. Ankle-foot orthoses. In: Biomechanical basis of orthotic management./ edited by P Bowker, DN Condie, DL Bader, DJ Pratt.- Oxford: Butterworth-Heinemann, 1993. p99-123.
- 86 FULFORD G E. Orthotics in cerebral palsy. In: The advance in orthotics./ edited by G Murdoch.- London: Edward Arnold, 1976. p443-451.
- 87 GUESS V S. Control of lower-extremity movement in cerebral palsy: selective action bracing. In: Principles of lower-extremity bracing./ edited by J Perry.-Alexandria, VA: APTA, 1967. p63-69.
- 88 MEADOWS C B et al. The use of polypropylene ankle-foot orthoses in the management of the young cerebral palsied child: a guide based on clinical experience in Dundee, Scotland.- Dundee: Tayside Rehabilitation Engineering Services, 1980.
- 89 SHARRARD W J W. Indications for bracing in cerebral palsy. In: The advance in orthotics./ edited by G Murdoch.- London: Edward Arnold, 1976. p453-461.

CONCLUSIONS AND RECOMMENDATIONS

The conference accepted the initial proposition that lower limb orthoses may be used in patients with cerebral palsy to achieve four goals

- to prevent and/or correct deformity
- to provide a base of support
- to facilitate training in skills
- to improve the dynamic efficiency of gait

The analysis of the evidence for such claims presented by **Stuberg** and the presentations by **Ziv**, **Kluzik** and **Meadows** on the scientific basis for such claims were considered and the undernoted conclusions and recommendations agreed.

- 1 The existing body of literature on the effects of orthotic intervention in cerebral palsy is, for the most part, seriously, scientifically and experimentally flawed with very few studies graded above Sackett's Level V. (Sackett, 1986)
- 2 The available scientific evidence on the causes of deformity would appear to suggest that muscle growth is reduced in the presence of spasticity which may lead to the development of deformities. "Static" positioning, such as is applied in most existing orthotic designs, is probably less effective than the "dynamic" application of force in preventing or correcting such deformities.

NB Deformities which are a result of abnormal <u>muscle</u> growth may be complimented, especially in the growing child with open epiphyses, by skeletal or joint deformities resulting from abnormal <u>bone</u> growth.

- 3 The development of more effective orthotic designs for this role will depend on further basic research to determine the optimum level and rate of application of the preventative or corrective forces.
- 4 The basic premise that distal stabilization of the lower limb joints leads to improved proximal control is supported. The immediate biomechanical effect of such action may readily be observed; however the longer term motor learning effect requires to be demonstrated.
- 5 The principle that good foot and leg position provides valuable feedback was also supported; however the use of such information would depend on the existence of an adequate control strategy.
- 6 It is recognised that appropriate orthotic designs, by controlling the position of the joints they encompass, may alter the biomechanical demands placed upon more proximal joints when walking. In general, this change results in more normal external joint moments and this effect may additionally avoid overactivity of certain muscle groups. Whether these orthoses also produce a motor learning effect still requires to be demonstrated.

- 7 It was noted that footwear modifications, alone or in conjunction with orthoses, may produce similarly beneficial effects.
- 8 Further studies of the effects of orthotic intervention are urgently required. Whilst it would be highly desirable to propose Sackett Level I and Level II studies it is recognised that the multifactorial nature of cerebral palsy would make this very difficult. Realistically, therefore, Level III studies (probably multiple single case studies) appear to be indicated, possibly in multicentre collaborations.
- 9 The conduct of even this type of study requires an adequate experimental design. Unfortunately, many clinical practitioners lack knowledge of research methods and must, therefore, either obtain training in this subject or seek assistance from qualified colleagues.
- 10 The data to be collected in such studies must include adequate information regarding the patient's medical history, the type of intervention and an appropriate measure of the patient's functional status pre and post intervention, both in the short and the long term.

The conference considered the presentations by **Neville** on the medical aspects of the cerebral palsies and by **Boyd**, **Fisk** and **Carter** on the therapeutic and surgical approaches to their management and their relationships with orthotic intervention, reaching the undernoted conclusions and recommendations.

- 11 Assessment of the child with cerebral palsy with a view to providing treatment must commence immediately when it is apparent that the child is failing to meet appropriate developmental milestones.
- 12 Assessment is a team activity and requires the participation of qualified individuals from the following professions in collaboration with the child's parents or other carers.
 - Paediatric Medicine
 - Paediatric Orthopaedics
 - Paediatric Neurology
 - Physical Therapy
 - Occupational Therapy
 - Orthotics
 - Psychiatry

portion

7:--:4

13 The assessment process should be structured so as to obtain objective data regarding the status of the child's neuro-musculo skeletal systems, abilities (gait if appropriate) and quality of life.

14 Following initial assessment, realistic treatment goals require to be established and the appropriate intervention specified (ie surgery, therapy, orthotics or other). Once again the involvement of the child's parents or carers in this process is vital.

- 15 Regular review/reassessment must take place to evaluate the effect of previous interventions, to establish if the treatment goals have been achieved and to specify, if appropriate, revised goals and associated further intervention.
- 16 It is essential that detailed records of the findings of all assessments and/or reviews are maintained with the associated treatment record. Video recordings are recognised as having particular value to record gait.
- 17 It is important to ensure that therapy and orthotic intervention is fully coordinated.
- 18 Recent scientific research on biomechanical aspects of lower limb function, the effects of soft tissue adaptation and motor learning has challenged previous ideas on therapeutic practise and opened up new avenues for the use of orthoses in conjunction with therapy to achieve the various aims.
- 19 Existing orthotic designs are probably adequate for the envisaged roles; however, more sophisticated measurement techniques may be required to assess the outcome and permit fine tuning in a clinical situation.
- 20 In general, surgical intervention should be considered either when there is a specific pathological indication such as a dislocating joint or, alternatively, when functional improvement has ceased and therapeutic/orthotic intervention has proved ineffective.

The conference considered the presentations by Cusick, Small, Drake, Lin, Supan and Weber on current orthotic practise and Vaughan and Major on orthotic biomechanics and concluded that -

21 Orthotic intervention for children with cerebral palsy may be considered as relating to three levels of function

pre standing
NB For some individuals, this will be the highest level they will ever attain.
standing
walking

22 The goals of orthotic intervention may be defined as, for the <u>pre-standing</u> child:

- to minimise or prevent deformity and hence maintain joint ranges of motion

- to achieve trunk control and thereby sitting balance, thus promoting upper limb and oromuscular function and allowing the child to interact with the environment

Vecondos

vev w

for the standing child:

- all those objectives defined for the pre-standing child and in addition

- to facilitate efficient balanced standing by providing the minimum appropriate support and thus creating an environment within which it will be possible to develop optimum control strategies

and for the walking child:

- all those objectives defined for the pre-standing and the standing child and in addition

- to attain efficient purposeful gait by facilitating desirable patterns of motion and resisting undesirable joint patterns of motion, thereby allowing the child to participate in activities of daily living.

23 The information which is required to determine the specific type of orthotic intervention and as a result the most appropriate orthotic design is for the <u>prestanding</u> child:

- the medical and social history

- the diagnosis

- the functional gross motor status and hence any specific motor or sensory impairments

- skeletal abnormalities

- the home, school and other relevant environments

- behavioural features (eg compliance and tolerance to proposed form of intervention

- relevant associated conditions (eg gastro-oesophageal reflux, epilepsy etc)

for the standing child:

- all of the information already specified for the pre-standing child and in addition

- the standing posture

- a balance assessment

and for the walking child:

- all the information already specified for the pre-standing and standing child and in addition

- a gait assessment

NB The Gage criteria for efficient gait were recognised as an appropriate tool for this purpose. (Gage, 1991)

24 The specification of the orthotic intervention required to achieve the defined goals requires the following information -

- the joints and segments to be encompassed by the orthosis

- the intersegmental orientation of the segments - the type of control to be applied to the joints encompassed by the orthoses - the form of any sensory input - the type of control which it is intended should be exerted on any joints not encompassed by the orthoses (eg the effect of a Floor Reaction Orthosis on knee joint motion) NB It was noted that the American Academy of Orthopaedic Surgeons system for orthotic prescription does encompass all these requirements. (McCollough et al, 1991) 25 A range of orthotic designs is currently available which will, when appropriately prescribed and correctly fabricated and fitted, satisfy the range of clinical and functional objectives considered as being necessary. 26 The evidence for any specific tone reducing or tone inhibiting effect of an orthosis is inconclusive; however it is clear that a close fit with accurate anatomical contouring will optimise the function attainable. Finally, the conference considered the presentations by Masso and Becher on the integration of treatment for children with cerebral palsy and concluded that -27 The care provided for people with cerebral palsy needs to be multidisciplinary, ideally should be community based, goal orientated and agreed amongst all the interested parties. Orthotic care cannot effectively be provided in isolation. The Conference strongly recommended further initiatives by ISPO in this sphere of patient care. 11 28 ISPO should act to obtain improved evidence of the effect of orthotic intervention by: - coordinating the development of a standardised protocol to record the requisite information regarding patient status, type of orthotic intervention and outcome of intervention - establishing a multicentre orthotic treatment evaluation project based on the above mentioned protocol.

29 ISPO should examine the feasibility of conducting multidisciplinary instructional courses designed to disseminate the principles of treatment agreed by the Conference allied to practical instruction on all forms of intervention by recognised experts from all the concerned disciplines.

References:

Gage J (1991) Gait Analysis in Cerebral Palsy. MacKeith Press, London, 63-67.

19

14

McCollough N C et al (1970). A New Approach to Patient Analysis for Orthotic Prescription. Artificial Limbs: 14, No. 2: 68-80.

Sackett D L (1986). Rules of Evidence and Clinical Recommendations on the use of Antithrombotic Agents. Chest, 39 (2 Suppl): 25-35.

PART 2

THE PRESENTATIONS

The following seventeen papers are the edited and in some cases revised versions of the papers presented by the invited reviewers at the conference. They represent the personal views of the presenters based on their knowledge of the literature and their clinical experience. They are also the basis on which the conference reached its conclusions and recommendations.

References

In the following papers, references to articles or books listed in the Selected Bibliography prepared for the conference are identified by a numeral, eg (43), which corresponds to the numbering of the bibliography.

Articles or books referred to which are not contained in the Selected Bibliography are identified by author(s) and date, eg (Smith and Brown, 1975) and listed in alphabetical order at the end of each paper.

THE MEDICAL ASPECTS OF THE CEREBRAL PALSIES THAT ARE RELEVANT TO ORTHOTIC MANAGEMENT OF THE LOWER LIMBS

Brian Neville MB FRCP, David Scrutton MSc MCSP, and Kate Richardson MCSP.

It is the purpose of this contribution to identify some of the key issues that arise within the cerebral palsies. It will, therefore, consider the usefulness of the concept of cerebral palsies: examples of the clinical setting in which orthoses might be used; the issue of setting priorities; and some of the developmental neurological considerations which are crucial to intervention in this difficult group of disorders.

The cerebral palsies are defined as motor disorders with neurological signs resulting from damage from the developing brain which is itself static and has occurred in early life. Excluded from the definition arbitrarily are motor delay without specific neurological signs in pure mental retardation, spinal cord damage, hydrocephalus, brain tumours, metabolic disease, well documented syndromes with a motor component, motor problems of cerebral origin in spina bifida and disorders of fine motor control. Many such patients of course need similar management. The purpose of the exclusions is mainly to remove progressive pathology. That the brain pathology is static is a conclusion of detailed investigation and very often of medium term follow-up.

It is arbitrary to define the phenotype consequent upon early damage to the nervous system by the motor component of the disability syndrome and cognitive, behavioural, seizural or special sensory definitions would be just as appropriate. In practice what is required is a description which includes all deficits.

In general, all pathological processes in the developing nervous system can produce all phenotypes but in different proportions.

The statement that the prevalence of cerebral palsies has remained at about 2.5 per 1,000 live births hides changes in the individual components, particularly an increase in severely affected very pre-term babies.

The following clinical syndromes are in general use:-

Spastic diplegia, spastic tetraplegia, spastic hemiplegia, dyskinetic cerebral palsy, ataxia cerebral palsy and the Worster-Drought syndrome or bulbar variant.

Actiology is best examined as risk factors rather than single causes but the availability of modern imaging is clarifying the situation.

The care provided for people with cerebral palsy needs to be multidisciplinary, community based, aim orientated and agreed amongst all interested parties. There is a heavy responsibility for medical services to ensure that they are available to people with severe physical and cognitive disability.

Early damage to the developing nervous system produces its own phenotypes and simplistic analogies, for example between the orthopaedic management of polio and of the cerebral palsies has proved disastrous.

Within this complex and variable field, proof that an intervention is effective in modifying the outcome can be difficult to obtain, but much of the care that is provided comes within the minimum civilised response to the family predicament.

It is important to recognise the emotional reaction that parents are having to cope with which has been likened to bereavement for the child who might have been, but with the child's continuing presence making for difficulty in this process, thus the sequence of shock - denial - anger - depression - guilt - and coming to terms is likely to be in progress during management.

Although the specific syndromes of cerebral palsy have different emphasis for their causes and underlying pathology, the work of Bengt and Gudrun Hagberg in Goteburg has emphasised the importance of risk factors which are more often multiple in the causation of the cerebral palsies. If congenital hemiplegia is taken as an example the child with congenital hemiplegia should be walking before the age of 2 and if that is not the case then the disease is either bilateral or accompanied by severe learning difficulties which effectively comes to the same thing. The studies of Paul Uvebrant and Laas Martin Wickland in Goteborg have shown that congenital hemiplegia may be caused by early maldevelopment, later cortical and subcortical damage or periventricular lesions. They interestingly found that in those with the periventricular lesions like those more commonly seen in pre-term diplegia, the leg was more severely affected than the arm, giving one understandable pathological correlate. The work of Robert Goodman has illustrated the importance of psychiatric disease in people with the cerebral palsies. Congenital brain damage is a very high risk factor for psychiatric abnormality, the psychiatric diagnosis being made in 35-40% of children with congenital hemiplegia and maladjustment is at an even higher rate. Such issues are crucial in setting management priorities.

Spastic diplegic commonly but not invariably following pre-term birth has a range of severities and similar pathology can be associated with predominately lower limb involvement to increasing upper limb and bulbar involvement but with relative sparing of cortically based disability, ie epilepsy, learning disorder and behaviour disorder. In North America, children with significant upper limb involvement within this sequence are often designated quadriplegia.

Dyskinetic or athetoid cerebral palsy tends to follow a diplegic sequence and relative freedom from cortically based disabilities and also less liability to fixed contracture development.

Children with spastic tetraplegia are amongst the most disabled people in society with very high levels of cognitive deficit, epilepsy, feeding problems and special sensory defects, with very little chance of future mobility and very high risks of developing deformity, particularly of the hips in windsweeping and the spine. Children with spastic tetraplegia commonly survive to adulthood and thus plans need to be laid early on from oesophageal reflux and later from hip dislocation. They have a high rate of illness, commonly suffer poor nutrition which needs to be remedied by various forms of

artificial feeding and their multiple medical problems which include a high rate of respiratory disease and epilepsy which require active management.

Carers of children with cerebral palsy have many concerns. They are often doubtful about the child's long-term survival and need their particular child's problems placed in context. Their feelings of bereavement and anger may make medical management very difficult. They have often been through quite a prolonged period of doubt about the prognosis. They have a very high care load in some forms of cerebral palsy and do not always find the professional advice and resources that they feel they need.

The approach to the management of children with cerebral palsy is aim orientated with specific goals which are worthwhile, relevant to the child's condition, achievable, explicit in both the type of intervention, the time over which it should occur and how it is to be audited and agreed amongst all members of the team.

Since early preventative intervention is obviously the ideal, it is important to point out that the earlier one attempts therapy, the greater the chances of loosing specificity, efficacy and assessability. Put briefly, it means that many young babies with motor delay or deviant motor development do not go on to develop cerebral palsy and achieve therapeutic cures which are an illusion. Interventions are of 3 types.

- 1 Global, for example schools of therapy and the use of drugs.
- 2 Regional, for example selective dorsal rhizotomy.
- 3 Local Botulinum toxin, surgery and orthotic management.

It is difficult to construct research which is able to assess the efficacy of early global treatments, whereas it should be easier when looking at more confined local treatments.

Orthoses are used in the cerebral palsies for the following purposes:-

1 <u>The prevention of deformity</u>. Examples of this include management of the hips and spine in tetraplegia. The work on the natural history of non-ambulant children with cerebral palsy by David Scrutton has now begun to provide clear guidelines on hips at high risk of dislocation. The long term outcome of progressive dislocation associated with massive windsweeping and scoliosis make this a painful, uncomfortable, potentially preventable outcome for those who survive to adult life. The logical intervention is a very early attack upon axial and lower limb asymmetry. As yet no intervention study has demonstrated this effect.

Potential deformities of the ankles, feet and wrists in hemiplegia and of the ankles, feet, knees and hips in diplegia also come into this group of potentially preventable deformities. Since the neurology is often not pure, a dystonic foot posture may appear and disrupt motor progress severely and is a lot more difficult to manage.

2 <u>To provide a base of support</u>. Examples of this are the provision of AFOs in

dynamic equinus, including dystonic equinus, children with ataxia and of seating for pelvic asymmetry.

- 3 <u>The provision of training</u>. Examples of this are the use of an AFO in hemiplegia to encourage knee and hip control, of the use of wrist splints to encourage hand function in hemiplegia and of AFOs in diplegia to encourage more appropriate knee use. The occasional use of twisters in diplegia probably fall into this category as well.
- 4 <u>Improving the efficiency of gait and cosmesis</u>. Examples of this include the use of an AFO in diplegia, the use of a Meyer brace for hip adduction which may also increase the base of support and certainly the appearance of gait and the use of floor reaction orthoses in diplegia. Occasionally a spinal brace may improve gait stability.

Bracing may of course be a crucial part of post-operative care when its aims include training and improving the efficiency of gait.

The factors which may limit motor progress include unrecognised progressive disease, weakness, hypertonia, the appearance of dystonia, ataxia, deformity, severe cognitive deficit insufficient or inappropriate therapy, lack of drive and increasing age.

If orthotic management of the lower limbs in cerebral palsy is to be successful, it needs to be practised as part of an informed team with all of the medical and therapeutic skills available. It needs to take place in an environment which is friendly and appropriate for young children. The different levels of service provision need to be integrated. The service for physical disability needs to be run effectively from a business point of view so that market forces are appropriately managed.

Some of the key issues referred to in this discussion are summarised in the attached appendix. (App1)

Appendix I

THE CEREBRAL PALSIES

Some Key Issues for their Effective Management

- 1 Disorders of the developing nervous system appear by slow revelation of deficits in functions appropriate to the child's age and deviant features.
- 2 The phenotype is the result of the effects of compensatory function within the nervous system in the face of early damage.
- 3 Cerebral palsies are defined as motor disorders with neurological signs resulting from damage from the developing brain which is itself static and has occurred in early life. Excluded from the definition arbitrarily are motor delay without specific neurological signs in pure mental retardation, spinal cord damage, hydrocephalus, brain tumours, metabolic disease, well documented syndromes with a motor component, motor problems of cerebral origin in spina bifida and disorders of fine motor control. The purpose of the exclusions is mainly to remove progressive pathology.
- 4 The statement that brain pathology is static is a conclusion of detailed investigation and very often follow-up.
- 5 It is arbitrary to define early damage to the nervous system by the motor component of the disability syndrome and cognitive, behavioural, seizural or special sensory definitions would be just as appropriate. In practice what is required is a composite of major deficits.
- 6 In general, all pathological processes in the developing nervous system can produce all phenotypes but in different proportions.
- 7 The statement that the prevalence of cerebral palsies has remained at about 2.5 per 1,000 live births hides changes in the individual components.
- 8 The following clinical syndromes are in general use:-

Spastic diplegia, spastic tetraplegia, spastic hemiplegia, dyskinetic cerebral palsy, ataxic cerebral palsy and the Worster-Drought syndrome or bulbar variant.

- 9 Aetiology is best examined as risk factors rather than single causes but the availability of modern imaging is clarifying the situation.
- 10 The care provided for people with cerebral palsy needs to be multidisciplinary, community based, aim orientated and agreed amongst all interested parties. There is a heavy responsibility for medical services to ensure that they are available to people with severe physical and cognitive disability.

25

- 11 Early damage to the developing nervous system produces its own phenotypes and simplistic analogies, for example between the orthopaedic management of polio and of the cerebral palsies has proved disastrous.
- 12 Within this complex and variable field, proof that an intervention is effective in modifying the outcome can be difficult to obtain, but much of the care that is provided comes within the minimum civilised response to the family predicament.
- 13 The Damocles effect! Shock denial anger depression guilt coming to terms.

THE AIMS OF LOWER LIMB ORTHOTIC TREATMENT IN CEREBRAL PALSY: A CRITICAL REVIEW OF THE LITERATURE

Wayne Stuberg PhD PT PCS

INTRODUCTION

Lower limb orthotic management for children with cerebral palsy has been reported to have many goals. The information upon which these goals are based ranges from anecdotal clinical reports to controlled single-subject and small group research. The goals of orthotic management that were identified as the basis for this conference were: 1. prevention and/or correction of deformity, 2. to promote a base of support for standing, 3. to facilitate the training of standing and walking skills, and 4. to improve the efficiency of movement with an emphasis on walking. The purpose of this paper was to review the Selected Bibliography prepared for the Conference using an established methodology to judge the scientific rigor of the reports to establish the likelihood that a stated goal was supported by scientific research.

In reviewing literature to gain clinical insights into the orthotic management of children with cerebral palsy it is often difficult to judge the scientific merit of recommendations. Many variables effect recommendations including the specific movement disorder of the individual, the subject groupings, use of controls, or general methodology that was applied in the study. In cerebral palsy management the problem of uniformly applying recommendations from the literature is compounded as the nature of cerebral palsy is non-uniformity. Although children may have been diagnosed with spastic diplegia or hemiplegia and may look clinically similar, it has been shown that the movement disorder within these subcategories can be markedly variant (Simon, Duetsch, Nuzzo, et al, 1978; Winter, 1987).

There does, however, appear to be four common goals or "aims" of orthotic treatment for children with cerebral palsy that were recurrent when the literature was reviewed. Those aims were to; 1. prevent or correct deformity, 2. promote a base of support for standing or walking, 3. facilitate the training of standing and walking skills, and 4. improve the efficiency of movement with particular attention to walking.

The purposes of this paper were to utilize an established methodology to validate recommendations made in the literature concerning the aims of lower extremity orthotic management in cerebral palsy and to present an brief overview of the literature on the topic.

METHODS

In 1986, D.L. Sackett published a method for clinicians and researchers to apply when judging conclusions from the scientific literature for the management of patients. Sackett's rules of clinical evidence provide a guideline to weigh the scientific merit of the published literature including parameters such as the type of randomization that was used, the likelihood of false positive or false negative results, whether controls were utilized and the number of subjects and groupings that were included in the study. Table 1 lists the five levels of evidence as published by Sackett.

Eighty two abstracts or papers and seven textbook chapters were reviewed and categorized by level of evidence using Sackett's methodology for each of the four aims identified. Recommendations were then made concerning the type and number studies that fall into each level of evidence. A grade "A" recommendation is supported by at least one level 1 randomized trial, but preferably more, grade "B" is supported by at least one, but preferably more level 2 trials, and grade "C" by evidence from levels 3-5. Therefore, an aim of management that carries a grade "A" recommendation provides the clinician with strong support from the literature. A grade "B" recommendation carries some support in the literature and a grade "C" recommendation indicates that it is basically clinical inference that is supporting the aim and not strong scientific data. An aim with a grade "B" or "C" does not, however, mean that the aim does not have clinical or scientific merit, but that it may not adequately substantiated. This point is very important when one considers the relative paucity of research using more rigorous methodological design in investigating the efficacy of lower extremity orthotic management in cerebral palsy.

RESULTS

Tables 2-5 list the references reviewed and the level of evidence assigned for each of the aims. The designation of "NA" is not utilized by Sackett, but was added by the author to indicate when a clinical testimonial was given in a paper in support of an aim.

The aim to prevent or correct deformity was subdivided into its two areas for discussion. Nineteen papers were identified that addressed the issue of deformity prevention through the use of orthotic intervention in children with cerebral palsy (Table 2). Two articles were categorized at level III, two at level V and the remainder were NA as reports of the author's clinical experience. The article by Watt and colleagues (78) is one of the level III articles supporting the use of inhibitive casts, solid ankle ankle-foot-orthoses (AFOs) and physical therapy services in reducing and preventing recurrence of ankle contractures. Twenty eight subjects with a diagnosis of cerebral palsy with spastic hemiparetic, diplegic or quadriplegic involvement completed the study. Methods included a nonrandomized sample treated with three weeks of inhibitive casting, physical therapy services at least twice per week followed, and then continuous wear of the AFOs until a five month follow-up. No change in static muscle tone nor developmental skills were reported, but an improvement in passive ankle dorsiflexion and videotape gait analysis was seen following the casting. Regression of the ankle range of motion and gait changes were reported at the follow up.

Eight papers were identified that addressed the issue of deformity correction through the use of orthotic intervention (see Table 3). Two articles were categorized at level III, two at level V and the remaining four as NA. Anderson and colleagues (1), a level III paper, reported on 23 children with cerebral palsy having knee flexion contractures of greater than 20° that were treated with night splints. The splints were worn bilaterally for 13 weeks followed by unilateral wear for 10 weeks to evaluate carryover of the treatment. Passive range of motion measures were recorded prior to, during and following the intervention to assess the amount of deformity correction. For individuals older than 16 years, a 45% reduction in contracture was seen from an initial mean of 54° and a 69% improvement for the individuals younger than 16 from an initial mean of 34^{0} of contracture. The authors also reported excellent carryover at follow-up ten months after the initial application of the splints. Aim two was to promote an improvement in the base of support through the use of orthoses. Four articles were identified that addressed aim two; two at level III, one at level V and the other as NA Table 4). As an example, a single subject design was employed by Harris and Riffle (27) to evaluate the improvement in balance and posture of a 4.5 year old child with diagnosis of spastic quadriplegia. A pre-post design with inhibitive AFOs was utilized with qualitative assessment from photos of the child's standing posture and measurement of the time of standing balance. Five trials were recorded over five sessions in both the pre and post phases. The authors reported a significant improvement in standing time, from less than 20 seconds without the AFOs to over two minutes by the end of the treatment phase. Considerable improvement in posture was also reported and demonatrated through use of the photographs.

Twenty five articles were identified that addressed aim three, facilitation of training of standing or walking skills (Table 5). Three papers were classified as level III, three as level V and the remaining 19 as NA. Two of the level three papers utilized a single subject design with the other by Sussman and Cusick (69) reported a cohort of 52 children who were treated with inhibitive casts and AFOs. The primary diagnosis of the children in the Sussman and Cusick study were spastic diplegia and quadriplegia with an age range from 10 months to 15 years. Children were categorized into six functional levels from pre-standing to ambulating without assistive devices. Casts were worn for an average of 42 days with a variety of orthoses provided following cast removal. Children were provided with physical therapy services concomitantly with the casting making the treatment effect of the casts and orthoses unclear, however, at six weeks following cast removal significant improvement in the children's functional capabilities were reported.

Thirty four papers were identified that supported the aim that orthoses improve the efficiency of movement (Table 6). One of the papers was classified as level II, 15 at level III, eight at level V, and the remainder as NA. Unlike the previous aims that had papers at only level III or below, one paper that included a small randomized clinical trial was found to support aim two. In the paper by Bertoti (4), sixteen children were randomized into a cohort who received only physical therapy services and a cohort that received physical therapy and inhibitive casting. The children ranged in age from 10 months to 9 years. Diagnoses of the children included spastic diplegia, hemiplegia and quadriplegia. Select gait parameters were analyzed pre casting, following the 10 week cast intervention, and at follow-up. The author reported a clinically significant improvement in stride length for the children in the group who were casted with no significant difference in stride width, foot angle or foot clarity (ink images of the footprints).

CONCLUSIONS

The four aims originally outlined for the use of lower extremity orthoses in the management of children with cerebral palsy were supported by numerous clinical case reports, many reports at the level III & V criteria of Sackett and only one report at level II(aim 4). No level I evidence was found to support any of the aims of orthotic management.

In making an estimate of the scientific rigor of our treatment aims, Sackett's criteria for grading recommendations was utilized. An aim is supported by a grade "A" recommendation when there is strong support in the scientific literature including randomized clinical trials in support of the treatment as opposed to a "C" recommendation that is supported primarily by case reports that carry a high risk of error. The assigned grades for each aim based on the literature reviewed in this study were:

<u>Aim</u>		<u>Grade</u>
1 A .	Prevent Deformity	С
1 B .	Correct Deformity	С
2.	Promote a Base of Support	С
3.	Facilitate the Training of Skills	С
4.	Improve the Efficiency of Movement (Gait)	B/C

There is a significant body of literature that supports the aims identified for orthotic management in cerebral palsy, but not at a level of scientific validation that is desirable. As orthoses have been utilized for decades in cerebral palsy management many of the papers reviewed were from more than a decade in the past and the level of research design is not of the same rigor as the more recent studies. Significant research remains to be completed to verify each of the aims to not only further validate orthotic managment, but also to provide clear guidelines for the use of orthoses in the area of cerebral palsy management.

REFERENCES

Mhr U, Von Wendt L. Influence of different sitting positions and abduction orthoses on leg muscle activity in children with cerebral palsy. Dev Med Child Neurol 1993 35(10), 870-880.

Sacket D L. Rules of evidence and clinical recommendations on the use of antithrombotic agents. Chest 1986 <u>89(2)</u>, 2-35.

Simon S, Deutsch S, Nuzzo R et al. Genu recurvatum in spastic cerebral palsy. J Bone Joint Surg 1978 <u>60A</u>, 882-894.

Winter T, Gage J, Hicks M. Gait patterns in spastic hemiplegia in children and young adults. J Bone Joint Surg 1987 <u>69A</u>. 437-441.

Sackett's Levels of Evidence

- 1. Randomized trial with low false-positive and low false-negative errors.
- 2. Randomized trial with high false-positive and high false-negative errors.
- 3. Nonrandomized concurrent cohort comparisons between contemporaneous patients. For example, controlled single-subject, pre-post or matched case-control series.
- 4. Nonrandomized historical cohort comparisons. For example, nonexperimental studies, such as comparative and correlational descriptive and case studies.
- 5. Case series without controls, ie. case reports.

TABLE 2

Aim 1A; Prevention of Deformity

Author(s)	Level of Evidence
Fuldner & Rosenberger (1958)	NA
Pollock (1965)	NA
	NA
Phelps (1966)	
Sharrard (1967)	NA
Stamp (1962)	NA
Kendall & Robson (1966)	NA
Messinger & Haviland (1975)	NA
Sharrard (1976)	V
Fulford (1976)	NA
Cusick & Sussman (1982)	V
Mills (1984)	III
Bunch & Dvonch (1985)	NA
Watt, Sims, et al. (1986)	III
Shamp (1989)	NA
Schaars, Hendriksen, et al. (1990)	NA
Weber (1991)	NA
Knutson & Clark (1991)	NA
Rogers & Vanderbilt (1992)	NA
McDonald & Valmassy (1992)	NA

Aim 1B - Correction of Deformity

Author(s)	Level of Evidence
Pollock (1965)	NA
Stamp (1962)	NA
Allen (1962)	NA
Demopoulos & Eschen (1976)	V
Sharrard (1976)	V
Anderson, Snow, et al. (1988)	Ш
Watt, Sims, et al. (1986)	Ш
Rogers & Vanderbilt (1992)	NA

TABLE 4

Aim 2 - Promote a Base of Support

Authors	Level of Evidence
Fuldner & Rosenberger (1958)	NA
Sussman & Cusick (1979)	III
Anderson & Meadows (1979)	V
Harris & Riffle (1986)	Ш

Aim 3 - Facilitate the Training of Skills

Author(s)	Level of Evidence
Thompsen (1957)	NA
Fuldner & Rosenberger (1958)	NA
Stamp (1962)	NA
Allen (1962)	NA
Pollock (1965)	NA
Garrett (1966)	NA
Phelps (1966)	NA
Kendall & Robson (1966)	NA
Bryce (1976)	NA
Demopoulos & Eschen (1976)	V
Fulford (1976)	NA
Sussman & Cusick (1979)	III
Gans, Erickson & Simon (1979)	V
Cusick & Sussman (1982)	V
Rosenthal (1984)	NA
Bunch & Dvonch (1985)	NA
Harris & Riffle (1986)	Ш
Taylor & Harris (1986)	III
Diamond (1986)	NA
McDonald & Valmassy (1987)	NA
Stamp (1989)	NA
Hylton (1989)	NA
Sundance (1989)	V
Knutson & Clark (1991)	NA
Rogers & Vanderbilt (1992)	NA

Aim 4 - Improve the Efficiency of Movement (Gait)

Author(s)	Level of Evidence
Murray & Greenfield (1970)	NA
Messinger & Haviland (1970)	NA
Sharrard (1976)	V
Demopoulos & Eschen (1976)	V
Fulford (1976)	NA
Anderson & Meadows (1979)	V
Gans, Erickson & Simon (1979)	V
Nuzzo (1980)	V
Duncan & Mott (1980)	Ш
Cusick & Sussman (1982)	V
Rosenthal, Deutsch & Miller (1985)	V
Diamond (1986)	NA
Bertoti (1986)	II
Harris & Riffle (1986)	III
Robson (1987)	NA
Lange (1987)	NA
Hinderer, Harris et al. (1988)	III
Khodadadeh & Patrick (1988)	III
Thomas, Sarwark & Dias (1989)	III
Binder & Eng (1989)	NA
Sundance (1989)	V
Shamp (1989)	NA
Mossberg, Linton & Kriske (1990)	III
Embrey, Yates & Mott (1990)	Ш
Rose, Ounpuu & DeLuca (1991)	III
Lough & Soderberg (1991)	Ш
Butler, Thompson & Major (1992)	Ш
Weber (1992)	NA
Rine, Ward & Lindeblad (1992)	III
Rogers, Albert & Schrag (1992)	Ш
Myhr & vonWendt (1993)	П
Drake & Boyd (1993)	III
Koop, Sonstein, et al. (1993)	Ш
Ounpuu, Bell, et al. (1993)	ш

THE SCIENTIFIC BASIS OF TREATMENT TO PREVENT AND CORRECT DEFORMITY

Israel Ziv MD DSc FRCSC and Lisa Cardamone BA MD

STATEMENT OF THE PROBLEM

The abnormalities associated with cerebral palsy (CP) are at the cerebral cortex and throughout the entire neural pathway, including muscles and joints. As a consequence, deformities ie contractures, occur because of muscle imbalance and shortening.

A spastic muscle has a shorter muscle belly than a normal one and it is not fibrosed (Bax and Brown, 1985, Lieber, 1986). It has increased tendon reflexes, increased resistance to passive motion, motor dysfunction and decreased strength, speed, and excursion (Landau, 1974, Rushworth, 1980). The increased resistance or muscle tone is specific to constant velocity stretch (Young and Wiegner, 1987). Despite the decrease in the ability of the spastic muscle to extend or stretch, the number of sarcomerers, fiber length, and structure visualised under light and electron microscopy is normal (Tardieu et al, 1982a, 1982b, O'Dwyer et al, 1989) The contractures are secondary to prolonged abnormal muscle function (O'Dwyer et al, 1989), decreased excursion (Silver et al, 1985) or length (Cosgrove and Graham, 1994). An animal model is needed to learn more about spastic muscle shortening and contractures.

(a) Current Treatment

Because spasticity is a manifestation of a very heterogeneous lesion (Truscelli et al, 1979) the success of attempts to treat the disorder has varied. Involuntary contractures and spasms may recur after casting or surgical lengthening. Therefore, the aim of management should be to suppress the abnormal muscle shortening and contractions (O'Dwyer et al, 1989). Immobilization of the spastic muscle by placing the limbs in a plaster cast in a lengthened position stimulates the addition of sarcomeres. Indirectly, maintaining the limbs in extension leads to increased range of motion (ROM) of the joint by lengthening the muscle, tendon or both (Tardieu et al, 1977, 1982a, 1982b). Lengthening of the tendon and/or performing a transfer by surgery is another means to achieve the same objective.

(b) Background

1. Muscle adaptation to function and metabolism

Increased muscle activity leads to increased protein synthesis (Goldspink and Williams, 1981), number of sarcomeres (Tabary et al, 1976) and hypertrophy. The number and length of sarcomeres are dependent on length of the muscle and not tension. Plantarflexors are an example of adaptation to function as they are six times stronger than dorsiflexors (Silver et al, 1985), seen also in children with Cerebral Palsy The tendon grows in the younger individual to maintain the overall muscle-tendon length (Williams and Goldspink, 1971, 1978, Tardieu et al, 1977).

Muscle growth in length is usually in response to bone growth. Insulin and insulin-like growth factors (IGFs) stimulate carbohydrate, fat and protein metabolism and growth differentiation in skeletal muscles (Alexandrides et al, 1989). There is a marked predominance of IGF-II receptors in fetal muscle that declines in early postnatal life. Muscle mass is decreased in pancreatic agenesis and increased in fetal hyperinsulinemia (Underwood and D'Encole, 1984).

The response of a growing muscle that is immobilized in a lengthened position is a brief addition of sarcomeres, whereas the response of adult muscle is constant, and sarcomeres retain their optimum tension (Williams and Goldspink, 1973, Tardieu et al, 1977). Shortening occurs if a muscle is immobilized in a shortened position, so that the length of its sarcomeres is less than optimal. The muscle responds with loss of sarcomeres (Williams and Goldspink, 1971, 1976, Tabary et al, 1972). Denervation also decreases muscle growth (Williams and Goldspink, 1976 and 1978). The shortened muscle adaptation is myogenic and occurs more slowly if muscle is denervated (Goldspink et al, 1974). The loss of sarcomeres also occurs when muscle is shortened by electrical stimulation (Tabary et al, 1981).

Connective tissue of muscle has a crucial role in contractures (Blondet et al, 1989 and O'Dwyer et al 1989). Immobilization not only causes changes in the number and length of sarcomeres, but also causes an increase in the perimysium and endomysium (Williams and Goldspink, 1984). Changes in the orientation of collagen fibers leads to the reduced ability of the muscle to extend (Tabary et al, 1972, 1981).

The area of the largest addition of sarcomeres, at the ends of myofibrils (Williams and Goldspink, 1971), is responsible for the increased length and is called "the muscle growth plate" (Ziv et al, 1984). The length of sarcomere remains constant within a given muscle at all ages. Generation of tension that may stimulate growth is achieved by optimal overlap of actin and myosin filaments at each sarcomere (Tabary et al, 1972).

2. The myotendinous junction (MTJ)

Tension, estimated at 16×10^3 N/m² (Tidball and Daniel, 1986, Alexandrides et al, 1989) is transmitted through the special ultrastructure of the basal lamina in the MTJ. Deep invaginations of the sarcolemma, which are covered by a thick lamina and have a high concentration of nuclei (Nishikawa, 1981, Kvist et al, 1991) form a large area of contact between the muscle fibril and the tendon. There are focal adhesion plaques at the myofibril terminals in which a series of proteins were found. Desmin strengthens the cytoplasmic cytoskeleton (Tidball, 1992) and dystrophin is required for the normal association of the thin filament with the membrane at the MTJ (Tidball and Law, 1991). Since type I muscle fiber is involved in continuous support of posture, the basal lamina is thicker and has longer invaginations (Kvist et al, 1991) when compared to type II. Both lamina densa and lucida of the basal lamina contain glycoseaminoglycans, fibronectin and laminin (Kvist et al, 1991).

Acetylcholinesterase

The role of acetylcholinesterase (AChhE) in the MTJ is still obscure; it was detected in the laminal rara of some rat muscles (Nishikawa, 1981). The activity or uptake of the enzyme was increased in the younger rat, in fibrils with wider Z lines and did not disappear after denervation. It could not be found in atrophic muscles (Nishikawa, 1981) therefore it is postulated that AChE is myogenic.

Surface and intracellular pool of acetylcholine receptors (AChR) were detected during synaptogenesis of the frog skeletal muscle (Goldfarb et al, 1990). The newly synthesized acetylcholine contributed initially to the intracellular receptors than to the surface AChRs. The continual increase in the surface AChRs during development shows that the rate of synthesis is greater than the rate of insertion which exceeds the rate of internalization. Light microscope radioautography revealed uniform distribution of intracellular AChR's along the length of muscle fibers, which occurs in the absence of any preferential concentration of intracellular AChR's in the subsynptic region. Our findings of increased AChE uptake at the musclulotendinous junction of eight-day-old spastic mice (Ziv and Rang, 1984, unpublished data), and the non-specific localization of AChR may have some role in muscle growth and the development of spasticity.

3. <u>Muscle in cerebral palsy</u>

Spastic calf muscles are characterized by decreased extension and excursion (Truscelli et al, 1979). Passive resistance to stretch, which exists in paralyzed limbs of humans as well as primates (Truscelli et al, 1979, Hufschmidt and Mauritz, 1985, Tardieu and Tardieu, 1987) is due partially to structural changes in the muscles themselves. These changes include the number of sarcomeres and disproportion in type of fiber and connective tissue of spastic muscles.

There are two types of deformities in children: one responds to stretching by application of a cast and the other to surgical lengthening of the Achilles tendon. Some children who undergo lengthening of the heel cord may experience a recurrence of the deformity due to a relative overgrowth of bone in relation to muscle.

Clostridium toxins

Muscle injection with a sublethal dose of tetanus toxin leads to tetanic spasm (Edstrom, 1970, Huet de la Tour et al, 1979 and Wright and Rang, 1990).

Botulinum toxin is produced by Clostridiym botulinum and causes paralysis by blocking the presynaptic release of acetylcholine from vesicles at the neuromuscular junction. Type A of this toxin can alleviate muscle spasms due to excessive neural activity of central origin or to weaken a muscle. Local injections of botulinum toxin are safe and effective in the treatment of strabismus, essential blepharospasm and hemifacial spasm (NIH, 1990). Clinical studies demonstrate symptomatic relief after injections in other conditions characterized by involuntary spasms, such as in spasmodic torticollis, oromandibular dystonia, spasmodic dysphonia, hand cramps, limb dystonia, anismus and urinary detrusor sphincter dyssynergia. Repeated injections may be needed to sustain long term relief over long periods of time. Some patients develop antibodies to the toxin which usually occur when a dose over 300 MU (mouse units) is being used. The long term effects of the injection of botulinum toxin is unknown.

4. Animal models

The objectives are to stimulate spasticity and demonstrate pathology similar to the human (Rang and Wright, 1989, Wright and Rang, 1990). However, the central nervous system of mammals is resistant to insults that create spasticity in the human. Usually, the higher mammal will recover or die and spasticity will be of short duration (Wright and Rang, 1990). Early flaccid paralysis is manifested in decerebrate cats, which develop late spasticity (Burke, 1983).

There are a number of considerations in selecting a suitable animal model for study of muscle in CP. Primates are expensive and recover spontaneously, decerebrate models are acute and not chronic and mice are too small (Wright and Rang, 1990).

- Brain damage in primates

Several varieties of surgical ablations to the brain produce severe spasticity followed by a flaccid period of four weeks (Tasker et al, 1975, Edner et al, 1982). A rhesus monkey that was anoxic for 45 min and was born with flaccid paralysis and delayed development recovered at six weeks and was not different from his twin (Ziv et al, 1984).

Spinal cord lesions

Ischemic cord lesions caused by aortic clamping produce a preferential death to the majority of internuncial fibers which are inhibitory. The infusion of saline to the cerebrospinal space produces death of both anterior horn and intermuncial neurons (Gelfan, 1966). Transection of the mid thoracic cord in cats produces disproportion of muscle fiber type (Mayer et al, 1984) and since each type of fiber has different resistance, muscle resistance to stretch increases and abnormal twitch responses occur.

Genetically spastic mice

Spastic mice (Jackson Lab, Bar Harbor, ME) (Chai, 1961) demonstrate spasticity and decreased presynaptic inhibition due to abnormalities in the bindings sites of benzodiazepines, glycine-dependent conduction (White and Heller, 1982, Biscoe et al, 1984, Biscoe and Duncan, 1986, Becker, 1990), Gamma amino butyric acid (GABA) (Biscoe and Fry, 1982, Biscoe and Duncan, 1986) and strichnine, which antagonizes the synaptic action of glycine (White and Heller, 1982). There is an abnormal twitch response in these mice (Ranatunga and Wylie, 1980). The extracellular space within the muscle fascicles is reduced (Blondet et al, 1989).

Homozygous spastic mice carry a single locus recessive mutation (Chai, 1961) on chromosome 3 (Lane, 1972) but appear normal at birth. At 2-3 weeks of age they develop rapid tremor, rigidity, gait is slow and characterized by tiptoe walking, and prolonged righting reflex. Electromyographic studies demonstrate abnormal electrical bursts during activity (Heller and Hallet, 1982). The spastic mice have a high mortality rate at weaning age.

THE GROWTH OF THE SPASTIC MUSCLE

Most of the growth of calf muscles which occurs at the MTJ follows the growth of bone. Spastic muscles grow 55% slower than bone, and most inhibition of growth occurs at the MTJ. The pathophysiology of muscle and bone growth retardation is unknown (Ziv et al, 1984, Young and Wiegner, 1987).

(a) Methods

At the age of five days, fine wires (0.03mm diameter) were inserted during open surgery under microscopic magnification into 12 litters of heterozygous spastic mice (B6C 3 a/s spa/+; Jackson Lab, Bar Harbor, ME). Three wires were inserted to the gastrocnemius muscle, one to bone ad mid-tibia, and one to the Achilles tendon. Serial radiographs were obtained until 90 days after labelling the soft tissues. Fine-grain radiographs (Faxitron) were obtained while the mice were put in carved foam so as to maintain the same leg position at each radiograph.

Computer analysis was performed of digitized data at 0.025mm resolution (HP 9845B). Error was estimated at 2.0% with the measurement of adjacent wires at tibial diaphysis of 5 mice.

Muscle growth was evaluated by the calculation of the percentage of the length increase in relation to bone length and by absolute daily increase.

Muscle growth index =
$$\frac{dM}{dB} \times 100$$

where dM is daily muscle growth and dB is daily bone growth (mm).

Preliminary histological studies of muscles and myotendineous junction with H&E, Alkaline phosphatase (Burstone, 1958) and Acetyl choline esterase (Karnovsky and Roots, 1964) were also performed.

(b) **Results**

- 1. Bone growth of 4%/day reduced to 1%/day at 4 weeks of age.
- 2. 72% of bone growth occurs at the proximal tibial epiphysis, which appeared to be linear with tibial growth rate.
- 3. Bone growth in spastic mice was 3%/day and reduced to 0.8%/day at 4 weeks. A similar rate was demonstrated in both groups.
- 4. Tendon growth: 1.1%/day at young age reduced to 0.7% later⁵³. Thirty-four percent of tendon growth occurs at the bone tendon-junction.
- 5. Two-thirds of muscle growth occurs at the MTJ, whereas 1/3 of growth is at the origin.
- 6. Spastic muscle grew 45% less.
- 7. Growth index in normal mice was 99 ± 0.29 and spastic had 55 ± 0.39 .

FUTURE DIRECTIONS AND PRELIMINARY DATA

(a) Interventions around the MTJ

The shortening of the muscle belly in the spastic mouse is not secondary to muscle contracture or shortening, since growth spurt in these mice precedes the deformity. Growth regulation occur via tensile forces acting at the MTJ that is sensitive to external influences (Ashmore, 1982). Tendon lengthening may diminish that essential tensile stimulus.

1. <u>Tenotomy</u>

Preliminary unpublished data demonstrated inhibited growth in the tenotomized muscle of mice. Tendon Achilles lengthening is good for adults because their muscles lose the ability to lengthen by growth.

2. Bone lengthening

Instantaneous lengthening of bone stimulated muscle growth in spastic mice and needs further investigation (unpublished data).

3. MTJ cautery

Cauterization of the MTJ resulted in shorter muscles and served as evidence that this region is the muscle growth plate (unpublished data).

(b) **Botulinum toxin**

1. Spastic mice

A prospective randomized study of injection of botulinum toxin A was performed in spastic mice at six days of age. The spastic muscles at maturity were 16% shorter than those of the unaffected siblings, and those spastic muscles that were injected with the botulinum toxin grew within 2% of the unaffected siblings (Cosgrove and Graham, 1994).

2. Cerebral palsy

The consequences of injections of botulinum toxin to 26 children with cerebral palsy were monitored by clinical examination and gait analysis (Cosgrove et al, 1994). Doses of 100-400 units of the toxin were given and improved the gait for 2-4 months. There seemed to be an inverse relationship between the therapeutic response and the patient's age. The younger patients demonstrated increased passive ankle dorsiflexion. Since the improvement in ambulation was demonstrated in young children, this injection may enhance muscle growth (Cosgrove et al, 1994, Cosgrove and Graham, 1994). This study also showed a reduction in spasticity in CP and improved sagittal plane kinematics in the knee and ankle (Cosgrove et al, 1994). There were no detected side effects at six months, which was the longest follow up. The limitation of this study is still the risk of the patient developing antibodies to the toxin secondary to the need for repeated injections.

(c) Apoptosis - Programmed Cell Death

Apoptosis occurs during normal embryonic or fetal development, normal tissue turnover, atrophy and toxin exposure. The initial morphologic changes include reduction in nuclear size and condensation of chromatin in crescentic caps at the nucleus periphery (Schwarz, 1992, Schwarz et al, 1993). There is also a loss of specialized surface structures and adaptation of smooth contour. In contrast to cell death, there is no evidence of mitochondrial swelling or changes in permeability of cell membrane. In later stages of apoptosis, there is blebbing at the cell surface and fragmentation of both cytoplasm and nucleus.

1. Rat premature muscle

Newborn Sprague-Dawley rats demonstrated apoptotic activity in their calf muscles. There was an increased maximal uptake of peroxidase labellings of 3'-OH DNA (Apoptag, Oncor, Inc., Gaithersburg MD) at six days in relation to one and ten days of age.

2. Spastic mice

Prospective immunohistological samplings and analysis of calf muscles from newborn mice are being studied. Four litters of heterozygous parents were biopsied at five and nine days of age. Preliminary results show an increased amount of apoptosis at the MTJ in the spastic mice at nine days of age compared to their normal siblings.

REFERENCES

Alexandrides T, Moses A C, and Smith R J. Developmental expression of receptors for insulin, insulin-like growth factor I (IGF-I and IGF-II in rat skeletal muscle. Endocrinology 1989 <u>124</u>, 1064.

Ashmore C R. Stretch-induced growth in chicken wing muscles: effects on hereditary muscular dystrophy. 1982 Am J Physiol <u>242-C</u>, 178.

Bax M C O and Brown J K. Contractures and their therapy. Develop Med Child Neurol. 1985 <u>27</u>, 423.

Becker C-M. Disorders of the inhibitory glycine receptor: the spastic mouse. FASEB Journal 1990 <u>4</u>, 2767.

Becker C-M, Hermans-Borgmeyer I, Schmitt B and Betz H. The glycine receptor deficiency of the mutant mouse spastic: evidence for normal glycine receptor structure and localization. J Neurosci. 1986 <u>6(5)</u>, 1358.

Biscoe T J and Duchen M R. Synaptic physiology of spinal motoneurons of normal and spastic mice: an in vitro study. J Physiol -London 1986, 379:275.

Biscoe T J and Fry J P. Some pharmacological studies on a spastic mouse. Br J Pharmacol 1982 75:22.

Biscoe T J, Fry J P and Rickets C. Changes in benzodiazepine receptor binding as seen autoradiographically in the central nervous system of the spastic mouse. J Physiol 1984, 352:509.

Blondet B, Duxson M J, Harris A J, Melki J, Guenet J -L, Pincon-Raymond M and Rieger F. Nerve and muscle development in paralyse mutant mice. Develop Biol 1989, 132:153.

Burke D. Critical examination of the case for or against fusimotor involvement in disorders of muscle tone. In Esmedt J E (ed): Motor Control Mechanisms in Health and Disease. New York, Raven Press, 1983.

Burstone M S. Histochemical demonstration of acid phosphatses with aphthol AS phosphates. J Natl Cancer Inst 1958 21:523.

Chai C K. Hereditary spasticity in mice J Heredity 1961 52:241.

Cosgrove A P, Corry I S and Graham H K. Botulinum toxin in the management of lower limb in cerebral palsy. Develop Med Child Neurol 1994 <u>36(5)</u>, 386.

Cosgrove A P and Graham H K. Botulinum toxin A prevents the development of contractures in hereditary spastic mouse. Develop Med Child Neurol 1994 <u>36</u>, 379.

Edner T J, Bloedel J R, Vitek J L and Schwarz A B. Effects of cerebellar stimulation on the stretch reflex in the spastic monkey. Brain 1982 105:245.

Edstrom I. Selective changes in the sizes of red and white muscle fibers in upper motor neuron lesions and Parkinsonism. J Neurol Sci 1970 <u>31</u>, 537.

Gelfan S. Altered spinal motor neurons in dogs with experimental hind limb rigidity. J Neurophysiol 1966 29, 583.

Goldfarb J, Cantin C and Cohen M W. Intracellular and surface acetylcholine receptors during the normal development of a frog skeletal muscle. J Neurosci 1990 10(2), 500.

Goldspink G, Tabary C, Tabary J C, Tardieu C and Tardieu G. Effect of denervation on the adaptation of sarcomere number and muscle extensibility to the functional length of the muscle. J Physiol 1974, 236-733.

Goldspink G and Williams P E. Development and growth of muscle In Guba F, Marechal G and Takacs O (eds). Mechanism of Muscle Adaptation to Functional Requirements. Advances in Physiological Sciences. Vo 24. New York, Pergamon. 1981..

Gundlach A L. Disorder of the inhibitory glycine receptor: inherited myoclonus in Poll Hereford calves. FASEB Journal 1990 <u>4</u>, 2761.

Heller A H and Hallett M. Electrophysiological studies with the spastic mutant mouse. Brain Res 1982, 234-299.

Huet de la Tour E, Tabary J C, Tabary C and Tardieu C. The respective roles of muscle lengthy and muscle tension in sarcomere number adaptation of guinea-pig soleus muscle. J Physiol 1979 <u>75</u>, 589.

Hufschmidt A and Mauritz K -H. Chronic transformation of muscle in spasticity: a peripheral contribution to increased tone. J Neurol. Neurosurg. Physchiatry 1985 <u>48</u>, 676

Karnovsky M J and Roots L. A "direct coloring", thiocholine method for cholinesterases. J Histochem Cytochem 1964 12, 219.

Kvist M, Jozsa L, Kannus P, Isola J, Vieno T, Jarvinen M and Lehto M. Morphology and histochemistry of the myotendineal junction of the rat calf muscles. Acta Anat 1991, 141:199.

Landau W M: Spasticity. The fable of a neurological demon and the emperor's new therapy. Arch. Neurol 1974 <u>31</u>, 217.

Lane P W. Two new mutations in linkage group XVI of the house mouse. J Hered. 1972 <u>63</u>, 135.

Lieber R L. Skeletal muscle adaptability. I: Review of basic properties. Develop Med Child Neurol 1986 <u>28</u>, 390.

Mayer R F, Burke R E, Toop J, Walmsley B and Hodgson J A. The effect of spinal cord transection on motor units in cat medial gastrocnemius muscles. Muscle & Nerve 1984 7, 23.

NIH. Clinical use of botuolinum toxin. Consensus Dev. Conf. 8, 1990.

Nishikawa M. Histo- and cytochemistry of acetylcholinesterase activity at the myotendinous junction in skeletal muscles of rats. Acta Histochem Cytochem 1981 14(6), 670.

O'Dwyer N J, Neilson P D and Nash J. Mechanisms of muscle growth related to muscle contracture in cerebral palsy. Develop Med Child Neurol 1989 <u>11</u>, 199.

Ranatunga K W and Wyle S R. Isometric contractile properties of fast-and-slowtwitch muscles in normal and spastic mice. Exp Neuro 1980, <u>70</u>, 205.

Rang M and Wright J: What have 30 years of medical progress done for cerebral palsy? Clin Orthop Rel Res 1989, 247:55.

Rushworth G: Some pathophysiological aspects of spasticity and the search for rational and successful therapy. Int Rehab Med 1980 $\underline{2}$, 23.

Schwarz L M. Insect muscle as a model for programmed cell death. J Neurobiol. 1992 23(9), 1312.

Schwartz L M, Jones M E E, Kosz L and Kuah K. Selective repression of actic and myosin heavy chain expression during the programmed death of insect skeletal muscle. Develop Biol 1993, 158:448.

Silver R L, de la garza J and Rang M. The myth of muscle balance. A study of relative strengths and excursions of normal muscle about the foot and ankle. J Bone Joint Surg 1985 <u>67B</u>, 432.

Tabary J C, Tabary C, Tardieu C, Tardieu G and Goldspink G: Physiological and structural changes in the cat's soleus muscle due to immobilization at different lengths by plaster casts. J Physiol 1972, 224-231.

Tabary J C, Tardieu C, Tardieu G and Tabary C. Experimental rapid sarcomere loss with concomiant hypoextensibility. Muscle & Nerve 1981 <u>4</u>, 198.

Tabary J C, Tardieu C, Tardieyu G, Tabary C and Gagnard L. Functional adaptation of sarcomere number of normal cat muscle. Journal de Physiologie 1976 <u>72</u>, 277.

Tardieu C, de la Tour, E H Bret M D and Tardieu G. Muscle hypoextensibility in children with cerebral palsy. 1. Clinical and experimental observations. Arch Phys Med Rehab 1982, 63:67.

Tardieu C, Tabary J C, Tabary C and Huet de la Tour E. Comparison of the sarcomere number adaptation of young and adult animals. J Physiol 1977 <u>73</u>, 1045.

Tardieu G and Tardieu C: Cerebral palsy. Mechanical evaluation and conservative correction of limb joint contractures. Clin Orthop Rel Res 1987 219:63.

Tardieu G, Tardieu C, Colbeau-Justin P and Lespargot A. Muscle hypoextensibility in children with cerebral palsy. II Therapeutic implications. Arch Phys Med Rehab 1982 <u>63</u>, 103.

Tasker R R, Gentili F, Sogabe K, Shanlin M and Hawrylyshyn P. Decorticate spasticity: A re-examination using quantitative assessment in the primate. Can J Neurol Sci 1975 <u>2</u> 303.

Tidball J G: Desmin at myotendinous junctions. Exp Cell Res 1992, 199-206.

Tidball J G and Daniel T L: Myotendious junctions of tonic muscle cells: Structure and loading. Cell Tissue Res 1986, 245-315.

Tidball J G and Law D J: Dystrophin is required for normal thin filament-membrane association at myotendon junctions. Am J Pathol 1991 <u>72</u>, 138.

Truscelli D, Lespargot A and Tardieu G. Variation in the long-term results of elongation of the tendo Achillis in children with cerebral palsy. J Bone Joint Surg 1979 <u>61B</u> 466.

Underwood L E and D'Encole A J. Insulin and insulin-like growth factors/somatomedings in fetal and neonatal development. Clin Endocrinol Metab 1984 <u>13</u>, 69.

Videman T. An experimental study of the effects of growth on the relationship of tendons and ligaments to bone at the site of diaphyseal insertion. Acta Orthop Scand Suppl 1970 131:1.

White W F and Heller A H. Glycine receptor alteration in the mutant mouse spastic. Nature 1982, 298:655.

Williams P E and Goldspink G: Longitudinal growth of striated muscle fibres J Cell Science 1971, <u>9</u>, 751.

Williams P E and Goldspink G. The effect of imobilization on the longitudinal growth of striated muscle fibres. J Anatomy 1973, 116:45.

Williams P E and Goldspink G. The effect of denervation and dystrophy on the adaptation of sarcomere number to the functional length of the muscle in young and adult mice. J Anatomy 1976, 122:455.

Williams P E and Goldspink G. Changes in sarcomere length and physiological properties in immobilized muscle. J Anatomy 1978, 127:459.

Williams P E and Goldspink G. Connective tissue changes in immobillised muscle. J Anatomy 1984, 138:343. Wright J and Rang M. The spastic mouse and the search for an animal model of spasticity in human beings. Clin Orthop Rel Res 1990, 253:12.

Young R R and Wiegner A W. Spasticity Clin Orthop Rel Res 1987, 219:50.

Ziv I, Blackburn N, Rang M and Koreska J. Muscle growth in normal and spastic mice. Develop Med Child Neurol. 1994, 26:94.

THE SCIENTIFIC BASIS OF TREATMENT II TO PROVIDE A BASE OF SUPPORT AND TO FACILITATE LEARNING

JoAnn Kluzik MS PT

INTRODUCTION

Children who have cerebral palsy frequently demonstrate postural control limitations in sitting and standing as a major impairment to function. Inefficient and atypical movement patterns are often employed to maintain balance and to achieve functional motor skills in an upright position (Bobath and Bobath, 1975, Bly, 1981, Nashner, Shumway-Cook and Marin, 1983, Olney and Wright, 1994). One goal of lower extremity orthotic intervention has been to stabilize specific lower extremity joints in optimal biomechanical alignment with the purpose of increasing joint and thus postural stability (30, 58, 83, 84, 85, Cusick, 1990). It has been thought that this increased level of stability will enhance the level and quality of motor function in a given position and thus lead to increased motor skill development.

Several motor control deficits may contribute to dysfunctional postural control in children who have cerebral palsy. Abnormal sequencing of muscle activation and disturbed timing relationships, insufficient force production, abnormal biomechanical alignment and therefore inefficient or ineffective balance strategies, excess cocontraction and abnormal muscle tone have all been cited as potential underlying motor impairments (Barolat-Ramona and Davis, 1980, Dietz and Berger, 1982, Nashner, Shumway-Cook and Marin, 1983, Fetters, 1991, Olney and Wright, 1994). A normal standing balance response during a task involving a small amplitude displacements consists of an ankle strategy with a tightly controlled distal to proximal sequence of muscle activation (Woollacott and Sviestrup, 1992). Children with spastic hemiplegia and diplegia demonstrate both abnormal and more variable muscle sequence timing relationships during standing balance tasks, including an abnormal proximal to distal sequence of muscle activation (Nashner, Shumway-Cook and Marin, 1983). This proximal to distal sequence is inefficient biomechanically, resulting in excess hip motion and center of mass displacement. The postural task of keeping the trunk upright and center of mass over the base of support is thus more difficult. In order to balance, children with cerebral palsy may stiffen extremities and trunk to compensate for inability to produce movements which are efficient, precisely timed and graded to match task and environmental demands. Excess recruitment of many muscle groups (overflow activity) and excess cocontraction (co-activation of antagonistic pairs) is a frequently reported observation of children with spastic cerebral palsy in standing and gait activities (Knuttson and Martensson, 1980, Dietz and Berger, 1982, Nashner, Shumway-Cook and Marin, 1983, van den Biggelaar & Berbrayer, 1990).

Studies demonstrating the efficacy of orthoses in achieving increased stability and motor function exist, but are limited in number and in strength of research design. Young children with cerebral palsy have demonstrated improved postural control in stance when wearing "inhibitive ankle-foot orthoses" (72), Dynamic Ankle-Foot Orthoses (DAFOs) (27), fixed ankle-foot orthoses "tuned" to minimize torque at the knees (9) and "tone reducing casts" (4, 29, 69, 78).

Improvements cited in these studies are primarily in quality of postural control, with minimal demonstrable change in functional skill level. Two of these studies, however, did report significant functional change following a period of orthotic intervention. Harris and Riffle (27), in a single subject study of a five year old child with spastic quadriplegia, found improved duration of independent stance. Butler et al (9) in a study of six 3-6 year old children with either spastic hemiplegic or diplegic cerebral palsy, found improved balance in both kneeling and standing. Some children in this study also developed the ability to initiate balance reactions at the ankle following perturbation in a standing position.

Qualitative improvements reported in the above cited studies are mainly subjective in nature. Notable qualitative improvements in standing postural control reported in the above studies include improved head and trunk control (69), improved symmetry, (72), increased heel contact (29, 72, 78) and decreased high guard position (29, 72). In addition to improved standing postural control, Hinderer et al (29) also reported increased ability to sit independently and to move from squat to stand without support, both of which indicate improved trunk and postural control. Bertoti (4) in the only study noted above that utilized an experimental design with both treated and control groups, found improved trunk control in tasks such as sitting and quadruped with casting intervention. Taylor and Harris (72) noted improved upper extremity function on the Peabody Fine Motor Scale during wear of inhibitive ankle-foot orthoses by a six year old boy with spastic diplegia. The authors postulate that increased stability and postural control may have contributed to gains made in upper extremity skill level.

Improvements in gait of children with cerebral palsy through lower extremity orthotic intervention can be found in the literature, though outcome measures and specific intervention protocols vary widely across studies. Reported positive effects include improved functional level of ambulation (69), increased stride length (4, 29, 73), reduced Physiological Cost Index (PCI) with implied reduced energy cost (43), reduced toe walking and/or increased foot to floor contact (9, 29, 41, 52, 78), more upright trunk and improved ground reaction force vector alignment through the knee (9) increased symmetry (41), decreased excessive knee flexion (20) and elimination of the high guard position (29). Given the important postural control task elements of gait, many of the cited improvements in gait may relate to improved postural stability facilitated by the orthosis cast. Winter (1989) suggests that gait can be broken down into three sub-tasks consisting of: 1) maintaining the upper body upright against gravity (preventing collapse of the lower extremities); 2) maintaining balance in the anterior-posterior planes of movement (keeping center of gravity over the base of support; and 3) controlling foot trajectory. The first two of these task elements are postural control tasks that must be timed specifically to meet balance demands in response to the quickly changing center of gravity in forward progression. If orthoses can enhance postural stability during forward progression, it seems logical that the functional skill of gait would become more attainable as well as more efficient.

While few existing studies validate the theory that lower extremity orthotic intervention leads to increased stability and balance and consequently improved motor function, a rationale for using orthoses to enhance stability, and thus function, can be developed from motor control and motor learning literature. Rationales for employing lower limb orthoses or casts to enhance postural control and gait in children with cerebral palsy generally fall into two realms, biomechanical alignment and neurophysiological mechanisms (62). These two dimensions are highly inter-related, each directly affecting the function of the other. For example, a change in biomechanical alignment at a joint will produce a change in sensory feedback from the joint and muscles around the joint, and thus affect movement responses through both spinal cord and long loop feedback pathways (Nashner 1977, Brooks, 1986, Chez, 1991). The biomechanical change at one joint in the closed kinematic chain will also affect the biomechanical alignment at more distal and proximal joints via muscle and joint linkages (Norkin and Levangie, 1992). Many of the neural effects purported to occur due to orthoic intervention may directly relate to affordances controlled biomechanical alignment provides.

Three rationales can be derived from combined biomechanical and neurophysiological principles to explain why lower extremity orthoses might enhance postural control. First, orthoses may assist in reducing degrees of freedom of motion at specific lower extremity joints, "simplifying" the postural control task for the child by reducing available movement options. Second, optimal biomechanical alignment provided by an orthosis may minimize demands of the postural control task by: 1) enabling a broader and more stable base of support; 2) altering joint moments to reduce muscular forces required to attain and sustain upright and aligning center of mass to stay more tightly. within the base of support; and 3) altering muscle length to place muscles at their optimal length for recruitment, power and efficiency of performance. The third rationale that can be developed relates to sensory feedback aspects of motor tasks. Through externally provided alignment, the braced child receives proprioceptive and visual feedback about a biomechanically efficient position for function. Additional tactile and pressure input at points of contact with the orthosis are provided as well. This sensory feedback may facilitate more effective and efficient balance strategies to occur. More importantly, this feedback may enhance motor learning of more efficient movement strategies by the child.

REDUCED DEGREES OF FREEDOM

The concept of reducing the degrees of freedom to simplify the postural control task is similar to the frequently cited purpose of applying orthoses or casts distally (foot/ankle) in order to enhance control proximally (29, 30, 69). When available foot and ankle motions are controlled by an orthosis, the child does not have to muscularly control that joint. The work of constraining joint motion is done for the child by the external device. The child needs only to actively control the joints that remain free to move.

The above concept can be viewed within a model of motor control proposed by Bernstein and others (Bernstein, 1967, Tuller et al, 1982, Turvey et al, 1982, Scholz, 1990). This model suggests that a critical component of motor control is the ability to reduce the degrees of freedom of motion (such as which muscles are recruited, number of motor units that are active, amount of force generated and directions and degrees of motion at each joint) that must be individually controlled. Coordinative structures that link the various degrees of freedom to behave as one unit have been proposed as an essential mechanism for simplifying the task of control. One example of a neural coordinative structure in postural control is the tight coupling between sequence and timing of muscle activation coordinative structure in postural control is the tight

coupling between sequence and timing of muscle activation in standing balance that was described above. This tight coupling gradually develops in normal children as the practice pull to stand, standing and stepping activities and appears to require an element of motor learning (Woolacott and Sveistrup, 1992). In children with spastic cerebral palsy, this tight coupling does not develop (Nashner, Shumway-Cook and Marin, 1983). These children are unable to generate specifically timed muscle forces to stabilize the ankle, then hip, then trunk. Excess cocontraction and hypertonus as well as primitive reflexive patterns are commonly reported during balance activities (16, 25, 62). The upper extremities may assume a high guard position with arms stiffly held in flexion (72, Bobath and Bobath, 1975). Cocontraction stiffens the joints and minimizes the amount of displacement that would ensue from internally or externally generated destabilizing forces (Daimano, 1993). The muscle stiffening behaviors observed in children with cerebral palsy may be an attempt to stabilize each joint to compensate for inability to orchestrate the specifically sequenced and tightly coupled muscle action needed to control joint movement and keep center of mass over a stable base of support. An orthosis can work as an external stabilizer of the talocrural joint, subtalar joint and arches of the foot. Degrees of freedom of motion at the ankle and foot requiring active control by the child will be decreased. Further, because the lower extremity is a biomechanically and muscularly linked system the orthoses at the ankles may also limit degrees of freedom at more proximal joints (Norkin and Levangie, 1992). With reduced degrees of freedom, the motor control task demands placed on the child are simplified. If the lower extremities are more tightly kept over the base of support, less effort will be required to control the trunk. The child is more likely to be able to produce a successful trunk and hip balance response without the need for excess muscular activity (cocontraction, overflow). Quality and efficiency of standing balance and related tasks should improve. The high guard position may disappear. The arms may be freed for other activities as they are not constrained to the task of maintaining the trunk upright. As mentioned earlier, both Hinderer et al (29) and Taylor and Harris (72) found reduced high guard positioning of the upper extremities following orthotic intervention.

OPTIMAL BIOMECHANICAL ALIGNMENT

The second rationale supporting use of lower extremity orthoses for enhancing postural control relates to specific effects of altered biomechanical alignment. One significant effect of the alignment ankle-foot orthoses provide is the broadening of the base of support by bringing the entire foot into contact with the floor and by distributing the weight more broadly across the sole. Bunch and Dvonch (84) point out that in normal standing, the foot has a trapezoid shape with a perpindicular line to the center of gravity. Many children with spastic cerebral palsy tend to bear weight primarily on the ball of the foot and with weight centered more medially (Cusick, 1990, Perry, 1992). This reduces the base of support and makes the postural task more demanding. If orthoses are applied to increase foot contact with the support surface and to shift the distribution of weight on the foot more laterally (through alignment correction and posting), base of support is increased, thus easing postural control demands.

Alignment at each lower extremity joint and of the trunk affects the amount of muscular force required to maintain upright. The amount of active force required is dependent on joint moments created by gravitational forces. The magnitude of force

and the location of the line of gravity descending from the superimposed mass determines the joint moment, or torque. In perfectly erect and quiet stance, gravitational forces, which are directly downward, produce minimal joint torque (McCollum and Leen, 1989, Perry, 1992). In normal stance, force requirements are minimized because postural alignment assumed minimizes joint moments. In spastic cerebral palsy, the line of gravity is often posterior to the ankle joint, creating a plantar flexion moment (56). This may be due to relative plantar flexion at the ankle joint secondary to gastroc-seleus overactivity or abnormal sequencing of muscle activation, or to actual tightness in the proximal joints must compensate for the effects of ankle alignment. Excessive hip flexion and anterior pelvic tilt to maintain balance may result, an energy costly postural control task. Positioning the ankle joint in neutral or slight dorseflexion brings the weight line anterior to the ankle joint, thus reducing muscular force demands at this joint. Aligning the foot and ankle in a more biomechanically efficient position in stance subsequently influences alignment at all proximal joints in the closed kinematic chain. Since more proximal joints no longer need to compensate for a plantar flexor moment at the ankle, active control demands should be reduced proximally.

Joint alignment affects not only biomechanical forces and joint moments, but determines length of muscles crossing the joint. Muscle length determines the amount of tension that can be generated within specific muscle groups due to length-tension curve properties of muscles (Gossman et al, 1982). Children with cerebral palsy chronically assume postures and stereotypical movement patterns which place the muscles in either overshortened or overlengthened positions (Bly, 1981, Bobath and Bobath, 1975, Olney, 1994). Through the external control of an orthosis, muscles can be brought closer to their physiological resting state where peak tension can be generated. The orthosis can assist in preventing weakness secondary to length changes in the muscle.

When a muscle is placed in an overshortened position for a prolonged period of time, the number of sarcomeres decreases and the muscle is less capable of force generation (Gossman et al, 1982). The peak tension curve shifts so the muscle is now strongest in its shortened position. The muscle itself becomes less extensible and elastic (Romanini et al, 1989). The muscle's antagonistic muscle groups are now placed at a disadvantage as they must overcome the passive resistance the shortened muscle gives in order to generate force.

Weakness also results when a muscle is placed in an overlengthened position for long periods of time (Gossman et al, 1982). In this case, the muscle adds sarcomeres, but the sarcomeres become shorter. When the muscle is now placed at the alignment needed for functional skill performance (eg overstretched hip extensors now asked to fire with hips actually placed in extension), the muscle cannot easily generate force. Positioning through orthotic intervention may facilitate maintenance of optimal muscle lengths for force production ability and prevent over-shortening or over-lengthening weakness. Since inability to produce sufficient force at the correct time is a major impairment in cerebral palsy, preserving optimal muscle length is important for maximizing function.

SENSORIMOTOR LEARNING

The third and final rationale to be discussed centers on sensory consequences of lower extremity orthotic intervention. Sensation produced when the body is in optimal biomechanical alignment may facilitate improved postural control responses. Muscle and joint proprioception and pressure and tactile sensation from the sole of the foot provide information which influences postural control strategies used (Nachner, 1977, 1979). Alignment contributes to the input these sensory receptors will receive. Perhaps as, or more importantly than the immediate effect of sensory input upon postural control, is the potential value of these sensations for facilitating motor learning. Sensory information received during movement while the lower extremities are in more stable and effective alignment for balance may teach the child sensations of this alignment and subsequently lead to motor learning of more adaptive and efficient postural responses.

Joint and muscle proprioception, in addition to visual and vestibular information, is thought to contribute significantly to postural strategies selected and produced by the nervous system (Nashner, 1977, 1979, Horak and Nashner, 1986, Shumway-Cook and Horak, 1986). Distal joints and muscle groups are thought to be particularly important because they provide specific information about body alignment relative to the support surface. In mature quiet standing, an ankle strategy is the primary postural action used to maintain position. The ankle strategy has been discussed previously and is a precisely sequenced pattern of muscle activation in response to low amplitude sway about a neutral ankle joint. Long loop reflexive responses elicited via proprioceptive input has been shown to be a critical element in the ankle strategy response. If the foot and ankle are at their physiologic end ranges with muscles over shortened or overlengthened, the ankle strategy is likely to be ineffective in maintaining balance. Use of orthoses can realign the ankle and foot near neutral where more optimal balance strategies may now be facilitated.

Deep pressure and tactile input through the sole of the foot produced by weight bearing has been shown to influence standing postural responses (19, 62). Duncan proposes four reflexes which can be activated by stimulating specific zones of the sole. One of these reflexes occurs when pressure is given over the ball of the foot. Toe grasp and plantar flexion are facilitated. Children with cerebral palsy frequently exhibit a tendency towards toe grasp and plantar flexion when standing. Weight is commonly shifted forward and distributed primarily over the ball of the foot, providing deep pressure input to this area. The postural response facilitated is additional plantar flexion and toe grasping, an inefficient and energy costly movement strategy. Orthoses enable the foot to be brought more fully in contact with the supporting surface so that weight may be shifted onto various parts of the foot (44, 84). Efficient and automatic distal responses may be easier to facilitate with orthoses because the areas of the sole of the foot receiving pressure input have been altered.

Spasticity and hypertonus are frequently identified as primary impairments to functional balance and gait. Children with spastic cerebral palsy often demonstrate strong extensor tone throughout the lower extremities and in particular in the plantar flexors (25, 29, Bobath and Bobath, 1975, Bly, 1981). A common goal of orthotic and casting intervention is to inhibit abnormal muscle overactivity and/or tone and this is thought to be accomplished through the influence of sensory feedback mechanisms

(19, 29, 62, 69). It has been proposed that deep pressure to a tendon, such as to the Achilles tendon, is inhibitory to that muscle via autogenic inhibition through the Golgi tendon organs (Carlson, 1984). Orthoses which keep the toes extended and prevent toe flexion prevent excess toe grasping (56). Unweighting the metatarsal heads (via a metatarsal bar or pad) is also proposed to inhibit toe grasping (56, 62, 69).

While the above discussion may hold true, recent research questions the assumption that tone is a primary impairment. Tone may actually be a secondary and functional compensatory phenomenon that results from dysfunctional timing and force control abilities (Nashner, Shumway-Cook and Marin, 1983, Fetters, 1991). Much of the hypertonus and muscular overactivity that occurs may be an active attempt to stabilize the extremities and trunk when more efficient movement patterns are not available to the child because of primary and secondary effects of central nervous system damage. Rather than reducing tone because of sensory input that "inhibits" the tone at a neurophysiologic level, orthoses may actually succeed in reducing tone because they provide alignment which enables the child to successfully generate the forces required for successful postural control without the need for hypertonus.

Physical therapy intervention for children with cerebral palsy and neurologically impaired adults has in recent years increasingly incorporated principles derived from motor learning research (Gordon, 1987). Research suggests that movement is learned through active, goal directed, task specific practice (Carr and Shephard, 1987). If a child repeatedly assumes and functions in a biomechanically inefficient pattern, such as crouched stance or stance with weight primarily on the ball of the foot, the movement responses needed to maintain balance in these postures will become well learned. Unfortunately, considerable energy will be required to maintain upright. The longer the child practices this inefficient pattern, the more entrained and automatic the movement pattern and postural control strategies will be. If a child will receive sensory information about this more efficient movement and through repetition of the pattern, hopefully learn to reproduce the pattern more easily and more automatically.

Active (as opposed to passive) movement is considered essential for motor learning to take place (Carr and Shephard, 1987). With an ankle-foot orthosis, the greatest degree of activity and learning will occur proximally. The foot and ankle will need minimal active control since the orthosis is providing external stabilization. The knee, hip and trunk must, however, be controlled actively. Improvement in proximal control for standing balance in children with cerebral palsy was reported by Butler et al, (9) following a four to six month intervention with "tuned" fixed ankle orthoses. If an orthosis allows carefully controlled degrees of movement in selected planes at the ankle and foot, some degree of distal control has been reported, although with stroke patients and not children with cerebral palsy (44, Karas, 1989). Mueller et al, (44) utilizing a single subject design, demonstrated improved active ankle control in stance with a DAFO as the orthotic intervention. Karas, (1989) in a single case report, noted similar improvements with a supramalleolar ankle-foot orthoses.

Orthoses have been shown to affect postural control and to enhance performance of functional tasks such as standing balance, gait, gross motor skill performance, and upper extremity function, albeit in studies of limited strength of design and subject number. This paper has developed a rationale, based on biomechanical and

neurophysiological principles, to support orthotic intervention for improving postural control and motor skill performance in children with cerebral palsy. Lower extremity orthoses may enhance postural control and motor skill performance by providing more optimal biomechanical alignment, improved stability and altered sensation that may contribute to enhanced control and motor learning. Research is needed to explore these purported effects of orthoses and to determine the overall efficacy of orthotic intervention in influencing active postural control and motor skill development.

REFERENCES

Barolat-Ramona G and Davis R. Neurophysiologican mechanisms in abnormal reflex activities in cerebral palsy and spinal spasticity. Journal of Neurology, Neurosurgery and Neuropsychiatry 1980 <u>43</u>, 333-342.

Bernstein N A. The Coordinations and Regulation of Movements. Oxford: Pergamon Press. 1967.

Bly L. Abnormal motor development. In D S Slaton (Ed) Development of movement in infancy. Chapel Hill: University of North Carolina. 1981.

Bobath B and Bobath K. Motor Development in the Different Types of Cerebral Palsy. London: Heinemann Medical, 1975.

Brooks V B. The Neural Basis of Motor Control. New York: Oxford University Press, 1986.

Carlson S J. A neurophysiological analysis of inhibitive casting. Physical and Occupational Therapy in Pediatrics 1984 $\underline{4}$, 31-42.

Carr J H and Shepherd R B. A motor learning model for rehabilitation. In Movement Science. Foundation for Physical Therapy in Rehabilitation. Rockville MD: Aspen, 1987.

Cusick B D. Progressive Casting and Splinting for Lower Extremity Deformities in Children with Neuromotor Dysfunction. Tuscan: Therapy Skill Builders, 1990.

Damiano D L. Reviewing muscle cocontraction: Is it a developmental, pathological or motor control issue? Physical and Occupational Therapy in Pediatrics 1993 <u>12</u>, 3-20.

Dietz V and Berger W. Normal and impaired regulation of muscle stiffness in gait: A new hypothesis about muscle hypertonia. Experimental Neurology 1982 79, 680-687.

Fetters L. Cerebral palsy: Contemporary treatment concepts. In M J Lister (Ed) Contemporary Management of Motor Control Problems Proceedings of the II Step Conference. Alexandria V A: APTA. 1991.

Ghez G. Posture. In E R Kandel, J H Schwartz and T M Jessel (Eds) Principles of Neural Science. New York: Elsevier Science. 1991, 596-607.

Gordon J. Assumptions underlying physical therapy intervention: Theoretical and historical perspectives. In J H Carr and R B Shepherd (Eds) Movement Science: Foundations for Physical Therapy in Rehabilitation. Rockville M D: Aspen. 1987, 1-30.

Gossman M R, Sahrman S A and Rose S J. Review of length associated changes in muscle. Physical Therapy 1982 <u>62</u>, 1799-1808.

Horak F and Nashner L M. Central programming of postural movements: adaptation to altered support surface configuration. Journal of Neurophysiology 1986 <u>55</u>, 1369-1381.

Karas M A. Management of gait of adult hemiplegia with a supramalleolar AFO. A case report. Neurology Report 1989 <u>13</u>.

Knuttson L M and Martensson A. Dynamic motor capacity in spastic paresis and its relation to prime mover dysfunction, spastic reflexes and antagonistic co-activation. Scandinavian Journal of Rehabilitation Medicine 1980 <u>12</u>, 93-106.

McCollum F and Leen T K. Form and exploration of mechanical stability limits in erect stance. Journal of Motor Behaviour 1989 <u>21</u>, 225-244.

Nashner L M. Organization and programming of motor activity during posture control. Progress in Brain Research 1979 <u>50</u>, 177-184.

Nashner L M. Fixed patterns of rapid postural responses among leg muscles during stance. Experimental Brain Research 1977 <u>30</u> 13-24.

Nashner L M, Shumway-Cook A and Marin O. Stance postural control in select groups of children with cerebral palsy: Deficits in sensory organization and muscular coordination. Experimental Brain Research 1983 <u>49</u>, 393-409.

Norkin C C and Levangie P K. Joint Structure and Function. 2nd Ed, 69. Philadelphia: F A Davis. 1992.

Olney S J and Wright M J. Cerebral Palsy. In S K Campbell (Ed) Physical Therapy for Children. Philadelphia: W B Saunders. 1994, 489-523.

Perry J. Gait Analysis. Normal and Pathological Function. Thorofare NJ: Slack. 1992.

Romanini L, Villani C, Meloni C and Calrisi V. Histological and morphological aspects of muscle in infantile cerebral palsy. Italian Journal of Orthopaedics and Traumatology 1989 <u>15</u>, 87-93.

Scholz J P. Dynamic pattern theory - some implications for therapeutics. Physical Therapy 1990 70, 827-843.

Shumway-Cook A and Horak F. Assessing the influence of sensory interaction on balance. Physical Therapy 1986 <u>66</u>, 1548-1550.

Tuller B, Turvey M T and Fitch H L. The Bernstein perspective II: The concept of muscle linkage or coordinative structure. In J A S Kelso (Ed) Human Motor Behavior: An introduction. Hillsdale NJ: Lawrence Erlbaum Associates. 1982, 253-270.

Turvey M T, Fitch H L and Tuller B. The Bernstein perspective I: The problem of degrees of freedom and context-conditions variability. In J A S Kelso (Ed) Human Motor Behavior: An introduction Hillsdale NJ: Lawrence Erlbaum Associates. 1982, 239-252.

van den Biggelaar J and Berbrayer D. Gait analysis in patients with cerebral palsy. University of Toronto Medical Journal 1990 <u>67</u>, 14-19.

Winter D A. Biomechanics of normal and pathological gait: Implications for understanding human locomotor control. Journal of Motor Behavior 1989 <u>21</u>, 2337-355.

Woolacott M and Sveistrup H. Changes in the sequencing and timing of muscle response coordination associated with developmental transitions in balance activities. Human Movement Science 1992 <u>11</u>, 23-36.

THE SCIENTIFIC BASIS OF TREATMENT III TO IMPROVE THE DYNAMIC EFFICIENCY OF GAIT

C B Meadows BSc PhD

INTRODUCTION

Observation of the gait patterns of children with cerebral palsy indicates that these are different from normal children. The variation from normal depends upon the type of cerebral palsy and its severity in any given child. Its is also influenced by the stage of development of that child and any day-to-day factors such as tiredness. It is therefore a gross oversimplification to say that there is a "typical" gait pattern, however most of us in the field would probably recognise a child with cerebral palsy by observation of their gait pattern. Perhaps the most common characteristics are an apparent lack of smooth control of the motion of the limbs with high energy consumption. In other words for a child with cerebral palsy walking is difficult and requires a lot of effort.

In the context of this presentation, "dynamic efficiency" is the degree to which the gait is well-controlled and energy-efficient.

This presentation will look firstly at the biomechanics of normal locomotion, secondly at the biomechanical aspects of locomotion of children with cerebral palsy, thirdly at the effect of one form of intervention (AFOs) and finally extrapolate this experience to other forms of management.

It is appreciated that in order to function, children with cerebral palsy have to do many more things other than walking - such as rising to the standing position or performing complex functions in a sitting position. Walking is therefore not the only important goal. However perhaps more scientific evidence exists currently which relates to walking rather than any other function.

BIOMECHANICS OF NORMAL LOCOMOTION

Because of gravity, body weight acts vertically downwards (Fig. 1). To balance this there is an equal and opposite ground-to-foot force, or "ground reaction force". This is due to a basic natural law of mechanics. In order to understand the biomechanics of locomotion it is more appropriate to consider the upwardly acting ground reaction force rather than thinking of body weight acting downwards

The ground reaction force is vertical when standing still, and equal to body weight. However, when walking, the magnitude of the ground reaction force varies between slightly more or slightly less than body weight. Because of the addition of small horizontal shear forces, the ground reaction force is no longer vertical but varies from leaning backwards in early stance to forwards in late stance.

The effect of the ground reaction force is to generate a turning effect (called an external moment) at the joints. The magnitude of these is dependent on the size of the force and the perpendicular distance of its line of action from each joint (similar to a



When standing still body weight acts vertically downwards.



To balance this there exists an equal and opposite force acting vertically upwards – the "ground-to-foot force".

When walking, horizontal shear forces are generated between feet and ground either', forwards or backwards.

When vertical and horizontal forces are added together resultant force vector leans away from vertical, either forwards or backwards.



Ð

M

If the force vector, f, passes a distance, d $\frac{1}{1000}$ from a joint an external moment, m, is a generated tending to make the joint flex or extend. In this example the knee will tend to flex.

m = f x d

To balance this, muscles must produce equal and opposite internal moments at the joints, in this example the knee extensors. Note also that the external moment at the hip will tend to cause extension and at the ankle dorsiflexion.

The moments generated depend on the magnitude of the force, f, and the perpendicular distance, d. Thus if this distance is large the moment generated will be high.

 $M = f \times D$

Similarly if the ground-to-foot force is large the moment will again be high.

 $M = F \times d$

Fig 1 - Biomechanics of normal locomotion

М

lever arm). To balance this, the muscles generate an equal and opposite turning effect (called an internal moment) at each joint. The external moments thus indicate the demand being placed on the neuromuscular system. If the force is large or the perpendicular distance is large then the external moments will also be large. The muscles will therefore have to develop a similarly large internal moment to balance this. Obviously if the forces *and* the distances are large then the moments will be very large!

Walking is therefore a highly complex and constantly varying interaction of ground reaction force and muscular activity to maintain a dynamic equilibrium. In normal locomotion walking tends to be carried out in as efficient a manner as possible avoiding the generation of unnecessarily high forces and maintaining the ground reaction force in as close alignment with the joints as possible. This minimises the size of the moments being generated. In other words we make walking as easy to control and as energy-efficient as possible for ourselves!

Instrumented gait analysis systems consist of equipment such as special television cameras or other sensors to monitor limb motion (kinematic aspects) and force plates to measure the ground reaction forces (kinetic aspects). There are many systems now commercially available and these present the data in many different ways.

Normal child locomotion appears to be similar to adult locomotion although slightly more "bouncy", in other words the variation of the ground reaction force above and below body weight is proportionately greater than adults (Figure 2.A) (Meadows, 1984, Mann, 1994).

CEREBRAL PALSY GAIT

Examination of the biomechanical aspects of the gaits of children with cerebral palsy indicates that these are often very different from normal children (Meadows 1984). In particular the ground reaction forces generated may be very high during parts of the stance phase and very low at others. For example the diplegic child illustrated in Figure 2.B exhibits very high impact forces as indicated by the long arrows in early stance (over one and a half times body weight), tends to rebound off the ground in mid stance indicated by the relatively short arrows, and then fails to achieve a significant push-off in late stance indicated by the relatively small second "peak" of ground reaction forces. In addition the ground reaction forces are not always closely aligned with the joints indicating that at times high moments are being generated.

There is therefore a significant demand being placed on the child's neuromuscular system, often with wide and rapid fluctuations of external moments, to which it has to respond. The degree to which an individual child achieves success depends among other things on the severity of the condition. Thus children with cerebral palsy are not able to achieve gaits with the same ease of control and energy efficiency enjoyed by their normal peers.

There is obviously a huge variation of gait patterns displayed by different children with cerebral palsy and it is therefore difficult and potentially misleading to make generalisations. However there are a number of similarities easily observed visually

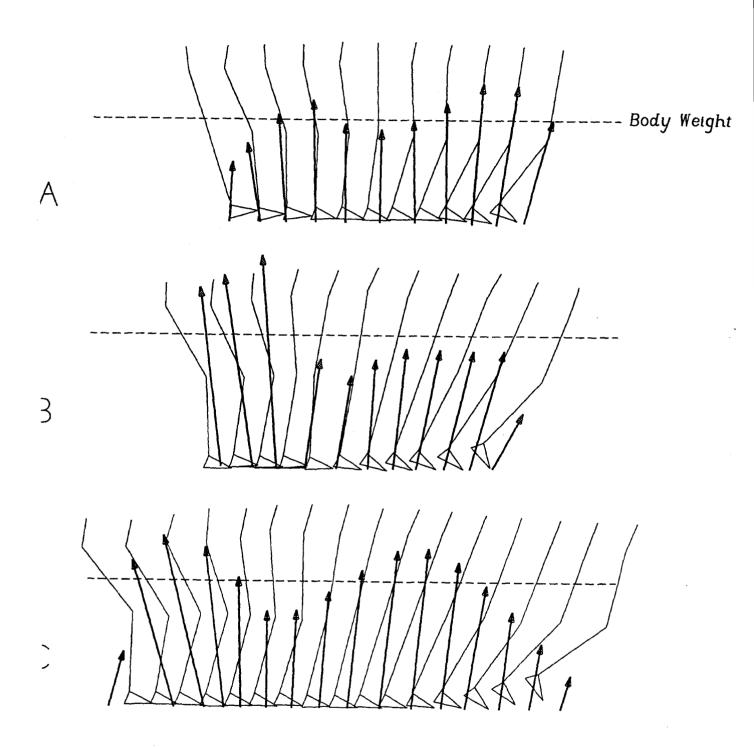


Fig 2 - Stick leg and force vector diagrams

- Normal child barefoot А
- В
- Diplegic child barefoot Same diplegic child with AFO and rocker sole С

such as the lack of smooth control of the gait patterns and the apparent high energy consumption described above. Treatment of various sorts - orthotics, physiotherapy, orthopaedic surgery or drugs - on their own or in combination are generally aimed at improving voluntary control and reducing energy consumption.

THE INFLUENCE OF AFOs

Central to the improvement of voluntary control and reduction of energy consumption is the modification of the undesirable biomechanical features of the gait pattern. In order to illustrate this process it is possible look at one specific form of intervention, the use of AFOs and associated footwear adaptations, and examine the biomechanical effects that result.

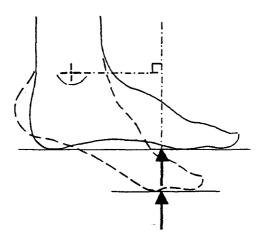
It is interesting to note that the effect of the AFO on the external moments generated at the ankle are not as significant as the effect on the moments at the knee and hip. This appears to be because the perpendicular distance between the line of action of the ground reaction force and the ankle joint may be at times little changed compared with barefoot despite the point of application being retained more posteriorly. This is due to the foot being maintained in a plantigrade attitude with the AFO rather than a plantarflexed attitude without the AFO (Fig.3). The stiffness characteristics of the AFO will to a certain extent relieve the demand on the plantarflexors which might be beneficial, however this aspect needs to be investigated further.

In Fig 4 it can be seen that in those children who have a tendency to walk with knees in extension in mid stance the effect of an appropriate AFO-footwear combination is a reduction in the external moment generated. This is achieved by the forward-leaning shank attitude moving the knee anteriorly and the origin of the reaction force being located more posteriorly on the foot.

Perhaps the most surprising effect relates to the external moments generated at the hip joint and the child's ability to support their body weight in late stance.

Gait studies have shown that in many children with cerebral palsy the second peak of the vertical component of the ground reaction force is diminished and may even be less than body weight (Meadows, 1984, Hullin, 1993). This indicates that the child has difficulty in generating the necessary muscle moments during single support to resist the downward movement of the body or generate sufficient "push-off" forces. In the extreme cases when the second peak does not reach body weight the child is actually in the process of collapsing! This is illustrated in Fig 2.B. There is therefore a significant incentive to get the contralateral foot forwards quickly to arrest the collapse. The child cannot therefore slow down because if he does he will fall down! How often do we tell the children to slow down and walk properly! The very high first peak of the ground reaction force is due to the high impact forces as the child recovers from the collapse at the end of the previous stance phase of the contralateral leg.

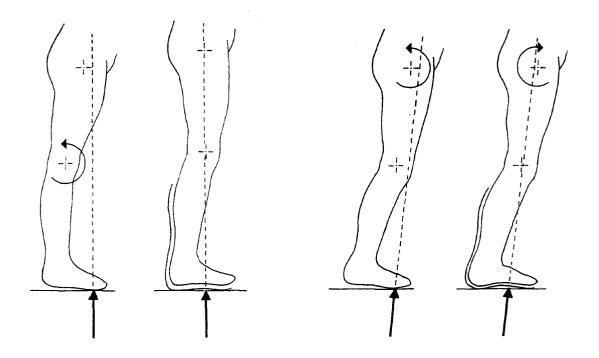
When an appropriate AFO-footwear combination is worn, the child is now apparently able to support body weight and generate increased push-off forces (Fig 2.C). As he is no longer falling down he is able to slow his gait pattern down and learn to walk with increased control and greater efficiency. The lack of high impact forces at the early part of stance phase indicates that the collapse at the end of stance phase of the



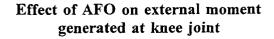


Effect of AFO on external moment generated at ankle joint

(Note that it is possible to have no change in lever arm in plantigrade position despite more posterior point of application of ground reaction force)









Effect of AFO on external moment generated at hip joint

contralateral leg did not occur. It would appear therefore that the demand on the child's neuromuscular system has been reduced to beneficial effect.

Examination of the external moments at the hip reveals the biomechanical reason for this observation (Fig 5).

When the child is barefoot or wearing an inappropriate AFO-footwear combination the origin of the reaction force in late stance is towards the front of the foot. The line of action of the force passes in front of the hip joint generating an external flexion moment. At this stage of stance phase the child requires to use the hip extensors to resist downward motion and generate push-off. Usually their muscle strength is insufficient to generate an adequate internal hip extension moment.

With an appropriate AFO-footwear combination the origin of the reaction force is maintained more posteriorly, the line of action also is moved more posteriorly generating a reduced external flexion moment, or sometimes behind the joint generating an external extension moment. This effect reduces the demand on the hip extensors down to a level which can be achieved thus preventing the collapse, or even *assists* the muscles when the external moment is in extension. In addition it is likely that this reduced demand also enables the child to achieve finer control of the muscle activity and therefore improve the quality of the gait pattern. Gait analysis reveals that the actual linear shift of the line of action at the hip may be relatively small, perhaps less than 20mm, which indicates why the child's gait can be influenced by relatively small changes in the angle of the AFO or details of the footwear adaptations. For instance the use of a rocker sole can easily achieve a posterior shift in the location of the ground reaction force of more than 20mm.

So far this discussion has dealt with what might be termed the "external characteristics" of the AFO-footwear combination(88). This includes the influence on external moments, the alignment of all the lower limb segments, including their relationship to the ground, and the nature of the associated footwear. However it is also interesting to consider the effect of the "internal characteristics", ie the control of the AFO on the ankle-foot complex itself. This is both the relationship of the segments encompassed by the AFO and the forces required to maintain this.

Concern has often been expressed by sceptics of the use of AFOs that the imposition of a "brace" in a neurological condition such as cerebral palsy may stimulate muscle activity, increase spasticity and result in the generation of unacceptably high interface forces. The author's personal clinical experience suggests that in most cases this does not appear to happen assuming that both the internal and external characteristics of the AFO are optimised for an individual child (88).

In order to investigate this situation a small trial was conducted involving the use of a purpose-built force transducer located between the ankle strap of the AFO and the dorsum of the child's foot (Meadows, 1984). This permitted measurement of the magnitude and duration of the forces required to resist plantarflexion of the child's ankle. The conclusion of this pilot study was that, contrary to concerns, the muscles remained relaxed during periods of inactivity by the child, in other words tone was not being increased by the presence of the AFO. During periods of activity the forces required to resist plantarflexion and maintain the slightly dorsiflexed setting of the

ankle were significantly lower than would have been required to resist the neuromuscular activity equivalent to toe-walking which would have occurred in the children tested without the use of the AFO. This was a very small trial and needs to be repeated on a larger scale to obtain any firm conclusions! However it did suggest that far from increasing tone a tone reduction effect might occur and that the principal internal effect of the AFO is to gently retain the corrected position of the ankle rather than resisting large muscle forces.

The AFOs involved in this trial were relatively simple designs when compared with more recent developments with "tone reducing" AFOs. It is probably reasonable to assume that similar effects could be expected with tone reducing AFOs and tone reducing casts. It would be interesting to measure these to see if they are any better!

In conclusion it can be seen that the biomechanics of cerebral palsy gait is different from normal children and that the effect of one form of intervention - AFOs - can be dramatic. It can be seen that changes in the characteristics in the AFO-footwear combination can alter the internal and external "biomechanical environment" within which the child has to function (88). It is also possible to appreciate that significant changes can be brought about by relatively small changes in prescription.

DISCUSSION

From the above experience it can be seen that the biomechanical demands being place on the child with cerebral palsy are significant. The neuromuscular system has to respond to this demand as best it can within its functional limitations. Experience with one form of treatment - AFOs - indicates that these biomechanical demands can be modified altering the demand on the child's neuromuscular system and therefore its ability to cope. It is proposed that this is a mutually interactive process with alterations in the response of the neuromuscular system in turn influencing the biomechanical picture. It is believed that the role of AFOs is to modify this interactive process externally so that the child can cope with the demand, gain better voluntary control, and by a process of learning make further, longer term improvements in function (9, Butler and Major, 1992).

It is therefore considered reasonable to postulate that other forms of treatment, eg orthopaedic surgery or physiotherapy, will have similar biomechanical influences. For instance the principal effect of a TA lengthening may be to achieve a more posteriorly located ground reaction force in late stance and thus influence the moments generated at the hip. The most significant result may thus be the generation of an adequate second peak of ground reaction force with all the associated benefits described above rather than the more "simple" achievement of "getting the heels down".

Similarly, when a physiotherapist is handling a child during a treatment session he/she influences the alignment of the various segments of the legs and trunk. The physiotherapist can sense when the child is more relaxed and in better control of muscle activity resulting in improved gait pattern. It may well be that the reasons for this improvement are biomechanical - in a similar way to that illustrated previously with AFOs. A physiotherapist may thus be considered to be a "controller of the biomechanical environment".

An alternative description of a physiotherapist in this context might be an "intelligent orthosis" on the basis that whilst many of the biomechanical effects attained by the physiotherapist are similar to those attained by an AFO, he/she has the added benefit of sensing the reaction of the child's neuromuscular system and can modify the input accordingly. In this way the optimum biomechanical environment can be achieved unlike the situation with an AFO whose characteristics are selected beforehand, which cannot respond to the child's neuromuscular reaction, and which can only be modified off the child!

Using a similar analogy an orthopaedic surgeon may be considered to be an "adjuster of the biomechanical environment" since again, like AFOs, there is no opportunity for on-line feedback of the child's reaction to the surgery. Unfortunately tendons cannot be lengthened and shortened until the optimum response is achieved! Usually a one-off adjustment is all that is possible. At least with AFOs adjustments can be made off-line until the best compromise can be determined by an iterative process.

There is an obvious need to conduct biomechanical studies to examine the effects of other forms of treatment in cerebral palsy. However the author believes from his experience in the management of children with cerebral palsy that there is likely to be significant biomechanical commonality between the various forms of treatment recognised as having benefit in cerebral palsy. It is difficult and dangerous at this stage to suggest that one form of treatment may be "better" than any other. However it is likely that children with cerebral palsy might benefit from combined programmes using various forms of treatment whose biomechanical influences are similar or at least compatible.

The author has to confess to his own particular "biomechanical paradigm" by which he believes that *most* forms of treatment -eg physiotherapy, orthotics or orthopaedic surgery- are primarily biomechanical influences to which the child's neuromuscular system responds, interacts and, hopefully, adapts.

Whatever the nature of the treatment programme adopted, the target of "dynamic efficiency" with good control and low energy consumption remains the same.

REFERENCES

Butler P B and Major R E. The learning of motor control: biomechanical considerations. Physiotherapy 1992 78, 1-6

Hullin, M G. The biomechanical assessment of gait in children with neuromuscular disorders. MD Thesis, University of Cambridge, 1993.

Mann, A.M. Maturation of normal child gait. Personal communication, 1994. (PhD Thesis, University of Strathclyde, Glasgow, in preparation).

Meadows, C B. The influence of polypropylene ankle-foot orthoses on the gait of cerebral palsied children. PhD Thesis, University of Strathclyde, Glasgow, 1984.

HOW DO THERAPISTS ACHIEVE THESE AIMS?

Roslyn Boyd BSc BApp Sc

INTRODUCTION

In discussing how physiotherapists achieve these aims it is important to recognise how much our profession has progressed in the last few years, towards use of current scientific evidence on the effects of adaptive soft tissue changes, the progression of deformity and our changing understanding of motor control.

The aims of the use of lower limb orthoses have been stated as:

- 1 To prevent and/or correct deformity
- 2 To provide a base of support
- 3 To facilitate training in skills
- 4 To improve the dynamic efficiency in gait

These aims are clear and appropriate for children with cerebral palsy, in helping them to overcome their problems with motor control. They focus on several features of the upper motor neurone syndrome such as:

- weakness
- increased muscle stiffness
- contracture
- lack of dexterity and poor co-ordination

Professor Roberta Shepherd has offered some changes to these aims in order to reflect the changing view of the clinical practice as it becomes more scientifically based.

As deformity is a sequel to soft tissue stiffness and lack of extensibility combined with skeletal growth, then:

Aim (1) should become "To prevent and/or correct adaptive soft tissue and skeletal changes which may lead to deformity."

Aim (2) remains "To provide a base of support". This aim needs to be investigated further to account for our increasing understanding of the literature on the development of postural adjustments.

Aim (3) should become "To facilitate motor training and the learning of functional tasks (or skills)."

As it is important to make clear that gait is only one of many actions that need to be dynamically efficient. Activities such as reaching, sitting to standing, stepping up onto a step are equally as important and should be trained and analysed more closely.

Aim (4) should therefore become "To improve the dynamic efficiency of functional motor actions including gait."

It is important for us to ensure that the use of a device enhances rather than restrains progress. There is a greater need to test the carry over effects of orthoses to account for the influences of growth, changes in body weight and height in the longer term. The effects need to be tested in and out of the orthoses in the immediate and longer term.

Dr Shepherd felt that the order of aims should also reflect a more dynamic view of orthotics to become, in priority, Aim 2,3,4,1.

However, the order of priority may be influenced by the expected level of functional skill in the group of children with cerebral palsy being considered. On this basis two groups of children may be identified:- static and dynamic.

The "Static" group includes those with a low level of independent functional skills that are not likely to achieve independent mobility. In this group there needs to be a greater emphasis on prevention and correction of soft tissue and skeletal changes and deformities. As hip dislocation in a patient with cerebral palsy results in significant morbidity in terms of pain, Cooperman et al (1987) reports up to 50% in adult life, then the prevention of dislocated hips and spinal deformity is a priority and must be addressed early in the child's life.

The aims of the orthoses in this static group also are to provide a base of support for interaction/communication, visual skills, upper limb skills for activities of daily living and assisted mobility as well as independent feeding.

It is vital to be realistic in setting aims and whilst training in functional skills is still important that it should not be at the expense of preventing deformity. This is the group of children which the physiotherapist and orthotist often have the most difficulties in managing.

The "Dynamic" group of children who are those expected to achieve some independence in upright standing and possibly mobility. In this group there should be a greater emphasis on:

- Aim (3a) "Facilitating motor training and the learning of functional tasks and skills.
- Aim (4a) "Improving the dynamic efficiency of motor actions including gait".

The use of lower limb orthotics in the dynamic group are sometimes used by physiotherapists aimed at providing a variable range of movement in order to:

- provide support for weak muscles
- opposition for "spastic muscles"
- to enhance muscle actions
- to protect muscles post surgically

• to improve the appearance of gait parameters when the patients lack selective control.

The prevention of adaptive soft tissue changes and skeletal deformity would also be important, but this aim may well be achieved by concentrating, very early on, aims (3) and (4). Functional motor training or other interventions to improve the biomechanics of the movement so that deforming forces may be reduced in order to prevent later problems.

THE CHANGING ROLE OF ORTHOTICS IN PHYSIOTHERAPY

There have been two dominant schools of thought for physical therapists over the last forty years. There has been a strong emphasis on neurodevelopmental treatment according to Dr and Mrs Bobath, particularly in North America and the United Kingdom. There has been an emphasis on "hands on therapy" aimed at reducing spasticity and facilitating "more normal movement".

The use of orthotics and techniques of serial casting were less in favour with this group than the second group represented by the work of Phelps, the more orthopaedic approach to management. This attitude appears to have limited the physiotherapy collaboration with Orthotists and Bioengineers in developing a more productive relationship.

The Orthopaedic group emphasised the use of callipers, boots and irons and serial casting to provide support for the limbs for upright weight bearing and to attempt to influence the progress of deformity. Both these approaches have changed to move with current times, with increasing surgery and greater use of orthotics in many clinical situations.

The two groups were fairly diverse in their nature of approaching the problem of movement in the cerebral palsy child, but have now started to come closer together as a scientific knowledge and changes and improvements in technology have provided more options. The greater scientific knowledge also encourages questioning and examination of the basic assumptions underlying clinical practise.

In those days it was as Disrelli said, a situation where "people argue the most about things they know the least about", the problem being that there was little scientific evidence for much of the accepted practice.

Paediatric Neurological Physiotherapy practice has been largely based on the experience of clinical practice and on techniques and methods, handed down from senior therapists. There has been little effort to achieve satisfactory objective measurement tools.

As more sophisticated measurement techniques become available it is necessary to learn to adapt these for use in the clinical situation and more accurately measure the outcomes of various treatment approaches including the use of orthoses. Expertise from the field of biomechanics, applied biomechanics and kinesiology can help us in measuring parameters that can be quantified, such as force production, torque, kinematics and kinetics. Over the last 20 years there has been a strong emphasis on orthotics aimed at supporting the limb in more normal positions and preventing deformity. This led to designs aimed at mimicking functional positions such as subtalar neutral in the foot in an attempt to reduce spasticity. There has been an enthusiasm more recently for dynamic orthotics allowing movement in a controlled range.

Current literature on control of neural input to muscles challenges previous ideas on therapeutic practice which were directed at reducing spasticity. The recent literature on motor learning gives ideas for modifying and fine tuning the use of orthoses to achieve the various aims.

NEURAL MECHANISMS

Recent evidence in upper motor neurone syndromes has indicated that movement disorders resulting from a neural lesion may be compensatory, rather than being the primary problem. An attempt by remaining tissue to compensate for loss of neural tissue, which manifests itself as a learned pattern of movement.

In a study by Neilsen and McCaughey (1982) a group of adults with cerebral palsy learned to decrease the muscle tone around their elbow, but this did not lead to a significant functional improvement as would be expected if the agonist muscle were inhibited by an overactive antagonist.

The work of Sahrmann & Norton (1977) produced some evidence that spasticity may not be a direct cause of the disordered movement. Slowness of the movement resulted directly from insufficient muscle.

Dietz (1983) noted that children with cerebral palsy demonstrated a heightened threshold to activate the stretch reflexes in spastic muscle and there was a high degree of co-contraction occurring during stance phase to get stability while the other leg was off the ground. This co-contraction has also been noted developmentally in young infants as a normal developmental phenomena (Vaughan 1992).

Vaughan (1992) felt that co-contraction accompanies both spasticity and also postural instability. It may be a natural compensation to restrict freedom of movement of the limb and protect joints from unpredicted perturbations. When adults learn a new skill they use co-contraction to reduce error and control the movement. (Martenuik et al 1987).

Burke (1988) explores this from a neurophysiological viewpoint and argued that the primary problem is decreased cortical input to the underactive muscle. Further work is necessary in children with cerebral palsy. Enhancement of techniques in electromyography helps in looking at the phasic action of certain muscle groups in the gait cycle, but does not quantify the degree of output of motor units. Future developments in this area will hopefully give greater clarity to these ideas.

DYNAMIC SYSTEMS APPROACH TO MOTOR DEVELOPMENT

It is useful to look at the problems in movement for the cerebral palsy child in terms of the "Dynamic Systems approach to Motor Development" by Kamm, Thelen and Jensen (1990).

This approach helps physiotherapists to take account of all the following subsystems and their interactions:

- neural mechanisms
- muscle biology and skeletal developmental
- motivation/arousal level
- physiology and biomechanics

All within an environmental context.

There has been an overemphasis on the neural mechanisms subsystem in therapeutic practice which has often confused the aims of treatment and led to a lack of consideration of the other subsystems.

Heriza (1991) emphasised muscle strength, body build and the effects of gravity on movement of skills which is a useful modification of this theory when considering problems of the child with cerebral palsy.

Zelazo (1983) found that it was possible to retain automatic walking reflexes in babies by practising of the reflex. Thelen and Fisher (1982) provide a good example of this in their comparison of the kinetics of spontaneous kicking in infants and new-born stepping. they found evidence that the two movements had similar kinematics.

Although there are similar requirements to perform both behaviours, new-born stepping disappears while kicking retained. Thelen and colleagues suggested biomechanical reasons for this, as the effects of gravity are different in standing to those in supine. It is important to consider the relationship of the moving parts to gravity. It may be assisted or inhibited by gravitational forces, therefore the kinematic differences between kicking and stepping may be the result of contextual differences in relation to gravitational forces (Van Vliet, 1992).

In standing, because gravity acts differently, it may be more difficult to produce the stepping movements. It is important for a therapist to apply this sort of analysis to the management of neurologically impaired children where an orthosis would be necessary to provide a basis of support for training and to reduce the effects of how forces act upon the limb and body segments or to enable the continuation of practice of certain movements, in positions that the child cannot achieve independently.

Therapeutic practice should not be geared to achieve stability in certain positions such as standing as a pre requisite before training stepping movements. It is important to practise both activities concurrently even if the movement cannot be performed in an ideal pattern. It is necessary to practise stepping for later transfers and upper limb skills in more upright positions rather than waiting to achieve control of standing in suitable alignment. Orthoses will be useful in this practice, eg even bicycles can mimic some of the motor actions required for stepping.

The David Hart Walker has recently been developed in England and its main benefit is that it enables the continuation of practice. The physiotherapist cannot propose that this orthosis will lead to independent walking but it does enable dynamic maintenance of muscle length in the "Static Group". It may be useful in younger children under 2 years to enable more practice of walking than caregivers are able to provide.

Orthoses can also be used to provide a stable base enabling body or limb support for activities, this enables the training of motor tasks, training of postural adjustments, the active maintenance of muscle lengths and the opportunity to achieve success when it was otherwise not possible.

Current evidence questions the assumption that decreasing spasticity will allow normal movement to occur and suggests that spasticity and abnormal movement patterns may not be the only problem. Often weakness and lack of neural input to the biomechanically disadvantaged muscle may have a greater effect. It seems that the focus should be on training movement rather than decreasing spasticity.

Some of the recent literature on motor learning in adults can be applied to practice, however it must be remembered that children with cerebral palsy may not learn in the same way. This literature may establish exactly how children with cerebral palsy learn new tasks, and give indications for assisting learning.

It is important to recognise all the problems that influence muscle control in attempting to understand the complexity of the problem with the neural mechanisms (Sugden and Keogh, 1990). These problems of muscle control include:

- control of single motor units
- muscle tone and spasm
- organisation of movement
- signal detection in sensory receptors.

THE BIOMECHANICAL SUBSYSTEM

There has been a long history of use of physiotherapy techniques to alter muscle length and to attempt to reduce "muscle tone".

Serial casting techniques have had mixed popularity over the years at it was observed that the effects often did not last as the child grew and sometimes the children were weak and lost functional skills after casting. So often there has been a "recipe for treatment" of two weeks in casts then change and to continue for a total of 6 weeks. This often resulted in a loss of standing and walking abilities due to weakness or possibility of a "rebound" effect due to overstretching.

Current literature on the adaptive soft tissue changes and the mechanism of deformity help to give more clear information on the effects of such techniques as serial casting. These techniques need to be fine tuned to achieve a better result. Muscle length factors are very important in influencing how well a movement can be performed. Muscles adapt their length if immobilised in a position for a considerable period of time, beginning to change their structure after only 24 hours. In shortened muscles, sarcomers will decrease in number and increase in individual sarcomere length (Grossman et al 1982). Animal studies have demonstrated that the primary effect on muscle length occurs within the first 48 hours after which the effects of atrophy are seen (Williams and Goldspink 1973).

The physiological consequences of this are that the length /tension relationship of the muscle changes so that the maximum tension is now developed in a shorter range than before. Clinically, many examples are seen of this, but may be attributed mostly to neurophysical factors.

SERIAL CASTING

Reported effects of serial casting in children with cerebral palsy include:

Hylton (1984) felt that tone could be reduced by serial casting and which was shown when the child showed increased passive movement or either a heel/toe or flat footed gait. It was assumed that spasticity and hypertonia could be changed to that which is described as "normal" by providing specific inputs (Carlson 1984).

Scientific evidence for the central effects on muscle tone with such techniques as serial casting were not conclusive. Studies of the biomechanics of gait and the changes have been much more useful and objective (29, Bertoli 1986).

Herbert (1988) describes the shift in the length/tension relationship over time. This curve will rapidly resume its previous position once immobilisation ceases, if a new range of movement is not elicited. The effects of casting have often been seen to diminish and disappear over time, presumably as the muscle re-adapts to resume its previous length/tension relationship.

Motor training should be undertaken to utilise the achieved range and help to achieve a new dynamic control of muscle action and help to overcome the problems that occur with growth in the child with cerebral palsy.

Serial casting should now be regarded as a technique to immobilise the muscle and bone, in a certain position and to achieve a change in the length/tension curve, if the muscle is able to adapt to the new condition. Tardieu et al (1982) reported two types of muscle, one which reacted in this way and another group of muscles which reached a plateau. This requires further investigation as the clinical pnenomenum of overstretching resulting in rebound may be evidence of this. It is necessary to determine the optimum tension for effectively stretching the muscle and develop a clinical tool for measuring this. It is also important to look at the timing of our use of serial casting. Serial casting is a cheap and efficient tool for elongating the muscle, where there is a static muscle length problem which can still respond to stretch.

Serial casting has probably become less popular, especially with orthopaedic surgeons because the muscles were overstretched or weakened and appropriate training was not

conducted afterwards for a portion of each day to achieve dynamic control of the new length.

It is important to re-adapt our casting techniques to repeated serial changes of cast over a three to five day period once a week instead of the one to two weeks previously adopted in many clinics.

MAINTENANCE OF CORRECTION

Serial casting can be an effective technique for lengthening the muscles, but however it is also important to take into account the maintenance of this correction if the effects of the casting are to be maintained. The child must then learn to recruit altered patterns of muscle use, which will be reflected in functional movement and more stable posture.

A more dynamic muscle balance must be achieved to maintain the effects of casting and this may help to overcome the problems that occur with growth in spastic muscles. Ziv (1984) reported that muscles in spastic mice grow at 55% of the rate of muscles in normal mice.

The management of static muscle contractures require continuous stretching initially to alter the muscle length, achieving maximum elongation within 48 hours in a given position. Once lengthened the muscle requires at least 6 hours of stretching in 24 hours in order to be able to maintain this length (Tardieu et al 1988).

Recent studies by Williams (1988) in animals suggested a lesser time of 2 hours in 24 hours only would be necessary in order to maintain muscle length. Tardieu recommended an optimum passive tension. With such diversity in optimal timing and required tensions further investigation is required.

Clinical practice has been influenced by this work in the United Kingdom. Evidence that more growth hormone is released at night suggested that serial stretching should be undertaken at night.

The physiotherapist must balance up a stretching programme for an optimal time each day with the motor skills she will teach the care giver to practice with the child each day. In the dynamic group night time bracing may be useful in allowing the child to be active for most of the day. However, the therapists must consider which muscles can be effectively stretched, eg soleus muscle in ankle foot orthoses (AFOs), gastronemius muscle with knee ankle foot orthoses (KAFOs) but this will not influence the hamstring muscle.

Parents find it difficult to administer our regimes of night time bracing. Better designs of orthoses for comfortable night wear still enabling change of position are required for the "static group" of children. They require the maintenance of length of the hip adductors on a stable pelvis as a priority for night bracing as early as the first two years of life. The effects of stretching muscles in a "loaded" or weightbearing position as opposed to an unloaded position have not been reported. The advantages of motor learning by dynamic use of orthotics must also be considered when preventing soft tissue changes.

In the author's clinic it has been found very useful when examining the child to make a subjective analysis of the static and dynamic stiffness of muscles. Using the techniques of (Reimers 1980) dynamic muscle stiffness is also assessed by looking at the "catch" range of movement. The limb is moved passively and quickly through a complete range of movement and a "catch" may be felt at a particular point. This is useful in determining in a clinical setting the functional range of movement that the children optimally appear to use. It is often an early clinical indication of an impending contracture. These measures, along with standardised measures such as Migration Percentage (MP) (Reimers 1980) and acetabular index (Hilgenreiner, 1925) are taken to monitor the progression of hip deformity.

A prospective study of hip development in children with motor problems is currently being conducted to determine the natural history of the disease (Scrutton and Baird, 1991). This study includes standardised X-rays at approximately 6 monthly intervals. Early results indicate the need for early intervention. Clinical indicators include dynamically tight adductors and medial hamstrings. Physiotherapists need to be aware of this in their programmes of motor training for children under 1 year.

Early results support the commencement of orthotics at less than 2 years before extensive skeletal remodelling has occurred under the effect of deforming muscle forces.

Fine tuning of clinical practice in use of conservative techniques can be demonstrated by the following clinical example:

The child with equinus gait or toe/toe gait may have a specific muscle contracture in his gastronemius which may benefit from serial casting, using 48 hour periods immobilised in serial cast with enough stretch to achieve passive tension until clinically applicable range of movement at the ankle joint has been gained. This needs to be followed by the use of various orthoses to retrain motor control and selective activity of muscles in the new range.

It is also important to consider the differences between stretching young and old muscles. Tardieu (1977) reported in animals the effect of stretch on young and adult muscles. In the first five days of stretch, both demonstrated an increase and addition in sarcomeres. An increase in the overall muscle length continued to occur in adult muscle. However in the younger individual there was elongation in the tendon to accommodate the inability to stretch the muscle.

This may demonstrate why some children who spend a long time in ankle foot orthoses with sustained stretch have short muscle bellies and long tendons on their gastrocnemius. This means that it is necessary to be selective in the timing of use of fixed orthoses to enable some active recruitment of muscles through the full range. Immobilisation also shows an increase in the perimysium and endomysium (Tabary et al, 1972 and 1981, Williams and Goldspink, 1984). Some reports emphasise how the change in orientation in collagen fibres leads to the reduced ability of the muscle to extend. These changes in orientation figures may account for the changes in muscle stiffness. It is important for the child to clinically maintain full excursion of the limb segment as well as practising fine control of the movement within a specified range.

The author's centre has used Botulinum Toxin to assess the effects of surgery for diagnostic purposes. Cosgrove and Graham (1994) have reported improved gait parameters and reduced energy cost eluding to enhanced muscle growth in children with cerebral palsy. The long term effect on the muscle is not known, but it would be useful to conduct a comparative study between serial casting and the use of Botulinum Toxin with appropriate functional measures.

Further evidence of the effect of muscles on application of serial casts and surgical lengthening of tendons in cerebral palsy children is necessary. Tenotomy on growing muscles in animal studies (Goldspink et al, 1974) has demonstrated effects on muscle growth and there would be an expected decrease in afferent input to the muscle.

Thus achieving a balance between conservative and surgical treatment is necessary in the cerebral palsy infant.

Physiotherapists must recognise the need for bony surgery, particularly for rotational deformities which cannot be altered by conservative means. Orthoses such as twisters will rarely impact on the deformity and will often lead to overstretching of the knee ligaments. These orthoses may be useful where surgery to correct the deformity may not be a realistic option (some children may not tolerate or co-operate with post operative management).

Post operative use of casting and orthoses have often involved extended periods of immobilisation leading to weakness and increased muscle spasms when they eventually come out of the casts. Internal fixation of bony surgery would enable early active assisted movements and assisted active movements. Continuous Passive Motion (CPM) machines have been used with considerable success in the author's clinic post operatively, particularly after multilevel bony surgery. This technique appears to have reduced weakness and incidence of muscle spasm resulting from long periods immobilised in plaster of paris spikas. Post operative management places greater emphasis on strengthening weak muscles. Orthoses are used to vary the range of practice with more confidence that the joint will maintain a stable position.

The study of Butler et al (9) is an excellent example of the possible training effects of fixed ankle foot orthoses (AFOs). Six children in fixed AFOs had their footwear tuned after one month of wear. Tuning was designed to correct the biomechanical environment to allow motor learning to occur. Footwear was tuned by analysis using Force Vector Visualisation (two dimensional) system. The result was reassessed three months after the AFOs had been weaned off. The benefits of the study were in the motor learning effect, however the effects of growth need to be considered and whether the technique will enable maintenance through rapid growth periods. The implications for physiotherapists is in working closer with our bioengineering colleagues and designing motor training with these principles in mind.

The two dimensional Force Vector System (Stallard 1987) is now portable for use in many situations and will give us a more accurate measure of biomechanical effects. The limitations of 2 dimensional analysis must be considered. This technique provides a useful clinically available measurement tool which will be much more effective than our current subjective visual estimation of a correct biomechanical alignment.

In addition to a greater need for dynamic orthoses to practice controlled ranges of movement it is necessary to consider the importance of variability of practice to achieve better retention of skills and the ability to still perform in an altered context.

It is necessary to consider the effects of training in a variety of situations for generalisation of the skill to occur. Physiotherapy is aimed at designing a specific motor task for carers to train. The child needs specific motor tasks with appropriate targets. Walking in the controlled context supplied by rigid AFOs must be balanced with targeting specific movements such as stepping on and off a step at different heights and walking on uneven surfaces.

Evidence from the motor learning literature encourages practice under different conditions so that the individual can get better at perceiving what critical features of the environment they have to attend to. Practice needs to be conducted throughout a portion of the child's day to enable retention of skills. This is where physiotherapists and the carers that they train cannot use their hands as "intelligent orthoses" for significant enough portions of the day and variable practice can be achieved with the aid of appropriate orthoses.

Behavioural factors such as the use of feedback and whether the environment is conducive to learning also influence how patients perform. Altering the environment by placing the target (eg a toy) appropriately for the required action may be more effective learning than manual guidance. Orthoses can help to set up support for this targeted training.

Another assumption underlying much of our clinical practice is that recovery from brain damage followed by an orderly sequence of development (Gordon 1987).

This has led to a somewhat rigid application of neurodevelopmental sequence in treatment, for example it may be necessary to achieve stable sitting balance before progressing onto dynamic sitting balance.

Research by Von Hofstein (1982) looking at the development of reaching skills in young infants and early fine motor actions showed that in the normal infant there is "in parallel" development of proximal and distal control of the upper limb not proximal to distal as previously described by Bobath. This suggests a more flexible approach should be taken as to the order in which different tasks are trained in children with cerebral palsy.

Orthoses could be important here in providing a base of support for stability of certain segments in order to try and gain more control over other segments.

Butler and Major (1992) proposed "Targeted motor training in upright in children with motor problems". This model is based on biomechanical principles rather than a

neurophysiological model. They designed equipment to support the trunk with a gradual increase in the degrees of freedom allowed. After practice, support is reduced to a lower level so that voluntary control can be trained again with another degree of freedom of movement. The concept is important in enabling the child to practice voluntary actions under the influence of gravitational forces by himself for longer periods and not necessarily with the aid of manual guidance. It must be considered whether practice of the whole movement immediately after this training is necessary as accurate controlled movement can only be learnt through the experience of the whole movement.

Another simple example is given by comparing slow and fast adduction movements of the arm in standing. In this example, performing the same movement at two different speeds results in different muscles performing the movement. (Van Vliet, 1992). An example of an environmental constraint on movement is given by the example of standing up from a high versus a low chair. From the low chair, the quadriceps muscle group must generate a greater torque to extend the knee than from a higher chair.

Hirschfield (1992) recently reported the use of body shells as a base of support to enable the practice of actions in standing and the resultant development of balanced standing. A 12 year old diplegic child had EMG measured on his leg muscles while holding onto a walking aid in standing with or without a shell orthosis to support his trunk and legs. Standing without the shell all leg muscles were activated continuously.

In the shell orthosis EMG in leg muscles approximated to "normal" and the child did not need to hold onto the walking aid. It is likely that holding onto a support in standing causes postural adjustments which in unsupported standing would usually involve all the lower limb muscles to ensure balance appeared to be using lower muscles to provide a rigid pillar and prevent collapse rather than ensure both support and postural adjustment.

Studies by Nashner et al (1983) looked at the postural adjustments that occur in normal and cerebral palsy children when their balance is perturbed. Normal children responded distally and then more proximally whereas children with spastic cerebral palsy responded proximally with hip flexors before distal initiation of the dorsiflexors to restore balance. In training normal postural adjustments as physiotherapists we must consider that training may be restricted by fixed AFOs. More work is required in this area and consideration given to its therapeutic implications.

Orthoses may be used in the management of hip problems in cerebral palsy children.

The hip abduction and spinal orthosis (HASO) as described by Bower (1990) and modified by Drake (17) was developed as an alternative to special seating devices which have provided function sometimes at the cost of development of deformity. There is only one commercially available seating system in the United Kingdom which provides more than 10 degrees of abduction of the hip in sitting. The HASO was modified to provide more than 10 degrees of abduction of the hip in sitting. The HASO was modified to provide better pelvic control and greater hip abduction to maintain the length of the hip adductors for a greater portion of the child's day as well as providing a stable and symmetrical base for learning upper limb tasks. Follow up of children using the HASO for at least 5 years has demonstrated a maintenance or improvement of hip migration (MP) according to Reimers (1980) and acetabular index (Hilgenreimer, 1925). If bracing was commenced at the appropriate time (at 40% migration) in the static group of children, these children are not expected to achieve independent sitting or standing.

The HASO has maintained post operative hips after soft tissue surgery and bony surgery if there is compliance with use. A subjective evaluation of functional skills by questionnaire has demonstrated at least a maintenance if not an improvement in skills.

An indication for earlier follow up has been demonstrated by a review of results of soft tissue surgery in the author's clinic (Cornell et al 1994). The success of outcome of soft tissue surgery in the author's unit depended on the amount of hip migration and the acetabular index as well as compliance with post operative bracing. Success was not related to age at surgery. The results of this study demonstrated a need for the commencement of treatment before migration percentage moved beyond 40%.

In the functionally less able group of children (Static group) the implications are that this amount of hip migration may develop within the first few years of the child's life so that objective measurement is essential and appropriate postural management is required earlier. Physiotherpists should be at the forefront of measurement and preventative postural management.

The dynamic group of children with cerebral palsy with migrating hips of over 40% appear to benefit from use of the Meyer Brace as designed by Dr Paul Meyer in Cambridge (Meyer, 1990). This brace is currently being trialed on a group of 15 children at the Newcomen Centre. It appears to offer exciting possibilities for maintaining length in the hip adductors in those children who demonstrate dynamic tightness. The brace enables independence in practising skills in a required range of abduction. The ability of the brace to achieve functional carryover with improved muscle balance between the hip adductors and abductors is not yet known.

This brace has been clinically very useful for children who require a wider base of support and may have poor clearance due to scissoring gait. It provides similar possibilities to the Rancho Hip Brace, but is lighter in weight and allows greater freedom of movement. It has been developed in three sizes and enables sitting, rolling, standing and stepping where the child has sufficient control with a maintained range of abduction. It is not designed to control internal rotation of the hips, but can influence it.

MEASUREMENT TOOLS

The physiotherapist requires objective and user friendly clinical tools for measurement of aims of management. The benefits of 3 dimensional gait analysis have been demonstrated in examining the effects of orthoses eg: Posterior Leaf Spring Orthosis (Ounpuu et al, 1993) and the results of surgery. Such laboratories may not be readily available or may be too costly for measurements of some conservative treatments in a representative group of patients.

The options available must be considered and collaboration with bioengineers established to develop new measures.

Suggestions for the objective measurement of muscle tone include the modified Ashworth Scale (Ashworth, 1964, Bohannon and Smith 1964). The Lidcombe template (Mosely 1991) uses a known torque applied to the ankle in a given direction and the range of movement at the ankle joint is measured. Tardieu (1982) used a similar device but ensured relaxation of the triceps surae by ischaemia of the muscle to ensure the measured passive torque. The Chaucer Template development at the University of East London, measures wrist flexors in a similar way.

EMG is useful in looking at the phasic action of muscles and whether they are continuously activated or acting in appropriate timing with the movement (57).

Force Vector Visualisation (Stallard 1987) is a useful objective measure which uses real time sychronisation of joint moments during visual representation of gait. It is a useful tool which is now available as a portable system.

Functional measures such as the Gross Motor Function Measure (GMFM) (Russel et al 1989) looks at 88 items of motor function to quantify functional ability. It is not a qualitative measure.

Two dimensional video analysis enables closer analysis of the events of a movement action. When evaluating motor actions it is necessary to consider timing muscle action, the degree or error and the target expected. Fetters (1990) suggested the use of movement units which looks at the portion between one acceleration and one declaration of a total reaching task.

CONCLUSION

Physiotherapists must utilise the scientific literature on biomechanics, the effects of soft tissue adaptations and motor learning. The profession has moved from therapeutic practice based on artistry to more scientifically based clinical reasoning.

With their expert training in the study of the quality of movement and how to train movement they may be considered "Movement Scientists". The scientific literature helps them to "fine tune" their techniques and become more effective in long term carry over. They may no longer rely on their hands as "intelligent orthoses". They must instruct care givers in appropriate training of functional motor tasks and work with their colleagues in other disciplines to develop clinically objective measures.

Their aims in use of orthoses and the designs used may not alter significantly in the future as there are already many sophisticated designs to choose from. However, more advanced measurement tools are available to enable selection and fine tuning of training in orthoses.

REFERENCES

Ashworth B. Preliminary trial of carisoprodol in multiple sclerosis. The Practitioner 1964 <u>192</u>, 540-542.

Bertoli D B. The effect of short leg casting on ambulation in children with spastic diplegia. Archives of Physical Medicine & Rehabilitation 1986 <u>70</u>, 481-489.

Bohannon R W and Smith M B. Interrater reliability of a modified Ashworth scale of muscle spasticity. Physical Therapy 1987 <u>67</u>, 206-207.

Bower E. Hip abduction and spinal orthosis in cerebral palsy. Physiotherapy 1990 <u>10</u>, 658-659.

Burke D. Spasticity as an adaptation to pyramidal tract injury. In Waxman S G (ed) Advances in Neurology, 47. Functional recovery in neurological disease. Rovan Press, New York, 401-423, 1988.

Butler P B and Major R E. The learning of motor control: biomedical considerations. Physiotherapy 1992 78, 1, 6-11.

Carlson S J. A neurophysiological analysis of inhibitive casting. Physical & Occupational Therapy in Paediatrics 1984 4(4), 31-42.

Cooperman D R. Hip dislocation in spastic cerebral palsy: long term consequences. Journal of Paediatric Orthopaedics 1987 <u>7</u>, 268-276.

Cornell M, Spencer J S, Boyd R, Baird G, Collins D. A review of soft tissue surgery in cerebral palsy. (in press) Developmental Medicine & Child Neurology 1994.

Cosgrove A P and Graham H K. Botulinum Toxin A prevents the development of contractures in hereditary spastic mouse. Developmental Medicine & Child Neurology 1994 <u>36</u>, 379.

Dietz V and Berger N. Normal and impaired regulation of muscle stiffness in gait: a new hypothesis about muscle hypotonia. Experimental Neurology 1983 <u>79</u>, 680-687.

Fetters L. Measurement and treatment in cerebral palsy: an argument for a new approach. Physical Therapy 1990 <u>71</u>, 244-247.

Goldspink G, Tabary C, Tabary J C, Tardieu C, Tardieu G. Effect of denervation on the adaptation of sarcomere number and muscle extensibility to functional length of the muscle. Journal of Physiology 1974 <u>236</u>, 733.

Gordon J. Assumptions underlying physical therapy intervention. In Movement Science: Foundations for Physical Therapy in Rehabilitation. Carr J H, Shepherd R B, Gordon J, Held J H. Aspen, Rockville 1987.

Grossman M R, Sahrmann S A, Rose S J. Review of length associated change in muscle. Physical Therapy 1982 <u>62(12)</u>, 1799-1808.

Herbert R. The passive mechanical properties of muscle and their adaptations to altered patterns of use. The Australian Journal of Physiotherapy 1988 <u>34(3)</u>, 141-149.

Heriza C B. Implications of a dynamic systems approach to understanding infant kicking behaviour. Physical Therapy 1991 <u>71</u>, 222-235.

Hilgenreiner H. Zur Frudiagnose und Fruhbehandlung der Angeborenen Huftgelenkverenkung. Medizinsche Klinic 1925 <u>21</u>, 1385-1425.

Hirschfeld H. Postural control: acquisition and integration during development. In H Forssberg and H Hirschfeld (eds). Movement Disorders in Children, 199-208, 1992.

Hylton N M. Postural and functional impact of dynamic AFOs and foot orthoses in a paediatric population. Journal of Prosthetics & Orthotics 1983, 199-208.

Hylton N. Techniques of inhibitory casting. Printed hand out available from Nancy Hylton, Children's Therapy Centre 26461, 104th Ave. S E Kent, WA 98031, 1984.

Kamm K, Thelen E, Jensen J L. A dynamic systems approach to motor development. Physical Therapy 1990 <u>70(12)</u>, 763-774.

Marteniuk R G, Mackenzie C L, Jeannerod M, Athenes S, Dugas C. Constraints on human arm movement trajectories. Canadian Journal of Psychology 1987 <u>4</u>, 365-378.

Meyer P. Personal communication Addenbrooks Hospital, Cambridge 1990.

Nashner L M, Shumway-Cook A, Marin O. Stance posture in select groups of children with cerebral palsy: deficits in sensory organisation and muscular condition. Experimental Brain Research 1983 <u>49</u>, 393-409.

Nielson P D, McCaughey J. Self regulation of spasm and spasticity in cerebral palsy. Journal of Neurology, Neurosurgery and Psychiatry <u>45</u>, 320-330.

Ounpuu S, Bell K, Davis R, De Luca P. An evaluation of the Posterior Leaf Spring using gait analysis (abstract). Journal of Paediatric Orthopaedics 1993 <u>14</u>, 1.

Reimers J. The stability of the hip in children: a radiological study of results of muscle surgery in cerebral palsy. Acta Orthopaedica Scandinavica 1980 Suppl No. 184.

Russell D J, Rosenbaum P L, Cadman D T et al. The Gross Motor Function Measure: a means to evaluate the effects of physical therapy. Developmental Medicine & Child Neurology 1989 <u>31</u>, 341-352.

Sahrmann S A, Norton B J. The relationship of voluntary movement to spasticity in the upper motor neurone syndrome. Annals of Neurology 1977 <u>2</u>, 460-465.

Scrutton D and Baird G. Personal communication "The SETHRA Hip Project", 1992.

Shepherd R. Personal communication to ISPO "How do physiotherapists achieve these aims" 1994.

Stallard J. Assessment of the mechanical function of orthoses by Force Vector Visualisation". Physiotherapy 1987 <u>73</u>, 398-402.

Sugden D A and Keogh J F. Problems in movement skill development. University of South Carolina Press, 1990.

Tabary J C, Tabory C, Tardieu C, Tardieu G, Goldspink G. Physiological and structural changes in the cats soleus muscle due to immobilisation at different lengths by plaster cast. Journal of Physiology (London) 1972, 224-231.

Tabary J C, Tarieu G, Tardieu C, Tabary C. Experimental rapid sarcomere loss with commitant hypoextensibility. Muscle & Nerve 1981 <u>4</u>, 198.

Tardieu C, de la Tour E H, Bret M D, Tardieu G. Muscle Hypoextensibility in children with cerebral palsy. Clinical and experimental observations. Archives of Physical Medicine & Rehabilitation 1982 <u>63</u>, 67.

Tardieu C, Tabary J C, Tabary C, de la Tour E H. Comparison of the sarcomere number adaptation of young and adult animals. J Physiology 1977 <u>73</u>, 1045-1055.

Tardieu G, Tardieu C, Colbeau-Justin P, Lespargot A. Muscle hypoextensibility in children with cerebral palsy: 2 Therapeutic implications. Archives of Physical Medicine & Rehabilitation 1982 <u>63</u>, 103.

Tardieu G, Lepargot A, Tabary C, Bret M D> For how long must the soleus muscle be stretched each day to prevent contracture? Developmental Medicine & Child Neurology 1988 $\underline{30}$, 3-10.

Thelen E, Fisher D M. Newborn stepping: an explanation for a "disappearing reflex". Developmental Psychology 1982 <u>18</u>, 760-775.

Von Hofsten C. Eye-hand co-ordination in the newborn. Developmental Psychology 1982 <u>18</u>, 450-461.

Williams P E and Goldspink G. The effect of immobilization on the longitudinal growth of striated muscle fibres. Journal of Anatomy 1973 <u>116</u>, 45.

Williams P and Goldspink G. Connective tissue changes in immobilised muscle. Journal of Anatomy 1984 <u>138</u>, 343-350.

Williams P E. Effect of intermittent stretch in immobilised muscle. Annals of Rheumatic Diseases 1988. <u>47</u>, 1014-1016.

Zelazo P R. The development of walking: new findings and old assumptions. Journal of Motor Behaviour 1983 <u>15</u>, 99-137.

Ziv I, Blackburn N, Rang M, Koneska J. Muscle growth in normal and spastic mice. Developmental Medicine and Child Neurology 1984 <u>26</u>, 94.

HOW DO SURGEONS ACHIEVE THESE AIMS? I

John R Fisk MD

Bax's (1964) original definition for cerebral palsy, "a disorder of movement and posture due to a defect or lesion of the immature brain", is well to remember because of its inconclusiveness and its simplicity. The role of the orthopedic surgeon is to facilitate that movement, maintain a functional posture and prevent deformity, and remember that he or she is dealing with an immature brain and therefore will see changes as a child grows and develops. In a phrase, it is to strive for the proper indications for intervention. One must remember that an orthopaedic surgeon not only operates, he or she is intimately involved with the diagnosis, the functional evaluation and the prescription of the many other treatment modalities.

When considering surgery, there are three categories of indications; (1) to prevent problems, ie deformities, pain and loss of function (2) to improve function but when doing so we must agree upon criteria for its evaluation and (3) to provide comfort and ease the burden on those caring for the individual.

There are many good historical perspectives on early approaches to the care and treatment of patients having cerebral palsy. Such names as Little (Rang, 1966), Freud (1968) and Osler (1987) are note worthy. They all dealt with the definition of a beguiling affliction found in children. When evaluating these individuals the first and most important step is to formulate an accurate diagnosis. The clinician must rule out other similar yet progressive neurological diseases and make sure that there is not a hereditary component as is the case in hereditary spastic paraplegia. In addition, the physician must have a clear understanding of normal child development so that an appropriate evaluation is performed with a proper perspective for what is normal for age.

There are critical dynamics affecting the child and parents while proceeding through different stages of development. How the family relates to and handles the grieving process over the loss of expectations when confronted with a disabled child greatly influences their acceptance of recommended therapies.

Dr James Cary (Gage, 1991) outlined his principles for treating the handicapped child: (1) define the end product and long range objectives, (2) identify the child's problems with precision, (3) analyze growth effects with and without treatment, (4) consider valid treatment alternatives, (5) treat the whole child.

The classic forms of clinical presentation of cerebral palsy are spastic hemiplegia, spastic diplegia, total body involvement (spastic quadriplegia) and other. A single extremity involvement, unusual though occasionally seen and central hypotonia which does not involve spasticity but has the same centrally based motor dysfunction that cerebral palsy has are frequently included in other category. Seventy per cent of the patients seen at the author's centre have spastic diplegia and it is with this group that

most of the observations concerning orthopaedic intervention for lower extremity problems will be made. Sixty-five per cent of these individuals are born prematurely.

Their spasticity is a symptom of their overall disability which comes form a brain dysfunction. If this spasticity were removed their brain dysfunction and consequently their motor control disability would remain.

Spastic quadriplegia involves all four extremities. The term "total body involvement" is preferred because these individuals not only have a spasticity involving all of their limbs, they also have difficulty in dealing with secretions, problems of nutrition, communication disorders and trunk balance. No single system is any more or less important that any of the other systems and all must be addressed when making therapeutic decisions. The approach should be to achieve comfort and provide for decreased demands on care providers. This may be summarized by stating that the indications for therapy are, "creature comforts and nursing care ease". Proper foresight and planning can prevent many problems with this group.

The hips can never be ignored. If overlooked, a high percentage will ultimately dislocate. Such an occurrence can lead to pain resulting in increased spasticity from irritability as early as the teenage years. Problems inherent with dislocation are better prevented than they are treated after they occur. Typically, soft tissue releases are indicated for the 0-3 year age range. At the first signs of progressive hip subluxation or when there is greater than fifty percent lateral migration of the femoral head, surgery is indicated. X-rays need to be taken every one to two years and the migration percentage calculated. Cooke (1989) prefers to use the acetabular index and feels it is the best prognostic indicator. Elective adductor tentotomies and, depending on the degree of spasticity, obturator neurectomies are indicated in this group of patients.

At age three to six, soft tissue release and femoral osteotomies may be necessary. Beyond age six and up until age twelve, a Pemberton of Salter innominant osteotomy may be necessary. Staheli (1991) has had good experience with his acetabuloplasty. Over age twelve, a Chiari osteotomy is necessary.

Attention needs to be paid to the hamstring muscles in order to avoid sacral sitting. Generally, a popliteal angle of greater that 45° begins to give evidence of problems. The hamstrings in the total body involved child may be released distally or proximally. Problems with over lengthening in this patient group have not been encountered by the author.

The position of the pelvis deserves careful attention when evaluating the sitting posture of a totally involved child. It may either be posteriorly tilted due to tight hamstrings resulting in sacral sitting or there may be obliquity. The obliquity is unrelated to the prognosis for hip subluxation but is significant in skin tolerance and sitting balance.

Scoliosis occurs in greater than 25% of immature patients with spastic quadriplegia. It can be relentlessly progressive and rarely responds to bracing. It may lead to the need for adaptive seating, may facilitate hip subluxation due to poor acetabular coverage and frequently requires surgery. Indications for surgery are to maintain sitting balance and avoid cardiopulmonary compromise due to severity of deformity.

The feet also deserve concern in this group. Although they rarely become functionally ambulatory, it is necessary for shoe wear and in assisting with transfers to have a plantigrade foot. Proper tendon surgery will facilitate this. The results of the use of

orthoses in preventing deformities in the total body involved patient group have been discouraging. Surgery is generally indicated when deformities become progressive but orthoses may help to prevent recurrence of deformities after surgery.

For patients having spastic diplegia and spastic hemiplegia, the orthopaedist's role, where possible, is to improve function. Before undertaking this it is essential to agree on how to measure function. Work in the Motion Analysis Laboratory has aided greatly in this determination. The parameters of gait, ie velocity, stride length and cadence may be considered, but the true measurement of improved function is whether it is easier to walk. Energy consumption appears to be the best criteria for demonstrating this. Heart rate has been one of the criteria for this but recent work by Gage and Koop at Gillette Children's Hospital suggests improved oxygen consumption.

Principles of gait were initially suggested by Jacqueline Perry (1985). These have been modified by Gage (1991) and are, (1) stability in stance, (2) clearance in swing, (3) pre-position of the foot in swing, (4) adequate step length and (5|) energy consumption. Each ambulatory child is looked at with these five principles in mind asking the question, "can we make an improvement?".

STABILITY IN STANCE: largely deals with the subtalar joint. A planovalgus foot frequently occurs in the diplegic child, whether this is extrinsic imbalance or secondary to an equinus deformity is yet to be determined. By restoring proper alignment and stability here, the individual has a much more stable platform on which to bear his/her weight. The author's centre has a large experience with subtalar arthrodesis. More recently the Evans calcaneal osteotomy has been used to lengthen the lateral border of the foot. This lateral procedure appears to do a better job of correcting forefoot valgus while providing for improvement in hind foot position. Surgery generally is not indicated until age four to seven. Prior to that time a high profile UCB or appropriately designed AFO can usually control the joint. As the child grows larger, both the body mass and increased strength overcome these devices making surgery necessary. The subtalar arthrodesis uses the technique of Dennyson and Fulford (1976). The calcaneal osteotomy was recently re-introduced by Moscowitz. In an adult a triple arthrodesis may be helpful.

Appropriate tendon transfers and lengthenings are necessary to better balance the foot and ankle before undertaking these bony procedures. Recent evidence by Etnyre et al (1993) indicate that tendon lengthenings provide for a better functional excursion but do not weaken the muscles.

CLEARANCE IN SWING: Lower extremity deformities such as equinus posturing of the feet, inadequate knee flexion or weaknesses of dorsiflexion at the ankle and/or hip flexors can be factors of concern here. Equinus deformity is aided by appropriate tendon lengthenings and use of ankle foot orthoses. Adequate knee flexion can be greatly aided by evaluating the function of the rectus femoris muscle. If EMG testing indicates poor timing and if the Duncan-Ely test is positive, rectus femoris transfer to the Sartorius may aid in greater knee flexion during swing. Hip flexor weakness must be avoided. Injudicious iliopsoas lengthenings may lead to this. Intermuscular psoas lengthenings over the brim of the pelvis is advocated when indicated. **PRE-POSITION OF THE FOOT IN TERMINAL SWING**: Adequate dynamic positioning of the foot during swing phase is necessary for pre-positioning in terminal swing before heel contact. Adequate dorsiflexion can be provided in the young with AFO's but must be addressed surgically when power overcomes external support. Inversion may be due either to an overactive tibialis posterior muscle or imbalance in the anterior tibial muscle. A split anterior tibial tendon transfer and/or intramuscular posterior tibial tendon lengthening may be necessary. An intramuscular paraneal tendon lengthening may help overactive eversion.

A mild dorsiflexion assist AFO will help with foot clearance in swing, however, too active a dorsiflexion assist may cause reflex spasticity resulting in increased plantar flexion.

ADEQUATE STEP LENGTH: The most common deterrent for adequate step length is overactive hamstring activity. This prevents the leg from swinging out in front of the trunk. It is felt that hamstring lengthenings may improve this, however Thometz et al (1989) demonstrated no changes in gait parameters.

The plantar flexion-knee extension couple needs to be considered as well. An overactive gastrocnemnius-soleus group will frustrate the second rocker of mid-stance causing knee hyperextension and shortening the step length. It is necessary to obtain a full 10° of dorsiflexion of the ankle for proper rollover.

ENERGY CONSERVATION: Mosberg (43) has demonstrated improved heart rates using AFOs in spastic diplegics. Studies yet unpublished indicate improved oxygen consumption with appropriate lower extremity surgical intervention. This further data is awaited with interest.

"How long should one use an AFO?" is frequently asked by parents. A critical answer might be as long as they fulfil the purpose for which they were prescribed. This begs the question, of course, "for what purpose were they prescribed?". Corrective surgery may replace their purpose. As with an earlier Polio experience, if therapy is planned to make a child orthosis free at the end of growth, using them is better accepted during growth. Careful assessment pre and post prescription must be made with all of the patients to ensure that therapeutic goals are met.

By way of conclusion it is necessary to address the question of when to operate. Historically, one procedure was done and an observation made and then another procedure etc until a child was spending each and every birthday in hospital for an additional orthopaedic event. With proper evaluation and diagnosis using the Motion Analysis Laboratory, multiple procedures can be done at one sitting, minimizing hospitaliztion and maximizing return to function. What age, however, is best? The author's personal view is that the best answer to this question is when all other approaches fail. When progress ceases and it can be improved with surgical intervention, then surgery should be undertaken. In some cases where hip stability is of concern, this may be in early infancy. In other areas this may not be until closer to the adolescent growth spurt. Remember that surgical intervention is meant to prevent deformities and when they cannot be otherwise prevented it is indicated. It may also improve function and when it cannot be otherwise improved, surgery is indicated. Some centers feel that any and all anticipated surgeries should be done early to facilitate a more normal motor development. This is an ideal consideration but data is lacking to support this contention and unfortunately, children are not always seen early enough. All decisions for therapy must be made by the team. It has been frequently noted that this involves in addition to the various groups of therapists, orthotists and physicians, the patient themselves and the patient's family. Their goals are the most important. All must participate in decisions for therapy.

REFERENCES

Bax M. Terminology and Classification of Cerebral Palsy. Dev Med Child Neurology, 1964 <u>6</u>, 295-297.

Cooke P H, Cole W G, Curey R D. Dislocation of the hips in cerebral palsy. Natural history and predictability. J Bone and Joint Surgery, 1989 <u>71B</u> (3), 441-6.

Dennyson W G and Fulford G E. J Bone and Joint Surgery, 1976 58B; 507-

Etnyre B et al. Preoperative and postoperative assessment of surgical intervention for equinus gait in children with cerebral palsy. J Pediatric Orthopaedics, 1993 <u>13</u>, 24-31.

Freud S. Infantile Cerebral Paralysis, (Trans. Russin, L A), Miami. University of Miami Press. 1968.

Gage J R. Gait analysis in cerebral palsy. New York, MacKeith Press, 1991, 118-

Osler W. The cerebral palsies of children: classics in developmental medicine. Philadelphia, J B Lippincott, 1987, 36-43.

Perry J. Normal and pathologic gait, In Bunch W H (Ed), Atlas of Orthotics, 2nd Edition, St Louis, C V Mosby, 1985, 76-111.

Rang, M. Cerebral palsy: William John Little, 1810-1894, In Rang H. Anthology of Orthopaedics, Edinburgh and London, E and S Livingstone, 1966, 48-52.

Staheli L T. Surgical management of acetabular dysplasia. Clin Orthop, 1991 264, 111-21.

Thometz J, Simons S, Rosenthal R. The effect on gait of lengthening of the medial hamstrings in cerebral palsy. J Bone and Joint Surgery, 1989, 71(3), 345-53.

HOW DO SURGEONS ACHIEVE THESE AIMS? II

George M Carter MB BS FRACS FA(ORTH)A

INTRODUCTION

In order to discuss the place of surgery in cerebral palsy and its relation to other modalities in treatment such as physical therapy and orthoses it is necessary to understand the genesis of the malfunction.

One must understand the neurological lesion and its musculoskeletal effects as well as the pathogenesis of the function and the eventual deformities which result from lack of treatment.

The aims of treatment will be discussed under the four main topics suggested as a framework for this discussion.

- to prevent deformity (or correct or minimise its effects)
- to achieve a stable base
- to aid achievement of motor skills
- to improve the dynamic efficiency of gait.

TO PREVENT DEFORMITY

Types of Deformity:

- Dynamic

No deformity at rest or asleep Deformity occurs on initiating or sustaining activity.

- Passive

Deformity occurs at rest but can be corrected by manual pressure with patient relaxed or asleep.

- Fixed Deformity

Cannot be corrected by manual pressure even asleep or anaesthetised.

Pathogenesis of Deformity

Usually goes through the above stages progressively. It may go through the following stages:

- (a) muscle spasm
- (b) reduced excursion
- (c) reduced growth
- (d) contracture
- (e) torsion
- (f) angulation
- (g) instability

- (h) dislocation
- (i) arthritis

The speed of moving from stage to stage varies greatly and must be carefully monitored and recorded by <u>all</u> clinicians looking after these people.

Spasm mediated by

overaction of muscle spindle lack of supraspinal inhibition cospasticity and reduced discrimination.

Reduced range of contraction

A muscle belly can only contract approximately to 60% of its resting length due to damage of the actin and myosin rods (Haines, 1932)

Reduced power of contraction

maximum power developed when contraction begins with muscle at just over its resting length (Reimers, 1990, Fulford, 1990) the antagonists to the spastic muscles are often overstretched and contract only weakly (until the spastic muscle is released or stretched) (Booth, 1977, 1979, 1982) muscle shorter than its resting length (in spasm) is also weak.

Reduced growth in muscle

contracted muscle grows more slowly (may be at half the normal rate) (Rowe and Goldspink, 1969, Williams and Goldspink, 1971, Tardieu, 1972, Tardieu and Tardieu 1987) normally muscle length is half of adult at age 4 years stimulus to growth is stretch beyond resting length (Tardieu and Tardieu, 1987) "growth plate" is the musculotendinous junction (Rowe and Goldspink, 1969, Williams and Goldspink, 1971, Tardieu, 1972, Tardieu and Tardieu, 1987)

Growth in tendon

stimulus is tendon stretch + tension Therefore spasm causes overgrowth EG patellar tendon "growth plate" is the bone-tendon junction (Tardieu and Tardieu, 1987)

Growth in bone

genetic, endocrine, biochemical factors cyclical loading is the main mechanical stimulus Woolf's Law and hypertropy eg short or long lateral side of foot torsion of femur/tibia

Anatomical site of deformity

- muscle/tendon complex remains the major site until very late
- capsule and ligaments
 - (a) rarely under 9 years
 - (b) even then, only in permanent sitters or subluxed/dislocated joints
 - (c) prevented by B D ranging
- All sites including bone and joints rapidly progress if dislocation is present.

Treatment of Deformity

Dynamic Deformity

Orthotic Treatment

- Tone reduction
- Achieve joint locking (knee straight at stance)
- Increase base of support
- Guide movements

Physical Therapy

- Prestretching
- Posturing

Surgical Treatment

- Muscle lengthening decreases deforming force
- Transfer of tendons to correct imbalance ie SPLATT in inverted foot of hemiplegic
- Rhizotomy superselective to reduce deforming forces in muscle groups

Passive Deformity

Orthoses

- guiding orthoses ie articulated AFO or floor reaction
- lock joints
- tone reduction

Physical Therapy

- Aim to increase motor power

Surgical

- Motorise joints ie peroneal transfer
- Transfer
- Fusion of joints ie subtalar fusion
- Tenodesis ie toe clawing

Fixed Deformity

- Physical therapy has now become limited
- Orthoses are limited in effectiveness
- Serial casting
- Resting orthoses to limit progression

Surgical Treatment

- More urgent and effective
- Open reduction of dislocation
- Arthroplasty
- Osteootomy
- Fusion

ACHIEVEMENT OF A STABLE BASE

A stable base requires:

A balance of subtalar, ankle, knee or hip in the ambulant patient or an interaction of the seat, pelvic stabilisers and spine and hip abductors.

Footwear

- broad heels
- floats
- bowling soles
- ankle hightops

Orthoses

- subtalar control
- "posting"
- in shoe fit
- AFOs

Surgery

- Fusion subtalar
 - triple
- Rebalance stance phase tibialis posterior transfer peroneus brevis transfer

91

tibialis anterior transfer lengthenings, ETA, hamstrings adductors

ACHIEVEMENT OF MOTOR SKILLS

Depends on desired skill requiring aid.

Sitting - may be required for computer operation or for driving a crane Hip and spinal stability may be the priority

Transferring from chair to car May require hamstring or extensor surgery

Walking Skills

May require surgery for:

- scissoring
- crouch gait
- to get a plantar grade foot
- to get swing phase knee flexion

IMPROVEMENT IN DYNAMIC EFFICIENCY OF GAIT

Work against gravity is the major energy wasting activity in cerebral palsy patients. Aim is to limit the rise of the centre of gravity.

- promote knee flexion in swing
- promote ankle dorsiflexion in swing
 - This allows the pelvis to dip, avoiding a rise on the swinging side.
- allow some knee flexion in midstance, ie rectus femoris transfer to hamstrings (Reimers, 1990)
- avoidance of crouched gait
- avoidance of internal rotation

Energy cost of walking and energy consumption in diplegia may be two to four times normal. This has been shown to be reduced in the diplegic by surgery (Gage, 1991).

REFERENCES

Bleck E E. Editorial comment. Clin Orth 1990 253, 2-3.

Blundell Jones G. Paralytic Dislocation of the Hip. J Bone Joint Surgery 1962 <u>44B</u>, 573-587.

Booth F W. Time course of muscular atrophy during immobilization of hind-limbs in rats. J Appl Physiol 1977 <u>43</u>, 656-661.

Booth F W. Effect of limb immobilization on skeletal muscle. J Appl Physiol 1982 <u>52</u>, 1113-1118.

Booth F W and Seider M J. Recovery of skeletal muscle after 3 months of hind-limb immobilization in rats. J Appl Physiol 1979 <u>47</u>, 435-439.

Gage J R. Gait analysis in cerebral palsy. London McKeith Press 1991.

Fulford G E. Surgical management of ankle and foot deformities in cerebral palsy. Clin Orth 1990 253, 55-61.

Haines R.W. The laws of muscle and tendon growth. J Anat 1932 66, 578-585.

Hoffer M M, Stein G A, Koffman M and Prieto M. Femoral varus de-rotation osteotomy in spastic cerebral palsy. J Bone Joint Surg 1985 <u>67-A</u>, 1229-1235.

Reimers J. Functional changes in the antagonists after lengthening the agonists in cerebral palsy. Clin Orth 1990, 253, 30-37.

Rowe R W D and Goldspink G. Muscle fibre growth in five different muscles in both sexes of mice. J Anat 1969 <u>104</u>, 519-538.

Tardieu G and Tardieu C. Cerebral palsy: mechanical evaluation and conservative correction of limb joint contractures. Clin Orth 1987 <u>219</u>, 63-69.

Williams P E and Goldspink G. Longitudinal growth of striated muscle fibres. J Cell Science 1971 <u>9</u>, 751-767.

Young R R and Wiegner A W. Spasticity. Clin Orth 1987 219, 50-62.

AN OVERVIEW OF COMPONENTS AND CONCEPTS INVOLVED IN ORTHOTIC PRESCRIPTION FOR CHILDREN WITH CEREBRAL PALSY

Beverly Cusick MS PT

THE CHALLENGE

Orthoses of specific configuration are commonly prescribed for diagnoses such as hemiplegia, diplegia, or quadriplegia, or for isolated deformities such as equinus. Equinus deformity - evidence of inadequate ankle dorsiflexion (DF) range of motion (ROM) - rarely occurs as an isolated problem in children with CP. The diversity of accompanying musculoskeletal components can escape consideration in this diagnosisbased approach to orthotic prescription. The associated factors include bone length and configuration, joint alignment and mobility, soft tissue extensibility, muscle strength and composition, and postural alignment - all of which combine to contribute to each child's problems of deformity and function.

Researchers who attempt to lump these musculoskeoletal variations under common diagnostic groups, and then to compare effects of two orthotic designs of one design and no intervention, cannot help us to learn about orthotic efficacy in the context of specific compositions of coexisting variables. Similarly, researchers who offer singlesubject designs and case studies without detailing the child's musculoskeletal composition cannot expect that their results will be replicated in cases with common diagnoses and different clusters of musculoskeletal components (Carlson et al, 1995).

In most medical facilities, one clinician on each management team either elects to or is expected to exercise responsibility for orthotic prescription. My experience suggests that training programs in the fields of physical medicine, orthopaedics and physical therapy feature brief, introductory-level lectures on orthoses, identifying the most common designs and naming a few indications for use, as if selecting one that will fulfil the desired functional goals is simple. Orthoses are expensive, and double so if ineffective or painful. It is unusual that any individual on the team, including the orthotist, receives specialized training to gain clinical assessment and problem-solving skills needed to prescribe optimally-effective orthoses.

Generating a Comprehensive, Context-Based Orthotic Prescription

Standardized training and licensure in orthotic prescription will be of little practical value until researchers establish orthotic efficacy in a patient-specific context of existing mechanical, strength and functional variables. Because the factors that could influence orthotic outcome are so diverse, an educated and appropriate prescription is best derived by employing the collaborative scope of knowledge and resources of all involved team members, including the child and caretakers.

To make clinical judgements as effectively as possible, I encourage team members to gather objective findings regarding musculoskeletal status and functional abilities, and to apply known principles of physics, kinesiology, kinetics and the closed kinetic chain to their review of the clinical findings. The problem-solving process that leads to orthotic prescription should then address a full array of available design options and their recommended criteria for use. The following discussion addresses various aspects of clinical assessment and application of scientific principles to orthotic design selection, and incorporates several orthotic design ideas along the way.

OBJECTIVE CLINICAL OBSERVATIONS

Various team members should be trained to skilfully contribute to the knowledge base leading to a program of therapeutic management and orthotic prescription, establishing reliable evidence of pre-intervention status against which to compare outcome, and providing the following data for consideration relevant to orthotic design selection:

- Musculoskeletal and anthropometric findings viewed as mechanical considerations, including body weight relative to height, bone structure, pelvic and lower extremity joint alignment and mobility in all three planes, and passive soft tissue extensibility which acknowledges "first catch" end ranges. These findings should be reviewed in the context of known age-related norms.
- Standardized functional assessment. The Gross Motor Function Measure has achieved acceptable reliability and validity as a functional assessment tool for children with CP (Young and Wright, 1995).
- Cardiovascular status and an account of typical daily demands on endurance (Rose et al, 1989, Jones and McLaughlin, 1993).
- Muscle strength at appropriate functional muscle lengths and in functionally applicable context. Consider potential influences on posture and movement control of inadequate central nervous system (CNS) maturation, true muscle weakness and physiologic muscle adaptation and transformation.
- Weight distribution on the foot in stance and gait.
- Proximity of body segments and load-bearing joints to the normal ground reaction force (GRF) vector in both sagittal and frontal planes.
- Characteristics of gait, obtaining objective measures for future comparison.
- Assessment of balance evidence of ataxia.
- An appraisal of primary (originating) and secondary (compensatory) problems that influence function.
- Context for orthotic use: therapeutic environment with frequent sessions of supervised training by caretakers or therapist vs. periodic consults and no ongoing intervention.
- Consideration of apparent potential to alter the loading forces on the foot or to significantly change a pathologic gait pattern through training, as distinguished from the goal of using mechanical adjustments to protect the loaded foot from enduring the full brunt of deforming forces over many years.

THE SCIENCES: PHYSICS, KINETICS, BIOMECHANICS OF THE CLOSED KINETIC CHAIN

Computerized gait analysis using force plate data to calculate load distribution and displacement, ground reaction forces, kinematics, resultant forces and moments acting at the joints, and the exchange between potential and kinetic energy during the gait cycle provides the rehabilitation team with scientific concepts and principles which are directly applicable to orthopedic, therapeutic and orthotic interventions, including the following (Olney, 1989, Vaughan et al, 1992, Gage 1994).

- The influence of lever arm length on muscle function around joint axes in all segments of the lower extremity.
- The interrelationship of mechanical and alignment features via the closed kinetic chain.
- Principles of kinetics energy storage and release as they apply to mechanical alignment and efficiency of muscle activity in normal and pathologic gait.
- Results of studies on energy efficiency and oxygen consumption during normal and pathologic gait.

ORTHOTIC DESIGN CONSIDERATIONS

The field of orthotics has experienced a blossoming of ingenuity and adaptation of old designs to incorporate new management concepts and technological advances. Their repertoire of options in orthotic design and materials has undergone a dramatic expansion, currently providing a variety of trim lines: ankle-height, short-shaft, full-foot-length, partial-foot-length, submalleolar and articulating orthoses; various joints and motion-assist options; and plastic compounds that offer different callibers of flexibility. These design options are ideally considered by the management team in the context of clinical findings and goals, and embrace the following elements:

- Evidence of the magnitude of resistance to manual deformity correction, to gain insight about:
 - The feasibility of orthoses for correction versus accommodation
 - The need for soft tissue preparation prior to introducing orthoses
 - The magnitude of corrective forces required and the associated necessity to disseminate the forces over a large surface area.
- Segments to be included in the orthosis.
- Properties of selected materials that will influence comfort, effective stabilization and durability.
- The weight of selected materials relative to available muscle strength.

- Desired joint motion: solid versus articulating; variable motion stops; the magnitude of excursion allowed by the stop; motion assist options; adjustability.
- Posting considerations in sagittal and frontal planes.
- Different orthotic devices provided to achieve different clinical management and functional goals.
- Low-temperature plastic, low-cost prototypes to supplement the problemsolving, prescription process by providing clinical trials of questionable designs (Sussman and Smith, 1987, Cusick 1988, 1990).
- Low-temperature plastic splints as temporary and readily changeable orthotic interventions to escort an infant or child through a recovery period following brain injury, nerve root or soft tissue surgery, flexible clubfoot deformity reduction, or to prevent contracture formation during prolonged skeletal traction procedures.

I have selected a few items on this list of considerations to discuss in more detail in this paper.

CLINICAL OBSERVATIONS

Ideally, the orthopedist, physiatrist and physical therapist can provide the orthotist with a review of any child's passive mechanical properties of lower extremity bone structure, joint mobility and soft tissue extensibility, as these observations are essential to designing management programs beyond the scope of orthotic intervention. The professional orthotist should be able to comprehend the significance of these clinical observations as they pertain to potential orthotic efficacy. This ideal accumulation and transfer of information often fails to occur. While we all work to acquire standardized and reliable skills in musculoskeletal assessment, the orthotist who receives a vague orthotic prescription and no pertinent clinical findings should be able either to request the needed information from other team members, or to quickly execute a variety of tests of musculoskeletal status, muscle strength, loading patterns and gait function to gain insights needed to fill the prescription knowledgeably.

Musculoskeletal Status

Barber (1993) suggests that clinical orthotists gain clinical assessment skills to use regularly in their professional practice, to accurately ascertain joint mobility and muscle strength status and tendency to fatigue, I would add that the orthotist should be prepared to knowledgeably anticipate or promptly correct problems with functional outcome that pertain to musculoskeletal status including bone structure and soft tissue extensibility.

Example (1): structural alignment influence - f emoral torsion deformity

Increased medial femoral torsion (MFT) is a common problem of structural immaturity in children with CP (Tachdjian, 1990, Laplaza et al, 1993). Torsional deformity in the femur typically requires orthopedic reduction via osteotomy rather than orthotic correction, and can impose embarrassing consequences on orthotic outcome if not identified prior to intervening. MFT is usually expressed in gait as excessive medial rotation of the transcondylar (ie knee joint) axis during swing and stance phases when the pelvis is aligned in the transverse plane. If the tibia is normally aligned with the femur, an intoe gait pattern (medial foot progression angle (FPA) occurs (Svenningsen et al, 1989). Foot pronation commonly accompanies increased MFT (Powell, 1983)

Orthotic Implication - increased MFT

If forefoot abduction accompanies foot pronation sufficiently to reduce the appearance of femoral torsion-related into gait, orthotic correction of foot pronation results in markedly increased into eing. If the tibia and fibula have modelled in increased lateral torsion to accommodate prolonged excessive lateral rotation forces, then orthotic intervention would probably have little effect on foot progression angle (FPA). A musculoskeletal assessment prior to intervening orthotically will alert the team to such potential orthotic effects, and they can warn caretakers in advance of the potential for a dramatic change in FPA on applying orthoses that diminish the deformity-masking compensation.

Example (2): structural imbalance in the foot-forefoot varus deviation

Children with a history of weightbearing on a pronated foot typically reveal significant forefoot varus deviation - a structural imbalance within the tarsus which limits forefoot eversion ROM when the subtalar joint (STJ) is aligned in full congruity (Grumbine, 1987, McPoil, 1987, Tiberio, 1988, Michaud, 1993).

Orthotic implication - forefoot varus

Having orthotically reduced excessive calcaneal eversion associated with weightbearing pronation, a forefoot varus deviation leaves the previously-loaded medial forefoot suspended in a position of inversion inside the orthosis. Without adequate forefoot eversion ROM the necessity to distribute loading forces over the greatest plantar surface area drives the entire foot to pronate inside the orthosis, usually be abducting the forefoot and adducting the talus and leg. Selecting a more flexible plastic orthotic shell without varus posting as needed might meet comfort requirements, but deprives the foot of stable alignment at propulsion and promotes persistent medial rotation of the leg throughout second rocker via the closed kinetic chain (Michaud, 1993). Early degenerative knee joint changes and knee pain in adulthood can be expected if walking in significant pronation is the child's primary means of locomotion (Clancey, 1983, Duckworth, 1983, Coplan, 1989, Michaud, 1993).

The only reason to modify a positive cast to "correct" the residual forefoot varus remaining after gentle correction was applied at molding should be to provide a plantigrade floor on which to provide an interior, modifiable forefoot varus post. Acknowledgeable first ray PF as a component of foot stability in normal stance and propulsion and design any forefoot varus post to permit the first ray to plantarflex a few degrees, by crushing a relatively compressible foam, for example, while more firmly supporting the remaining metatarsal heads in varus deviation gradually resolves,

reduce the magnitude of the post by grinding under the first ray, or remove the post as your assessment findings suggest.

Example (3): joint mobility problem

An axial tibiofibular (leg) rotation bias with knee flexed (and often with it extended) is evident when passive transverse-plane rotation of the leg unit (excluding the foot) into either medial or lateral rotation is significantly limited (McCrea, 1985). This rotary bias reveals either developmental immaturity, popliteus muscle contracture or overlengthening, or abnormal modelling effect on the collateral ligaments at the knee joint. Gage (1991) states that the knee (ie leg under the femur) rotates in the transverse plane 10°-15°. However, that magnitude of motion occurs with the knee joint extended and the achievement of ligamentous integrity. With increasing knee flexion, the let rotation range increases, totalling up to 60° in non-disabled children ages 2-10 years, and approximating 40° in adult women (Lehmkuhl and Smith, 1983, Cusick, 1994). Total rotation ROM often exceeds 60° and rotary biases occur in children who routinely Wsit with feet turned medially or laterally, or in those who show ligament laxity.

Given that the femur shows normal medial torsion, a child with a significant medial axial leg rotation bias typically shows a medial foot progression angle (FPA) in gait. This problem is frequently (mis)diagnosed as "internal tibial torsion" in young children who are assessed in sitting position with the knee flexed 90° (Rosen and Sandwick, 1955, Weseley et al, 1981). If the child is very young - 2 years or younger - the knee ligaments typically reveal a normal laxity that permits demonstration of a medial rotary bias with knee extended as well as flexed. If a child with a medial rotary bias walks with flexed knees, as do many children with diplegia, the magnitude of intoeing in gait increases due to the increased range of transverse-plane mobility evident with knee flexion. Persistence of this rotary bias can be expected to lead to bony trans-malleolar axis and the frontal plane with the knee axis aligned on the frontal plane and the knee joint extended. Several studies indicate that in adulthood, lack of lateral tibiofibular torsion is associated with medial knee joint arthritis (Turner and Smillie, 1981, Hagi and Suzaki, 1986, Yagi, 1994, Turner, 1994).

Orthotic implication - medial axial tibiofibular rotation bias

If the child is less that 5 years of age, consider instituting gentle night splinting - using for example a customized adaptation of the Wheaton telescoping brace^a - and a daytime intervention using rigid taping techniques or elastic rotation-assist strapping to reduce the FPA in functional context (Kendall et al, 1993, Sarver, 1994). If these interventions are introduced, establish immediate and long-term efficacy with footprint studies, and note any changes in axial lateral leg rotation at the flexed knee joint through periodic and precise ROM assessment. My management goal for limited lateral rotation is 30° of lateral tibiofibular rotation with knee flexed 90°. This measurement is obtained with the child positioned in prone, using the longitudinal axis of the fully congruent foot as a reference axis off the longitudinal axis of the posterior thigh (Cusick and Stuberg, 1992).

^a Wheaton Telescoping Brace - AliMed Inc 297 High Street, Dedham, MA 02026

Example (4): soft tissue extensibility problem

Functional ROM for muscle and related tissues occurs at "first catch" or the initial end range of a quickly, easily and passively-imposed excursion (Reimers, 1974, 1992, Sapega et al, 1981, Sapega, 1988, Tardieu and Tardieu, 1987). Maitland refers to the same phenomenon as Resistance-1 (R₁) (Maitland, 1977). This initial end range can also be interpreted as the resting muscle length (L1) - the point on the stress-strain curve at which tension is noted in response to passive stretch (Herbert, 1988). Muscles can exert maximum force at the length at which the contractile filaments overlap optimally, which is also their resting length. If length varies by more that 50% above or below the muscle's resting length, active contraction drops to zero. Within that excursion from resting muscle length, the tension generated by a muscle reduces. The sum of these active and passive tensile components is the overall length-tension relationship (Rab, 1994).

When assessing passive ankle DF ROM, extend the knee and maintain the foot joints in full congruity and prohibited from permitting forefoot DF that occurs with pronation. Use the fibula and the plantar plane of the foot as reference axes (Siebel, 1988, Bohannon et al, 1989, McPoil and Brocato, 1990, Michaud, 1993). In children, first catch (if present) should normally occur at or near 0° of DF (Tardieu and Tardieu, 1987). My observations of non-disabled children suggest that on measuring passive ankle DF ROM in the manner described the younger the child, the more difficult it is to discern first catch end range, and that when evident, it commonly occurs at 5° to 10° of DF. Maximum passive end range easily achieves 15° to 30° of DF in children age 1-5 years (Tardieu and Tardieu, 1987, Sutherland et al, 1988, Cusick, 1995).

My clinical and teaching experiences involving musculoskeletal assessments of hundreds of children with CP, undertaken by hundreds of clinicians with my supervision, suggest to me that children with chronically overactive triceps surae muscles reveal first catch (R_1) passive ankle DF ROM with the knee extended to be an obvious and highly reliable clinical finding (informally but consistently observed to fall within 4° of variance between testers). Recalling that R_1 end range is also the functional end range and occurs at the muscle's resting length, I suggest that this finding is more clinically significant than the less reliable maximum end range, the latter of which generally never appears in spontaneous ambulatory function. Tardieu and Tardieu (1987) and Sapega (1988) suggest this distinct R_1 end range shows evidence of proliferation and increased longitudinal orientation of intracellular connective tissues in the context of hypertonic muscle use. The excursion between R_1 and maximum end range might be limited to less than 10° or resisted throughout the range in the presence of muscle tissue transformation (Williams and Goldspink, 1984, Handelsman and Glasser, 1986, Lieber, 1986, Tardieu and Tardieu, 1987, Rab, 1992).

Orthotic implication - shortened triceps surae muscles

The classic problem of tolerance for wearing ankle-foot orthoses (AFOs) in children with hypertonic CP can be assigned to two common factors: 1) difficulty "keeping the heel seated" in the AFO which sets the ankle at 0° PF, often requiring padded instep straps applied at maximum compressive tension; and 2) skin breakdown. In these cases, the soleus muscle typically shows adequate extensibility and the gastrocnemius

muscle, which spans three shortened gastrocnemius muscle on slack. Therefore, when the child stands and walks, the gastrocnemius fails to elongate and the heel rises. The pronated foot usually attempts to provide the expected sagittal plane mobility at the forefoot by pronating in the orthosis. If the foot supinates with equinus, when forced into the same AFO, it too is poorly controlled and endures excessive pressure at contact points with the device. In these instances, common orthotic outcomes include:

- The child displays "noncompliance" in effecting the proposed wear schedule
- The compliant child's skin breaks down or forms bursae or callouses
- Any existing midfoot pronation deformity worsens
- The family "loses" the appliance between clinic visits.

Prior to intervening with any orthosis, shortened soft tissues should be gently elongated for prolonged periods (minimum 20 minutes per stretch) to try to restore adequate soft tissue extensibility by effecting normal physiologic adaptation (Bohannon and Larkin, 1985, Kisner and Colby, 1990). Conservative measures include:

- Serial casting (Westin and Dye, 1983, Gritzka and Gerlach, 1986, Tardieu and Tardieu, 1987, Herbert 1988, Cusick, 1990a, Mazur et al, 1992, Berghof et al, 1995)
- Positioning to apply prolonged, loaded elongation to the ankle, with the foot supported to prevent pronatory deviation (Bohannon and Larkin, 1985)
- Night splinting (Baumann and Zumstein, 1985, Handelsman and Glasser, 1986)

In the absence of facilities to provide soft tissue preparation, the orthosis should accommodate for the lack of adequate DF with a plantarflexed ankle position and heel wedge posing, to relieve the foot of pronatory of supinatory stress and to provide maximum possible vertical loading on the heel pad (Russell, 1995).

Orthotic implication - shortened hamstrings and hip flexor muscles

If an extending influence on the knee joint is expected in the context of crouch posture, wait to issue DF-resist AFOs until significant shortening of hamstrings, hip flexors and any existing knee flexion contractures are reduced (26, Cusick, 1988, Cusick, 1990) Otherwise, the orthoses might relieve the hamstrings of a measure of oversuse, but the desired mechanical effect on knee angle will be compromised. Floor reaction orthoses used to reduce knee flexion in gait are contraindicated in the presence of triceps surae contracture.

Weight Distribution on the Foot in Stance and Gait

Aharonson et al (1980) and Cavanagh et al (1987) used computerized photosensors on a force plate to calibrate load distribution on the foot in static stance in non-disabled children, age 4 years, and in non-disabled asymptomatic adults. Both researchers obtained essentially the same averaged findings. Weight was distributed as follows: 60% on the heel pad, 35% on the metatarsal heads and toes, and 5% on the midfoot area, indicating a weightbearing ratio of heel to metatarsal heads of close to 2:1. The more pronated feet showed increased loading on the midfoot and anterior medial forefoot.

Orthotic implication: abnormal load distribution on the foot

The restoration of appropriate distribution of body weight on the segments foot is a fundamental therapeutic and orthotic goal. Consider the relationship between weight distribution and the proximity of the center of mass to the heel pad and weight bearing of the lower extremities (Kendall et al, 1993).

Aharonson et al (1992) used medial heelwedges (varus posts) to reduce clacaneal eversion associated with pronation to 0° in children ages 4-6 years. The researchers observed significant normalization of the load distribution area on the feet as a result. Aharonson's subjects did not exhibit CP, but many children with CP exhibit pronation and anteriomedial load distribution problems. Rose et al (1992) found similar effects of more exaggerated medial and lateral heel wedging which resulted in redistribution of loading forces to the side of the foot that was lowermost on the wedge.

Proximity of Body Segments and Load-bearing Joints to the Vertical Ground Reaction Force (GRF) Line

Close proximity between the load-bearing and the GRF vector in sagittal and frontal planes indicates optimum mechanical circumstances for energy efficient use of the musculature (Stallard, 1987, Weber, 1990, Gage, 1991, Rab, 1994). Several researchers have demonstrated this principle by evaluating the energy demands of flexed-knee gait on non-disabled adults and found that the imposed knee flexion angle increases, energy cost measured as oxygen uptake increases along with EMG activity in vastus lateralis, gluteus maximus and soleus muscles (Winter, 1983, Cerny et al, 1994, Cuddleford et al, 1995). Cuddleford et al (1995) detected that energy cost increased significantly at 40° of knee flexion in 7 non-disabled adults, and suggested that this finding cannot adequately represent the energy cost incurred in spastic diplegia due to the potential influences of various other postural, musculoskeletal and mechanical problems.

Orthotic implication - GRF/joint proximity

Use orthoses and joint-stabilizing splints to achieve optimum sagittal-plane knee alignment in stance and gait, and to train the child to use muscle groups in appropriate biomechanical context (Butler and Nene, 1991, Butler and Major, 1992). This concept will be addressed later in this paper in more detail in a discussion regarding manipulating degrees of freedom and targeting joint to simplify motor training.

Primary (Originating) and Secondary (Coping or Compensatory) Problems that Influence Function

Thomas et al (1992) studied the effect on muscle activation, represented by EMG output, of mechanically altering lower-extremity joint alignment in 6 non-disabled adults to mimic three alignment patterns seen commonly in children with diplegia. The

authors suggest that gait analysis results be correlated with clinical (ie musculoskeletal) examination. These findings raise the question of the origin of altered muscle function - and particularly of hypertonic muscle function - seen in children with diplegia. Which comes first?

- Weakness leading to alignment problems that result in predictable compensatory muscle firing patterns?
- Joint melalignment due to ligament laxity and structural immaturity?
- Unhibited spinal-level reflex muscle activity leading to abnormal postural alignment problems?

Management implications of primary and compensatory factors

Primary and secondary deviations together diminish energy efficiency. By addressing the primary problem, the coping deviations spontaneously disappear (Gage, 1991). When the primary problem is weakness in the extensor muscle groups at the hip, knee and ankle, with profound foot pronation, overuse of the quadriceps in lengthened position of the hamstrings and of the triceps surae are predictable compensations. Orthotically holding the ankle in DF addresses one compensation without addressing the primary problem of excessive anterior weight distribution. When weakness-related malalignment is the primary problem, conservatively reduce compensatory soft tissue contractiures to support the goals of orthotic intervention, and use orthotic, therapeutic and positioning interventions to restore more normal muscle function and alignment (Herbert, 1988, Farmer and Butler, 1994).

Orthotic implication - example: knee hyperextension

Quadriceps weakness in children with CP, leading to mild to moderate knee hyperextension during the stance phase of gait, can often be managed by applying a resilient knee splint that effectively prohibits knee extension for 3 to 8 hours per day (Cusick, 1988, Romero and Rice, 1992). The knee splint may be made either with a low-temperature plastic (Cusick, 1990) or a tubular, elasticized support adapted with posterior and velcro straps (LaRock, 1994). McConnell's biomechanical rigid skin taping techniques have also been applied effectively to this problem (McConnell, 1986, Hilyard, 1990, Sarver, 1994, Martin, 1995). By choosing among these interventions which are directed to the knee joint specifically, rather than applying a dorsoiflexed AFO, the clinician can avoid imposing prolonged orthotic lengthening and weakening of the triceps surae and the orthotic restriction of ankle plantarflexion, particularly during first and third rockers. These interventions mechanically limit terminal swing knee extension for the duration of the training period, but that feature of gait is typically undeveloped among children with CP (Hicks et al, 1988).

When knee hyperextension occurs in gait secondary to limited ankle DF ROM, try serial casting to improve DF range before addressing knee hyperextension directly (Baumann and Zumstein, 1985, Gritzka and Gerlach, 1986, Cusick, 1990a, Mazur et al, 1992, Berghof et al, 1995). In the absence of facilities to restore adequate ankle DF ROM, apply an articulating, semi-solid or solid AFO (as the child's functional skills indicate), having set the ankle in enough PF to protect the foot joints from secondary

deforming strain, and contouring and lining the interior plantar surface with a textured material to stabilize the foot against forward shear. Then provide a heel lift or wedge of adequate height to use the posterior shaft section to apply floor reaction forces that will oppose knee hyperextension (9).

I recommend engaging the ambulatory child in closed-chain strengthening of the ankle and foot musculature during the course of any ankle-foot orthotic intervention that features a stop, using a low-cut splint for training if needed to maintain the foot in desired weightbearing alignment. Continue to monitor the effects of and adjust either intervention, and, depending on the severity of the problem, within 6 to 12 months the knee hyperextension problem usually diminishes significantly or resolve and the intervention can be terminated (Butler and Major, 1992).

CNS Maturation, Muscle Strength and Muscle Transformation

EMG studies of non-disabled infants achieving independent walking reveal patterns of coactivation which actively reduce - though they do not eliminate - the degrees of freedom of lower extremity joint motion. The infant also gains control in upright postures in a proximal-to-distal sequence. Most of these patterns of prolonged muscle activity resolve to mature reciprocal patterns by the age of 4 years (Okamoto and Kumamoto, 1972, Berger et al, 1982, Sutherland et al, 1988, Sutherland and Valencia, 1992).

CNS Maturation: Infants and children (20 months through age 6 years) with diplegic and hemiplegic CO who achieve independent ambulation (on a treadmill) without assistive devices show persistence of immature muscle activation and coactivation patterns, and a failure of their gait patterns to mature (Leonard et al, 1991). Children with CP typically show less control over distal segments than proximal, overusing the hip musculature in stepping and walking (Olney, 1989, Gage, 1994). The ankle plantar flexors are the primary tibial stabilizers in gait, and weakness of the same muscle group is the primary cause of crouch posture in children with CO (Sutherland, 1978, Sutherland et al, 1980).

Muscle strength: Optimum muscle function demands the following components (Lieber, 1986a, Rang et al, 1986, Herbert, 1988, Tardieu et al, 1988, Gage, 1991).

- the proper number of sarcomeres in series for adequate velocity of contraction
- Ample sarcomeres in parallel to provide adequate tensile force
- Functionally-appropriate muscle fiber type composition: Type I (phasic, fast twitch) or Type II (tonic, slow twitch).
- Normal joint configuration and alignment to provide the optimum functional force couple and normal lever arm between the muscle force vector and the axis of rotation
- Normal precontractile muscle elongation.

A muscle which is composed mainly of slow fibers (Type II) will generate less force than a muscle of equal size but composed of fast fibers (Type I) (Lieber, 1986a)). Hypertonic muscle and adjoining connective tissues typically fail to respond to stretch imposed by bone growth by growing at a normal rate, and so generate fewer sarcomeres in series (Ziv et al, 1984, Rang et al, 1986, Gage, 1991). Muscle atrophy occurs in conjunction with loss of contractile force and is commonly observed in the distal lower extremities of children with DP (Handelman and Glasser, 1986). Shortened muscles do not achieve precontractile elongation in function.

As the non-disabled child grows, muscle mass increases at a greater rate than muscle strength. For children with CP who enter adolescence and experience its rapid increase in body size, this muscle mass/strength imbalance can be of a significant functional importance (Gage, 1991, Rab, 1994). Kramer and McPhail (1994) evaluated twelve adolescents with mild CO and determined that for eleven of them, measurements of increased isokinetic knee extensor strength, obtained with a dynamometer, modestly correlated with energy efficiency index scores during fast versus comfortable walking, and with gross motor ability (assessed using components of the Gross Motor Function Measure.

Citing Cavagna et al (1968) Gage states that if a normal muscle is stretched just prior to contraction, it will contact with greater power. During normal gait, most of the lower extremity major muscle groups are stretched by inertial and/or ground reaction forces just prior to the onset of their contraction. Examples: at the termination of second rocker, the gastrocnemius and soleus are elongated, ideally allowing 10° of tibial progression over the stable (congruent vs pronated) foot in preparation for the ensuing propulsive power burst, and the hip flexors are elongated (ideally 0°) at terminal stance just prior to third rocker and the onset of swing.^B

The common impression that triceps surae muscle strength in equinus deformity is excessive and overpowers the antagonists has been refuted by research results which suggest that the gait-related level of EMG activity in the lower extremities is generally *reduced* in children with CP, particularly in the triceps surae (Berger et al, 1982). Ankle kinetics at third rocker reveal a significant reduction in power in children with hemiplegia (Olney et al, 1990). The antagonists in over-lengthened position are also weak and lose mechanical advantage to impose normal lengthening on the hypertonic, shortened agonists via reciprocal innervation.

Muscle transformation: In the context of spasticity, motor units transform to regulate muscle tension at a lower level of neuronal organisation, enabling the child with CO to support bodyweight during gait (Leonard et al, 1991). As ordinarily phasic (Type I) muscles are routinely used tonically, Type I fibers disappear or convert to Type II, slow-twitch fibers (Handelsman and Glasser, 1986, Gage, 1991). Fast active movements requiring precise timing become impossible (Leonard et al, 1991).

Numerous animal studies have consistently shown sarcomere (muscle cell) number reduction in series and consequent loss of muscle length secondary to prolonged

^B For a detailed discussion of normal and abnormal muscle actions at each sub-phase of gait and related kinetics and kinematics in the three cardinal planes, the reader is referred to Gage (1991) chapters 4 and 5. Dr Gage and Gillette Children's Hospital have also produced 2 excellent instructional videotapes: <u>Normal Gait and Principles of Pathologic Gait in Cerebral Palsy</u>. For information, contact GCH, 200 East University Avenue, St Maul MN 55101

immobilization of nonspastic muscles in the shortened range and in response to excessive contraction imposed by electrical stimulation. Mechanically shortened muscle shows reduced amounts of passive and active tension compared with normal tissues. Peak tension occurs at approximately the point in the range at which the muscles are immobilized (Tabary et al, 1972, Tabary et al, 1976, Tardieu and Tardieu, 1987, Williams and Goldspink, 1978, Cusick, 1995). Shortened muscle cannot achieve adequate pre-contractile elongation and therefore loses the enhancement of the power of the ensuing contraction.

Example: Many children with hypertonous lack specific muscle strength - a function of sarcomeres in parallel; adequate contractile velocity - a function of the number of sarcomeres in series; and adequate extensibility of the antagonist muscle groups - a function of muscle transformation and physiologic length adaptation - to achieve preswing propulsion as an energy source for forward momentum, and to achieve normal pre-stance foot positioning. Furthermore, contraction times are often prolonged in the hypertonic and shortened triceps surae muscles and their phasic function and timing at third rocker are usually impaired (Gage, 1991, Dietz and Berger, 1995).

Therapeutic and Orthotic implications - muscle transformation/weakness

Evaluate active control of isolated joint motions needed to effect efficient gait function, and consider whether orthotic features would enhance, substitute for or influence those motions. For example, to address the problem of limited active ankle DF at terminal swing, consider adding a DF-assist feature to an articulating AFO (3). Because atrophy of the triceps surae musculature typically occurs in children who wear AFOs which stop PF at 0° or in 5° DF, whenever possible, permit a few degrees of PF in an articulating system for first and third rocker, even if a DF assist is needed for toe clearance in swing phase. Institute a daily regimen of closed-kineticchain strengthening - even 10 minutes per day - to activate and strengthen the calf musculature when a solid or articulating ankle system that restricts ankle PF is required for community ambulation.

Incomplete terminal swing knee extension is a difficult problem because of the combined effects of the presence of immature CNS control mechanisms, muscle weakness and muscle transformation. I would advise therapists to begin work on improving terminal swing knee extension by equipping and training the child's caretakers to institute a daily positioning program to gradually, passively elongate shortened hip flexors, adductors and hamstrings as a mechanical prerequisite to achieving the functional pelvo-femoral disassociation and knee joint ROM needed or all activities, including gait. Hamstrings strength is required for active hip extension as a source of forward momentum during early second rocker (Hoffinger et al, 1993). I therefore prefer to try to preserve any strength potential by using conservative rather than surgical lengthening techniques, the latter of which have as vet proved to be less that satisfactory (Hoffinger et al, 1993, Baumann, 1994, Gage, 1994a). First catch passive knee extension with hip flexed 90° (and the child in supine position) should occur with the knee in less that 30° of flexion (Katz et al, 1992). I also work for active hamstrings elongation via reciprocal inhibition by prescribing active knee extension exercises with hip flexed in sitting (Kendall et al, 1993, Kramer and McPhail, 1994, Conti and Girolami, 1995).

In the event that inadequate terminal knee extension is at least in part a function of a diminished propulsive moment at pre-swing, I work for more efficient forward momentum by stabilizing the foot in congruity for all weight-bearing activities; instituting daily closed-chain calf muscle strengthening exercises on narrow bobble boards; and the use manual assist in gait training to achieve more elevation of the center of mass at midstance (Olney, 1989).

I suggest these orthotic design features to attempt to increase terminal knee extension in swing:

- Provide an orthosis which aligns the foot in a stable configuration to promote more efficient propulsion prior to the swing phase.
- Add potential propulsion assist by significantly reducing (not eliminating) flexibility of the distal sole under the full length of the toes (Lehmann, 1993).
- Add DF resist if appropriate to try to gain more influence from the distal toe spring (Lehmann, 1993).
- Consider adding a dynamic (elasticized) knee extension assist splint or orthosis^C for functional training (47).

Orthotic Implication: adjust degrees of freedom to reduce proximity of the loadbearing joints to the GRF

Compared with a solid AFO, an ankle-height orthosis that permits free ankle motion facilitates transitions from floor to standing, stair climbing and rising from a chair. It is also undeniably cosmetically appealing. My experience suggests that this orthotic design is most useful for independently ambulatory children who use no assistive devices and who present with minimal to moderate pronatory foot and ankle alignment problems. However, when distal stabilization is needed at the ankle as well as the foot to gain more efficient proximal joint alignment and function, the low-cut orthosis can become a functional disadvantage. Clinicians who are actively engaging children in movement training programs are pressing orthotic engineers to provide increasingly "tunable" and influential orthotic designs and joints for children who require more or less ankle stability for different situations.

When proximal and distal weakness is evident, try providing more stabilization at the ankle and foot joint excursions with coactivation. The child with neuromotor deficit cannot elicit appropriate muscle coactivation. Consider instituting training periods of selected joint motion restriction to simplify the child's motor learning strategy at targeted joints, such as the hip or the ankle and foot (Reyerson, 1988, Butler and Nene, 1991, Butler and Major, 1992, Farmer and Butler, 1994).

Examples: Using short-leg casts designed to align and stabilize the foot and leg, we discovered the principle of targeting joints for focused postural and gait training by

^c SKO Knee Extension Assist Orthosis - Scott Orthopedics, 1831 East Mulberry, Ft Collins, CO 80524

limiting degrees of freedom distally. We routinely observed improved weightbearing alignment and function at the trunk, hip, and knee while the child wore the casts (13).

Applying the same principle to management of persistent knee flexion in stance and swing, I apply a knee extension assist splint to one knee to simplify the achievement of organized weight shifts onto the ipsilateral foot and to facilitate kicking a ball with the opposite foot. Ball kicking is a phasic activity that involves the normal components seen at terminal swing. I also use knee extension assist splints or cylindrical, fulllength, bivalved casts to help in using a small bobble board successfully for specific closed-chain strengthening of the ankle and foot musculature, and to more easily address problems of strength and mobility at the hips.

To manage varying ankle joint control demands imposed by different functional circumstances, try applying an anterior shell to an articulated device. Dual axis ankle joints mounted to ankle-height, total contact foot orthoses on metal uprights with a calf cuff offer some ankle motion adjustment options (Lehmann, 1993, Small, 1993, Harris, 1994). Seelye (1994) uses 1/8-inch thickness Aquaplast-T^{tm D} as a medium for making temporary and prototype devices. He has modified the standard solid AFO trim lines by cutting a deep, narrow, posterior U-cut to the proximal calcaneus, flattening the sides of the shaft section slightly, and adding wide velcro straps to close the entire shaft section. The straps reduce but do not eliminate sagittal plane motion, and can be adjusted in length and elasticized to vary the available range of ankle motion.

PRINCIPLES OF MECHANICS, KINETICS AND THE CLOSED KINETIC CHAIN

Clinical researchers have produced volumes of data and literature pertaining to mechanical features of normal and abnormal gait and other functional skills. Their observations have provided the clinical management team with new insights into designing orthotic interventions for children with CP that will more effectively enhance the children's functional status and reduce the destructive effects of abnormal forces on load-bearing joints.

The Principle of the Force Couple (M = F X D)

In biomechanics, a force couple operates in such a way that as the muscle shortens and becomes mechanically weaker, the perpendicular lever arm (D) of the muscle force vector (F) to the axis of rotation lengthens, thereby maintaining a constant magnitude of the moment (M) produced by the muscle (Gage, 1991). The influence of normal bone design on muscle forces demonstrates this principle. For example, the long posterior segment of the calcaneus sets the tendon of the ankle plantaflexor Force at a considerable perpendicular Distance from the ankle joint axis, effectively increasing the PF Moment both for eccentrically generating the PF power burst during third rocker.

Applying these fundamental principles of leverage and the length-tension relationship to the joints and muscles of the foot, Michaud (1993) illustrates and describes the

^D Aquaplast-Ttm is available from Smith & Nephew/Rolyan. One Quality Drive, Germantown, WI 53002

relationship between the triplanar inclination (or pitch) of a foot joint axis and function of the muscles acting upon that axis. When the foot joints are aligned in stable configuration - neither pronated nor supinated, such that radiologic findings would fall within age-related normal limits - one can expect normal muscle function to occur across the joint axes. When the foot joints are abnormally expected to adversely affect the magnitude and nature of related muscle Forces: shortened muscles gain perpendicular Distance from the deviated axes, and thereby lose their corrective Moment.

Management implications of lever arm dysfunction

To maintain optimum lever arm advantage at the pelvis and segments proximal to the foot, use rigorous positioning, strengthening of antagonist muscles and conservative contracture management techniques. Seek timely orthopedic assistance, using osteotomies to restore optimum bony lever arms (Cavagna et al, 1968). Institute postural and weight shift training that, if necessary, incorporates splints or orthotic means of reducing excessive deviation of load-bearing joint axes from the ideal **GRF** vector, with the goal of reducing the associated increased work load on the antigravity musculature.

Pertaining to the foot, know the normal and ideal age-related architecture of the weightbearing foot and use the plantar, medial and lateral aspects of the orthosis to achieve appropriate alignment in all three planes. Restore optimum axial inclinations to all joints of the foot to attempt to restore normal potential Moment to the musculature.

Principles of Kinetics

The remarkable efficiency of normal gait is the product of a combination of mechanical and muscle activation factors. Eccentric (lengthening) muscle activation prevails in normal gait. Eccentric muscle action represents negative work, spends metabolic energy more efficiently than concentric (shortening) muscle activation, and provides elements of shock absorption, joint stabilization and deceleration. Concentric muscle action provides acceleration, represents positive work, and puts mechanical energy into the walking system by using more metabolic energy (Winter, 1978, Olney, 1989, Gage 1994, Rab, 1994).

The primary source of power needed for walking is gained by 3 power bursts: two at the hip and one at the ankle. The ankle plantarflexion power burst at propulsion provides about 45% of the power required for walking and contributes energy for swing-phase momentum of the same limb (Robertson and Winter, 1980, Winter, 1980, Gage, 1994). The hip flexors at initial swing rapidly add 20% walking energy to that provided by the ankle PF burst at propulsion, and assist the hip extensors on the stance side to raise the trunk and pelvis (body center of mass) to the maximum distance from the foot at midstance. Following weight assumption, hip extension during early second rocker provides about 30% of walking power (Gage, 1989, 1994).

Only two smooth rises and falls in kinetic energy should occur during the gait cycle, as the inverse patterns of rises and falls in potential energy, Inman (1993) suggests that muscle activation normally contributes about 50% to the total energy cost. Disrupting

the smooth exchange of potential and kinetic energy requires additional metabolic resources that increase the energy cost of walking (Olney, 1989).

Therapeutic implication of kinetic events

Olney (1989) advises clinicians to try to minimize isometric muscle activity in gait, as it constitutes an efficient use of energy spent without the advantage of achieving movement. She also advises us to strive to effect the positive work normally gained from power bursts at the ankle and hip. the therapist's tools include strengthening in functional context in the closed and open kinetic chains, maintaining normal joint alignment whenever possible, and occasionally using functional electrical stimulation as an adjunctive modality (Carmick, 1993, Lutz et al, 1993).

Orthotic implications of kinetics

A solid ankle substitutes a part for the decelerating function of the soleus, but fails to permit the normal lengthening of the triceps surae muscle that occurs at terminal stance. The solid ankle also provides static versus dynamic stabilization of the tibia against falling forward on the foot during second rocker. Any orthosis that stops ankle PF at 0° or less imposes an abrupt moment on the smooth exchange of energy during the stance phase and prohibits the $10^{\circ}-20^{\circ}$ of PF that normally occurs with the ankle PF power burst at propulsion. Isometric ankle PF might occur inside the orthosis, but that energy is not spent in generating movement as the orthosis blocks the foot against plantarflexing.

An articulating ankle with free DF and limited PF permits the tibia to free-fall over the foot, allows lengthening of the triceps surae musculature but offers no decelerating influence on the tibia. Dual axis ankle joints using a spring as a PF stop might offer a measure of the desired effect, but can be expected to facilitate toe drag in swing. In my experience, elasticized posterior DF check straps routinely fail to effectively mimic the soleus in second rocker and have shown no evidence of facilitating propulsion at third rocker. The check strap idea seems sound as a DF-resist and PF-assist, but the materials available to implement the concept effectively are not yet adequate.

Other available options for regulating but not restricting ankle motion include 2 modifications to the solid AFO: gradually trimming back the anterior borders at the malleoli and cutting a deep narrow posterior U to the proximal calcaneus. The former adaptation allow the device to strain into dorsiflexion in increasing increments, but it prohibits PF. The latter U-cut adaptation employs variable strapping systems on the shaft section (described previously) and offers a means of passively reducing rather than blocking ankle DF at tibial progression and ankle PF at propulsion in cases in which hip, knee and ankle extensor weakness is not significant enough to result in severe calcaneus deformity and associated crouch posture.

I anticipate the inevitable development of affordable orthoses that can enhance the propulsive moment using energy storage and release mechanisms which mimic the decelerating moment of second rocker and the accelerating power burst at propulsion. Some of the following suggestions are therefore somewhat futuristic:

- Ideally, apropulsive, shock-absorbing foot pronation resolve to congruity at terminal stance and the foot converts to a rigid lever in supination at third rocker, enhancing the mechanically propulsive ankle PF moment. Orthoses that promote propulsion should therefore restore adequate congruency of the subtalar and midtarsal joints at terminal stance, in preparation for energy efficient propulsion, and they should permit the 10°-20° of unrestricted ankle PF mobility that occurs at third rocker if at all possible.
- Currently, articulated orthoses provide only sagittal-plane motion. I suggest that small increments of frontal plane motion be provided as well. I can accomplish this adaptation be carving a vertical slot at each joint to permit the mechanical axis to slide with heel inversion of about 5° while the calcaneal mold secures the hindfoot in the device. An elasticized eversionresist/inversion-assist band added to the medial aspect of the ankle joint might further enhance the supination moment at propulsion.
- I formally request that biomechanical engineers design a **DF-resist** feature for articulating devices which offers the progressing tibia <u>an energy-storing</u>. <u>resilient anterior stop</u> versus an abrupt block. The DF-resist would ideally compress to allow up to 10° of tibial progression and return the stored energy as added PF force when the heel rises. Hydraulics or spring steel technology might eventually foster this dynamic capability (Herr, 1994).
- Rocker soles on shoes, when combined with DF resist (not necessarily a solid ankle), can mechanically enhance forward momentum by increasing the speed of progression over the shoe at third rocker.
- Having secured the tibia against excessive forward progression (ankle DF resist), I can suggest two device modifications that are designed to provide spring-like resistance to passive dorsiflexion of the MTP/toe area at third rocker:
 - Stiffen, but do not make rigid, the distal floor of the device under the toes and metatarsal heads (Lehmann, 1993).
 - Add a carbon steel propulsion plate^E of appropriate stiffness to the plantar orthosis or to the interior sole of the shoe under the orthosis. The concept is already used in prosthetics, but the desired spring effect as propulsive assist has not yet been documented to my knowledge.

The expected gain in PF moment at propulsion may be attributed to either or both of the following events: as a return of energy stored by dorsiflexing the device and taxing its elastic recoil properties; and by increasing the length of the plantar lever arm over which the body must progress throughout third rocker.

- Herr (1994) has developed and patented a carbon steel "propulsion sole" sole that is expected to go into production in 1996. The sole is comprised of 2

^E Springlite "toe filler" carbon steel propulsion plate: CRP inc. dba Springlite, 1006 West Beardsley Place, Salt Lake City, UT 84119

layers of carbon steel. The uppermost layer is bent upward at the region of the metatarsophalangeal joints, achieving maximum height under the heel, and is melded to the flat bottom layer. The elevated posterior section absorbs shock at first rocker by compressing. Through second rocker, however subtly, the upper sole strains to return to the original elevated position, effectively thrusting the body forward with increasing vigor as the heel is unloaded.

The 2 layers of flexible carbon steel under the metatarsal heads and toes presumably serve as distal spring by resisting passive toe extension and assisting PF through third rocker (as described previously). I plan to collaborate with Mr Herr to evaluate the propulsive efficacy of this adaptation in conjunction with a DF-resist AFO for children with crouch gait as soon as a prototype is available.

Biomechanical Aspects of the Closed Kinetic Chain

Given adequate ligament support at the knee and hip joints and a competent talocrural mortice, change sin foot position predictably alter proximal limb and pelvic alignment and related muscle activation through the closed kinetic chain (CKC). Calcaneal eversion drops the sustentaculum tali out from under the medial talar neck. In response, the talus adducts (ie rotates medially) and plantarflexes (and probably also everts) (Perry, 1983, Michaud, 1993). The structural mortice at the talo-crural joint commits the malleoli, and thereby the leg, to follow this talar migration in any plane other than sagittal. Ligament and muscle attachments at the knee and hip communicate the resulting medial deviations of the leg in the transverse and frontal planes to the knee and hip as medial femoral rotation and adduction (Coplan, 1989, Michaud, 1993).

During normal gait, the advancing swing limb and rotating pelvis impose lateral torque forces on the stance limb and foot (Michaud, 1993). these torque forces provide the modelling mechanism for torsional reduction in the femur and lateral torsion in the leg unit during skeletal growth (Tachdjian, 1990).

In the context of the pronation-related alignment disturbances just described, the center of mass is displaced anteriorly and predictable patterns of muscle activation occur to sustain the upright position. To experience the altered load distribution on the foot, the interference with lateral weight shift, the loss of propulsion and of terminal swing knee extension, and the onset of compensatory isometric muscle activity, please stand, shift your pelvis from side-to-side and walk with full bilateral foot pronation. Lack of weight shifting onto the heels and lateral pillars of the feet due to persistent loading on the medial/anterior aspects also results in weakness of the muscles of foot supination and first ray PF.

Orthotic implication of the CKC

Use the foot as a link in the CKC to help to align the proximal load-bearing joints and to facilitate more efficient muscle function at the knee, hip and pelvis. Several clinicians have compared barefoot standing to standing in orthoses, and reported improved radiologic findings when the child was x-rayed while wearing the orthoses (30, 58, Bleck and Berzins, 1977, Bordelon, 1980). I recommend effectively resolving

pronation before working through therapeutic exercise and gait training to alter the modelling moments afforded by converting weight bearing torque forces from medial to lateral. Early intervention takes advantage of the plasticity of the skeletal system if modelling effect is a management goal.

Embrey et al (20) reported the results of a single case study featuring a child with diplegia and knee flexed gait. The subject was evidently neither hypertonic nor spastic, as she showed no ROM restrictions and was not described as "spastic diplegic". The authors found that implementing ankle-height total contact orthoses produced an immediately apparent (though minimal) reduction in the angle of knee flexion at midstance in gait. This result suggests that knee alignment improved via the CKC because the calcaneus and talus were reportedly restored to a more appropriate weightbearing alignment in the orthoses.

If the foot is stabilized orthotically for maximum propulsion, the shock-absorption afforded by first rocker does not occur within the foot structures, and should be added to the heel of the shoe as a cushion. Up to 5° of varus posting under the pronating foot can provide a mechanical assist to reducing knee and hip flexion via the ankle mortice by using calcaneal inversion and the sustentaculum tali to abduct and dorsiflex the talus. Varus posting can impose lateral instability on the foot and ankle, particularly if the walking base is narrow, so safety precautions and close monitoring are required and a short shaft section might be added to a foot support system for protection against lateral sprains. See the previous section for other ideas regarding dynamic supination-assist/pronation-resist design suggestions.

Provide Different Orthotic Devices for Different Purposes

The time has come to dispense with the notion that one device meets all management needs. A solid AFO selected to manage contracture of the triceps surae muscles and worn all day can, in the absence of a diligent program of closed-chain strengthening activities to build triceps surae strength, promote atrophy in the very muscle group that needs normalized function and strength in gait. A device prescribed for a child at one point in time might need replacing with one of a different design 4 or 6 months later as functional skills improve or deformity reduces.

Interventions for contracture management - such as night splinting (60) or two hours of weightbearing in 10° of ankle dorsiflexion in a total-contact AFO to maintain DF ROM after casting (DeLacey, 1994) - differ greatly from those designed to enhance function. The therapeutic exercise setting which features guided joint placement and weight shifts differs greatly from a 3-hour excursion at a shopping mall. Orthoses prescribed with cost containment as the primary concern (the familiar on-device-peryear regulating from funding sources) deny the growing child the optimum fit and potential contribution of the orthoses to the course of deformity management and developmental habilitation, and can instead become a deterrent to progress.

I suggest that a cost-effective solution to the problems of meeting management demands with a variety of devices and of providing clinical trials of questionable orthotic prescriptions is available to orthotists and therapists who can demonstrate suitable skills (Sussman and Smith, 1987, McClure et al, 1994). Consider complementing high-temperature orthoses designed for durability with custom-molded low-temperature Aquaplast-Ttm splints designed for other management purposes. Splints take a fraction of the time to fabricate compared with comparable orthoses; are direct-molded on the child; and since they cost considerably less than their orthotic counterparts, for the same allocation of funds several splints could be made over the course of the same time allotment assigned to the orthotic prescription, for infants, toddlers and preschoolers who are experiencing rapid growth and significant change in status.

Similarly, while an older child recovers from traumatic brain injury or selective dorsal rhizotomy or orthopedic surgery, we can provide custom-molded splints to address the short-term positioning, alignment and stabilization needs, and adjust or remake them to address changing functional or management requirements. The role of splint-maker should fall to the most capable and most interest team members. The problem of establishing billing mechanisms for orthotists that will support the routine use of splints as prototypes, as alternatives to more costly orthoses for very young children and as supplemental orthotic interventions, cannot be insurmountable if their value is realized.

SUMMARY

This discussion is an overview which uses examples to raise many issues pertaining to clinical preparation for orthotic design selection. Inevitably, some factors have escaped consideration in this paper. Managing CP is a complicated and often arduous, ongoing process. Orthotic efficacy is not an isolated factor in the context of the problems associated with CP. Instead, it manifests the combined efforts of all participating members of the management team, including the child and family. No single team member can claim responsibility for functional outcome.

The clinical management team must account for its contribution to the child's functional abilities by using such objective clinical measures as soft tissue extensibility, muscle strength, joint alignment, oxygen consumption, velocity, step and stride length, kinematics and kinetics and measures of gross motor function; and such subjective measures as reports of pain or satisfaction on scales of 1 to 10. However, I believe that prevention is worth more than cure, and propose that if we successfully protect load-bearing joints from enduring destructive deforming forces that logically and reportedly lead to premature degenerative changes, then in 20 or 30 years our efficacy will be evident in a population of adults with CP who can participate as community members without enduring limitations on their quality of life imposed by a combination of longstanding muscle weakness, muscle overuse syndromes or joint pain.

Meanwhile, new research findings and technology inspire us to continually reconsider our methods, redirect our efforts and refine our management protocols. Standing knee-deep in this fast-moving river of new resources and their influences on management rationale and procedures, team members and caretakers must share the load of learning and hold each other steady. Collectively, we're the human bridge that the child with cerebral palsy crosses into adulthood.

REFERENCES

Aharonson Z, Voloshin A, Steinbach T B, Brull M A and Farine I. Normal footground pressure pattern in children. Clinical Orthopaedics and Related Research 1980 150, 220-223. Aharonson Z, Arcan M and Steinbach T V. Foot-ground pressure pattern of flexible flatfoot in children, with and without correction of calcaneovalgus. Clinical Orthopedics and Related Research 1992 <u>278</u> 178-182.

Barber E L. Strength and range of motion examination skills for the clinical orthotist. Journal of Prosthetics and Orthotics 1993 5(2), 49-51.

Baumann J U and Zumstein M. Experience with a plastic ankle-foot orthosis for prevention of muscle contracture. Developmental Medicine and Child Neurology 1985 27(5), 83 Abstract.

Baumann J. Personal communication. November 22, 1944.

Berger W, Quintern J and Dietz V. Pathophysiology of gait in children with cerebral palsy. Electroencephalography and Clinical Neurophysiology 1982 <u>53</u>, 538-548.

Berghof R, Doederlein L and Seibel A. The effect of 4-week short-leg casting on gait in cerebral palsy children. Gait and Posture 1995 3(2), 101.

Bleck E E and Berzins U J. Conservative management of pes valgus with plantarflexed talus, flexible. Clinical Orthopedics and Related Research 1977 <u>122</u>, 85-94.

Bohannon R W and Larkin P A. Passive ankle dorsiflexion increases in patients after a regimen of tilt table-wedge board standing. Physical Therapy 1985 <u>65(11)</u>, 1676-1678

Bohannon R W, Tiberio D and Zito M. Selected measures of ankle dorsiflexion range of motion: Differences and Correlations. Foot and Ankle 1989 <u>10(2)</u>, 99-103.

Bordelon R L. Correction of hypermobile flatfoot in children by moulded insert. Foot & Ankle 1980 <u>1(3)</u>, 143-150.

Butler P B and Nene A V. The biomechanics of fixed ankle foot orthoses and their potential in the management of cerebral palsied children. Physiotherapy 1991 77(2), 81-88.

Butler P B and Major R E. The learning of motor control: biomechanical considerations. Physiotherapy 1992 <u>78(1)</u>, 6-11.

Carmick J. Clinical use of neuromuscular stimulation for children with cerebral palsy. Physical Therapy 1993 <u>73(8)</u>, 505-513.

Cavagna G A, Dusman B and Margaria R. Positive work done by a previously stretched muscle. Journal of Applied Physiology 1968 <u>24</u>, 21.

Cavagna P R, Rodgers M M and Liboshi A. Pressure distribution under symptom-free feet during barefoot standing. Foot and Ankle 1987 7(5), 262-276.

Cerny K, Perry, J and Walker J M. Adaptations during the stance phase of gait for simulated flexion contractures at the knee. Orthopedics 1994 <u>17(6)</u>, 501-513.

Clancey W G. Anterior cruciate ligament functional instability. Clinical Orthopaedics and Related Research 1983 <u>172</u>, 102-106.

Conti R and Girolami G. The effects of a combined muscle strengthening program and NDTA Network 1995 May 1-11.

Coplan J A. Rotational motion of the knee: a comparison of normal and pronating subjects. Journal of Orthopedic and Sports Physical Therapy 1989 <u>March</u>, 366-369.

Cuddleford T J, Thomas S S, Alona R and Freeling P. The effect of flexed knee posture on energy consumption. Gait and Posture 1995 3(2), 105.

Cusick B. Splints and casts: managing foot deformity in children with neuromotor disorders. Physical Therapy 1988 <u>68(12)</u>, 1903-1912.

Cusick B D. Progressive casting and splinting for lower extremity deformities in children with neuromotor deficit. Tuscan AZ: Therapy Skill Builders 1990.

Cusick B. Serial casts: their use in the management of spasticity-induced deformity. Tuscan AZ: Therapy Skill Builders 1990-a.

Cusick B. Developmental Features Chart. Course syllabus - Developmental Biomechanics: A Study of Growing Lower Extremity Structures and Related Assessment Procedures. Complilation of findings obtained during 9 course lab sessions, including 50 non-disabled children. May 11 1994.

Cusick B D. Unpublished data from course-related assessment labs on non-disabled children. 1995.

Cusick B and Stuberg W A. Assessment of lower-extremity alignment in the transverse plane; implications for the management of children with neuromotor dysfunction. Physical Therapy - Pediatric Orthopedics Series 1992 71(1), 3-15.

DeLacey M. Personal communication. Senior Physiotherapist, Queensland Spastic Welfare League, PO Box 386, Fortitude Valley, Queensland, Australia 4006. November 11 1994.

Dietz V and Berger W. Cerebral Palsy and muscle transformation. Developmental Medicine and Child Neurology 1995 <u>37</u>: 180-184.

Duckworth T. The hindfoot and its relation to rotational deformities of the forefoot. Clinical Orthopaedics and Related Research 1983 <u>177</u>, 39-48.

Farmer S E and Butler P B. Conservative treatment of crouch gait. Gait and Posture 1994 2(1), 41-42.

Gage J R. An overview of normal and cerebral palsy gait. Journal of Biomechanics 1989 <u>4(2)</u>, 379-401.

Gage J R. Gait analysis in cerebral palsy. Clinics in Developmental Medicine No. 121 New York, HY. Cambridge University Press 1991. Gage J R. Clinical use of kinetics for gait pathology in cerebral palsy. Gait & Posture 1994 2(1), 36-37.

Gage J R. From the past to the future on "a less travelled road". Gaig & Posture 1994-a 2(1), 39-41.

Gritzka T L and Gerlach C. Serial short-leg casts for the treatment of equinus deformity in cerebral palsy. Developmental Medicine and Child Neurology 1986 28(5). Supplement 53, Abstract 8.

Grumbine N A. The varus components of the forefoot in flatfoot deformities. Journal of the American Podiatric Medical Association 1987 77(1), 14-20.

Handelsman J E and Glasser R A. A comparative histological and clinical evaluation of cerebral-palsied muscle. Development Medicine and Child Neurology 1986 <u>28(5)</u>, Supplement 53:35 Abstract.

Harris D R. Personal communication. Orthotic and Prosthetic Specialities, Laurel MD July 30 1994.

Harrison A. Spastic cerebral palsy: possible spinalintermeuronal contributions. Developmental Medicine and Child Neurology 1988 <u>30</u>, 769-780.

Herbert R. Passive mechanical properties of muscle and their adaptations to altered patterns of use. Australian Journal of Physiotherapy 1988 <u>34(3)</u>, 141-149.

Herr H. Personal cummunication. Engineering Sciences Building, Miomechanics laboratory, Harvard University, Cambridge MA, June 20 1994.

Hicks R N, Durinick J R and Gage J R. Differentiation between idiopathic toe-walking and cerebral palsy. Journal of Pediatric Orthopedics 1988 <u>8(2)</u>, 160-163.

Hilyard A. Recent developments in the management of patello-femoral pain: The McConnell program. Physiotherapy 1990 <u>76(1)</u>, 559-565.

Hoffinger S A, Rab G T and Abou-Ghaida H. Hamstrings in cerebral palsy crouch gait. Journal of Pediatric Orthopedics 1993 <u>13(6)</u>, 722-726.

Inman V T. Human locomotion. Clinical Orthopedics and Related Research 1993 288, 3-9 (Reprint of his 1966 publication).

Jones J and McLaughlin J F. Mechanical efficiency of children with spastic cerebral palsy. Developmental Medicine and Child Neurology 1993 <u>36</u>, 614-620.

Katz K, Rosenthal A and Yosipovitch Z. Normal ranges of popliteal angles in children. Journal of Pediatric Orthopedics 1992 <u>12</u>, 229-231.

Kendall F P, McCreary E K and Provance P G. Muscles: testing and function, fourth edition with posture and pain. Baltimore MD: Williams and Wilkins 1993.

Kisner C and Colby L A. Stretching. In C Kisner and L A Colby (Eds): Therapeutic Exercise - Foundations and Techniques, second edition 109-142 Philadelphia PA, F A Davis Company, 1990.

Kramer J F and MacPhail H E A. Relationships among measures of walking efficiency, gross motor ability and isokinetic strength in adolescents with cerebral paalsy. Pediatric Physical Therapy 1994 6(1), 3-8.

LaPlaza F J, Root L, Tassanawipas A and Glasser D B. Femoral torsion and neck-shaft angles in cerebral palsy. Journal of Pediatric Orthopedics 1993 <u>13</u>, 192-199.

LaRock K. Personal communication. Department of Physical Therapy, The Children's Hospital, Denver CO, 1994.

Lehman J F. Push-off and propulsion of the body in normal and abnormal gait. Clinical Orthopedics and Related Research 1993 <u>288</u>, 97-108.

Lehmkuhl L D and Smith L K. Brunnstromm's clinical kinesiology, fourth edition, 292 Philadelphia PA, F A Davis Company, 1983.

Leonard C T, Hirschfeld H and Forssberg H. The development of independent walking in children with cerebral palsy. Developmental Medicine and Child Neurology 1991 <u>33</u>, 567-577.

Lieber R L. Skeletal muscle adaptability. II Muscle properties following spinal cord injury. Developmental Medicine and Child Neurology 1986 <u>28</u>, 533-542.

Lieber R L. Skeletal muscle adaptability. 1 Review of basic properties. Developmental Medicine and Child Neurology 1986-a <u>28</u>, 390-397.

Lutz G E, Palmitier R A, An K N and Chao E Y S. Comparison of tibiofemoral joint forces during open-kinetic-chain and closed-kinetic-chain exercises. Journal of Bone and Joint Surgery 1993 <u>73-A(5)</u>, 732-739.

Maitland G D. Peripheral manipulation, second edition. Boston: Butterworths 1977.

Martin T. Personal communication. Rocky River, OH, 1995.

Mazur J M, Shanks D E, Cummings, McCluskey W P, Federico L and Goins M. Nonsurgical treatment of tight achilles tendon. In M D Sussman (Ed): The diplegic child: evaluation and management. Rosemont II: American Academy of Orthopaedic Surgeons 1992, 343-353.

McClure P W, Blackburn L G and Dusold C. The use of splints in the treatment of joint stiffness: biologic rationale and an algorithm for making clinical decisions. Physical Therapy 1994 <u>74(12)</u>, 1101-1107.

McConnell J. The management of chondromalacia patellae: a long-term solution. The Australian Journal of Physiotherapy 1986 32(4), 215-223.

McCrea J. Pediatric orthopedics of the lower extremity: an instructional handbook. Mount Kisco, NY: Futura Press 1985. McPoil T G. Anatomical characteristics of the talus in relation to forefoot deformities. Journal of the American Podiatric Medical Association 1987 <u>72(2)</u>, 77-80.

McPoil T G and Brocato R S. The foot and ankle: biomechanical evaluation and treatment. In J A Gould (Ed): Orthopedic and Sports Physical Therapy, second edition: St Louis C V Mosby Co 1990, 293-321.

Michaud T C. Foot orthoses and other forms of conservative foot care. Baltimore MD: Williams and Wilkins, 1993.

O'Dwyer N J, Neilson P D and Nash J. Mechanisms of muscle growth related to muscle contracture in cerebral palsy. Developmental Medicine and Child Neurology 1989 <u>31</u>, 543-552.

Okamoto T and Jumamoto M. Electromyograph Study of the learning process of walking in infants. Electromyography 1972 12(2), 149-158.

Olney S J. New developments in the biomechanics of gait with cerebral palsy. Topics in Pediatrics - Lesson 1. Alexandria VA: American Physical Therapy Association Inc 1969.

Olney S J, MacPhail H E A, Hedden D M, Boyce W F. Work and power in hemiplegic cerebral palsy gait. Physical Therapy 1990 <u>70(7)</u>, 431-438.

Perry J. Anatomy and biomechanics of the hindfoot. Clinical Orthpedics and Related Research 1983 <u>177</u>, 9-15.

Powell H D and Cantab M A. Pes planovalgus in children. Clinical Orthopedics and Related Research 1983 77, 133-139.

Rab G T. Diplegic gait: is there more than spasticity? In M D Sussman (Ed): The diplegic child: evaluation and management. Rosemont IL: American Academy of Orthopaedic Surgeons 1992.

Rab G T. Musle. In J Rose and J G Gamble (eds). Human walking. Baltimore MD: Williams and Wilkins 1993.

Rang M, Silver R and de la Glaza J. Cerebral palsy: In W W Lovell and R B Winter (Eds): Pediatric orthopedics, second edition. Philadelphia PA: J B Lippincott Company 1986 345-396.

Reimers J. Contracture of the hamstrings in spastic cerebral palsy. Journal of Bone and Joint Surgery 1974 <u>56-B(1)</u>, 102-109.

Reimers J. Clinically based decision making for surgery. In M D Sussman (ed): The diplegic child. Rosemont IL: American Academy of Orthopedic Surgeons 1992.

Reverson D D. The foot in hemiplegia. In G Hunt (Ed: Physical therapy of the foot and ankle: New York: Churchill Livingstone 1988, 109-131.

Robertson D G E and Winter D A. Mechanical energy generation, absorption and transfer amongst segments during walking. Journal of Biomechanics 1980 <u>13</u>, 845-854.

Romero P W and Rice J. Clinical use of the knee hyperextension block orthosis. Pediatric Physical Therapy 1992 4(4), 205-207.

Rose J, Gamble J, Medieros J and Burgos A. Energy cost of walking in normal children and those with cerebral palsy: Comparison of heart rate and oxygen uptake. Journal of Pediatric Orthopedics 1989 <u>9(3)</u>, 276-279.

Rose N E, Feiwell L A and Cracchioio A III. A method for measuring foot pressures using a high resolution, computerized insole sensor: the effect of heel wedges on plantar pressure distribution and center of force. Foot and Ankle 1992 <u>13(5)</u>, 263-270.

Sapega A, Quedenfeld T C, Moyer R A and Butler R A. Biophysical factors in rangeof motion exercise. The Physician and Sports Medicine 1982 <u>9(12)</u>, 57-65.

Sarver W. Personal communication. Wendy lives in Northport, NY, 1994.

Steelye R. Personal communication. Orthotic and Prosthetic Specialities, Laurel MC, 30 July 1994.

Seibel M O. Foot Function: A Programmed Text. Baltimore MD: Williams and Wilkins 1988.

Small G. Personal communication 10-11 September 1993.

Stallard J. Assessment of the mechanical function of orthoses by force vector visualization. Physiotherapy 1987 <u>73(8)</u>, 398-402.

Sussman M D and Smith M S. Instant orthoses: the aquaplast AFO. Developmental Medicine and Child Neurology 1987 <u>29(5)</u>, Supplement 55 (Abstract).

Sutherland D H. The pathomechanics of crouch gait in spastic diplegia. Orthopedic Clinics of North America 1978 <u>9(1)</u>, 143-154.

Sutherland D H, Cooper L and Daniel D. The role of ankle plantarflexors in normal walking. Journal of Bone and Joint Surgery 1980 <u>62-A</u>, 354-363.

Sutherland D H, Olshen R A, Biden E N and Wyatt M P. Anthropometric measurements and developmental screening. In Sutherland et al (eds): The Development of Mature Walking, New York NY, Cambridge University Press 1988, 36-37.

Sutherland D N and Valencia F. Pediatric gait: Normal and abnormal development. In J C Drennan (Ed) The child's foot and ankle, New York NY: The Raven Press Ltd 1992.

Tabary J C, Tabary C, Tardieu C, Tardieu G and Goldspink G. Physiological and structural changes in the cat's soleus muscle due to immobilization at different lengths by plaster casts. Journal of Physiology (London) 1972 <u>224</u>, 231-244.

Tabary J C, Tardieu C, Tardieu G, Tabary C and Gagnard L. Functional adaptation of sarcomere number of normal cat muscle. Journal of Physiology 1976 <u>72</u>, 277-291.

Tachdjian M O. Torsional (or rotational) deformities of the lower limbs. In Pediatric Orthopedics, second edition, Volume 4, Philadelphia PA, W B Saunders Company 1990.

Tardieu G and Tardieu C. Mechanical evaluation and conservative correction of limb joint contractures. Clinical Orthopedics and Related Research 1987 <u>219</u>, 63-70.

Tardieu C, Lespargot A, Tabary C and Bret M-D. For how long must the soleus muscle be stretched each day to prevent contracture? Developmental Medicine and Child Neurology 1988 <u>30</u>, 3-10.

Thomas S S, Moore C, Kelp-Lenane C and Norris C. The effect of joint position on dynamic electromyography and joint moments during gait. Developmental Medicine and Child Neurology 1992 <u>34(9)</u>, Supplement 66:16 Abstracts. (Fuller report to appear in Gait & Posture late in 1995.)

Tiberio D. Pathomechanics of Structural Foot Deformities. Physical Therapy 1988 68(12), 1840-1949.

Turner M S. The association between tibial torsion and knee joint pathology. Clinical Orthopedics and Related Research 1994 <u>302</u>, 47-51.

Turner M S and Smillie I S. The effect of tibial torsion on the pathology of the knee. Journal of Bone and Joint Surgery 1981 <u>63-B(3)</u>, 396-398.

Vaughan C, Davis B L and O'Connor J C. Dynamics of human gait. Champaign IL, Human Kinetics Publishers 1992.

Weber D. Clinical aspects of lower extremity orthotics. In Biomechanics in Gait: Weber and M Argo (Eds) Oakville, Ontario, Elgan Enterprises, 1990.

Weseley M S, Barenfield P A and Elsenstein A L. Thoughts on in-toeing and Outtoeing. Twenty Year's Experience with over 5,000 Cases and a Review of the Literature. Foot and Ankle 1981 2(1), 49-57.

Westin G W and Dye S. Conservative management of cerebral palsy in the growing child. Foot and Ankle 1983 4(3), 160-163.

Williams P E and GoldspinkG. Changes in sarcomere length and physiologic properties in immobilized muscle. Journal of Anatomy 1978 <u>127</u>, 459-468.

Winter D A. Energy assessments in pathological gait. Physiotherapy Canada 1978 <u>30</u>, 183-191.

Williams P E and Goldspink G. Connective tissue changes in immobilized muscle. Journal of Anatomy 1984 138-143.

Winter D A. Overall principle of lower limb support during stance phase of gait. Journal of Biomechanics 1980 <u>13</u>, 923-927.

Winter D A. Knee flexion during stance as a determinant of inefficient walking. Physical Therapy 1983 $\underline{63(3)}$, 331-333.

Yagi T and Sasaki T. Tibial torsion in patients with medial-type osteoarthritic knee. Clinical Orthopaedics and Related Research 1986 <u>213</u>, 178-182.

Yagi T. Tibial torsion in patients with medial-type osteoarthritic knees. Clinical Orthopedics and Related Research 1994 <u>302</u>, 52-56.

Young NL and Wright JG. Measuring pediatric physical function. Journal of Pediatric Orthopedics 1995 <u>15</u> 244-253.

Ziv I, Blackburn N, Rang M and Korseska J. Muscle growth in normal and spastic mice. Developmental Medicine and Child Neurology 1984 <u>26</u>, 94-99.

THE ORTHOTIC MANAGEMENT OF THE FOOT IN CEREBRAL PALSY

Gregory J Small CO

A well-molded, anatomically contoured foot interface provides the necessary purchase to align and hold the structures of the foot and to facilitate dynamic balance reactions. It is the biomechanical alignment of the foot structure that enables the successful transmission of the ground reaction forces. The dynamic response of the foot to the ground reaction forces during closed kinetic chain weight bearing results in postural adaptations higher up the lower limb structure.

"Biomechanical alignment is important for posture and movement. Alignment provides mechanical advantage for muscle activity and can provide stability with a minimum amount of muscle action. Posture is most efficient when the weight line passes through a joint in such a way that the joint is stable.

Mobility is necessary for efficient posture and movement. Range of motion is necessary for correct joint position for a specific activity. Muscles and fascia need to elongate and shorten appropriately so that correct biomechanical alignment can be achieved.

Biomechanical alignment is important for proprioception. Neuromotor activity depends on proprioceptive input from muscles and joints. Joint alignment appears to be crucial in determining muscle activity. For example, loading a joint (weight bearing) produces a normal postural response only if the joint is in correct alignment. Traction or compression at a joint can result in more or less muscle activity" (Cupps, 1988)

It is widely believed that contact at key points within the foot interface can also facilitate muscle action. If the interface is shaped to achieve contact at these key points it can provide active stimulation rather than simple passive support. Care must be taken, however, that this shaping does not provide painful stimulus resulting in withdrawal rather than muscle activation.

During the past 35 years the profession of orthotics has advanced considerably, especially in technical aspects. In some ways, however, very little has changed.

Kendall and Robson wrote in "Lower Limb Bracing in CP" in the Journal of Clinical Orthopedics, 1966, "It is obvious that the indications and the type of brace used not only vary with the clinical pattern of each individual patient, but also vary according to the objective that is hoped to achieve and even from center to center. A world wide survey conducted by this unit into the role of lower limb bracing in CP provided no less than 29 different indications, many of which constituted direct contraindications. Therefore at present it cannot be stated that there is unanimous agreement on this subject."

From this same time period Dr Winthrop Phelps wrote, "There have been remarkable advances in the treatment of CP patients over the last 30 years or so But there have also developed many fads which have come and gone. Therefore it is important not to publicise new methods of treatment until they have been tried out long enough to

determine their value in relation to other forms of treatment and to have careful evaluation of end results."

Much of the available literature on this subject reports that the use of well-designed, properly prescribed orthoses has a positive effect on the stance, posture and gait pattern of CP children. Studies show increased velocity and stride length, better balance reactions, as well as reduced energy expenditure and oxygen consumption (9, 26, 30, 33, 41, 43, 72)

A point of contention arises today within the orthotist/therapist/physician team regarding orthotic prescription principles. Traditionally orthotists have designed devices to enforce complete control with the aim of achieving a more normal gait pattern using a "brace what you see" approach. This approach has its merits in certain situations, however when the patient is a developing child receiving extensive therapy, the therapist quite often prefers a less controlling device which would allow modern, progressive therapy treatment techniques to encourage the child's own development. Orthotic design for the developing child thus employs a more goal oriented approach. The orthosis is used as a habitation aid or tool and its design reflects the specific therapeutic goal. (56)

Current foot management systems are based on three principles: biomechanical, inhibitive and facilitative. The first of these principles, the one most familiar to orthotists, is biomechanical. (62)

In order for a biomechanical foot system to be effective, it must hold the foot in an ideal or stable alignment allowing the motions of pronation and supination to occur within the normal limits of the gait cycle. With the foot held in this alignment, forward tibial progression is accompanied by elongation or stretch of the triceps surae muscle group facilitating a normal muscle pattern. (30)

This ideal foot alignment has been described as Feiss' Line, however it should be remembered that this is an ideal alignment and that humans do exhibit variations. Maintenance of this Feiss' Line or close to it establishes a point around which maximum foot function can occur. (Michaud, 1992)

In closed chain pronation when the foot is contacting the ground, the combination of ground reaction forces and the body weight line acts to plantarflex and adduct the talus, thereby dorsilflexing and abducting the forefoot at the mid tarsal joint. This movement of the talus down and is accompanied by internal rotation of the tibia and fibula and the eversion of the calcaneous.

The amount of pronation which will occur may be excessive when the triceps surae is spastic or contracted as is often the case in CP patients. Forward progression of the tibia is then accompanied by extreme dorsiflexion and abduction of the forefoot. In some cases the talus contacts the floor, bringing with it the internally rotating tibia and fibula. In this manner the dorsiflexion motion takes place at the mid tarsal joint instead of at the ankle joint. The foot remains pronated through the entire stance phase of gait. (80)

The reverse sequence of events is true if the foot is maintained in a supinated position. Ground reaction forces act to dorsiflex and abduct the talus. The forefoot plantar flexes and adducts while the tibia and fibula rotate externally and the calcaneous inverts.

A total foot system is created when the inhibitive and facilitative components are added to the biomechanical alignment. In 1960 Dr William Duncan published his now_classic article in the Journal of Bone and Joint Surgery entitled "Tonic Reflexes of the Foot: Their Orthopedic Significance in Normal Children and Children with CP." In this paper Duncan identified four areas of the foot and their tonic reflex action when stimulated. These areas are:

- 1 Toe grasp reflex or flexion and adduction of the toes. This reflex is stimulated at the ball of the foot near the base of the second and third toes.
- 2 Inversion reflex of the foot in response to stimulus near the head of the first metatarsal.
- 3 Eversion reflex of the foot in response to stimulation near the head of the fifth metatarsal.
- 4 Dorsiflexion reflex in response to simulation of the central portion of the plantar surface of the heel.

Duncan further stated "These are superficial reflexes in that they are elicited by stimulation of the skin alone. These reflexes normally disappear in an orderly sequence during the first years of life. If one or more reflexes fail to disappear, a reflex induced deformity may occur. The concept of this deformity is that if a reflex induced deformity may occur. The concept of this deformity is that if a reflex movement repeatedly occurs, the involved structures will eventually assume the distorted attitude. Such deformities are more common in patients with CP in whom disappearance of these reflexes is frequently delayed.

Not only can these reflexes lead to deformities but they also interfere with the timing and coordination of appropriate muscle function during standing and ambulation. The objective of inhibiting these tonic reflexes led to the design of the now familiar inhibitive foot plate. The cut-outs or recesses are designed to reduce the stimulus to the reflex areas. The addition to this footplate of the anatomical contours of the dynamic arches of the foot provides additional distribution of ground reaction forces resulting in a further reduction of stimulus at the reflex areas, the maintenance of biomechanical alignment and the facilitation of the muscles of the foot. The apices of the contours are designed to provide activation of the muscles much like tapping a muscle to make it respond. The footplate thus encourages balance reactions and provides key points for the patient to find the body midline (30, 62, Hylton, 1989, Cusick, 1990)

REFERENCES

Cupps B. Paper presented to the American Academy of Orthotists and Prosthetists, 1988.

Cusick B. Progressive Casting and Splinting for Lower Extremity Deformities in Children with Neuromotor Dysfunction. Therapy Skill Builders in Tucson, Arizona, 1990.

Duncan W R. Tonic reflexes of the foot. Journal of Bone and Joint Surgery 1960 <u>42A</u> (5).

Hylton N. The use of dynamic ankle foot orthoses and their impact on balance and upper body function. Neurology Report 1989 <u>13(3)</u>, 15-18.

Michaud, T C. Foot Orthoses and Other Forms of Conservative Foot Care, Williams and Wilkins, Baltimore, 1993.

ORTHOTIC MANAGEMENT OF DEFORMITY IN CEREBRAL PALSY

Chris Drake LBIST DipOTC

INTRODUCTION

The prevention of lower limb and hip deformity in Cerebral Palsy and its subsequent management is complex and difficult. The reasons that deformity occurs in the young cerebral palsy child is mainly a combination of poor postural positioning and the inability of the child to move away from the deforming effects of gravity (7, 63, Fulford and Brown, 1976,) Poor positioning combined with muscle imbalance means that the deforming effects upon the cerebral palsy child can become overwhelming and severe which can be extremely difficult to control and manage.

Therefore the aims of orthotic management should be targeted at attempting to overcome muscle imbalance and to correct postural alignment from the earliest age possible in an attempt to prevent soft tissue contracture and subsequent joint and skeletal deformities. The prescription of orthoses and their design and construction is very difficult due to the complexity of cerebral palsy and its presentation. The type of cerebral palsy, plays a major role in determining:

HOW are orthoses to be used?

WHEN are they to be introduced?

WHAT do we want them to achieve?

In general Cerebral palsy can be split into two classifications 1 Neurological 2 Anatomical.

NEUROLOGICAL		ANATOMICAL	
1	SPASTIC	1	HEMIPLEGIA
2	ATHETOID	2	DIPLEGIA
3	ATONIC	3	QUADRIPLEGIA
			(TOTAL BODY INVOLVEMENT)
4	DYSTONIC		
5	RIGIDITY		

The assessment and decision making process of determining orthotic prescription should be and must be carried out in a Multidisciplinary environment involving the Orthotist, Physiotherapist, Surgeon, Physician, Parents or Carers and the Child. The aims of orthotic management should meet with realistic goals. Careful examination and assessment (31) helps us to target our orthotic treatment to achieve the desired outcome. In many cases orthotic management will compromise with the desire to prevent deformity and allow dynamic movement. This conflict is difficult to balance as it may be the development of a contracture that prevents and hinders function or alternatively it may be the application of orthoses which prevents and inhibits function and movement. Therefore, the prescription of orthoses must fit in to the child's overall management plan if they are to become useful in the management of deformity.

In general, hemiplegic children will normally walk with or without aids at an early age but they may require Ankle Foot Orthoses (AFOs) or some type of Foot Orthoses (FOs) and will usually be of a dynamic type which facilitates movement but also prevents the development of deformity such as an Equinus foot and ankle. The beneficial effects of the application of AFOs has been demonstrated clinically as well as in gait laboratories (9, Meadows, 1984). They show that prevention of deformity can also lead to improved function.

When assessing for lower limb orthotic management the whole of the lower limb must be looked at as a complete unit instead of the foot and ankle alone. For example when a contracture of gastrocnemius is present it can not only produce an Equinus foot and ankle but also a knee flexion deformity this is due to it being a two joint muscle. In this case the fitting of an AFO to correct the equinus only, will inevitably lead to an increase in knee flexion deformity and possible a compensatory hip flexion deformity. The knee flexion deformity must also be managed if the deformity is to be controlled.

Spastic diplegia can present asymmetrically in varying degrees of severity. It can be from very mild, when the child will become a good functional walker without the need of a great deal of orthotic management, to very severe, with no prospect of walking at all. In general the majority will walk at a later age and will most probably benefit from some kind of walker such as rollators or posterior walkers. The use of AFOs (Butler and Nene, 1991) and Dynamic Ankle Foot Orthoses (DAFOs) (20, 30, 62) have been clinically noted to have beneficial effects both on the management of deformity and for dynamic function.

Knee orthoses for the use in walking have been little used due to the impact they have on the restriction of gait with the exception of the Swedish hyperextension orthosis (Cusick, 1990) which allows free knee flexion but controls knee hyperextension.

In the past the application of Knee Ankle Foot Orthoses (KAFOs) were used (66) but they are relatively unused today due to detrimental impact on function and efficiency of walking,

Few Quadriplegic children, who are amongst the most severely affected, will become functional walkers (Fixsen, 1992) and are consequently the most at risk from developing deformities of the lower limbs, most commonly at the hip (Scrutton, 1978, Vidal et al, 1985). In these children postural positioning of the pelvis, hips and knees becomes more important than looking solely at the position of the foot and ankle, although foot position cannot be ignored due to its importance in stabilising the body in sitting and standing. With these children it is the proximal problems that demand the most orthotic attention. They tend to present with severe spasticity and suffer more from the effects of asymmetrical muscle tone the effects of gravity and poor positioning.

The development of hip subluxation in this group is widespread and can be catastrophic (7, Beals, 1969, Vidal et al, 1985, Scrutton, 1989). Hip deformity seems to be associated with scoliosis and Pelvic obliquity (Cooke et al, 1989), although it is still difficult to confirm whether it is spinal or hip/pelvic deformity which occurs first. It still seems to be in dispute if indeed they are related at all. It is obvious, however, clinically that a large number of cerebral palsy children with wind swept hip deformity (adduction/internal rotation on one hip, abduction/external rotation of the opposite hip) (Fulford and Brown, 1976) also present with scoliosis. It is this complexity of spasticity and lower limb deformity that makes this group of children the most difficult to treat and manage with orthoses.

The use of hip abduction orthoses (17, 40, Thompson, 1957, Nakamura and Ohamu, 1980, Bower, 1990) have been described for the management of hip deformity. There is, however, currently a lack of published data that supports claims that the use of hip abduction orthoses can influence the development of the hip joint and decrease the likelihood of bony deformity. It has been noted that clinically that they seem to assist in the maintenance of muscle length, range of motion of the hip, an improvement in posture and also assist in the management of tone (6, Nakammura and Ohamu, 1980, Hoffer, 1985).

Night time positioning of the severely affected quadriplegic child can be useful as it may be a time when the child relaxes and good orthotic management can take advantage of long periods of time to gently stretch without causing stress to the child. It seems obvious that we should take advantage of this time to apply orthoses when the sleeping hours may be the longest in a young child's life. The application of a sustained low grade stretch seems to be very beneficial in assisting with the control and management of contractures (Tardieu et al, 1988).

In many circumstances it is not the use of orthoses alone that brings about any modification of deformity but a combination of surgical intervention, targeted physiotherapy as well as the application of orthotic principles that bring about change.

It also goes without saying that it requires hard work, dedication and resilience from parents and lets not forget The CHILDREN!

FOOT ORTHOSES (FOs)

The use of foot orthoses in the management of the cerebral palsy child is widespread and the devices range from simple supportive boots with adaptations (60, 66, American Academy of Orthopaedic Surgeons 1975) to complex multi material biomechanical functional foot orthoses (30, 34, 58, Philps, 1990). All of these orthoses report to act upon stabilizing the hind and mid foot during the stance phase thus preventing foot and ankle deformity from occurring during weight bearing.

The use of Heel cups of UCBLs (University of California Biomechanics Laboratory Orthosis), Supramalleolar Orthoses (SMOs), (62, Cusick, 1990) and Dynamic Foot Orthoses (DFOs) (5), can control and stabilize the mid and hind foot only during the

stance of gait. They offer relatively little control during the swing phase of gait or when non weight bearing. This means they have limited use in the management of the more profoundly handicapped quadriplegic child who may have little or no weight bearing.

Foot orthoses can be highly beneficial in improving dynamic function and when weight bearing exert correctional forces which can control Valgus or Varus deformities by stabilising the hind, mid and forefoot. The literature reviewed contained many variations on a theme regarding the design of the foot orthotics but they had many features in common

- Hindfoot stability (close molding around heel)
- Rear foot posting (valgus or varus, extrinsic or intrinsic)
- Stabilizing effect mid foot (medial & lateral extensions. Valgus arch supported).
- Toe and Metatarsal support (Tone management?)
- Casts usually taken in sub talar neutral.
- Contoured sole plates to assist in foot stabilisation (Reduce Tone?)
- Manufactured from: Semi flexible material (polypropylene, polythene etc) Rigid material (acrylics)

ANKLE FOOT ORTHOSES (AFOs)

As with foot orthoses, Ankle Foot Orthoses (AFOs) often use the same biomechanical principles of achieving sub talar neutral foot position within the orthoses while weight bearing. They can also control the foot and ankle during swing phase and non weight bearing (3, 9, 58, 62, 85, Meadows, 1984). This makes them more appropriate for a wider spectrum of children with differing levels of spasticity and functional ability they can offer control over many problems associated with cerebral palsy. Such as:

- Equino-varus/valgus
- Equinus without varus or valgus
- Valgus/varus Hindfoot
- Hyperextension (Genu-recurvatum) of the knee
- Knee Flexion contracture

- Crouch gait (Hip/knee flexion combined with Internal Rotation and Hyper dorsi flexion at the ankle)

The presentation of the child's clinical requirements will influence AFO design but all will have basic key design elements incorporated.

- Maintenance of subtalar neutral foot position.
- Full foot piece to prevent plantar flexion/dorseflexion
- Medial and lateral trim lines which control hindfoot alignment.

- Forefoot extensions to control pronation/supination/abduction and adduction of the mid and forefoot

- Heel retaining strap to prevent heel lift and anterior slip
- Toe lift and metatarsal support to assist with tone management?

At the initial stages of AFO design the fixed AFO was used to control plantar flexion and valgus/varus stresses but this also limited dorsi flexion which in the case of the dynamic toe walking hemiplegic child was inhibiting their habilitation.

Due to this problem of holding back the child's gross motor skills by preventing active dorsi flexion, the hinged AFO was gradually developed and now many designs of joints are available. The hinged AFO and its use in the management of an equinus gait has been noted to have a beneficial effect on the child's walking and gross motor skills most notably with hemiplegic children. (41, Drake & Borsteinas, 1993). The design of most hinged AFOs utilizes a back stop to prevent unwanted plantar flexion.

Hypertension is a deformity that is usually associated with an equinus deformity, common in hemiplegic children (Meadows, 1984, 1985). This deformity can mask the true problem of an equinus gait. If there is a problem of an equinus deformity the application an AFO should be considered hinged or fixed depending on the individual child. The ankle angle can be set dorsiflexed to overcome hyperextension by positioning the tibia in a forward angle inducing a flexion moment at the knee so as to resist the hyperextension. Rosenthal et al (59) stated that an optimum angle of 5 degrees of dorsi-flexion at the ankle could help in the prevention of between 5 degrees to 20 degrees of hyperextension. Pitching the tibia forward or fine tuning the AFO by fitting heel raises to the base of the AFO or footwear (Butler & Nene, 1991), brings the ground reaction behind the knee joint therefore inducing a knee flexion moment and preventing hyperextionsion.

The hinged AFO seems to be very beneficial for a large number of hemiplegic children with dynamic toe walking gait, but great care has to be taken when attempting to fit the hinged AFO to Diplegic children.

Knee flexion deformity induced by hyper activity of the hamstrings, combined with hip flexion deformity, internal rotation, hyper dorsi-flexion and weakness in the quadriceps manifests as crouch gait. This condition is also exacerbated by surgical over lengthening of Tendo-Achilles and leaving Hip and Knee deformities uncorrectd.

In a diplegic child with crouch gait the application of a hinged AFO would be a contraindication as the unlimited dorsi flexion allowed by the free hinge would allow the deformity to increase. In these cases the use of a Ground Reaction Orthosis (GRO or Saltiel orthosis or talus control AFO) can be useful to assist in the prevention of crouch gait (26).

The success of the GRO is very dependable on pre-Orthotic assessment. If there are any hamstring contractures they should be surgically corrected. Out of the eleven subjects who were in the study by Harrington, Lin & Gage (26), eight of the children underwent hamstring release before the fitting of the GRO.

As the name indicates the GRO relies on body weight to induce a turning moment that counteracts the knee flexion component and therefore over activity in the hamstrings on walking will impact heavily on the effect the orthosis has on reducing knee flexion. Also any fixed contractures of hips and knees or internal/external rotation at the hips will also reduce the effectiveness of the GRO.

The use of AFOs for night resting has been used for many years but there is little information published regarding their effect upon decreasing deformity. The problem with using only an AFO to prevent a plantar flexion deformity occurring is they can only help in the reduction of the contracture when the soleus muscle is affected because it is a single joint muscle. If gastrocnemius is contracted then the child only has to flex their knee to reduce the effect of the stretch.

Night AFOs do have a place in the management of deformity and their role may well become more prominent following the reports from Tardieu (1988). He states in his study that if the soleus muscle is stretched above a minimal threshold of six hours out of a twenty four, there is likely to be no progression of a contracture.

The six hour threshold was found from twenty four hour data recordings of the activity in the soleus muscle in normal children. Although this report was specific to the soleus muscle it is probable that it may have significant implications in the use of orthoses in preventing deformity in other muscles and joints.

KNEE ANKLE FOOT ORTHOSES (KAFOs) AND KNEE ORTHOSES (KOs)

The use of Knee Ankle Foot Orthoses (KAFOs) and Knee Orthoses (KOs) was common in the past. Stamp (66) describes KAFOs for the treatment of knee contractures with Dial locks to gradually reduce flexion followed by fitting of a flexion joint when correction had been achieved. He also advocated the use of old orthoses for night use to assist with the prevention of contractures.

Knee flexion deformity is the most common problem associated with the spastic diplegic child and also seems to be the most difficult to treat. The application of KOs (1), or long leg plasters, (Cusick, 1990), for day or night use seems to have a beneficial effect on reducing hamstring contracture.

Anderson used soft thermoplastic foam KOs (no feet included) which were applied at night during the hours of sleep (length of duration not reported) to a group of children who presented with severe knee flexion deformities. They were used over the 10 month period and after follow up of the 25 children she reported an average decrease in knee flexion contracture of 24 degrees.

Cusick reported a single case study of a severe spastic diplegic child who presented with popliteal angles of 64 degrees on the right and 60 degrees on theleft. She applied long leg casts which included the feet and ankles. The casting in the case report lasted for 45 days with 3 changes of casts on the right leg and 4 changes of cast on left. At the end of the casting she reported a reduction in the popliteal angle on the right from 64 degrees to 8 degrees. The left had decreased from 60 degrees to 16 degrees. Following the casting the child was fitted with GROs. The hip flexion deformity that was present before treatment still remained and this highlights the problems of associated deformity which appears when a deformity exists elsewhere.

The Swedish knee cage, (American Academy of Orthopaedic Surgeons, 1975, Cusick, 1990) can be used for the prevention of mild hyperextension while still maintaining dynamic function. Other knee orthoses have been used (Scaramuzza, 1967) when there is weakness in the knee extensors. The use of full leg resting or night KOs (1)

with or without Abduction bar are used to help passive night time positioning and also Post Operatively.

The use of twister orthoses is noted to have a desirable effect on internal rotation, (66) but care has to be taken not to use an excessive external force so as not to create secondary rotary deformities at the knee, tibia or hip. An average period of use without a rest from a twister orthosis would be approximately 6 months to avoid this occurring.

In general the use of KAFOs for the cerebral palsy child is relegated to the past due to the detrimental effects they have on function and efficiency of walking.

HIP ABDUCTION SPINAL ORTHOSES (HASOs)

The management of the hip and its position in cerebral palsy is complex and difficult because the majority of the children will have four limb involvement and may also be hypertonic, thus the problem of severe spasticity must be overcome as well as deformity.

There are many aims of management in the quadriplegic child, not just to position the hip, but also to control pelvic alignment, maintain control over flexion and extension and provide a good base of support for sitting and attention.

The use of Hip Abduction orthoses has been in use for many years (23, 40, 75) but little published data on its effect on the CP hip. Nakamura and Ohamu (1980), designed an orthosis which was used at night on a group of seven cerebral palsy children. All the children either stood with orthoses or walked during the day. The Hip brace was applied at night and they were followed up after 16 months later. No radiological improvements were shown. It was reported however that the range of movement at hip was improved.

The majority of the children who suffer most from the effects of hip deformity have four limb involvement with the added problems of increased spasticity and spinal deformities (Cooke et al, 1989). They also have little volitional movement and are susceptible to the deforming effects of poor positioning (63, Fulford and Brown, 1976). Therefore the design of HASO must have certain standard features:

- Fit close to body contours
- Attempt to control pelvic obliquity
- Control range of hip abduction/adduction
- Control range of hip extension/flexion
- Enable sitting/standing and lying
- Be lightweight
- Easy to doff and don

Bower (1990) and Drake and Boyd (17) describe a hip abduction orthosis which consists of a complete spinal component (Bi-valved) with an anterior and posterior polypropylene shell. Attached to the shell are metal side members with hip joints which lock in 90 degrees of flexion and 180 degrees of extension with free movement in-between. Thigh cuffs are added and extend to cover the femoral condyles. A rigid adjustable abduction bar is positioned in-between the thigh cuffs to control the amount of abduction and adduction.

The close fit of the orthosis allows precise control over the hip joint even with a child who presents with severe spasticity. The angles of abduction are set between 25 degrees and 35 degrees (50 degrees to 70 degrees included). The orthosis is used not only to assist in hip alignment but also used as a positioning device to help with the day-to-day management of the child at home and in school. Severe spasticity can be managed within the orthosis and it is worn for a minimum of eight hours out of twentyfour in line with the report by Tardieu et al, (1988).

Clinically there have been immediate benefits in the posture and function of children when wearing the orthosis and high compliance and tolerance has been reported. Experience gained over the last five years in the Newcomen Centre at Guys Hospital has helped us to be more selective in the appropriate prescription, which is of vital importance.

Children who have scoliosis as well as hip problems and postural instability may require the spinal component to be used in an environment where the hip joints and thigh components are not required. The orthosis now has detachable hip and thigh components. This allows continual control of the scoliosis but permits more active movements at the hips for specific times in the child's programme which require more freedom.

Great care and attention has to be taken in the correct assessment, prescription, casting and fitting. It should be carried out by a **Multidisciplinary Team** including Carers, Orthopaedic Surgeon, Paediatric physiotherapist and Orthotist. Currently research is being undertaken to see if the objective data correlates to the positive subjective data over the last five years.

CONCLUSION

The use of Orthoses in the management of Cerebral Palsy is complex and difficult. It has many conflicts of approach with regards to Functional and Passive positioning.

Treatment should be targeted as the whole body function of the child and take into account the environment which surrounds them. The desire to prevent or correct deformity must be balanced with the desire to facilitate movement and achieve dynamic control.

There are many types of orthoses used by many practitioners all of whom tend to have strong views on the correct approach of management but at the end of the line there are the children and their families who live with their orthoses and success cannot be gauged on prevention of deformity alone.

A Hip Orthoses applied correctly may have a dramatic effect on the child's postural ability, daily management and functional ability but it may not change the eventual outcome a progressive hip disorder, is it success or failure? Alternatively the fitting of bilateral AFOs to a diplegic child which corrects the majority of deformity but reduces their ability of function dynamically, is it success or failure?

The prescription of Orthoses must be carried out in a **Multidisciplinary Team** environment if the goals of management are met and if the use of Orthotics is to be maximised to its full potential.

REFERENCES

American Academy of Orthopaedic Surgeons. The Atlas of Orthotics. The C V Mosby Company, St Louis, 1975.

Beals R K. Developmental changes in the femur and acetabulum in spastic paraplegia and diplegia. Dev Med Neurol 1969 <u>11</u>, 303-313.

Bower E. Hip Abduction and spinal orthosis in cerebral palsy. Physiotherapy 1990 <u>76</u> (10), 658-659.

Butler P B, Nene A V. The biomechanics of fixed ankle foot orthoses and their potential in the management of cerebral palsied children. Physiotherapy 1991 <u>77 (2)</u>, 81-88.

Cooke P H et al. Dislocation of the hip in cerebral palsy: natural history and predictability. J Bone and Joint Surg 1989 <u>71-B</u>, 441-446.

Cusick B D. Progressive casting and splinting for lower extremity deformities in children with neuromotor dysfunction. Therapy Skill Builders, Arizona, 1990.

Drake C J, Borsteinas R. Results from a comparison study of the effectiveness of the fixed ankle for foot orthosis versus the hinged ankle foot orthosis. Newsletter ISPO-UKNMS 1993 <u>Summer</u>, 23-24.

Fixsen J A. Orthopaedic surgery for cerebral palsy. Hospital update (UK) 1992 November, 803-805.

Fulford G E, Brown J K. Position as a cause of deformity in children with cerebral palsy. Dev Med Child Neurol 1976 <u>18</u>.

Hoffer M. Management of the hip in cerebral palsy. J Bone and Joint Surg 1985 <u>67-</u> <u>A</u>, 1229-1235.

Meadows C B. The influence of polypropylene ankle-foot orthoses on the gait of cerebral palsy children. PhD Thesis 1984, University of Strathclyde.

Nakamura T, Ohamu M. Hip abduction splint for use at night for scissor leg of cerebral palsy patients. J Orthot and Prosthet 1980 <u>34</u>, 13-18.

Philps J W. The functional foot orthosis. Churchill Livingston Press 1990.

Scrutton D. Developmental deformity and the profoundly retarded child. In Care of the Handicapped Children, 1978, Spastics Int Publications, Heinemann, London.

Scrutton D. The early management of hips in cerebral palsy. Dev Med Child Neurol 1989 <u>31</u>, 108-116.

Tardieu C, Lespargot A, Tabary C, Bret M D. For how long must the soleus muscle be stretched each day to prevent contracture? Dev Med and Child Neurol 1988 <u>30</u>, 3-10.

Vidal J, Deguillaume P et Vidal M. The anatomy of the dysplastic hip in cerebral palsy related to prognosis and treatment. Int Orth (SICOT) 1985 <u>9</u>, 105-110.

ORTHOTIC MANAGEMENT OF THE CEREBRAL PALSY CHILD

Robert S Lin CPO

The analysis of normal human locomotion is an exceedingly complex task. Yet before one can begin to assess different gait pathologies, the understanding of normal gait is a prerequisite.

There are four primary requirements for normal gait. The first of these is stability in stance, which relates to the phase of gait when the supporting extremity is the "single" or primary support of the body mass. The function of the supporting extremity during stance phase may be profoundly affected by the external support provided by an orthotic device. The center of mass should be aligned over the base of support if at all possible to reduce or eliminate angular moments acting upon the lower limb joints. Ideally, the muscles balance the ground reaction forces or inertial forces, resulting in joint stability.

Stance phase can be further broken down into three rockers. The degree in which any orthosis impacts on the smooth translation through these rockers will affect all normal parameters of gait, ie single support time, step/stride length.

The period of first rocker begins at initial loading (previously referred to as heel strike) and continue until foot flat during which the ankle joint typically experiences a plantarflexion moment. If, however, first rocker is impeded by the orthotic design (eg solid ankle AFO), the heel will act as a fulcrum and can result in an unstable situation. An orthotic design which allows controlled plantarflexion will enable the ground reaction force to be absorbed and effect a smooth first rocker. A well-designed Posterior Leaf Spring (PLS) AFO (or dorsiflexion assist) or an articulating (hinged) AFO with visco-elastic plantarflexion stop will both mimic the normal plantarflexion movement of the ankle. A SACH-type of shoe modification is also capable to a degree of simulating plantarflexion, even when a solid ankle design is used.

The period of second rocker relates to mid-stance and is concerned with the advancement of the tibia over the ankle foot complex from a position of approximately 10° plantarflexion to a 10° dorsiflexed attitude. Free or controlled sagittal plane ankle motion should be considered if the patient has the stability to accommodate this degree of mobility. An articulating AFO with free dorsiflexion or an elastic posterior check strap will enable forward progression of the body mass over the supporting limb, as will an optimally designed PLS-AFO. It should be noted that if a PLS-AFO is too stiff for the specific patient and resists tibial advancement, the ground reaction forces will advance in front of the knee and cause a hyperextension moment at mid-stance, similar to the effect of a tight heel cord. If an orthotic design that does not allow sagittal plane motion is used a rocker bottom sole should be considered to facilitate progression through stance, thus maximizing the quality of the 2nd rocker.

The period of the third rocker is from heel-off to push-off. If an orthosis restricts plantarflexion, it will impede third rocker and may affect other parameters of gait, leading to a shortened step length (due to early heel rise), a slower cadence and ineffective load transfer to the contralateral limb.

The second requirement of normal gait, swing phase clearance, can be very effectively assisted by an orthotic design which crosses the ankle joint, thus resisting plantarflexion. It should be noted that the PLS-AFO should be preset at a 5° dorsiflexed attitude to account for normal deflection (due to the weight of the foot and shoe) during mid-swing.

While orthoses may have a profound effect upon the function of the ankle foot complex during swing phase, they cannot substitute for inadequate knee and/or hip flexion. The presence of the latter abnormality will result in inadequate swing phase clearance.

The third requirement for normal gait is swing phase pre-positioning. If one assumes the ankle-foot complex is optimally managed and any knee deformity has been addressed, then the remaining critical element is the hip joint function. It is welldocumented that an imbalance of the hip musclature as well as spasticity, can result in adductor tightness, rotary deformities and gait deviations such as the Trendelenburg deviation.

The CP hip orthosis can be used diagnostically or definitively to address the typical adducted, scissoring or windswept gait encountered in the spastic diplegic population. This design incorporates two thermoplastic femoral cuffs with key supracondylar compression and a narrow medio-lateral design. The mechanical hip joints are such that they allow free flexion, extension and abduction, but can be adjusted relative to the adduction stop. The hip orthosis may be used to determine the appropriate form of treatment, whether orthotic or other. It may also be used to augment therapy sessions and for the pre-operative assessment of function.

Several considerations will influence the choice of trimlines for any thermoplastic ankle foot orthosis. Transverse plane deformities are prevalent in the CP child and may be a result of abnormal alignment or control of the mid-tarsal joint. The presence of a midfoot break or abduction/adduction angulation will affect the manner in which the foot will accept weight during stance. Careful attention must be given to the control of the foot as a supporting structure so that ground reaction forces can be approximately aligned in front of the knee if desirable, as in the case of the Floor Reaction AFO. In addition, if the trimlines encompass the shafts of the first and fifth metatarsals, this will maximize control of both the mid-tarsal and subtalar joints.

Irrespective of the design of thermoplastic AFO employed, the integrity of the toe plate will affect the heel-toe lever arm and the ability to achieve a normal metatarsophalangeal toe break. The restriction of this motion can also affect step length (the fourth and final requirement of normal gait). The plate should, therefore, be thinned or even cut back unless the presence of severe toe flexor spasticity takes precedence.

In summary, all orthotic designs must make sense! The presence of key 3-point force systems and the concept of total contact or strategic lack of contact, should be carefully considered in the design of any orthotic system.

The greatest advance in the field of orthotics over the past decade has not been in the introduction of new materials, designs, or components, but rather the enhanced

understanding of the biomechanical implications of orthotic management on the extremity involved ... in other words, kinesiology.

THE USE OF ORTHOTICS TO IMPROVE THE DYNAMIC EFFICIENCY OF GAIT

Terry J Supan CPO

INTRODUCTION

The role of orthoses in the care and treatment of individuals having cerebral palsy is no different than that for any other condition: to protect a part, to prevent deformity and/or to improve function. With this population, there is a limited need for protection and a questionable ability to prevent deformity. The primary goal in cerebral palsy is to help the individual walk better.

By understanding normal gait and the pathological and pathomechanical (Fish and Nielsen, 1993) patterns of cerebral palsy, custom made orthoses for ambulation can be utilized to their best biomechanical advantage. Gage's five principals of efficient gait (Gage, 1991) must be kept in mind when performing a patient evaluation. Is there instability during stance? Is there insufficient foot clearance during swing? Is there a problem with pre-positioning of the foot at the end of swing? Is there a difference in the stride length or is it inadequate? Is there increased energy consumption? If the answer to any of these questions is yes, then the goal of any orthotic or surgical intervention should be to create a more efficient gait.

After a patient's gait pattern has been evaluated, orthotic recommendations are made based on the biomechanical ability of a given device to improve the gait. Recent design changes have greatly improved our ability to have a positive impact and as new components become available, orthotic design will continue to alter prescription recommendations.

FOOT ORTHOSES

The goal of University of California at Berkley Laboratory (UCBL) foot orthosis is to stabilize the subtalar joint without restricting ankle motion (Carlson and Berglund, 1979). Foot motion may also be controlled depending on the pliability of the foot and how distal the trim line is. Trim lines may vary widely. Some practitioners will bring the sides to a supramalleolar level and call it a SMO. The important consideration is that there must be good hind foot correction and control to be able to maintain proper alignment through the remainder of the foot. Except in the very young, too much pressure under the navicular area will only cause discomfort and skin irritation.

ANKLE FOOT ORTHOSES

The pre-made ankle foot orthosis is used primarily for swing phase control. It provides dorsiflexion assist and reduces foot slap. Occasionally a pre-made device can aid in evaluating the orthotic impact on an individual before prescribing a custom made orthosis. Seldom are they of adequate fit to be used for extended periods of time.

The custom made solid ankle foot orthosis can be made in different styles (Glancy and Lindseth, 1972). Specific motions can be controlled around the foot depending on how the orthosis is designed. It increases stability of both the ankle and the subtalar

joint and will affect knee control as a result of the alignment of the tibia. A hyperextended knee secondary to a plantar flexed foot can be very positively influenced by preventing the offending plantar flexion (9, 59). As with the UCBL, hind foot control is the key to affecting proper posture in the remainder of the foot. This AFO will provide dorsiflexion assist during swing phase.

The custom made dorsiflexion assist orthosis is trimmed more posterior to the malleoli than the solid ankle AFO. This allows more flexibility and will have less effect on the knee during third rocker. It will provide limited stability at the ankle but will not control subtalar motion unless it is specifically designed with a varus/valgus control trim. Historically the metal spring loaded dorsiflexion assist AFO was utilized to aid with clearance in swing phase. It is still seen where physiology and biomechanics have been lost sight of. The stretch reflex, pathologic in the individual having cerebral palsy is only worsened by the addition of a spring under extreme tension. Plantar flexion inhibition is the function that is desirable, not augmented dorsiflexion.

The spiral orthosis (Lehneis, 1974) and the thermoplastic elastomer orthosis (Sutton, 1990) function differently than the standard polypropylene orthoses by allowing a more gradual plantar flexion. The first is designed to absorb and utilize the torques which are in normal walking. It has limited stability at the ankle and the subtalar joint. The second provides a dorsiflexion assist and allows a limited plantar flexion resistance because of the elastomer material. These functions are inherent to their materials and design.

The custom made articulated orthosis has undergone the most changes in recent years (3, 34, 80) Modifications have allowed the orthotist to concentrate on preventing unwanted ankle motion while allowing more normal kinematics. Improved gait is beneficial in the child with cerebral palsy during the development stages because the orthosis will interfere less with neuromuscular pattern formation. This orthosis can be designed to stabilize the subtalar joint, while preventing unwanted ankle motion, it may assist in preferable ankle motions; and it will give limited control of knee motion. All of these functions are the result of the way that the orthosis is designed and the type of modifications that are made.

The articulated ankle foot orthosis can be set up to range from a free to a rigid ankle. This type of versatility can allow one device to function as clinical needs very from a post surgical state to complete convalesence (62). There can be excellent subtalar control and still allow free ankle motion. With an adjustable posterior stop the orthotist can better evaluate and maintain the desired knee posture during stance phase by controlling the effect of the orthosis on the second rocker. Finally, a dorsiflexion assist spring can be incorporated into the design. Care must be taken, as was alluded to earlier, not to introduce an unwanted spastic response to a potential stretch caused by the spring.

The articulated AFO best takes into consideration control of unwanted dynamics in the rockers of the foot and ankle while facilitating the desirable rockers. With a 90 degree plantar flexion stop there is a swing phase control and pre-positioning for stance. During stance there is subtalar control for stability and facilitated second rocker motion. A flexible toe plate will allow forefoot dorsiflexion of the third rocker while

limitation in the articulation prevents excessive plantar flexion in early swing. The net effect is a smoother roll over.

FLOOR REACTION ANKLE FOOT ORTHOSES

Two types of floor reaction orthoses, solid and articulated are frequently utilized. Originally designed to provide the amount of force needed to prevent unwanted motion and be able to provide more stability to the knee for a patient having post polio paralysis (Salteil, 1969) the floor reaction orthosis has been modified over the years (26). The idea behind this orthosis is that while it stabilizes ankle and subtalar motion, it eliminates knee flexion because of the floor reaction forces onto the knee and it may provide dorsiflexion assist.

The custom made articulated floor reaction orthosis is a more recent example of the orthosis trying to better facilitate normal motion (Gage, 1991) The concept is to minimize orthotic involvement at the first and third rockers while assisting a knee extension moment at second rocker. This orthosis will stabilize the ankle and subtalar joint, allow first rocker plantar flexion and prevent second rocker dorsiflexion so that the tibia cannot progress anteriorly over the top of the talus. The result is to prevent knee flexion in stance. Free dorsiflexion of the toes at third rocker is dependent on how the orthosis is trimmed in the metatarsal area. Rubber bands may be added for dorsiflexion assist. These bands add control of first rocker and will slow plantar flexion in the minimally involved patient during loading response.

TONE INHIBITION

Along with the many design alterations (articulated versus non articulated, spiral versus conventional, location of trim lines and choice of materials) AFOs have been designed to have tone reducing capabilities. They have been a natural out growth of inhibitive casting (19, 25, 29, 68, 69, 78). Modifications of the trim lines and the foot plates are examples of these modifications. The principles behind tone inhibition are to increase the pressure along the metatarsal arch and the peroneal arch, provide relief under the metatarsal heads, provide a dorsiflexion moment to the toes, provide control of the proximal portion of the calcaneous in the areas medial and lateral to the Achilles tendon, relief on the plantar surface of the calcaneous and to provide support in the sustentacular tali area of the calcaneous (30, 62). As with the casting, controversy exists about their effectiveness. There are many articles (20, 27, 44, 72, 82) in the recent literature claiming far reaching benefits from the tone reducing orthoses. Some claim short term alterations in tone, others go so far as to suggest that there may be a lasting change in primitive reflexes. Unfortunately none of these studies are controlled or randomized. Most are single observer reports. Another problem with interpreting reports in the literature have to do with tone reducing orthoses is the inconsistency in the material used, the basic designs, the trim lines and the areas of relief. Evidence is still lacking for the role of so called tone reducing orthoses (Bleck, 1990).

REPLACEMENT AND ADJUSTMENT

Within the field of Orthotics there are currently no known published studies that have addressed the nature and frequency of adjustments and subsequent replacement of orthoses prescribed for the lower limb. At Southern Illinois University School of Medicine a retrospective study was performed to provide information to determine the duration a thermoplastic AFO will fit appropriately and when subsequent adjustment and replacement was indicated. Clinical records of children fitted with thermoplastic AFOs from 1982 to 1992 were reviewed. Neuromusculoskeletal functional status appears to determine the AFO design prescribed and its replacement. Adjustments to thermoplastic AFOs primarily involve heat flaring to alter plastic contours and provide sufficient space for donning, doffing and pressure relief over regions of bony prominence.

Sixty one percent of all of these patients were diagnosed with varying types of cerebral palsy. Spastic diplegia made up the largest group with forty-five percent. Spastic hemiplegia and total body involvement had similar amounts with twenty-six and twenty-two respectively.

The most commonly used AFOs used were solid ankle designs and articulated. Since the study was completed the articulated floor reaction has become more widely used with the diplegia patient. There was a twenty-five percent increase in the amount of heat flare adjustments of the articulated AFOs but surprisingly only a five percent increase at the ankle region itself.

The replacement of the orthosis was necessary for boys at seventeen months and twelve months for girls for the whole patient population. There was a slight decrease in longevity for the children with cerebral palsy.

RECOMMENDATION

Our experience has shown that the orthotic design should be based on the biomechanical needs of the patient. Children with total body involvement have used solid ankle AFOs to maintain ankle position and KAFOs to help prevent ankle and knee deformities. The child with spastic hemiplegia needs an AFO with the ankle held in dorsiflexion and a proximal trimline closer to the knee center to better control hyperextension of the knee. The ankle should be allowed to dorsiflex to provide a dynamic stretch on the gastroscoleous. The child with diplegia normally starts with solid ankle AFOs and progresses to either articulated AFOs or articulated floor reaction AFOs, the latter if knee control during stance phase is necessary. Base the orthotic design on the need to meet Gage's principals of efficient gait.

REFERENCES

Bleck, E E. Current concepts: Management of the lower extremities in children who have cerebral palsy. IBJS 1990 <u>72-A</u>,140-144.

Carlson M J, Berglund G. An effective orthotic design for controlling the unstable subtalar joint. Orthot Prosthet 1979 <u>33</u>, 39.

Fish D J, Nielsen J P. Clinical assessment of human gait. J Prosthet Orthot 1993 <u>5(2)</u>, 39-48.

Gage J. Gait Analysis in Cerebral Palsy. MacKeith Press, London, 1991.

Glancy J, Lindseth R E. The polypropylene solid-ankle orthosis. Orthot Prosthet 1972 <u>26</u>, 14-26.

Lehneis H R. Plastic spiral foot-ankle orthoses. Orthot Prosthet 1974 28, 3-13.

Salteil J. A one-piece laminated knee locking short leg brace. Orthot Prosthet 1969 23, 68-75.

Sutton R. Thermoplastic elastomer (TPE): the TPE ankle-foot orthosis and the TPE biomechanical foot orthosis. J Prosthet Orthot 1990 2(2), 164-172.

GAIT RELATED ORTHOTIC PRESCRIPTION CRITERIA FOR CHILDREN WITH CEREBRAL PALSY

Don Weber BSc CO

INTRODUCTION

"Normal gait is dependent upon a coordinated series of events which moves the body through space with the minimal energy expenditure. Any neuromuscular abnormality which interferes with this pattern and results in a loss of coordination will give rise to increased energy requirements" (Mann, 1983). In addition to energy efficiency the gait pattern must also maintain stability in terms of balance (centre of gravity over base of support) and in terms of joint stability. Children with cerebral palsy have inefficient gait patterns due to motor dysfunction, joint contractures and abnormal muscle phasic activity. Common abnormal gait patterns seen in this population include, decreased velocity, decreased stride length, prolonged stance phase, toe strike before heelstrike, inefficiency of movement with faulty weight transfer and limb circumduction, a negative base of gait in diplegics with adductor spasticity and crouch gait (Mann, 1983).

The objective of this paper is to investigate the improvement of efficiency and stability during the ambulation of children with cerebral palsy when using lower extremity orthoses. The review of different types of orthoses is limited to custom plastic orthoses which are lighter, more cosmetic and offer significantly improved control and function as compared to conventional metal and leather devices.

Categories of Orthoses

The effect on gait of different orthoses are evaluated under different categories from minimal control to maximal control of the lower extremity as follows:

Shoes/Shoe Modifications Foot Orthoses/Heel Foot Orthoses/Supramalleolar Ankle Foot Orthoses Articulated (Single - Axis) Ankle Foot Orthoses Rigid Ankle foot Orthoses Tone Reducing/Inhibiting Ankle Foot Orthoses Hip Knee Ankle Foot Orthoses

Each of the above categories of orthoses will be investigated under the following headings:

Indications

- Which pathological joint motions seen during standing and ambulation indicate the use of this category of orthosis.
- Which functional and developmental factors suggest the use of this design of orthosis

Orthoses control the lower extremity in two ways during ambulation. The type of biomechanical control will be distinguished where relevant.

<u>Three point pressure control</u> (3PP control) stabilizes the joint in all phases of gait and does not depend on the inclination of the walking surface, the position of the foot at foot contact and the type and function of the shoe used with the orthosis.

The ground reaction force control (GRF control) uses contact with the ground to control the lower extremity and therefore is only effective during stance phase. Using GRF control the position of the joint during foot flat is dependent on the inclination of the walking surface. The position of the foot at weight acceptance dictates which side of the joint the line of action will fall and therefore if the pathological motion is increased or decreased. The width and rigidity of the shoe and the ability of the shoe to lock onto the orthosis produces the effective lever arm of the GRF and the position of the lower extremity during stance respectively.

Contraindications

- Which factors contraindicate the use of this category of orthosis.

Type of Orthosis

Each type of orthosis in the category will be discussed under the following headings:

<u>Design Features</u> - including: materials, structure, components, trimlines, static and dynamic features etc.

<u>Clinical Experience</u> - A review of clinical experiences involving these types of orthoses.

<u>Scientific Research</u> - A review of scientific studies (including comparative and descriptive studies) investigating the use of this type of orthosis. Each of the studies is described in some detail in terms of numbers, types and ages of the children with cerebral palsy, the types of orthoses, the length of time the orthoses were used, the outcome measures and the results. The practitioner can then decide if the relevance and strength of the study results are enough to change his or her practice.

SHOES/SHOE MODIFICATIONS

Indications

- Shoes are (with a few exceptions) an integral part of the orthotic management of the lower extremity

Design Features

Custom shoes with appropriate reinforcing may be used for mild medial/lateral control problems.

Characteristics of off the shelf shoes to be used with orthoses:

- Sturdy construction of the upper to prevent movement of the orthosis in the shoe during ambulation.
- Secure anterior closure of the shoe (ideally laces) maintains the foot in the orthosis and prevents the shoe from slipping off.
- A wide toe box creates space for the orthosis and forefoot in the shoe.
- The sole of the shoe creates a non-slip surface for the orthosis during stance phase.

External shoe modifications are used to aid in the control of the lower extremity during stance phase (GRF control):

- Shoe lifts accommodate for leg length discrepancies and ensure that the pelvis is level during stance phase.
- A heel lift accommodates for a plantarflexion contracture This allows forward progression and minimizes knee hyperextension through midstance.
- Medial or lateral wedges accommodate for fixed varus or valgus respectively during stance phase.
- Flares or buttresses (fig 1A) increase medial or lateral control of the foot during stance phase (GRF control) when lower profile orthoses are used (supramelleolar orthoses and below). Used with AFO's, buttresses (fig 1B) and flares on shoes have some influence on the medial/lateral stability at the knee (GRF control).

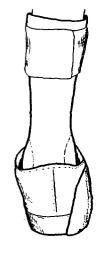
Clinical Experience

Custom shoes are used in some cases for children with mild spasticity (less than 2 on the scale of Ashworth) up to age 12. these shoes incorporate a reinforced shaft and tongue along with soft inlays to control valgus and varus deformities (60).

When using low profile orthoses (supramalleolar and below), the structure and type of sole on the shoe in combination with the extrinsic posting material of the orthosis will determine the medial/lateral motions of the foot during stance phase.

Rocker bottom soles decrease the flexion/extension moments about the knee when using rigid AFOs during stance phase. This reduces knee flexion instability from heel





A - Medial Buttress

7

B - Medial Buttress with AFO



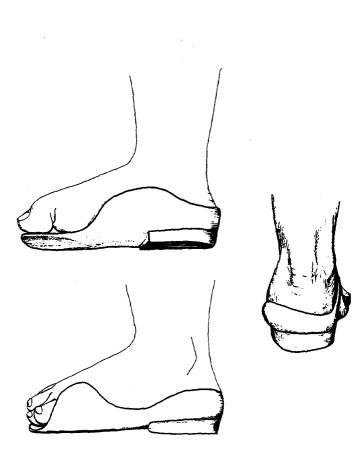


FIGURE 2 - Heel Foot Orthosis

strike to foot flat, minimizes excessive knee hyperextension at toe off and produces a smoother forward progression of the body during all of stance phase.

Scientific Research

Using shoe lifts to accommodate for tibial length differences and even the pelvis in stance, 8 of 10 children with hemiplegia were said to have amore even gait with shoe lifts on. No signs of knee hyperextension or toe drag was noted (79).

FOOT ORTHOSES/HEEL FOOT ORTHOSES/ SUPRAMALLEOLAR ANKLE FOOT ORTHOSES

Indications

Indications for the use of this category of orthoses include:

- mediolateral instability of the subtalar joint (GRF control)
- midfoot instability resulting in:

*forefoot abduction or adduction (3PP control) *forefoot valgus or varus (GRF)

- *forefoot desciferion (CDE)
- *forefoot dorsiflexion (GRF)
- mild to moderate spasticity (62)
- need for reduction of hypertonic foot reflex activity (62)
- ability to achieve heel strike (62)

Contradictions

Contraindications for the use of this category of orthoses include:

- lack of voluntary dorsiflexion control
- moderate to severe M/L spasticity (not controllable by the GRF control)
- fixed equinus

Foot Orthoses

Design Features

Foot orthoses extend over the plantar surface of the foot with minimal medial/lateral contact. Incorporated with a sturdy shoe, forefoot and medial/lateral control can be achieved. Both "cut and paste" and custom casted designs have been used:

- Cut and paste (no positive cast) designs incorporate a shoe insert offoam or leather that is cut to the shape of the shoe. Navicular and/or metatarsal pads are added as needed.
- In custom casted foot orthoses (from positive cast), a variety of foams and plastics are used to cover the plantar surface of the foot. Different densities of posting material at the heel and metatarsal heads is used to maintain the foot in the corrected position.

Clinical Experience

Cut and paste orthoses is a simple, quick and inexpensive way to control mild supination/pronation problems seen during stance phase.

Foot orthoses are used for mild hypertonic reflex activity seen clinically as mild toe clawing with stable subtalar and ankle joints (62).

Heel Foot Orthoses (Fig 2)

Design Features

Heel foot orthoses are custom casted plastic orthoses with trim lines inferior to the malleoli. Medial/lateral walls and intrinsic and/or extrinsic posting prevent foot deformities:

- Various combinations of medial/lateral walls at the metatarsal heads, midfoot and calcaneus control abduction/adduction of the forefoot.
- Intrinsic posting of the forefoot is achieved by the addition of plaster to the positive cast on the plantar surface of the medial or lateral metatarsal heads decreasing forefoot varus or valgus respectively. A reduction of a part (correctable portion) of the forefoot valgus and varus deformity is allowed to occur during stance phase.
- Extrinsic posting of the forefoot is achieved by the addition of a wedge of material external to the orthosis on the medial and/or lateral plantar surface of the orthosis. This accommodates for the rigid non-correctable portion of forefoot valgus or varus deformity during stance phase. Extrinsic forefoot posting can also be used to maintain a neutral forefoot position and prevent some supination/pronation of the subtalar joint from heel off to toe off.
- Extrinsic posting of the forefoot is achieved by the addition of a wedge of material external to the orthosis on the medial and/or lateral plantar surface of the orthosis. This accommodates for the rigid non-correctable portion of forefoot valgus or varus deformity during stance phase. Extrinsic forefoot posting can also be used to maintain a neutral forefoot position and prevent some supination/pronation of the subtalar joint from heel off to toe off.
- Extrinsic posting of the hindfoot accommodates for fixed valgus or varus deformities and maintains the foot in subtalar joint neutral during stance phase.

Extrinsic posting of the hindfoot is essential for stabilizing the orthosis in the shoe when the GRF is the only stabilizing force being used.

- Extrinsic posting of the hindfoot accommodates for fixed valgus or varus deformities and maintains the foot in subtalar joint neutral during stance phase. Extrinsic posting of the hindfoot is essential for stabilizing the orthosis in the shoe when the GRF is the only stabilizing force being used.
- Supination/pronation of the foot about the subtalar joint is achieved by an intimate locking of the foot/orthosis/shoe combination so that there is no movement between them. Extrinsic plastic heel posts incorporated at the time of molding maximise the stability of the orthosis in the shoe.

The type and thickness of materials dictates the flexibility of the device:

- Thinning the plastic in selected areas allows for the increased flexibility allowing some motion of the foot and a "soft feel" during stance. The types of plastics from rigid to flexible may include: ortholen, polypropylene, subortholen, colyene, modified polyethylene. Thinner plastics are typically used (1/8" - 3/32").
- Bonding a second layer of plastic to the plantar surface of the orthosis during molding reinforces the foot plate to stabilize against excessive forefoot dorsiflexion in an unstable midfoot.
- The materials used for extrinsic posting of the forefoot and hindfoot vary from high to low density according to the amount of supination/pronation to be allowed during stance phase. Posting material from high to low density may include: ucolene, neoprene (crepe), extra-firm nickoplast, medium density EVA, nickoplast, EVA. Similar materials added externally between the forefoot and hindfoot posts increase the rigidity and change the dynamics of the midfoot during stance.
- Padding added inside the orthosis may be needed to accommodate for bony prominences (navicular, base of the 5th metatarsal, etc) to prevent skin problems.

Clinical Experience

Dynamic hallux varus secondary to spasticity of abductor hallucis muscle can be controlled with medial extension (58).

Supramalleolar AFOs

Design Features

Supramalleolar AFOs are included in the same category as heel foot orthoses category since they provide a similar functional control of the foot during gait. They have similar design features to heel foot orthosis with the addition of medial and lateral trim lines extended to just proximal to the malleoli; a posterior trimline cut down to just

proximal to the calcaneus to allow free plantarflexion; an anterior opening at the malleoli level for free dorsiflexion; and a circumferential design.

The original design (fig 3) the dynamic ankle foot orthosis (DAFO), outlined by Hylton (30) incorporated the following characteristics:

- A very thin circumferential shell of plastic (3/32" polypropylene) allows slight movements to improved balancing strategies while maintaining foot stability. The increased flexibility of the plastic shell produces a "soft feel" for improved tolerance of the orthoses by the children. The use of total contact and containment controls spasticity and decreases edge pressure problems.
- A custom contoured footplate provides support and stabilization for the dynamic arches of the foot. Aggressive modifications of the footplates provide the following functions:

* Modification of the distal aspect of the plantar surface of the calcaneus (increased pressure) prevents the calcaneus from plantarflexing and adducting.

* Modification of the lateral arch acts as a counter pressure for medial arch modifications and decreases tone.

* Metatarsal arch modification (increased pressure) provides long axis alignment, resists pronation, decreases metatarsal pressure (inhibitory) and marks the sensory centre of the foot.

- * Toe dorsiflexion aids in tone inhibition and intrinsic long stretch.
- Extrinsic foam posting on the entire planar surface of the orthosis locks the orthosis into the shoe and allows slight supination/pronation motions within the shoe.
- Ankle and forefoot straps on the dorsum of the foot lock the orthosis securely on the foot.

(Note - With the addition of medial and lateral extensions up the tibia and a posterior calf strap to this orthosis, control of mid swing plantarflexion may be achieved for mild to moderate spasticity (62). This would then be classed as a true AFO.)

A non-circumferential design of supramalleolar orthosis (fig 4) has been used:

- Forefoot and midfoot "tabs" of plastic (3/32" - 1/8" plastic (colyene)) which wrap around to the dorsum of the foot control abduction/adduction instabilities of the midfoot. The tabs are thinned and flared as they round the dorsal surface of the foot. This allows the tabs to be flexed and locked over the dorsum of the foot when the shoe is tightened producing excellent containment and control of the foot and minimal skin pressure and bulk problems within the shoe.



FIGURE 3 - Dynamic Ankle Foot Orthosis (DAFO)

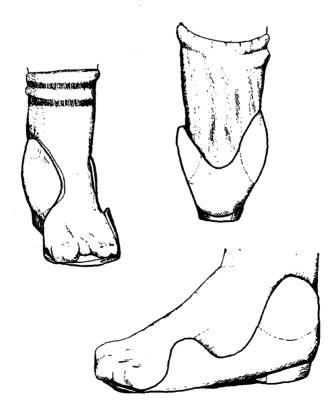


FIGURE 4 - Supramalleolar AFO (non - circumferential)

- Generous padding of the lateral malleolus, medial malleolus and medial arch create soft pressure tolerant surfaces for the bony prominences. The medial and lateral malleolar walls when secured into high top running shoes lock onto the foot mediolaterally maximizing control of the subtalar joint with minimal pressure problems.
- No straps are needed but the use of a high top shoe is recommended to maintain the stability of the orthosis around the foot. The orthoses may be left in the shoes as the flexibility of the plastic allows easy entry of the foot.
- Extrinsic hindfoot posting (colyene, added at the time of the original molding) prevents movements of the orthosis in the shoe and maximises the lever arm of the GRF during stance phase. An extra layer of plastic is sometimes applied to the plantar surface (added at time of original molding) for the extra rigidity when excessive midfoot instability is present.

Clinical Experience

The use of thin flexible plastics and internal padding for this type of orthosis allows for slight supination and pronation of the foot while still maintaining gross control of foot deformities. The ability to leave the orthoses in the shoe and the "soft" feel of these orthoses has greatly increased tolerance and compliance.

Scientific Research

Two single case studies using DAFOs found:

- A more immediate decrease in knee flexion at initial contact in gait cycle of a two year old child with spastic diplegia when using the orthosis with NDT therapy compared to NDT therapy alone (20).
- Improvements in symmetry and standing balance (longer duration, better recovery from loss of balance) were demonstrated in a 4 year old child with spastic quadriplegia (27).

ARTICULATED (SINGLE-AXIS) ANKLE FOOT ORTHOSES

Indications

When variable amounts of ankle motion allow a more functional gait pattern:

- A plantarflexion stop prevents plantarflexion of toe walkers in stance (3 PP control) knee hyperextension from foot flat to toe off (GRF control) and plantarflexion in swing phase (3 PP control)
- A dorsiflexion stop resists knee flexion for a mild crouch gait pattern (GRF control)

- Free motion in dorsiflexion encourages normal tibial excursion over the foot with stretching of the calf muscles during stance phase (GRF control).

Use of a hinged AFO requires five or preferably ten degrees passive ankle dorsiflexion (without compromising neutral STJ and MTJ positions).

Hinged AFOs are used to control moderate to severe spastic deformities of the subtalar joint (3PP control).

Midfoot instabilities controlled by hinged AFOs include:

- forefoot abduction or adduction (3PP control)
- forefoot valgus or varus (GRF)
- forefoot dorsiflexion (GRF)

Contraindications

Contraindications for the use of hinged AFOs include:

- hamstring muscle contractures and/or moderate to severe loss of ankle, knee and hip extensors resulting in a crouch gait pattern.
- when ankle dorsiflexion during gait is completely restricted by severe triceps surae spasticity.
- fixed plantarflexion contractures.
- excessive fixed equinovarus deformity.
- an unstable midfoot in subtalar joint neutral.

Design Features (Fig 5)

Hinged AFOs extend from distal to the fibular head to the toes and incorporate a hinge at the ankle joint. They can incorporate similar design features to control the midfoot as a supramalleolar AFO. Current pediatric designs of ankle joints which are molded into the orthosis include: Gillette double flexure joints, ortholen (Chedoke) joints, Oklahoma joints, Gaffney joints, USMC Select joints.

A plantarflexion stop can be achieved by the following methods:

An extra piece of plastic is molded inside the main plastic at the achilles tendon. The cut line for the articulation of the orthosis goes through the middle of this internally molded plastic to provide a broad plantar stop. The protruding part of the screw can be embedded in crepe rubber to minimize the "clicking noise" when the plantarflexion stop engages. A relatively noise free

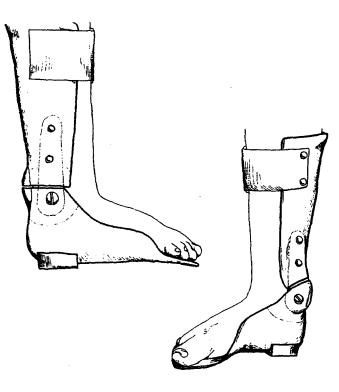
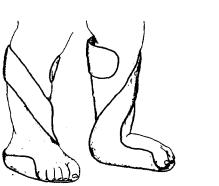


FIGURE 5 - Hinged AFO with Ortholen (Chedoke) Joints



Ż

7



A - Hemispiral Orthosis

B - Neurophysiological AFO

FIGURE 6

alternative is the use of a small rod of plastic which is glued into a hole drilled in the plastic stop. The stop is adjusted by cutting down the length of the rod.

- A commercially available vacuum moldable posterior dorsi/assist plantar stop component allows for an adjustable plantarflexion stop (3).
- The introduction of various cams into the USMC select joint creates a number of different plantarflexion stops.

A dorsiflexion stop/assist is achieved by:

- A strap (dacron) riveted across the posterior articulation.
- A self tapping screw mounted into the posterior aspect of the ortholon (Chedoke) joints creates an adjustable dorseflexion stop.
- The introduction of various cams into the USMC select joint creates a number of different dorsiflexion stops.
- A commercially available vacuum moldable posterior component creates for a dorsiflexion assist (3).

The hybrid plastic design (inner liner of flexible plastic) can be used with hinged AFOs (details of hybrid system under rigid AFO)

Clinical Experience

Using a hinged AFO, control of the motions of the lower extremity changes according to the movements allowed by the mechanical ankle joints:

- Free motion hinged joints control subtalar joint motion (3PP control) but allow unrestricted dorsiflexion/plantarflexion.
- A plantarflexion stop at 10-15 degrees dorsiflexion prevents knee hyperextension in stance phase (GRF) control) maintains foot dorseflexion in swing phase (3PP control) and allows normal forward progression past midstance.
- A dorsiflexion stop at 5-10 degrees dorseflexion limits the crouch pattern (GRF control) and creates a lever arm for push off.

Movements of the ankle axis are closely associated with movements of the subtalar and midfoot joints through closed kinetic chain motion. Two concerns involving closed kinetic chain motion must be addressed when prescribing a hinged AFO which allows free dorsiflexion:

- The increased range of dorsiflexion during stance phase permitted by the use of a hinged AFO must be secured before increased motion in ankle dorsiflexion is allowed.

The use of hinged AFOs is contraindicated for excessive instability of the midfoot in the cerebral palsy child with moderate to severe spasticity. This may be explained as follows: As the tibia rotates forward in stance phase, the spastic triceps surae blocks ankle dorsiflexion and transfers the forces and moments of weight bearing to the midfoot. The hinged AFO allows this motion resulting in collapse of the unstable midfoot against the orthosis during stance producing excessive pressure problems (80).

Improvements in the gait patterns using hinged AFOs have also been suggested:

- In a child with full passive range of motion and some active dorsiflexion a hinged AFO allows ankle motion and a more normal gait pattern (84).
- The use of hinged AFOs allow stretch of calf muscles resulting in decreased spasticity, longer stride length, improved balance and increased ROM in the ankle (56).

Two additional orthotic designs which do not have hinged joints but allow motion at the ankle axis through deformation of the plastic include:

- A hemispiral orthosis (fig 6A) spirals around the leg and attaches to a footplate allowing controlled varus with resisted dorsiflexion/plantarflexion. It has not been commonly used because of problems with fabrication, fit, breakage and structural strength (22).
- The neurophysiological AFO (fig 6B) (non articulated) has unique trimlines which include narrowing of the foot plate (allowing dorsiflexion/plantarflexion) and cutouts from above the medial malleolus around to and including the achilles tendon (allowing inversion/eversion). Indications for use include mild to moderate spasticity, normal passive dorsiflexion with knee in flexion, minimal to moderate varus instability and a need for reduction of hypertonic foot reflex activity (62).

Scientific Research

Three studies found various kinematic improvements in gait when using hinged AFOs:

- In 15 children with mild to moderate spastic diplegia greater midstance ankle dorsiflexion and faster walking velocity was found with hinged AFOs compared to shoes only. No differences was found in the emg at vastus lateralis, medial hamstrings, anterior tibialis and lateral gastrocnemius across all groups (37).
- In a group of 24 cerebral palsy children wearing "posterior leaf spring orthoses", a significant increase in ankle dorsiflexion during swing was observed compared to no orthosis. No difference was found in ankle power generated between the two test conditions (48).
- In a single case study of a four and one half year old child, hinged orthoses were found to be superior when compared to rigid orthoses in terms of: a more

natural ankle motion during stance, greater symmetry of segmental lower extremity motion and decreased knee motions during stance (41).

In a group of 36 cerebral palsy children with spastic diplegia, 18 were randomized to a braced group (hybrid hinged AFOs with variable plantar/dorsiflexion stops) and 18 to a non-braced group. After 4 months both groups were assessed using the Gross Motor Functional Measure (a quantitative gross motor functional measure for cerebral palsy) the Gross Motor Performance Measure (a qualitative gross motor functional measure for cerebral palsy) and range of ankle dorsiflexion. No statistically significant difference resulted between the groups when measurements were taken with the orthosis off of the braced group. Statistically significant differences were seen in the alignment segment of the Gross Motor Performance Measure when measurements were taken with braces on the braced group.

RIGID ANKLE FOOT ORTHOSES

Indications

General indications for the use of rigid AFOs include:

- when maximum stability and immobilization is needed for the ankle, subtalar and midfoot joints in all planes during swing and stance phase (3PP control).
- when closed chain motion control of knee and hip position is needed:

* to prevent knee hyperextension in stance phase (GRF control)

* to prevent crouch gait pattern in stance phase for patients with moderate to severe loss of knee and hip extensors (GRF control)

- post surgical applications
- for moderate to severe spasticity
- to protect an unstable midfoot from the closed chain effects of ankle dorsiflexion when a spastic triceps surae is active during stance phase.

Functional indications for the different types of rigid AFOs are outlined below:

- functional indications for standard rigid AFOs (83).

* child is ready to stand but unable to balance on feet which are in pathlogical position (equinus, equino-valgus, varus, equinovarus).

* child stands on heels but walks on toes.

* child pulls up to standing on toes and stays there.

- functional indications for a floor reaction orthosis (for crouch gait) (43)
 - * overactive hamstrings (with weak quadriceps) which leads to overflexed knees.
 - * surgically overcorrected heel cord.
 - * over extended heel cord due to poor protection after surgery.
 - * over lengthened heel cord from long term flexion pattern.
- functional indications for plastic hybrid orthoses
 - * control of severe spasticity of STJ and MTJ
- functional indications for circumferential orthoses:
 - * in infants with "chubby" feet the circumferential design offers increased control of excessive amounts of soft tissue.
 - * to avoid pressure problems on bony infant foot.
 - * if a rigid orthosis causes increased spasticity, the flexibility of the circumferential design may minimize this.

Contraindications

- when some ankle motion increases function

Standard Rigid Orthosis (custom plastic) (Fig 7)

Design Features

Various trim lines and strapping systems create various types of control including the following:

- A trim line behind malleoli creates a strong plantarflexion stop, weak dorsiflexion resist and an inversion/eversion stop for mild to moderate spasticity of the subtalar joint.
- A trim line in front of the malleoli creates a strong plantarflexion/dorsiflexion stop of the subtalar joint for moderate to severe spasticity.
- A strap anterior to the ankle joint and angled down at 45 degrees creates an optimally directed middle force for the plantarflexion stop three point pressure system.

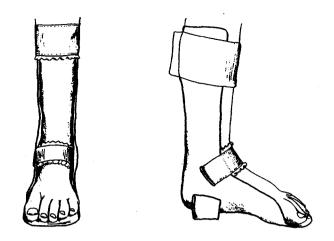


FIGURE 7 - Standard Rigid Ankle Foot Onthosis (Custom Plastic)

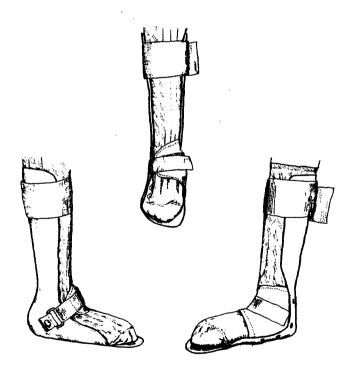


FIGURE 8 - Rigid AFO with Moulded Leather Strap

- A molded leather strap (fig 8) encasing the medial malleolus and/or medial arch and attached by a pull back strap to the lateral wall (with anterior trim line) creates an effective control of a severely spastic pronation foot or accommodative control of a fixed planovalgus deformity (rocker bottom foot). The alternate configuration using a lateral molded strap controls the comparable equinovarus problem. Bioflex (Erkoflex) may be used.
- Various combinations of medial and lateral walls at the metatarsal heads, midfoot and calcaneus control abduction/adduction motions of the forefoot.
- The distal plantar trimline of foot plate when cut to the tips of the toes allows for growth, dorsiflexion of the toes during push off and a flat base for toe clawing. The plastic on the plantar surface is thinned from proximal to the metatarsal heads to the end of the foot plate to create flexibility for the toe dorsiflexion. The trim line is cut back to the toe crease of growth and toe clawing is not a problem.

The position of the orthosis (degrees of dorsi/plantarflexion) produces different effects on various parts of the gait cycle:

- A dorsiflexion stop at 0-4 degrees dorsiflexion prevents knee flexion in a crouch gait pattern. Increased ankle dorsiflexion may be needed to accommodate for knee flexion contractures sometimes associated with a crouch gait.
- A plantarflexion stop in 5-7 degrees dorsiflexion prevents knee hyperestension from heel strike to midstance and allows forward progression from midstance to heel off.
- Greater than 7 degrees of dorsiflexion may be needed to accommodate for knee and hip flexion contractures.

Clinical Experience

The rigid AFO typically produces problems at heel strike and push off:

- At heel strike the plantar stop of a rigid AFO produces a prolonged knee flexion moment and possible knee instability. This has been experimentally documented as outlined by Condie (85). An external cushioned heel wedge or a rocker bottom sole aid in decreasing this effect.
- From midstance to toe off a rigid AFO positioned at 0 degrees dorsiflexion resists forward progression of the body and creates a strong extension moment at the knee. A rocker bottom sole on the shoe and/or slight dorsiflexion helps to reduce these two effects (85).

Molded leather straps over the medial or lateral malleolus are used to control severe medial/lateral spasticity of the subtalar joint or accommodate for contracted unstable midfoot deformities.

The use of rigid AFOs produces a more stable gait pattern (increased velocity and stride length) (58).

Growth adjustments of rigid plastic orthoses have been attempted with the use of inner liners (pelite) to accommodate for circumferential changes and telescoping uprights for length changes (14).

The use of rigid AFOs can create other problems with function (83).

- W sitting produces excessive internal/external rotation force at knee.
- Restricted dorsiflexion interferes with a normal reciprocal crawling action.
- Toeing in during gait becomes more evident when an AFO is applied due to blocking of the compensatory action of the subtalar joint.

Scientific Research

A number of energy studies of children with cerebral palsy using rigid orthoses have been completed:

- In a study of 35 diplegics (12 with orthoses), the CP children demonstrated three times the energy expenditure in walking compared to normal. Energy expenditure of diplegics increased with age due to increased body weight, poor motor control, spasticity and impaired balance reactions (84).
- In a study of 18 children with spastic diplegia, heart rate was used as an indicator of efficiency. A statistically but not clinically significant decrease in the heart rate was found when using the orthoses compared to without orthoses (43).
- EMGs showed a decreased amount of cocontraction therefore increased efficiency in a comparative study of 17 cerebral palsy children with orthoses compared to without orthoses (29).

Gait analysis studies of children with spastic diplegia and hemiplegia comparing rigid AFOs to no orthosis conditions have been completed:

- Seventeen children with cerebral palsy (classification?) demonstrated increased velocity, increased stride length and joint motions closer to normal when using AFOs compared to no orthoses (73).
- Improved midfoot alignment in rigid brace versus shoe was found in a study of 15 spastic diplegics (37).

- Genu recurvatum was eliminated in 12 children (7-9 years/classification?) with cerebral palsy when using a rigid AFO (Risebtgak et al, 1975).
- A group of ten spastic diplegics (1-5 years) demonstrated no significant effect of AFO on cadence, velocity or stride length between no orthosis and rigid AFO (52).
- a six year old spastic diplegic using supramalleolar AFOs extended to mid calf posteriorly demonstrated no difference in duration of standing and balance with and without orthoses. Video observation showed the "high guard" upper extremity position when not wearing orthoses compared to a more relaxed position with orthoses on. Improved fine motor skills were shown when using the orthoses (tone reducing) (72).
- Eleven hemiplegics (mean age 6 years) demonstrated decreased ROM at the knee and increased push off of the normal side when using AFOs on the effected side compared to no orthosis (61).
- Four diplegics and one hemiplegic (mean age 5 years) with knee hyperextension problems completed a gait assessment (no orthosis). Rigid AFOs were fit and fine tuned with ground reaction force monitoring (using heel wedges) to minimise the ground reaction force moment at the knee. After 4-6 months in the orthoses the children were again assessed (with no orthoses). Improvements in knee extension moments (decreased), foot/ground contact and stance phase posture were seen. The authors suggested this demonstrated a motor learning effect from the orthoses (9).

Floor Reaction Orthosis (custom plastic)

Design Features

Two possible designs of floor reaction orthoses are:

- Posterior Entry Design (Fig 9): A plastic shell extends from mid-patella to the proximal edge of the metatarsals and completely surrounds the lower leg and foot except for a posterior opening to allow entry of the leg. Reinforcement of the plantar surface and up into the medial arch (with second layer plastic) is needed for sufficient rigidity of the foot section. The proximal anterior shell in combination with the shoe over the posterior calcaneus provides an effective force couple to prevent forward angulation of the tibia (crouch gait) in stance phase.
- Proximal Entry Design (Fig 10): From mid-gastrocnemius down the trim lines are similar to a standard rigid AFOs (with trim lines in front of the malleoli). From mid-gastrocnemius up the plastic curves around to enclose the leg anteriorly up to and including the PTB. The posterior aspect of the shell is cut down to allow entry to the foot. Reinforcement of the plastic structure around the malleoli and on the plantar surface of the foot is sometimes needed for a structurally stable dorsiflexion stop. The force couple to prevent dorsiflexion is

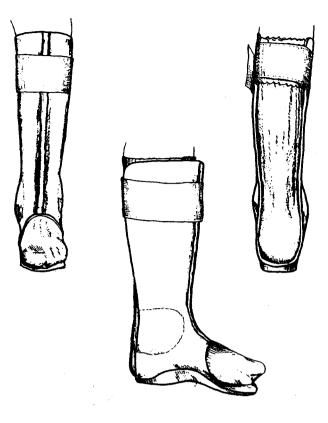


FIGURE 9 - Floor Reaction AFO: Posterior Entry Design

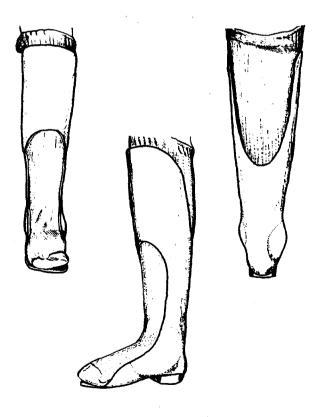


FIGURE 10 - Floor Reaction AFO: Proximal Entry Design

provided by the proximal anterior shell and the contact point at the posterior calcaneus.

At foot flat with the shoe on, an anterior tilt of the tibia of 3-5 degrees is the typical dorsiflexion angle of the orthosis to allow forward progression of the body past midstance. The dorsiflexion angle set in the orthosis may decrease to protect weak quadriceps or increase to accommodate for knee flexion contractures.

Clinical Experience

Both floor reaction designs provide optimal dorsiflexion stop mechanics (GRF control) for crouch gait patterns but each has inherent problems:

Problems with the posterior entry design include:

- difficulty in donning if the width of the patient's metatarsal heads are wider than the midfoot.
- making growth or other adjustments around the midfoot and ankle is difficult.
- the hindfoot is not completely contained in the plastic producing less effective medial/lateral control of the calcaneus.

Problems with the proximal entry design:

- difficulty donning the orthosis through the proximal end of the orthosis.
- structural stability of the orthosis for a dorsiflexion stop on an obese person may be difficult to attain.

Scientific Research

In case report of an 11 year old child with spastic diplegia using anterior floor reaction orthoses, improvements were seen in single support time and stride length compared to the no orthosis condition. Energy consumption was unchanged between the braced and unbraced group (26).

Hybrid Plastic Orthoses (Fig 11)

Design Features

The hybrid plastic orthoses combine a flexible plastic inner shell (modified polyethylene) and a rigid plastic outer shell (polyproylene or colyene).

Similar control features as the standard rigid AFO are possible when using the different shells

- the outer shell:

* gives the structural rigidity to the orthosis where needed.

* may be flared in selected areas to allow small movements of the foot within the orthosis or to decrease pressure problems.

* selected cutouts of the outer shell helps decrease bulk.

- the inner shell:

- * gives flexible dynamic control of the foot within the orthosis
- * gives decreased pressure problems due to flexibility.

Clinical Experience

The hybrid design is most useful for severe inversion/eversion spasticity. The inner shell provides flexible, circumferential and dynamic control in selected areas to help reduce skin problems. The outer shell provides substantial rigidity to the overall structure of the orthosis to control the high forces involved.

Circumferential Orthoses (Fig 12)

Design Features

The circumferential orthosis completely surrounds the lower leg and foot with a flexible plastic. A cut line down the centre of the front and the thinner plastic on the anterior tibia and dorsum of the foot allows donning of the orthosis.

Two alternate designs include:

- an inner layer of foam and a thin outer layer of polypropylene (22)
- a modified plyethylene outer shell with pads at the malleoli and navicular.

Clinical Experience

Slight movement of the foot due to the flexibility of the orthosis decreases skin pressure problems. Spasticity may also decrease due to the softer material which does not stimulate a spastic response compared to an AFO fabricated of a more rigid plastic.

The circumferential design is particularly useful in controlling the excess tissue seen in infant feet.

TONE REDUCING/INHIBITING ANKLE FOOT ORTHOSES (AND CASTS)

Indications

Tone reducing orthoses aim to reduce reflex induced deformities and/or elimination of extensor thrust.

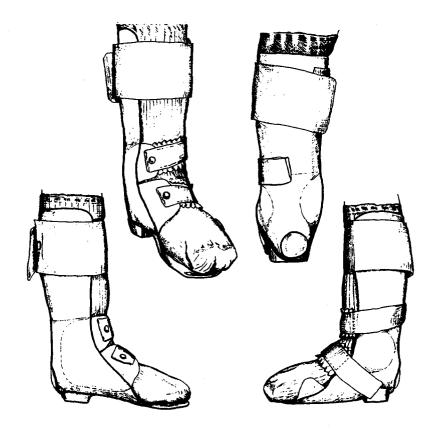


FIGURE 11 - Hybrid Plastic AFO

Į

7



FIGURE 12 - Circumferential AFO

Tone reducing orthoses are used as an adjunct to therapy supplying the "extra pair of hands" to control the ankle/foot while allowing the therapist to concentrate on more proximal control. They are a first stage device to enhance standing balance and facilitate early gait training.

Custom Plastic Orthoses with Plantar Surface Modifications

Design Features

The custom plastic tone reducing orthoses have similar design characteristics of standard rigid AFOs with the addition of the following inhibitive modifications of the foot plates (fig 13A) (56).

- Increased pressure from the proximal medial calcaneus to under the talus relieves weight bearing on navicular and promotes dorsiflexion during weight bearing increased pressure on the lateral aspect of plantar surface at the cuboid promotes peroneal activity as well as a stabilizing effect on the gluteus medius.
- A metatarsal pada or bar along with relief of the metarsal heads inhibits toe grasp. Extension of the digits is also used to inhibit toe grasp. An alternative modification is a toe crest at the PIP joints to unweight the metatarsals at late stance phase and inhibit reflex hypertonicity (62).

Clinical Experience

Two possible mechanisms have been suggested for the tone reducing effect:

- The neurophysiological effect uses cutaneous stimulation and joint position to reduce tone. Inhibition of the tonic reflexes through the use of appropriately modified orthoses reduces reflex hypertonicity of the foot (19, 62). Positioning the foot in the "quiet zone" of 10-20 degrees dorsiflexion and calcaneal varus may reduce tone through elongation of the spastic triceps surae and peroneal muscles respectively (22).
- Increased stability created by the orthosis may reduce the biomechanical demand on the neuromuscular system and may directly reduce muscle tone (85).

Scientific Research

In a study of 10 spastic diplegics ranging from 1-5 years of age, a gait video was critiqued by 8 therapists. The gait of 6 children looked best in 3 arch orthosis (inhibitive), 3 children looked better in one arch orthosis (standard rigid) and one looked better in no orthosis (52).

TRAFO (tone reducing ankle foot orthoses) (Fig 13B)

Design Features

The position of the ankle/foot in the TRAFO includes (25).

- ankle joint immobilization at 90 degrees
- subtalar joint held in maximally congruent position
- long axis of midtarsal joint is pronated
- oblique axis of midtarsal joint is supinated
- toes are dorsiflexed

The design features of the TRAFO include:

- A two part rigid anterior/posterior shell (synthetic casting tape) formed directly on the patient and extending from the proximal calf to the distal edge of metatarsal heads.
- Anterior/posterior shells are held together with a figure 8 strap at the ankle and pull back strap at the proximal calf.
- A plantar plate (ie crepe) is adhered to the scotch cast shell and extends 1/4" ti 1/2" past the distal border of the toes. The plantar plate may incorporate abiplane rocker that permits medial/lateral motion of the leg about the base of support (25).
- The inside is completely lined with cotton stockinette and an anterior pad (if necessary).
- A foam wedge is placed under the toes to extend the MCP joints and flex the distal joints.

Inhibitive Plaster Casts

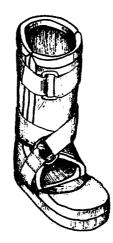
Design Features

Inhibitive plaster casts are circumferential plaster wraps directly applied to the patient and incorporating tone reducing foot plates.

Characteristics of tone inhibiting casts include (25).

- hyperextension of the toes





A - Tone reducing foot plate

B - Tone Reducing Ankle Foot Orthosis

FIGURE 13



FIGURE 14 - Twister Cables

1

- pressure under metatarsal heads
- stable ankle position
- deep tendon pressure along calcaneal tendon

Clinical Experience

Inhibitive plaster casts are useful when the children first starting to stand. The orthoses act as a second pair of hands creating a stable base of support facilitating motor function and reducing hypertonia. They inhibit abnormal patterns of muscle action and facilitate the development of more normal patterns of muscle action (25).

À

Clinical experience with 99 patients using short leg casts (69) found the following.

- Immobilization of an unstable foot and ankle reduces tone and facilitates mobility of the knees, hips and trunk.
- The casts were followed by custom molded orthoses (ie polyprop AFO, UCBL, etc) for continuing passive and dynamic control of deformities.

Scientific Research

A number of studies have investigated gait and muscle tone when children with cerebral palsy are using inhibitive plaster casts:

- A single subject design study using two children with spastic diplegia (ages 3.5, 5 years) compared tone reducing casts to standard plaster casts. Increased stride length and better alignment of the foot was found in the tone reducing casts (29).
- Sixteen children (3 quadriplegics, 6 hemiplegics, 7 diplegics) were divided into casted and uncasted groups. The casted group used bivalved plaster casts with foot plates. A significant increase in stride length was found in the casted group (3).
- In a group of 86 children with poor prognosis of walking (using a locomotor prognosis system) 60 percent became ambulatory after the use of inhibitive casts and standard neurodevelopmental program (19).
- Thirty-two children (17 diplegics, 8 quadriplegics, 3 hemiplegics) used inhibitive plaster casts. The results showed no difference in deep tendon reflexes, ankle cionus, plantar responses and developmental motor skills two weeks and five months after cast removal. Improvements were seen in increased range of motion and increased foot floor contact (78).

HIP KNEE ANKLE FOOT ORTHOSES

In the past conventional metal and leather KAFOs and HKAPFOs have been used with cerebral palsy to control gait. Current orthotic management uses very few orthoses above the AFO level with the possible exception of orthoses for internal rotation of the hip.

Indications

- mild to moderate spastic internal rotation which interferes with ambulation.

Contraindications

- severe spastic internal rotation.

Orthoses to Control Transverse Hip Rotation

Design Features

Two types of orthoses to control transverse hip rotation have been used:

- Twister cables (Fig 14): A flexible rod attaches to a pelvic band proximally and the tibial section of an AFO distally. Used bilaterally or unilaterally, this orthosis controls internal external rotation about the hip. The flexible rod can be composed of a tightly coiled spring or solid plastic rod. Free motion hip and knee joints may also be added.
- Spiral strap: An elastic strap spirals around the leg from the proximal AFO to a pelvic band. Tension on the strap controls internal rotation.

Clinical Experience

Twisters are used for int/ext rotation problems (5, 15, 47, 60, 86, 87) The clinical experience of these authors suggests:

- Twisters are used to control excessive internal rotation not associated with spastic internal rotator muscles (86).
- External rotation bandages are used for extreme internal rotation causing stumbling and falling and spasticity not to exceed 2 on Ashworth scale. The author suggests that twister cables are not functional after 11 years of age (60).

REFERENCES

Mann R. Biomechanics in Cerebral Palsy. Foot and Ankle 1983 4, 114.

Risebtgak R J M, Deytscg S D M, Nukker W et al. A fixed-ankle below-the-knee orthosis for the management of gneu recurvatum in spastic cerebral palsy. J Bone Joint Surg 1975 <u>57a</u>, 545-547.

CURRENT ORTHOTIC PRACTICE IN RELATION TO THE IMPROVEMENT OF GAIT EFFICIENCY

Richard E Major BSc, Cert Ed, C Eng, M BES

INTRODUCTION

Before attempting to discuss this subject in any detail it is worth considering the meaning of the word efficiency which in the context of gait is taken here to be "achieving a desired goal whilst minimising energy consumption and mechanical stress on the biological system". Although a large body of literature exists in the field of lower limb orthotics related to cerebral palsy it is interesting to note that very few papers present any measured parameters and those that do often measure factors such as step and stride length, width of base, cadence, velocity or ground reaction force which are not necessarily related to efficiency. For instance, if stride length is increased due to orthotic intervention is this a good or bad thing in terms of efficiency? Similarly, observations of ground reaction forces during gait may be thought to give indications of mechanical stress but in practice measurements of the moments experienced at different joints will be much more significant. In reviewing the literature, papers which do not present data relating to gait efficiency have, in the main, been excluded.

The use of orthotics to prevent joint contracture and deformity is a key element in improving efficiency of locomotion. This is because of the adverse moments due to gravity and inertia that develop about joints in the presence of deformity. There are many examples in the literature (Winter 1987, Gage 1991) of the nature and variation of joint moments during the gait cycle but few authors point out the vital significance to gait efficiency of maintaining these at a minimum. The form of the skeletal structure tends towards minimising these moments during activity but contractures and deformity often defeat this objective.

When reviewing the literature for efficiency information it is interesting to note that AFOs feature prominently and, as will be shown, the important aspect of these orthoses is that they influence joints other than those they cross.

ENERGY

There are few reported examples of energy changes in cerebral palsied gait due to orthotic intervention. Campbell and Ball (10) reported on the energy cost of ambulation for 22 cerebral palsied children obtained using gas exchange methods. Little information was given of the orthoses used by 11 of the children and no conclusions were drawn relating orthotics and energy costs. Both Butler et al (1984) and Mossberg et al (43) have measured energy expenditure using the concept of Physiological Cost Index (PCI) first proposed by MacGregor (1981) which employs changes in heart rate combined with speed to give a reading in heart beats per metre. Hence a reduced reading indicates higher efficiency. The first of these papers reports two subjects where PCI was reduced from 1.53 to 0.98 and 1.7 to 0.7 but the exact details of orthotic intervention were not provided, being outside the scope of this paper. The more recent paper by Mossberg and co workers studied 18 children with and without AFOs, which were presumably of the fixed variety, and reported an average reduction in PCI from 1.51 to 1.34 when AFOs were used. In discussing their results the authors considered the length of time the orthoses had been used looking for evidence that longer term users would be more disadvantaged walking barefoot due to an orthosis induced learning effect. They suggested this effect was proposed by Butler et al (1984) but no evidence of a disadvantage was found and it was concluded that if there was a learning effect this probably occurred in under 4 months which was the shortest period of AFO use in the study. This concept of orthoses producing long term learning effects is worthy of further exploration.

ORTHOTICS AS THERAPY

"There is a great discrepancy between the number of brain-injured children and the number of physical therapists available to treat them. Too often the problem is solved by giving inadequate treatment, too few times, to too many children" This comment was made by Garret and co workers (23) nearly 30 years ago and yet it rings true today. They also made the statement "However, it is also well to consider that spasticity may be controlled by other methods besides the laying of the physical therapist's hands; a brace that could control spasticity would have an added advantage of being applied for a longer time than the physical therapist's hands are available". Thus it was recognised some time ago that there should be an active interrelationship between therapists and orthotists but few examples with demonstrated outcomes are to be found in the literature.

One approach is that described by Cusick and Sussman (13) where short leg casts were introduced for short periods as an adjunct to neurodevelopmental therapy. However, Watt et al (78) could not find evidence of any long term benefits from this approach and concluded that further investigation is required.

Another example of considering the links between orthotics and therapy grew from examining issues related to the learning of upright motor control in a gravitational and inertial situation (Butler and Major, 1992). This introduced a new concept of Targeted Learning which identifies the most proximal joint at which control is judged to be deficient and then blocks motion below this level and creates a new "pseudo" base of support at the segment immediately below the identified joint. By maintaining the new base in the correct orientation and providing suitable perturbations to challenge the neuromuscular system it is postulated that motor learning will be enhanced. Thus if control is assured down to the hip/knee area then fixed AFOs are an expression of this new theory. It can be seen in retrospect, that the first hint as to its application when using AFOs was given by Meadows et al (88) who demonstrated that a fixed AFO can harness ground reaction forces to modify external moments (perturbations) experienced by joints at knee level and above thus providing the "correct" biomechanical feedback.

This work was further expanded by Butler and Nene (1991) who used a video display of ground reaction forces overlaid on an image of the patient walking (Stallard 1994) so that estimations of joint moments could readily be made to aid clinical decision making. This technology could not be accessed by Meadows and co workers but is now commercially available. From both papers it is evident that the correct mechanics are unlikely to be achieved from the orthoses "as supplied" and that optimisation of the alignment will probably be required. It is also evident from the work of Butler and Nene that this is more than a simple biomechanical realignment of forces as described by Condie and Meadows (85) and represents a direct interaction with the active control system.

MOTOR LEARNING

The evidence that motor learning does indeed take place was provided by Butler et al (9) where marked reductions in the external knee moment arms tending to cause knee extension were demonstrated in eleven limbs of six cerebral palsied children during barefoot walking monitored prior to and after 6 months use of suitably "tuned" fixed AFOs. Fig 1 which is derived from this study shows the variation of knee extending moment arm produced by the ground reaction force plotted against percentage of stance phase. The knee moment arm was chosen, as opposed to the moment, since it is believed to reveal the underlying problem of motor control which could be masked by compensatory strategies were the knee moments to be reported. Note that all results are obtained during barefoot walking so as to exclude any direct effects of orthoses or footwear during the assessments. It can clearly be seen that the large abnormal knee extending moment arm near mid stance demonstrated before orthotic intervention is very much reduced after six months use of "tuned" fixed AFOs. Although these are averaged results the effect could be seen in each of the 11 limbs studied.

Although not formally reported in the literature, there is evidence from ongoing review of patients at Oswestry that this correction can be maintained long after the orthoses have been withdrawn with the longest case to date exceeding 3.5 years. Even in cases where a child reverts to a previous walking style, for example due to a growth spurt, it has been observed that a short orthotic re-intervention can easily restore the situation. Thus it can be seen that new, powerful and lasting techniques are opening up for the combination of orthotics and therapy.

It is interesting to note that Butler and Nene (1991) report that a heel raise as small as 3mm can be significant in modifying gait parameters (representing an angular change of about 2 degrees in floor/shank angle) whereas Condie and Meadows (85) state "Clinical experience suggests that the plantar flexion tone may be reduced if a position of 10°-20° of ankle dorsiflexion is adopted" suggesting a tolerance of 10 degrees in casting position. This wide variation may reflect a genuine difficulty in casting to a precise angle and when taken in conjunction with the motor learning evidence would suggest that routine optimum "tuning" should be undertaken for these orthoses.

ADDITIONAL EVIDENCE

The value of targeted motor learning is not limited to the use of AFOs. In an application of the theory to hip/knee control Farmer and Butler (1994) have not only demonstrated improved control but have also produced a lengthening of short hamstrings. Thus it may be possible to use conservative interactions with the control mechanism to influence muscle length. This is very much preliminary work which needs further investigation.

Further evidence of the efficacy of this approach is provided by Butler (1993) where trunk control has been targeted. Here six children without sitting balance achieved this milestone in periods ranging from 11-25 weeks and all maintained the ability after

withdrawal of the intervention with the longest follow up being 71 weeks. Although this is not directly relevant to gait and lower limb orthotics it does hold out the potential of introducing cerebral palsied children to walking training who would otherwise remain chair bound.

STABILITY, ENERGY AND CONTROL

From the forgoing it will be evident that the main factors that have been discussed are the energy cost of walking, stability as provided by orthotic intervention and the state of the control system. These three factors are interrelated and can be considered in a graphical manner (Fig 2) as described by Major and Butler (1995).

It is important to recognise that this is an hypothetical graph and that the exact shape of the line has not been determined although the general trend indicated is self evident. Also note that the Control axis on the right hand side is inverted. The broken lines are used to show the relationship in normals where an inherently low stability skeletal structure leads to the ability to ambulate at low energy levels but requires a very high degree of active control. If this control mechanism is compromised for any reason (in this case through cerebral palsy) the diagram shows, as dotted lines, that additional stabilisation is required which might take the form of a walking aid, an orthosis or both. However, reference to the diagram indicates that in this case it is unrealistic to expect normal energy costs of locomotion. Additionally this diagram reveals the benefits of working to improve the compromised control system, by whatever means, since this is likely to lead to a reduction in orthotic need and energy cost of walking.

This diagram my be of help in the decision making process when planning treatment, both to aid determining the best approach and also to give an indication of what outcomes might realistically be expected.

CONCLUSION

It is hoped that the work discussed here will prove useful to clinicians, therapists and orthotists as they attempt to tackle the difficult problems presented by cerebral palsy. Most of the papers referenced have been (or will be) published in this decade which would appear to justify the optimistic view that forward progress in this area is being made which is ripe for further exploitation. However it should be noted that many of the paper titles include, quite deliberately, the word "preliminary" which underscores the fact that much additional work is required both in expanding the application of new theory and proving the efficacy of application.

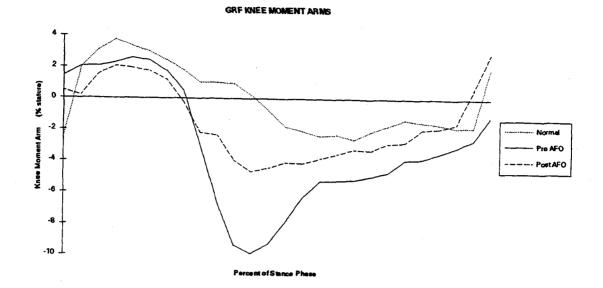


Fig 1

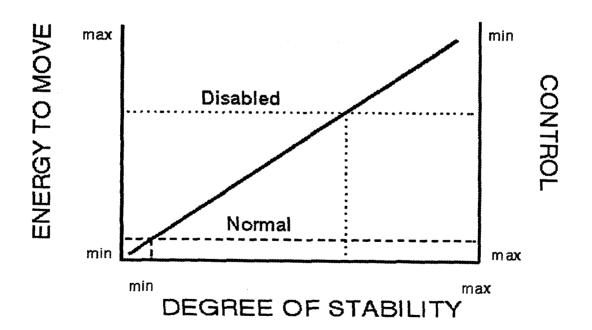


Fig 2

REFERENCES

Butler P, Engelbrecht M, Major R E, Tait J H, Stallard J and Patrick J H. Physiological cost index of walking for normal children and its use as an indicator of physical handicap. Dev Med and Child Neurol 1984 <u>26</u>, 607-612.

Butler P B and Nene A V. The biomechanics of fixed ankle foot orthoses and their potential in the management of cerebral palsied children. Physiotherapy 1991 <u>77</u>, 81-88.

Butler P B and Major R E. The learning of motor control: biomechanical considerations. Physiotherapy 1992 78, 1-6.

Butler P B. The physiotherapy management of motor impairment. PhD Thesis, Birmingham University Department of Surgery, 1993.

Farmer S E and Butler P B. Conservative treatment of crouch gait. Gait and Posture 1994 2, 41-42.

Gage J R. Gait analysis in cerebral palsy. MacKeith Press, 1991.

MacGregor J. The Evaluation of Patient Performance Using Long-Term Ambulatory Monitoring Technique in the Domicliary Environment. Physiotherapy 1981 <u>67</u>, 30-33.

Major R E and Butler P B. Discussion of segmental stability with implications to motor learning. Clinical Rehabilitation 1995 $\underline{9}$, 167-172.

Stallard J. Clinical experience in rehabilitation using the ORLAU Video Vector Generator. Proceedings of Biokinetics, 1994, Ludencheid 1994, 18-22.

Winter D A. The biomechanics and motor control of human gait. University of Waterloo Press, 1987.

BIOMECHANICS OF ORTHOTIC MANAGEMENT OF GAIT IN SPASTIC DIPLEGIA

Christopher L Vaughan PhD, Warren E Carlson MS, Diane L Damiano PhD PT, Mark F Abel MD

INTRODUCTION

The causes of cerebral palsy (CP) are many and varied. Although the aetiology of CP is difficult to establish, the primary lesion occurs during one of the following three time periods: before birth (prenatal), during birth (perinatal) and after birth (postnatal) but before 24 months. According to some studies (Eiben and Crocker 1983), the proportion of types is about 80% spastic (pyramidal disorder), 10% pure athetoid, and 10% ataxic, these latter two being extrapyramidal. Clinically, spasticity is characterized by a disharmony of muscle movements brought on by hyperactive stretch reflexes and hypertonicity of the anti-gravity muscles. The increased hypertonus causes overdevelopment of the spastic muscle groups and coupled with the marked under-development of their antagonists, this imbalance will often lead to deformities of certain major joints such as the hips, knees and ankles. Patients with spastic CP who are able to walk usually adopt an equinus (foot down) style of gait, have poor fine motor control and their spastic responses are velocity dependent. In this report we will only be concerned with spastic diplegic patients as they are very appropriate candidates for the use of AFOs, and their incidence - as a percentage of all children born with CP - has increased dramatically in the past three decades (Bleck, 1987).

There are a number of treatment modalities used to improve the function of the patient with spastic CP: physical and occupational therapy, orthoses, orthopaedic surgery (tendon lengthening) and neurosurgery (rhizotomy) and neuro-inhibitory drugs. The orthotic approach, because it is conservative, has a certain appeal. It is therefore not surprising that the application of orthoses to children with CP is now extremely widespread, and has been embraced by certain sections of the clinical community (34, Cusick, 1988). However, little objective data about the effects of these devices exists, and with some health care centers charging in excess of US\$1000 for a pair of AFOs, it is imperative that more objectivity be brought to bear on the problem.

HISTORY OF BRACE AND CAST TREATMENT

Bracing and the use of casts in treating children with CP goes back at least three decades (66, Te Groen and Dommisse, 1964). Perhaps the most widely cited work that forms the basis for much of today's treatment rationale stems from a paper by Duncan (1960) who identified four tonic reflexes of the foot: the toe-grasp, inversion, eversion and dorsiflexion reflexes of the foot. He hypothesized that there are certain "reflexogenous areas" and that a knowledge of these areas could be used to inhibit some of the reflexes in the child, thus leading to a reduction in tone. In a later follow-up to this work, Duncan and Mott (19) studied the use of inhibitive casting and concluded that the casts were a useful adjunct for management of foot deformity and associated proximal hypertonicity. The evidence that they provided, however, was highly subjective and did not provide definitive evidence, nor did they show the mechanism by which the casts function.

The early work of Duncan (1960) was also the basis for the casting strategy used by clinicians here at the University of Virginia in the late 1970s (68, 69). This work has been widely quoted in the literature, although here too the successful outcome was largely based on subjective rating. More recently Cusick (1988) provided a historical perspective of orthotic management and strategies based on her personal experience, but failed to provide the scientific evidence necessary to support her claims.

There are three groups of clinicians who have enthusiastically endorsed the benefits of orthoses for children with CO. These have included orthopaedic surgeons (19, 58, 59, Meyer, 1974, Westin and Dye, 1983), physical therapists (30, 82, Ford et al, 1986) and orthotists (56, 62, Lima, 1989).

Unfortunately none of these authors provided the objective data necessary to substantiate their claims. Their emphasis has been "this is how we do it" rather than "do these AFOs really improve function and if they do what is the mechanism?". It is evident from this brief review of the literature that there are a plethora of papers extolling the virtues of AFOs, but few have addressed the important questions of objective evaluation.

EFFECT OF ORTHOSES ON WALKING FUNCTION

There are various goals in applying ankle foot orthoses to children with spastic cerebral palsy. These can include (34): (1) correction or prevention of deformity; (2) correction of joint alignment and improved mechanics; (3) control of undesirable motions; (4) stabilization of weak muscles; and (5) reduction of abnormal muscle tone. As alluded to above, very few (if any) of these goals have been tested with the appropriate degree of scientific rigor. We describe in this section those few papers where an effort has been made to provide objective data to support the rationale for AFOs.

The effects of AFOs on walking function have been studied by some physical therapists using simple gait analysis techniques. These have included stride length and foot angle using footprint techniques (4, 29) as well as cadence, velocity or stride length. We would argue that while these studies have provided some useful information, they fall short on the type of data that should be generated. Their kinematic data are merely a measure of the *effects* of the AFOs, but say nothing about the underlying *causes* which produced the observed changes. More detailed kinetic measurements, which may *explain* the underlying causes are required. Such data can only be generated using an integrated system which incorporates 3D kinematics, EMG and force plates (Gage 1991).

Brodke et al (1989) were concerned about the potential penalty that AFOs might impart to walking ability. They studied five normal children with and without AFOs and suggested that their study provided baseline information for evaluation of new orthotic designs and materials for children. However, we seriously question whether an AFO on a normal child tells us anything about its efficacy on a population with CP where the intention is to provide stability and decrease muscle tone. Thomas et al (73) used kinematic and EMG information to evaluate 17 children with and without AFOs. They demonstrated an improvement in ankle motion for all children, and also an increase in the hip and knee motions for at least 80% of the patients. Perhaps their most interesting finding was a significant reduction in co-contraction of some muscle groups and more phasic patterns in the tibialis anterior, gastrocnemius, vastus lateralis and medical hamstrings. They therefore speculated that these changes in muscle function decreased the energy consumption required for walking, a conclusion reached by Mossberg et al (43).

The group with perhaps the most experience in studying the biomechanics of AFOs has been based at the Dundee Limb Fitting Centre in Scotland (83, 85, Meadows, 1985). They compared the dynamics of a normal child's gait to that of children who have spastic diplegia (with and without AFOs). Their data were summarized by means of a case study. For a patient without AFOs, the ground reaction force (GRF) during early stance was higher than normal. During late stance it was lower than normal, very often less than body weight, indicating an inability to generate a push-off force. The external moments (or torques) generated at the joints were generally greater in magnitude than normal. The use of an appropriate AFO reduced the high impact forces in early stance, while the vertical reaction forces in late stance were increased, indicating an ability to support body weight and to generate push-off. These changes were accomplished by a re-alignment of the GRF which led to an increased hip extensor moment.

Another British group, based at the Orthotic Research and Locomotor Assessment Unit in Oswestry, have also used the idea of combining the ground reaction force vector and joint centers to estimate joint moments (9, Butler and Nene, 1991, Butler et al, 1992). A video image of the patient at different times during stance phase was overlaid with a thin white line representing the magnitude, point of application and line of action of the ground reaction force. They provided some evidence that an appropriately prescribed AFO for 5 children with spastic hemiplegia served to reduce the large knee moments that are characteristic of these children's gait (9). One of the most appealing features of their approach is that it facilitates the fine-tuning of the orthosis during the fitting process. This opens up the possibility of a gait laboratory being used not only to assess locomotor status but also to improve function in a realtime rehabilitation context.

When control of the ankle joint is paramount, the two primary design variations include the solid (sometimes referred to as the standard or fixed) AFO, which restricts ankle and foot motion in both directions, and the hinged AFO, which allows freedom of motion in dorsiflexion (Jordan et al, 1994). Proposed advantages of the hinged AFO include more normal motion of the ankle by facilitating tibial translation during the stance phase of gait (4) with a resultant increase in gait velocity when compared to the use of a rigid AFO (37). A decreased knee extensor moment, similar to the normal pattern, was reported by Middleton et al (41) for the hinged AFO condition. However, all their conclusions are based on a single case study and cannot easily be generalized. Although Lough and Soderberg (37) studied 15 children with spastic diplegia under both the fixed and hinged AFO conditions, their results have not yet been published in full in a peer-reviewed journal, and are not yet readily available.

OUR EXPERIENCE AT THE UNIVERSITY OF VIRGINIA

Gait analysis was performed on 11 children, ages 4-11, with spastic diplegia. A fourcamera system was used to obtain 3D kinematic data, while two force plates provided ground reaction force data. An inverse dynamics approach, combining anthropometric, kinematic and force plate data, was employed to calculate joint moments and powers (Vaughan et al, 1992). Two orthoses were studied: a standard ankle foot orthosis (AFO) and a supramalleolar orthosis (SMO). A repeated measures A-B-A-C crossover design with analysis of variance (ANOVA) was used to examine the relative efficacy of the two orthotic designs.

Condition	<u>Day</u>	Activity
Α	1	Cease wearing orthosis (if already doing so)
	30	Gait analysis without orthoses, but with shoes
В	31	Begin wearing orthoses (AFOs or SMOs, as randomly assigned)
	60	Gait analysis with orthoses
Α	61	Cease wearing orthoses
	90	Gait analysis without orthoses, but with shoes
С	91	Begin wearing orthoses (SMOs or AFOs, whichever not worn in B)
	120	Gait analysis with orthoses

For each condition studied, at least three walking trials were recorded and ensemble averages of the data obtained. For the repeated measures ANOVA, a statistical significance level of p<0.05 was set. The two baseline conditions are referred to as B1 and B2 while the brace conditions are identified as AFO and SMO. The parameters studied in the statistical analysis were termporal-distance factors (stride length, cadence and velocity), sagittal plane ranges of motion (excursions) and sagittal plane moments at the hip, knee and ankle joints and maximum power generated at the ankle. The subject data averages and ensemble means and standard deviations for the temporal-distance and kinematic parameters are presented in Table 1, while the relevant kinetic parameters are summarized in Table 2. Statistically significant differences are highlighted in bold with the key at the bottom right of the tables indicating the specific comparisons.

For stride length, there was a significant difference between Baseline 1 (0.81m) and each of the other conditions: AFO (0.92), Baseline 2 (0.89m) and SMO (0.93m). The increase in stride length between Baselines 1 and 2 was unexpected but could probably be attributed to the children gaining confidence in the gait laboratory environment. This possibility is also supported by the statistically significant increase in velocity between Baseline 1 (0.87 m/s) and Baseline 2 (1.02 m/s). These differences between Baselines 1 and 2 notwithstanding, the following general conclusions may be drawn from the data presented in Tables 1 and 2 and Figures 1 through 3:

- 2 AFOs significantly increase the dorsiflexion angle at the ankle joint at initial foot strike
- 3 SMOs do not restrict the ankle range of motion
- 4 Wearing either AFOs or SMOs does not significantly change the range of motion at the knee and hip joints

¹ AFOs significantly reduce the total excursion at the ankle joint

- 5 Wearing AFOs or SMOs does not, in general, reduce the undesirable plantar flexion moment peak at 20% of the gait cycle in spastic CP gait
- 6 The plantar flexion moment generated at the ankle during push-off increases when wearing SMOs but this increase is not significant
- 8 Wearing either AFOs or SMOs does not significantly change the extension moments at the knee and hip joints
- 9 The maximum power absorbed at the ankle following initial foot contact decreases significantly when wearing AFOs
- 10 The maximum power generated at the ankle during push-off decreases significantly while wearing AFOs
- 11 The maximum power generated at the ankle during push-off is unchanged when wearing SMOs
- 12 The maximum power generated at the ankle during push-off is significantly less when wearing AFOs than wearing SMOs.

On the positive side, the AFOs functioned successfully by limiting the range of motion at the ankle, positioning the foot appropriately prior to initial foot contact, absorbing less power following initial foot contact and generating a larger ankle moment during push-off. From a negative point of view, the AFOs did not decrease the undesirable plantar flexion moment peak at 20% of the cycle and their use led to a reduction in the ankle power generated during push-off. Since joint power is defined as the dot (scalar) product of joint moment and joint angular velocity

$\mathbf{P} = \mathbf{M}$

it was the reduction in angular velocity which contributed to the decrease in power. The slope of the ankle angle curve is clearly less steep when AFOs are worn (cf. Figure 1, between 50% and 60% of the cycle).

In contrast to the AFOs - which altered the ankle joint mechanics quite noticeably - the SMOs would appear to have elicited almost no changes at all (cf Tables 1 and 2, Figures 1 through 3). This is an important finding: SMOs are widely prescribed and, because of their cosmetic appeal, are well tolerated by patients and their families. It should be stressed, however, that the primary function of SMOs is to control mid-foot instability which can lead to varus angulation of the forefoot. The parameters measured in this first phase are primarily limited to the sagittal plane, whereas the action of SMOs is likely to have an impact in the transverse and frontal planes. While these other data were gathered, they were far more variable than the sagittal plane data (cf Table 1 and 2) and so no statistical comparisons were performed.

While Meadows (1985) has argued and shown some evidence, based on a single case study, that standard AFOs can decrease the ground reaction force - and thus the undesirable plantar flexion moment - after foot contact, our general findings did not support this argument (Table 2, Figure 2). As can be seen in Figure 4, one of our subjects experienced slightly greater moments when using the orthoses. There were a few others, however, who did show a marked reduction in this early moment peak, particularly when wearing the AFO (cf Figure 5). These data suggest that there may be some scope to use the gait laboratory as a rehabilitation tool where the child would be evaluated immediately after the fitting of an orthosis to see what effect, if any, it has on altering the child's function (Butler and Nene 1991).

Even though the repeated measures cross-over design is a powerful statistical approach, the relatively small subject pool - just 11 patients - is a shortcoming in this preliminary study. There are another 5 subjects who are either still busy with the study or their data analysis has not been completed yet, but the total of 16 is still less than our original target of 24 in the first two years of the project. There were two primary reasons for this low number: only mildly involved children were selected for the study, and the experimental design - which required two months of no bracing - deterred quite a few families from participating. As part of its regular clinical service over the past four years, the Motion Analysis Laboratory at the University of Virginia has studied over 60 children with spastic cerebral palsy who wore orthoses (in addition to the 16 enrolled in the current study) at the time of their gait evaluation.

FUTURE WORK

Although the primary focus of this report has been on the effects that orthoses have on level gait, it should be recognized that this is not the only reason for their prescription. AFOs can be worn as night-time splints to prevent joint contractures and they also have to function during other activities of daily living besides walking. Six of the 11 patients were studied while ascending and descending a set of instrumented stairs. Not surprisingly, the AFOs restricted ankle motion when compared to baseline and SMOs (22 degrees versus 39 and 37 degrees respectively) and the lack of dorseflexion compromised the patients' ability to ascend stairs. Since the hinged AFO facilitates dorsiflexion while limiting plantarflexion and thus controlling the dynamic equinus, it should be included in any future studies. The Gross Motor Function Measure (GMFM) has been developed recently to document motor status and to measure change over time or as a result of the intervention in children with CP (Russell et al 1989). This measure encompasses a range of motor activities that a child might employ throughout the course of a day and includes the following five dimensions: (1) lying and rolling; (2) sitting; (3) crawling and kneeling; (4) standing; and (5) walking, running and jumping. The specific purpose of the GMFM is to quantify "how much" motor function a child is able to demonstrate without regard to the quality of the motor patterns. In conclusion, we are recommending that future studies include:

- (1) Comparison of the standard (fixed) AFO versus the hinged AFO;
- (2) Analysis of both level gait and stairclimbing;
- (3) Evaluation of overall locomotor function using the GMFM.

REFERENCES

Bleck E E. Spastic diplegia: orthopaedic management in cerebral palsy. In Clinics in Developmental Medicine (Ed E E Bleck), Lippincott, Philadelphia 1987 <u>99</u>, 282.

Brodke D S, Skinner S R, Lamoreux L W, Johanson M E, St Helen R, Moran S A, Ashley R K. Effects of ankle-foot orthoses on the gait of children. Journal of Pediatric Orthopaedics 1989 <u>9(6)</u>, 702-708.

Butler P B, Nene A V. The biomechanics of fixed ankle-foot orthoses and their potential in the management of cerebral palsied children. Physiotherapy 1991 <u>77</u>, 81-88.

Cusick B D. Managing foot deformity in children with neuromotor disorders. Physical Therapy 1988 <u>68(12</u>, 1903-1912.

Duncan W R. Tonic reflexes of the foot: their orthopedic significance in normal children and in children with cerebral palsy. The Journal of Bone & Joint Surgery 1960 $\underline{42A(5)}$, 859-868.

Eiben R M, Crocker A C. Cerebral palsy within the spectrum of developmental disabilities. Comprehensive Management of Cerebral Palsy (Eds G H Thomson et al) Grune and Statton, NY, 1983, 19-23.

Ford C, Grotz R C, Shamp J K. The neurophysiological ankle-foot orthosis. Clinical Prosthetics and Orthotics 1986 <u>10(1)</u>, 15-23.

Gage J R. Gait Analysis in Cerebral Palsy, MacKeith Press, Oxford, England. 1991.

Jordan K, Arkenau K, Baker A, Turner D, Worrell T. Effects of hinged ankle-foot orthoses on stride length, width and foot angles in a child with developmental delay and hypotonia. Physical Therapy 1994 $\underline{74(5)}$, S147.

Lima D. Overview of the causes, treatment and orthotic management of lower limb spasticity. Journal of Prosthetics & Orthotics 1989 2(1), 33-39.

Meadows C B. Mobility of the cerebral palsied child: A research report - The first 20 years: 1965-1985. Tayside Rehabilitation Engineering Services, Dundee, Scotland 1985.

Meyer P R. Lower limb orthotics. Clinical Orthopedics and Related Research 1974 102, 58-71.

Russell D J, Rosenbaum P L, Cadman D T, Gowland C, Hardy S, Jarvis S. The gross motor function measure: A means to evaluate the effects of physical therapy. Dev Medicine and Child Neurol 1989 <u>31</u>, 341-352.

Te Groen J A, Dommisse G F. Plaster casts in the conservative treatment of cerebral palsy. South African Medical Journal 1964, 502-505.

Vaughan C L, Davis B L, O'Connor J. Dynamics of Human Gait, Human Kinetics, Champaign IL 1992.

Westin G W, Dye S. Conservative management of cerebral palsy in the growing child. Foot and Ankle 1983 <u>4(3)</u>, 160-163.

aStride Length (m)bCadence (steps/min)SubjectBase 1AFOBase 2SMOS10.630.770.760.68S20.630.770.760.68S30.861.111.031.00S40.901.131.021.20S51.030.980.970.97S61.061.121.061.29S70.740.760.830.83S100.830.840.900.88S110.660.770.650.76Mean0.610.790.650.76S110.660.841.060.98 ¹⁰ 0.810.670.68 ¹⁰ 0.99 ¹¹ S20.610.730.650.77S20.6610.730.69 ¹⁴ 0.99 ¹¹ S20.610.730.660.63S30.791.371.171.18S60.900.810.840.80S30.791.371.171.18S60.970.061.0635.7S40.900.810.04S40.970.94S50.900.810.84S40.971.02S50.970.16S60.971.101.111.100.83S40.971.22S50.971.06S40.971.24		_	r	<u></u>			ı .	,			
S1 0.71 0.77 0.81 0.79 S2 0.63 0.77 0.76 0.66 S3 0.86 1.11 1.03 1.00 114.5 1	.	а					b	1			
S20.630.770.760.66S30.861.111.031.00S40.901.131.021.20S51.030.980.970.97S51.081.121.061.29S61.081.121.061.29S70.740.760.830.83S80.810.850.860.93S100.830.840.900.88S100.830.840.900.88S100.650.150.130.18Std. Dev.0.150.150.130.18SubjectS20.660.841.08S10.650.840.90S10.650.840.98S20.610.770.750.650.841.080.98S20.610.790.79S50.900.810.84S60.971.011.04S55.55.6263.7S60.970.97S70.850.870.170.161.24S55.55.62S81.001.02S101.271.001.271.001.170.860.190.870.870.860.190.180.190.121.00110.140.180.190.180.190.1											
S30.861.111.031.00S40.901.131.021.20S40.901.131.021.20S51.030.980.970.97S61.081.121.061.29S70.740.760.830.83S100.660.770.650.76S110.660.770.650.76Mean0.810.89 ¹¹ 0.89 ²¹ 0.89 ²¹ Std Dev.0.150.150.130.18CVelocity (m/s)1.101.06S20.610.730.750.60S20.610.730.750.60S20.610.730.750.60S20.610.730.750.60S40.971.221.121.28S50.900.810.840.80S60.971.101.04S50.526.623.7S60.971.101.210.180.190.150.20S10.610.726.31S101.271.001.17S110.646.3.8S110.678.82SMO0.180.19S110.160.20S120.180.19S130.180.72S140.180.72S140.635.7S528.428.2S2 <t< td=""><td></td><td></td><td></td><td></td><td></td><td></td><td></td><td></td><td></td><td></td><td></td></t<>											
S4 0.90 1.13 1.02 1.20 S5 1.03 0.98 0.97 0.97 S6 1.08 0.97 0.97 127.5 129.0 131.0 127.5 98.5 102.5 98.5 102.5 98.5 102.5 98.5 102.5 118.0 120.5 115.5 S7 0.74 0.76 0.83 0.83 0.83 0.83 0.83 0.83 0.83 0.83 0.84 0.20.5 118.0 120.5 115.0 120.5 131.0 127.5 138.0 148.0 130.0 120.5 131.0 122.5 131.0 122.5 131.0 122.5 131.0 122.5 131.0 120.0 130.0 <th< td=""><td></td><td></td><td></td><td></td><td></td><td></td><td></td><td>1</td><td></td><td></td><td></td></th<>								1			
S51.030.980.970.970.97S61.081.121.061.29S70.740.760.830.83S80.810.850.860.83S90.671.020.970.85S100.830.840.900.88S110.660.6810.6890.150.150.150.130.150.150.130.93S110.650.641.080.810.770.650.76Mean0.610.730.75S60.971.101.06S20.610.730.75S60.971.101.06S40.971.221.12S81.001.031.04S60.971.101.061.110.161.24S70.850.871.01S81.001.03S110.180.149S60.971.101.010.130.94S110.611.010.180.190.150.2933.326.2S166.436.45166.235.15222.933.322.933.326.2S166.436.45528.428.232.526.9S463.463.55528.428.23536.5		1									142.5
S61.081.121.061.29S70.740.760.830.83S80.810.850.830.83S90.671.020.970.93S100.830.840.900.88Mean0.61 ^{11,231} 0.92 ^[11] 0.99 ^[2] 0.93 ^[3] Std. Dev.0.150.150.150.130.150.150.150.160.77Std. Dev.0.150.150.16SubjectBase 1AFOBase 2S40.971.371.17S50.900.810.840.971.101.061.24S50.900.810.84S101.271.10S60.971.10S60.971.10S60.971.101.001.02 ^[10] S60.971.01S60.971.101.100.830.94S110.811.010.811.001.02 ^[21] 1.001.02 ^[21] 1.00S1166.145.06749.149.251053.657.75222.933.326.229.933.326.29493.928.428.229.526.63951.061.145.662.344.07.449.149.349.2 <t< td=""><td></td><td>1</td><td></td><td></td><td></td><td></td><td></td><td></td><td></td><td></td><td>127.5</td></t<>		1									127.5
$ \begin{array}{c c c c c c c c c c c c c c c c c c c $											98.5
$ \begin{array}{c c c c c c c c c c c c c c c c c c c $							а. -			120.5	115.5
$\begin{array}{c c c c c c c c c c c c c c c c c c c $		I								146.0	149.5
S10 S11 Mean0.63 0.66 								148.0	145.0	144.0	141.5
S10 S11 0.83 0.66 0.84 0.77 0.90 						0.93		131.0	129.5	151.0	
S11 Mean0.66 0.81 ^[12,3] 0.77 0.650.76 0.76149.5156.5152.0148.0Mean0.81 ^[12,3] 0.92 ^[1] 0.89 ^[2] 0.93 ^[3] 0.93 ^[3] 130.2 ^[6] 130.2 ^[6] Std. Dev.0.150.150.130.18129.5131.7138.0 ^[6] 130.2 ^[6] SubjectBase 1AFOBase 2SMO60.258.557.160.6S20.610.730.750.6060.258.557.160.6S30.791.371.171.1856.355.360.356.1S40.971.221.121.2857.853.754.057.7S50.900.810.840.8035.754.951.244.9S60.971.101.061.2459.556.263.769.7S70.850.871.011.041.0951.244.99.730.2S90.721.101.210.9439.547.147.648.8S101.271.001.070.9235.87.411.3S1d. Dev.0.180.190.150.209.35.87.411.9S222.933.326.219.935.921.221.421.1S370.667.469.663.831.311.725.320.9S463.463.255.757.226.9<				0.84	0.90	0.88		182.5	142.0		
Mean Std. Dev. $0.81^{[12,3]}$ $0.92^{[1]}$ $0.89^{[2]}$ $0.93^{[3]}$ Std. Dev. 0.15 0.15 0.13 0.18 CSubject AFO $Base 1$ AFO $Base 2$ SMO S1 0.65 0.84 1.08 0.98 60.2 58.5 57.1 S2 0.61 0.73 0.75 0.60 60.2 58.5 57.1 S4 0.97 1.22 1.12 1.28 57.8 53.7 54.0 S5 0.90 0.81 0.84 0.80 35.7 44.9 39.3 41.9 S6 0.97 1.10 1.04 1.09 55.5 56.2 63.7 69.7 S8 1.00 1.03 1.04 1.09 51.2 44.9 90.7 30.2 S9 0.72 1.10 1.21 0.94 54.9 46.8 45.1 49.2 S11 $0.87^{[2]}$ 1.00 $1.02^{[2]}$ 1.00 9.3 5.8 7.4 11.3 S12 22.9 33.3 26.2 19.9 35.8 7.4 11.3 S2 22.9 33.3 26.2 19.9 35.8 7.4 $11.9.7$ S3 70.6 87.4 69.6 63.8 31.3 11.7 25.3 20.9 S4L $Dev.$ 0.18 $0.72.7$ 38.9 12.7 30.4 31.6 S4 63.4 63.4 65.7 57.7 26.9 24.0 12	S11		0.66					149.5	156.5		
Std. Dev. 0.15 0.13 0.13 0.13 0.15 0.15 0.16 10.15 0.16 11.12 0.18 11.12 12.2 SMO Subject Sase 1 AFO Base 2 SMO 0.665 0.664 1.08 0.16 AFO Base 2 SMO Sign Colspan="2">Sign Colspan="2">AFO Base 2 SMO Sign Colspan="2">Sign Colspan="2"Sign Colspa= Sign Colspa="2"Sign Colspan="2">Sign Colspa= Sign Co	Mean		0.81 ^[1,2,3]	0.92 ^[1]	0.89 ^[2]	0.93 ^[3]		129.5		138.0[6]	130.2[6]
$\begin{array}{c c c c c c c c c c c c c c c c c c c $	Std. Dev.		0.15		0.13						
Subject S1Base 1AFOBase 2SMOS10.650.841.080.98S20.610.730.750.60S20.610.730.750.60S30.791.371.171.18S40.971.221.121.28S50.900.810.840.80S60.971.101.061.24S70.850.871.011.04S81.001.031.041.09S10.811.010.830.94S110.811.010.830.94Mean0.87 ^[2] 1.001.02 ^[2] S166.148.067.2S166.148.067.2S222.933.326.2S222.933.326.2S463.464.357.1S528.428.232.5S662.344.0S166.148.0S528.428.2S222.933.3S463.464.3S749.143.5S662.344.0S749.143.5S662.344.0S749.143.5S951.061.3S850.543.1S951.061.3S850.5S1053.6S2.750.6S2.750.6 <td></td> <td></td> <td></td> <td>···</td> <td>· • • • • • • • • • • • • • • • • • • •</td> <td></td> <td></td> <td>L</td> <td></td> <td></td> <td></td>				···	· • • • • • • • • • • • • • • • • • • •			L			
Subject S1Base 1AFOBase 2SMOS10.650.841.080.98S20.610.730.750.60S20.610.730.750.60S30.791.371.171.18S40.971.221.121.28S50.900.810.840.80S60.971.101.061.24S70.850.871.011.04S81.001.031.041.09S10.811.010.830.94S110.811.010.830.94S110.811.010.830.94Mean0.87 ^[2] 1.001.02 ^[2] S166.148.067.2S222.933.326.2S1222.933.326.2S463.464.357.1S528.428.232.5S662.344.061.0S749.143.551.5S662.344.0S749.143.5S662.344.0S749.143.5S662.344.0S749.143.5S662.344.0S749.143.5S662.344.0S749.143.5S951.061.3S1053.655.7S750.6S2.7 </td <td></td> <td>с</td> <td>·</td> <td>Velocit</td> <td>v (m/s)</td> <td></td> <td>ь _</td> <td></td> <td></td> <td>n (degrees)</td> <td></td>		с	·	Velocit	v (m/s)		ь _			n (degrees)	
S1 0.65 0.84 1.08 0.98 S2 0.61 0.73 0.75 0.60 S3 0.79 1.37 1.17 1.18 S4 0.97 1.22 1.12 1.28 S5 0.90 0.81 0.84 0.80 S6 0.97 1.01 1.06 1.24 S7 0.85 0.87 1.01 1.04 S8 1.00 1.03 1.04 45.4 Mean $0.87^{[2]}$ 1.00 $1.02^{[2]}$ S1 66.1 48.0 67.2 S1 66.1 48.0 67.2 S1 22.9 33.3 26.2 S4 63.4 64.3 57.1 S5 28.4 28.2 32.5 S6 62.3 44.0 61.0 S7 49.1 43.5 51.5 49.1 43.5 51.5 49.1 43.5 51.5 88 50.5 43.1 49.1 43.5 51.5 88 50.5 43.1 51.0 61.3 58.8 51.5 43.1 43.3 54.5 27.0 7.7 28.3 51.5 48.8 22.1 6.6 23.7 50.6 52.7 50.6 52.7 50.6 52.7 50.6 52.8 55.6	Subject	-	Base 1			SMO	u				
$\begin{array}{c ccccccccccccccccccccccccccccccccccc$											
$\begin{array}{c ccccccccccccccccccccccccccccccccccc$											
$\begin{array}{c ccccccccccccccccccccccccccccccccccc$											
$\begin{array}{c ccccccccccccccccccccccccccccccccccc$											
$\begin{array}{c ccccccccccccccccccccccccccccccccccc$											
S7 0.85 0.87 1.01 1.04 45.4 44.4 49.9 47.0 S8 1.00 1.03 1.04 1.09 39.5 47.1 47.6 48.8 S10 1.27 1.00 1.17 0.96 39.5 47.1 47.6 48.8 S10 1.27 1.00 1.17 0.96 39.5 47.1 47.6 48.8 Mean $0.87^{[2]}$ 1.00 $1.02^{[2]}$ 1.00 93.5 47.1 47.6 48.8 S10 $0.87^{[2]}$ 1.00 $1.02^{[2]}$ 1.00 9.3 5.8 7.4 11.3 Mean 0.18 0.19 0.15 0.20 9.3 5.8 7.4 11.3 S2 2.9 33.3 26.2 19.9 35.0 35.0 13.9 28.1 19.7 S2 22.9 33.3 26.2 19.9 35.0 13.3 11.7 25.3 20.9 S4 63.4 64.3 57.1 68.7 25.8 10.9 20.0 31.4 S5 28.4 28.2 32.5 26.9 24.0 12.4 29.4 35.1 S6 62.3 44.0 61.0 72.7 7.7 28.3 35.1 S7 49.1 43.5 51.5 48.8 22.1 6.6 23.7 20.6 S10 53.6 55.7 57.2 62.0 23.1 10.1 21.3 20.8 S11 62.0 $47.$											
$\begin{array}{c c c c c c c c c c c c c c c c c c c $											
$\begin{array}{c c c c c c c c c c c c c c c c c c c $											
$\begin{array}{c c c c c c c c c c c c c c c c c c c $											
S11 0.81 1.01 0.83 0.94 Mean $0.87^{[2]}$ 1.00 $1.02^{[2]}$ 1.00 Std. Dev. 0.18 0.19 0.15 0.20 SubjectBase 1AFOBase 2SMOS1 66.1 48.0 67.2 63.1 S2 22.9 33.3 26.2 19.9 S3 70.6 87.4 69.6 63.8 S4 63.4 64.3 57.1 68.7 S5 28.4 28.2 32.5 26.9 S6 62.3 44.0 61.0 72.7 S6 62.3 44.0 61.0 72.7 S9 51.0 61.3 58.8 71.3 S10 53.6 55.7 57.2 62.0 S11 62.0 47.8 56.8 60.5 Mean 52.7 50.6 52.8 55.6											
Mean Std. Dev. $0.87^{[2]}$ 1.00 $1.02^{[2]}$ 1.00 Std. Dev. 0.18 0.19 0.15 0.20 $Std. Dev.$ 0.18 0.19 0.15 0.20 $Subject$ S1 S2 S2 S2 S3 S4 S5 S5 S5 S6 S6 S6 S1.0 AFO $Base 2$ $Base 1$ SMO $Std. Dev.$ $Base 1$ AFO $Base 2$ $Base 2$ SMO $S1$ S2 S2 S2 S2 S3 S4 S5 S5 S5 S6 S6 S6 S8 S0.5 S1.0 66.1 48.0 48.0 67.2 63.1 66.1 48.0 67.2 63.3 57.1 68.7 57 49.1 43.5 51.5 51.5 51.5 51.5 51.5 51.5 51.5 51.6 62.3 43.1 43.5 51.5 51.5 51.6 48.8 52.7 50.6 52.8 55.6 55.7 57.2 62.0 51.1 62.0 47.8 56.8 60.5 60.5 52.8 55.6 43.1 43.3 54.5 23.6 51.6 49.3 49.2 51.0 51.0 51.3 51.5 51.5 51.5 51.5 51.5 51.5 51.5 51.5 51.5 51.5 51.5 51.5 51.6 49.2 51.0 51.0 51.3 52.7 50.6 52.8 55.6 49.2 51.6 51.7 57.2 57.2 62.0 23.1 10.1 21.3 $24.6^{[4]}$ $25.4^{[5]}$ Mean 52.7 50.6 52.8 55.6 55.6 52.8 55.6 52.8 55.6 49.2 51.6 51.3 51.5											
Std. Dev. 0.18 0.19 0.15 0.20 Std. Dev. 0.18 0.19 0.15 0.20 Std. Dev. 0.18 0.19 0.15 0.20 Std. Dev. 0.18 0.19 0.15 0.10 Subject AFO Base 1 AFO Base 2SMOS1 66.1 48.0 67.2 63.1 $S2$ 22.9 33.3 26.2 19.7 22.9 33.3 26.2 SMO 35.0 66.1 48.0 67.2 63.4 68.7 22.9 33.3 26.2 SMO 31.3 11.7 25.3 20.5 12.0 21.4 21.4 $S3$ 70.6 87.7 28.4 28.2 32.5 51.0 62.3 44.0 61.0 72.7 38.9 12.7 30.4 35											
$\begin{array}{c c c c c c c c c c c c c c c c c c c $]									
SubjectBase 1AFOBase 2SMOS1 66.1 48.0 67.2 63.1 35.0 13.9 28.1 19.7 S2 22.9 33.3 26.2 19.9 20.5 12.0 21.4 21.1 S3 70.6 87.4 69.6 63.8 31.3 11.7 25.3 20.9 S4 63.4 64.3 57.1 68.7 25.8 10.9 20.0 31.4 S5 28.4 28.2 32.5 26.9 24.0 12.4 29.4 35.1 S6 62.3 44.0 61.0 72.7 38.9 12.7 30.4 31.6 S7 49.1 43.5 51.5 48.8 22.1 6.6 23.7 20.6 S8 50.5 43.1 43.3 54.5 27.0 7.7 28.3 35.1 S9 51.0 61.3 58.8 71.3 23.6 14.1 26.0 22.0 S10 53.6 55.7 57.2 62.0 23.1 10.1 21.3 20.8 S11 62.0 47.8 56.8 60.5 15.8 19.3 16.6 21.4 Mean 52.7 50.6 52.8 55.6 $26.1^{[1]}$ $11.9^{[1.4.5]}$ $24.6^{[4]}$ $25.4^{[5]}$	Sid. Dev.	I	0.18	0.19	0.15	0.20		9.3	5.8	7.4	11.3
SubjectBase 1AFOBase 2SMOS1 66.1 48.0 67.2 63.1 35.0 13.9 28.1 19.7 S2 22.9 33.3 26.2 19.9 20.5 12.0 21.4 21.1 S3 70.6 87.4 69.6 63.8 31.3 11.7 25.3 20.9 S4 63.4 64.3 57.1 68.7 25.8 10.9 20.0 31.4 S5 28.4 28.2 32.5 26.9 24.0 12.4 29.4 35.1 S6 62.3 44.0 61.0 72.7 38.9 12.7 30.4 31.6 S7 49.1 43.5 51.5 48.8 22.1 6.6 23.7 20.6 S8 50.5 43.1 43.3 54.5 27.0 7.7 28.3 35.1 S9 51.0 61.3 58.8 71.3 23.6 14.1 26.0 22.0 S10 53.6 55.7 57.2 62.0 23.1 10.1 21.3 20.8 S11 62.0 47.8 56.8 60.5 15.8 19.3 16.6 21.4 Mean 52.7 50.6 52.8 55.6 $26.1^{[1]}$ $11.9^{[1.4.5]}$ $24.6^{[4]}$ $25.4^{[5]}$		r						·		<u> </u>	
S1 66.1 48.0 67.2 63.1 35.0 13.9 28.1 19.7 S2 22.9 33.3 26.2 19.9 20.5 12.0 21.4 21.1 S3 70.6 87.4 69.6 63.8 31.3 11.7 25.3 20.9 S4 63.4 64.3 57.1 68.7 25.8 10.9 20.0 31.4 S5 28.4 28.2 32.5 26.9 24.0 12.4 29.4 35.1 S6 62.3 44.0 61.0 72.7 38.9 12.7 30.4 31.6 S7 49.1 43.5 51.5 48.8 22.1 6.6 23.7 20.6 S8 50.5 43.1 43.3 54.5 27.0 7.7 28.3 35.1 S9 51.0 61.3 58.8 71.3 23.6 14.1 26.0 22.0 S10 53.6 55.7 57.2 62.0 23.1 10.1 21.3 20.8 S11 62.0 47.8 56.8 60.5 15.8 19.3 16.6 21.4 Mean 52.7 50.6 52.8 55.6 $26.1^{[1]}$ $11.9^{[1.4.5]}$ $24.6^{[4]}$ $25.4^{[5]}$.	е					f				
S222.933.326.219.920.512.021.421.1S370.687.469.663.831.311.725.320.9S463.464.357.168.725.810.920.031.4S528.428.232.526.924.012.429.435.1S662.344.061.072.738.912.730.431.6S749.143.551.548.822.16.623.720.6S850.543.143.354.527.07.728.335.1S951.061.358.871.323.614.126.022.0S1053.655.757.262.023.110.121.320.8S1162.047.856.860.515.819.316.621.4Mean52.750.652.855.626.1 ^[1] 11.9 ^[1.4,8] 24.6 ^[4] 25.4 ^[5]											
$\begin{array}{cccccccccccccccccccccccccccccccccccc$		(
$ \begin{array}{c ccccccccccccccccccccccccccccccccccc$											
$\begin{array}{c ccccccccccccccccccccccccccccccccccc$											20.9
$ \begin{array}{c ccccccccccccccccccccccccccccccccccc$											
$ \begin{array}{c ccccccccccccccccccccccccccccccccccc$											
$ \begin{array}{c ccccccccccccccccccccccccccccccccccc$											
$ \begin{array}{c ccccccccccccccccccccccccccccccccccc$										23.7	20.6
$ \begin{array}{c ccccccccccccccccccccccccccccccccccc$									7.7	28.3	35.1
$ \begin{array}{c ccccccccccccccccccccccccccccccccccc$								23.6	14.1		
S11 62.0 47.8 56.8 60.5 15.8 19.3 16.6 21.4 Mean 52.7 50.6 52.8 55.6 $26.1^{[1]}$ $11.9^{[1,4,5]}$ $24.6^{[4]}$ $25.4^{[5]}$					57.2	62.0		23.1			
Mean 52.7 50.6 52.8 55.6 $26.1^{(1)}$ 11.9 ^(1,4,5) 24.6 ⁽⁴⁾ 25.4 ⁽⁵⁾	S11		62.0	47.8	56.8						
	Mean	ſ	52.7		52.8			26.1 ^[1]			
	Std. Dev.	Ì	15.1								
								·	i <u></u> i <u></u>		

Table 1

Subject data averages and ensemble means and standard deviations for selected temporal-distance and kinematic parameters. Key at right is for statistically significant differences from a repeated measures ANOVA with p < 0.05.

[1] Base 1 vs AFO

[2] Base 1 vs Base 2

[3] Base 1 vs SMO

[4] AFO vs Base 2

[5] AFO vs SMO[6] Base 2 vs SMO

	a	Max H	in Extensio	n Moment (Nm/ka)	7	L				
Subject	_	Base 1	AFO	Base 2			b	Max Kne	e Ext. Mor	nent Peak	l (Nm/kg)
S1		0.85	0.75	0.98	<u>SMO</u>	-		Base 1	AFO	Base 2	SMO
S2		1.27	0.55	1.27	0.92			0.31	0.51	0.28	0.26
S3		0.96	1.18	0.70	0.66			0.68	0.50	0.49	0.54
S4		0.88	1.18	0.70	0.99	1		0.27	0.75	0.30	0.26
S5		1.45	1.38	1.35	1.33			0.35	NA	0.15	NA
S6		1.91	1.87	1.35	0.79			0.91	0.14	0.53	0.46
S7		0.74	0.72	0.95	2.58			0.32	0.39	0.68	0.78
S8		1.07	1.49	1.85	0.85			0.27	0.68	0.23	0.67
S9	l	1.03	1.05	1.19	1.33			0.22	0.26	0.30	0.55
S10		1.03	2.10		1.04			0.05	0.45	NA	0.18
S11		0.64	1.18	1.34	1.32	ļ		0.73	0.36	0.62	0.14
Mean	ŀ	1.07	1.18	0.56	0.86	4		0.32	0.40	0.40	0.22
Std. Dev.	ł	0.36	0.47		1.15	4	i	0.40	0.44	0.40	0.41
	L	0.00	0.47	0.39	0.53	J	l	0.26	0.18	0.18	0.22
	сſ	Max Kne	e Ext. Mom	ent Peak 2	(Nm/ka)	1	d	Mon Art	(- Ol		
Subject	-	Base 1	AFO	Base 2	SMO	1	a	Max Ank	ie Pia. Mon	nent Peak 1	
S1	F	0.10	0.38	0.19	0.28	1		Base 1 0.78	AFO	Base 2	SMO
S2		0.48	0.38	0.41	0.62				0.44	0.71	0.75
S3	- (0.29	0.39	0.39	0.31	l		0.44	0.78	0.47	0.77
S4		0.22	0.33	0.27	0.46			0.23	0.45	0.39	0.57
S 5		0.34	0.05	0.42	0.53			0.48	0.09	0.56	0.29
S6		0.27	0.70	0.63	0.33			0.91	0.86	0.68	0.73
S7		0.28	0.22	0.42	0.20		[0.59	1.01	0.69	1.05
S8		0.09	0.15	0.15	0.40			0.91 1.12	0.94	1.12	1.49
S9		0.13	0.35	0.27	0.22		}		1.29	1.09	1.35
S10		0.42	0.25	0.23	0.14			1.08 1.12	0.80	1.29	0.57
S11		0.24	0.18	0.24	0.34		1	0.98	0.85	0.81	0.80
Mean		0.26	0.31	0.33	0.37		H	0.98	1.08	0.89	0.91
Std. Dev.	Γ	0.12	0.17	0.14	0.15			0.31	0.78	0.79	0.84
	_						Ĺ	0.01	0.34	0.28	0.35
	e [Max Ankl	e Pla. Mom	ent Peak 2	(Nm/ka)		fΓ	Max Ankl	Push off	Power Gen.	(AA(A)-)
Subject		Base 1	AFO	Base 2	SMO		•	Base 1	AFO		
S1		0.86	0.66	0.70	0.78		-	1.13	0.87	Base 2 1.10	SMO
S2		0.84	1.09	0.80	1.05			0.79	1.01	0.94	1.30
S3	{	0.71	0.82	0.80	0.88			1.50	1.84	0.94 2.73	0.88
S4		0.97	0.99	0.97	0.93			1.59	1.30	2.73	1.84
S5		1.02	1.10	0.97	0.90			1.60	0.55		2.07
S6		0.91	1.17	0.94	1.12			1.39	1.34	1.06 2.00	1.25
S 7		0.65	1.22	0.82	0.87			1.07	0.71	2.00	2.15
S8		0.78	0.91	0.85	0.77			1.21	0.99	1.22	1.21
S9		0.92	0.90	0.69	0.99]	0.97	1.28	1.49	1.86
S10		0.70	1.35	1.01	1.01			1.95	0.73	2.06	1.52
S11	L	0.98	0.98	1.00	1.07			1.66	1.01	2.06	0.98
Mean		0.85	1.02 ^[4]	0.87[4]	0.94		F	1.35	1.05 ^[4,5]	1.50 ^[4]	2.96
Std. Dev.		0.13	0.19	0.12	0.11		\vdash	0.35	0.37	0.54	1.64 ^[5]
								0.00	0.07	0.54	0.61

Table 2

Subject data averages and ensemble means and standard deviations for selected kinetic parameters. Key at right is for statistically significant differences from a repeated measures ANOVA with p < 0.05.

[1] Base 1 vs AFO

Ankle Range of Motion (degrees)

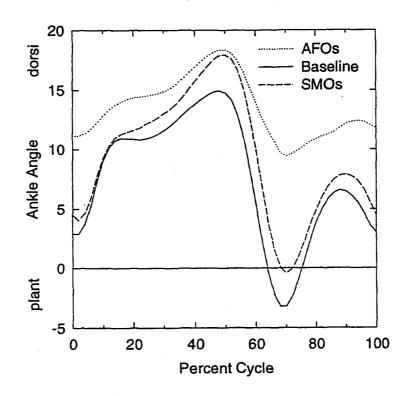


Figure 1. Ensemble averages for sagittal plane ankle angle based on 11 spastic diplegic subjects

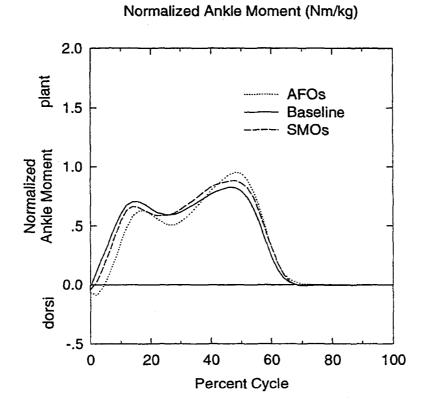
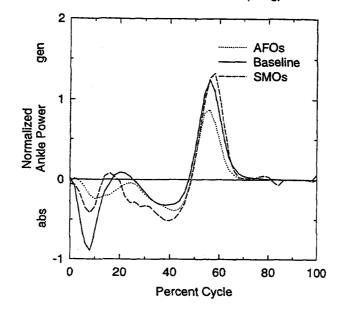
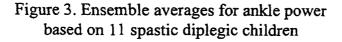


Figure 2. Ensemble averages for sagittal plane ankle moment based on 11 spastic diplegic children

Normalized Ankle Power (W/kg)





Normalized Ankle Moment (Nm/kg) Subject S8

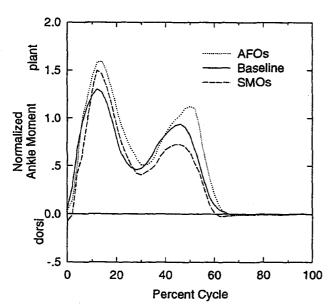


Figure 4. Ensemble averages of sagittal plane ankle moment for subject S8.

2.0 plant AFOs 1.5 Baseline SMOs Normalized Ankle Moment 1.0 .5 0.0 dorsi -.5 0 20 40 60 80 100 Percent Cycle

Normalized Ankle Moment (Nm/kg) Subject S4

Figure 5. Ensemble averages of sagittal plane ankle moment for subject S4.

THE INTEGRATION OF ORTHOTIC TREATMENT IN AN OVERALL MANAGEMENT PROGRAMME

Jules Becher MD

INTRODUCTION

Traditionally, medical disciplines are cure-oriented. The interventions are based on the medical model: identification of symptoms of illness, putting a diagnosis, identifying pathological processes and detection of the aetiology lead to a treatment to cure a patient. If cure is not possible, a care-oriented approach is necessary. Rehabilitation medicine focuses not only on how to cure a patient, but also on the consequences of the disease for the person's daily life. The ICIDH (International Classification of Impairment, Disability and Handicaps) offers a framework for a methodological approach for intervention (Bennekom et al, 1995). Impairments are the consequences of a disease on organ level. A special interest of the rehabilitation medicine is the relation between impairments and disability: which impairments give high risk on disabilities? The third stage of consequences of a disease is the level of handicap, defined as the social consequences of disabilities, for instance lack of social relations, lack of work. The final aim in the rehabilitation treatment is to diminish or to prevent handicap, by prevention of lessening disabilities and relevant impairments.

This concept is only elaborated for adults. The interest for functional assessment at the level of disabilities in child rehabilitation is increasing (15, Feldman and Haley, 1990).

PRINCIPLES OF INTERVENTION IN CHILD REHABILITATION

Intervention in child rehabilitation is complicated by the facts that children have to grow and to develop. Also, the parents and the child have to deal with the handicap.

Intervention is possible at three levels, the three Rs of intervention: the level of Remediation, of Redefinition and Re-education. These levels are hierarchical.

Remediation

Remediation aims to change the conditions of the child into normal. In regard to Cerebral Palsy (CP) remediation concerns pre-, peri-, and postnatal care to prevent serious brain damage. Even with neurological pathology at the age of one year, cure into normal is possible in regard to the motor impairments. It could be possible that orthotic treatment in young children (0-4 years old) add some benefit in preventing deformity, providing a base of support and facilitating training motor skills. Randomised Clinical Trials are needed to evaluate the effects of orthotic treatment. Physiotherapy only (according to the Bobath method) was not effective in regard to improvement of skills (Palmer et al, 1988)

Redefinition

Redefinition concerns the process of changing the expectation of the parents and changing the interpretation of behaviour of the child. Redefinition for the parents

means coping with the handicap. In cerebral palsied babies, the process of redefinition is complicated by the lack of appearance of handicap of the baby. When remediation couldn't be achieved, the process of redefinition must start. Early detection of serious brain damage in CP children is possible with the aid of diagnostic investigations in the first three months after birth. The physician had to discuss the changed expectations about the development of the child with the parents, for instance by showing CT or MRI scans. Also, information had to be given about the short term natural course and treatment possibilities. A delicate problem is to support parents with their feelings of guilt about the handicap of the baby.

In that way, a change in the interpretation of behaviour of the child can be achieved (for instance the excessive extension pattern of a CP baby is easily interpreted as an ability to stand).

Redefinition for the child means coping with the frustrations about the disabilities. This process starts at the developmental age of 1.5 year! It is important that the parents give information to their child about the nature of the illness (a stable condition, caused at birth; the child has no blame; therapy is required to improve abilities, but does not cure).

Re-education

Re-education concerns adaptation of behaviour of the parents to the conditions of the child. The main therapeutic goal in the first year is to support the parents in their perceived problems to guide them to re-education. The feeling that you help them "to do everything possible for the development of their child" makes that possible. After one year, the developmental possibilities of the child are getting clear, so gradually information over associated disabilities can be introduced. When the process of redefinition of the parents breaks down in the first years, the process of re-education will not be successful. That can be the true reason for therapeutic failures.

Effective re-education means adaptation to the conditions of the child (and not turning the conditions into normal), home programmes for daily care and education are obligatory. In most CP children, orthotic treatment is a component of the re-education programme.

GOALS OF TREATMENT

Two goals of treatment in CP children can be distinguished:

- 1 Prevention of secondary impairments
- 2 Improvement of abilities.

Prevention of secondary impairments concerns mainly prevention or treatment of musculocutaneus deformities. Specially, secondary impairment of spine and hip function can cause increase of disabilities.

Improvement of abilities is the main task in the treatment of CP children, not only in regard to the gross motor skills, but also to the fine and perceptual motor skills, social, mental, emotional and communicational skills. There is too much to do in too little

time! Special risks in the education programme of a CP child are the lack of experience in daily life, for instance, shopping with the parents, playing in a recreation ground with other children), mental under-estimation (particularly in dyskinetic or atactic children) and an unbalanced rehabilitation programme. In the first three years of life, the development of motor abilities and development of communicational skills have the priority. In the age of a toddler, social abilities and fine motor abilities had to develop. At the school age, development of cognitive functions, perceptual motor skills and self esteem is important. Priority planning in the time is a basic condition for a balanced rehabilitation programme (5).

Orthotic treatment and orthopaedic surgery are supplementary to each other in the treatment of impairments and disabilities. As orthopedic surgery is a serious event for a child, vulnerable developmental periods had to be taken into account in planning surgery.

TEAMWORK

Child rehabilitation is teamwork. A team needs a coordinator. Team members must be familiar with each other's skills. Discussing problems at the level of disabilities will overcome the problems of differences in jargon. Goals of treatment should be set on the level of abilities. The way of making decisions by the team must be clear. Disagreement must be discussed in the team, never with the parents nor the child. Although the orthotist isn't mostly a member of the rehabilitation team, agreement between the physician, the physiotherapist and the parents are requirements for the orthotist to manufacture an orthosis which reached the proposed goal.

THE PROCESS OF PRESCRIPTION

The process of prescription of an orthosis starts with the observation of a problem, at the level of impairment of disability, in relation to the total level of abilities and the functional prognosis. The burden of the family must also be considered before proposing the use of an orthosis. Working in a rehabilitation team, the observed problem and the proposed orthosis should be discussed only with the members of the team who deal with the observed problem. The next step is explanation of the observed problem to the parents and, if possible, to the child. After that, a therapeutic intervention, such as an orthosis, can be proposed. The following subjects must be explained:

- the expected effect of the orthosis

- the advantages and disadvantages in use (for instance facilitating standing and walking with support, hampering crawling and independency in clothing)

- the use of the orthosis: donning and doffing, time of use, places at risk for pressure sores

- the moment and way of evaluation of the effect of the orthosis

- alternatives for treatment (for instance surgery, serial casting, the natural history).

The process of manufacturing the orthosis can start after permission of the parents (and the child). In the Netherlands, the physiatrist and/or the physiotherapist is used to have a joint consultancy with the orthotist. Appointments have to be made about the following aspects:

- the purpose of the orthosis in terms of effect on impairment and disability
- the medical requirements in detail
- the choice of material, the lock and the design.

In manufacturing the model of plaster, the orthotist or an assistant must be familiar with the inhibiting techniques to reduce spasticity and to stretch muscles. The orthosis must be controlled on fitting and functioning in trial. After giving the finishing touch to the orthosis, the parents are instructed at delivery on use, pulling on and off and appointments are made about evaluation or replacement. The therapists must be instructed in use and had to report about the effect. To enhance the participation, the child can be asked to use self-made calendars for registration, the use and discomfort of the orthosis.

CONCLUSION

Integration of orthotic treatment in the rehabilitation programme is a complicated issue. This article did focus on the general conditions required for successful (orthotic) intervention and has given attention to the process of prescription, fitting, delivery and evaluation of an orthosis. The way of working presented seems self-evident. However, looking at daily practice, it is hard to fulfil all conditions mentioned. Also, the knowledge required for a detailed medical and biomechanicl prescription and proper manufacturing is considerable. Finally, the conclusion must be that orthotic management in cerebral palsied children has to be team work and the team, giving attention to all aspects of development, has to be specialised in child rehabilitation.

REFERENCES

Bennekom van C A M, Jelles F, Lankhorst G J. Rehabilitation Activities Profile: the ICIDH as a framework for a problem-oriented assessment method in rehabilitation medicine. Disabil Rehabil 1995 <u>17</u>, 169-75.

Feldman A B, Haley S M. Concurrent and construct validity of the pediatric evaluation of disability inventory. Phys Ther 1990 <u>70</u>, 602-610.

Palmer F B, Shapiro B K, Wachtel R C. The effects of physical therapy on cerebral palsy. A controlled trial in infants with spastic diplegia. New England J Med 1988 318, 803-808.



Price

Members of ISPO- \$20.00Non-members- \$30.00